U.PORTO

INSTITUTO DE CIÊNCIAS BIOMÉDICAS ABEL SALAZAR
UNIVERSIDADE DO PORTO

Final Training Report

Integrated Masters Degree in Veterinary Medicine

Small Animal Medicine and Surgery

Maria Ferreira Gonçalves Dias de Morais

Supervisor:
Professor Doutor Augusto José Ferreira de Matos

Co-Supervisors:
Dr. Christopher Seymour (Royal Veterinary College)
Dr. Michał Dubiel (Animal Medical Centre Referral Services)

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Abstract

During the last four months I did my final training in Small Animal Medicine and Surgery. This training took place in the Animal Medical Centre Referral Services (AMCRS) in Manchester for 9 weeks and then I spent another 9 weeks in the Queen Mother Hospital for Animals - Royal Veterinary College in London (QMHA).

During my training in AMCRS I did different rotations in Anaesthesia, Ophthalmology, Soft Tissue Surgery, Internal Medicine and Diagnostic Imaging. I assisted specialists and interns in consults, participated actively in in-patient care, discussed cases and prepared diagnostic plans and treatments, monitored Anaesthesia cases and performed Soft Tissue Surgery as a main surgeon under supervision. I could also get some insight of the positive and negative aspects of running a business, and daily routine in a first opinion/referral practice.

In the QMHA I did a 7 week rotation in Anaesthesia and Analgesia Service and 2 weeks in Emergency and Critical Care service. At this time I could take advantage of all the good outcomes of a training in an University. I was working in the Hospital, having the opportunity to view advanced techniques that are not routinely performed elsewhere and enjoy the Lecturers, Specialists, Residents and Intern's teaching. Adding to that I could visit the library and read extensively. During the training I became more passionate about Anaesthesia and Analgesia and it was a big motivation for me to pursue a better education in the field.

After this training period I feel that my objectives where fulfilled and I had a better outcome than I could expect.

Index of Abbreviations

ºC - degrees Celsius

ACh - acetylcholine

ALT - alanine aminotransferase

AMCRS - Animal Medical Centre Referral Services

AP - alkaline phosphatase

AST - aspartate transaminase

BID - *Bis in die*

bpm - beats per minute

CEPSS - congenital extrahepatic portosystemic shunt

cm - centimetre

cmH_2O - centimetres of water

CNS - central nervous system

CO_2 - carbon dioxide

CRI - continuous rate infusion

CRT - capillary refill time

CSL - compound sodium lactate or Hartmann's solution

CVP - central venous pressure

DV - dorsoventral

GABA - gamma-Aminobutyric Acid

h - hour

id - intradermal

im - intramuscular

IOP - intraocular pressure

IOL - intraocular lens

iv - intravenous

IU - international units

kDa - kilodaltons

L - Litre

LIU - lens-induced uveitis

LMN - lower motor neuron

µg - microgram

mg - miligram

$MgSO_4$ - magnesium sulphate

mL - millilitres

mmol - milimole

PCV - packed cell Volume

PDS - polydioxanone suture

pH - potential of hydrogen

PLR - pupillary light reflex

po - *per os*

PRA - progressive retinal atrophy

PSS - portosystemic shunt

QID - *quater in die*

QMHA - Queen Mother Hospital for Animals

REM - rapid eye movement

RR - reference range

s - second

sc - subcutaneous

SID - *semel in die*

TID - *ter in die*

TP - total proteins

TS - total solids

Index

Soft Tissue Surgery Case – Diaphragmatic Hernia

Patient characterisation and reason for visit: Rocco is a 3 month-old male Yorkshire Terrier and weights 2 kg. He presented in the Queen Mother Hospital for Animals (QMHA) Emergency and Critical Care Service with a history of dyspnoea for 8 hours.

History: Rocco was acquired 4 weeks before and he has been well since then. He has been eating, drinking, urinating, and defecating normally, with no vomiting, diarrhoea, coughing, or sneezing, and had no previous episodes of dyspnoea. He had been a bright, happy puppy. Earlier that day, the owner noted him to be dyspnoeic and orthopnoeic. He was unable to settle and a clicking noise was heard on inspiration. No traumatic episodes were noted Rocco is bright and bouncy and jumps up stairs and off furniture. Rocco presented to his primary vets where radiographs under sedation raised questions about the integrity of his diaphragm due to suspicion of his liver in the thorax.

Physical and respiratory examination: On presentation Rocco was bright, alert and responsive, although he was intermittently markedly lethargic and dyspnoeic. He had pink and moist mucous membranes that cyanosed with handling and excitement. He was both dyspnoeic and tachypnoeic. Abdominal palpation was unremarkable. Femoral pulses were synchronous, strong and symmetrical. Peripheral lymph nodes were normal. Lung and heart sounds were reduced on the left hemithorax. Heart rate was 120 beats per minute (bpm), respiratory rate 80, capillary refill time (CRT) 1 s (second) temperature 38 ºC. Respiratory movements were adequate, nares were symmetrical, moist, no discharge was noted, and bilateral air stream present. Frontal sinuses, oropharynx, larynx, and trachea had nothing abnormal to describe. Thoracic wall was integrate.

Problems list: Dyspnoea, muffled heart sounds tachypnoea, intermittent lethargy, cyanosis with excitement.

Differential diagnosis: Traumatic diaphragmatic hernia, peritoneopericardial diaphragmatic hernia, lung lobe torsion, foreign body, cardiac right-left shunt, laryngeal paralysis, pneumonia, pneumothorax, pleural effusion.

Diagnostic tests: Packed cell volume (PCV) and total solids (TS) were 31% and 57g/L, respectively. Venous blood gas and electrolyte panel revealed a mild hyponatraemia [135.7mmol/L; reference range (RR) 140-153] and a mild anaemia consistent with age (29%; RR 35-55). Emergency ultrasound revealed a right displaced cardiac silhouette. There was no peritoneal, pericardial nor pleural effusion.

Thoracic X-ray (Annex I, figure 1 and 2): multiple gas filled small intestinal loops were within the left hemithorax extending cranial to the cardiac silhouette. Normal aerated left lung was no longer visible. There was a slight shift of the cardiac silhouette to the right hemithorax. The cardiac silhouette was poorly visible in the lateral projection and there was border effacement of the left side of the cardiac silhouette in the dorsoventral (DV) projection. There was effacement of the left side of the diaphragm in the DV view.

Abdominal X-ray: Gas filled gastric silhouette causing caudal displacement of the left kidney. Overlying the gastric silhouette there was granular radiopaque material. The outline of the liver is visible caudal to the diaphragm. Part of the small intestine was visible in the caudal abdomen.

Procedures performed: Rocco was admitted to the intensive care unit and received oxygen in the incubator until exploratory celiotomy was performed.

Pre-anaesthetic medication: methadone 0.2 mg/kg iv 15 minutes before induction, and 20 mg/kg cefuroxime iv was administered for prophylaxis.

General anaesthesia: co-induction with propofol 3 mg/kg iv and midazolam 0.3 mg/kg iv,. Fluid therapy with Hartmann's solution (CSL) at 5 ml/kg/h. Rocco was maintained on a combination of 100% oxygen and 2,1% sevoflurane. He had an episode of bradycardia treated with glycopyrrolate 5 µg/kg iv. His breathing was assisted with intermittent positive pressure ventilation.

Surgery: Abdominal and caudal half of the thoracic region were clipped and skin was prepared for aseptic procedure. In theatre he remained in dorsal recumbency. Midline ventral skin incision was performed extending 7 cm from xiphoid cartilage toward pubis. Subcutaneous tissue was bluntly dissected, exposing the external fascia of the rectus abdominis muscle. Branches of the caudal superficial epigastric vein were cauterized. After identification of *linea alba* the abdominal wall was pulled upwards and a 1 cm incision was made in the cranial portion *linea alba* with a scalpel blade. The inner aspect of the abdominal wall was palpated for adhesions, and as none were found the incision was extended caudally with a scissors. The falciform ligament was ruptured manually. Then a 2 cm radial diaphragmatic rupture was found in the left dorsolateral corner, through which his spleen and a large proportion of the jejunum had herniated. The rupture was chronic in appearance. The abdominal contents were returned to anatomical location. Diaphragm defect edges were debrided and repaired with 3-0 polydioxanone suture (PDS) in a modified continuous pattern. The thorax was drained through the diaphragm on closure to re-establish negative pressure within the thoracic cavity. The remainder of abdominal exploration was otherwise unremarkable. Closure was done with 2-0 PDS simple continuous suture for the linea alba and intradermal skin sutures.

Rocco went to the intensive care unit for recovery after the surgery. He has hypothermic (34 ºC) so he was kept in the incubator and oxygen therapy was made. 5 hours later he was bright, alert, responsive and eating. In the following day he was transferred to the soft tissue service wards. After the surgery he remained in the hospital for 5 days where his pain was managed with buprenorphine 0.02 mg/kg iv *quarter in die* (QID - 4 times a day).

Rocco was discharged and his owners were advised that he should remain rested during the following 4 weeks to allow proper healing. Furthermore owners were advised to keep him strictly rested in cage rest during the 2 following weeks. Walks should be always short and never off-lead. It was reinforced that he should not jump or climb stairs. After this 4 week period, exercise should be gradually increased if he was doing well.

Also, his owners were advised to keep Rocco from licking his wound to allow proper healing, and for that the use of a buster collar was advised, specially for times when he was left unattended and at night. His wound was to be checked daily to ensure there was no redness, heat, swelling, discharge or malodour.

Discussion: Hernia is a protrusion of an organ or part through a loss of continuity in a wall of an anatomical cavity. The three parts of a hernia are the ring, the sac and the contents. The hernial sac consists of a tissue involving the herniated contents. The nature of the contents can be predicted by the anatomical region where the hernia is located, but it is not reliable as mobile organs such as the omentum and intestines can be involved at most times. Herniation may occur following a modification on the function of the body cavities or organs involved. Diaphragmatic hernias are usually characterised by etiology, as these are not true hernias as described previously. The viscera are not contained in a sac, but lie free inside the pleural cavity or pericardial sac.[1]

Trauma is the most prevalent cause of diaphragmatic hernia in dogs and cats. 77-85% of the cases were traumatic in origin and 5-10% were congenital and the remaining idiopathic. Road traffic accidents where the main cause of trauma, followed by kicks, falls and fights. Injury to the diaphragm can either be direct, with a penetrating object (sharp object stab, projectile) rarely encountered in animals, iatrogenic, or they can be indirect. Indirect trauma leads to a sudden increase in intra-abdominal pressure and at the same time forceful stroke to the abdominal wall and if the glottis is open, it induces a fast lung deflation, producing a large pleuroperitoneal pressure gradient.[1]

The diaphragmatic muscles are more often ruptured when compared with the central tendon. Crural muscles are rarely involved. Left-sided hernias are more often encountered. In humans, it is believed this is due to a protective effect to impact that liver has on the right side of the diaphragm. The organ most frequently herniated is the liver, followed by the small intestine, stomach, spleen, omentum, pancreas, colon, cecum and uterus.[1]

Many diaphragmatic hernias are diagnosed weeks after injury. The animals can present in shock acutely after trauma or it can be an incidental finding. Usually animals present with respiratory signs including dyspnoea and exercise intolerance, or gastrointestinal signs such as anorexia, vomiting, diarrhoea, weight loss, or they can be nonspecific, like depression.[2]

Clinical signs can include pale or cyanotic mucous membranes, tachypnoea, tachycardia, and/or oliguria. Cardiac arrhythmias are common and associated with high morbidity. If the liver is herniated, hydrothorax can arise, caused by entrapment and venous occlusion.[2]

Diagnosis of pleuroperitoneal diaphragmatic hernia is usually performed by radiography or ultrasound. Diaphragmatic hernia radiographs will show loss of the diaphragmatic line and cardiac silhouette, dorsal or lateral displacement of lung fields, pleural effusion, failure to find stomach or liver in the abdomen. This diagnoses can be difficult if only a small portion of an organ is herniated. If the herniation is not obvious ons radiographs, ultrasound of the diaphragmatic silhouette can aid.

