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FORTY YEARS ON: Looking back to see ahead

Robert Cook[1]

Changing something is like moving a graveyard - you never know how many friends the dead have until you try to move them. - Woodrow Wilson

April Fool's Day, 1969, was day one of my 8-years at Balaton Lodge in Newmarket, where the Equine Research Station of the Animal Health Trust was then located. The focus of my research was the head, neck and chest of the horse. This essay is a sketch of the work I did there, reviewed with the benefit of hindsight. Let the biopsy begin.

The endoscope I used from 1969-2003 was the same as I had used since 1960; a one meter, rigid rod with glass optics, and a terminal bulb powered by a 4.5 volt battery (Cook 1970). Rather grandly, it was called a rhinolaryngoscope ('rhino' = nose; larynx = voice box). It was an instrument for inspecting gun barrels, masquerading under a new name. As a borescope, it was safe enough for the operator, the instrument and the gun but not when used for examining the throat of a fully conscious horse. No equine sedatives had been developed in the seventies, so the 'gun' was loaded. The likely impact of the vulcanite eyepiece on the operator's eye could be softened with a wrap of sponge rubber. The throat was seen 'as through a glass darkly.' The number of veterinarians worldwide that had used such an instrument could be counted on one hand with several fingers missing. By trial and error, I learned how to conjure this device into the back of a horse's throat without bending the horse, the instrument or myself. To examine the guttural pouch meant burying it up to its hilt. The horse became a 'sword-swallower'(Fig 1). Curiosity overcame temerity and much was learned. Endoscopy became as indispensable for the diagnosis of ear, nose and throat diseases, as radiography for the diagnosis of lameness.

When fiberscopes became available, the human oesophagoscope served as an equine laryngoscope (Cook 1974a). Their flexibility made it safer for the patient and veterinarian. It was also more 'democratic' as endoscopy was no longer the preserve of a foolhardy few. The vastly improved lighting permitted photography. For recording purposes in the early days, I had to paint a watercolour.

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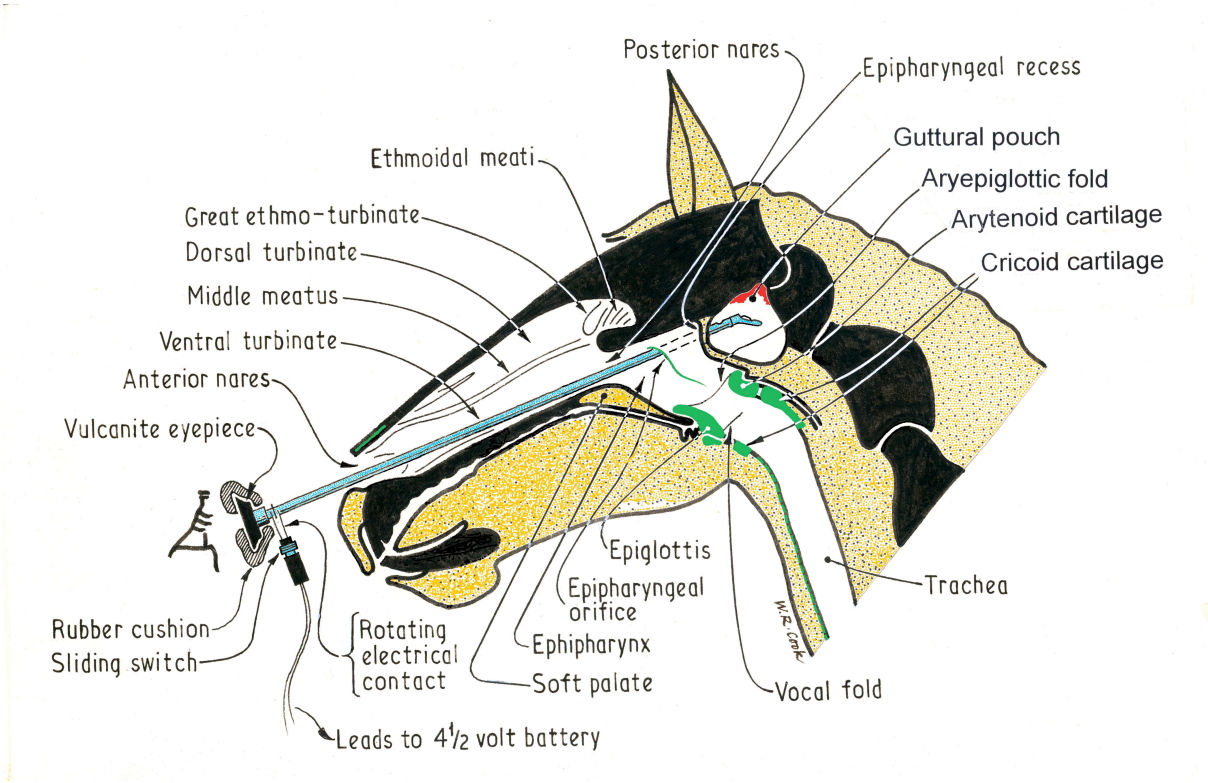

Figure 1. Normal anatomy and 70s style guttural pouch endoscopy. The red area shows the fungal mat of guttural pouch mycosis.

