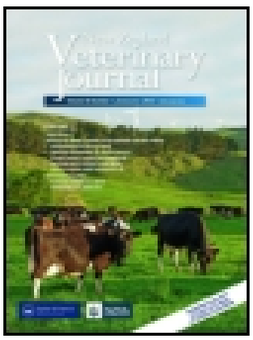

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CLINICAL COMMUNICATION

Laryngeal paralysis secondary to cervical bite injuries in five dogs

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ABSTRACT

Case histories: Medical records of a veterinary hospital in Belgium were reviewed for dogs (n = 5) that presented between 2016 and 2019 with laryngeal paralysis secondary to bite wounds to the cervical region received while fighting with other dogs. The time elapsed between the trauma and presentation was from a few hours up to 5 days.

Clinical findings and treatment: Bilateral laryngeal paralysis was identified in three dogs and unilateral laryngeal paralysis in two dogs via endoscopic assessment of laryngeal function. The primary concomitant lesions included tracheal injury in 3/5 dogs and oesophageal injury in 1/5 dogs. One dog with bilateral laryngeal paralysis was treated medically as no signs of dyspnoea were present. Surgical management was elected in 4/5 dogs based on evaluation of their clinical status and lesions revealed by endoscopic examination of upper gastrointestinal and respiratory tracts. Dogs underwent surgical procedures that were determined to be appropriate for treatment of the lesions identified on clinical examination, diagnostic imaging, and endoscopy. The cervical region was explored through a ventral midline approach in 2/4 cases, to close tracheal perforations. Temporary tracheostomy was performed in 2/4 cases. Procedures to correct brachycephalic airway obstructive syndrome were performed in 2/4 cases. Cricoarytenoid lateralisation was performed in 2/4 dogs. Dogs were hospitalised for 2–10 days and received antimicrobial therapy before surgery and for 2–3 weeks after surgery. Physical examination and respiratory function were normal in 3/5 dogs 4–6 months after discharge. Information regarding outcomes for two cases was obtained from the owners by telephone assessment 1–6 months after surgery. The owner of each dog reported the respiratory function to be excellent.

Diagnosis: Uni- or bilateral, transient or permanent laryngeal paralysis with concomitant oesophageal, tracheal, or laryngeal lesions following cervical dog bite injuries diagnosed by endoscopic examination of upper gastrointestinal and respiratory tracts.

Clinical relevance: This case series describes the diagnosis and management of dogs with laryngeal paralysis secondary to cervical dog bite injuries. To the authors' knowledge, this is the first published report documenting bilateral laryngeal paralysis secondary to cervical dog bite injuries. Clinicians should be aware of this pathology and the importance of investigating laryngeal function in dogs presenting with cervical bites, particularly those with inspiratory dyspnoea. Upper airway and digestive endoscopy are recommended for complete assessment of cervical traumatic injuries.

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Laryngeal paralysis; cervical; bite; fighting; dog; wound

Introduction

Fights between dogs frequently result in injuries to the cervical region. Previous studies have reported that 20–29% of bite wounds occur in the cervical region, and this is the most frequent location for bite wounds in medium and large breed dogs (Shamir *et al.* 2002; Basdani *et al.* 2016). During dog fights, the skin and/or underlying tissues may be lacerated, stretched, crushed, and/or avulsed (Shamir *et al.* 2002). Due to the proximity of vital structures, wounds to the cervical region can lead to laryngeal paralysis, laryngeal avulsion, tracheal tear, oesophageal tear, and other conditions, which may be life-threatening.

Laryngeal paralysis is the inability to abduct the arytenoid cartilages and vocal folds during inspiration resulting in partial airway obstruction. The aetiology may be congenital (hereditary laryngeal paralysis or congenital polyneuropathy), or acquired (trauma, neoplasia, polyneuropathy, endocrinopathy) (Kitshoff *et al.* 2013). Innervation to the laryngeal muscles is provided by the cranial and caudal laryngeal nerves which originate from the vagus nerve (Hermanson and Evans 1993). The cranial laryngeal nerve leaves the vagus at the distal ganglion, which lies external to the tympano-occipital fissure, and ventral and medial to the tympanic bulla. It then passes ventral to the larynx, supplying the cricothyroideus muscle and receiving sensory fibres from laryngeal mucosa (Evans and Kitchell 1993). The vagus nerve runs through the vagosympathetic trunk dorso-parallel to the common carotid artery, and within the thoracic cavity, branches into the recurrent laryngeal nerves. The left recurrent laryngeal nerve leaves the left

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vagus, arches around the aorta, ascends along the trachea, and ends as the left caudal laryngeal nerve. The right recurrent laryngeal nerve leaves the right vagus, loops around the right subclavian artery and ascends along the trachea to end as the right caudal laryngeal nerve (Evans and Kitchell 1993). All intrinsic laryngeal muscles except the cricothyroideus are innervated by the caudal laryngeal nerve (Hermanson and Evans 1993). Therefore, traumatic damage (e.g. bite wounds, surgical trauma, tumours) to the cervical region may cause acquired laryngeal paralysis (Monnet and Tobias 2018).