A hernia can be misdiagnosed if severe contusions are present in the lung, because in these cases it is very similar to liver in appearance on ultrasound. Also a normal mirror-image artifact should not be mistaken for herniated liver. A positive contrast celiography may also be helpful. Specific laboratory findings are not common. In cases of liver herniation, serum alanine aminotransferase (ALT) and serum alkaline phosphatase values can be increased.[2]

If the animal presents dyspnoeic, oxygen should be delivered by face mask, nasal insufflation or oxygen cage. Positioning in sternal recumbency with the forelimbs elevated can help ventilation. If pleural effusion is detected, thoracocentesis should be performed. If the animal is in shock, fluid therapy and antibiotics should be started.[2]

Acute diaphragmatic hernias may have a lower mortality than chronic diaphragmatic hernias. However the prognosis with surgery is good to excellent. If the lungs have severe contusions, the patient condition should be first stabilised before undergoing surgery. If the stomach is encountered distended within the thoracic cavity, this can rapidly lead to respiratory impairment and death. Therefore these patients should go to surgery as soon as they can be safely inducted to anaesthesia.[2]

As the animal's breathing is compromised, drugs with minimal depressant effects should be elected. This was seen with Rocco: because propofol is a deep respiratory depressant and can even generate apnoea after induction, a co-induction plan was followed with midazolam and propofol. Ventilation equipment should be readily available. Intermittent positive pressure ventilation should be performed, but carefully avoiding high inspiratory pressures if possible. Nitrous oxide is contraindicated in these patients. Adequate pain management is crucial in respiratory compromised patients, as these patients need to take slow deep breaths after surgery, and if painful they will take fast shallow breaths.[2]

Repair of chronic diaphragmatic hernia in dogs can be performed with an abdominal flap graft obtained from the peritoneum and transverse abdominal muscle caudal to the diaphragm.[2]

A non-absorbable (like polypropylene) or an absorbable (like polydioxanone or polyglyconate) suture should be used to close the diaphragm.[2]

As the diaphragmatic defect is repaired, air should be drained from the lungs. Thoracic drainage techniques do not ensure communication between both hemithorax, therefore if a small mediastinal puncture is made, it might not eliminate the desired amount of air of the bilateral pneumothorax. Therefore, a large puncture is suggested in the caudal mediastinum[3]

After surgery, patients should be monitored for hypoventilation and oxygen should be provided as required. Reexpansion pulmonary oedema is a possible negative outcome associated with rapid lung reexpansion after repair, so inflation of the lungs should be done carefully with low pressure as the herniated organs are repositioned within the abdominal cavity.[1,2]

Pneumothorax is the most common complication after surgical treatment. This is most seen if the hernia is chronic and adhesions are present. Reexpansion pulmonary oedema is also encountered

if lungs have been chronically collapsed.[2] Mortality rates after peritoneopericardial hernia correction surgery are reportedly high because of the former.[4]

Prognosis is excellent when the animal survives the early postoperative period. If proper technique is performed recurrence is rare.[2]

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Neurology Case - Tetanus

Patient characterisation and reason for visit: Simba is a 10,5 year-old crossbred neutered male dog and he weights 21,7 Kg. Simba presented to the QMHA referred by local vets for further management of suspected tetanus. **History:** 10 days before referral Simba went out on a walk and came back with intermittent front left lameness. He presented to own vets 4 days later for concerns about his vision due to blurry eyes noticed by his owner. In the course of consultation lameness, progressive hyporexia and lethargy where mentioned by the owners. According to history, he was in owners possession since last year and no previous medical history other than low grade heart murmur was known. He enjoys exercise - swimming, chasing rabbits and going down fox and badger holes. Owner also has chickens and horses. Simba is fully vaccinated, receives Advocate® monthly and Drontal® every 6 months. He has been on meloxicam once daily for suspected cruciate ligament injury - last dose 1 day before admittance to own vet. On presentation Simba was trembling and panting, his temperature was 39.3 ºC. He was lame on the left forelimb, 5th digit on that limb was painful and swollen. Both hindlimbs showed moderate weakness. He appeared cross-eyed, both conjunctiva inflamed and had bilateral cataracts. Menace and pupillary light reflex (PLR) normal, no muscle wastage, proprioception normal on all 4 limbs, hopping and panniculus were normal. Haematology biochemistry and electrolytes were unremarkable. At this time treatment for suspected tetanus was initiated: Metronidazole 10 mg/kg IV *bis in die* (BID, twice daily), single administration of Tetanus antitoxin 51,5-154,5 IU/kg IV and single administration of Buprenorphine 20 µg/kg IV. Throughout the week he was dysphagic and on and off food. His ears were erect, enophthalmos and third eyelid protrusion were noted. He went progressively stiff, face and neck felt tender. He was presenting tremors which did not seem to respond to diazepam but no trismus. **Physical and neurologic examination:** When Simba presented to the QMHA he was alert and moderately responsive. He remained in lateral recumbency with all limbs in rigid extension and marked tremors. When assisted he was able to stand up remaining in wide stance. He had a mild *Risus Sardonicus* and erect ears. Body score was 4/10, dehydration status below 5%, rectal temperature was 39.4 ºC, mucous membranes were pink with a CRT of 1. Heart rate 60 bpm, panting and normal lymph nodes. Thoracic auscultation was affected by acoustic artefacts due to tremors. Peripheral pulses were normodynamic and synchronous. Nail bed of the fifth digit of left forelimb was swollen and erythematous. Cranial nerves were normal, palpation revealed increased muscle tone, spinal reflexes were normal, postural reactions were not performed due to his stiffness and temperament. **Problems list:** tremors, increased muscle tone, generalised stiffness, *Risus Sardonicus*, pyrexia, nail bed infection of fifth digit of left forelimb, historic heart murmur. **Differential diagnosis:** Tetanus, Polymyositis, Hypocalcemia, strychnine poisoning, seizures, meningitis, LMN disease. **Diagnostic tests:** Haematology, and electrolytes had nothing abnormal to describe. Blood glucose was 5.1 mmol/L. Emergency blood gas analysis revealed a mild respiratory alkalosis. Oblique and dorsopalmar radiographs of the left forelimb (Annex II, figures 1 and 2) revealed distal phalanx of the fifth digit with a moderately heterogenous and radiolucent appearance with mild diffuse osteolysis and soft tissue swelling in association. This was consistent with osteomyelitis or neoplasia, the former being more likely in Simba's case.

Diagnosis: Tetanus graded as class III severity (Annex II, table 1). **Treatment and progression:** Simba remained in intensive care during his 8 day stay in the QMHA. He was kept on fluids, CSL 3ml/kg/h IV, Maropitant 1 mg/Kg IV *semel in die* (SID once daily) and Omeprazole 1 mg/kg iv SID for vomiting. Muscle relaxants and sedatives, Diazepam 0.5 mg/Kg IV SID, Acepromazine 0.03 mg/Kg IV as required and Methacarbamol 50 mg/Kg PO *ter in die* (TID, three times daily), continued Metronidazole 10 mg/Kg IV BID for antibiosis, and Buprenorphine 0.02 mg/Kg IV TID for pain relief. Simba was kept in a quiet, dark and comfortable bed, with as little manipulation and traffic as possible. Ears were plugged with cotton buds to protect from sounds. He underwent a general anaesthesia for left forelimb digit radiography and amputation of his fifth digit given the active infection and radiographic evidence of osteomyelitis. Bacterial culture of the amputated toe revealed no bacterial isolates after 48h aerobic and anaerobic incubation. An urinary catheter was placed during the first half of his stay. An oesophagostomy tube was placed under the same anaesthetic, due to concerns about progression of the disease. Positioning was checked radiographically. He improved over the days and in the last day he went home eating well, very bright although he still had abnormal facial spasticity. Simba went home with Methocarbamol 45 mg/kg TID PO, Meloxicam 0.2 mg/kg BID PO for five days, Tramadol 2-4 mg/kg PO and Acepromazine 1 mg/Kg PO as needed. **Discussion:** Tetanus is a state of sustained muscular contraction without the periods of relaxation caused by repetitive stimulation of the motor nerve trunk at very high frequencies that individual muscle twitches cannot be distinguished from one another.[1] In clinical medicine, the term *tetanus* is generally used to describe the disease caused by *Clostridium tetani* potent toxin, *tetanospasmin*, produced within the body by the pathogen's vegetative form.[1,2] *C. tetani* is a motile, gram-positive, non encapsulated, anaerobic, spore-forming bacillus. This bacterium is ubiquitous, being found in faeces of humans and animals (mainly horses) and soil, where conditions like increased moisture, fertilization and cultivation favours their survival.[1,2,3] These spores can survive adverse weather conditions in the absence of direct sunlight for months or years. Spores are resistant chemical and physical inactivation than other microorganisms, however the vegetative phase is not. The organism is typically nonpathogenic if ingested, considering the organism remains in the gastrointestinal tract.[3] For germination to occur, and disease to develop, it's essential that the spores are introduced into wounds or penetrating injuries and are kept in anaerobic conditions. This mostly occurs at the site of injury, in a necrotic tissue, abscess, in presence of a foreign body or other microorganisms. Infections are also described after surgical contamination, including ovariohysterectomy, by inadequate sterilisation of instruments or as a complication of improper management of wounds.[3] Two toxins of *C. tetani* have been identified: *Tetanolepsin* and *Tetanospasmin. T*he first causes haemolysis during rapid in vitro growth and it's not considered clinically relevant. The second produces marked effects on neurologic function. Tetanospasmin is a dimer composed of a heavy chain (H) (100 kDa) that binds to gangliosides on nerves and subsequently internalises and transports proteins, and a light chain (L) (50 kDa), a metalloprotease that blocks the release of neurotransmitters.[2] The toxin enters the nervous system at the neuromuscular end plate and ascends via retrograde transport within the axons of nerves to the spinal cord within hours, where it transsynaptically invades the inhibitory interneurons and then disseminates throughout the central nervous system.[1,2,3] It can also reach

the central nervous system throughout the blood stream, lymphatics or cerebrospinal fluid.[3] The L chain cleaves a membrane protein present in synaptic vesicles, synaptobrevin, responsible for the release of the inhibitory neurotransmitters glycine in the spinal cord and gamma-amino butyric acid (GABA) in the brain-stem motor nuclei. This results in unopposed excitation within the nervous system.[1] Glycine is the neurotransmitter for primary inhibitory interneurons such as the Renshaw cell.[1] GABA is the inhibitory transmitter for descending pathways. Thus, it occurs permanent LMN firing.[1] Ultimately, the toxin may also act directly on the cerebral cortex, resulting in seizures, and the hypothalamus, causing autonomic nervous system dysfunction.[3] The binding of the toxin is irreversible and thus recovery depends on the production of new axon terminals, which explains the delay in recovery.[3] The prevalence in dogs and cats is relatively low when compared with horses and humans.[2] This is why, these more susceptible species are vaccinated against the disease and being a rare disease in dogs and cats it is considered that they don't require immunoprophylaxis.[2,3] The mortality rate in dogs is up to 50%, but with early diagnosis and intensive therapy and nursing, the prognosis is good and up to 90% of the cases survive.[3] The dog and the cat might have a natural resistance to tetanospasmin due to an inability of the toxin to penetrate and bind to nervous tissue. Clinical signs depend on the species susceptibility and amount of toxin[1]. Cats appear to have frequently the localised form, with extensor rigidity in a limb, and dogs are frequently strike with generalised tetanus, resulting in severe muscular contraction.[2,3] In dogs, younger animals appear to be more susceptible and have increased risk of developing more severe clinical signs.[1,4] The signs usually occur 5 to 10 days after infection, as it happened to Simba, assuming that the lesion found in the toe was the site of inoculation of the agent.[1,2] This time varies and is shorter if the wound is closer to the CNS, has increased number of organisms, and has a more anaerobic environment, and may take up to 3 weeks to show due to increased resistance.[2] If the toxin travels in a nerve of a limb, that limb shows signs first, then the opposite limb and eventually the whole body.[1] If the toxin is in the systemic circulation, then the signs will appear first in the head, with protrusion of the nictitating membrane, sunken eyes and contraction of the facial mastication muscles, and then spread to the entire body.[1,4] Amongst the generalised signs, dogs show increased muscle tone and stiffness, usually in all four limbs and in the muscles of the head. In the early typical signs we can recognise the lips drawn, making it look like a "grin", often known as "*risus sardonicus*", the ears are contracted towards each other, specially in dogs without pendant ears, trismus, stiff gait and tail elevation.[1] Later on, the animal is recumbent with extension of all four limbs and opisthotonus.[1] Auditory and visual stimulation enhance these signs, resulting in violent painful spasms.[5] Death may occur after respiratory complications related to inability to breathe due to rigidity of the respiratory muscles, aspiration pneumonia as result of laryngeal paralysis, or from cardiac arrhythmias, consequent to autonomic dysfunction.[1] Autonomic effects seen include cardiac arrhythmias like bradyarrhythmias, including atrioventricular blocks and atrial standstill. Furthermore, tachyarrhythmias, including supra ventricular tachycardia and accelerated idioventricular arrhythmias can occur. Tachypnoea and increased inspiratory stridor may appear consequent to laryngeal paralysis. Mechanical ventilation is needed if respiratory failure due to respiratory muscle paralysis initiates. Hiatal hernia and megaoesophagus may be present and result in regurgitation.[1] The exact mechanism of hiatal hernia is often congenital, but in