Colour key: black = bone; yellow = soft tissue; green = cartilage

Since the 70s, endoscopy has advanced anatomically and technologically. The lung can now be 'scoped as well as the stomach. Video endoscopy of the respiratory tract of the standing horse was followed by treadmill endoscopy and, most recently, by dynamic endoscopy – examination of a ridden horse's throat at the gallop. This is not the moons of Jupiter but it is a new world for equine veterinary medicine. Theodore Rozsak wrote, "*Nature composes some of her loveliest poems for the microscope and telescope.*" Today he might have included the endoscope.

Back in the age of the borescope, I had described a fungal disease of the strange evagination of the horse's Eustachian tube, its guttural pouch (Cook 1968).[2] One of the clinical signs of this 'new' disease (guttural pouch mycosis) was nose bleeding. Ferocious and sometimes fatal haemorrhages occurred from aneurysms on the internal carotid artery. With the help of Dr. D. Hawkins, a medical radiologist from Cambridge, we developed an angiographic technique for identifying these aneurysms (Cook 1973, Colles and Cook 1983). The damaged artery was then ligated before the horse bled to death. In the 70s, the cause of the disease was unknown and, even today, it is still classified in the mainstream literature as of unknown aetiology. I am of the opinion that the problem arises from a temporary blockage of the horse's Eustachian tube (Cook 1986). At its middle ear end, the

[2] The Eustachian, auditory or pharyngo-tympanic tube links the throat to the middle ear.

tube is a short (6-7mm) and extremely narrow channel through bone. My hypothesis is that this capillary tube is easily blocked by inflammatory swelling during a respiratory infection (Fig 5a). This would lead to the development of a vacuum in the middle ear, followed by a transudate of serous fluid. Once the inflammation regresses, serum oozes out and adheres to the roof of the guttural pouch, providing the ideal culture medium for the growth of a fungal mat in this warm, humid and poorly ventilated cavity (Fig.1). Fungal spores are a constant contaminant of the guttural pouches of a stabled horse.[3]

Looking back, it is surprising to be reminded that a chronic cough in the horse used to be a clinical puzzle. The puzzle resolved into either 'dust or donkeys.' Maurice Round at the Equine Research Station had recently shown us that the donkey is an asymptomatic host for the equine lungworm. Horses grazing the same pastures often developed parasitic bronchitis. But stable dust was a much more common cause of chronic bronchitis ('broken wind'). Many horses at the time were housed in barns that doubled as storage facilities for slowly decaying vegetation – hay and straw.

In the 70s, I examined many racehorses suffering from what was referred to as a 'broken blood vessel.'[4] No one had asked which vessel it was that 'broke.' It was assumed to be somewhere in the nose and, accordingly, the condition was called epistaxis. The borescope enabled me to rule out the head as the source of the blood and conclude that these horses were bleeding from the lungs (Cook1974b). Richard Pascoe in Australia confirmed this in 1981 by fiberscope surveys, naming the problem 'exercise-induced pulmonary haemorrhage' (EIPH). Subsequent surveys indicated that EIPH affects over 95% of Thoroughbred racehorses. This sounds like a harsh judgment on the Thoroughbred and a new slant on the 'blood' horse - when they run they bleed. But in fairness, the problem is not restricted to the Thoroughbred. It occurs during exercise in many other breeds and occupations.

Since the 70s, much has been written about EIPH but debate continues concerning its cause. In my opinion, the only explanation consistent with the facts is that it is caused by upper airway obstruction (Cook 1988, Cook et al 1988) (Fig 2). For many years I thought that palsy of the voice box (recurrent laryngeal neuropathy or 'Roaring') was the major culprit. Recurrent laryngeal neuropathy (RLN) is certainly a common disease and whatever causes EIPH has to be virtually ubiquitous. However, since 1998 I have realized that something even more consistent with the facts has been staring us in the face for 5000 years – the horse's bit. I now believe that, though RLN is a common and serious cause of airway obstruction and 'bleeding,' the bit is an even more constant cause. EIPH is a misnomer. It is neither exclusively 'exercise-induced' nor a true haemorrhage. The problem used to occur in plow horses that were 'roarers' but it can also occur in the stable if an otherwise healthy horse gets cast with its head twisted. A more accurate name and one that reflects the cause is that of a rare disease in man - negative pressure pulmonary

[3] Fungal spores are no respecters of equine 'royalty.' His skull now in the Newmarket Racing Museum, tells me that Hyperion may have been a lucky survivor from guttural pouch mycosis.

[4] An early 'breaker of blood vessels' b. 1716 was a son of the Darley Arabian named 'Bleeding Childers.' He never raced but was put to stud as 'Bartlet's Childers' and became an ancestor of Eclipse.

oedema (NPPO). An internet search will reveal that NPPO in man is analogous to EIPH in the horse.[5]

The bit suffocates a horse because it permits air into the digestive part of the throat and triggers movement of the root of the tongue. These and other factors elevate the soft palate which, in turn, obstructs the respiratory part of the throat (Fig 3, 5 & 6).

Salix is not a remedy for suffocation. The causes of airway obstruction must be removed. RLN will be difficult to remove, though its incidence might be lessened by more discriminating breeding. But the bit could and should be removed (Fig 4). A change in the rules of racing may be required to permit the use of a bitless, painless and more effective bridle (Cook 1999, Cook and Strasser 2003, Cook and Mills 2010).[6] Such a change would enhance the welfare of the racehorse, improve the

Figure 2. The red portions of the lung show the distribution of haemorrhagic pulmonary oedema that occurs as a result of any upper airway obstruction (in this case, caused by bit-induced poll flexion). The 'top-and-tail' distribution of the oedema in both lungs follows from the principles of aerodynamics. The small arrows in the throat indicate the dynamic collapse of the soft walls of the throat that result when the suction pressure of inspiration increases because of poll flexion.

