

# Satellite Article

## Surgery for obstruction of the equine oesophagus and trachea

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### Introduction

A case report in this issue (Gehlen *et al.* 2005) describes an unusual relationship between anomalies in the cervical trachea and oesophagus of a 10-year-old Friesian stallion. The close anatomical proximity of the 2 lesions to each other and timing of onset of clinical signs raise the possibility of a cause and effect relationship. Unfortunately, the true nature of the relationship is complicated by a lack of history for the 6 years of the horse's life preceding transfer to the present owner, lack of clinical signs for the first 30 months in the present ownership and lack of clinical signs attributable to the oesophageal lesion. The latter is all the more remarkable because accumulation of ingesta in the oesophageal diverticulum, sufficient to cause visible swelling in the neck, preceded clinical signs of the tracheal problem by 12 months and was considered responsible for the tracheal deformation and obstruction. However, the tracheal lesions were the only ones causing clinical signs in this case, specifically a loud respiratory noise and marked dyspnoea. The cause of these lesions therefore remains speculative, although the authors' proposal that trauma was responsible is plausible, and trauma could certainly be the most common cause of oesophageal and tracheal lesions in horses. Treatment in this case would be extremely difficult and possibly would afford little success. Management by dietary manipulation would seem reasonable to prevent any future problems with the oesophageal lesions, but the respiratory noise remains in this horse, as expected.

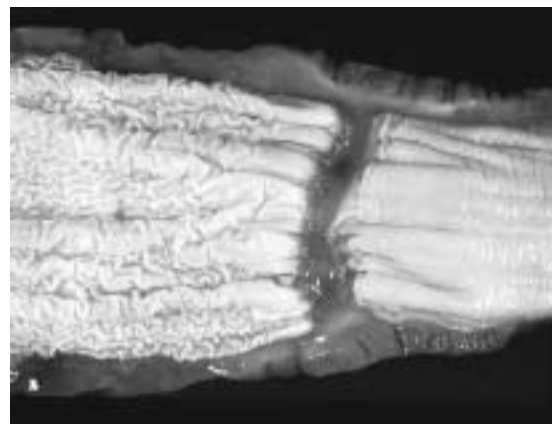
The purpose of this article is to address some of these issues and review available information on the diseases encountered in this case. In addition, the treatment options described for similar oesophageal and tracheal diseases will be discussed, with particular regard as to how they may, or may not, apply to the horse described in the case report.

### Oesophageal stenosis or stricture

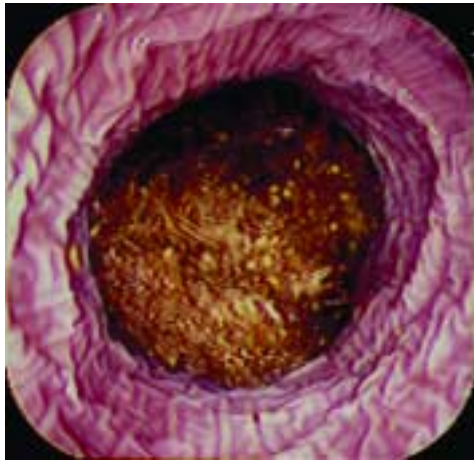
Oesophageal stenosis refers to narrowing of the oesophageal lumen from any cause, whether intra- or extraluminal. Oesophageal stricture can follow any physical, chemical or infectious damage to the oesophageal wall, especially if the

injury is so severe that it heals through fibrous protein synthesis and wound contraction, and is directed around the oesophageal circumference (**Fig 1**). Superficial mucosal defects heal by epithelialisation, whereas full thickness injury to mucosa and oesophageal muscle heal by scarring and stricture formation. Extraluminal causes of oesophageal stricture include trauma to the neck from kicks or collision with objects (Freeman 1982). Congenital strictures and those caused by extension of lymph node abscesses are rare (Priester *et al.* 1970; Deegen *et al.* 1976; Freeman 1982). Oesophageal stenosis from any cause is manifested as recurrent food impactions (**Fig 2**) that can cause proximal oesophageal dilatation (**Fig 1**), weight loss and aspiration pneumonia (Freeman 1982).