tetanus is not well known, although it is hypothesised that it can be caused by abnormalities in oesophageal motility and tone, combined with diaphragmatic spasms.[4] Some dogs experience urinary retention and require catheterisation.[1,4] Continuous muscle activity likely leads to hyperthermia, often seen in affected patients.[1] The diagnosis of generalised tetanus is based on the typical clinical signs *risus sardonicus*, trismus, ocular abnormalities, erect ears, altered facial expression, generalised muscle rigidity with spastic tetraplegia, hyperaesthesia and hypersensitivity to auditory stimuli. A search for an inciting wound must not be forgotten, as wound debridement can minimise further toxin elaboration, although the inciting wound is not always found. Differential diagnosis can be excluded based on a lack of consistent history, presence of concurrent signs and progression of the disease. Haematology and biochemistry indicate nonspecific changes, but are imperative as a guide to supportive care.[5] Culture of the wound site with clostridial organisms growth is supportive of the diagnosis, but in many cases may result in a false negative, and intra-abdominal infections, such as metritis and abscesses may be a cause. In young dogs, the loss of deciduous teeth may also be a source of infection.[1,5] Electromyography is described particularly helpful in diagnosis of focal tetanus, showing prolonged insertional activity and spontaneous continuous motor unit discharges that occur in both the agonist and antagonistic muscle groups.[1] Measurements of serum antibody titers to tetanus toxin can be used to substantiate the diagnosis.[1,2] The goals of the treatment are to neutralise unbound toxin, inhibit further growth of *C. tetani* with antibiotics, and provide supportive care until the effects of the toxin have worn off. To neutralise unbound toxin, tetanus antitoxin should be administered. Its efficacy is unproven and the optimal dose is not well defined, as the antitoxin will bind to the circulating toxin unbound to the CNS or yet to be formed, but as the toxin travels within axons, it is not clear what role it plays in peracute stages of the disease, but it will not speed recovery.[1,2,5,6] Antitoxin should be administered early in the course of disease, and preferably by IV route, however it should be given cautiously as it is associated with high prevalence of anaphylaxis.[1,2] A dose of 100 to 1000 IU/Kg is recommended for dogs. An intradermal (ID) test injection of 0.1 to 0.2mL of antitoxin should be performed before IV administration. Epinephrine, antihistaminics and corticosteroids should be readily available in case of an anaphylaxis event.[1,2] Metronidazole has become the drug of choice for treatment of tetanus in human patients because it is bactericidal against anaerobes, rapidly achieves therapeutic concentrations in nearly all body fluids and tissues, including abscess cavity, and *in vivo* activity is not inhibited by local pH or inactivating enzymes, although it has a higher risk of causing toxicity, when compared with penicillin and tetracyclines.[1,2,6] Penicillin was considered the antibiotic of choice in the treatment of tetanus, but its structure is similar to GABA, thus it may act as a competitive inhibitor, potentiation its inhibition in the disease. At high concentrations, penicillins may cause CNS hyperexcitability and seizures.[1] Antibiotics used in the treatment of tetanus include metronidazole 10 mg/kg administered PO every 8 hours, penicillin G 20,000 to 100,000 IU/kg, administered IM or SC every 6 to 8 hours and clindamycin 3 to 10 mg/Kg administered PO, IV or IM every 8 to 12 hours. Antibiotics should be administered for a minimum of 10 to 14 days.[1] If patients develop aspiration pneumonia or urinary tract infections, culture and antibiogram should be performed and choice of antibiotic treatment should be made according to the results.[5] Patients suffering severe muscular spasms, hyperexcitable states and seizures

require sedation and muscular relaxation. In addition, patients with tetanus should be kept in a quiet and dark environment and stimulation must be avoided.[1,5] Phenothiazine tranquilizers, depress descending excitatory neurons in the brainstem, such as acepromazine or chlorpromazine, and should be given to patients that are cardiovascularly stable. Acepromazine at 0.02 to 0.05 mg/kg every 4 to 8 hours is an ideal first choice drug. Adjunct sedation may be provided with diazepam at 0,5 to 1 mg/kg IV when required, or midazolam CRI at 0.2 to 0.5 mg/kg/h. Phenobarbital is used to control seizures if diazepam is not sufficient. Glycopyrrolate can be used to treat bradycardia. Other drugs like methocarbamol, dantrolene and baclofen have been used for muscle relaxation, but their effect is variable.[5] Pyridoxine (vitamin B6) can increase GABA concentrations in the body as it is a coenzyme involved in its production. Severely affected patients may require anaesthesia with propofol infusions. Buprenorphine at 0.01 to 0.03 mg/kg IV every 6 to 8 hours should be administered to control pain.[1,5] Magnesium sulphate ($MgSO_4$) is thought to be beneficial in tetanus from its action as a nonspecific calcium blocker. At the neuromuscular junction, magnesium decreases calcium intake to presynaptic terminals leading to decreased acetylcholine (ACh) release. Furthermore, it decreases the sensitivity of postsynaptic motor endplates to ACh resulting in muscle relaxation.[7] Any wounds or abscesses should be debrided under general anaesthesia. The wounds should be extensively cleaned and managed as necessary. Many patients have pharyngeal or laryngeal dysfunction and in these cases feeding tubes should be considered. Nutritional requirements in tetanus are increased and patients should be closely monitored for weight loss. Patients should also be monitored closely for upper respiratory tract obstruction, aspiration pneumonia and hypoventilation. If hypoventilation is observed, positive pressure ventilation is required. Urinary catheterisation may be considered and in these cases urinalysis should be included in daily monitoring. The patient should be turned regularly to prevent pressure sores and as the patient improves, physiotherapy concentrating on passive motions can be used to prevent joint stiffness.[5] Reports of long-term complications from tetanus after recovery are rare. Increased movements during sleep, specially in REM phase were reported by authors a few days up to a few months after recovering from tetanus. After infection, patients do not acquire long-term immunity and are at risk of re-infection.[5]

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Anaesthesia and Analgesia Case - Extrahepatic Portosystemic Shunt

Patient characterisation and reason for visit: Rosie is a 21 weeks old crossbred entire female dog and weights 4 Kg. Rosie presented to the QMHA referred from her local vets for further investigation of abnormal behaviour episodes and abnormal bile acid stimulation test results with the clinical suspicion of a portosystemic shunt (PSS). **History:** During the week prior to the referral consult, Rosie presented to her local vets after an episode of abnormal behaviour which involved her becoming unsteady on her feet, circling, ptyalism and head pressing. She was drinking a lot but not passing a lot of urine. She had been off colour and lethargic for over 2 weeks prior to the visit. Blood analysis showed elevated ALT and low platelets. Treatment with enrofloxacin and dexamethasone was started. After treatment, she seemed brighter and more relaxed. 3 days after a bile acid stimulation test, showed high level of bile acids in both pre and post prandial samples. Rosie was of similar size to litter-mates when the owners brought her at 2 months of age, she has put on weight and seemed to be growing normally. Semi-formed faeces and fresh blood was passed 1 day before the referral. She was fully vaccinated and due her next dose of advocate in that week. **Physical examination:** On presentation, Rosie was subdued but responsive. Body score 4/9, dehydration status below 5%, mucous membranes were pink and moist with a CRT of 2 seconds, rectal temperature was 37.9 ºC. Heart rate 96 bpm and respiratory rate was 36. On thoracic auscultation, her lung sounds were prominent but with no crackles or wheezes. Abdominal palpation was comfortable and unremarkable. Peripheral pulses normodynamic and synchronous. Peripheral lymph nodes were within normal limits. **Neurological examination:** Rosie was obtunded, ambulatory, normal posture and gait. Postural reactions were unremarkable except for hopping, which was decreased in pelvic limbs. Menace response was bilaterally decreased, vision also seemed mildly decreased in both eyes. The remaining cranial nerves responses were adequate as well as palpation and spinal reflexes. **Lesion Localisation:** Forebrain. **Problems list:** Unsteady on her feet, circling, hypersalivation, head pressing, lethargy. **Differential diagnosis:** PSS, canine distemper, toxoplasmosis, recreational drugs toxicity, hydrocephalus, hypoglycemia, hyper or hypocalcemia, hypocalemia, hypercalemia, hypophosphatemia, head trauma, epilepsy. **Diagnostic tests:** **Haematology**: mild neutrophilia and a mild anaemia without a regenerative response of 34%. There was also decreased MCH and low MCHC. **Biochemistry**: hypoproteinaemia, hypoalbuminaemia, hypoglobulinaemia, high inorganic phosphorus, low urea, low creatinine, hypocholesterolaemia and hyperbilirubinaemia. Ammonia levels were elevated and urianalysis showed red blood cells but otherwise was unremarkable. **Abdominal ultrasound:** a singe large extrahepatic portocaval shunt. There was secondary microhepatica, bilateral nephromegaly and one small cystholith. **Diagnosis:** PSS – congenital extra-hepatic (CEPSS). **Treatment:** Surgery to ligate the CEPSS, after a medical treatment with amoxicillin and lactulose for a period of two to four weeks to try to reduce the production and absorption of encephalopathic toxins. **Anticipated critical events in surgery:** Haemorrage, hypotension, hypothermia. **ASA classification:** II/III category. **General Anaesthesia:** **Pre- anaesthetic medication:** Pethidine 5mg/kg IM, 15 minutes before induction. **Induction:** propofol (to effect) 4mg/kg IV followed by endotracheal intubation. **Maintenance in prep room:** T-piece with 2.25% sevoflurane and 100%

oxygen. **Fluid therapy:** CSL 5 mL/kg/h. **Lumbosacral epidural anaesthesia** (annex III, figure 1)**:** preservative free morphine 0.1mg/kg. **Local anaesthesia:** TAP block with ropivacaine 2mg/kg. **Maintenance in theatre:** closed circle at low fresh gas flow, 1.5% sevoflurane and 100% oxygen. **Ventilation:** Intermittent positive pressure ventilation, pressure of inspired air of 8 cmH_2O, tidal volume 40-50 mL and respiratory rate 12-20. **Monitoring** (annex III, Figure 2, 3)**:** To assess cardiovascular system, heart rate and rhythm were monitored with electrocardiograph, pulse oximeter, and arterial blood pressure measured every 5 minutes with a sphygmomanometer with Doppler flow probe, after arterial line placement attempted for this purpose without success. Respiratory system monitored parameters: mucous membranes, tidal volume with spirometer, blood gases, capnograph and capnometer, pulse oximeter. Temperature was measured with an oesophageal thermometer and blood glucose assessed every 30 minutes. During the initial 15 minutes of anaesthesia bradycardia (80 bpm) and a 2nd degree atrioventricular block, Mobitz II were noted and managed with glycopyrrolate 5 µg/kg iv, which caused a transient tachycardia. This was repeated at minute 160 and no tachycardia was noted. At 70 minutes clavulanate potentiated amoxicillin was administered for infection profilaxis. Blood glucose was 2,9 mmol/L at minute 80, thus 2,5% dextrose at a constant rate infusion of 4 mL/kg/h was added and CSL rate was decreased to 3 mL/Kg/h. Liver manipulation at minute 95 led to an increase in heart rate, blood pressure and respiratory rate, signs of possible pain, managed with 0,1mg/Kg methadone IV. Extra-hepatic shunt occlusion trial induced a systolic pressure transient drop from 94 to 64 mmHg. Portal blood pressure was measured after occlusion, ranging from 8 to 50 cmH_2O. For this reason only partial occlusion of the shunt was performed, using a Prolene suture. Rosie was under anaesthesia during 160 minutes. Her heart rate range during anaesthesia was 80-210 bpm, respiratory rate 12-30, blood pressure 70-133, end tidal CO_2 35-44 mmHg and blood oxygen saturation above 97%. Temperature dropped to 33,7 ºC. **Recovery:** Ventilator was switched off, sevoflurane as well and Rosie was extubated as soon as she had laryngeal reflexes. Temperature 33,9 ºC, heart rate 110, 20 breaths per minute and systolic blood pressure 110 mmHg. Seizure activity and systemic hypotension were monitored in intensive care unit. She was kept in the incubator to increase her temperature and minimise heat loss. CSL and 2,5% dextrose fluid rate was 4 ml/Kg/h and decreased to maintenance, 2 ml/kg/h, 5 hours after surgery. At this time temperature was 37,6 ºC. Blood glucose levels were measured every 3 hours being within normal limits. Pain was assessed according to Glasgow composite pain scale and methadone was given when pain score was above 3/20, this was every 4 hours. She started eating low-protein renal diet 4 hours after surgery. After 6 days with no post-op complications and marked demeanour and overall condition improved Rosie was discharged with the following therapeutic plan: Amoxicilin/ Clavulanate 12,5 mg/kg PO BID and Lactulose 0,75 ml/kg TID PO until next appointment with own vets, Levetiracetam 20 mg/kg PO TID for 7 days and renal diet. A re-examination was scheduled 12 weeks after to assess total occlusion of the shunt. **Discussion:** PSS induce reduced portal vein flow due to bypass of normal portal blood draining from the stomach, intestines, pancreas and spleen directly into systemic circulation, ignoring its usual passage through the liver.[1] When portal blood bypasses the liver, many substances enter the systemic circulation without metabolization. Also, hepatotrophic substances from the pancreas and intestines do not reach the liver, leading to