[5] Pulmonary oedema in man is accompanied by acute chest pain and a sensation of drowning. If the horse experiences anything similar, this plus the sudden onset of exhaustion might account for a so-called mis-step, followed by a fall and a catastrophic injury. In other words, there may be a connection between 'broken blood vessels,' broken wind in its widest sense, and broken legs.

[6] The crossunder bitless bridle is now marketed worldwide by many companies but, as the Chairman of BitlessBridle Inc., - the first company to successfully introduce the design - I unapologetically declare a conflict of interest. For more information visit the educational website www.bitlessbridle.com

the safety of the jockey and reduce accidents. On the basis of experience with this bridle in other disciplines, I predict that a racehorse without a foreign body in its mouth will run faster.

In 1969, the radiography facilities at the Equine Research Station were unremarkable but subsequently became outstanding. Bob Crowhurst in Newmarket, the veterinary advisor to Lady Beaverbrook, referred one of her horses from Major Hern's yard at Lambourn. Sea Epic, her favorite horse, 'swallowed its tongue' or – as this problem came to be called – developed dorsal displacement of its soft palate (DDSP). I wish I had known then what I know now about the cause of DDSP but, in the event, I operated on the horse and resected a small portion of its soft palate (staphylectomy). When her horse was discharged, Lady Beaverbrook mentioned that she would like to give us something and, several months later, we heard from Bob Crowhurst, that she wanted us to tell her what we would like. We had no idea what size of gift she had in mind, so we originally suggested some quite modest piece of equipment. I don't know what was proposed at first or even, with tongue in cheek, on the second occasion but it was not enough. "No' she said, "I want to give you a <u>real</u> present." So now, with some trepidation, it was proposed that Lady Beaverbrook might like to give us a Siemens radiography unit, complete with fluoroscopy and tomography.[7] This she did and the generous gift enabled us to overcome many barriers to diagnosis.

Trying to cure a disease without knowledge of its cause is like sailing the Atlantic without a navigator. DDSP is a common scourge of the racehorse. Thanks to fluoroscopy and a study of Wood's comparative anatomy of the mammalian throat, I learned a little about the problem but not the cause. Once again, it is only in the last decade that light has dawned (Cook 2002, 2005). I now propose a unifying hypothesis, consistent with the facts, that explains the cause of DDSP and NPPO. First, the bit causes elevation of the soft palate, with or without DDSP. This causes obstruction of the airway and, with or without a degree of RLN, this in turn causes 'bleeding' (NPPO).[8]

During a race, a gale whistles back and forth in a horse's throat, not less than 120 times a minute. Bit-induced tongue movement and air entry causes instability of the horse's long soft palate. The soft palate elevates and - from time to time - becomes completely unbuttoned from the voice box, i.e., DDSP (Figs. 5 & 6). DDSP results in a horse asphyxiating and making the characteristic death rattle in its throat but elevation alone – though less dramatic - is still serious. The increased suction force of obstructed inspiration and the accelerated and turbulent airflow, may also cause the roof of the throat to collapse (Fig.2). Soft palate elevation and DDSP both cause suffocation and precipitate intense fatigue. Such a horse may fall and, in falling, break a leg.[9] Elevation and DDSP is a normal part of swallowing but not of running.

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[7] In 1877, the cofounder of the company, Ernst Werner von Siemens, was a manufacturer of dynamos. He funded a chair for the physics dean, Heinrich Friedrich Weber, at the Swiss Polytechnic Institute. The dean's classes were taken by a student with an intense interest in dynamos. The student's name was Einstein.

[8] Dynamic collapse of the windpipe is probably a part of this cascade.

[9] DDSP is a functional problem and, by itself, leaves no post-mortem evidence. At autopsies on racecourse fatalities, laryngeal and lung lesions should be looked for, otherwise such fatalities may be ascribed to breakdowns and fractures and be incorrectly classified as musculo-skeletal rather than respiratory in origin.

To place a bit in the mouth of a horse about to run is akin to muzzling a horse about to eat.

Figure 3. A bit triggers the wrong set of responses for exercise. It stimulates digestive responses (chewing, salivation and swallowing) rather than the respiratory and cardiovascular responses that are needed (increased respiratory rate, heart rate and blood supply to the muscles of locomotion). The two diagrams show that the configuration of the throat has to be fundamentally different for each function. The soft palate and the cartilages of the voice box act like switch plates to either direct air into the lungs or food and water into the stomach. At bitted exercise, swallowing may only occur intermittently but partial elevation of the soft palate is common because of digestive reflexes, tongue movement and the entry of air into the mouth and back of throat. Elevation of the soft palate, from this and other causes, obstructs the airway (see also Fig 6).