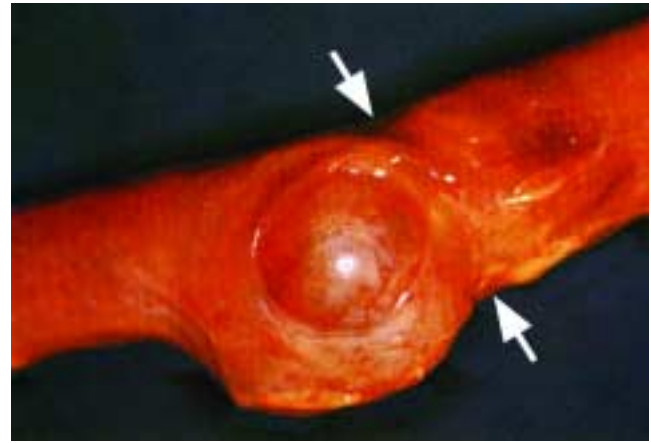
A positive (barium sulphate) or negative (air) contrast oesophagogram can define the stenotic segment, sometimes with abnormal mucosa and annular indentations in the barium column immediately cranial and caudal to the stricture. Barium is superior for demonstrating mucosal lesions, because it will coat and be retained in the mucosal defect for some minutes after the barium column has passed through. Oesophagoscopy should be used to further evaluate mucosal integrity. If the



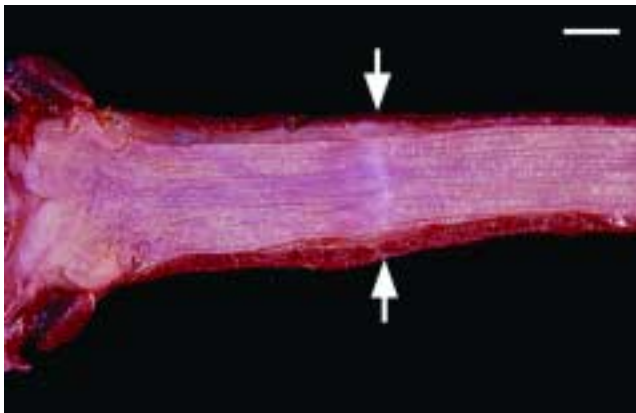
**Fig 1:** A circumferential ulcer that caused chronic oesophageal stenosis and dilatation proximal to it (left), as evident by the proximal oesophageal widening and herringbone pattern in the mucosa. The latter is caused by wrinkling of the redundant mucosa in the nondistended state.



**Fig 2:** Typical endoscopic appearance of a food bolus impacted in the proximal oesophagus at the site of stricture.



**Fig 4:** Typical appearance of a pulsion diverticulum proximal to a stenosis (arrows), with mucosa bulging through a defect in the muscular wall. From Deegen *et al.* (1976), reproduced with permission.



**Fig 3:** Oesophagus at necropsy of a horse that had recurrent choke, annular mucosal ulceration and then stricture formation. Although residual scarring was minimal (between arrows), it was sufficient to cause recurrent obstruction and failure to thrive. Bar = 2 cm.



**Fig 5:** Xeroradiograph of the cervical oesophagus of a horse with a traction diverticulum in the site of a healed oesophagostomy. The longitudinal folds of the oesophagus are intact in the fundus of the diverticulum and there is no disruption of the mucosal lining. External marker is at the site of the original skin incision. From Freeman and Naylor (1978), reproduced with permission.