Nerve injuries have been divided into three broad categories, associated with the anatomical extent of nerve injury and with the chance of a spontaneous recovery of function after trauma. Neurapraxia, the mildest type of injury, does not involve loss of nerve continuity and causes transient functional loss. Axonotmesis occurs when there is complete interruption of the nerve axon and surrounding myelin while the perineurium and epineurium are preserved. Axon and myelin degeneration occur distal to the injury, causing complete denervation. Prognosis for recovery is usually good because the remaining uninjured mesenchymal layers provide a path down which axons can regrow. Neurotmesis involves complete transection of a nerve. Functional loss is complete and recovery without surgical intervention does not usually occur because of scar formation and loss of the mesenchymal guide that directs axonal regrowth (Burnett and Zager 2004).

The larynx of animals with severe cervical injuries or evidence of inspiratory stridor should be directly inspected to identify laryngeal paralysis and rule out other causes of laryngeal stridor such as laryngeal obstruction (Broome *et al.* 2000; Jordan *et al.* 2013). Peroral laryngoscopy is considered to be the gold standard for diagnosis of these conditions (Broome *et al.* 2000; Radlinsky *et al.* 2009). When laryngeal inspection is inconclusive, administration of doxapram hydrochloride may be useful as it stimulates vigorous respiration which in dogs with laryngeal paralysis may lead to the collapse of the arytenoids during inspiration (Tobias *et al.* 2004).

While traumatic damage to the cervical region is common during dog fights, cervical bite wounds as a cause of acquired laryngeal paralysis remains rare. In a retrospective study gathering 56 cases of airway injury associated with cervical bite wounds, two dogs suffered from documented damage to the recurrent laryngeal nerve resulting in unilateral laryngeal paralysis which was not clinically relevant (Jordan *et al.* 2013). Here we report the diagnosis, management, surgical procedures, and outcomes for five dogs with acquired uni- or bilateral laryngeal paralysis secondary to cervical bite injuries.

Case histories

Medical records were reviewed to identify dogs diagnosed with laryngeal paralysis secondary to dog bites to the cervical region between 2016 and 2019 at the Clinique Vétérinaire Universitaire (Faculty of Veterinary Medicine of the University of Liège; Liège, Belgium). Cases were included if they presented with uni- or bilateral laryngeal paralysis assessed by endoscopic examination and a recent history of dog bites to the cranio-cervical region. Information obtained from medical records included the signalment, the time elapsed since the bite occurred, clinical signs at presentation, results of diagnostic imaging and endoscopic examination, and if performed, the surgical findings and outcome. Surgical data gathered included the details of anaesthesia and procedure performed. Post-operative information obtained included type and duration of medical treatment, complications associated with the injury or treatment and duration of hospitalisation. Information regarding follow-up and outcome were also reviewed. For dogs that had not been physically examined after recovery, owners were contacted by telephone.

Five dogs of different breeds met the inclusion criteria and were included in the study (Table 1). The median age of the dogs was 7 (min 2, max 13) years and the median weight was 11.2 (mean 12.1; min 2.2, max 24.6) kg. The time elapsed since the dog fight varied from a few hours to >72 hours with 4/5

Table 1. Signalment, weight, time elapsed since injury at presentation, and location and type of wounds of five dogs that presented to a veterinary hospital in Belgium with laryngeal paralysis secondary to dog bites to the cervical region.

						Bite wound	
Case	Breed	Sex	Age (years)	Weight (kg)	Time elapsed (hours)	Location	Type and status
1	French Bulldog	FN	2	11.2	<24	Ventral cervical region and pinna	Punctiform
2	Miniature Pinscher	MN	9	5.4	24–72	Ventral cervical and perineal regions, limbs	Mild, moderate depth, mild sero-sanguinous discharge
3	Mixed breed	M	13	17.1	>72	Dorsal cervical	Punctiform, healing phase
4	Chihuahua	F	3	2.2	<24	Ventral cervical	Mild, moderate depth, mild sero-sanguinous discharge
5	English Bulldog	M	7	24.6	24–72	Dorsal cervical region and pinna	Punctiform

F = female; M = male; N = neutered.

presenting within 72 hours. One dog was referred 5 days after the trauma due to worsening general condition and dyspnoea, suspected to be secondary to bronchopneumonia. All dogs were reported to be clinically normal before the event.

Clinical and diagnostic findings

All dogs presented with wounds to the cervical region caused by dog bites. For 3/5 dogs the cervical wounds were located ventrally while for 2/5, the wounds were in the dorsal cervical region. Three dogs also had wounds in other locations. The location and type of wounds are described in Table 1. Other commonly recorded clinical signs included dyspnoea, S/C emphysema, tachycardia, stertor, and stridor (Table 2). The results of neurological examinations were within normal limits for all dogs. A grade 3/6, left-sided heart murmur was incidentally detected in case 4.

Case 1 was initially stabilised with oxygen supplementation (face mask followed by nasal cannula) and an I/V bolus of 10 mL/kg isotonic crystalloid fluids (Ringer Lactate Vetoflex; Bioluz, St Jean de Luz, France) within 15 minutes before any diagnostic procedures were performed. Based on clinical examination the other cases were judged not to require stabilisation before diagnostic evaluations were performed. A venous blood sample was obtained from all dogs for a complete blood count and serum biochemistry analysis. The results of this analysis revealed mild deviations consistent with dehydration in cases 1 and 5 and moderate elevations of white blood cells and platelets in case 3 consistent with inflammation (Table 2).

