

# BIT-INDUCED FEAR: A welfare problem & safety hazard for horse and rider

W. Robert Cook FRCVS, PhD<sup>1</sup>

## Part II: BITS AND DISEASES

Table I provides a summary of the 40 or more bit-induced diseases that I have recognized in the last ten years, the evidence for which has already been published. This is not the place to repeat the evidence but, by way of exemplifying the problems that bits cause, I will comment on two of the major problems, the headshaking syndrome and airway obstruction. In Part III I will focus on another problem which is the most serious of all and hence the title of this trilogy...the problem of bit-induced fear.

<b>BIT-INDUCED DISEASES</b>		
#	<b>ORAL &amp; DENTAL DISEASES</b>	<b>NOTES</b>
1	Sore mouth (gingivitis) and lips (dermatitis)	Dermatitis can lead to sarcoid formation at the corner of the mouth
2	Mandibular periostitis	'Bone spurs' on the bars of the mouth (very common)
3	Hypersalivation at exercise	'Drooling of 'ropes' of saliva, sometimes admixed with air so that the saliva foams
4	Sequestrum formation on the bars	Rare
5	Laceration of the tongue	Including amputation of the apex of the tongue
6	Mandibular fracture	For example, from a loose horse treading on a trailing rein
7	Temporo-mandibular joint disorders	Evidence questionable but frequently suspected (Cook 2006)
8	Dental pain (toothache)	From bit trauma to unerupted vestigial wolf teeth in the lower jaw
9	Dental pain (toothache)	From bit trauma to wolf teeth in upper jaw (less of a problem than #7)
10	Erosion of canine teeth	In the gelding.
11	Dental pain (toothache)	In the mare, from trauma to vestigial canine teeth in the lower jaw
12	Erosion of premolars	Especially the first cheek tooth in lower jaw (common)
13	Shedding of premolars in lower jaw	Especially the first cheek tooth
14	Alveolar periostitis	Sequel to paradontal disease and shedding
<b>RESPIRATORY DISEASES</b>		
1	Elevation of the soft palate	From multiple bit causes (e.g. poll flexion, open mouth, gagging reflexes): Causing asphyxia
2	Dorsal displacement of the soft palate	Sequel to #1. Causing asphyxia and suffocation at exercise
3	Dynamic collapse of the nasopharynx	Sequel to #1 & #2. Additional cause of asphyxia
4	Epiglottal entrapment	Sequel to an open mouth allowing air to enter the oropharynx and exposing the ventral epiglottal mucosa to the negative pressure of inspiration
5	Dynamic collapse of the larynx	Sequel to obstruction of nasopharynx caused by #s 1-4 above
6	'scabbard' trachea	Long-term sequel to obstruction of nasopharynx and larynx at exercise
7	Hypoxaemia at exercise	Low blood oxygen from upper airway obstruction
8	Asphyxia-induced pulmonary edema	So-called EIPH or 'bleeding': caused by any upper airway obstruction
9	Small airway disease (bronchiolitis)	Sequel to upper airway obstruction
10	Pulmonary congestion	Sequel to upper airway obstruction
11	Synchronous diaphragmatic flutter	'Thumps' or spasm of the diaphragm, perhaps triggered by dehydration

<sup>1</sup> Professor of Surgery Emeritus, Tufts University, Cummings School of Veterinary Medicine. USA  
Chairman, The Bitless Bridle Inc.  
Current address: 206, Birch Run Road, Chestertown, MD 21620 USA  
Tel: (410) 778 9005 E-mail: drcook@bitlessbridle.com