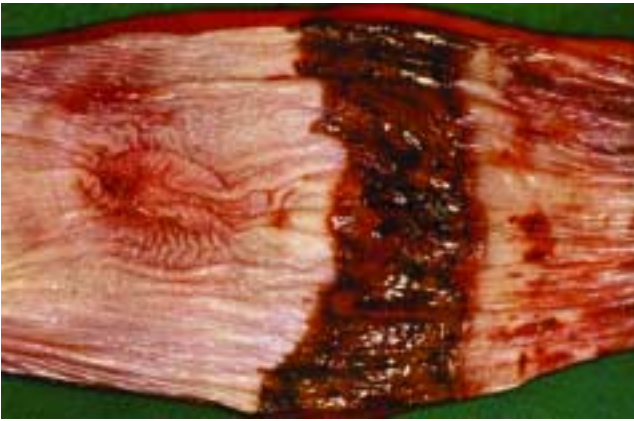
stricture resulted from external injury to the caudal cervical oesophagus, there may be scars in the overlying skin and muscle. The oesophageal mucosa of the horse described by Gehlen *et al.* (2005) appeared to be intact at the stenosis and the oesophageal stenosis was attributed to tissue masses. Based on these descriptions, the images provided and the absence of signs of oesophageal obstruction, the true nature of the stenosis is unknown. If impactions occur at the point of stenosis in the future, they could cause mucosal ulceration and haemorrhage through repeated abrasion (**Fig 1**) and thereby induce scar formation and stricture (**Fig 3**).

### Oesophageal diverticulum

Oesophageal diverticula are well known but uncommon lesions in horses (Craig *et al.* 1989), and the 2 types, pulsion diverticulum (**Fig 4**) and traction diverticulum (**Fig 5**), have been well defined by Gehlen *et al.* (2005). However, the information available to the authors is insufficient to place the diverticulum

of this report in one category or the other, and surgical exposure would be needed to do so in this case. A pulsion diverticulum is a congenital or acquired circumscribed sac of mucosa protruding through a defect in the muscular coat of the oesophagus (**Fig 4**; Deegen *et al.* 1976; Freeman 1982). A pulsion diverticulum can be caused by overstretching and separation of muscle fibres rostral to a stenosis (**Figs 4** and **6**) or an external injury to the ventral aspect of the neck (Deegen *et al.* 1976; Freeman 1982). However, in this case, the diverticulum formed caudal to a stenosis, which is unusual and would rule out a typical cause and effect relationship between the stenosis and diverticulum (**Fig 6**).

Because of its long communication with the oesophagus (20 cm), the diverticulum in this horse resembled a traction diverticulum. A traction diverticulum rarely causes clinical signs because it has a wide neck that does not entrap food material, is shallow and retains a sufficient amount of normal muscle layer so peristaltic waves can keep it empty (**Fig 5**; Freeman



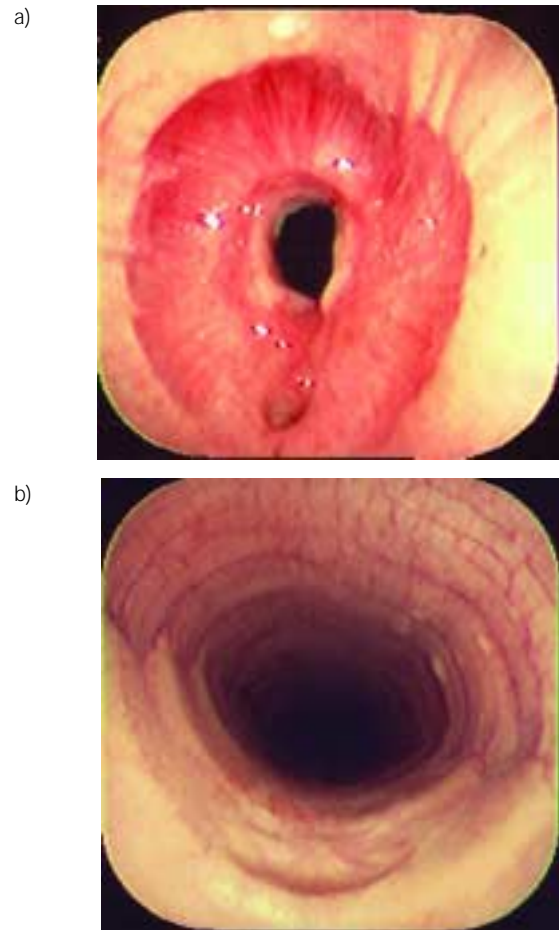
**Fig 6:** Same pulsion diverticulum as in Figure 4 (wrinkled mucosa to the left), opened to show its relationship with a circumferential ulcer distal to it that caused sufficient obstruction to force the mucosa through the oesophageal wall. From Deegen *et al.* (1976), reproduced with permission.