insufficient hepatic development or atrophy.[1] Hepatic insufficiency or hepatic encephalopathy often develops.[1] CNS function is altered in hepatic encephalopathy due to hepatic insufficiency.[1] Patients can present with signs from mild depression to coma.[2] It is important that these signs are managed medically prior to surgery if possible.[2] PSS can be congenital or acquired, and intrahepatic or extrahepatic. CEPSS are typically single anomalous blood vessels that bypass blood from the liver to the systemic circulation and represent 63% of PSS in dogs.[1] Purebred dogs have increased risk of CEPSS and it is more frequently diagnosed in miniature and toy-breed dogs.[1] CEPSS are often congenital and more commonly found in animals younger than 2 or 3 years-old.[1] Affected animals are often evaluated due to delayed growth or weight loss.[1] In addition, intermittent anorexia, depression, vomiting, polydipsia, polyuria, ptyalism (mainly in cats), pica, amaurosis and behavioural changes.[1] Haematologic findings can include low packed cell volume and total solids, microcytosis with normochromic erythrocytes, possibly due to low serum iron concentrations, mild non regenerative anaemia, target cells or poikilocytosis.[1,2] Biochemical tests frequently present a decrease in serum albumin and cholesterol increased bilirubin, bile acids and fasting ammonia concentrations.[1,2] Reduced conversion of ammonia to urea in the hepatic urea cycle causes low blood urea nitrogen concentrations.[1] Additional abnormalities may consist of mild to marked increases in serum ALT, aspartate aminotransferase (AST) and AP.[1] In certain occasions these patients may have prolonged bleeding time. Blood glucose can also be decreased.[2] Urinary precipitation of ammonium biurate crystals occurs due to hyperuricemia and hyperammonemia that lead to increased urinary excretion of rate and ammonia.[1] If urate calculi develops, signs of urolithiasis may consist of hematuria, pyuria, and proteinuria.[1] Serum bile acids have been the standard hepatic function test in dogs and cats for years, but nowadays their limitations are recognized.[1] Medical management of Hepatic encephalopathy signs combine a low-protein diet, antibiotics and lactulose, thus aids to reduce ammonia production and other putative compounds such as mercaptans.[2] Patients with PSS have increased incidence of hyperdynamic circulation with increased cardiac output and decreased systemic vascular resistance.[5] Volatile anaesthetic agents induce changes in blood circulation affecting the liver.[2] These agents reduce cardiac output and systemic arterial pressure, thus decreasing liver blood flow.[2] Metabolization occurs in the liver to some extent and metabolites are slowly excreted.[2] Studies found metabolites in urine for many weeks after general anaesthesia.[2] The liver becomes susceptible to hypoxaemia and hypotension with decreased hepatic perfusion at baseline and furthermore under general anaesthesia.[5] Hepatic blood flow may be reduce by 30-50% with anaesthetic agents.[5] Studies show that isoflurane, sevoflurane and desflurane have the least effect on hepatic circulation in patients with chronic liver disease, and methoxyflurane undergoes the greatest hepatic metabolism.[2,4] Therefore these agents are preferred for patients with liver disease.[2,4] When in contact with soda lime or Baralyme, Sevoflurane breaks down producing Compound A, which is potentially hepatotoxic.[2] However it is unlikely that Compound A can reach toxic values with low flow breathing systems.[2] Further parameters that can affect hepatic perfusion intraoperatively include hypotension, hemorrhage and vasoconstrictive drugs.[5] The volume of distribution of neuromuscular blocking agents is increased in patients with liver disease, thus require increased doses to achieve desired effect.[5] Atracurium and cisatracurium may be that agents of choice because they are not eliminated by the liver or

kidneys.[5] Central depressant drugs may have abnormally increased effect in patients with hepatic disease.[2] Chronic hepatopathy is associated with increase in GABA receptors resulting in higher cerebral sensitivity, and increased blood-brain barrier permeability.[2,4] Decreased ability to metabolise and inactivate drugs outcomes hepatic insufficiency.[2] Hypoproteinaemia induces a decrease in volume of distribution of drugs bound to albumin, which can be followed by overdose.[2] Preoperative assessment should evaluate if it is likely that intraoperative colloids or blood products will be needed.[2] Blood or plasma should be administered fresh, as ammonia concentrations increase with storage time.[2] Hyperventilation must be avoided, as alkalosis may increase brain ammonia concentration.[2] The choice of drugs should fall into agents that do not require extensive metabolism or into agents that can be reversed.[2] Studies show that opioid analgesics have little or no adverse effects on the liver, but their actions can be intensified in these patients due to higher cerebral sensitivity and increase in the unbound fraction of these agent. Intravenous morphine administration in dogs causes histamine release. In this species it leads to hepatic congestion as histamine induces spasm of the hepatic vein.[2] Opioids are metabolized in the liver, thus their effects may last longer in animals with PSS.[2] In addition opioids can induce a significant decrease in the release of antidiuretic hormone and therefore in urine output. Premedication with an opioid and atropine is opportune. Phenothiazines have relatively long duration of action, affect systemic blood pressure and lower seizure threshold, so it is prudent to avoid them.[2] Phenothiazine administration has also been associated with thrombocyte disorders, and their use can exacerbate any coagulopathy in the patient with liver disease.[4] α2-agonists should not be used due to the intense cardiovascular effects of these drugs.[2] After administration of xylazine and detomidine bradycardia, atrioventricular block or alterations in plasma glucose may occur.[4] In paediatric patients drugs which require much metabolism should be avoided.[3] Also, drugs that reduce heart rate should be avoided.[3] Pethidine is often favoured in paediatric patients.[3] Pethidine has approximately 1/10 the potency of morphine.[2] It is a selective μ agonist, with excellent spasmolytic and sedative properties, thus it is appropriate for visceral pain. Contrasting with morphine, lacks in unpleasant side effects except for histamine release, which leads to hypotension.[2] Induction either with propofol or isoflurane in oxygen, via face mask.[2] After induction the animal is intubated and anaesthesia maintained with either isoflurane or sevoflurane. Prolonged recoveries are seen with the use of Thiopentone and Ketamine causes CNS side effects, even though it is used in hepatic disease.[2] Diazepam is considered cardiovascularly safe when used intravenously at lower doses than 0.2 mg/kg. Because of liver biotransformation, patients with severe hepatopathy may take longer to recover from diazepam action.[4] Endogenous benzodiazepine-like substances are associated with hepatic encephalopathy development, thus benzodiazepines use is controversial in patients with liver disease.[2,4] Propofol redistribution and metabolism are extremely rapid.[4] It is mostly eliminated by the kidneys (90%).[4] Fluid therapy is crucial, so a intravenous catheter should be placed prior to surgery. If total protein (TP) values are bellow 50 g/L a colloid or fresh frozen plasma should be administered at 5 ml/kg/h during anaesthesia.[2] This prevents hypovolemia, low oncotic pressure and fresh frozen plasma has the added benefit of providing clotting factors.[2] Hypoproteinaemia and hypothermia predispose patients to hypotension.[4] That can also be exarcebated by isoflurane-induced peripheral vasodilation, so arterial blood pressure should be

monitored.[4] Indirect techniques to assess blood pressure can be used such as a Doppler probe, but it is best to resort to direct techniques with an arterial catheter. The dorsal pedal artery is the most commonly used to place an arterial line, but auricular and lingual arteries are also an option.[2] Direct blood pressure monitoring allows faster assessment of changes in blood pressure.[2] Arterial lines also allow arterial blood sampling for blood gas analysis during anaesthesia.[2] PCV and TP can also be evaluated during surgery to assess hemorrhage, although this is not common in CEPSS surgery.[2] Hepatopathy can lead to medication-refractory hypotension, thus catecholamine infusions such as dobutamine or dopamine can have variable responses.[4] Surgical retraction of the liver and compression of the caudal vena cava may increase vagal tone, leading to further decrease in venous return, cardiac output, and arterial blood pressure.[4] In normal dogs, portal pressure usually ranges between 8 and 13 cmH_2O.[4] After occlusion, this pressure should not exceed 20-23 cmH_2O.[4] Central venous pressure (CVP) in this cases is an indicator of blood flow from the intestines to the vena cava.[4] A decrease in CVP after shunt occlusion is important to estimate portal resistance and to predict post-operative complications due to portal hypertension.[4] CVP should not decrease more than 1 cmH_2O 3 minutes after shunt occlusion.[4] Blood glucose is measured and hypoglycaemia should be treated with administration of 5% dextrose in the IV fluids.[2] Temperature monitoring is crucial in these patients, as surgical preparation, opened abdominal cavity and large surface area to volume ratio in small patients highly increases risk of hypothermia.[2] Anaesthetics cause CNS depression, thus decreasing hypothalamus sensitivity to decrease in temperature.[6] Hypothermia has a dangerous general depressant effect, specially when shivering ceases bellow 34 ºC.[3,6] This reduces metabolic rate that leads to decreased drug metabolism, thus a prolonged anaesthesia recovery, also drug overdoses may occur. Furthermore lowers cell sodium pump activity followed by cell swelling electrolyte abnormalities and pH imbalances, such as hyperkalaemia, acidosis, and initial hyperglycaemia.[3,6] PCV increases after cell swelling and acidosis and hypothermia contribute to splenic contraction.[3] This increases myocardial demands.[3] One stage prothrombine time and activated partial thromboplastin time become prolonged as platelets concentrate in blood sinusoids in liver, bone marrow and spleen.[3] Conduction velocities in excitable cell membranes are decreased, therefore the myocardium becomes irritable when bellow 30 ºC and ventricular arrhythmias occur. Anti-cholinergic non-responsive bradycardia develops. Authors believe the fibrillation threshold is 28 ºC.[3] Hypothermia also has a negative effect in CNS activity which leads to reduced minimal alveolar concentration.[3] Confusion develops below 35 ºC, unconsciousness bellow 30 ºC and cerebral activity chests below 18 ºC.[3] Baroreceptor reflexes are depressed resulting in hypotension and decreased cardiac output.[3] Reduced respiratory muscle activity, mucociliary activity, tissue oxygen delivery, and immune cell function are observed. Shivering starts above 34 ºC leading to an increase in oxygen demand.[3] Heat loss should be minimised as soon as the animal is anaesthetised.[3] Methods include insulation with blankets, warm bedding, wrapping of extremities which have high surface area:volume body parts leading to fast heat loss, forced warm air blankets, heat lamps, and electrical heat pads.[3,6] Heat an moisture exchangers humidify inspired gases and reduce evaporative losses.[6] Preparation for surgery with excessive scrub solutions or spirit is to be avoided and the patient should be kept dry as much as possible.[6] Rebreathing systems will also retain heat.[6] Intravenous fluids should be warmed prior to

administration.[6] Incubators design for human infants or animals deliver a temperature-controlled environment for postoperative care and gentle warming.[6] Opioids can be used for postoperative pain management.[2] Parenteral lumbosacral extradural preservative-free morphine injection is executed after induction at 0.1 mg/kg, diluted to a volume of 0.3 mg/kg with a maximum volume of 6 mL.[2] Transversus abdominis plane block can also be performed to provide local anaesthesia to the abdominal wall.[7] This block is a regional anaesthetic technique and it is ultrasound guided.[7] Using local anaesthetic as ropivacaine injection within the fascial plane that overlies the transversus abdominis will decrease opioid consumption and patient satisfaction for up to 48 hours.[7] Postoperative monitoring for 24-48 hours is needed due to risk of portal hypertension after occlusion of the shunt. Seizures may also occur postoperatively and are usually refractory to the standard methods of treatment.[2] Authors describe dogs started on levetiracetam therapy 6.5 days before surgery were less likely to have postoperative seizures, compared with dogs who did not receive anticonvulsant drugs.[8] Drug absorption after PO administration is fast, with peak plasma concentration occurring 37 minutes after the 1st administration.[8] Levetiracetam should be administered at 20 mg/kg PO TID.

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Ophthalmology Case - Immature Cataract

Patient characterisation and reason for visit: Bertie is a 6 year and 3 month-old , male neutered, English Cocker Spaniel with 13.4 Kg. Bertie came to the QMHA, referred from his local vets for further cataract assessment. **History:** Two years ago Bertie had a history of bilateral conjunctivitis and no obvious tear deficiency. This was treated with Fusidic Acid eyedrops and did not recur. A year later Bertie developed a bilateral mucopurulent discharge, treated again with Fusidic Acid eyedrops and improvement of the discharge was noted. At this time he had some lens changes affecting the right eye. Tears were not measured then. Cataracts were suspected to be developing in both eyes. His owners reported that there has been some change in his vision. He was occasionally bumping into their legs, and was not as good at chasing his ball. **Physical examination:** Bertie was bright, alert and responsive. Body score 5/9, dehydration status below 5%, mucous membranes were pink and moist with a CRT of 1 seconds, rectal temperature 38.9 ºC. Respiratory rate was 28 and heart rate 120 bpm. No abnormal findings in the remaining parameters (abdominal palpation, thoracic auscultation and lymph nodes). **Ophthalmic examination** revealed *Oculus uterque* (OU) opened and comfortable. There was an intermittent menace response OU and pupillary light reflexes were as expected OU. Schirmer tear test readings were 21 mm/min and 22 mm/min in *oculus dexter* (OD) and *oculus sinister* (OS) respectively. Examination revealed mild distichiasis affecting all four eyelids but no conjunctival hyperaemia. Corneas appeared healthy. There was no aqueous flare. Lens opacification (anterior and posterior) were present in both eyes (Annex IV, figure 1), more advanced in the OS. Intraocular pressures were 11 mmHg OU. Funduscopy was restricted by the lens and very limited in the OS. In OD there was some patchy hyperreflectivity in the tapetal fundus, otherwise funduscopy was as expected for a healthy dog. In the consulting room he struggled to navigate around the room with the lights off, but managed well with the lights on. **Problems list:** Decreased menace response, distichiasis, bilateral lens opacification, nyctalopia. **Differential diagnosis:** Cataracts, progressive retinal atrophy (PRA), vitamin A deficiency, retinal dysplasia, lenticular nuclear sclerosis. **Diagnostic tests:** Haematology revealed mean corpuscular haemoglobin concentration above the higher limit [24.7 pg, reference interval (RI) 19.5-24.5 pg], plasma colour was consistent with mild lipaemia and haemolysis. Red cell morphology with mild anisocytosis. Occasional smudged reds were noted, which may reflect the lipaemia of the plasma and a low numbers of eccentrocytes were observer, consistent with oxidative damage. Neutrophils occasionally had foamy and basophilic cytoplasm, suggestive of toxic change and recent acute demand. Biochemistry revealed total protein and albumin within superior limit (71.2 and 39.3 respectively, RI 49-71 and 28-39). CH Genetic test for PRA was performed, but due to prolonged time of results these were not available. Ocular ultrasonography (Annex IV, figures 2 and 3) revealed increased echogenicity within the lateral aspect of the lens and posterior part of the capsule of the lens was slightly irregular in the OS. Right eye had slightly increased peripheral echogenicity of the lens, mostly at its posterior aspect. No evidence of virtual or retinal abnormalities were noted in OU. Diagnosis: Incipient bilateral cataract and possible PRA. **Diagnosis:** Immature bilateral cataracts OU (final diagnosis) and PRA (provisional diagnosis, with