<b>NERVOUS DISEASES</b>		
1	Scared horse syndrome (extremely common)	'spooky' 'hot' and apprehensive; caused by oral pain or anticipation of such pain (stress)
2	<b>Trigeminal neuralgia (common)</b>	The 'headshaking' syndrome
3	Agrophobia	Unwilling to leave the barn
4	Many character changes bordering on the psychological but explainable as physiological responses to oral pain	Behavioral changes triggered by oral pain are not 'vices' but normal physiological responses, though they are often inconvenient for the rider and the source of accidents to horse and rider
<b>CARDIOVASCULAR DISEASES</b>		
		evidence uncertain
1	<b>sudden death from heart failure</b>	Sequel to pulmonary congestion
2	<b>Atrial fibrillation</b>	Sequel to dehydration
<b>MUSCULO-SKELETAL DISEASES</b>		
		sequelae to bit-induced oral pain, hypoxemia and premature fatigue
1	<b>'bridle lameness'</b>	Sequel to oral pain
2	Incoordination	Mimics EPM and may be mistaken for
3	Sprains and strains of neck & back	Especially from bit-induced over-bending ('Rollkur')
4	Tendonitis & desmitis	Sprained tendons & ligaments ('breakdowns')
5	Arthritis	Sprained joints
6	Metacarpal osteitis	'sore shins' (from a horse being constantly on the forehand)
7	Occipital exostoses	sequel to over-bending ('Rollkur')
8	Fractures of the base of the skull	From rearing and falling over backwards
9	Fracture of paramastoid process	From rearing
10	Broken back	Sequel to rearing and falling: leading to euthanasia
11	Carpitis	'chipped knees'
12	Sesamoiditis	Chip fractures of sesamoid bones
13	Long bone fractures	Sequel to falls caused by premature fatigue and hypoxemia: leading to euthanasia
<b>METABOLIC DISEASES</b>		
1	<b>Dehydration</b>	Sequel to sore mouth and disinclination to drink
2	<b>Rhabdomyolysis ('tying-up')</b>	Sequel to stress (i.e. oral pain or anticipation of oral pain)

**TABLE I: A list of over 40 diseases caused either directly or indirectly by the bit.** Those 22 diseases that, in my experience, are specific to the bit and not caused by any other factor are printed in red. The first three respiratory diseases are ones that, until now, have always been classified as of unknown cause. Many of the other diseases are most frequently caused by the bit (e.g. the scared horse syndrome) but they are not exclusively caused by the bit. The five items printed in blue are diseases for which the evidence for including them as being even partially bit-induced is flimsy. They are added in the belief that a causative connection may nevertheless be present and, therefore, these suggestions are put forward as possible candidates. This approach is considered to be justified as most of the 'blue' diseases are currently recognized as being of unknown cause anyway and proposals for plausible causative factors are needed.

### **Bit-Induced Headshaking Syndrome.**

Twenty eight years ago, I published a four-part article on headshaking in the horse (Cook 1979a, b; 1980a, b). This was the first major contribution to the literature on a problem that had long been a source of distress to the horse, not to mention owners and veterinarians. In this series of articles, I described the history and clinical signs of the syndrome and recommended a protocol for the investigation and clinical examination of affected horses. But, in spite of having spilt so much ink, I made it clear that I did not know the cause of the problem and, therefore, could offer no cure. Thirteen years later I published some further ideas on this problem, though still without claiming victory on the vital question of cause and cure (Cook 1992). By this time, many other workers had been

researching the problem but none had been able to recommend an effective cure.

Over the last decade, however, my continuing research has provided convincing evidence which fails to falsify the hypothesis that the bit is the most common cause of the headshaking syndrome and that the syndrome, when bit-induced, is a manifestation of trigeminal neuralgia (Cook 1999b, 1999c, 2000, 2002a, and 2003).<sup>2</sup> As the syndrome has been such a recalcitrant problem in the past, it is good to be able to report that here, at last, is an aetiology that is consistent with the evidence. Such a conclusion is supported by the substantial success in treating the syndrome by removing the proposed cause...the bit. The results provide further compelling evidence that the bit is not only an inefficient method of communication but also one that is physiologically contraindicated.

In the past, many different diseases have been proposed as the hypothetical cause of the seven or more clinical signs that have been thought to define the headshaking syndrome (Cook 2003). Sadly, none of these varied explanations have been supported by a convincing resolution of the syndrome following therapy for any one of the proposed diseases.

The syndrome is named after its most prominent and disturbing sign; the persistent, violent, and spasmodic, vertical tossing of the head at exercise. Other characteristic signs include but are not limited to muzzle rubbing, sneezing, snorting, and a general hypersensitivity and shyness when handled around the mouth, face and ears (see Table I in Part III of this article). High ambient temperatures exacerbate these signs and so also does bright sunlight. For this reason, headshaking horses are often described as being photophobic.

