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CASE REPORT

Companion or pet animals

Complete tracheal obstruction during anaesthesia for ventral slot decompression surgery in a dog

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Abstract

An 8-year-old, female Yorkshire terrier presented to the neurology department with a 3-month history of progressive ambulatory tetraparesis. A diagnosis of intervertebral disc protrusions at the levels of C3–C4 and C6–C7 was made using magnetic resonance imaging. Ventral slot decompressive surgery was carried out at both sites. When accessing the C6–C7 disc space, placement of Gelpi retractors resulted in complete tracheal obstruction, characterised by an absence of end tidal carbon dioxide measurement. This occurred in conjunction with the cessation of abdominal and thoracic respiratory movement despite the use of positive pressure ventilation. Removal of the Gelpi retractors resolved the tracheal occlusion and permitted the resumption of ventilatory support. To facilitate this surgical approach, the trachea was reintubated with a longer endotracheal tube to protect the patency of the airway. To the authors' knowledge, this is the first report of tracheal obstruction as a complication of a ventral slot procedure.

KEYWORDS
anaesthesia, dogs, neurosurgery, ventilation

BACKGROUND

Ventral slot surgery is a frequently performed technique in a referral neurology setting for the treatment of cervical intervertebral disc disease.[1] Cervical neurosurgical techniques have previously been shown to have a higher morbidity and mortality in the peri-anaesthetic period than that in the general population described by Brodbelt et al.[2,3] Studies looking at adverse events and complications arising from cervical decompressive surgery have not highlighted tracheal occlusion as a potential risk.[4,5]

Airway obstruction may lead to hypoxia and hypercapnia with subsequent cardiac arrest and death, if left untreated.[6,7] Swift resolution is therefore required to prevent these catastrophic complications for the patient. To the authors' knowledge, the occlusion of the airway by the placement of surgical retractors has not been described in dogs before this report. It is hoped that the information provided here may assist others when troubleshooting ventilatory problems.

CASE PRESENTATION

An 8-year-old, female Yorkshire terrier presented to the neurology department for investigation of a 3-month progressive history of tetraparesis, which was now severely affecting its gait. A degree of neck discomfort was also described by the owner. There had been temporary improvement of the neurological signs after a course of prednisolone prescribed for otitis externa by the referring veterinary surgeon, which was continued at a dose of 2.5 mg orally, once daily.

On neurological examination, the dog's sensory status and cranial nerve function were normal. A 'two-engine' tetraparetic gait was present, where the hindlimbs moved asynchronously to the forelimbs. Occasional collapsing of the hindlimbs was also observed. Postural reactions were absent from the hindlimbs and mildly delayed in the forelimbs. Spinal reflexes were normal in the hindlimbs; in the forelimbs, a reduction in shoulder flexion was present upon limb withdrawal. Discomfort was evident on extension of the cervical spine.

Magnetic resonance imaging (MRI) was recommended, so the dog was presented to the anaesthesia department, with the possibility of surgery based on diagnostic findings. During preanaesthetic physical examination, the dog was bright and responsive. Mucous membranes were pink and moist. Lung sounds were bronchovesicular with a costo-abdominal breathing pattern and respiratory rate of 24 breaths per minute. The heartbeat was regular, with no murmur detectable on auscultation, good peripheral pulse quality and

heart rate of 112 beats per minute. The presence of a distended abdomen and significant deposits of fat over the thoracic cavity placed the body condition score of this dog at 8 out of 9. The rectal temperature was 38.6°C.

Premedication was provided using dexmedetomidine (2 μg/kg, Dexdomitor 500 μg/ml; OrionPharma) combined with methadone (0.3 mg/kg, Methadyne 10 mg/ml; Jurox) by intramuscular injection. After 15 minutes, an intravenous catheter was placed. Oxygen was provided by mask for 5 minutes before induction of general anaesthesia with propofol (PropoFlo Plus 10 mg/ml; Zoetis) to effect; a total dose of 3.9 mg/kg was needed. Oral endotracheal (ET) intubation was performed with a cuffed ET tube with a 6.5-mm internal diameter inserted to a depth of 18 cm. Anaesthesia was maintained with isoflurane (Isofane 100% w/w; Henry Schein) delivered in 100% oxygen through a Bain breathing system. Following a period of stabilisation and monitoring, the dog was moved to the MRI. In MRI, mechanical ventilation of the lungs was initiated with a Pneupac (Pneupac VentiPAC; Smiths Medical) ventilator connected to a circle breathing system. This was set to deliver a rate of 24 breaths per minute, and a peak inspiratory pressure of 13 cmH_2O was generated.