DNA test pending results). **Procedures:** Despite Bertie had possible PRA he appeared to have remaining day vision, and could therefore benefit from cataract surgery, which was performed a week later. Bertie struggled to navigate around the examination room with the lights off, but managed well with the lights on. These findings, together with the examination findings and signalment, made suspicion that Bertie was affected by PRA. Some dogs with PRA will have no vision at all after cataract vision and other can regain or continue to have some degree of day vision for a year or, possibly more. Dogs that have cataracts in the early to mid-disease process of their PRA, especially if they are still day-visual at the onset of cataract development are the more likely candidates that could benefit from cataract surgery. This was discussed at length with owners and an appointment was made for phacoemulsification. 2 hours before surgery 1% tropicamide and 0,03% flurbiprofen were administered OU every 30 minutes. After anaesthetic induction, neuromuscular block was performed with Atracurium 0,5 mg/kg iv for the eye to remain central. Also antibiotic, Cefuroxime 20 mg/kg, methadone 0,2 mg/kg and carprofen 4 mg/kg were injected IV. Then, routine bilateral phacoemulsification was performed, with intracameral administration of epinephrine. Trypan blue was used to improve visualisation of the anterior capsule and a ease capsulorhexis. The elected technique as “divide and conquer”. Following ultrasonic irrigation and aspiration of the lens a foldable single piece 14 mm internal diameter intraocular lens (IOL) Acrivet® square-edge haptic 60V-14 was injected into the intact capsule in both eyes. Intracameral carbachol was injected to prevent glaucoma. Incision was closed with 9-0 Vicryl®. After surgery 1 drop OU of solution with 0,1% dexamethasone, 3.5 mg neomycin and 10000 units of polymyxin B sulphate (Maxitrol®) was administered QID, cephalexin 20 mg/kd PO BID and intraocular pressure (IOP) was measured OU Q4h. Anaesthesia and recovery were uneventful. Bertie was discharged with Cephalexin 10mg/kg PO BID during 7 days, Carprofen PO 4mg/kg SID for three weeks, Maxitrol® 1 drop OU QID for one week, then drop OU TID in the following week. Following that, Maxitrol® was to be replaced with a solution with 0,1% dexamethasone (Maxidex®), 1 drop OU BID for one week, and 1 drop OU SID from the next week on, until indicated otherwise by a Veterinary Ophthalmologist. Lastly, 1 drop OU of 2% dorzolamide and 0,5% timolol solution (Cosopt®) BID for seven days. **Discussion:** The lens has a biconvex body, which is transparent and avascular. It is composed by the capsule, anterior epithelium and lens fibers and divided into cortex and nucleus. In order the lens to grow, layers of fibers are produced in the equatorial area and sit on top of the former layers, forcing older fibers toward the lens centre. As the older cells form tighter layers, the nucleus becomes denser and harder. In dogs this starts to become visible as a greyish blue haze at 6 years of age. Energy requirements of the lens are satisfied by metabolism of glucose. The lens is composed of 35% protein and 65% water and low in minerals. Proteins can be either soluble proteins (crystallin) or insoluble (albuminoid), being the former the most abundant. When animals age the content of soluble proteins decreases in relation with insoluble proteins. Cataract development involves a comparable process.[1] Advanced nuclear sclerosis is frequently confused with cataracts by practitioners and owners. In order to distinguish the two, use of mydriatics and retroillumination will highlight the cataract opacities and differentiate them from transparent nuclear sclerosis. In most animals, nuclear sclerosis still allows visualisation of the fundus. The exact biochemical disorders responsible for the development of cataracts is not

well known, with exception of diabetic, galactosemic and experimental cataracts. However, lens opacity may arrive with detrimental effects affecting lens nutrition, energy or protein metabolism, and osmotic balances. These disturbances will cause irreversible changes in lens protein concentrations, metabolic pumps, ionic contents and antioxidant mechanisms. Activity of the epithelial Na^+/K^+ adenosine triphosphate pump lowers, resulting in a shift in the ionic balance within the lens, and antioxidant activity in the lens consequently decreases. Also, proteolytic enzyme activity increases in the lens, breaking down cell membranes and degrading lens protein. Morphologic changes in the lens capsule, epithelium and fibers lead to these events, leading to loss of transparency due to lens fibers rupture, cell death, and water-cleft formation. It is uncommon that the opacification will spread throughout the entire lens cortex. Sometimes they cease further development and have affection to vision. Extensive impregnation of fluid into the cortex will cause at other times complete opacification may be rapid. Cataract can have a varying age of onset, speed and extent of progression, appearance, and etiology. These differ in nature and appearance, numerous methods of classification are commonly used (Annex IV, table 1). Inheritance is the most prevalent cause of cataracts in many canine breeds. According to stage of cataract development, they can be incipient, immature, mature and hypermature. In the former the sight is preserved and there is early, focal opacity. Immature show more extensive opacity, and most of the lens is involved in the pathologic process. The transparency of the lens is not totally lost, allowing slight tapetal reflection, although the fundus may be partially obscured ophthalmoscopically, as seen with Bertie. Vision is affected (just as it is affected by a dirty car windshield or by unclean glasses), but the animal can still see. Mature: The eye is functionally blind due to total lens opacification. Tapetal reflection is absent, and the fundus can no longer be examined ophthalmoscopically. A number of mature cataracts begin to liquify due to proteolysis (lens reabsorption), and progress to hypermaturity. This process usually starts from the cortex spreading to the nucleus. The debris of lens proteins pass through the lens capsule into the anterior chamber. Thus, the lens volume diminishes and the capsule has a wrinkled appearance as a consequence. Without cortex inside the capsule, the nucleus and may sink to its bottom. It should be noted that in young dogs vision can be recovered, as resorption can be so extensive that involves the majority of the cataractous lens. This will lead to secondary inflammation that must be treated aggressively. In older dogs lens resorption can occur as well, although it does not lead to vision recovery.[1] Cataracts etiology may be hereditary, congenital, acquired, senile, diabetic or secondary to other ocular disorders. In Bertie's case, PRA was a strong suspicion, and it is known that PRA or other forms of generalised retinal degeneration are an important cause of secondary cataracts. The exact mechanism is unknown, but it is proposed that the degenerated retina release toxic products may be the cause. Degenerative rod outer segments can release water-soluble dialdehydes from peroxidation of photoreceptor lipid membranes that may diffuse through the vitreous. In earlier stages, these lens opacities form equatorial and posterior cortical vacuoles, Y-suture changes, and a higher diffuse in relucency to the posterior subcapsular region. Clinical presentation of PRA in middle-aged English Cocker Spaniels and Miniature Poodles is frequently cataract formation. In these cases owners erroneously attribute the vision loss to the

cataracts. When gathering the history, owners mention that before the formation of cataracts there was vision impairment, more noticeable under poor lighting conditions. PLR may give an indication of retinal function, but it is unreliable thus electroretinography is preferred before considering cataract surgery. Dazzle reflex may be present in affected patients because of residual retinal function.[2,3] Lens-induced uveitis (LIU) occurs as lens antigens are present in the aqueous humour, leading to an inflammation of the eye. Leakage is observed from the lens into the anterior chamber after the breakdown of lens protein in cataracts, thus it is called *phacolytic uveitis.* The degradation and resulting LIU accompany the severity of cataracts, and are limited in mature cataracts and more extensive in hypermature cataracts. Phacolytic uveitis is a humoral and cell-mediated immune reaction of the uvea to the released lens protein. Proteins are separated from the immune system before birth. Thus they are recognized as foreign when outside the lens and inflammation occurs. Inflammation is less severe in younger animals. Clinical presentation of uveitis include photophobia, corneal oedema, blepharospasm, ciliary injection, aqueous flare, miosis, a dark iris, and hypotony. Medical treatment must be started as soon as LIU is detected, as progression may severely effect the prognosis of cataract surgery. Progression of LIU may lead to glaucoma and posterior synechia. LIU following traumatic rupture of the lens capsule is known as *phacoclastic uveitis*.[1] The most seen complications of LIU are glaucoma and phthisis bulbi. [1]Cataract removal is usually recommended when demonstrable visual deficits are noted. Astuteness of the owner to detect clinical changes and the level and type of activity, and acuity of other special senses, such as hearing and smell, of the dog will vary. Clinically evident changes in behaviour associated with poor vision are not detected by the owner until the cataract is 40–50% complete. Advanced cataracts are not actually associated with PLR abnormalities (from absence of light transmission to the photoreceptors). If surgery is not performed in cataract patients, the condition will continue to progress, and leakage of lens proteins can occur. As it was discussed before, this leakage is followed by LIU requiring medical treatment. Hypermature cataracts are also at risk of subluxation or lunation, which can also bring further complications. Most blind dogs develop permanent behavioural changes and are limited functionally, but they can adapt within their environment. Common behaviour changes noted in blind dogs include a tendency to stay closer to the owner and a more cautious approach to the environment. Owner should be sensitised to adequate fencing or containment of the dog when outdoors, minimal changes of furniture within the home, and limited access to stairs, decks, or pools.[3] For several centuries, many compounds have been used topically and systemically to either delay the formation of cataracts or cause their dissolution. Controlled studies of these substances have indicated no efficacy to date.[1] Perioperative medications aim mydriasis of the pupil to facilitate visualisation of the cataract during surgery and prevent miosis, prevent inflammation and bacterial growth. If LIU is present, medication should be initiated several days prior to surgery and the clinical signs of LIU controlled. Topical broad-spectrum antibiotics and corticosteroids are administered every 6 h, starting 24–48 h before to surgery. The pupil is dilated using a parasympatholytic such as 1% atropine or 1% tropicamide on the day of surgery. Topical corticosteroids and NSAIDs are administered every 30 min starting 1–2 h prior to surgery. A systemic NSAID and systemic broad-spectrum antibiotic are administered right before surgery. Postoperatively, topical antibiotics and corticosteroids are

continued. IOP should be measured closely. Patients are recommended cataract surgery when IOP values are within 15-20 mmHg.[1] ***Phacoemulsification*** starts with a small incision at the limbus and tearing of a round portion of the anterior lens capsule. Then a special probe cracks the lens with high-frequency ultrasound waves. Next robotic irrigation and aspiration of the lens debris is performed. The incision at the limbus, when compared with other techniques, allows faster surgery healing and leads less prevalence of complications, as it is only the size of the phacoemulsification probe. Thus this technique results in a more comfortable post-operative period for the patient.[1] Severe hyperopia develops after surgery, as the patient doesn't have the lens to focus on objects. Implantation of an artificial intraocular lens (IOL) aid the patient to achieve emmetropia, thus corrects the visual deficit. It is believed that IOL should have nearly 41 diopters. These lenses are starting to be widely implanted by veterinary ophthalmologists nowadays, and significant enhancement in visual acuity is noted. Because the cornea is the major refractive organ of the eye, plays a major role in accommodation and focusing in animals. For this reason, postoperative vision without an IOL is possible. As lens capsule is opened during surgery, lens proteins are in contact with aqueous humour and it can lead to LIU. Treatment involves aggressive administration of mydriatic agents and topical and systemic anti-inflammatory drugs. Prophylaxis can start several days before surgery and this also guarantees a dilated surgery during the surgery. Topical and systemic antibiotics are also an option of treatment and also prophylaxis for glaucoma can be added.[1] IOP must be checked postoperatively. Prognosis of treatment is better in dogs who undergo cataract surgery, when compared with medical treatment. Furthermore, higher success rate is seen for hypermature and mature cataracts, when compared with immature cataracts. Studies show that after phacoemulsification a high percentage (>80%) of the dogs had good or excellent vision with or without IOL placement.

