New Service coming to Willows

Radioactive Iodine treatment for hyperthyroidism in cats.
Gold Standard treatment for hyperthyroidism in cats.

Willows is delighted to announce that we will be launching a new service for 2018! We have been updating you on our exciting new building extension which has been an ongoing project over the past months, and with this we will be opening a dedicated facility for the treatment of feline hyperthyroidism, leading the way for a complete solution for your hyperthyroid patients. We are now taking bookings for January 2018.

The service will be led by our Feline Medicine Specialist Stephanie Lalor, BVetMed(Hons) MANZCVSc DipECVIM-CA MRCVS, RCVS Specialist in Small Animal Medicine (Internal Medicine), European Specialist in Small Animal Internal Medicine, RCVS Specialist in Feline Medicine and lead radioactive iodine nurse, Vicky Maude.

Radioactive iodine therapy can be considered the gold standard therapy with a reported success rate of 95%. It is also ideal for patients that are unsuitable for medical treatment or surgery. Most cats are no longer hyperthyroid after one treatment of radioactive iodine. The radioisotope 131I is injected subcutaneously, after which the overacting thyroid tissue takes up most of the 131I as it prepares to make thyroid hormones. Here the 131I emits beta particles, which will destroy the abnormal thyroid tissue while sparing all other organs. The radioactivity is very local and there is no hair loss or other side effects that are associated with radiotherapy. It is well documented that 20% to 25% of hyperthyroid cats have multiple areas of hyperfunctional thyroid tissue, which is destroyed with radioiodine treatment regardless of location. All treatment regimes for hyperthyroidism can potentially unmask renal disease; therefore, it is important to monitor this closely.

Radioactive iodine therapy is safe and effective but does require hospitalisation of the patient for 2 weeks post treatment. The procedure does not necessarily require a general anaesthetic or sedation. Blood samples checking biochemistry parameters, total thyroxine (TT4) concentration, and urinalysis should be carried out before any treatment is administered to check for any underlying medical conditions.
7 year old, male entire German Shepherd Dog

Bob was presented for evaluation of PUPD, lethargy and a reduced appetite. The referring vet’s blood tests had showed azotaemia and probable hypercalcaemia. An ACTH stimulation test had been performed and excluded hypoadrenocorticism.

On referral, examination revealed some generalised muscle atrophy only. Ionised calcium measurement confirmed severe hypercalcaemia (1.96; ref: 1.12-1.40 mmol/l) and azotaemia (creatinine 175 umol/l).

A thoraco-abdominal CT scan was performed – selected images are shown on the right (pictures 1 & 2). The rest of the haematology and biochemistry was unremarkable.

What is your diagnosis or differential diagnosis? What are the treatment options for this condition? What is the prognosis?

...for the answer see the back page

RSA Insurance Group
Important Update

Willows now subsidises RSA Preferred Network Fee

In order to fully support our clients and our ongoing commitment to excellence in clinical care, Willows will now cover the cost of the £200 fee that may be charged to our clients by Royal Sun Alliance (RSA).

As practices are aware, if the case is an emergency or there is no relevant specialist on the ‘Preferred Provider List’ close to the client, the £200 payment should be waived by the RSA.

Willows is still firmly committed to their views regarding the RSA ‘Preferred Referral Network Scheme’, and remain as one of many centres providing excellent quality referral services which are not part of this scheme.
Total hip replacements are very well established at Willows. Our Specialists have built up a huge wealth of experience and expertise over the years. Willows’ orthopaedic Specialists have pioneered new techniques for performing hip replacements in small animals, and these techniques have been shown to give lower complication rates and excellent patient outcomes. Our orthopaedic surgeons are supported by a truly multi-disciplinary team in our state-of-the-art hospital.