Key: AC = arytenoid cartilages of the larynx ('flapper' cartilages); E = epiglottis; LP = laryngopharynx; OI = ostium intrapharyngium (the soft palate buttonhole); OP = oropharynx; SP = soft palate;

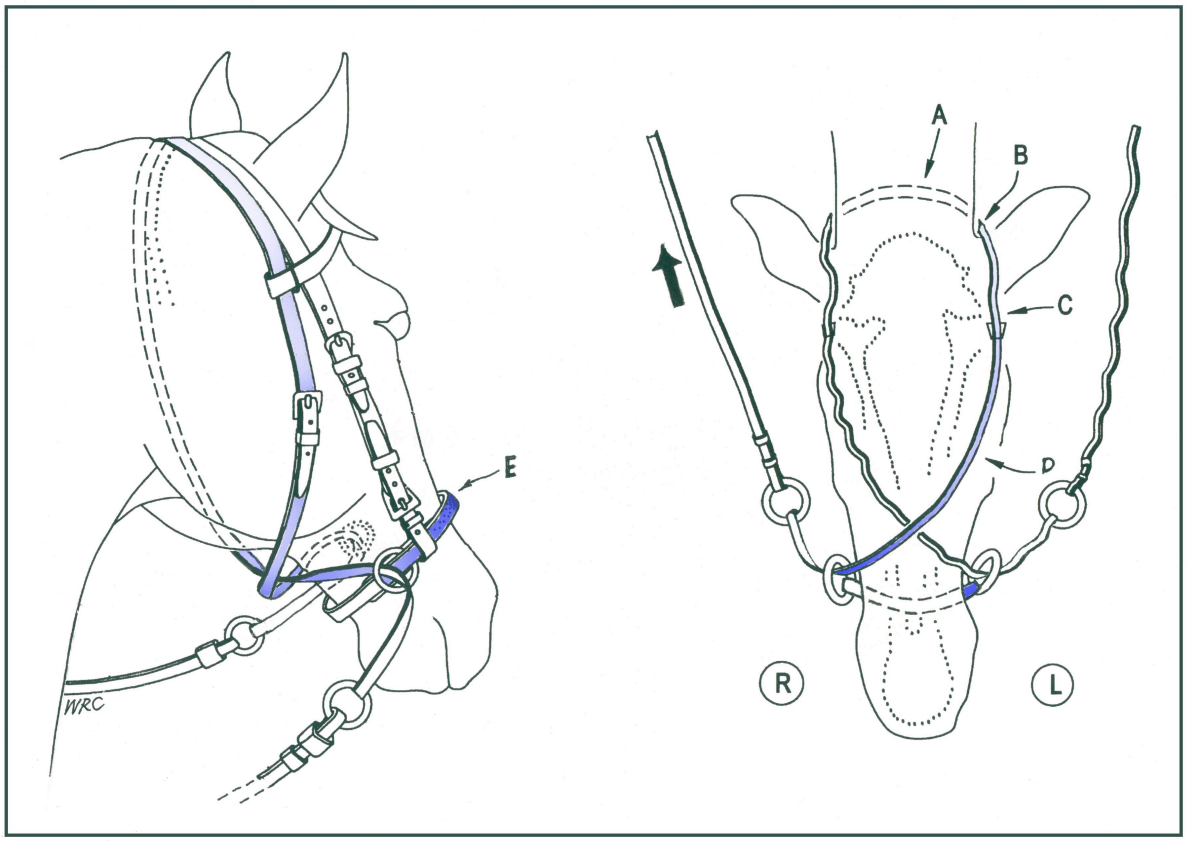

Figure 4. Crossunder bitless bridle. The diagram on the right shows the underside of the head. For a steering rein-aid, the right rein (thick arrow) distributes painless pressure on skin over the left half of the head (thin arrows A–E). For slowing or stopping, the rein-aid hugs the whole of the head. At no point is skin pressure anything but gentle. As indicated by gradation of colour, pressure diminishes from E to A.

At the Equine Research Station, as during my previous years at the Glasgow Veterinary School and the Royal Veterinary College, London, I continued to encounter horses that suffered from the headshaking syndrome. This all too familiar problem was the bane of the horse, owner and veterinarian as the syndrome was notoriously recalcitrant to treatment. For a dressage horse it was often career ending. The reason for our inability to cure headshaking was, of course, our failure to understand its cause. Soon after I left Newmarket and took up a post in the USA, I published four articles which, for the first time, fully described the syndrome and its investigation. Yet, in spite of spilling so much ink, I concluded the series by admitting that I did not know the cause and could offer no treatment (Cook 1979, !980a, b). Years later, I published another article, still with the same conclusion (Cook 1992). Late in my career,[10] I realize that the bit is the most common cause of headshaking (Cook 2007a, b, c).

