Bit-Induced Asphyxia In the Horse
(Elevation and Dorsal Displacement of the Soft Palate at Exercise)
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Equine practitioners must be disappointed when they read conference reports on clinical problems with which they are familiar and learn that the research is incomplete and solutions are not in sight. As "more research is needed' is the conclusion to so many research reports, it seems a pity not to announce the good news when a solution to a problem has been discovered. It is especially a pity when this applies to a problem that was caused by man in the first instance; is a problem that has been around for hundreds of years; is all too familiar to veterinarians; and is harmful to the welfare of both horse and rider.

Dr. Normand Ducharme's report of his invited contribution to this year's World Equine Veterinary Congress was entitled "Functional Relationship of Anatomy and DDSP" (J Equine Vet Sci, 2001; 21: 529-530). It commences "Dorsal Displacement of the Soft Palate (DDSP) was reported by Quinlan in 1949, yet our understanding of this disease is still incomplete." It continues with an outline of the current hypothesis being considered by Ducharme and others as to the possible causes of this problem. Ducharme classifies the causes as being some fault with either the intrinsic or extrinsic factors responsible for maintaining the functional integrity of the nasopharyngeal airway during fast exercise. The intrinsic factors relate to the health of the nasopharyngeal musculature, and the extrinsic factors to the health of the musculature that controls the position of the hyoid apparatus and larynx. From the report's conclusion it would appear that Ducharme and probably others currently regard DDSP as being caused by one or more diseases (emphasis added). But the identity, cause, and treatment of these diseases are not provided. His report concludes with the comment "At this time, only partial answers are available" and it would seem that no solutions to the problem of DDSP were proposed that practitioners could immediately apply.

With all due respect, I believe that members of this school of thought are asking the wrong question. If I understand their hypothesis correctly, they seem to be assuming that one or more diseases cause DDSP. Furthermore, they are basing their hypothesis and, therefore, their research efforts on the proposition that whatever diseases are causing DDSP accompanied by dysphagia in the horse at rest might be the same diseases that cause DDSP unaccompanied by dysphagia in the horse at exercise. In my opinion, this hypothesis can be refuted on the grounds that it is not consistent with the known facts about DDSP at exercise. Unlike the situation with dysphagia, no evidence of pathology accompanies DDSP at exercise. DDSP (the type that occurs at exercise) and DDSP (the type that occurs in conjunction with dysphagia) are, I believe, two quite different problems and need to be studied separately. Indeed, if we separate them we find that, although there is always a place for more research, we already have a working solution for the problem of DDSP at exercise.

Before going any further, it is necessary to define what is most commonly understood by the term 'dorsal displacement of the soft palate.' Typically, a horse with 'DDSP' is a racehorse that, if it were examined at rest, would be passed as fit to race. However, during a race this same horse will, from time to time (generally during the last third of a race),

develop severe respiratory distress. This is characterized by the sudden onset of a gurgling noise, like a death rattle, on expiration and/or inspiration, together with a marked reduction in speed or even a complete cessation of racing effort. In most cases, soon after coming to a standstill if not before, the noise will cease as abruptly as it commenced, and the horse will recover. A few horses, however, fall during the episode of asphyxia and break a leg in falling, while others suffer breakdowns &/or fatal pulmonary hemorrhages. In racing parlance, a 'gurgler' is said to have 'swallowed its tongue.' The almost ubiquitous use of a 'tongue-tie' is the trainer's effort at prevention. The logic, efficacy, and humanity of this measure is open to question2 and has never been assessed but its use is now so habitual that a tongue strap or bandage has become accepted as part of the standard racing tack. In any given horse, episodes of asphyxia are intermittent and generally unpredictable. Immediately prior to an attack, a horse may or may not have been making an inspiratory roaring noise (laryngeal stridor).

So much for the typical 'tongue-swallower' that actually displaces its soft palate from the epiglottis (ventral luxation of the palato-laryngeal junction). There is, however, a less dramatic but more prevalent version of the so-called 'soft palate problem' that occurs in both racehorses and non-racehorses. This is the horse in which there is elevation of the soft palate (ESP) but no displacement (Fig 8b). ESP may or may not be followed by DDSP (Fig 7). The clinical signs of ESP on its own include loss of performance, inspiratory laryngeal stridor (indistinguishable from that caused by recurrent laryngeal neuropathy), laryngeal fremitus and premature fatigue. In the following discussion of DDSP I shall not always refer to the possibility of ESP being an additional or alternative variant but readers should bear in mind that ESP is potentially a part of the DDSP phenomenon. I used to believe that laryngeal stridor was caused almost exclusively by recurrent laryngeal neuropathy. But I now realize that bit-induced ESP is an important differential diagnosis. Happily, differentiation is easy, using the new bitless bridle as an aid to diagnosis.

Returning now to commenting on Ducharme's report, we have known for some time that damage to the pharyngeal branch of the vagus nerve causes dysphagia and that this is sometimes accompanied by dorsal displacement of the soft palate in the horse at rest. Such damage is generally a sequel to guttural pouch mycosis (1). Similarly, we are aware that acute pharyngitis may also cause dysphagia, with or without DDSP at rest. The inflammation is generally associated with abscess formation in the retropharyngeal lymph nodes. Both these diseases draw attention to themselves in the stabled horse. The horse is clearly unwell and will usually be exhibiting many other clinical signs in addition to dysphagia, so much so that exercise is out of the question. Such presentations are quite different from the clinical histories of horses that develop DDSP at exercise only. The distinction is important and, to avoid confusion in our thinking, it seems necessary to qualify the acronym DDSP with 'at rest' or 'at exercise' (DDSPR or DDSPE).