**Total hip replacement clinic**
- Orthopaedic team consisting of six board-certified Specialists, supported by Specialists in anaesthesia and imaging
- One of the most experienced teams in the world for canine and feline total hip replacement
- Proven low complication rates and outstanding clinical results
- Comprehensive investigation of patients using state-of-the-art diagnostic systems
- Hospitalised patients benefit from 24 hour veterinary and nursing care
- Free telephone advice given with the minimum of delay
- Urgent and emergency cases seen as a priority
- Rapid reporting on all cases seen

We are pleased to see canine and feline patients for assessment and treatment of hip problems
- Small and large patients, from the smallest toy breed to the largest St Bernard
- Younger, middle aged and older patients, from puppies and kittens to elderly animals
- Hip dysplasia
- Legg Perthes disease
- Irreparable femoral head fractures
- Chronic hip luxation
- Severe osteoarthritis

We are able to optimise success rates and patient outcomes for all cases, thanks to our Specialists’ experience and expertise, state-of-art operating theatre facilities, and our postoperative recovery wards and intensive care unit which are staffed 24 hours a day, 365 days a year.

**References**


**CALL OUR TEAM ON 0121 712 7070 FOR FURTHER INFORMATION**
Bob, a young male Labrador, was presented to Willows earlier this year for further investigation of chronic mucopurulent nasal discharge. This had been present for the previous 3 to 4 months and was initially unilateral in nature before becoming bilateral. Bob had also had a single episode of epistaxis (left-sided) and was becoming progressively more lethargic. Various therapeutic trials had been prescribed (antibiotics, prednisolone, oclacitinib) without a favourable response.

On presentation to Willows, Bob was quiet but alert. His clinical examination was unremarkable besides the presence of depigmentation around his left nostril and evident mucopurulent nasal discharge. Some discomfort was perceived on palpation of the rostral nose.

The list of differential diagnoses for chronic nasal discharge is relatively short. In this case, the primary concerns were sinonasal aspergillosis, a nasal foreign body, a nasal tumour or lymphoplasmacytic rhinitis; aspergillosis was considered the most probable diagnosis based on signalment, the presence of depigmentation and nasal pain. The ability to investigate nasal diseases has been revolutionised with access to advanced imaging and consequently a CT scan of the nose was recommended.

The CT scan showed a destructive rhinitis in the left nasal cavity with mild extension into the right nasal cavity. The presence of significant turbinate destruction is strongly suggestive of sinonasal aspergillosis. The diagnosis can be confirmed by visualisation of fungal colonies (plaques) at rhinoscopy.

Sinonasal aspergillosis is a challenging disease to treat due to its invasive nature in a site that is quite difficult to access. Several treatment approaches (varying in invasiveness) have been described. These include rhinotomy/sinusotomy, sinus trephination, catheter implantation and endoscopic techniques. The current literature suggests that success rates do not differ significantly with the technique chosen but recovery time and invasiveness does differ. The authors feel that the likelihood of resolution is dependent on the completeness of debridement and prefer an endoscopic approach first-line. Bob was therefore taken to rhinoscopy in order to confirm the diagnosis and, if appropriate, to perform the first treatment.

A small flexible endoscope was used for direct rhinoscopy. Fungal plaques were identified bilaterally (see images). Thorough endoscopic debridement of all visible fungal colonies was performed. The endoscope was also advanced into the frontal sinus to remove further fungal material (see image). Following this, a foley catheter was placed into the nasopharynx to occlude the back of the nasal cavity and enilconazole instilled and left for a short period of time (an enilconazole soak). Bob was hospitalised overnight for monitoring (and to protect the owner’s carpets) before being discharged the next day. Already he was brighter and had a reduced nasal discharge.

Bob was returned to Willows for a follow-up examination 3 weeks later. By that time, the nasal discharge had resolved completely and Bob was much brighter, even behaving like a puppy again. In some cases, a repeat rhinoscopy will be performed at this stage to confirm that the fungal colonies have not returned. As the clinical response was complete in this case repeat endoscopy was not pursued. Bob has been clear of any nasal symptoms for the last 6 months but ongoing monitoring will continue; relapse is rarely observed.

As sinonasal aspergillosis is an invasive condition some cases will require more than one treatment in order to achieve resolution of the disease. Unfortunately, some dogs will be left with a degree of chronic rhinitis due to the turbinate damage and this may require other ongoing therapies.

The minimally invasive approach to sinonasal aspergillosis that is typically taken at Willows has excellent results with minimal recovery required and we therefore feel this is an excellent first-line option for these cases.
Pericardial effusion is a well described condition in which an abnormal accumulation of fluid builds up in the pericardial space. In certain cases this can become life threatening due to the increased intra-pericardial pressure leading to differing degrees of hemodynamic compromise (cardiac tamponade) (3). Pericardiocentesis is a relatively easy procedure that can often be performed in the conscious patient. However, some patients may require sedation (and in rare cases anaesthesia) and this can be challenging. In an emergency situation draining the effusion efficiently can greatly improve the condition of a patient with respiratory distress and/or cardiac tamponade and allow for a more thorough cardiac examination.

