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TITLE OF CASE ***Do not include "a case report"***

Anaesthesia management of a pug (in late stage pregnancy) with lung lobe torsion.

SUMMARY ***Up to 150 words summarising the case presentation and outcome (this will be freely available online)***

Anaesthetic management can be challenging when patients have multiple co-morbidities. This report presents one such case: a pug with brachycephalic airway syndrome, in late gestation, presented with a lung lobe torsion. Under general anaesthesia the dog had initially low PaO_2, with severe ventilation perfusion mismatch and pulmonary shunt, this was mildly improved with patient positioning with head-up tilted table in right lateral recumbency. Tension pneumothorax developed after initiation of positive pressure ventilation, and was resolved with rapid entry into thoracic cavity. Ventilation was adjusted with the aid of spirometry, blood gases, capnography and direct visualisation of lung tissue. The dog made a full recovery and was discharged from hospital 5 days post-operatively.

BACKGROUND ***Why you think this case is important – why did you write it up?***

Spontaneous lung lobe torsion (LLT) in dogs is an uncommon but life-threatening disease. Pugs as a breed appear to be overrepresented in the literature[1]. Pugs also present with brachycephalic airway syndrome (BOAS) and affected animals have small nares, hypoplastic trachea, elongated

soft palate and everted laryngeal ventricles, all contributing to increased upper airway resistance, obstruction may be further exacerbated due to increased negative intrathoracic pressure during respiration leading to laryngeal collapse[2]. These dogs are also predisposed to gastrointestinal complications including vomiting, regurgitation and ptyalism, with risk of aspiration pneumonia[3]. A pregnant animal presents with many physiological changes affecting multiple body systems, which will affect the conduct of anaesthesia. In addition, anaesthetic or physiological changes affecting the foetus should be considered, including maintaining adequate uterine blood flow[4].

Lung lobe torsion associated with a late stage pregnancy in a dog has not previously been reported in the veterinary literature. Anaesthesia management in such a case can be challenging, this case discussion highlight the anaesthesia concerns and discusses detection and management of tension pneumothorax.

CASE PRESENTATION ***Presenting features, clinical and environmental history***

A two year old, 8.9 kg female entire pug in late stage pregnancy (40 days into gestation) was referred to the hospital after a two day history of progressive respiratory compromise. The dog was initially treated for upper respiratory infection (meloxicam 0.17 mg kg^{-1} and Amoxicillin 17 mg kg^{-1}), but then cardiomegaly and pulmonary oedema was suspected, and treated with furosemide 5.6 mg kg^{-1} and meloxicam continued.

On presentation the dog was tachypnoeic and mouth-breathing with 89% haemoglobin oxygen saturation (SpO_2) while breathing room air. A basic blood profile was obtained showing respiratory alkalosis and hypokalaemia (see table 1). A small amount of pleural effusion was seen on thoracic ultrasound and the volume of the cardiac chambers was subjectively small. Conscious thoracic computed tomography angiography with oxygen support showed torsion of the cranial portion of the left cranial lung lobe (Figure 1), a multifocal alveolar pattern caudodorsally (Figure 2), mild regional thoracic lymphadenomegaly and an enlarged uterus at the edge of the scanned volume confirming the pregnant status of the animal (Figure 3). Methadone (0.1 mg kg^{-1}) was administered intravenously (IV) (120 minutes prior to induction), with a 45 ml bolus of compound sodium lactate (CSL) prior to starting maintenance fluids (2 mL/kg/hour) of CSL. The owner was consulted, and a lung lobectomy recommended.

Table 1 Blood results. Venous blood sample taken prior induction of anaesthesia during initial assessment of the dog and arterial blood samples taken during anaesthesia to assess acid-base status and respiratory system.