To investigate the extent of the dogs' injuries, a variety of imaging modalities were employed (Table 2). The most important findings revealed after evaluation of three-view thoracic radiographs were the presence of a S/C emphysema (3/4 dogs), pneumomediastinum (3/4 dogs), and pneumothorax (2/4 dogs). Two-view cranio-cervical radiographs revealed S/C emphysema (3/4 dogs) (Figure 1), focal thickening of the dorsal tracheal wall (2/4 dogs), and a poorly defined aspect of the cervical trachea (1/4 dogs).

Table 2. Clinical presentation, haematological abnormalities, results of endoscopic examination of upper respiratory and gastrointestinal tracts, and diagnostic imaging findings for five dogs diagnosed with laryngeal paralysis due to dog bites to the cervical region.

			Endoscopic examination		
Case	Clinical signs	Haematological abnormalities[a]	Laryngeal paralysis	Additional lesions	Diagnostic imaging findings
1	Dyspnoea, stridor, cyanosis, S/C emphysema, tachycardia	Haematocrit 67.5%	Bilateral	Elongated soft palate, laryngeal oedema and haematoma, partial tracheal section	Laryngeal and dorsal tracheal wall thickening, mild pneumomediastinum, S/C emphysema[b,c] Peri-laryngeal and laryngeal inflammation, focal effusion, S/C emphysema, probable cricoid cartilage tear, parietal haematoma, intraluminal secretions[d]
2	S/C emphysema, tachycardia	NAD	Bilateral	Punctiform tracheal tears, pharyngeal haematoma	Severe cervical S/C emphysema, pneumomediastinum, thickened dorsal tracheal wall ventrally to C5, moderate pneumothorax[b,c]
3	Tachypnoea, inspiratory dyspnoea, stridor	Leucocytes 20.38 $\times 10^9$/L Neutrophils 17.27 $\times 10^9$/L Monocytes 1.750 $\times 10^9$/L Platelets 535 $\times 10^9$/L	Bilateral	No other signs of trauma	Multiple B-lines in the left cranial hemithorax[e] Focal nonspecific pulmonary consolidation in the caudal portion of the left cranial lobe compatible with an atypical pneumonia, focal pulmonary contusions or atelectasia, fracture of 7th costal cartilage[b]
4	Inspiratory dyspnoea, stertor, S/C emphysema, tachycardia	NAD	Right unilateral	Tracheal granulomatous lesion, grade II tracheal collapse at the level of the granulomatous lesion, right oesophageal perforation at the beginning of the oesophagus, facing a left tracheal non-perforating lesion	Severe cervical S/C emphysema, moderate pneumomediastinum, poorly defined aspect of cervical trachea compatible with a tracheal tear, slight pneumothorax[b,c]
5	Inspiratory stertor, S/C emphysema, tachycardia	Platelets 609 $\times 10^9$/L	Left unilateral	Elongated soft palate, everted oedematous saccules, laryngeal collapse, proliferative lesion near left vocal cord, tracheal erythema	Severe cranial and cervical S/C emphysema[a,b] Severe S/C emphysema with reactional steatitis, thickened soft palate, discrete pneumomediastinum, left transverse fracture of laryngeal thyroid cartilage, granuloma with a suspected ventral tracheal lesion (first tracheal ring)[f]

[a]Reference intervals: haematocrit: 37.3–61.7%: leucocytes: 5.05–16.76 $\times 10^9$/L; neutrophils: 2.95–11.64 $\times 10^9$/L; monocytes: 0.16–1.12 $\times 10^9$/L; platelets: 148–484 $\times 10^9$/L.
[b]Three-view thoracic radiography.
[c]Two-view cranio-cervical radiography.
[d]Laryngeal ultrasonography.
[e]Thoracic and abdominal point of care ultrasonography.
[f]Cranio-cervical computed tomography.
C5 = fifth cervical vertebra; NAD = no abnormalities detected.

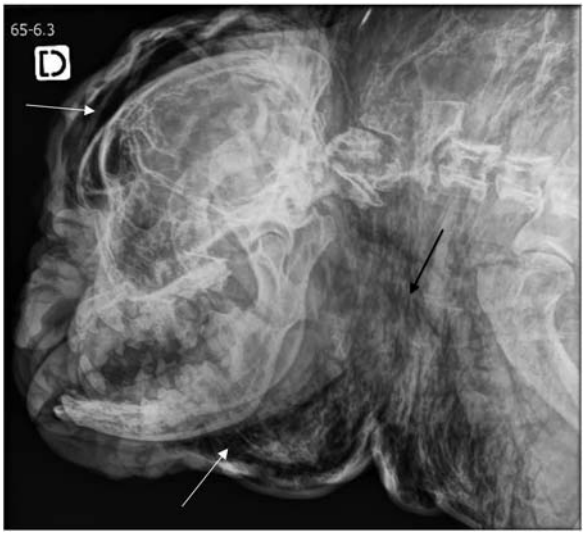

Figure 1. Right lateral radiographic view of the skull of a dog with laryngeal paralysis secondary to a dog bite injury to the cervical region (case 5) showing severe S/C emphysema (white arrows). The trachea is barely visible (black arrow).