and Naylor 1978). By comparison, the point of evagination is usually quite short in a pulsion diverticulum, which causes food to be retained intermittently in its fundus, thereby causing periodic obstruction (Aanes 1975; Hackett *et al.* 1978). The diverticulum in this report also had features of a pulsion diverticulum, such as location in the caudal third of the cervical oesophagus and its appearance as a bulge or swelling that could be eliminated by massage of its contents (Aanes 1975; Hackett *et al.* 1978; Freeman 1982).

On radiographs, a pulsion diverticulum appears as a club-shaped, pyriform or cone-shaped swelling with an accumulation of gas and inspissated food material in its fundus. Feeding barium sulphate in grain is more suitable as a contrast oesophagogram for pulsion diverticula because this mixture is more likely to gravitate into the depths of the diverticulum than liquid contrast (Hackett *et al.* 1978). With liquid barium sulphate, a traction diverticulum appears as a small protrusion of the barium column outside the normal oesophageal lumen that empties readily (Fig 5; Freeman and Naylor 1978). An intrathoracic pulsion diverticulum can assume enormous proportions and cause local compression atelectasis, because there are no solid structures surrounding it to prevent its expansion into the pleural cavity (Franken *et al.* 1978).

### Tracheal obstruction

As stated by Gehlen *et al.* (2005), tracheal obstruction is rare in horses and most cases are caused by injury, either from blunt trauma to the trachea or following tracheostomy (Fig 7). Congenital deformities of the trachea are also possible (Freeman 1990), although unlikely in a horse of this age at the time of recognition. Tracheal compression by extraluminal masses, such as lymph node abscesses (Randall and Myers 1973), lipoma (Yovich and Stashak 1984), mediastinal masses and tumours (Lane 1981), are rare causes of tracheal stenosis, but would share the same pathogenesis proposed for the horse in the accompanying report. It is very unusual for any



**Fig 7:** a) Tracheal stenosis with severe web formation caused by a tracheostomy that damaged cartilaginous rings and almost transected the trachea. b) A more typical appearance of the healed trachea after a tracheostomy that preserved tracheal rings by placing the incision between them.

type of oesophageal diverticulum to compress the cervical trachea, because there is little restriction to tracheal displacement away from the swelling in the ventral part of the neck. Compression seems more likely in the confined space of the thoracic inlet, but the oesophageal lesions were cranial to the thoracic inlet in this horse.

### Collapsed trachea

Collapsed trachea in horses is probably a congenital defect and usually involves the entire cervical and/or thoracic portions of trachea, unlike acquired tracheal stenosis, which usually involves a short segment of cervical trachea. The condition is also described as dorsoventral flattening of the trachea and 'scabbard' trachea (Freeman 1990). The affected tracheal rings form half circles with widely separated ends and the dorsal membrane that spans this space is thin and flaccid. As in dogs (Nelson 2003), miniaturisation appears to be a factor in development of collapsed trachea in horses, because ponies and miniature horses are predominantly affected (Delahanty and Georgi 1954; Rothenbacher 1965;



**Fig 8:** A 3-year-old Standardbred filly with an oesophagostomy tube in place. The tube was placed on the ventral midline of the neck, immediately caudal to the bandage, and maintained for 42 days.