These apparently disconnected signs can now be economically explained on the grounds that, collectively, they are all signs of facial neuralgia. Instead of having to propose a multiplicity of causes, none of which are entirely convincing, this is a unifying hypothesis that explains all the symptoms of the syndrome under one cause. Happily, the hypothesis withstands the crucial test in that removal of the proposed cause brings about either a complete resolution or at least a convincing regression of the signs.

Facial neuralgia in human medicine is also known as *tic douloureux* and trigeminal neuralgia. It is an acutely painful disease in man, hence the word 'neuralgia,' which simply means *pain along the course of a nerve*. The trigeminal nerve is the main sensory nerve to the face and the largest of all the cranial nerves. In man, the severe neuralgia is notoriously difficult to cure, as its cause is not well understood.

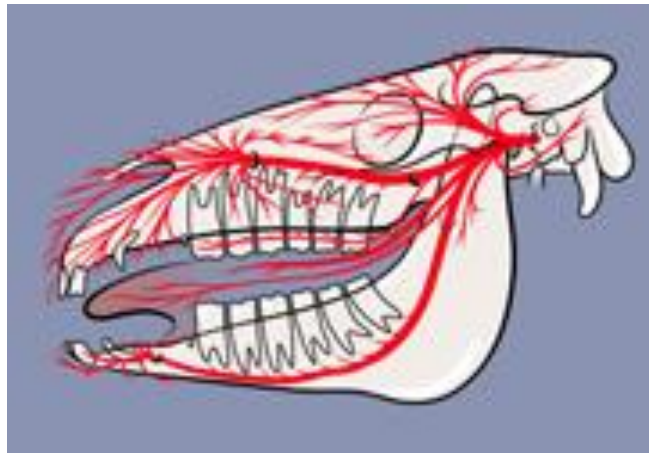
---

<sup>2</sup> There are isolated reports describing causes of headshaking other than facial neuralgia but these causes are rare and it is my experience that the bit is by far the most common cause and the first one to be ruled-out. "Common things commonly occur."

The trigeminal has three branches (Fig 1). The mandibular branch supplies sensation to the bone of the lower jaw, its teeth and to the related soft tissues of the tongue, chin, lips and gums. It also innervates the salivary glands<sup>3</sup> and skin of the ear. The maxillary branch supplies the bone, teeth, hard palate, soft palate, nasal mucous membranes, lips and gums of the upper jaw. The ophthalmic branch supplies sensation to the eye, eyelids, tear glands, skin of the forehead and the nearby nasal mucosa.

The null hypothesis, which has been disproved, proposes that removing the painful bit from the mouth of a headshaking horse and replacing it with the painless cross-under bitless bridle will not result in a significant amelioration of the headshaking syndrome.

Trigeminal pain may be transmitted directly to the brain or be sensed indirectly by a process of referred pain in which signals from any tributary of the trigeminal nerve in direct contact with the bit spread to other tributaries of the same nerve, resulting in pain being experienced in areas of the face that have no direct contact with the bit.



*Fig.1. The distribution of the three branches of the trigeminal nerve. To imagine the potential distribution of facial pain, start from any one of the areas that the bit contacts and follow the red pathway from there to the termination of the nerve in any other facial area. Some headshaking horses will experience pain or hypersensitivity in the region of the muzzle, whereas others may experience this around the eyes, ears or forelock.*

Direct stimulation of the brain by the acute pain in the mouth accounts for the violent head tossing. The more chronic pain probably initiates a dull bone ache similar to a bad toothache and accounts for a general tenseness of the jaw, neck, spine and limbs. This explains the stilted gait and short stride that so many bitted

---

<sup>3</sup> Hence the bit-induced stimulus to salivation that is physiologically contraindicated in the exercising horse (Cook 1999b)

horses exhibit; features that disappear when the bit is removed. When we have toothache we walk gingerly and do not feel like running. The horse may feel the same but when obliged to exercise experiences intermittent muscle spasms in the neck that produce the head tossing.