[10] I graduated from the Royal Veterinary College in 1952

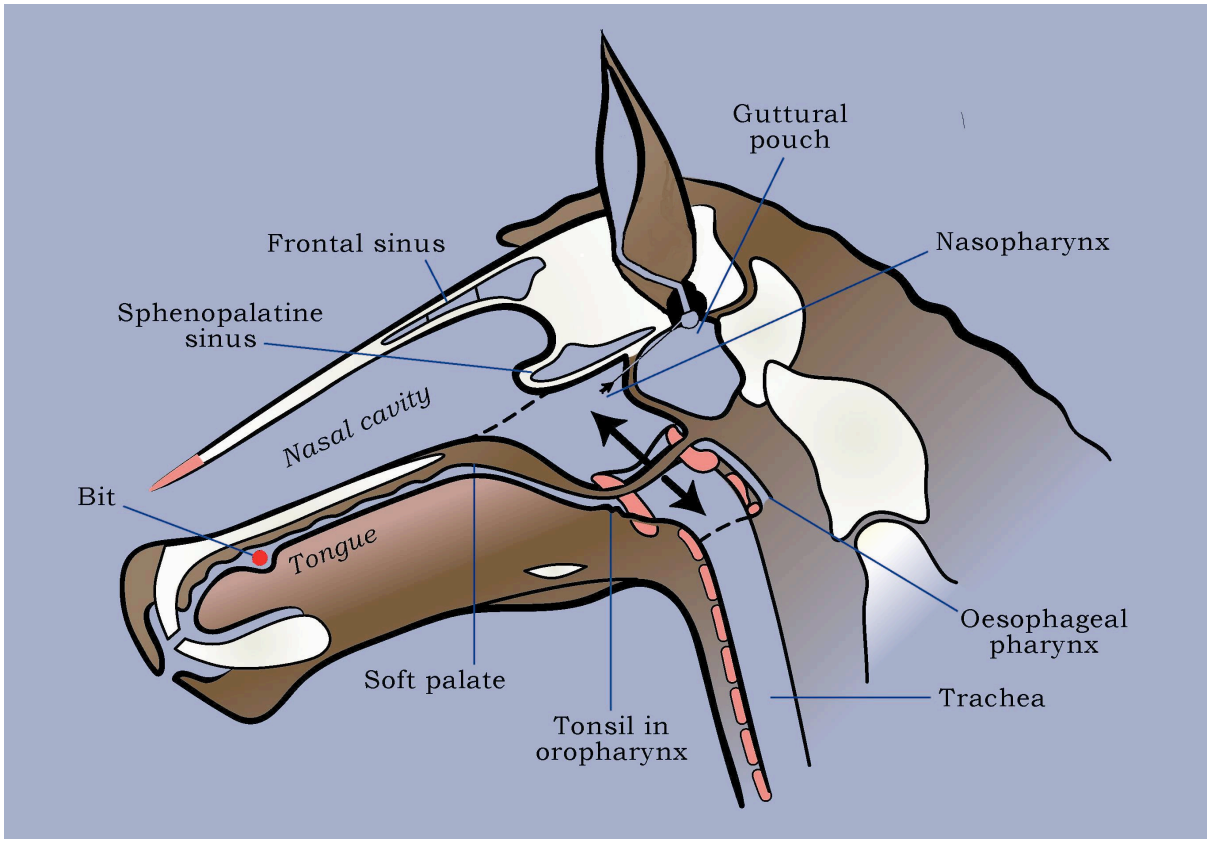

Fig. 5a. Normal anatomy. The horse can only breathe through its nose. In order to breathe freely at the gallop, the soft palate must stay in contact with the immobile root of the tongue and its buttonhole must firmly embrace the fully dilated voice box. But the bit causes tongue movement and RLN causes a failure of the voice box to open properly. The small arrow is the Eustachian tube. A magnifying glass will enable the capillary section of the tube to be seen as it connects with the spherical cavity of the middle ear.

Fig 5b. An enlarged view of the throat, illustrating the proper configuration of the soft palate buttonhole during exercise.[Errata: The diagram needs two corrections. A larger section of the epiglottis should be shown lying on the soft palate and the air space in front of the base of the epiglottis should be eliminated].

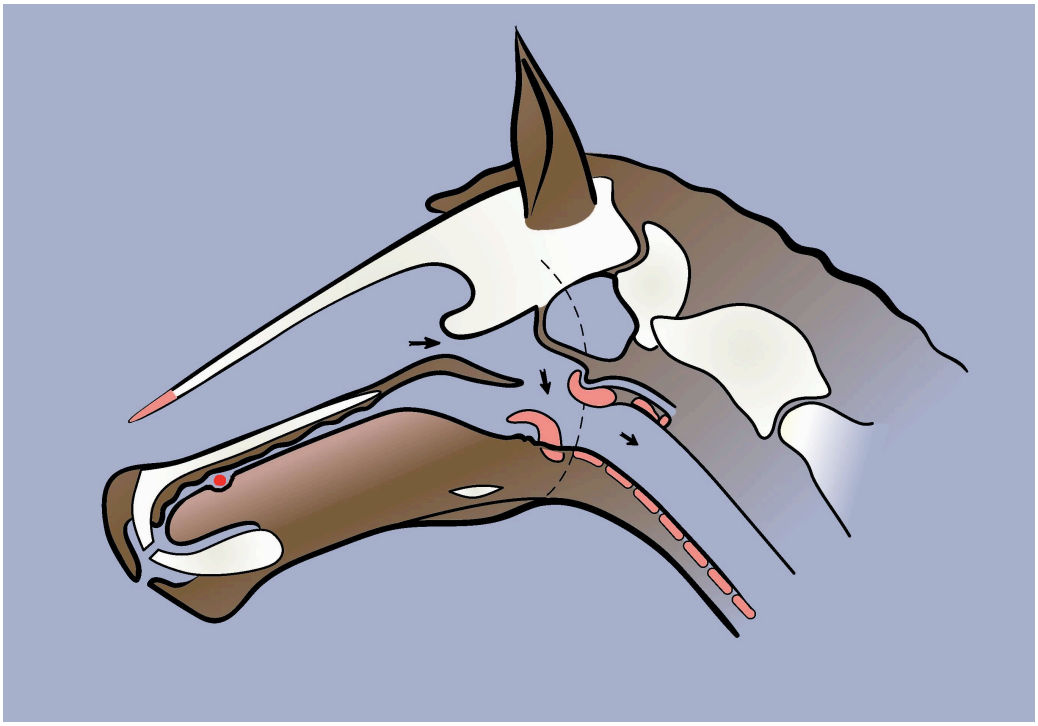

Fig. 6a. DDSP at the gallop causing suffocation. From left to right, the three arrows illustrate severe obstruction of the airway on inspiration. First, obstruction occurs at the junction between nasal cavity and throat; secondly at the entrance to the larynx; and thirdly dynamic obstruction of the windpipe occurs, especially at the entrance to the chest.