Compressive disc lesions at the levels of C3–C4 and C6–C7 were revealed by MRI, which required decompressive surgery by the ventral slot. During surgical preparation, infusions of fentanyl (Fentadon 50 μg/ml; Dechra) and ketamine (Ketavet 100 mg/ml; Zoetis) were initiated at rates of 5 μg/kg/h and 10 μg/kg/min, respectively. Fentanyl administration was subsequently altered according to physiological responses to surgery, whereas ketamine administration remained constant. The dog was moved to the theatre and positioned in dorsal recumbency in a rigid plastic cradle that secured both the thoracic and abdominal cavities. The neck was extended cranially with a downwards stretch, and the forelimbs were tied caudally to expose the ventral surface for surgical access. In the theatre, the lungs were ventilated through a circle breathing system using an Aestiva/5 anaesthesia machine and a Smartvent 7900 ventilator (Aestiva/5 7900; GE Datex-Ohmeda) with an integrated Datex-Ohmeda S/5 monitor (Datex-Ohmeda S/5; GE Datex-Ohmeda). The ventilator was set to deliver a tidal volume of 80 ml at a rate of 25 breaths per minute. This occasionally generated a peak inspiratory pressure of up to 22 cmH_2O, triggering the relief alarm set at 20 cmH_2O. At this point in time, the heart rate was stable at approximately 80 beats per minute with good synchronous pulses, and a mean blood pressure of 100 mmHg was measured noninvasively. Oxygen saturation was measured to be above 95%, and the end tidal carbon dioxide ($EtCO_2$) was measured to be between 5.2 and 6.3 kPa.

During the surgical approach and decompression of the C3–C4 disc space, anaesthesia was uneventful. As tissues were retracted laterally with Gelpi retractors to expose the C6–C7 joint space, there was a sudden loss of capnography trace, and the partial pressure of $EtCO_2$ dropped to 0 kPa. At this time, the ventilator bellows appeared to function mechanically (i.e., could be observed moving), and all breathing system components were still securely attached together; however, there was an absence of any ventilatory thoracic and abdominal movements. Pulse rate and characteristics had not changed from earlier assessments, and oxygen saturation was 100%. The pressure alarm, which had been set at 20 cmH_2O, was activated as the peak inspiratory pressure increased to 22 cmH_2O.

LEARNING POINTS/TAKE-HOME MESSAGES

- Capnography is an early indicator of complete ventilatory obstruction in dogs.
- Caudal cervical decompression surgery carries a risk of airway obstruction due to the challenges accessing the surgical site and the tissue retraction required.
- High airway pressures in obese patients, positioned in such a way as to reduce thoracic and abdominal movement, have the potential to mask ventilatory problems.

The ventilator module continued to display that a tidal volume of 50 ml was being obtained. After removal of a pair of Gelpi retractors from the neck by the surgeons, ventilation of the lungs resumed, as indicated by a return of the capnogram and movement of the thoracic and abdominal walls.

INVESTIGATIONS

Investigations as to the cause of the 0 kPa reading on the capnograph were carried out in a sequential manner. This included the assessment of:

- the dog's pulse;
- the connection of the breathing system components and attachment to the dog;
- the movement of ventilator bellows;
- capnography sampling line connection;
- abdominal and thoracic movement associated with ventilation;
- removal of all surgical instrumentation from the neck.

DIFFERENTIAL DIAGNOSIS

The following scenarios could have resulted in the reduction or absence of $EtCO_2$[8]:

- disconnection of breathing system components;
- ventilator malfunction;
- cardiovascular collapse;
- obstruction of breathing system/airway;
- accidental tracheal extubation;
- capnography malfunction; for example, sampling leak.

TREATMENT

This incident recurred during subsequent attempts to achieve suitable visualisation of the vertebral body. To permit the surgical approach, the trachea was reintubated. This was performed in dorsal recumbency to avoid repositioning the animal when an open surgical site was present. To facilitate this, a 1 mg/kg bolus of propofol was given when the delivery of inhalant anaesthetic was interrupted to allow detachment of the breathing system and extubation. The trachea was

reintubated with a 6.0-mm internal diameter armoured orotracheal tube to a depth of 24 cm, which was judged to be just caudal to the thoracic inlet. Following this, no further episodes of ventilatory failure occurred, and the surgery was completed without incident.

OUTCOME AND FOLLOW-UP

After reintubation with a longer ET tube, no further obstruction was observed during retraction and manipulation of the tissues, allowing adequate visualisation and an uneventful conclusion of the procedure by the neurosurgery team. The dog made a successful recovery from anaesthesia in the intensive care unit. Following a further 8-day stay in the hospital, the dog was discharged and showed excellent return of neurological function at its recheck appointment 6 weeks later.