Carrig *et al.* 1973; Martin 1981; Simmons *et al.* 1988). This size predisposition would make such a lesion unlikely in the horse in this report. Most affected animals are age  $\geq 10$  years at the time of diagnosis, and the disease is frequently diagnosed as an incidental finding at necropsy in horses or ponies that had a sedentary lifestyle and therefore appeared clinically normal (Delahanty and Georgi 1954; Rothenbacher 1965; Carrig *et al.* 1973; Martin 1981; Simmons *et al.* 1988). Clinical signs may develop or worsen in later life because ageing changes in affected cartilages, such as calcification and inflammation, can exacerbate the obstruction (Martin 1981; Simmons *et al.* 1988).

A 'honking' respiratory noise can become evident on inspiration and expiration (Martin 1981) and is usually exacerbated by exercise, although not consistently (Hanselka 1973). Dynamic tracheal collapse has been reported as a cause of exercise intolerance in a 2-year-old Thoroughbred filly (Tetens *et al.* 2000). Some horses cough (Simmons *et al.* 1988) and have signs similar to those of heaves-affected horses (Rothenbacher 1965); severe cases can become debilitated and have cyanotic mucous membranes (Delahanty and Georgi 1954; Rothenbacher 1965; Carrig *et al.* 1973). The ends of flattened tracheal cartilages can be palpated in some horses as sharp ridges along the course of the cervical trachea (Delahanty and Georgi 1954; Carrig *et al.* 1973) and the severe dorsoventral flattening of the trachea and flaccid dorsal membrane can be seen on endoscopy (Delahanty and Georgi 1954; Simmons *et al.* 1988) and on radiographs (Blikslager and Sweeney 1991).

## Oesophageal surgery

Oesophageal surgery in the horse can be daunting. The relatively poor healing of oesophageal repairs can be attributed to excessive motion during swallowing and respiration, poor suture holding in oesophageal muscle and mucosa, and absence of a serosal layer to seal anastomotic leaks. Also, horses feed over long periods, ingest coarse and abrasive food and their oesophagus must empty against gravity, features that provide a critical test of the integrity of an oesophageal repair

or anastomosis. The oesophageal lumen diameter is small in horses relative to that of other animals and additional narrowing is poorly tolerated (Fig 3). Depending on the surgery, integrity and location of the repair (cervical less critical than thoracic oesophagus), reintroduction to food should be gradual and slow. Liquid diets can be administered by nasogastric or oesophagostomy tube (Fig 8; Freeman and Naylor 1978), and horses should probably be denied solid food for at least a week after any surgery that breaches the mucosal lining and requires first intention healing. The most suitable form of solid food is a complete pellet diet soaked in warm water, although some horses find this unpalatable.

Surgical exploration in the horse described by Gehlen *et al.* (2005) would have provided useful information on the nature of the oesophageal and tracheal lesions. In general, surgery on these structures should be based on solid information about the nature of the problem beforehand, but this is not always possible, and it is not unusual for intraoperative findings to be different to those obtained preoperatively (Ford *et al.* 1991). The following are descriptions of possible surgical treatments for the lesions found in this horse, but the suitability of any treatment would have to be governed by intraoperative findings. In addition, staggering of the surgical treatments would be advisable, starting with exploration and removal of the oesophageal diverticulum, then observing the airway response to relief from compression. An unsatisfactory response would justify a later surgery to treat the tracheal defect.

## Surgery for oesophageal stenosis

Stenosis and strictures have been successfully treated with resection and anastomosis (Lowe 1964; Vaughan and Hoffer 1963; Suann 1982; Gideon 1984), mucosal resection and myotomy (Derksen and Stick 1983), patch grafting (Hoffer *et al.* 1977), combined oesophagostomy and cicatrix fenestration (Craig and Todhunter 1987), balloon dilation (Tillotson *et al.* 2003), oesophagomyotomy (Wagner and Rantanen 1980; Nixon *et al.* 1983) and combined oesophagomyotomy and oesophagopexy (Lillich *et al.* 2001). Mild cases are managed conservatively with a diet of grass and soaked, pelleted complete feeds (Todhunter *et al.* 1984). In the case reported by Gehlen *et al.* (2005), conservative management through dietary manipulation was an appropriate choice for the stenosis, especially because it did not cause signs of oesophageal obstruction.