Pain signals will be sent directly to the brain from any portion of the gums, lips, hard palate, tongue, and teeth that the bit may press upon. In the male horse, as can be seen in Figure 1, the roots of the canine teeth lie ventral to the bars. Pain from the canines when the horse is 'on the bit' may account for the higher incidence of headshaking in geldings than mares<sup>4</sup> and the tendency for headshaking to be a familiar problem among horses trained for dressage. A few horses of both sexes will have wolf teeth in the lower jaw (as depicted), though they are more common in the upper jaw. Those in the lower jaw will often go unreported because, being vestigial, they may never erupt. They lie under the gum just in front of the first cheek tooth

The indirect stimulation may take the form of hypersensitivity, tingling, 'pins and needles' or actual pain. Such sensations in the mandibular branch explain the resentment that many horses exhibit to being handled around the ears. Referred pain from the bars may also travel down the branch of the mandibular nerve that supplies sensation to the tongue. This could result in the tongue itself being hypersensitive and be yet one more reason why so many bitted horses are 'mouthy,' 'play with their tongue,' protrude it, retract it, or place it over the bit. The same mechanism can be invoked to suggest that trigeminal neuralgia may initiate toothache.

Referred pain in the maxillary branch explains the sneezing, snorting and muzzle rubbing of the headshaking syndrome. Referred signals down the ophthalmic branch explain the sensitivity to bright light, the rapid blinking spasms, and the shyness to handling around the forelock. Stimulation of the lacrimal glands also accounts for the nasal discharge and provides a further explanation for sneezing.

The clinical signs of the headshaking syndrome represent a pathological exaggeration of a normal response. A healthy horse will respond to a fly landing on its face with a toss of its head. A horse with trigeminal neuralgia behaves as though it is plagued with a swarm of particularly vicious biting flies that refuse to go away. Imagine this, coupled with raging toothache in both jaws, and the reader will have some idea of a horse's pain. Tic douloureux is one of the fiercest pains known to man.

Depending on the duration and severity of the neuralgia, removal of the bit brings about a regression of the signs or their complete disappearance. Affected horses may show a more or less immediate improvement or a progressive improvement over a period of several weeks or months. Presumably, many of

---

<sup>4</sup> Though as there are more geldings than mares in work this too could influence the sex bias

these horses will be shown, in due course, to have had bone spurs on the bars of their mouth but this correlation has not yet been made.

Most headshaking horses exhibit many more adverse behavioral signs than the seven or so signs that have traditionally been associated with this syndrome. I conclude that the headshaking syndrome is simply a subset of the much larger syndrome, aversion to the bit. In other words, facial neuralgia explains the seven or so traditional signs of headshaking but affected horses also exhibit many signs associated with the other six F's (Part III; Table I).

### **Bit-Induced Upper Airway Obstruction**

Some respiratory diseases that I believe to be caused by the bit include dorsal displacement of the soft palate (Cook 1999a, 2002a), epiglottal entrapment (Cook 1999b), and pulmonary bleeding (Cook 1999a, c). I now recognize that the bit is also a common cause of abnormal respiratory noise at exercise and responsible for 'thickness of wind' and 'roaring' in many horses (Cook 2003). I recommend that the bit should routinely be considered as a differential diagnosis for any horse that makes a roaring noise. Roaring is no longer a sign that is pathognomonic of recurrent laryngeal neuropathy. Deformity of the windpipe and further obstruction of the airway is also a common defect and occurs much more frequently than has generally been supposed. I am of the opinion that this is a long-term effect of airway obstruction caused by the bit. Finally, upper airway obstruction is probably a cause of small airway disease.

Because the root of the tongue is suspended from the hyoid apparatus, which also supports the larynx, any thing that causes the tip of the tongue to move, such as the bit, is likely to cause the root to move and, therefore, the larynx. When the larynx is tossing about like a ship in a storm, the airway will be obstructed. Because the soft palate lies in contact with the root of the tongue, any movement of the tongue also causes movement (i.e. elevation) of the soft palate, which in turn obstructs the nasopharynx.

The tongue is a powerful muscular organ, a highly tuned sense organ and has predominantly digestive functions. It is active during eating and drinking but should be at rest during exercise. As in all mammals, the horse has evolved to eat or exercise. It cannot carry out both activities simultaneously and should not try. And yet by placing a bit in its mouth this is precisely what a rider is demanding.