DISCUSSION

Significant perioperative complications have been reported during cervical decompressive surgery in dogs.[2,4] Deaths due to acute haemorrhage and bradycardia have been reported in the peri-anaesthetic period.[2] Both tachydysrhythmias and bradydysrhythmias have been reported by Stauffer et al.[9] Post-operatively, hypoventilation has been reported due to a worsening of neurological symptoms and deterioration of respiratory function.[2] Aspiration pneumonia was another reported complication causing morbidity and mortality in several cases.[2,4] Inadvertent fixation of the ET tube (preventing extubation of the trachea) has been reported following cervical surgery.[10]

To the authors' knowledge, this is the first case of ventilatory obstruction in a dog resulting from the surgical retraction of tissues in a ventral slot procedure. Several other causes of tracheal and airway obstruction have been reported in anaesthetised dogs. These range from material, such as blood clots, occluding the ET tube lumen[11] to herniation of the ET tube cuff[12] and tracheal collapse.[13] In these cases, the clinical indication of obstruction was often characterised by changes in the capnogram. Capnogram changes included alterations in the $EtCO_2$ and variations in the shape of the waveform. Both increases and decreases in $EtCO_2$ have been reported. Increases are likely to indicate a partial obstruction where alveolar ventilation is reduced but sufficient tidal volume is generated to allow exhalation and measurement of CO_2 in alveolar gas. Reductions in $EtCO_2$ indicate a greater degree of obstruction. In such instances, the volume of gas that is returned from exchange units in the lung is minimal. Thus, on expiration, any alveolar component is diluted in a larger volume of dead space gas, resulting in a lower $EtCO_2$ value. The absence of $EtCO_2$, as was present here, indicates that a total occlusion of the tube lumen was present as no gas movement occurred between the breathing system and the patient's lungs.[8,14]

During airway obstruction, failure to eliminate CO_2 will cause the arterial partial pressure of CO_2 ($PaCO_2$) to increase, resulting in hypercarbia and respiratory acidosis.[6] With sidestream capnography, if tidal volume is low or absent, fresh gas will be sampled. This leads to an inability to equate $EtCO_2$ to $PaCO_2$. Partial airway obstruction can cause a more gradual increase in expiratory phase two and an increase in the alpha angle of the capnogram.[15] In this case, these changes were not evident, as no capnogram was visible.

Due to the partial, progressive or protracted nature of the incidents reported by others, impairment in oxygenation was able to develop between 6 and 8 minutes.[12] This results in hypoxia, which can cause mortality of the animal.[13] The sudden onset of airway obstruction, followed by a rapid identification and resolution of the problem, meant that hypoxia did not develop in this case.

The real-time nature of capnography facilitated almost immediate detection of a ventilatory problem, allowing for troubleshooting to begin. Due to the small size of the dog in comparison to the large bulk of surgical drapes, continuous visualisation of ventilatory thoracic and abdominal movement and the breathing system connections proximal to the dog was not possible. In human paediatric patients, where a similar situation exists, capnography has been shown to be key in the early diagnosis of a range of critical events.[16] Capnography has been shown to provide early detection of extubation, tube obstruction or breathing system disconnection in dogs.[14]

During anaesthesia before surgery, an elevated level of $EtCO_2$ of 7.6 kPa was present with spontaneous breathing, so mechanical ventilation was initiated, with a peak inspiratory pressure of 13 cmH_2O and a rate of 24 breaths per minute. To deliver an adequate minute volume without the generation of excessive pressures, a high respiratory rate in combination with a smaller tidal volume per kilogram was used. Nevertheless, this dog was unable to maintain normocapnia because of obesity, which decreases thoracic and pulmonary compliance due to fat deposition around the thorax and functional residual volume (FRC) and increases airway resistance and the work of breathing with decreased efficiency of ventilation.[17,18]

The respiratory rate and inspiratory pressure needed to deliver an adequate minute volume increased once the dog was in theatre. The use of a rigid, non-distensible cradle with an obese dog in dorsal recumbency is likely to have posed significant restriction to thoracic expansion. Additionally, the forelimbs were retracted caudally over the thorax to maximise ventral cervical exposure, again impeding normal movement. The result of this was a requirement of a peak inspiratory pressure closer to 20 cmH_2O and delivery of a tidal volume of approximately 70 ml. It is also possible that if some partial airway obstruction was present at this time, inspiratory volume may have been larger than was able to be exhaled passively, hence building peak pressures; however, airway pressure appeared to fall to zero between breaths.