## Surgery for oesophageal diverticulum

Pulsion diverticula in the cervical oesophagus can be treated successfully by surgery (Hackett *et al.* 1978), whereas traction diverticula usually do not require treatment (Freeman and Naylor 1978). Diverticulectomy is the treatment of choice for a large pulsion diverticulum that has a narrow communication with the oesophageal lumen or for a diverticulum that is inflamed and necrotic (Frauenfelder and Adams 1982; Freeman 1982; Ford *et al.* 1991). At surgery, the diverticulum can be identified as thin-walled oesophageal mucosa bulging

through a linear tear in the muscularis (Ford *et al.* 1991). After the diverticulum has been isolated, its contents are gently massaged into the oesophagus or removed to the stomach by water delivered forcefully through a nasogastric tube (Ford *et al.* 1991). The oesophagus is packed off from surrounding tissues and the redundant mucosa excised, taking care to leave an ample oesophageal lumen, and closed with size 2-0 polydioxanone in a simple continuous or interrupted pattern. Alternatively, the mucosa of the diverticulum can be resected by placing a row of staples across the tented portion of mucosa with an automatic stapling instrument (Ford *et al.* 1991). The oesophageal muscularis and submucosa are then closed over the stapled diverticulum with a single layer of size 2-0 or 0 polydioxanone in an interrupted Lembert pattern (Ford *et al.* 1991) or a simple interrupted pattern (Frauenfelder and Adams 1982). A nasogastric tube should be able to be passed easily through the repaired section of oesophagus.

For small or shallow diverticula with an intact mucosa, the redundant oesophageal mucosa that forms the fundus of the diverticulum is inverted into the oesophageal lumen and the edges of the surrounding muscle wall defect are trimmed back to normal tissue (Aanes 1975; Hackett *et al.* 1978). This step may be difficult because the submucosal layer of a pulsion diverticulum is usually more vascular than normal and may be enveloped in scar tissue. Once the inversion is completed, the edges of the muscle defect are apposed with interrupted sutures of absorbable material. Lumen diameter can be preserved by repairing the muscle wall defect transversely to the long axis of the oesophagus or, in the case of a V-shaped defect, by closing it with a Y-plasty (Hackett *et al.* 1978). The main advantage of this method is that it avoids invasion of the oesophageal lumen, with the subsequent risk of leakage, sepsis and fistula formation (Aanes 1975).

### ***Surgery for compressed and collapsed trachea***

Removal of the oesophageal diverticulum might not have provided respiratory relief in the horse in the accompanying report, because the involved tracheal rings can remain collapsed after decompression by removal of an impinging mass or drainage of an abscesses (Randall and Myers 1973; Yovich and Stashak 1984). Tracheal reconstruction might be required (Yovich and Stashak 1984), although the repair will be difficult and carry a poor prognosis if a long segment of trachea is involved. Treatment methods in the cervical trachea include tracheal imbrication (Delahanty and Georgi 1954), extraluminal support (Simmons *et al.* 1988) and replacement of a segment of trachea with a prosthesis (Carrig *et al.* 1973). These methods are preferable to resection and anastomosis whenever possible, because complications are less severe, but they are of limited value if there is mucosal damage and severe cartilage injury. The most popular material for extraluminal support is a 60 ml polypropylene syringe case, cut in half longitudinally and with many small holes drilled in it (Hedlund and Tangner 1983; Yovich and Stashak 1984; Nelson 2003; Robertson and Spurlock 1986). If a long segment of trachea is involved, several individual plastic rings may be preferable to one solid piece

