Clinical Commentary

Equine oesophageal dysfunction

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Within this issue of Equine Veterinary Education, Cathcart et al. (2013) confirm that the reported mortality in equine cases of intrathoracic oesophageal oesophageal rupturing is 100%, thereby illustrating the impact of the integrity of the oesophagus in the equine species. Surgery, if applicable, might be an option to increase survival rate, although in a retrospective study only 2 out of 11 horses survived after cervical oesophageal perforation (Craig et al. 1989). Unfortunately, Cathcart et al. (2013) did not mention any history of passing of a nasogastric tube in any time span before referral for suspected colic in this case. The post mortem examination of this case describes the absence of haemorrhage at the edges of the oesophageal tear, indicating this defect to be matured >4 days. It also shows quite mature granulation tissue or even connective tissue (which is known to appear >8 days [Witte and Barbul 1997]) on macroscopic pictures surrounding the tunnelling defect but also linear in formation with the original linear occurrence of the oesophageal tear. This could be suggestive of an even longer existing problem than described for the histopathological changes of the pleural surface in this case report but is difficult to conclude without histopathology of the mucosal oesophageal defect. Iatrogenic perforation may occur in response to excessive force with a stomach tube against an obstruction or a compromised region of the oesophagus (Hardy et al. 1992). Predilection sites for obstructions are located in the proximal oesophagus, at the thoracic inlet, the mid-thoracic region at the level of the base of the heart or just cranial to the cardia (Feige et al. 2000) and one could argue the anatomical distribution to be likely the same for iatrogenic perforation due to passing of a nasogastric tube. Oesophageal mucosal perforations might heal by second intention [Lunn and Peel 1985] and are usually linear of form [Freeman 1989]. It is worth emphasising the potential risk of rupture of the oesophagus associated with nasogastric tubing especially in case of leaving it in position over several days (Hardy et al. 1992). Because of extremely uncommon occurrence of iatrogenic perforation due to passing of a nasogastric tube, there is no need to refrain a horse suffering from colic from nasogastric tubing.

The reported high lethality rate due to perforation in general might be related in part to the colonisation of the epithelium of the equine oesophagus with microorganisms. A permanent mucosal flora of the epithelium of the equine oesophagus has been suggested, predominated by facultative and obligate anaerobic species, which require targeted antimicrobial therapy. The genera isolated most frequently and in highest numbers included streptococci, Prevotella spp., Fusobacterium spp. and Actinobacillus equit. Only 2 groups of Enterobacteriaceae (Escherichia coli and Pantoea spp.) were regularly found and their abundance was lower than that of the other bacterial groups mentioned above (Meyer et al. 2010).

Although the prevalence of oesophageal dysfunction in most horse breeds is rare, abnormalities include oesophageal obstruction with complications such as stricture and diverticulum formation, (traumatic) oesophageal perforation, lack of oesophageal peristalsis and idiopathic muscular hypertrophy of the oesophagus (IMHO). A high incidence of lack of oesophageal peristalsis (and concurrent megaesophagus defined as an abnormal enlargement of the lower part of the oesophagus) has been reported in Friesian horses (Broekman and Kuiper 2002). Barium contrast oesophageal function testing remains the gold standard for diagnosis of lack of oesophageal peristalsis. As has been illustrated in this issue of Equine Veterinary Education by Cathcart et al. (2013), endoscopy might provide additional diagnostic information.

Indeed, the question remains of the aetiopathogenesis of IMHO. It seems most likely to presume a mechanical and/or functional obstruction aborad to the muscular hypertrophy of the oesophagus. In general, the histopathological abnormalities in IMHO usually end abruptly just before the cardia. As a consequence, dysfunction of the lower oesophageal sphincter (LES) might be hypothesised such as increased LES pressure and partial or incomplete LES relaxation. Of note, in 8 out of 31 cases of IMHO, the disorder was seen in concurrence with idiopathic hypertrophy of the funica muscularis of various other parts of the gastrointestinal tract predominantly the ileum [Benders et al. 2004]. Similarly, a mechanical and/or functional obstruction aborad to muscular hypertrophy of the ileum could be suspected i.e. dysfunction of the ileocaecal sphincter/ileocaecal valve. In agreement with the majority of IMHO cases, the case described by Cathcart et al. (2013) showed idiopathic muscular hypertrophy of the oesophagus only.