The most common causes of pericardial effusions are listed in table 1.

<table>
<thead>
<tr>
<th>DOG</th>
<th>CAT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic pericardial effusion</td>
<td>Feline Infectious Peritonitis</td>
</tr>
<tr>
<td>Cardiac neoplasia e.g hemangiosarcoma, chemodectoma, mesothelioma</td>
<td>Chronic Heart Failure</td>
</tr>
</tbody>
</table>

Table 1: Common causes of pericardial effusion in the dog and cat (2,3)

**CASE EXAMPLE**

An eight-year-old male entire springer spaniel presented to the cardiology service at Willows Referral Service for investigation and treatment of a pericardial effusion.

The patient had a short history of acute abdominal enlargement noticed by the owner 5 days prior to presentation. No other clinical signs were reported. Initial investigations with the referring veterinary surgeon had included an abdominal ultrasound which revealed ascites with hepatomegaly confirmed also via abdominal radiography. Two days post-investigations the patient developed bilateral jugular pulsing and a repeat ultrasound of the heart revealed a mild pericardial effusion. Thoracic radiographs were also taken (see figure 1) and the dog was referred for further investigations. No medications were prescribed prior to presentation.

**Table 2: Common clinical signs associated with acute and chronic cases presenting with pericardial effusion**

<table>
<thead>
<tr>
<th>ACUTE CASES</th>
<th>CHRONIC CASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia</td>
<td>History of ascites</td>
</tr>
<tr>
<td>Sudden onset exercise intolerance</td>
<td>Gradual exercise intolerance</td>
</tr>
<tr>
<td>Collapse</td>
<td>Lethargy</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>Gastrointestinal signs</td>
</tr>
<tr>
<td>Rapid death (if untreated)</td>
<td>Collapse</td>
</tr>
</tbody>
</table>

**Figure 1:** Right lateral thoracic radiograph showing slightly rounded cardiac silhouette

**Victoria Phillips**
BVetMed MRCVS
Anaesthesia Intern
On general examination the patient was bright, alert and responsive. Heart rate was 112-120 bpm with good quality pulses. No muffled heart sounds or murmur were detected on auscultation. Mucous membranes were pale pink and moist. Capillary refill time was < 2 seconds. Respiratory rate was 28 bpm with no increased effort. The patient was ambulatory with mild abdominal distention due to known ascites. Otherwise examination was unremarkable. Common clinical signs associated with pericardial effusion are listed in table 3.

A conscious echocardiogram was performed (see figure 2) and confirmed a mild pericardial effusion with right atrial tamponade. No significant structural abnormalities were found. A marked abdominal effusion was also present. A 20-gauge cephalic intravenous catheter was placed and the patient was started on crystalloid fluid therapy at a rate of 2 ml/kg/hr prior to the pericardiocentesis.

A therapeutic pericardiocentesis was performed. The patient received 0.3 mg/kg of butorphanol intravenously 10 mins prior to pericardiocentesis and was placed in left lateral recumbency. Oxygen was provided via a face mask at 4 L/min and a continuous electrocardiogram (ECG) and pulse oximeter were used for monitoring from the time of administration of the butorphanol. The right hemithorax was clipped and prepped appropriately. Lidocaine 1 mg/kg was administered subcutaneously and into the intercostal muscles at the site of needle insertion to provide local anaesthesia. It is important to ensure the pleura has been infiltrated with the local anaesthetic as needle penetration during the pericardiocentesis can cause significant discomfort (7). The clinical procedure will not be described here. The patient received a bolus of 5 ml/kg crystalloid fluids immediately before pericardial drainage. The patient reacted to initial stimulation and alfaxalone was then administered intravenously in small incremental doses and to effect providing a light sedation. A total of 8 mg (0.4 mg/kg) of alfaxalone was administered over 20 minutes. Equipment was readily available if induction of anaesthesia became necessary and all emergency drugs (atropine, adrenaline and lidocaine) were drawn up and labelled for administration if necessary.