Time from induction (min):	**Basic blood profile**	**Blood gas 1** *15*	**Blood gas 2** *55*	**Blood gas 3** *130*
Arterial / venous blood	Venous	Arterial	Arterial	Arterial
pH	7.51	7.34	7.25	7.24
PCO_2 (mmHg)	26	50	61	59
$EtCO_2$ (mmHg)		14	42	48
PO_2 (mmHg)		77	81	268
FiO_2 (%)		94	94	94
SaO_2 (%)		93	91	100
HCO_3^- (mmol/L)	19.2	24.8	24.8	23.4
Anion Gap (mmol/L)		21.4	22.6	23.0
tCO_2 (mmHg)		26.4	26.7	25.3
BE (mmHg)		- 1.2	- 3.3	- 4.6
tHb (g/dL)		11.3	10.4	9.8
Na^+ (mmol		158	157	158
K^+ (mmol/L)	3.4	3.5	3.4	3.7
Cl^- (mmol/L)		115	113	115
PCV (%)	37			
Total solids (g/l)	80			
Glucose (mmol/l)	7.4			
Lactate (mmol/l)	1.8			

On pre-anaesthetic examination, the dog was depressed, but responsive with respiratory compromise (tachypnoea respiratory rate (fR) 100 breaths per minute (bpm), shallow breaths, marked inspiratory and expiratory effort and intermittent mouth breathing). Her body condition score was 7/9, the mucous membranes were pink but dry with a capillary refill time of about 1.5 seconds. Rectal temperature was 37.7 C. The heart rate (HR) was 188, with a fair quality of the metatarsal pulses, and abdomen markedly distended.

Lung pre-oxygenation was performed using a Bain breathing system with 2 l/min oxygen delivery by mask. No further premedication was administered and anaesthesia was induced using propofol to effect (31 mg IV), the head was kept elevated until the trachea was intubated with a 4.5 mm internal diameter endotracheal tube (ETT) and the cuff inflated. Oxygen was supplied at 2 l/minute via a low resistance circle breathing system. No saliva or stomach contents were present in the oesophagus when suctioned. The dog was positioned into right lateral recumbency for surgical preparation, and moved to dorsal-oblique recumbency (for a short period) during clipping to facilitate access to the ventral and left lateral aspect of the thorax. Anaesthesia was maintained using total intravenous anaesthesia; propofol (0.2 mg kg^{-1} min^{-1}) and fentanyl (5 ug kg^{-1} hr^{-1}).

After induction, the HR was 195 bpm, fR 63 bpm (spontaneous ventilation), $EtCO_2$ 14mmHg (side stream capnography), and SpO_2 98%. Fluids (CSL) were continued at 45 ml/hr. An arterial cannula (22 gauge) was placed into the left metatarsal artery and a sample taken for blood gas analysis; this showed mild respiratory acidosis, hypoxaemia (see table 1), hypoventilation and severe ventilation / perfusion (V/Q) mismatch and pulmonary shunting affecting gas exchange (see table 2). Over the first 25 minutes of anaesthesia, HR gradually decreased to 160 beats per minute, fR ranged between 60-80bpm (spontaneous breathing), and $EtCO_2$ increased to 27 mmHg.

Table 2 Blood gas calculations. Using blood gas analysis, the following can be calculated or estimated to further assess ventilation. CO_2 (A-a) gradient; alveolar dead-space ventilation (hypoventilation, V/Q mismatch). O_2 (A-a) gradient; assesses degree of V/Q mismatch, pulmonary shunting and alveolar hypoventilation. Physiological dead space equation where sum of all parts of the tidal volume that do not take part in gaseous exchange is estimated

Time from induction:	**Blood gas 1** *15 min*	**Blood gas 2** *55 min*	**Blood gas 3** *130 min*
CO_2(A-a) gradient (mmHg)[*]	36	19	11
O_2 (A – a) gradient (mmHg) [†]	572	533	325
Physiological dead space [V_D/V_T] (%)[¤]	72	31	19
Minute Ventilation [fR x Vt] (ml/min)		960	1335
Alveolar ventilation (ml/min) [€]		662.4	1081.35