Severe peri-laryngeal and laryngeal inflammation, S/C emphysema and a parietal haematoma were diagnosed with laryngeal ultrasonography (case 1; Figure 2). Ultrasonographic examination of the thorax and abdomen of case 3 revealed multiple B-lines in the left cranial hemithorax consistent with pulmonary oedema, aspiration pneumonia, or contusion.

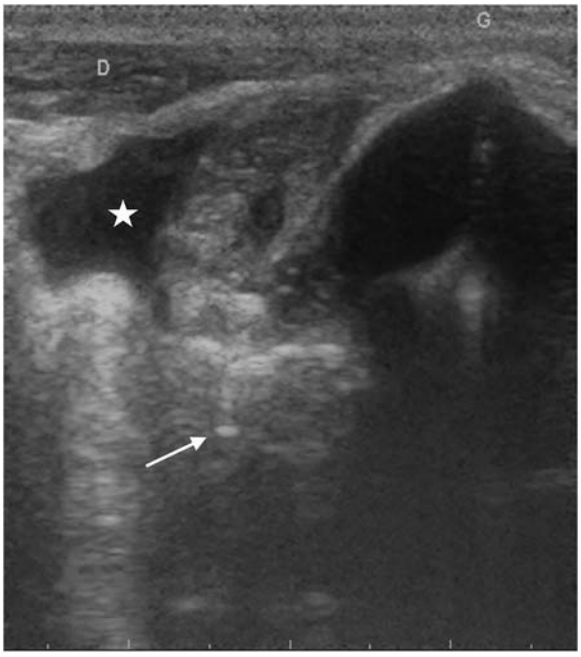

Figure 2. Transverse ultrasonographic image of the right laryngeal area of a dog with laryngeal paralysis secondary to a dog bite injury to the cervical region (case 1), showing swelling of the soft-tissues adjacent to the larynx with a small collection of anechoic fluid (star) and gas (hyperechoic spots; arrow). The dog's ventral aspect is at the top of the image and its right side is shown on the left of the image, with the larynx on the right.

Computed tomography of the cranio-cervical region of case 5 highlighted severe S/C emphysema, a thickened soft palate, discrete pneumomediastinum, left transverse fracture of the laryngeal thyroid cartilage, and a granuloma with a suspected ventral tracheal lesion. Detailed imaging findings are presented in Table 2.

All dogs were then anaesthetised using the same protocol to allow endoscopic examination of the upper gastrointestinal and respiratory tracts. In all cases, this was performed as follows. First, the pharynx, larynx, and trachea were inspected immediately after induction and before intubation. Once the dog was intubated, the gastrointestinal tract from oral cavity to pylorus was examined. Dogs were then extubated and laryngeal function was immediately and systematically reassessed after administration of I/V 1.1 mg/kg doxapram (Dopram-V; Vetoquinol, Lure, France). Anaesthesia was induced with I/V 0.2 mg/kg methadone (Comfortan; Eurovet Animal Health BV, Badel, Netherlands) and 0.2 mg/kg midazolam (Midazolam Mylan; Mylan BVBA, Hoeilaart, Belgium). Propofol (Diprivan; Aspen Pharma Trading Ltd., Dublin, Ireland) was then given I/V to effect (2–6 mg/kg) and anaesthesia was maintained with isoflurane (Iso-Vet; Piramal Critical Care BV, Voorschoten, Netherlands). Monitoring included heart rate, respiratory rate, temperature, non-invasive arterial blood pressure, pulse oximetry and capnography.

The first phase of endoscopic examination revealed an elongated soft palate in cases 1 and 5, laryngeal oedema and haematoma on the right arytenoid cartilage in case 1, a moderate haematoma of peri-laryngeal tissues in case 2, everted and oedematous saccules with a proliferative lesion dorsally and caudally to left vocal cord in case 5, tracheal injuries in cases 1, 2, and 4 (Figure 3(a)), and grade II tracheal collapse at the level of the tracheal lesion in case 4. The second phase of the endoscopic examination highlighted a perforating lesion at the proximal oesophagus, facing a left tracheal non-perforating lesion in case 4. Bilateral laryngeal paralysis was diagnosed in 3/5 cases (Figure 3(b)) and unilateral laryngeal paralysis was diagnosed in cases 4 and 5. Detailed endoscopic findings are presented in Table 2. No complications associated with endoscopic examination were noted.

Treatment and outcome

Four dogs were managed surgically and one dog medically (Table 3). Dogs were hospitalised for 2–10 days and underwent surgical procedures as determined appropriate for treatment of the lesions identified on clinical examination, diagnostic imaging and endoscopy (Table 3). Surgical procedures were carried out during the same anaesthetic episode as endoscopy. Copious lavage with sterile 0.9% NaCl