In man, achalasia is characterised by a lack of oesophageal peristalsis, increased LES pressure and partial or incomplete LES relaxation (Francavilla et al. 2009) and predisposes to formation of diverticuli (Debas et al. 1980) and megaesophagus [Vaezi and Richter 1999]. If lack of oesophageal peristalsis is associated with increased LES pressure and partial or incomplete LES relaxation this might also be classified as (primary) achalasia in the equine species. It might be hypothesised that IMHO and lack of oesophageal peristalsis/megaesophagus are both related to dysfunction of the lower oesophageal sphincter or, in other words, 2 sides of the same coin. Lack of oesophageal peristalsis in accordance with reduced LES relaxation might precede IMHO/megaesophagus. Idiopathic muscular hypertrophy of the oesophagus might be regarded as an adequate compensatory mechanism given the fact that it is usually reported as an incidental post mortem finding similar to Cathcart et al. (2013). Of note, the equine LES is already well known for its very high basal pressure.
In addition to the suggested spontaneous oesophageal perforation secondary to IMHO, one might also suggest that IMHO prevented the mucosal tear from extending towards the thoracic cavity for a thus far unknown period. Furthermore, the possible dysfunction of the LES in affected horses could also be a predisposing influence on iatrogenic perforation by nasogastric tubing if a nasogastric tube had been passed in this horse. In agreement with Cathcart et al. (2013), rupture of the oesophagus is an uncommon but catastrophic event in horses and also to the authors’ knowledge has thus far never been reported to be spontaneous defined as ‘developing without apparent external influence, force, cause, or treatment’ (Pease 2006).

Recently, a new method to assess neurophysiological function of the equine oesophagus was developed (van der Kolk et al. 2011). The procedure involved an endoscopically-guided percutaneous needle EMG procedure performed just caudal to the larynx and just cranial to the thoracic inlet (to monitor striated and smooth muscle neurophysiological activity, respectively) to visualise oesophageal motility (see Figs 1–3). Preliminary findings suggest that endoscopically-guided percutaneous needle EMG might become a valuable method in elucidating the pathophysiology of dysfunction of oesophageal motility in horses.

Intersitial cells of Cajal (ICC) form networks widely distributed within the gastrointestinal tract from the oesophagus to the internal anal sphincter. The ICC generate spontaneously active pacemaker currents that may be recorded as plateau and slow potentials. These pacemaker currents drive the spontaneous electrical and mechanical activities of smooth muscle cells. The role of the ICC and the enteric nervous system in the integrative control of

Fig 1: Overview of EMG procedure (Viking Quest; software version 11.0).© 2013 EVJ Ltd

Fig 2: Cranial position of EMG needle just caudal to the larynx in order to monitor striated muscle neurophysiological activity.
gastrointestinal function and particularly of spontaneous rhythmic activity, is still unknown. Nevertheless, it is likely that the ICC drive spontaneous rhythmic motility; however, a role for the enteric nervous system in the regulation of spontaneous rhythmic motility cannot be overlooked (Takaki 2003; De Ceulaer et al. 2007). In Friesian horses with oesophageal dysfunction, aperistalsis in smooth muscle was associated with the absence of almost any electrophysiological current (van der Kolk et al. 2011) suggesting a loss of neurogenic input at least of the level of the interstitial pacemaker cells of Cajal. In comparison, in human oesophageal achalasia, the myenteric neurons of the oesophagus were affected (Hirano 1999).

Indeed, further investigation is required into the aetiopathogenesis of equine idiopathic muscular hypertrophy of the oesophagus, particularly in relation to the function of the lower oesophageal sphincter and the interstitial pacemaker cells of Cajal.

Authors’ declaration of interests
No conflicts of interest have been declared.

Manufacturer’s address
Viking Quest, Natus Medical Incorporated, San Carlos, California, USA.

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