Horses with DDSPE as defined do not exhibit dysphagia. Such horses are essentially healthy in all other respects. This is not surprising because DDSP unaccompanied by dysphagia is a perfectly normal and physiological phenomenon under certain conditions. It is normal, for example, for elevation of the soft palate to occur during mastication and for DDSP to occur during swallowing. It is abnormal for ESP or DDSP to occur during fast exercise, because the horse is an obligate nose-breather and needs a fully patent nasopharyngeal airway. The cause of DDSPE is not disease but inappropriate usage. It is a management problem. DDSPE occurs because man expects horses to run with one or

more metal foreign bodies in their mouths; something we would never let our children do. These foreign bodies (bits) generate digestive system reflexes (including DDSP and salivation), oral pain and other physiological responses that are incompatible with maintenance of the functional integrity of the nasopharyngeal airway during exercise.

In two scientific articles (3,4), I have published a list of the bit-induced mechanisms that I consider are the cause of DDSPE. However, the first article was published some years ago and the second article was in German. As the articles dealt with all of the many and diverse problems caused by the bit, perhaps the connection between the bit and DDSPE has been overlooked. It may help if I provide another list. But before I do this, let me say that I am now of the opinion that the bit is by far the most common cause of either elevation or dorsal displacement of the soft palate at exercise. If there are other causes (such as subepiglottic cysts) these are quite rare.

Reasons why the bit causes elevation or dorsal displacement of the soft palate

1. **The presence of one or more bits in a horse's mouth triggers digestive system reflexes. The horse is being signaled to 'think eat.' This is fine as long as the horse is at rest but now a rider or driver sets the horse in motion and signals the horse to 'think exercise.' The horse is now being signaled to eat and exercise simultaneously, something for which it has not been evolved. The result is a neurological confusion at the level of the pharynx, where the digestive and respiratory pathways cross over. The pharynx can function as an oropharynx or a nasopharynx but it cannot serve both functions at the same time (Figs 1-3).**

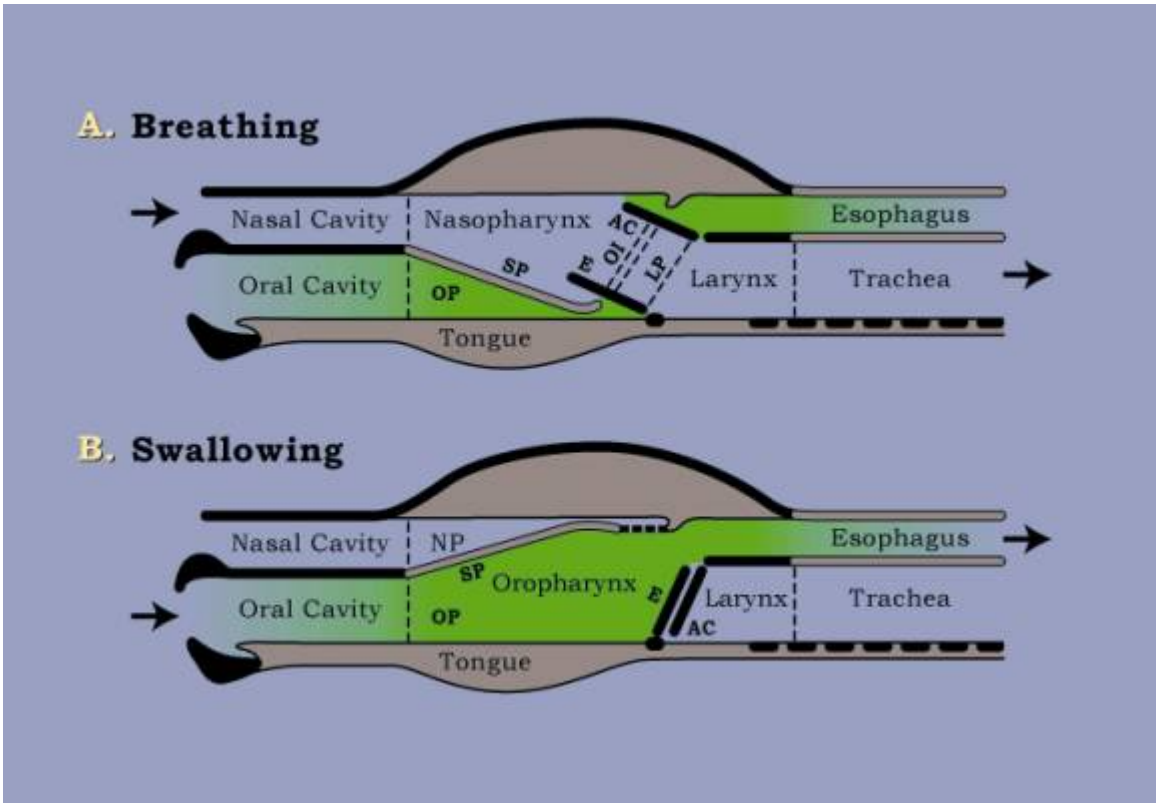

Fig 1. Showing the switching processes needed to change pharyngeal function from exercising to eating. For the sake of clarity, the mouth, oropharynx and esophagus are shown as actual spaces. However, except for those times when they contain food or liquid, these are - in normality - potential spaces only. Key: OI = ostium intrapharyngium; E = epiglottis; AC = arytenoid cartilages; NP = nasopharynx; OP = oropharynx; L = larynx; SP = soft palate; LP = laryngopharynx; EP = esophageal pharynx

a). Exercising: The soft palate is lowered to seal off the oropharynx and enlarge the nasopharynx. The arytenoid cartilages are raised to close the esophagus and open the larynx. The epiglottis is lowered to form a seal with the soft palate and, more than is apparent in this diagram, to smooth off airflow. The larynx now fits snugly into the soft palate buttonhole (ostium intrapharyngium)

b). Eating dry food or swallowing liquids: The soft palate is raised to close off the nasal cavity and prevent food or water entering. The arytenoid cartilages swing down to open the esophagus and close the larynx, so preventing food or liquid from inundating the lungs. Finally, the epiglottis swings back over the arytenoid cartilages.