The most common complications of the surgery are uveitis, corneal disease, intraoperative hyphema and glaucoma. Retinal detachment can also occur, but the prevalence of this complication is not a consensus.[4,5]

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Dermatology Case - Chronic Otitis Externa

Patient characterisation and reason for visit: Sefton is a 7 year, 3 month-old male 40 Kg Labrador . He presented at the QMHA for further investigation of his recurrent otitis. **History:** His ear disease had been managed since 5 years before, with periodic Otomax® and regular Malacetic Aural® ear cleaner. The intervals between Otomax® treatments gradually decreased, and ultimately no improvement had been seen. Reduced or absent hearing was reported in the right ear by his own vets. Sefton had changed to a rice based diet within the last 6 months, and his digestion had improved. He was being treated with advocate every 6-8 weeks, and a garlic pill was being administered daily. Firocoxib had been given as an analgesic the the previous couple of days. Sefton had had a benign tumour removed from his head two years ago. He was reportedly in good health, and lived with one other dog and a cat, neither of whom expressed signs of skin disease. **Physical examination:** Sefton presented bright alert and responsive. His body shape was adequate, mucous membranes where moist, pink, CRT 1s. Respiratory movements, peripheral lymph nodes, thoracic auscultation and abdominal palpation were uneventful. Respiratory rate 20, heart rate 120 bpm and rectal temperature 38.9 ºC. **Dermatological examination:** Coat was soft and glossy, generalised difficult plucking, more intense odour was found in the ears. Revealed abnormalities that were confined to the ears. Both external auditory meatus showed mottled hyperpigmentation, erythema and brown discharge (annex V, figure 1). **Problems list:** Recurrent bilateral otitis externa, hyperpigmentation, erythema and brown discharge in the ear. **Differential diagnosis:** food allergy, chronic otitis externa, yeast otitis, foreign body, atopy, histiocytoma, mastocytoma, **Diagnostic tests:** Computerised tomography scan of his bullae (Annex V, figure 2) revealed both sides filled with contrast-negative material. Advanced mineralisation of the external ear canals on both sides. Ear lavage and video otoscopy, which showed Both sides mildly stenotic with hyperplastic changes and the vertical and horizontal canals contained many short hairs with large amounts of brown/grey/pink debris in the lumen of the canals and tightly adherent to the hair shafts. Tympanic membranes were absent on both sides. The bullae and the canals were extensively flushed with saline but material adherent to hair shafts could not be completely removed. Haematology: Basophils very mildly elevated 0.04 10e9/l (RR 0-0 10e9/l). All other parameters were within normal limits. Serum biochemistry: Total protein mildly elevated 72.7 g/l (RR 49-71 g/l), mild hypernatraemia 154 mmol/l (RR 142- 153 mmol/l), mild hyperphosphatemia 2.20 mmol/l (RR 0.8-2 mmol/l), mildly elevated total bilirubin µmol/l (RR0-2.4 µmol/l), elevated ALT 343 U/l (RR 13-88 U/l) and elevated ALP 344 U/l (RR19-285 U/l). All other parameters were normal. **Diagnosis:** Chronic otitis externa associated with *Malassezia* and chronic otitis media. **Procedures:** while being investigated, owners noted that Sefton was painful in the ears, when these were cleaned. He started a treatment with Otoclean® to both ears SID, Canesten® BID to both ears, methylprednisolone 4 mg 7 tablets every other day for 10 days followed by 4 tablets every other day for 10 days. A bilateral total ear canal ablation and lateral bulla osteotomy were performed. A swab was taken from the right and left middle ear of culture and biopsy samples were taken from both ears. Sefton went home with cephalexin 22 mg/kg po BID for 8 weeks and carprofen 4 mg/kg BID po for 1 week.

Discussion: The pinnae and vertical canal are formed by the auricular cartilage. At the external orifice of the ear, the cartilage rolls into a funnel shape, becoming tube shaped as it reaches down into the lower portions of the ear canal. The ear canal can vary in length and it is divided in the horizontal and vertical portions. Starting at the pinnae, it extends in a rostral and ventral direction through the vertical portion and then bending medially forming the horizontal canal. Its lumen is 0.5-1 cm in diameter. Different breeds have different diameters, specially from the vertical portion of the ear canal. The skin that lines the ear canal is usually a relatively smooth surface, and has particular adnexa, like hair follicles, sebaceous and ceremonial glands particularly in the vertical canal. Ceremonial gland and hair follicle density varies between different breeds.[1]

Exfoliating cells and glandular secretions form the earwax and cerumen that is believed to have a protective role. Canine immunoglobulins A, G and M have been found in canine cerumen, being immunoglobulin G the most predominant. The epidermis of the ear canal has a clearing mechanism, through which epithelial cell migration, removes the epithelial cells, cerumen, trapped dirt and debris.[1]

Tympanic membrane is the epithelial physical structure that acts as a barrier from the outside environment to the middle ear. The normal tympanic membrane can be assessed through otoscopic examination. It is concave, translucent and has a white C-shaped area in the dorsal part. This area is where the manubrium of the males bone attaches. The tympanic membrane is divided into the pars flaccida and pars tensa. The former is the region next to the manubrium and the pars tensa is the ventral portion.[1]

Tympanic cavity and walls, medial wall of the tympanic membrane, the auditory ossicles and associated ligaments, muscles and nerves (chorda tympani and other smaller nerves), and the auditory tube form the middle ear. The auditory tube is the only communication to the outside environment, and opens into de nasopharynx. The tympanic cavity is formed by the dorsal, middle and ventral part. The smallest is the dorsal part, and it is where the head of the malleus and the incus sit. The middle part, also known as tympanic cavity proper, lays right next to to the tympanic membrane. The auditory tube opens in the middle portion of the tympanic cavity. The tympanic bulla is the largest portion and it is ventral. The tympanic bulla is elliptical shaped, and its dorsal aspect communicates with the middle part. Debris and toxins are typically trapped within the tympanic bulla when otitis media is present. Approach to this structure is difficult, and cannot be well examined by otoscopy. The facial nerve, vagus, carotid and lingual arteries travel next to it.[1]

Otitis externa is inflammation of the ear canal. It is usually classified as predisposing, primary, secondary, and perpetuating. In every case, the clinician should identify as many causes and factors as possible that may contribute to the otitis. Most chronic cases have at least one primary and several other causes or factors present. Failure to recognise and correct one or more of these causes may lead to treatment failures. [1]

Predisposing factors increase the risk of acquiring otitis externa, working in conjunction with primary, secondary and perpetuating factors. These may include the shape of the ear, climatic factors, feline nasopharyngeal polyps and ceruminal gland neoplasms.[1]

Primary causes directly induce otitis externa. Amongst the most commonly seen we can find atopy, food hypersensitivity, keratinisation disorders and ear mites. A range of parasites have been associated with otitis externa, being *Otodectes cynotis* the most common. But this parasite is rarely detected, possibly because only 1-2 mites can develop otitis externa. Another possibility is that the mites leave the canal or are destroyed by inflammation or secondary infection. If encounter a recurrent case, in-contact animals should be investigated as well. Hypersensitivities like atopy, food hypersensitivity and contact hypersensitivity can cause otitis externa. This can develop secondarily to trauma. Erythema of the pinna and vertical canal is a common feature of allergic otitis externa. Bacterial or yeast infection may arise from chronic inflammation. Ear disease is seen in up to 80% of dogs and cats with food hypersensitivity. Cocker spaniels and Labrador retrievers have been suggested as breeds more likely to present with otitis externa. More primary causes of otitis externa are foreign bodies, autoimmune diseases, viral diseases and others.[1]

Secondary causes include bacterial infection, yeast infection, and topical acquired irritant reactions . *Malassezia pachydermatis* is the most common yeast that leads to otitis externa. It has a peanut or bottle shape an may be encountered in as many as 36% of normal canine ears. In otitis it is found in up to 76% of the ears and frequently in combination with *Staphylococcus spp*. It is believed that *S. intermedius* produces a factor that stimulates the growth of *M. pachydermatis.* This yeast is a common complication of hypersensitivity disorders, and a superinfection may arise after antibiotic therapy. [1]

The perpetuating factors don't allow resolution of the otitis. Progressive pathologic changes occur changing the ear's anatomy. The chronically inflamed skin is stimulated to undergo numerous changes, including hyperplasia, oedema, fibrosis, ceruminal gland hyperplasia or inflamation. Studies show that breeds predisposed to otitis externa had more ceruminal glands when compared with the number of sebaceous glands. Inflammation and stenosis may be responsible for slowing, stopping and even reversing the migration pattern of cell migration. [1]

Progressive changes lead to a thickening of the skin, which eventually extends to both sides of the auricular cartilage. Then the canal lumen is stenotic and the skin folds. These fold act as sites for secretions and exudate entrapment and perpetuate opportunist microorganisms.[1]

The tympanic membrane thickens and becomes opaque or slightly pigmented. It may appear white, off-white, yellow, brown, or gray. Thus, it can be mistaken for exudates or keratin plugs and lead clinician to erroneously think the tympanic membrane is intact. The tympanic membrane is capable of restoring itself after rupture, it is common to have otitis media with an intact tympanic membrane. Usually, the tympanic membrane thickens in response to inflammation and polyploid

extensions of granulation tissue can arise into the middle ear cavity, which, in some cases, form adhesions with middle ear mucosa. [1]

Otitis media is inflammation of the middle ear. The normal middle ear cavity has commensal bacteria and occasional yeast and lacks in exudate or inflammatory cells. The presence of exudate within the tympanic cavity is difficult if not impossible to treat with topical therapy as the tympanic membrane blocks the access to the middle year. In more severe cases, the authors have found keratin plugs developing within the tympanic cavity. The keratin may serve as a culture medium for bacteria and a source for inflammation. Eventually, calcification may occur, which may be observed radiographically. In some cases, osteomyelitis of the bony wall or within the newly proliferated bone occurs. Osteomyelitis is difficult to treat medically and often requires surgery to alleviate.[1]

Clinical examination should look for neurological signs, such as vestibular syndrome or facial paresis secondary to otitis media. In cats with suppurative otitis externa it is crucial to test for retroviruses. Not less important is to examine the pharynx and larynx to detect nasopharyngeal polyps. Paraneoplastic disease can accompany otitis externa. Dermatological examination should be performed with care, as the disease is normally a local manifestation of other aetiologies. An otoscopic examination of the external ear canal must be followed. Nervous animals may require sedation because if affected, otitis can be quite painful. The auroscopic examination intents to look for foreign bodies, inflammatory changes and rupture or other abnormalities of the tympanic membrane, thus It should always permit its visualisation. Rupture of the timpanic membrane indicates otitis media. Further investigation includes direct microscopy, bacteriological culture and biopsy.[2]

Therapeutic plan envolves: **cleaning** being a crucial step, as inflamatory debris and pus can inactivate further topical treatment applied. Should be done with a aqueous solution when the tympanum membrane loses its integrity. Is to be repeated 2-3 times a week until the ear is free of unwanted material. The solution is flushed into the ear canal, the ear is gently massaged to emulsify the debris. Then the animal is let free, and will shake its head. The remnants in the ear canal are then dried with absorbent paper. **Topical therapy** should be directed towards the etiology of the disease. In Stefon's case it is *Malassezia*. Products that contain more than three different agents have low concentration of each agent, even though it involves a wide spectrum. Thus, resistances may appear.[2]

Topical therapy is the cornerstone of treatment. It should be directed towards the cause of the disease (e.g. acaricidal treatment in case of otoacariosis, antifungal treatment for *Malassezia* otitis, antibiotics for SOE). The use of products containing more than three different agents should be avoided, as the concentration of each active ingredient is decreased in such preparations. [2]

A number of different products are licensed for use in dogs and cats. to treat malassezia infection. Usually, acute otitis externa treatment with these should last 7-14 days. these include nyastin,

thiabendazole, pimaricin, miconazole and clotrimazole.. Silver sulfadiazine is also effective against Malassezia.[2]

Glucocorticoids are widely present in commercial topical anti-inflammatory preparations. These target pruritus, erythema, exudation and tissue proliferation, and helps promote drainage and ventilation. Care must be taken as usual with therapy with glucocorticoids, even though it is a topical agent and patients should undergo weaning.[2]

Local anaesthetics can provide pain relief. Systemical antibiotherapy is used in otitis media. Antibiotic selection should be based on culture and sensitivity findings. Treatment should be given for an extended period of time, involving several months.[2]

Surgery is indicated in otitis externa when diagnostic investigation followed by cleaning procedures and appropriate medical treatment don't lead to a cure. About 50% of the otitis media cases require surgery and total ablation of the external auditory canal along with ventral bulla osteotomy is the only way to treat the disease. Postoperative complications include transient Horner syndrome, pain, pinna necrosis and problems related with the incision.[2,3,4]