In the exercising horse at liberty, the lips are closed, there is no air in the oral cavity and oropharynx, the immobile tongue occupies the entire space within both these regions and salivation is in abeyance. In the exercising horse when ridden with a bit in its mouth, the seal of the lips is broken; the jaw may be frankly

open; air enters the oral cavity and oropharynx, the tongue is constantly on the move; and salivation is stimulated.

The above responses triggered by the bit are digestive system responses. All these responses are diametrically opposed to the respiratory system responses required for exercise. Because of these, a bit interferes with the horse's ability to breathe properly at exercise. A running horse takes one stride for every breath (Cook 1965). If it cannot breathe properly it cannot stride properly. As these two functions are impaired, together with other cardiovascular functions crucial to exercise, a bit prevents a horse from performing to its full potential. The details of how the bit causes confusion in the horse's throat have been explained and illustrated in previous articles (Cook 1999b, 1999c, 2000, 2002a). A summary follows here. A cascade of events occurs in which respiratory obstruction progresses from mild to severe and even fatal. In any one bitted horse, the degree of asphyxiation will vary according to the nature of the exercise.

- A bit is frequently, though incorrectly, used to bring about poll flexion
- Poll flexion obstructs the airway at the throat
- A bit causes a horse to open its mouth and move its tongue
- This allows air to enter the oropharynx, which elevates the soft palate and obstructs the nasopharynx.
- Elevation of the soft palate can lead to dorsal displacement of the soft palate (DDSP) and 'choking-up'
- It is also responsible for the development of epiglottal entrapment
- Obstruction of the nasopharynx can lead to a partial collapse of airway structures that lie caudally, e.g. dynamic collapse of the aryepiglottic folds.
- Negative pressures on inspiration become increasingly negative as the distance from the source of the obstruction increases. This effect is most severe at the termination of the airway, i.e. the lungs.
- Nasopharyngeal obstruction will lead, in the short term, to dynamic collapse of the cervical trachea.
- In the long-term, it explains permanent deformity of the cervical trachea ('scabbard trachea') and yet further obstruction caudal to the trachea
- The delicate air sacs of the lung cannot function properly because of the abnormally high suction pressures that an airway obstruction generates on inspiration
- All of the above factors traumatize the bronchioles of the lung and will lead, in my opinion, to the development of small airway disease.
- Abnormally low negative pressures on inspiration will suck heavily blood-stained fluid from the pulmonary capillaries into the pulmonary parenchyma, the alveoli and small airways.

- Pulmonary oedema develops. For a number of reasons, asphyxia-induced pulmonary oedema (AIPE) is a more appropriate name than exercise-induced pulmonary hemorrhage (EIPH)<sup>5</sup>
- In man, pulmonary oedema causes severe chest pains. The horse may also experience pain for the same reason. If so, thoracic pain would inhibit respiration and this, in turn, would exacerbate the asphyxia
- A racehorse may die from acute pulmonary congestion and heart failure
- Or it may simply stop racing because of pain, a lack of oxygen, respiratory distress and extreme fatigue
- Fatigue leads to falls
- Falls cause broken legs
- Broken legs lead to euthanasia

### **Bit-Induced Welfare and Safety Problems for Horse and Rider**

Any method of communication that triggers a hundred common and negative side-effects, some of which are fatal, has to be classified as contraindicated and unacceptable. However, if only because the bit inflicts avoidable pain (the definition of cruelty) its use should be subject to investigation. But in addition and quite aside from the humanitarian issue, the bit method is contrary to the best interests of a rider aiming at developing a harmonious partnership with her horse and achieving optimum performance. An athlete in pain cannot and will not perform well. Pain reduces both the ability and the desire to perform and is a potent source of accidents to rider and horse.

There is no right way of doing the wrong thing. In my opinion, the bit method of communication is inefficient, inhumane, and unsafe.

---

<sup>5</sup> Blood-stained oedema fluid leaks out of the waterlogged lung for the same reason that a fountain pen leaks at high altitudes during a flight (Cook 1999a). In the shod horse at the gallop, absence of the four supplementary vascular pumps that the unshod hooves normally provide, will be a further cause of poor circulation and, therefore, of pulmonary congestion. Such congestion may well be an additional cause of "bleeding."