Peak inspiratory pressures over 20 cmH_2O may increase the risk of lung injury during ventilation.[19] The relief pressure on the ventilator was therefore set at 20 cmH_2O to protect against this. When the obstruction event occurred, the high-pressure alarm of the ventilator was triggered. As this had occurred intermittently throughout the surgery before this point and continued after reintubation, high airway pressure was unlikely to be due to prior partial obstruction in this dog.[20] Alarm fatigue could be a risk in the anaesthesia environment and might hinder the identification of problems.[21] No other alarms (e.g., apnoea or hypoxaemia) were triggered due to the rapid correction of airway obstruction.

During troubleshooting, conflicting information was provided by the ventilator apparatus and capnogram. While the

capnograph had a reading of zero, the ventilator bellows were still functioning, and the flowmeter, found at the distal portion of the expiratory limb to the dog, continued to display 40–50 ml expired tidal volume on the ventilator control module. Given the absence of the capnogram and thoracic expansion, this misleading reading was created as a result of breathing system distensibility. This distensibility allowed for some movement of gas at the level of the flowmeter during intermittent positive pressure applied by the mechanical bellows. This effect has been described in the Aestiva/5 7900 anaesthesia machine (Aestiva/5 7900; GE Datex-Ohmeda) in laboratory situations, where an overestimation of tidal volume of up to 55% has been reported.[22] The use of spirometry at the level of the breathing system to ET tube connection (as recommended in human paediatric patients[23]) could have identified the absence of meaningful gas delivery to the dog's lungs sooner.

A dog's trachea may be more easily compressed if tracheal collapse is present. The Yorkshire terrier breed is overrepresented in cases diagnosed with tracheal collapse.[24] The pathogenesis of this disease is not fully understood, but results in weakening and malacia of the tracheal cartilage structure.[25,26] In conscious dogs, this leaves the trachea susceptible to collapse under the negative pressures associated with breathing or due to external mechanical pressure; for example, a neck leash. Although this was not detected on initial physical examination, this disease could have facilitated compression of the trachea by the surgical instrumentation to a greater extent than would have been seen in a normal, healthy dog. On a subsequent visit to the hospital, thoracic radiographs were taken, which showed slight tracheal narrowing at the thoracic inlet, compatible with a degree of tracheal collapse.

The difficulties encountered by the surgical team in generating suitable visualisation of the sixth cervical vertebral body resulted in significant manipulation of cervical structures. To visualise the space, lateral retraction of the trachea was needed. It is suspected that the tracheal manipulation caused airway occlusion due to the bevel of the ET tube being pressed against the tracheal wall. Given the lack of a Murphy's eye in the initial ET tube, the risk of full obstruction from bevel occlusion is more likely.[27] Alternatively, compression of an abnormally compressible trachea to the point of occlusion could have also resulted in obstruction. Both scenarios would have resulted in the loss of the capnogram and inability to ventilate, which has been described.

The communication with the surgical team to remove the instrumentation from the neck produced a temporary resolution of the problem, allowing the necessary window for a more permanent solution to be found for surgery to continue.

For reintubation, several factors were considered. The use of a longer ET tube, inserted to a point where the tip lay caudal to the thoracic inlet, was expected to aid in supporting a patent tracheal lumen and resisting external compression. The protection an armoured ET tube provides against kinking during bending[28] was the rationale behind this selection, as the surgical access to the vertebrae required significant deviation of the trachea. Murphy's eye supplies a secondary aperture, should occlusion of the primary distal aperture occur.[27] In this case, there was concern that the tracheal wall itself could obstruct the tube's primary aperture during surgical manipulation due to weaker tracheal cartilage. Thus, Murphy's eye was considered beneficial.

Capnography was a key feature in the identification of a ventilatory problem in this dog. A cascade of troubleshooting procedures allowed rapid identification of the problem and resolution to be achieved. Initially, this was obtained by removal of the surgical instrumentation from the operating site. A more permanent solution was found by reintubating the trachea using a longer, armoured ET tube with Murphy's eye. In dogs predisposed to tracheal collapse or malacia, radiographic assessment before cervical surgery may highlight an increased risk for tracheal occlusion.

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CONFLICTS OF INTEREST

The authors declare they have no conflicts of interest.

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ETHICS STATEMENT

This report retrospectively documents a complication event that occurred in clinical practice. Informed owner consent was obtained for the veterinary treatment of this dog. Rapid and successful resolution resulted in no adverse effects for the dog. The hospital consent form includes consent for the retention of data and anonymous use in retrospective publications.

AUTHOR CONTRIBUTIONS

AGM: Case management, writing, editing and review of manuscript. PJM: Case management, writing, editing and review of manuscript.

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