Fig. 6b. Enlarged view of the elastic-sided buttonhole, now no longer under tension and collapsed, severely obstructing airflow. The double ended arrow shows the turbulent flow and obstruction on expiration responsible for the characteristic 'death rattle' of DDSP.

The underlying reason for the head tossing spasms and other signs of facial pain is trigeminal neuralgia (tic douloureux). Nerve pain in the major sensory nerve to the head is, I conclude, triggered by the relentless and repetitive pressure of the bit on tongue, bone and teeth (Fig.7). In the 70s, in order to investigate these cases, I used to carry out a whole range of tests, mostly with unconvincing results. Life is simpler now, less expensive and more rewarding for all concerned. The first and most important step I recommend is removal of the bit. With this simple step, the majority of headshakers recover.

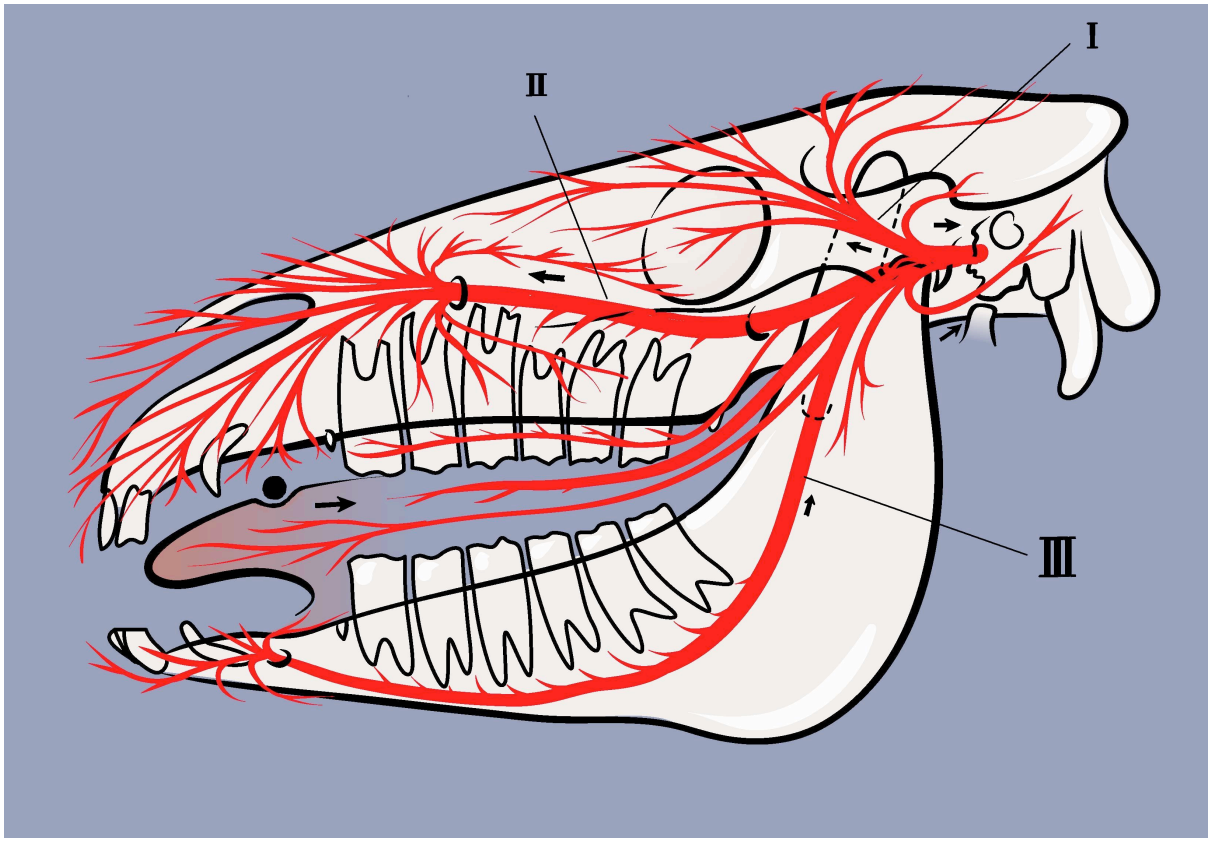

Figure 7. Distribution of the trigeminal nerve (cranial nerve V). This major sensory nerve has three branches: ophthalmic (I); maxillary (II); and mandibular (III). A feature of the nerve when over stimulated is to refer pain from any one section of the nerve to other sections of the same nerve (see arrows). For example, constant irritation of the tongue or the bars of the mouth by the bit, may result in the horse sensing pain in the muzzle, ear or eye. Symptoms would be, respectively, muzzle rubbing, refusal to permit an ear to be handled, and dislike of bright light (photophobia). Head tossing results from pain in one or more locations.

A colleague at Glasgow University, Professor Donald Lawson, used to preface his more *ex-cathedra* pronouncements with a smile and say, *"In my humble opinion. if I have such a thing."* I will borrow this preface in pronouncing that the bit is also the cause of epiglottal entrapment, 40 other diseases, over 200 behavioural problems and many accidents, some of which are fatal to horse and rider (Cook 2007b, c, 2009). My contention can be tested by removing the bit from any horse and counting the improvements that follow. A bit is to a horse what a virus is to a computer. When competition rules mandate one or more bits, a rider has the option of not competing or campaigning to get the rules reformed.