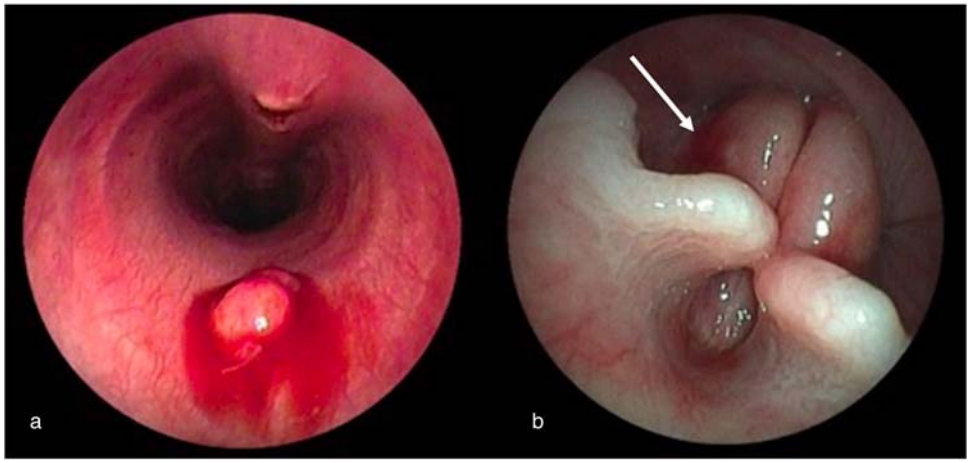

Figure 3. Endoscopic views of the larynx of two dogs with laryngeal paralysis caused by dog bites to the cervical region showing (a) two minor punctiform tracheal tears (case 2) and (b) bilateral laryngeal paralysis associated with mild oedema and small haematoma on the right arytenoid cartilage (white arrow) (case 1).

and soft-tissue debridement were performed prior to surgical repair. Puncture wounds were kept clean with the application of sterile gauze moistened with 0.9% NaCl 0.9% four times per day and allowed to heal by secondary intention. Polypropylene suture (Prolene 2-0; Ethicon, Johnson and Johnson Co., Somerville, NJ, USA) was used for cricoarytenoid and thyroarytenoid lateralisation procedures. If a dog was deemed to be uncomfortable or painful after surgery, analgesia was provided by administration of I/V 0.1 mg/kg methadone every 4 h until signs of pain upon clinical examination ceased.

The cervical region was explored through a ventral midline approach in cases 1 and 4 in order to close the tracheal perforation. Tracheal tears were sutured with 3–0 polydioxanone (PDS; Ethicon) in a simple interrupted pattern. In case 1, the right caudal laryngeal nerve was noted to have been transected. Primary nerve repair was not attempted.

Brachycephalic airway obstructive syndrome-related procedures were performed in 2/4 surgically managed cases and consisted of staphylectomy (2/2), wedge resection rhinoplasty (1/2), and sacculectomy (1/2) (Table 3). These procedures were performed when anatomical abnormalities related to this syndrome were deemed to be worsening dyspnoea.

Temporary tracheostomy was performed in 2/4 cases either to allow reduction of swelling (case 1) or

Table 3. Surgical procedures and medications administered during hospitalisation, with duration of hospitalisation (days) and number of days between surgery and the most recent follow-up assessment, for five dogs with laryngeal paralysis subsequent to a dog bite injury to the cervical region.

Case	Surgical procedure	Medications[a]	Duration of hospitalisation	Follow-up period
1	Surgical exploration, tracheal tear suture, temporary tracheostomy, staphylectomy, wedge resection rhinoplasty, left cricoarytenoid lateralisation	Amoxicillin-clavulanic acid: 20 mg/kg I/V every 8 h Methylprednisolone sodium succinate: 5 mg/kg I/V Acepromazine: 0.01 mg/kg I/V	8	180
2	None; managed medically	Amoxicillin-clavulanic acid: 20 mg/kg I/V every 8 h	5	150
3	Left cricoarytenoid lateralisation, left thyroarytenoid lateralisation, temporary tracheostomy, right cricoarytenoid lateralisation	Amoxicillin-clavulanic acid: 15 mg/kg S/C Amoxicillin-clavulanic acid: 20 mg/kg I/V every 8 h Enrofloxacin: 5 mg/kg S/C once daily Butorphanol: 0.2 mg/kg I/V Metoclopramide: 0.3 mg/kg I/V every 8 h[b]	10	30
4	Surgical exploration, suture of tracheal tear, oesophagostomy tube	Amoxicillin-clavulanic acid: 20 mg/kg I/V every 8 h Prednisolone: 0.5 mg/kg once daily 5 days	2	120
5	Staphylectomy, sacculectomy	Amoxicillin-clavulanic acid: 20 mg/kg I/V every 8 h Prednisolone: 0.5 mg/kg twice daily for 3 days, once daily for 3 days, once every other day for 4 days Omeprazole: 1 mg/kg twice daily 10 days	2	180

[a]Methylprednisolone sodium succinate (Solumedrol): Pfizer SA, Brussels, Belgium; acepromazine (Placivet) and prednisolone; Kela NV, Hoogstraten, Belgium; butorphanol (Butomidor): Richter Pharma AG, Wels, Austria; metoclopramide (Vomend): Eurovet Animal Health BV, Bladel, Netherlands; omeprazole: Eurogenerics SA, Brussels, Belgium.
[b]Furosemide (2 mg/kg I/M) and dexamethasone (0.1 mg/kg I/M) were given by the referring veterinarian before presentation.

due to the dog's dyspnoea and inability to maintain sufficient oxygen saturation (case 3). Cricoarytenoid lateralisation was performed in 2/4 dogs. Surgical management of cases 1 and 3 were not straightforward. After repair of the tracheal tear and correction of the elongated soft palate in case 1, significant laryngeal oedema was noted. Thus, the decision was made to perform a temporary tracheostomy. However, 6 days after the original tracheostomy, surgery to lateralise the left cricoarytenoid cartilage was performed since repeated attempts to remove the tracheostomy tube led to severe inspiratory dyspnoea and endoscopy did not show any improvement in laryngeal function. Post-operative visualisation of abduction of the arytenoid cartilages was adequate, and the dog recovered uneventfully, breathing normally.