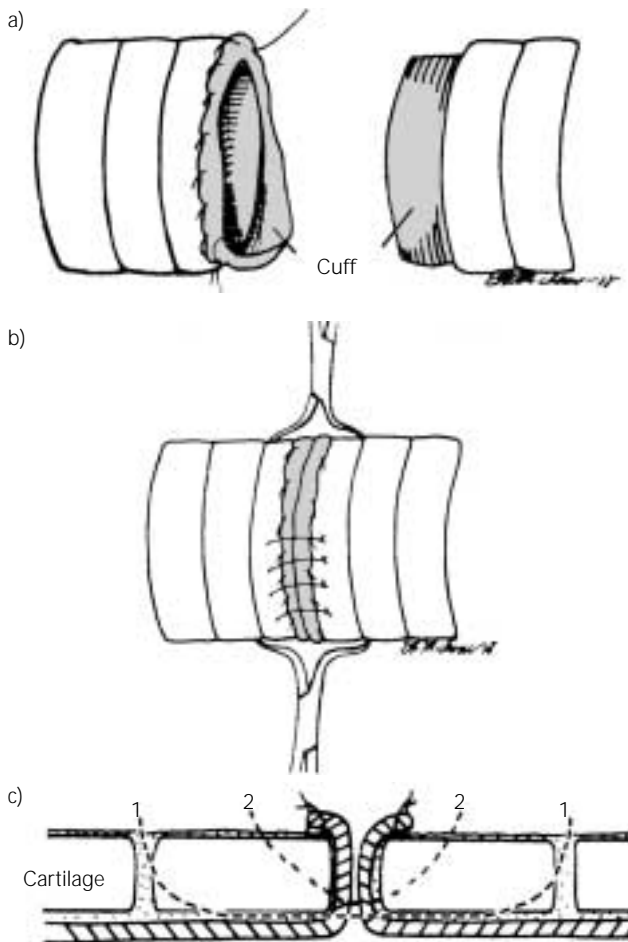
because they would permit more normal tracheal flexibility and movement (Nelson 2003). An alternative support is a wire spring covered with polyethylene tubing and spiralled onto the affected segment (Horney 1975). Several partial or full-thickness incisions may be necessary across the width of each affected tracheal ring to allow reshaping (Robertson and Spurlock 1986). Fine, monofilament, nonabsorbable sutures, such as polypropylene, are preplaced as needed around the affected segments of tracheal rings to elevate them to a normal position (Nelson 2003; Robertson and Spurlock 1986). Extraluminal supports in horses can eliminate noise and dyspnoea (Goulden 1977) and allow a successful racing career (Robertson and Spurlock 1986). Failures with external supports can be attributed to infection, soft tissue damage from movement against an unyielding implant, or damage to adjacent nerves.

### ***Tracheal resection and anastomosis***

Resection and anastomosis of the trachea are difficult in horses and should be reserved for those acquired tracheal stenoses in which other reconstructive techniques have failed or for horses with short segments ( $\leq 5$  tracheal rings) affected by mucosal fibrosis, induration, granulation tissue, polyp formation, intraluminal webbing (**Fig 7**), thickening of cut cartilage rings, dystrophic changes in affected cartilages, telescoping of cut edges of cartilages over each other and tracheal rupture (Freeman 1990; Kirker-Head and Jakob 1990). Three tracheal rings can be resected in a horse without much difficulty, but twice as much force is required to appose the ends when 5 rings are removed (Tate *et al.* 1981). Therefore, the applicability of anastomosis to the horse in the accompanying report would be determined by the condition of the tracheal rings and the number of rings involved.