[*] CO_2 (A-a) gradient (mmHg) = $PaCO_2 – EtCO_2$[7]
[†] O_2 (A – a) gradient (mmHg) = fiO2 x (atmospheric pressure – H_2O Pressure) – ($PaCO_2$ /respiratory quotient)] – PaO_2. Atmospheric pressure: 756 mmHg (at 43 meters above sea level), H_2O pressure is saturated vapour pressure of water at 37°C: 47 mmHg, respiratory quotient: 0.8[6].
[¤] V_D/V_T (%) = 100 x (($P_aCO_2 – EtCO_2$)/P_aCO_2)[6]
[€] Alveolar ventilation = (1 - V_D/V_T) x minute ventilation[6]

The dog was moved to the surgical theatre and placed in right lateral recumbency with the table tilted to provide a head-up angle (approximately 10º), this immediately improved $EtCO_2$ (40 mmHg). A left intercostal thoracotomy approach was started. After the initial superficial dissection, intercostal (2^{nd} - 6^{th}) nerve blocks were performed (a total of 1 mg kg^{-1} bupivacaine). A second blood gas sample was taken for analysis (30 minutes after positional change) showing worsening respiratory acidosis, mild improvements in oxygenation (see table 1), but markedly decreased alveolar dead-space ventilation (see table 2). During spontaneous ventilation, tidal volume (Vt) was measuring on average 20 ml (pitot tube spirometry), and this was used to adjust volume controlled Intermittent Positive Pressure Ventilation (IPPV), prior to entry into the thorax. IPPV was initially set at fR 50 bpm, inspiratory to expiratory ratio of 1:2, and peak inspiratory pressure limit set at 25 cm H_2O. Within one minute of starting IPPV, blood pressure decreased

with a depressed arterial waveform, $EtCO_2$ decreased to 12 mmHg and SpO_2 decreased to the lowest reading recorded (70%). A tension pneumothorax was suspected. The surgeon rapidly entered the thorax to release the intrathoracic pressure, the arterial trace returned to normal morphology, and the $EtCO_2$ slowly increased over 3-5 minutes. Within 5-8 minutes, the SpO_2 reached 100% and similar clinical values as previously were obtained (HR 155 – 165 bpm, EtCO2 38 mmHg and SpO2 100%, mean BP 93 – 98 mmHg). Ventilation was adjusted throughout surgery and included; inspiratory to expiratory ratio was reversed (2:1), slow increase in Vt (75 – 90 ml over 25 minutes) and decrease in fR (from 40 bpm to 25 bpm), the peak inspiratory pressure limit was never reached. Surgery time was 80 minutes.

A thoracic drain was placed and chest drained to negative pressure. A third arterial blood gas was taken for analysis; showing markedly improved oxygenation and alveolar ventilation, with improved alveolar gas exchange (table 1 and 2). Bupivacaine (0.6 mg kg^{-1}, q4h for 24 hours) was administered interpleurally, and the dog moved to intensive care unit (ICU). The dog was placed in oxygen enriched kennel for two days post-operatively. Analgesia consisted of methadone 0.2 mg kg^{-1} IV every 4 hours for two days, (followed by buprenorphine 0.02 mg kg^{-1} q6h until morning of discharge), paracetamol 10 mg kg^{-1} IV BID, and continued orally after discharge. In addition, maropitant 1 mg kg^{-1} IV SID and metoclopramide infusion of 2 mg kg^{-1} day^{-1} was administered during the first 48 hours post-surgery.

INVESTIGATIONS ***If relevant***

DIFFERENTIAL DIAGNOSIS ***If relevant***

TREATMENT ***If relevant***

OUTCOME AND FOLLOW-UP

The dog was discharged 5 days post-operatively continuing on oral paracetamol. Prior to discharge, an abdominal ultrasound examination confirmed viable foetuses (movement and heart beat) however it was not possible to count the puppies. The dog was returned to the care of the referring veterinarian. Unfortunately there was no follow up regarding the remainder of the dog's pregnancy.