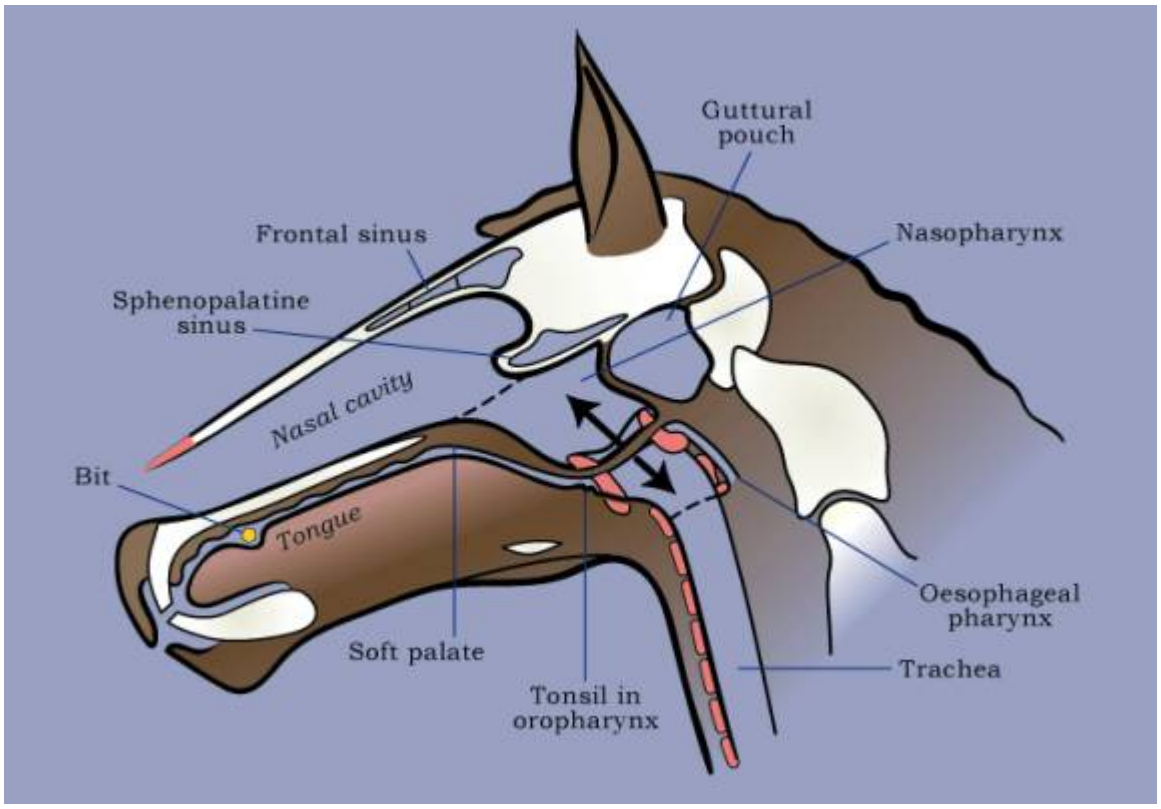

Fig 2. Sagittal section of the head and neck, showing the neutral poll position (jowl angle 87°) as for quiet breathing and slow paces. Note the position of the bit in relation to the tongue and soft palate. The double-ended arrow indicates what should be the direction of airflow. Note too the relationship between the soft palate and the larynx. At exercise, the larynx should fit tightly into the soft palate 'button-hole' (ostium intrapharyngium). There should be an airtight seal between the two so that no air can pass from the respiratory tract into the digestive tract or vice-versa. At fast paces, the poll should be extended so that the nasopharynx is stretched longitudinally, allowing it to resist dynamic collapse during inspiration. Because a bit is present, the lips are shown as being unsealed and there is air in the oral cavity and oropharynx. This is not physiological, as at times of exercise all air should be expelled from the digestive tract. Key: brown areas = soft tissue; white areas = bone; red areas = cartilage