Dr. Richard Archer, who was Director of the Equine Research Station during my time there, encouraged me to study for a PhD and arranged for me to be admitted at his college, Trinity Hall. Kenneth Wilsdon, the senior Ear Nose and Throat consultant at Addenbrookes Hospital in Cambridge volunteered to be my supervisor for a study of laryngeal paralysis in the horse.[11] He was most conscientious about his duties and would call in at Balaton Lodge for a chat every Thursday afternoon,

[11] It was many years later before I proposed the more accurate name recurrent laryngeal neuropathy (RLN).

after he had finished his clinic at the Newmarket Hospital. If I had to tackle some unfamiliar surgery, such as the repair of a cleft soft palate, I could appeal to him for support and he would scrub up with me. As Kenneth was deaf and dependent on lip reading, we communicated during these surgical duets by hand signals as we were both wearing masks. The tale has already been told of how my thesis travelled for three months on the London-Edinburgh line without being disturbed (Cook 1999).

The same reference describes my serendipitous discovery of a spinal reflex in the horse. By slapping a horse on the left saddle patch, the right side of the voice box gives an answering twitch and vice-versa. I called it the thoraco-laryngeal reflex and subsequently developed a computer-based instrument that measured the response time from slap to twitch (Cook and Thalhammer 1991). A healthy nerve pathway allowed the signal to reach its destination faster and in better 'shape' than a diseased one (Fig. 8). As an objective test for RLN it has merit but needs waveform analysis of the data to bring it to fruition. I hope that some researcher in the future will team up with a mathematician to complete this project. The same reflex proved valuable for the investigation of 'Wobblers' and other diseases of the spinal cord (Greet et al. 1980).

At Newmarket, I came to the conclusion that surgery could never be a satisfactory answer for RLN. The voice box is a valve that has to open for deep breathing and shut for swallowing. Any surgery aimed at fixing it in a fully open position is doomed. And anything less than fully open is insufficient for a racehorse.[12] As my endoscopy and postmortem studies were indicating that some degree of left-sided RLN was present in most horses, it seemed probable that the disease was inherited. Prevention was the logical approach. So I spent a number of years studying the genetics of the Thoroughbred. My plan was to measure its coefficient of inbreeding, in order to offer this as a selection tool. My reasoning being that the less inbred a yearling was, the less likely would it be to suffer from recessive defects during racing or transmit these when breeding. The ideal location of Newmarket close to Cambridge, made it possible to seek advice from a professional geneticist, Dr. A.E. Edwards. Like a true Cambridge academic, when he travelled the 15 miles to Newmarket he cycled. He was the first cyclist I knew who was wise enough to wear a helmet. We measured the coefficient of inbreeding for 490 horses at 12 generations but had inadequate computer power to complete the project (Cook and Kirk 1991). The baton needs to be picked up by others. Suffice to say that an earlier 20 generation sampling study had shown that 60 Thoroughbreds had an average degree of inbreeding greater than that of a brother-half sister mating (Mahon and Cunningham 1982). The degree of inbreeding of the modern Thoroughbred has yet to be measured but, if it ever is the information cannot be anything but bad news. The General Stud Book has now been a closed stud book for over 200 years. Inevitably, each succeeding generation is more inbred than the last. Three more generations have been born since Mahon's work. It is to be expected in such a population that the incidence of infertility, fragility and recessive diseases will rise. Neurological problems in all species often surface in inbred populations, so RLN in the Thoroughbred and other breeds is likely to be no coincidence.

[12] One of the first endoscopic revelations was an appreciation of the full range of movement of the larynx. The fully open position was far more open than had been previously assumed and this emphasized the inadequacy of anything that could be achieved by surgery.