In case 3, left cricoarytenoid cartilage lateralisation was also performed, as the rima glottidis was of insufficient size due to bilateral laryngeal paralysis. Cartilages were calcified and difficult to move but post-operative visualisation of the arytenoid cartilage abduction appeared adequate. The dog recovered uneventfully with a normal breathing pattern. However, it became severely dyspnoeic a few hours after surgery. A second endoscopic examination showed collapse of the left arytenoid cartilage. The surgery was repeated. The left arytenoid cartilage was partially cracked at the suture site and a thyroarytenoid lateralisation was performed. The dog recovered from anaesthesia uneventfully. Four days later, the dog again became dyspnoeic with a bilateral purulent nasal discharge. Thoracic radiography was repeated which showed lesions consistent with severe aspiration bronchopneumonia including increased opacity in the right and left cranial lobes, right middle lobe and accessory lobe with air bronchograms. As the dog progressively became unable to maintain sufficient oxygen saturation and dyspnoea recurred, a temporary tracheostomy was performed. At the same time, endoscopy of upper gastrointestinal and respiratory tracts was repeated. Right laryngeal paralysis was still present with a tendency during inspiration for paradoxical motion of the arytenoids. The rima glottidis was still very small despite thyroarytenoid lateralisation. Severe tracheo-bronchial inflammation and large amounts of purulent secretions primarily from the left cranial lobes of the lungs were observed. Lavage of these lobes was performed in an attempt to aspirate the secretions. Occasional erosions and ulcers in the pyloric antrum were also visualised. In order to enlarge the rima glottidis, a right cricoarytenoid lateralisation was performed. The dog improved rapidly, its tracheostomy tube was removed, and it was discharged 2 days later.

Despite bilateral laryngeal paralysis, case 2 was eupnoeic and since the tracheal tears were small, a conservative approach was chosen. Skin wounds were dressed with wet-to-dry bandages applied twice daily for 2 days followed by dressing with nitrofural ointment (Furacine; Limacom, Diepenbeek, Belgium) applied once a day until discharge. Respiration was monitored by respiratory rate, evaluation of the breathing pattern, and oxygen saturation.

Selection of antimicrobials and duration of therapy were based on the preference of the surgeon and consisted of I/V 20 mg/kg amoxicillin-clavulanic acid every 8 h during hospitalisation (Augmentin; GlaxoSmithKline Pharmaceuticals SA, Wavre, Belgium) for all dogs. Dogs received oral antimicrobial therapy for 2–3 weeks after surgery, as well as parenterally before surgery. Due to severe bronchopneumonia and declining respiratory condition, 5 mg/kg enrofloxacin (Baytril; Bayer Healthcare, Loos, France), given S/C once daily was added to the treatment protocol for case 3. All medications administered to the dogs are listed in Table 3.

Follow-up examinations were performed on three dogs (cases 1, 2, and 4) 4–6 months after surgery. This consisted of clinical examination and assessment of respiratory function, and for cases 1 and 4, endoscopy of the upper respiratory tract (Table 3). Physical examination and respiratory function were normal for all dogs examined. In case 2, where bilateral laryngeal paralysis was managed medically, normal laryngeal motion was noted at endoscopy. In case 4, endoscopy was repeated 1 and 4 months after surgery to monitor the surgical site in the trachea and the tracheal collapse. One month after the surgery, there was persistent right unilateral laryngeal paralysis, slight tracheal stenosis at the surgical tracheal site with a dorsal granuloma, and grade II tracheal collapse. Fluticasone (250-µg puffs; Flixotide; GlaxoSmithKline Pharmaceuticals SA, Marly-le-Roi, France) daily for 1 month was recommended to treat the tracheal granuloma. The dog was rechecked 4 months after admission and endoscopy revealed persistent right unilateral laryngeal paralysis, a small scar granuloma at the site of tracheal surgery, with associated grade I tracheal collapse. Information regarding outcomes for cases 3 and 5 was obtained from the owners by telephone assessment 1 and 6 months after surgery (Table 3). The owner of each dog reported its respiratory function to be excellent. All dogs recovered a good quality of life.

Discussion

This case series describes the management of 5 dogs with acquired laryngeal paralysis following cervical dog bite injuries. Three cases in this series had bilateral laryngeal paralysis. Bilateral traumatic laryngeal paralysis is a rare condition and, to the authors' knowledge, has not been reported secondary to cervical dog bite injuries. Our results show that when treated