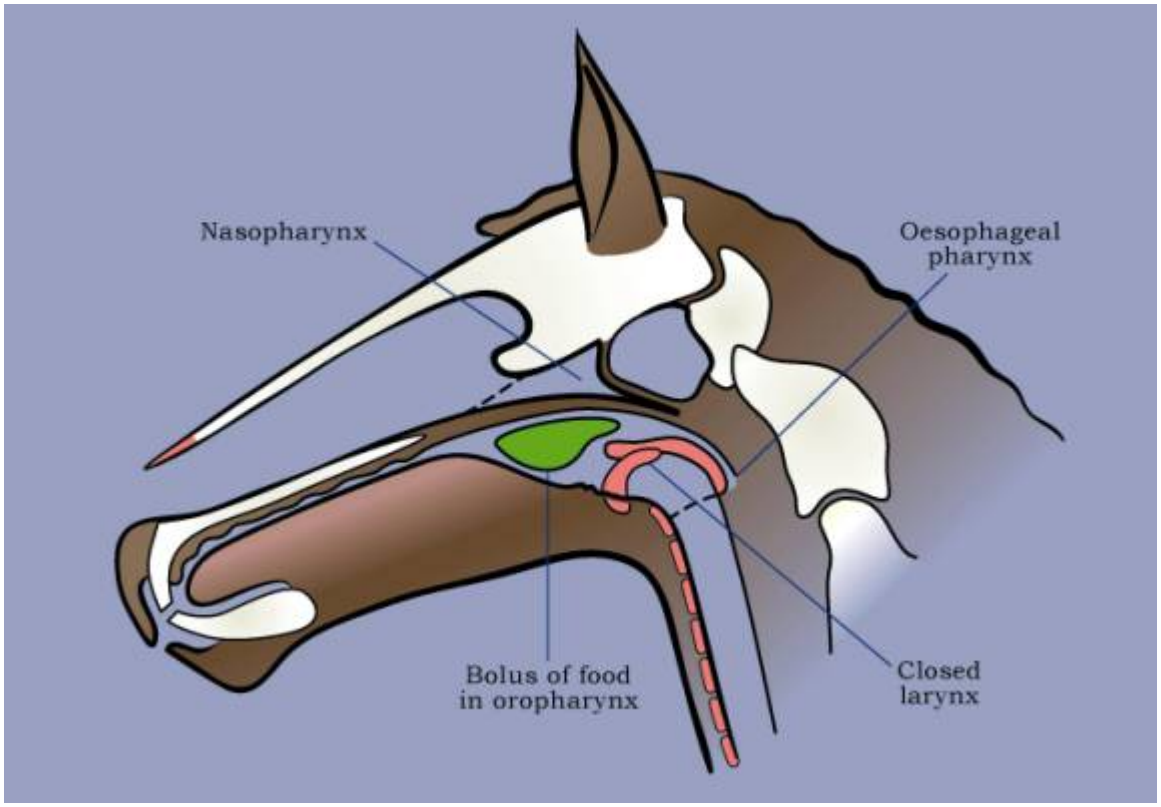

Fig 3. Showing the changes which take place in the pharynx when the digestive system is dominant, i.e. during swallowing of a bolus (green). Under these conditions dorsal displacement of the soft palate (accompanied by complete luxation of the palato-laryngeal junction) is normal. Closure of the laryngeal airway is a corollary to this situation.

2. **The mouth is one of the most sensitive parts of the horse's anatomy, being generously supplied with sensory and pain receptors through the mandibular and maxillary branches of the trigeminal nerve. Bit-induced facial pain might well have the feedback effect of reducing the tone of the nasopharyngeal musculature. This mechanism could be similar to the situation in man when sudden pain in the foot causes a person to fall, due to the limb musculature becoming momentarily**

incapable of bearing weight. Pain might also have the effect of destroying a horse's will to compete. If a horse 'stops trying', then loss of general muscle tone will follow. There will be a relaxation of the strap muscles of the neck (sternothyrohyoideus muscles) together with a tendency for the horse to drop its head and lose the atlanto-occipital extension on which longitudinal stretching of the nasopharynx depends for its ability to resist dynamic collapse on inspiration (2). One minute the jockey will have 'a double handful' as he might say, the next minute the reins will be slack and there will be 'nothing there.' Strong supporting evidence for the existence of bit-induced pain comes from a survey I have carried out recently on 65 skulls of horses five years old or older (Cook W.R: Unpublished material). 75% of these skulls exhibited bone spurs on the diastema of the mandible (Fig 4). The bit undoubtedly causes these exostoses and the pain associated with their presence must be excruciating.

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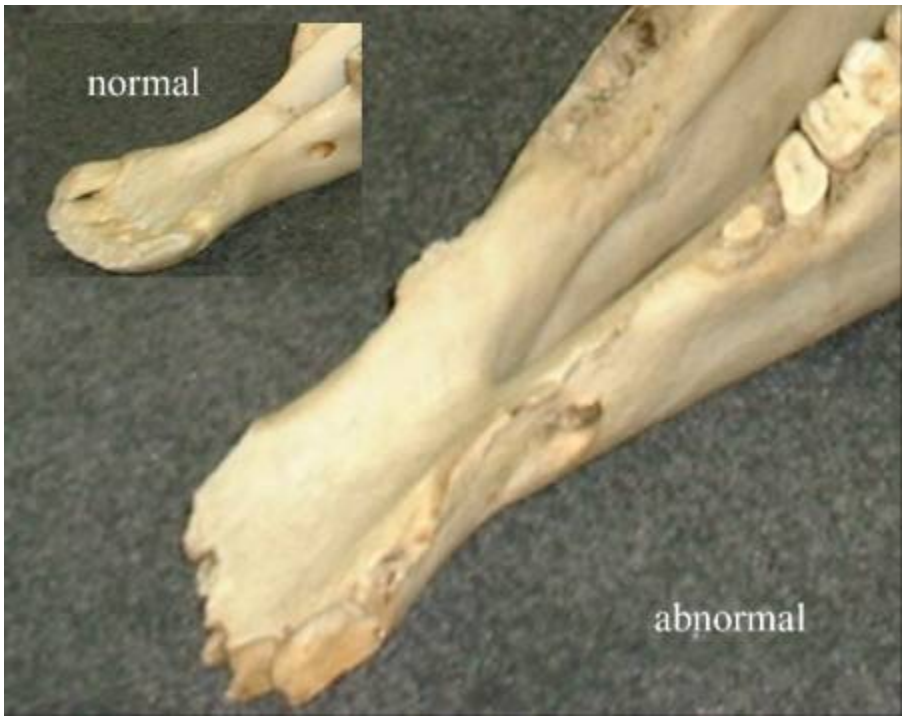

Fig 4. Showing the diastemas of a normal and abnormal mandible for the purpose of comparison. The inset mandible is that of a pony. It may or may not have had a bit in its mouth but the diastema is healthy with a smooth knife-edge to the diastema. This can be best seen in the furthest diastema that is shown in profile. Note, nevertheless, that the 'bars' of the mouth are not flat but knife-edged. Compare this with the abnormal mandible from an old horse and note the presence of a large exostosis on the right diastema. A similar exostosis is present on the left diastema, though it is more difficult to see in the photograph. It is located, as they all are, immediately dorsal to the mental foramen.