DISCUSSION ***Include a very brief review of similar published cases***

Anaesthesia concerns in a case such as this, involve a complex mixture of both upper and lower respiratory compromise. Lung lobe torsion is associated with hypoxaemia, alveolar hypoventilation and venous admixture[5]. Breed associated upper respiratory compromise included

partial upper airway obstruction and increased respiratory effort[2] and in this case the enlarged uterus caused cranial displacement of the diaphragm, decreasing functional residual capacity (FRC) and total lung volume (TLV) and thus lowering oxygen reserve capacity[4]. This combination of anatomical, physiological and pathological changes predisposed this dog to have severe respiratory compromise.

In a LLT, the affected lung lobe rotates around the bronchus, causing occlusion of the affected airway and veins. This causes engorgement of the alveoli with blood, vascular congestion forms in the affected area leading to accumulation of fluid and infiltration of inflammatory mediators causing progressive resorption of alveolar gases (absorption atelectasis) and a decrease of TLV[5]. This affects gas exchange and can lead to permanent damage to the affected lung architecture, including diffused necrosis and fibrin deposits. Clinically, these changes result in a lower Vt with a compensatory increase in respiratory rate attempting to maintain minute ventilation[5]. Ventilation and perfusion mismatch (V/Q mismatch) and pulmonary shunt can develop, due to LLT pathology, were alveolar ventilation is severely affected.

After induction of anaesthesia, the dog had initially a high fR and low $EtCO_2$, likely indicating poor alveolar ventilation, due to reduced Vt, most likely due to LLT, reduced TLV (enlarged uterus affecting diaphragm flattening) and V/Q mismatch. The main findings on initial blood gas evaluation (see table 1 and 2) included borderline hypoxaemia, wide O_2 - and CO_2 (A-a) gradients and 72% physiological dead space. Physiological dead-space is a calculation of anatomical and alveolar dead-space ventilation[6,7]. In this case high alveolar dead-space ventilation was most likely the contributing factor with low Vt, V/Q mismatch (likely due to LLT and perfused alveoli with zero or limited ventilation (V/Q mismatch)) and cranial displacement of abdominal organs (due to pregnancy) further decreasing TLV. Calculating O_2 (A-a) gradient gives an assessment of the degree of V/Q mismatch and alveolar hypoventilation[6]. This equation uses the respiratory quotient (RQ), which is determined by the amount of CO_2 produced relative to oxygen consumed, and is influenced by diet as well as disease state. In dogs the RQ is commonly set at 0.8[8] and so this was used in this calculation, however given the pregnancy status and health of this dog the estimate may be incorrect. Nevertheless, this calculation is of clinical relevance as it indicates the severity of pulmonary dysfunction.

Pulse oximetry indicated 98% oxygen saturation which highlights the limitation of this monitor to assess the respiratory system when using high inspired oxygen concentrations. Pulse oximetry measures the percentage (%) of haemoglobin oxygen saturation (SpO_2) and not the arterial oxygen

tension (PaO_2). Due to the shape of the oxygen-dissociation curve, large changes in PaO_2 may occur, with minimal changes in SpO_2 seen unless the PaO_2 is <100 mmHg. For this reason arterial blood gas analysis and EtCO2 should be used in conjunction with SpO2 values to fully evaluate the ventilation (including possible shunt or venous admixture) in a case such as this.

At the start of anaesthesia, there was a high suspicion of severe ventilation compromise and dead-space ventilation. Tension pneumothorax was a risk if the integrity of the alveoli were compromised, due to LLT, which was a reason for avoiding ventilation. Additionally, the extent of venous admixture was not immediately appreciated, as the first blood gas sample was taken just prior to moving into theatre. However it could be argued, mechanical ventilation using low Vt and high fR (to maintain minute ventilation with minimal risk of over inflation of the lungs) from the beginning of anaesthesia may have been advantageous.

Tilting the table (approximately 10º) was used to try to increase FRC and alveolar collapse by decreasing the pressure of abdominal contents on the diaphragm. This slight positional change, improved physiological dead space by 57% (Table 2), and further reduced hypoventilation (CO_2 (A-a) gradient). However respiratory acidosis and marked venous admixture was still present with little improvement of the PaO_2. This type of positioning may increase systemic vascular resistance index and decrease cardiac index[9], and any potential benefit should always be balanced against potential risks.