appropriately, airway injuries following cervical dog bites may have a good prognosis. One dog with bilateral laryngeal paralysis was treated medically as no signs of dyspnoea were present. This particular case was interesting as the bilateral laryngeal paralysis was transient and normal laryngeal function was confirmed 5 months after admission. Surgical management was elected in 4/5 dogs based on clinical evaluation and lesions revealed by endoscopic examination of upper gastrointestinal and respiratory tracts. In a retrospective study of 56 cases of airway injury associated with cervical wounds, two dogs presented with laryngeal hemi-paralysis but no signs of dyspnoea, and thus did not require surgical procedures (Jordan *et al.* 2013). Surgical intervention depends on the clinical status of the animal and is required if dyspnoea is worsening and/or S/C emphysema is present (Mitchell *et al.* 2000). The type of procedure was selected based on the mechanism deemed to be responsible for the clinical signs, or worsening dyspnoea. Minor tracheal tears with static emphysema and no respiratory distress, as in case 2, often do not require surgical intervention and may heal by second intention (Mitchell *et al*. 2000). However, in case 1, the tracheal tear was repaired surgically as it was thought to be exacerbating respiratory distress in the dog. Procedures related to brachycephalic airway obstructive syndrome were the only surgical procedures performed in case 5. We assessed that these procedures would result in significant improvement in the dog's respiratory condition, as swelling and S/C emphysema were contributing to deterioration of its respiratory status. For case 1, we elected to perform procedures to correct anatomical abnormalities related to brachycephalic airway obstructive syndrome during surgery to repair the tracheal tear. These procedures could have temporarily increased oedema and swelling in the pharynx and larynx and could have worsened dyspnoea, thus we felt that it would be more beneficial to correct the anatomical abnormalities during the same intervention rather than postponing surgery. The absence of tracheal collapse prior to the trauma in case 4 cannot be ascertained due to lack of prior endoscopic evaluation. However, the dog was reported to be clinically normal up until the event. We hypothesised that this focal tracheal collapse may have been due to the trauma, as it was at the same level as the tracheal injury.

Management of dogs and cats with cervical wounds can be challenging as concomitant lesions are common and laryngeal oedema and inflammation can worsen respiratory function (Millard and Tobias 2009). The frequency of concomitant lesions in this study was: tracheal tears (3/5), laryngeal and pharyngeal oedema (2/5), oesophageal tear (1/5), laryngeal injury (1/5). As these cervical lesions can be severe and have a significant impact on the clinical status and prognosis, they should be investigated during the initial diagnostic process, and where present, included in the global management of the case. None of the dogs in our study had any evidence of dysfunction of the vagosympathetic trunk other than laryngeal paralysis. We noted that 3/5 cases had ventral cervical wounds and 2/5 had dorsal cervical wounds. Caudal laryngeal nerve transection was observed during surgical exploration in case 1, in which the cervical wounds were ventral. Identification of the nerves was not sought during surgical procedures. We believe that trauma to the caudal laryngeal nerve may be more common as this nerve is anatomically more prone to injury than the cranial laryngeal nerve, due to its location and muscular coverage (Hermanson and Evans 1993). While for two dogs, wounds were only found dorsally in the cervical region, trauma to the caudal laryngeal nerve in these cases cannot be ruled out. Injuries to the airway have been documented even in cases of cervical bite trauma where no skin penetration was apparent (Jordan *et al.* 2013).

Diagnosis of laryngeal and tracheal injuries can be challenging. In a recent study of dogs with upper airway injury secondary to trauma, S/C emphysema and respiratory distress were present in 70% and 50% of cases respectively (Basdani *et al.* 2016). Radiographic findings associated with tracheal perforation include peritracheal air accumulation, S/C or deep fascial emphysema, and pneumomediastinum (Farrow 2003). However, radiographic evaluation of upper airway obstruction is limited because of superimposition and its static nature. Air-dilated laryngeal saccules are a common finding on lateral cervical radiographs in dogs with laryngeal paralysis (Stadler *et al.* 2011). Laryngoscopy is probably the most common method in general practice to diagnose laryngeal paralysis. We believe that in dogs with cervical injury, evaluation of the entire pharyngeal and laryngeal region, oesophagus, and trachea is fundamental. Management of dogs with bites to this region should not be limited to laryngeal evaluation with a laryngoscope. Endoscopy of the respiratory tract may be necessary to further locate and specify upper airway lesions. While endoscopy may worsen tracheal tears and pneumothorax, it allows evaluation of laryngeal motion and structure and can help to identify tracheal tears (Basdani *et al.* 2016). However, diagnosis of laryngeal paralysis can be difficult in the presence of oedema, haematoma, and bleeding which may inhibit laryngeal mobility. No complications associated with endoscopic examination were observed in the five cases described here.

Good agreement between laryngoscopic evaluation and ultrasonography of the larynx has been reported in dogs with laryngeal paralysis (Rudorf *et al.* 2001). Ultrasonographic investigation of the larynx can accurately indicate the presence of paralysis and confirm the uni- or bilateral nature of the disorder (Rudorf

et al. 2001). Ultrasonography of the larynx could therefore be useful as the animal does not need to be anaesthetised. However, air present in the S/C tissues secondary to bite injuries reflects sound waves and makes laryngeal evaluation more difficult. Computed tomography imaging of non-anaesthetised dogs with upper airway obstruction in a restraining device has been reported. However, to increase the likelihood of imaging dynamic laryngeal lesions in the most severely affected phase of respiration, dogs must be scanned multiple times. Sedation is often still necessary (Stadler *et al.* 2011).