3. **The bit causes movement of the jaw. This in turn causes movement of the pharyngeal and laryngeal airways that interferes with the smooth flow of air at high volume and velocity. Movement coupled with turbulent airflow would predispose to DDSPE</P< LI>**
4. **Similarly, bit-induced movement of the tongue will have the same effect. Because tongue and larynx are both suspended from the hyoid apparatus, any movement of the tongue will result in movement of the larynx (Fig 5). A general instability of the hyoid apparatus will lead to instability of the laryngo-palatal junction.**

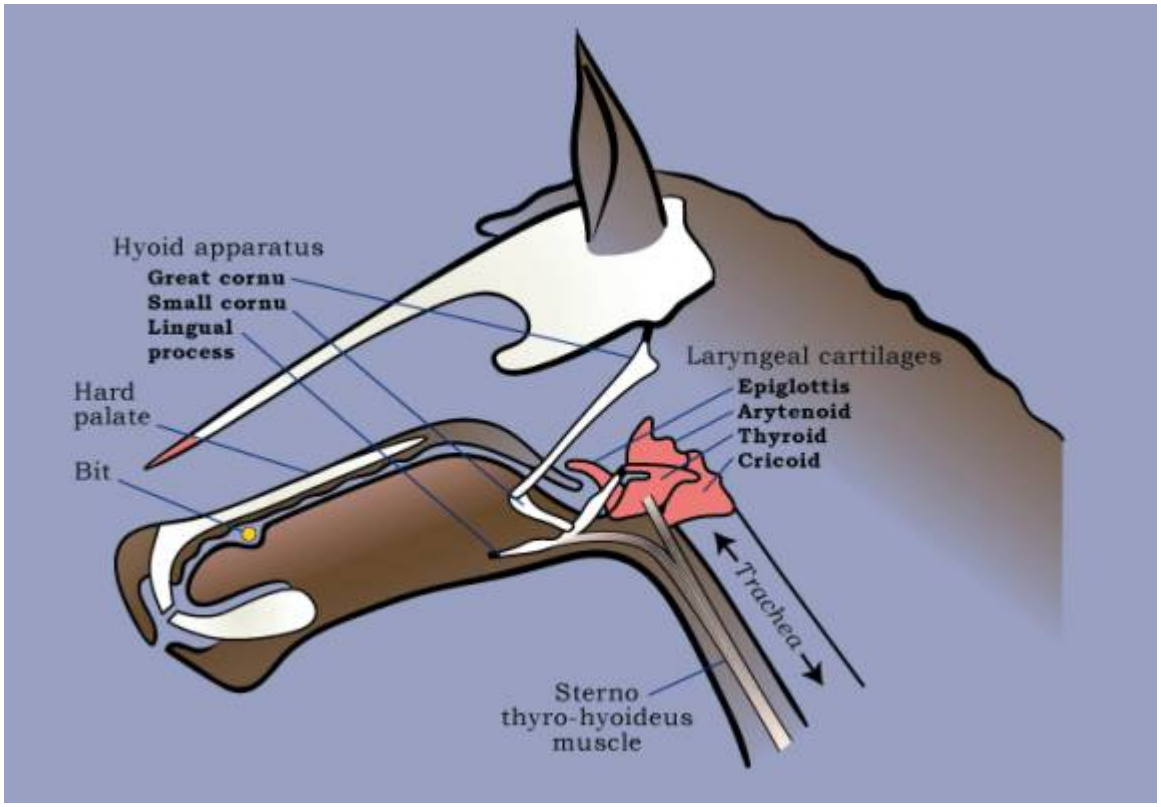

Fig 5. Showing how the larynx and tongue are both suspended from the base of the skull by the hyoid apparatus. As both share a common anchorage, any movement of the tongue caused by the bit is likely to move the larynx, which interferes with breathing.

5. **A horse that evades the bit by drawing the tip of its tongue behind the bit (a common occurrence) causes the root of its tongue to bulge upwards and elevate the soft palate (Fig 6)**