Positive pressure ventilation is required during thoracic surgery. In this case, ideally one-lung-ventilation would have been used. This requires either a specific endotracheal tube intubating the non-affected lung, or a bronchial blocker to prevent ventilation of the affected lung[10]. This prevents positive pressure ventilation causing excessive pressure to the LLT. However, a bronchus blocker and endoscopic assistance was not available. If the affected lung lobe cannot be isolated in patients with pulmonary lesions such as LLT, it is recommended to ventilate with a low Vt and higher fR to prevent excessive stretching of the affected lung which could cause haemorrhage into the unaffected lung leading to worsening gas exchange[11]. The severity of damage to the lung architecture was not known, and the risk of tension pneumothorax was a concern if a pre-existing defect in the affected lung could have permitted gas escape when positive pressure ventilation was applied. Pressure controlled ventilation may have been a more appropriate method of ventilation in this dog, as ventilation would be set at a specific pressure limit. However, the inspiratory pressure needed for adequate ventilation (due to cranial displacement of diaphragm) and not to increase the risk of tension pneumothorax was felt to be difficult to assess. Instead a conservative

attempt using volume controlled ventilation using the same Vt as during spontaneous ventilation was chosen with slightly lower fR, and pressure limit of 25 cm H_2O. Peak inspiratory pressure (PIP) of 10 – 15 cm H_2O in dogs is usually required and a pressure limit of 18-20 cm H_2O is commonly set[12]. In this case the pressure limit was set higher as the first blood gas result indicated severe respiratory compromise and there was concern that inadequate lung inflation might have been achieved without relatively high pressures. Although 25 cmH_2O is arguably high and a more conservative pressure limit may have been wiser, actual PIP did not reach the limit set. Airway pressures as high as 30 cmH_2O have been suggested to be required in animals with severe lung disease[12]. However, tension pneumothorax did develop and was recognised by the severe dampening of the arterial pressure waveform, sudden hypocapnia and desaturation noted on pulse oximeter. This is thought to occur as pressure rapidly building up, reducing lung expansion and causes blood vessel collapse, thus severely affecting cardiac output. After opening the chest and manual manipulation of the affected lung, lung ventilation improved.

Ventilation in this case was adjusted throughout anaesthesia. Vt was increased and fR decreased guided by spirometry and capnography. Unfortunately the peak inspiratory pressure (PIP) was not recorded, and thus cannot be reported on, however it was used as a guide to adjust the ventilation; if the PIP decreased with the same Vt (suggesting improved lung expansion), the Vt would be slowly increased. In an attempt to further improve alveolar ventilation, and re-expand previously compressed alveoli, a reverse inspiratory and expiratory time (2:1) was used for a short period when the chest was open. This prolonged inspiratory time, may assist with recruiting already collapsed alveoli whilst not affecting minute ventilation. However this can cause an intrinsic positive end expiratory pressure (PEEP); due to shorter expiratory time, and the decreased transpulmonary pressure and mean blood pressure[13], however this is less of a problem in an open chest[11]. In this case, mean blood pressure was not affected, intrinsic PEEP was not observed on pressure-volume loops on spirometry, and EtCO2 implied improvement in V/Q mismatch. Other methods of alveolar recruitment, e.g. specific manoeuvres with increasing pressure and PEEP for a brief period, could have been used as an alternative to using inverse ratio ventilation. Blood gases taken (see table 1 and 2) at the end of surgery (in sternal recumbency) showed markedly improved oxygenation, improved alveolar ventilation and improved venous admixture.