If tracheal resection and anastomosis are required, the technique described by Tate *et al.* (1981) is strongly recommended, because it minimises tension on the suture line and achieves precise anatomical reconstruction of mucosa and cartilage. To minimise tension, a martingale apparatus is used for anaesthetic recovery and for 3 weeks after surgery to maintain head and neck flexion (Tate *et al.* 1981). This is applied the week beforehand to allow the horse time to adjust (Tate *et al.* 1981). With the horse in dorsal recumbency, the trachea from the cricoid cartilage to the thoracic inlet is exposed and mobilised through a 40 cm long ventral midline incision, without any apparent risk of disrupting blood supply (Tate *et al.* 1981). Such mobilisation can be needed to reduce tension in the suture line, but damage to the vagosympathetic trunk or recurrent laryngeal nerve must be carefully avoided (Tate *et al.* 1981). The affected segment is resected, but a cuff of normal mucosa of 360° circumference is left to extend for at least 1 cm beyond the tracheal rings to be apposed (**Fig 9**). These mucosal cuffs are then reflected over the normal tracheal cartilages and sutured to the adventitia with fine, absorbable suture material (size 2-0 polydioxanone) in a simple continuous pattern (Tate *et al.* 1981).

The severed ends of trachea are drawn together by 4 Backhaus towel clamps (**Fig 9**) while the head and neck are



**Fig 9:** a) After the abnormal segment of trachea is resected, a cuff of mucosa (shaded) is left on each end of the remaining trachea, turned back and sutured to the adventitia. b) The 2 ends of the trachea are brought into apposition and sutured together with interrupted sutures of steel or size 1 polydioxanone. c) Methods of suturing the 2 end tracheal rings together so that the reflected ends of mucosa abut and thereby seal the anastomosis: the author prefers to use size 1 polydioxanone in a deeper and wider bite through each ring (dashed line 1) than was described in the original report, in which each stainless steel suture penetrated half the thickness of the tracheal cartilage (dashed line 2). With both methods, the mucosa is not penetrated. From Tate *et al.* (1981), reproduced with permission.

placed at a 90° angle. The approximated tracheal rings are anastomosed with simple interrupted sutures of 25 g stainless steel wire swaged to a curved cutting needle (Fig 9). Sutures are placed 0.5–1 cm apart and through half the width of each tracheal ring, without penetrating the tracheal mucosa (Tate *et al.* 1981). Sutures placed directly through cartilaginous rings can fracture the cartilage, but sutures that encircle the cartilage tend to telescope apposed tracheal rings. Risks of infection, granuloma formation and mucosal ulceration increase if suture material enters the tracheal lumen, especially braided material (Tangner and Hedlund 1983). The trachea is checked for air leaks by flooding the surgical site with antibiotic in saline solution and forcing air through the endotracheal tube with a ventilator.



**Fig 10:** Endoscopic view of a tracheal anastomosis 24 h after surgery for removal of 3 abnormal rings and a stenosis in the same horse as in Figure 7a. Note that a tracheostomy tube placed before surgery was still in place but was considered unnecessary after this endoscopic examination.



**Fig 11:** Endoscopic view of healed anastomosis in the same horse as in Figures 7 and 10 at 17 months after tracheal resection and anastomosis. The filly was in race training at this stage.

The author has modified this procedure by using a shorter ventral midline incision (15 cm) to remove 3 cartilaginous rings distorted by a tracheostomy error (Fig 7). Instead of wire, the tracheal rings were apposed with a simple interrupted pattern using size 1 polydioxanone and taking a substantial bite into the tracheal cartilages (Fig 9). The angled endotracheal tube described in the original report to intubate the distal segment (Tate *et al.* 1981) was not used. The outcome in this horse was a marked improvement in airway diameter (Fig 10), and the horse returned to race training (Fig 11).

## Conclusions

Some of the preceding diseases and treatments could apply to the horse in the accompanying report (Gehlen *et al.* 2005), although additional information about the tracheal and oesophageal lesions would be required before deciding on appropriate treatment. Also, many of these treatments

are aggressive and fraught with complications that could be more difficult to handle than the original problems. However, any progression of either lesion would be a reason to consider surgical management, while the other could be managed conservatively.

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