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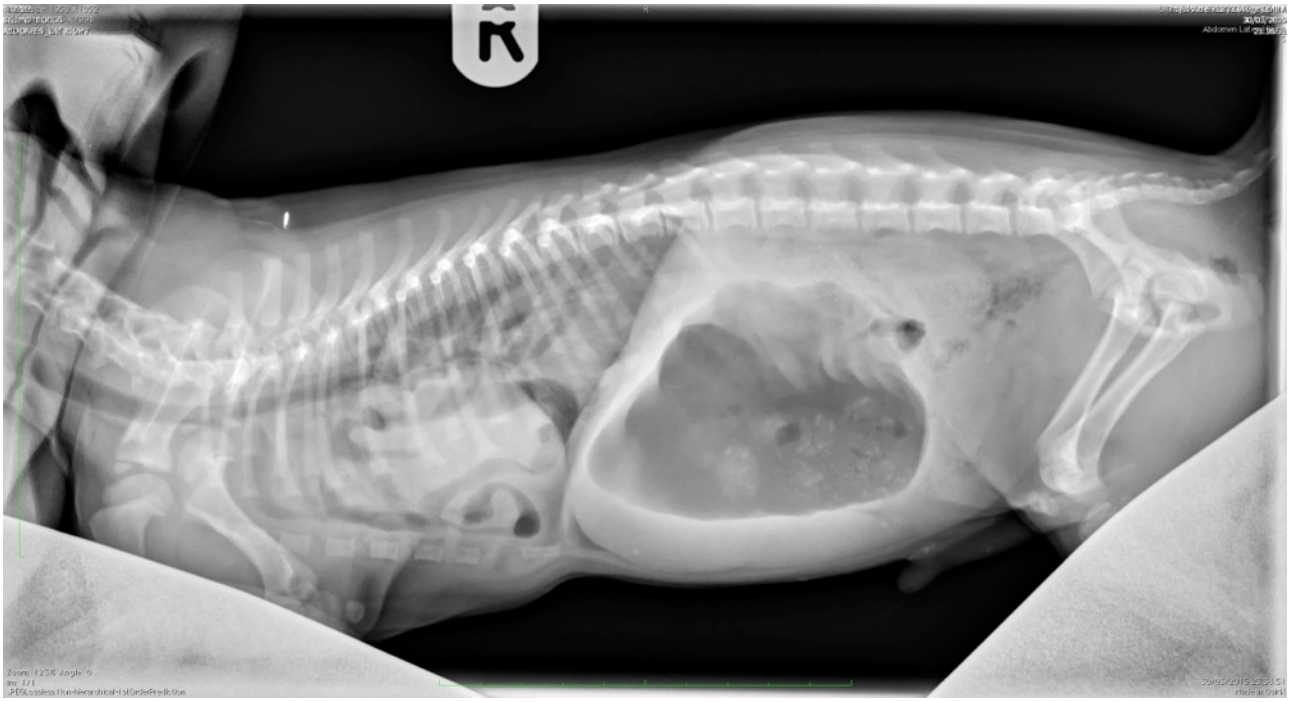
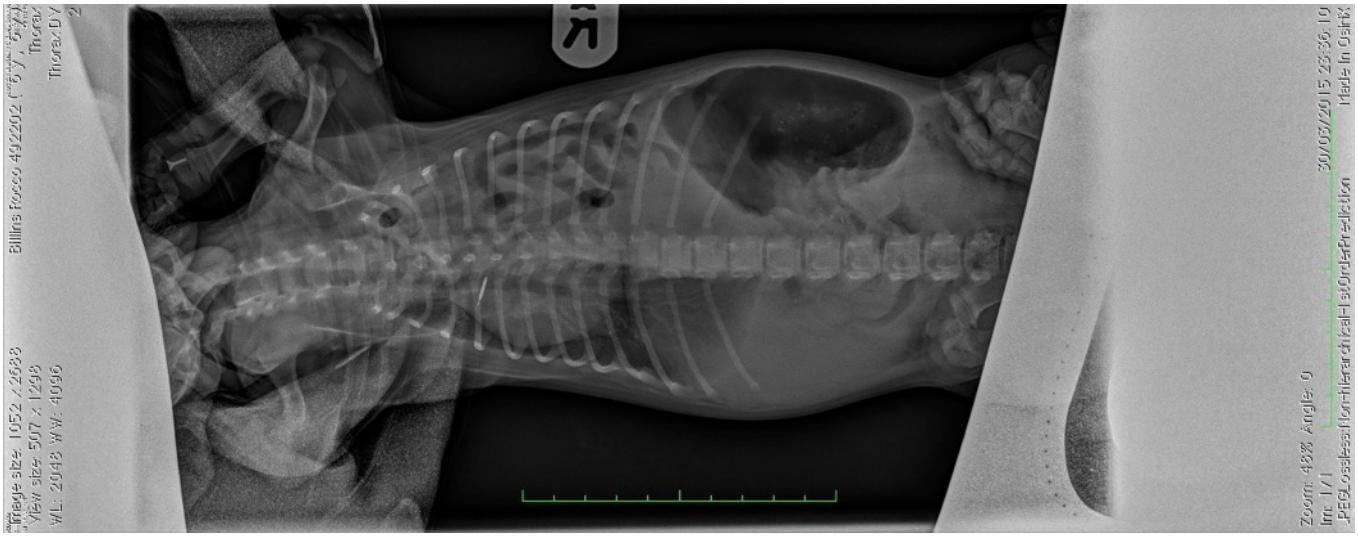
Annex I





Figures 1, 2. Left diaphragmatic rupture with small intestine filling the left hemi-thorax. The appearance of the stomach may suggest a more chronic process with chronic partial obstruction. Gastric displacement (90degree) and dilation.

Annex II

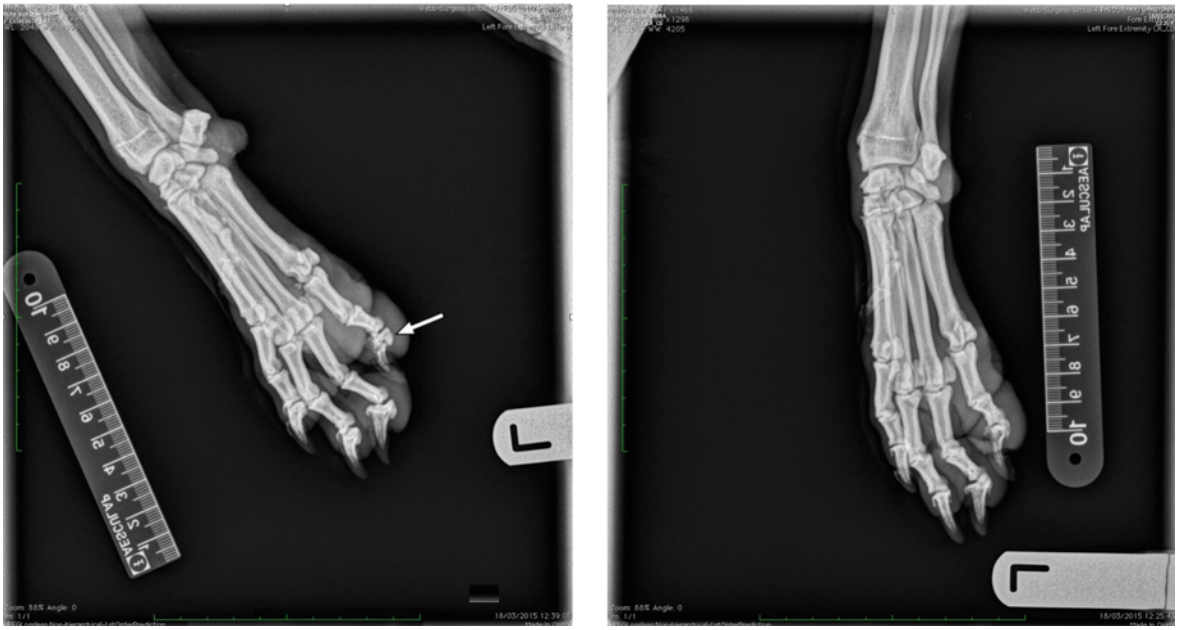


Figure 1, 2. The distal phalanx of the fifth digit has a moderately heterogenous and radiolucent appearance with mild diffuse osteolysis (arrow). There is moderate diffuse soft tissue swelling over the distal half of the digit (picture: QMHA).

Tetanus severity classification system developed for use in dogs.

Class	Clinical signs*
I	Any or all of the following: Miosis, enophthalmos, risus sardonicus, erect ears, or trismus Hypersensitivity to noise, light, or touch Ambulatory Absence of any class II, III, or IV signs
II	May include any or all class I signs Any or all of the following: Dysphagia Stiff gait, sawhorse stance, or erect tail Ambulatory Absence of any class III or IV signs
III	Must have class I or class II signs (requirement) Any or all of the following: Recumbency Muscle fasciculations or spasms Seizures Absence of any class IV signs
IV	Must have class I, II, or III signs (requirement) Any or all of the following: Bradycardia (heart rate ≤ 60 beats/min) or bradyarrhythmia Sinus tachycardia (heart rate ≥ 140 beats/min) or tachyarrhythmia Labile hypertension (mean arterial blood pressure ≥ 130 mm Hg or systolic arterial blood pressure ≥ 150 mm Hg) Labile hypotension (mean arterial blood pressure ≤ 60 mm Hg or systolic arterial blood pressure ≤ 80 mm Hg) Periods of apnea or respiratory arrest

*Note that dogs graded as class II need not have class I signs, but dogs in class III must have class I or II signs. Dogs graded as class IV must have class I, II, or III signs.

Table 1. Tetanus severity classification system similar to the tetanus severity classification system most commonly used in humans.

Annex III

Figure 1. Rosie's Epidural block with preservative-free morphine. In the picture palpation of the landmarks and needle insertion in the lumbosacral space is shown.

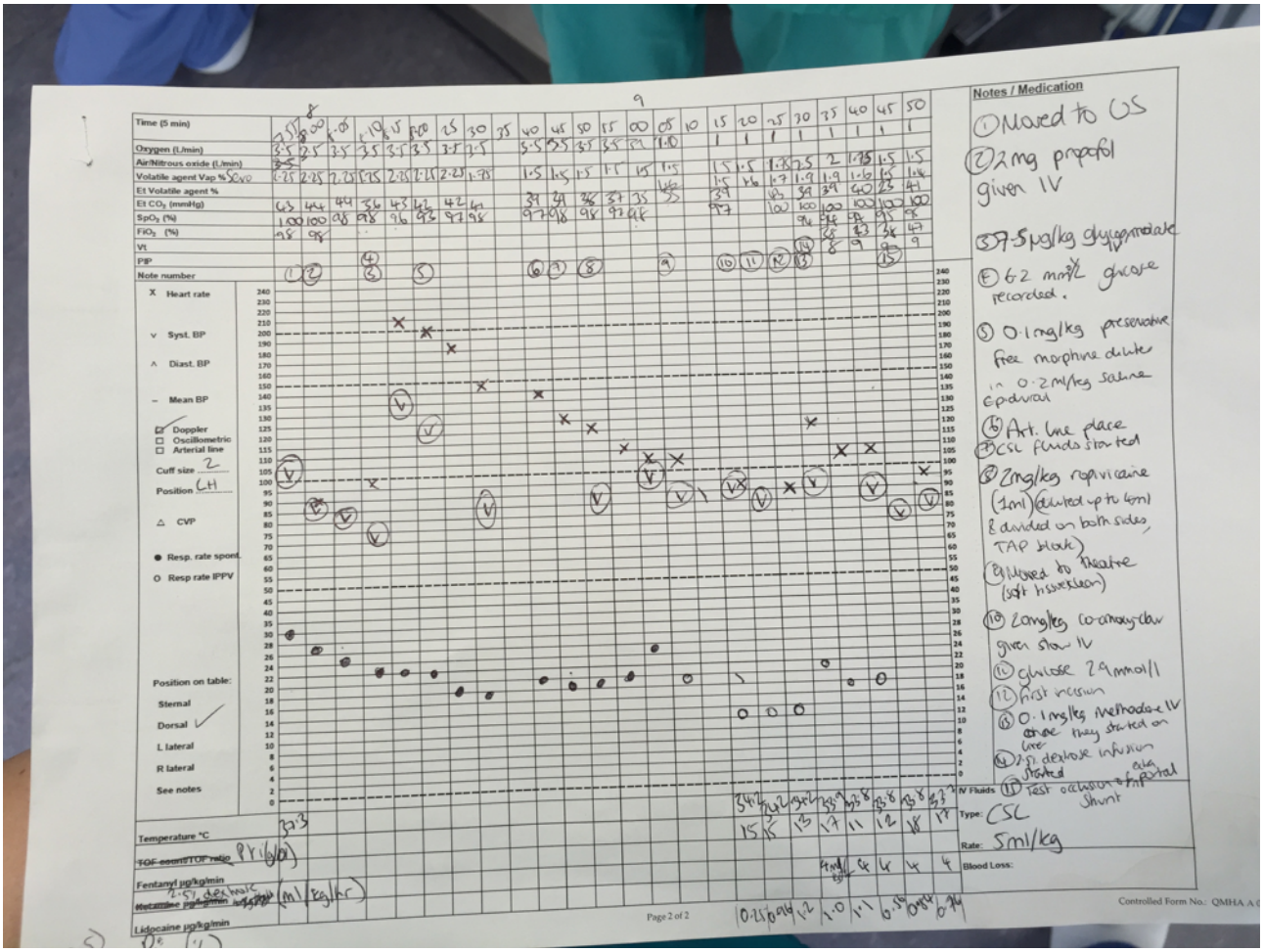


Figure 2. Anaesthetic monitoring sheet 1/2.

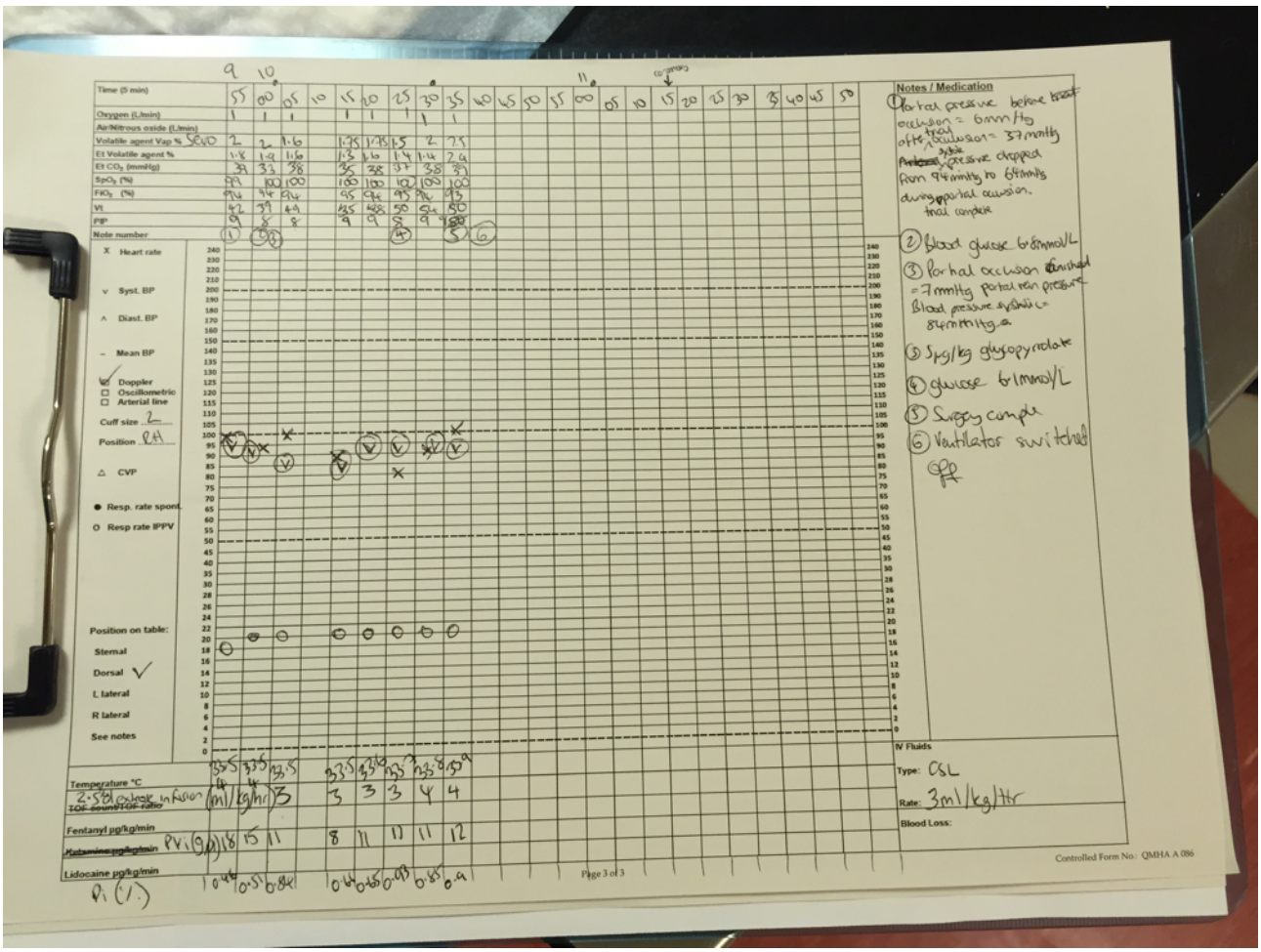


Figure 3. Anaesthetic monitoring sheet 2/2.