In addition to the LLT concerns, aspiration pneumonia is commonly reported in brachycephalic breeds, and is thought to be due to increased negative intrathoracic pressure predisposing to regurgitation and vomiting[3]. Aspiration pneumonia is also a risk factor during pregnancy, where there is delayed gastric emptying, decreased oesophageal sphincter tone and increased gastrin

levels, all of which can increase the risk of vomiting and regurgitation[4]. A patchy multifocal alveolar pattern was noted on the caudodorsal lung fields, potentially indicating non-cardiogenic oedema, or possibly aspiration pneumonia. During induction, keeping the head elevated until the airway is secure prevents any possible digesta entering the trachea.

In general, for non-obstetric procedures, preserving uterine blood flow for uteroplacental perfusion is important for foetal safety and prevention of premature labour. Uteroplacental perfusion is blood pressure dependant from the dam's circulation[4, 14]. Thus hypotension or increased vascular resistance (e.g. in response to pain or shock) can negatively affect the uteroplacental perfusion. In addition, during pregnancy there is an increased oxygen demand and metabolic rate and periods of hypoxaemia and hypercapnia have both been shown to have detrimental effects on the foetus. Hypoxaemia can cause uteroplacental vasoconstriction and reduction in perfusion, causing foetal hypoxaemia. Hypercapnia may cause foetal respiratory acidosis, uterine vasoconstriction and reduction in uterine blood flow[4,10]. In this case, hypotension did not develop, however severe ventilation impairment with severe hypoxaemia and mild hypercapnia did.

There are no anaesthesia agents licensed to use in pregnant animals, and all have a potential to cause a direct or indirect adverse effect to the foetuses, and no single protocol can be recommended for pregnancy or other non – obstetric procedures[14,15]. Most anaesthetic drugs cross the placenta, however this is less of a concern in a non-obstetric procedure, as drugs will return to the dam's circulation to be eliminated. In a late stage pregnancy non-steroidal anti-inflammatory drugs are often avoided, as they can increase the risk of miscarriage, cause malformation of foetuses in pregnancy and can cause premature closure or narrowing of the ductus arteriosus[15]. There are no licensed oral analgesics for pregnant animals available, however paracetamol (combined with codeine) is licensed for dogs for post-operative analgesia and there are no known contraindications for its use during pregnancy[16]. As this product is not available for intravenous injection, paracetamol [Perfalgan, Bristol-Myers Squibb Pharmaceuticals Ltd] was used initially according the off label use of drugs legislation by the prescription cascade in the United Kingdom (UK), where intravenous formulation of paracetamol is authorised in the UK for human use.

Pugs are over represented with LLT and the left cranial lung lobe reported to be the most common lobe affected[1,5]. It has been hypothesised that the potential reason for this is due to upper airway resistance due to breed conformation. It is not possible to be sure if the pregnancy status of the dog increased the risk of LLT developing or only if it was an unfortunate coincidence.

LEARNING POINTS/TAKE HOME MESSAGES ***3 to 5 bullet points – this is a required field***

- Lung Lobe Torsion can cause hypoxaemia, hypoventilation and venous admixture, due to lung pathology.
- Pulse oximetry may not indicate large changes in PaO_2 when using high inspired oxygen concentration.
- Blood gas calculations allow further assessment of ventilation including CO_2 (A-a) gradient, O_2 (A-a) gradient, minute ventilation and physiological dead space equation.
- Positioning with a head-up tilt may allow improved lung expansion in late pregnancy patients.

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FIGURE/VIDEO CAPTIONS ***figures should NOT be embedded in this document***

Figure 1. Three dimensional computed tomography reconstruction of the inflated lung parenchyma showing the abrupt discontinuation (arrow) of the bronchus of the cranial portion of the left cranial lung lobe that is torsed (delineated by red dotted line). Note the cranial displacement of the stomach (asterisk) causing compression of the left caudal lung.

Figure 2. Transverse view of thoracic computed tomography (lung window) highlighting the patchy alveolar pattern (arrowheads) in the caudal lung lobes.

Figure 3. Dorsal view of thoracic computed tomography angiography (soft tissue window) showing the left cranial lung lobe torsion (arrow) and distended uterus (asterisks). Note the cranial displacement of the diaphragm.

OWNER'S PERSPECTIVE ***Optional***

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