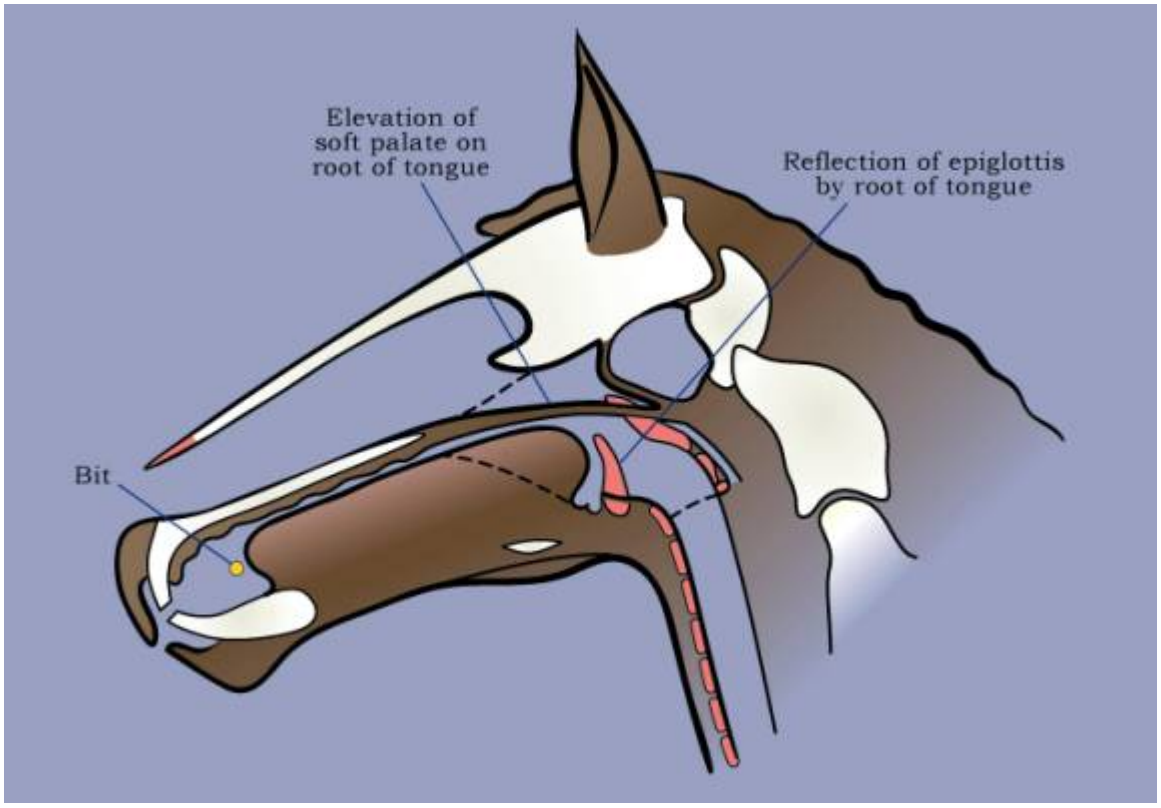

Fig 6. Illustrating the pathophysiology of "swallowing the tongue". If a horse evades the bit, the tip of the tongue is retracted and comes to lie caudal to the bit. The root of the tongue pushes the soft palate dorsally, obstructing the nasopharynx. The broken line indicates where the top line of the root of the tongue should lie (see Fig 2). If DDSP occurs (as in this diagram), the epiglottis also moves caudally and obstructs the entrance to the larynx (aditus laryngis). The horse 'chokes-up' and suffers momentary asphyxiation (see also ESP in Fig 8b).

6. **Presence of a bit breaks what should be an airtight seal of the lips in a horse at exercise. Focal pressure from the bit frequently results in a mouth that is not only poorly sealed but wide open; something one will never see in a horse running at liberty. When air gains entrance to the oral cavity it will also invade the oropharynx. As a result, the soft palate will become elevated on a bubble of air. Once air is present both dorsal and ventral to the soft palate, the aerodynamic conditions are favorable to the onset of a vibratory flapping that may end in DDSPE (Fig 7). Even if the soft palate does not become actually displaced from the epiglottis, the elevation will still constitute a serious impediment to airflow.**