We cannot be certain that the laryngeal paralysis identified in the dogs reported here was in fact caused by bite injuries as no dogs underwent endoscopy before presentation. However, none of the dogs were reported to have respiratory problems prior to the fight for which they were presented to the clinic. For dogs that present with laryngeal paralysis following traumatic injury, the possibility that the paralysis is reversible should be considered and used to guide decisions regarding surgical intervention. As some dogs tolerate uni- or bilateral laryngeal paralysis well, clinical evaluation is fundamental to determine whether surgery is required. The type of procedure should be based on what is deemed to be contributing to dyspnoea. Therefore, among the dogs included in this study laryngeal paralysis was not surgically corrected in 2/4 cases.

While the laryngeal nerves have been reported to regenerate following nerve damage (Crumley and McCabe 1982), recovery of function of a peripheral nerve is frequently unpredictable. Return of laryngeal function can be expected in cases of laryngeal nerve neurapraxia. Interestingly, a propensity for reinnervation after experimental nerve resection in cats was noted for the recurrent laryngeal nerve but not for the vagus nerve (Woodson 2007). These authors also noted preferential reinnervation of the laryngeal adductor muscles. This highlights the fact that reinnervation is influenced by the site of the nerve injury.

Electromyography of laryngeal muscles could have been performed to demonstrate denervation potentials 5 days after trauma and could have provided valuable diagnostic and prognostic information regarding patterns of nerve injury (Scott *et al.* 2010). However, some animals with electromyography results indicative of denervation have normal laryngeal function (Peterson *et al.* 1982; Monnet and Tobias 2018). Moreover, in one study, electromyography of the laryngeal musculature did not identify abnormal spontaneous activity despite the presence of histological abnormalities in laryngeal muscles of dogs affected by idiopathic laryngeal paralysis (Andrade *et al.* 2015). In our study, caudal laryngeal nerve neurapraxia and associated transient laryngeal paralysis were suspected in case 2. However, no electromyography testing was completed to assess this hypothesis. Lesions such as fractures of cartilage, oedema, or haematoma can be responsible for neurapraxia by compression or stretching of the nerves beyond their normal range but may also mechanically inhibit laryngeal function which potentially could improve with resolution of these comorbidities.

The laryngeal cartilages of case 3 were found to be calcified and were hard to move during lateralisation. Furthermore, hours after surgery, the cartilage cracked allowing collapse of the cartilage and return of dyspnoea. The presence of both calcification and ossification have been described in canine laryngeal cartilages (Gaskell 1974). Calcification of laryngeal cartilages is a normal ageing process in the dog. Thyroid and cricoid cartilages are the most commonly affected (Coulson and Lewis 2008). Trauma has also been reported to cause ossification of laryngeal cartilages (Manchi *et al.* 2016). Calcified cartilages can be more friable and result in suture breakage during the cricoarytenoid lateralisation procedure (Monnet and Tobias 2018). The calcified laryngeal cartilages noted in case 3 were presumably secondary to age, as trauma had occurred recently.

Temporary tracheostomy is recommended when postoperative inflammation may lead to airway obstruction (Jordan *et al.* 2013). However, temporary tracheostomy after unilateral cricoarytenoid lateralisation has been associated with higher morbidity (due to aspiration pneumonia, respiratory distress, and other causes) and mortality (MacPhail and Monnet 2001). Temporary tracheostomy was performed in case 1 to allow reduction of swelling, and in case 3 due to dyspnoea and inability to maintain sufficient oxygen saturation. No complications were noted in case 1. Case 3 was admitted with signs compatible with bronchopneumonia. Emergency temporary tracheostomy was performed because upper respiratory signs recurred a few days after left crico- and thyroarytenoid lateralisation. The dog also became unable to maintain sufficient oxygen saturation. Endoscopy revealed large amounts of purulent secretions primarily from the left cranial lobes of the lungs, consistent with bronchopneumonia. Bronchopneumonia did not worsen after the temporary tracheostomy and bilateral arytenoid lateralisation, and the dog improved quickly.

Dogs in our study were premedicated with methadone before endoscopic examination, and methadone was also administered for post-operative analgesia when required. A side effect of opioid administration in dogs is respiratory depression (Berg and Orton 1986). However, no significant decrease in alveolar ventilation has been identified following methadone administration in unanaesthetised dogs (Maiante *et al.* 2009). In contrast, Wilson and Monnet (2016) reported that the risk of aspiration pneumonia in the post-operative period for dogs treated surgically for

laryngeal paralysis was increased by post-operative administration of opioid analgesics. They speculated that reduced opioid administration permits dogs to regain sternal recumbency more rapidly after anaesthesia, rather than remaining in lateral recumbency for several hours after surgery. Case 3 presented with an aspiration pneumonia which worsened during hospitalisation. This dog may have been at an increased risk due to methadone administration, and we cannot exclude the possibility that the observed worsening of the dog's condition was due to aspiration pneumonia during hospitalisation. However, analgesia was considered essential to recovery after significant surgical procedures and dog bite wounds.

In conclusion, dog bite wounds to the cervical region can lead to laryngeal paralysis, which may be uni- or bilateral, transient, or permanent, and which may potentially require surgical intervention. Endoscopic examination of upper digestive and respiratory tracts as well as the assessment of laryngeal function are therefore advised in animals with cervical injuries. This is particularly important as some dogs can present with bilateral laryngeal paralysis in the absence of obvious clinical signs. Moreover, endoscopy enables the diagnosis of concomitant lesions, which may require surgery. Prognosis may be worsened if laryngeal paralysis is not detected early enough and if adequate management is not recommended.

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