Two hundred and twenty milliliters of serosanguinous fluid was drained and a sample sent for analysis. The patient recovered from sedation in the intensive care unit with continuous ECG monitoring and vital signs were recorded for the first few hours following the procedure. No additional drugs were administered. Repeat ultrasonography later the same day showed no recurrence of the effusion and no complications were encountered in this case following the procedure. Complications can occur and are listed in table 5.

The owner was contacted one week post pericardiocentesis and reported the patient to be doing well. Abdominal ascites had resolved without further intervention and there was no recurrence of the pericardial effusion or associated clinical signs. Considering the findings and the initial outcome at this stage the diagnosis would be idiopathic pericardial effusion.

Table 3: Common findings on physical examination (3) in a patient with pericardial effusion

<table>
<thead>
<tr>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Becks Triad: jugular vein distention (increased CVP), muffled heart sounds (reduced intensity of apex beat), systemic hypotension (reduced pulse pressure)</td>
</tr>
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</table>

Table 4: diagnostic findings consistent with pericardial effusion

<table>
<thead>
<tr>
<th>Diagnostic Findings</th>
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<tr>
<td>Echocardiogram (Gold standard) Anechoic or hypoechoic space between the epicardium and the pericardium, mass lesions</td>
</tr>
</tbody>
</table>

Table 5: Complications associated with pericardiocentesis (3)

| Complications | Cardiac Puncture Arrhythmias Atrial fibrillation Myocardial stunning Dissemination of infection or neoplasia |

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Pericardiocentesis is well described in the literature and a relatively common procedure to perform in practice providing immediate relief and improvement of cardiac output\(^1\). It is the treatment of choice to stabilise patients presenting with pericardial effusion and cardiac tamponade\(^2,^3\). Anaesthetic considerations prior to pericardiocentesis such as drug combinations, fluid therapy and monitoring, will most likely depend on the initial presentation of the patient.

If sedation or general anaesthesia is required it is beneficial in these patients to use a multimodal approach\(^4\). Collapsed patients presenting acutely may only require infiltration of local anaesthetic at the site of needle insertion to allow pericardiocentesis to be performed but many dyspnoeic patients will require additional drug therapy, especially those with severe respiratory distress.

Patients in which only local anaesthesia is not sufficient, that are dyspneic or anxious, will require light sedation to prevent any sudden movements during the procedure. Stress will also increase the level of circulating catecholamine’s which in a severely cardiovascularily comprised patient could be fatal. Sedation may also be beneficial to the less experienced practitioner to ensure the animal is as still as possible for echocardiography or finally sedation may be necessary for intravenous catheter placement if the patient will not co-operate.

If sedation is required, drug combinations will depend largely on the individual patient. Cardiac tamponade results in a decrease in venous return, stroke volume, cardiac output and ventricular filling. As intrapericardial pressure increases the right side of the heart collapses preventing blood ejection into the pulmonary artery. The body attempts to compensate for this by increasing heart rate and peripheral vascular resistance. Therefore, it is advised to avoid drugs which may cause myocardial depression, bradycardia (such as medetomidine) or vasodilatation (such as acepromazine)\(^4\). The aim of sedation is to allow pericardiocentesis to be performed efficiently whilst maintaining these compensatory mechanisms\(^5\).

A commonly used drug combination in a patient requiring sedation would be a benzodiazepine, such as midazolam 0.2 mg/kg IM or IV, and an opioid such as butorphanol 0.2-0.3 mg/kg IM or IV (1, 4). In more debilitated patients just the administration of an opioid maybe sufficient. In non-surgical patients with cardiac tamponade, butorphanol is the opioid of choice as with stroke volume being fixed, cardiac output is dependent on heart rate and butorphanol is less likely to decrease the heart rate compared to other opioids such as methadone\(^4\). Benzodiazepines have the potential to cause disinhibition and induce excitement rather than sedation and this should be kept in mind when administering this class of drugs to relatively healthy patients. An alternative, as in the case example above, would be butorphanol 0.2-0.3 mg/kg IV 10-15 mins prior to the procedure and alfalfalone 1-2 mg/kg IV titrated to effect as required. Alfalfalone can be titrated to effect to provide additional sedation as it has minimal cardiovascular effects used at low doses\(^6\). It is important in all patients to provide oxygen therapy using flow-by or ideally a mask if tolerated\(^7\).