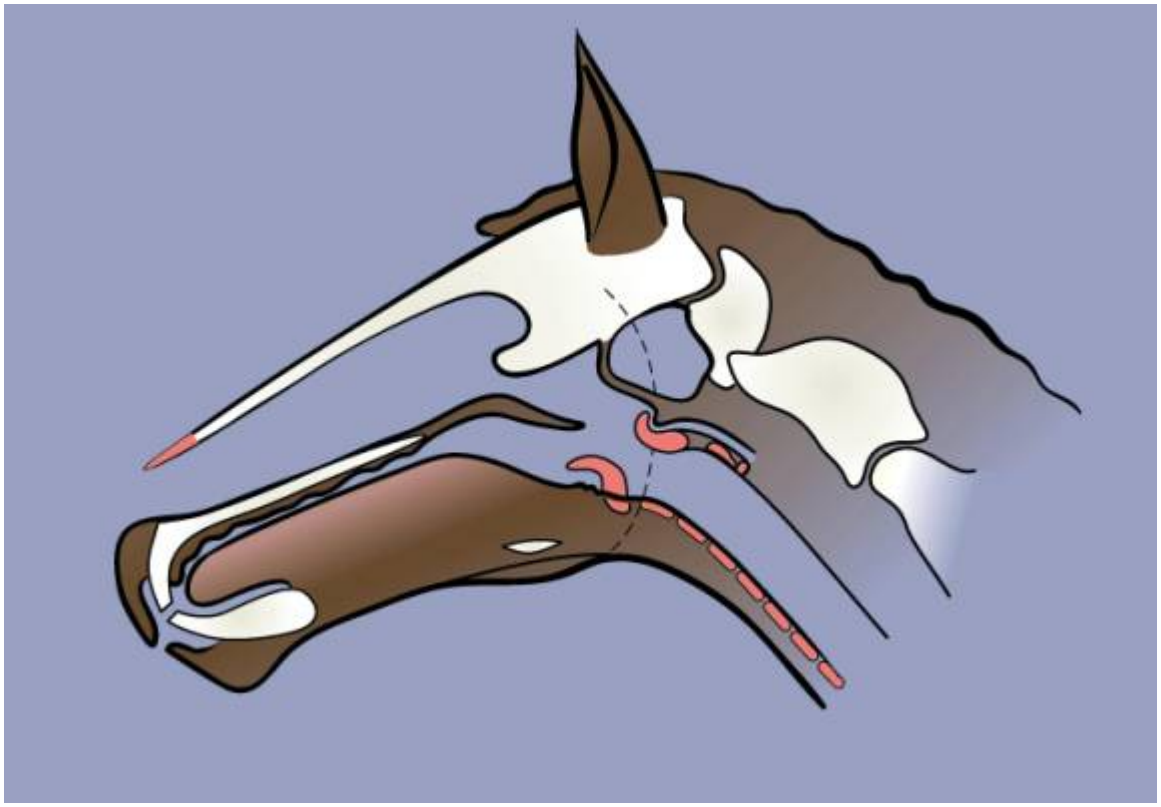

Fig 7. Showing the obstruction of the nasopharyngeal airway that occurs during DDSPE. The obstruction becomes most severe at the level of the posterior nares, which now becomes the narrowest point in the whole airway and its limiting factor with regard to airflow capability. Once the palate is raised, air enters the oropharynx at each expiration. This maintains the problem until such time as the horse can slow up and swallow.

7. **Bit-induced pharyngeal reflexes, such as gag reflexes (spasms of the pharyngeal musculature), might be expected as a result of the presence of a painful foreign body in the oral cavity. Gag reflexes will differ from swallowing reflexes but they may have the same effect as far as the soft palate is concerned.**
8. **Bit-induced salivation also disturbs respiration. This is yet another manifestation of the physiological contraindication that is invoked by expecting a horse to 'eat' and 'exercise' simultaneously. Under these conditions, parasympathetic and sympathetic nervous systems are both vying for dominance. Contrary to long-standing belief, horses at exercise should have a relatively dry mouth (sympathetic dominance). Excess quantities of saliva in the oropharynx might precipitate a true swallowing movement or a laryngeal spasm as a result of saliva creeping into the larynx. We are all aware that saliva is an irritant to the human laryngeal mucosa. The horse has a larynx that is relatively unresponsive to physical stimuli (e.g., the stomach tube) but perhaps it still responds to chemical stimuli.**

9. **Bit-induced poll flexion has the inevitable effect of destroying the longitudinal stretching of the nasopharynx upon which physiological tone in the walls of the nasopharyngeal airway depends (Fig 8). An ability to resist dynamic collapse of the nasopharynx does not, in other words, depend solely on good muscular tone in the relatively tiny stylopharyngeus muscle, the only dilator of the pharynx.**

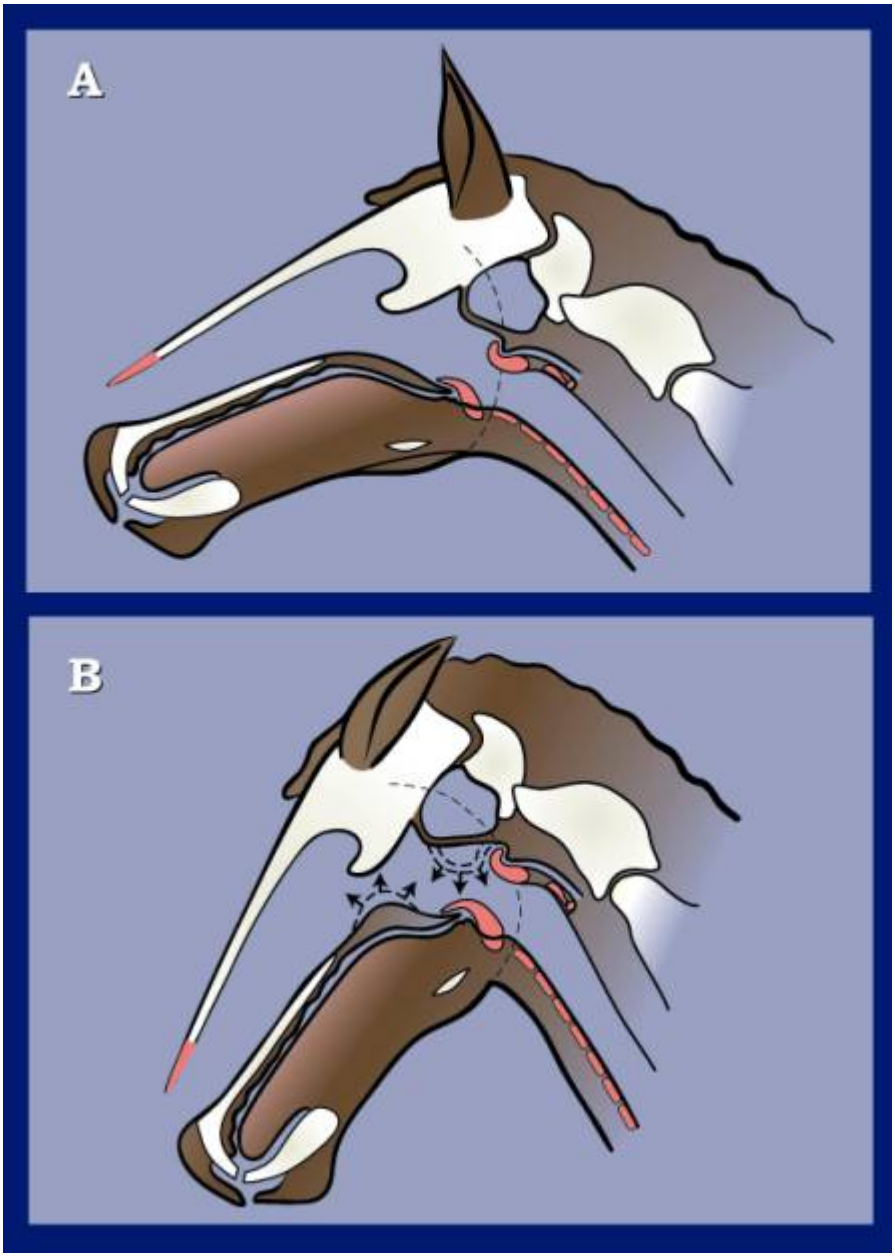

Fig 8. Showing the effect on the nasopharyngeal airway produced by different positions of the atlanto-occipital joint.
a) Full poll extension (jowl angle 140°): The airway is fully patent. The soft tissue boundaries of the nasopharynx are stretched longitudinally, which helps them resist the otherwise collapsing force of inspiration.
b) Partial poll flexion (jowl angle 70°): The airway is sharply bent and seriously