Annex IV

Figure 1. Bertie's eyes show bilateral lens opacification.

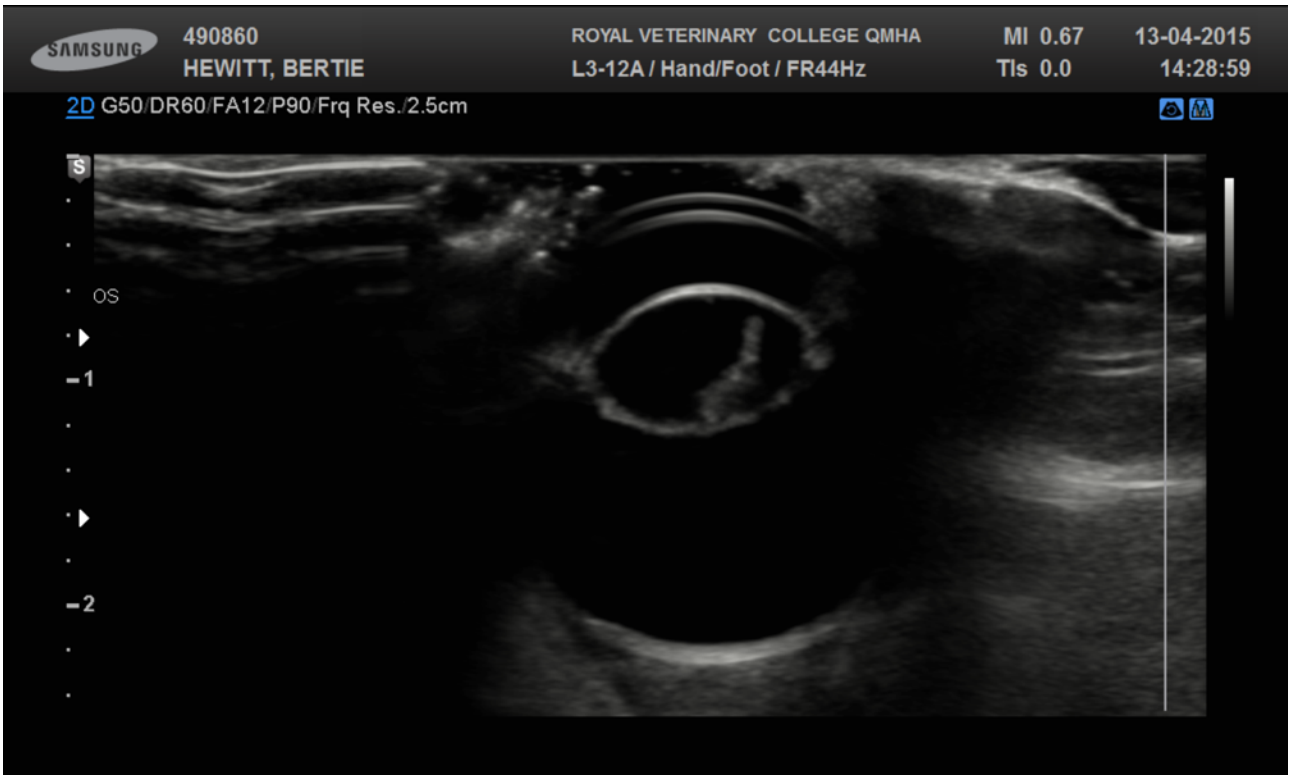


Figure 2. Left eye: increased relatively sharp and slightly echogenicity within the lateral aspect of the lens. The posterior part of the capsule of the lens is slightly irregular. No evidence of vitreal or retinal abnormalities.

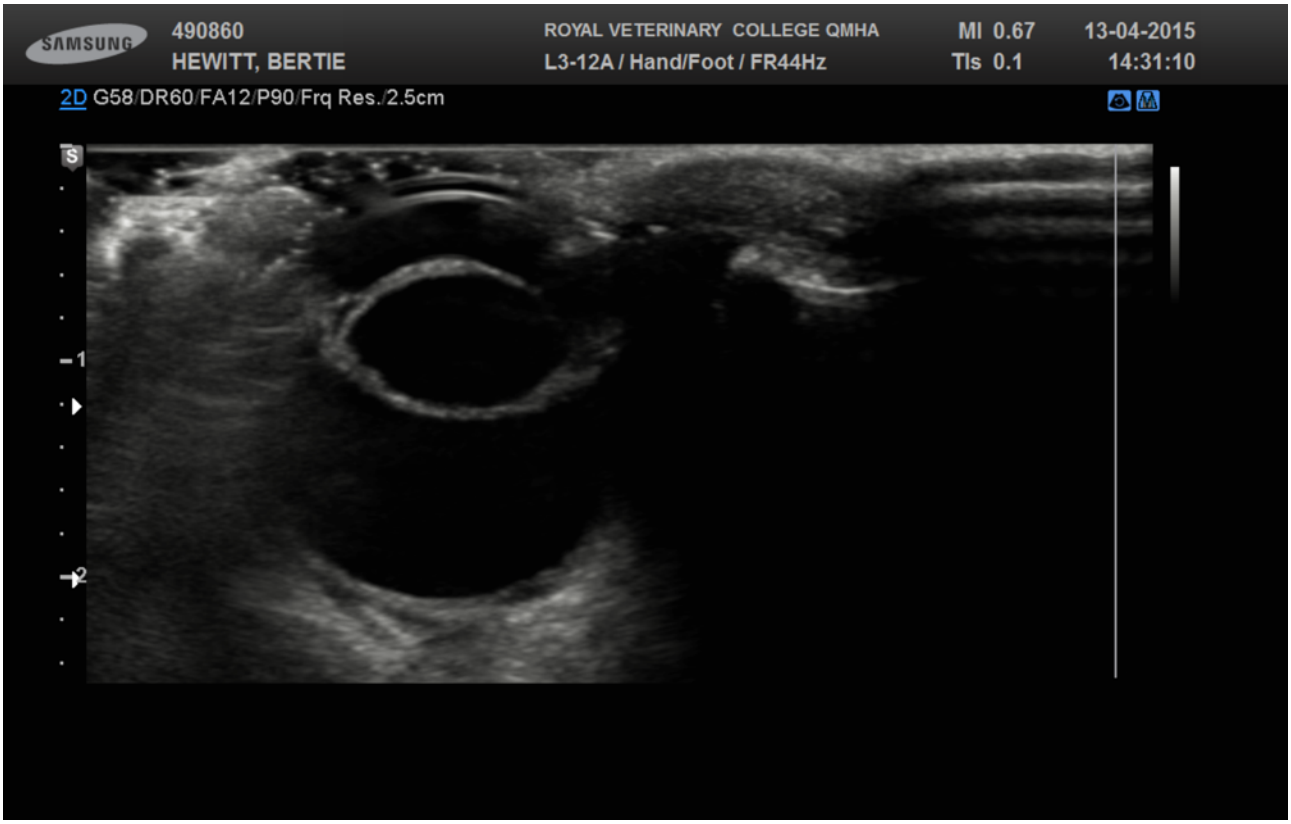


Figure 3. Right eye: slightly increased peripheral echogenicity of the lens, mostly at the its posterior aspect. No evidence of vitreal or retinal abnormalities.

Annex V

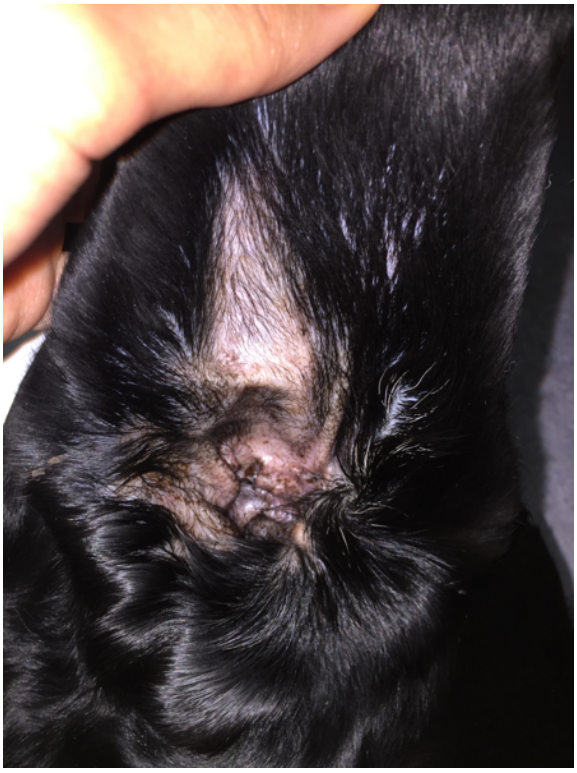


Figure 1. Sefton's ear showing brown discharge, redness and hyperpigmentation.

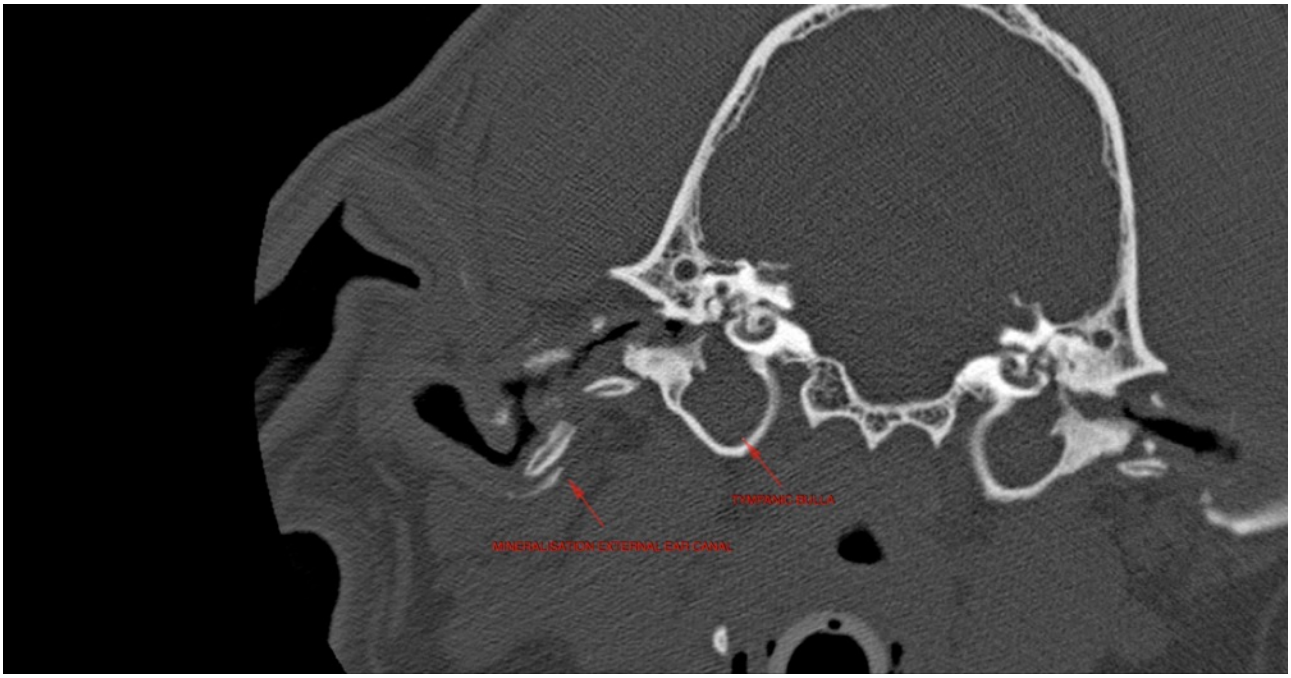


Figure 2. CT Scan: Bilateral filling of tympanic bulla and mineralization of the ear canal.