Intravenous fluid therapy is recommended prior to sedation or induction of anaesthesia to optimise preload and maintain cardiac filling pressures. Small boluses (2-5 ml/kg) before draining the pericardium is recommended\(^8\). Continuous ECG monitoring before, during and following the procedure for up to 24 hours is paramount\(^2,^7\). Electrical alternans (figure 3) and elevation of the ST segment is commonly seen in pericardial disease\(^7\). Contact of the catheter or needle with the heart can induce supra-ventricular or ventricular ectopy (figure 4) which may proceed further into ventricular tachycardia or even fibrillation. The presence of arrhythmias may necessitate withdrawal of the catheter completely and/or anti-arrhythmic therapy. Lidocaine (without adrenaline) should be available and pre-drawn into syringes in boluses of 2-3 mg/kg to administer in an emergency. Following drainage ECG complexes should return to normal size, heart rate should reduce if the patient has been tachycardic and pulse quality should improve\(^7\).
We are delighted to announce the arrival of four new members to the Willows’ team

Brian Watson  
Hospital Director  
Brian joined Willows in May 2017 as Hospital Director. Having worked in senior management roles within the veterinary industry over the past 10 years, Willows was an easy choice for the next step on his career journey.

Chiara Penzo  
DVM PhD Dip.ECVIM-CA (Oncology) MRCVS  
RCVS Specialist & European Specialist in Veterinary Oncology  
Chiara graduated with honours and achieved a PhD from University of Padua (Italy). She completed a one year small animal internship at the University of Glasgow and a three year residency in internal medicine and oncology at The University of Edinburgh.

Alexis Bilmont  
DVM DipECVS MRCVS  
European Specialist in Small Animal Surgery  
Alexis graduated from the Veterinary School of Maisons-Alfort (France) in 2006. He became a Diplomate of the European College of Veterinary Surgeons in 2017. Alexis’ clinical interests cover all aspects of orthopaedics and spinal surgery.

Julia Riggs  
MA VetMB FHEA MRCVS  
Clinician in Small Animal Surgery  
Julia graduated from the University of Cambridge in 2011, and completed her small animal surgery residency in 2017. Julia is interested in all aspects of small animal surgery, particularly surgical oncology, wound management/reconstructive surgery, and upper airway surgery.

WHAT WAS YOUR DIAGNOSIS?

7 year old, male entire German Shepherd Dog

The CT scan revealed multiple ‘punched out’ areas of osteolysis throughout the skeleton including the ribs and the vertebral bodies. There were some soft tissue masses associated with some of the rib lesions. A large unilateral renal mass was also seen though was considered extremely difficult to aspirate (ultrasound-guided fine needle aspirate) to achieve a diagnosis, though most renal tumours are malignant carcinomas.

Diffuse osteolysis is often a classical finding in dogs with multiple myeloma (MM), though these patients usually have severe elevation of globulin levels which is one of the additional hallmarks of the disease. It was therefore considered most likely that there was a primary renal carcinoma with diffuse metastasis to the skeleton, resulting in hypercalcaemia due to osteolysis +/- humoral factors elicited by the tumour cells. Dogs with MM also typically have haematological abnormalities due to overcrowding of the bone marrow by malignant plasma cells – such as anaemia, neutropenia or thrombocytopenia.

For confirmation of the suspected diagnosis, FNA of one of the rib lesions was performed which revealed a monomorphic population of malignant plasma cells confirming MM. As the serum globulin levels were normal, and serum protein electrophoresis failed to detect a monoclonal gammopathy, this case of MM was considered to be a rare “non-secretory” case, whereby the malignant plasma cell do not secrete excessive quantities of globulin.

Whilst MM is rarely curable and most dogs will eventually succumb to the disease, this was in fact good news for Bob considering the alternative (advanced and metastatic solid tumours such as carcinomas often have a poor outlook with chemotherapy). MM typically responds well to minimally invasive chemotherapy which is usually given orally. Median survival times of around 18 months are typically reported.

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BVM&S CertSAM DipECVIM-CA (Oncology) MRCVS  
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Committed to excellence