obstructed. The soft tissues of the pharynx further collapse into the airway (see broken lines) during inspiration. If, as in dressage, the horse works with its nasal bone vertical to the ground or, even worse, behind the vertical ('overbent'), the degree of airway obstruction would be even more severe. This diagram illustrates two possible versions of ESP. The central arrow above the soft palate indicates the level of the posterior nares.

10. **A galloping horse, when at liberty, chooses to extend its atlanto-occipital joint. A galloping horse, under saddle and when controlled by a bit, is often expected to perform with a considerable degree of poll flexion. Indeed, the mechanics of a bit are such that it is regrettably good at producing counter-productive flexion of the atlanto-occipital joint. Poll flexion increases respiratory resistance and this, on inspiration, will have the effect of exaggerating the negative pressure that is normally generated in the nasopharynx. This, in turn, will tend to result in elevation of the soft palate.4**

11. **Obviously, the bit is not the cause of recurrent laryngeal neuropathy but, when a bit is used (which it commonly is) and some degree of neuropathy is present (which it commonly is) then the conjunction of the two renders DDSPE more likely. The cascade effect can be explained as follows. First the bit causes airway obstruction in one or more of the ways described above. This increases the work of breathing and abnormally lowers the negative pressure in the nasopharynx and, therefore, larynx. Because of this, the cricoarytenoideus dorsalis muscles will fatigue prematurely, especially the left one, which is the one most likely to be affected with neuro-muscular degeneration. The left arytenoid cartilage and aryepiglottic fold will tend to collapse towards the midline. This loosens what should be an airtight seal at the level of the ostium intrapharyngium, allowing air to enter the oropharynx on expiration, making it more likely that DDSPE will develop. Additional mechanisms could be suggested. For example, the initial airway obstruction caused by the bit might be the cause of a relative hypoxia and this, in turn, would harm the function of all nerves, especially those that are already diseased.**

12. **As a single bit is enough to produce one or more of the above mechanisms, it would not be surprising if two bits were even more likely to have this effect. The Standardbred racehorse is expected to perform with two bits in its mouth; a regular bit and an overcheck. It is not, I feel, a coincidence, that the Standardbred is especially prone to DDSPE. An additional factor is that a driver, as opposed to a rider, is totally reliant on mouth contact for communication. The huge pressure on the horse's sensitive mouth that a seated driver can exert hardly bears thinking about. It would be interesting to have a measurement. Consider the effect of two bits, the mechanical advantage of long lines, and the ability of the driver with his feet braced on the sulky to throw his whole body weight against the virtually unprotected bars of the mouth.**

From the above arguments, I conclude that the bit is the major cause of DDSPE. In hindsight, it is now seems so blindingly obvious that I feel annoyed with myself for not having recognized this etiology years ago. I would suggest that in future the problem might be referred to as Bit-Induced DDSPE. But before anyone starts to use the acronym BIDDSPE let's simplify the nomenclature and at the same time select a name that describes the true nature of the problem by calling it 'bit-induced asphyxia.' Such a name is in accord with 'asphyxia-induced pulmonary edema', the name I prefer for exercise-induced pulmonary hemorrhage (5-8).

The above hypothesis carries with it the prediction that by removing the bit the prevalence of DDSPE should be significantly reduced in frequency or even eliminated. The acid test of this hypothesis will depend on racing stewards allowing horses to race without a bit in their mouths. Such a proposition is certainly feasible since the introduction of the new bitless bridle. Pain-free communication is recommended on the grounds of increased safety to horse and rider, and the resolution of many bit-induced problems (3,4). In the meantime, the hypothesis is being tested on horses in training and in non-racehorses. The good news is that it is withstanding these opportunities for refutation, in that 'gurglers' can be cured by removal of the bit, as can some 'roarers.'

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Tel: (410) 778 9005 E-mail: drwrcook@aol.com
2 Rational treatment is dependent on removal of the cause. As it is the bit that causes tongue movement and evasion of the bit, removal of the bit is the logical treatment, rather than some attempt to immobilize the tongue with a harness.
3 The Bitless Bridle. Bitless Bridle Inc. 2020, South Queen Street, York, PA 17403-

4829 Tel: 1 866 235 0938. Online at www.bitlessbridle.com
4 An increase of negative pressure on inspiration in the nasopharynx is also caused by an obstruction to airflow in the airway at any point rostral to the nasopharynx. Does this mean that any obstruction of the nasal cavity might be considered as a theoretical cause of DDSPE? Obviously, such obstructions are not bit-induced but, nevertheless, for the sake of completeness, should we consider choanal atresia or developmental stenosis, abnormal engorgement of the nasal mucosa (Horner's Syndrome), nasal polyps, tumours or ethmoid hematomata, paralysis of the anterior nares, and any conformational narrowness of the nasal cavity? My answer is 'No', none of these conditions are commonly discovered during examination of horses with DDSPE, and the clinical histories are quite different.