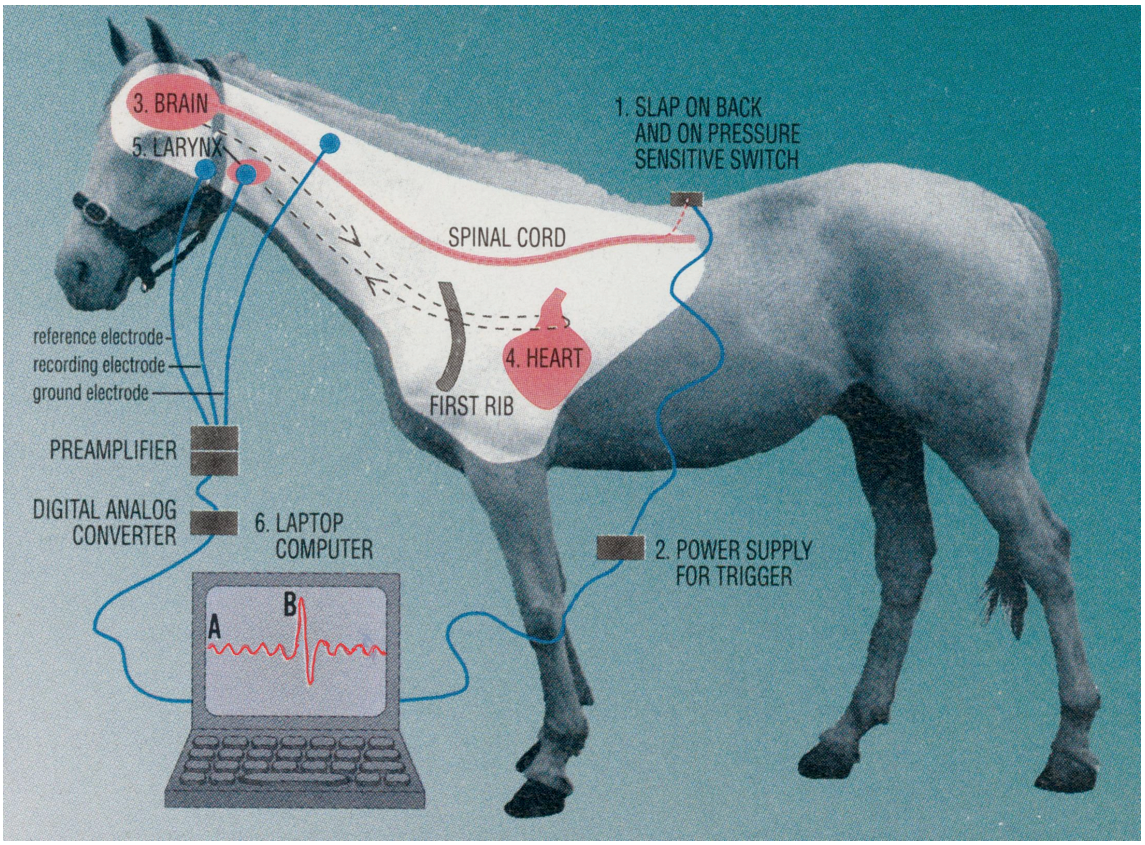

Figure 8. Nerve conduction test for the thoraco-laryngeal reflex, measuring the response time in milliseconds between back slap (A) and laryngeal twitch (B).

Equine dentistry in the 70s was not a specialty that had been widely adopted. Dentistry in practice consisted mainly of rasping teeth and removing temporary premolar caps. Referred dental cases mostly involved congenital defects of the jaw or sinus infections that required removal of an upper molar tooth. Since then, much work has been accomplished in dental care for the horse, with many new problems being identified and therapeutic procedures recommended. Concurrently, the natural horsemanship movement has developed and many horses, in order to go barefoot, are no longer stabled. A bonus of this more natural management of the horse will, I predict, be a decrease in dental problems.[13] Museum surveys have shown me that the oral health of feral horses is excellent. They do just fine without dentists or veterinarians. This in marked contrast to 66 museum skulls from domestic horses in which not less than 88% showed evidence of bit damage to the teeth or the bars of the mouth (Cook 2011).

The above paragraphs relate to only a small section of head, neck and chest diseases, though they are some of the most important and were, at the time, the most puzzling. It is instructive to look back on these with the benefit of hindsight, because – as it happens - all of them in the 70s were diseases of unknown cause. Even today, there is no consensus on their cause. Not everyone agrees with my unifying hypothesis. But anyone who has evidence to refute my hypothesis is free to publish it. They have had plenty of time and so far, no one has. The rules of the

[13] A decrease in other problems too, for example lameness and colic.

scientific game say that an unrefuted hypothesis merits acceptance as a basis for understanding and action, at the present state of knowledge. On the basis of 59 years experience as a veterinarian, I propose the following cause-based summary.

Guttural pouch mycosis is a management problem caused by stabling horses in a fungus-rich environment. The disease does not occur in horses kept at grass. Similarly, chronic bronchitis is a disease of stabled horses. 'Bleeding' is caused by the bit and RLN by breeding practices - two more management problems. The bit also causes DDSP, epiglottal entrapment, trigeminal neuralgia and a host of unwanted behavioural responses. I conclude that these are all diseases of domestication. In other words, they are caused by man. We humans are 'the management.'

The horse is a highly-tuned physiological entity, evolved over millions of years to survive and thrive in a natural environment. It cannot be 'supplemented' with foot and mouth metal without being harmed. A large mammal should not be kept in a small box. Horses are herd animals evolved to be easily frightened. They should be in the open, in company, grazing 14 hours a day with their heads at ground level,[14] secure in the knowledge that they are able to flee if necessary. Stabling a horse is stressful. It is like forcing a bat into the light. If we are to use them for high performance tests, let us at least wait until their bones have matured.

In my field of interest, there has been no time lag in the adoption of advances in diagnosis. There has, however, been an avoidable delay in the adoption of new information about the cause of disease and, therefore, its treatment and prevention. If diagnostic equipment can be quickly adopted, why not aetiological information? Perhaps aetiology suffers from the same problem as terminology. The physiologist Bernard Katz observed that, "*Certain scientists would no more use another's terminology than they would use another person's toothbrush."*

More careful scrutiny of breeding stock for pure-bred horses could bring benefits. But these changes will require difficult decisions by breed associations and will take much time and effort to introduce. In happy contrast, three management changes can be introduced over night by an owner. It has already been demonstrated that these can make immediate improvements to the welfare and safety of both horse and rider. In the last decade, thousands of owners, worldwide, have triumphantly shown us the way. In the 21st century, we can liberate the horse from stall and steel; from stable, bits and shoes.

[14] A position that facilitates postural drainage of lung, sinus and guttural pouch, and also promotes good hoof and dental care.

Acknowledgment

I am grateful to the Animal Health Trust for eight interesting years.

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There is properly no history; only biography

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