Welcome (cont.)

Continued from front page

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Mobile Expertise in Equine Diagnostics

We are committed to providing a high quality hospital-based clinical teaching practice and the technology we have in the hospital, along with the facilities and support staff are among the very best in this country and Europe. Hospitalisation is often crucial to allow the best care and most effective evaluation. We do however understand that there are times when your clients are unable to travel or it is not possible to bring horses into the hospital. With improvements in mobile diagnostic units we are sometimes happy to provide expertise ‘on the road’. Your clients should be aware that the cost may well be greater than a visit to the hospital, as it incurs costs related to travel. If you have a specific request to use us in this way, please contact the relevant clinical service. Surgery is not currently included in this approach as we strongly feel that, due to the nature of the surgical cases we are asked to evaluate, the best service to your client is provided when the surgery is carried out by a whole team of specialists (anaesthetists, surgical nurses, medical backup) who perform their relative duties in a facility designed for that purpose. It also means that we can achieve close post-operative monitoring in cases of predicted and unpredictable complications; as you know surgery can be unpredictable even with the best care. This mobile service is on an ad hoc basis, when other staff commitments allow, and in consultation with the senior clinicians only.

Our Clinicians

Medical

Professor Bruce McGorum
BSc, BVMS, Cert EM, DipECISM, MRCVS
Dr Scott Priece
BVMS, PhD, Cert EM, Cert EP, DipECISM, MRCVS
Dr John Keen
BvetMed, PhD, Cert EM, DipECISM, MRCVS
Dr Karen Blissitt
BVSc, PhD, DVA, DipECVA, MRCVS
Surgery

Professor Paddy Dixon
MVB, PhD, MRCVS
Dr Sarah E. Taylor
BVMS, PhD, Cert ES (Orth), DipECVS, MRCVS
Mr Eugene Cillan-Garcia
DVM, MRCVS
Mr Richard Readon
BvetMed (Hons), MVM, Cert ES (Orth), DipECISM, MRCVS
Neurology

Dr Caroline Hahn
DVM, MSc, PhD, DipECISM, DipECVN, MRCVS
Our Residents

Lucinda Meehan
BVSc, MRCVS
Justine Kane-Smyth
BVMS, MRCVS
Rachel Jago
BVMS, MRCVS
Tim Froydenlund
MA, BVM&S, MRCVS
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Welcome to our Spring Newsletter

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Three new residents have started this year, as three others have finished their specialist training. Tim Barnett left to take up a job with Rossdale’s Equine in Newmarket, John O’Leary left us for his Irish homeland, although does pop back to provide us with occasional locum help and Claire Stratford headed down south back home. They have all been a fantastic help in the hospital throughout the last
Research Update: Steroids and Laminitis

Ruth Morgan, under the supervision of Dr John Keen, is undertaking a 4 year BBSRC and Zoetis funded PhD evaluating the role of corticosteroids in metabolic syndrome. Cushings’ Disease and laminitis. This study is in collaboration with researchers at the University of Edinburgh Medical School who evaluate similar vascular diseases in humans. Preliminary results have proved very interesting and include the following:

- Steroid levels appear to be excessively high in fat tissues of horses that have the equine metabolic syndrome and that are predisposed to laminitis. This is similar to the situation in humans where many believe that systemic and/or local steroid levels may ‘drive’ the human metabolic syndrome and the predisposition toward cardiovascular diseases such as strokes and myocardial infarction.

- Equine blood vessels appear to behave differently from mouse and human blood vessels when bathed in steroids: while studies have shown that mouse or human blood vessels fail to grow in the presence of steroids, Ruth has found that equine vessels proliferate massively. This raises interesting questions about our assumptions of how steroids may affect blood vessels in the horses foot, but also the effects of steroids in wound healing in horses (we had assumed that steroids stopped blood vessel growth so contributing to delayed healing), how steroids regulate ovulation in the horse etc. This research is on-going for the next 3 years and some aspects are dependent on gaining access to laminitis clinical case material. If you know of horses or ponies destined for euthanasia as a result of repeated laminitis that are clinically stable and able to travel then John or Ruth would be very keen to hear from you. Please contact john.keen@ed.ac.uk in the first instance.

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Equine Scintigraphy

We are pleased to announce that our new Equine Scintigraphy Unit is installed and fully functioning!!

Our new, state of the art, purpose installed and fully functioning!!

Our new Equine Scintigraphy Unit is the first to be installed in Scotland. The improved images together with decreased scanning times and reduced stress for our patients combine to make scintigraphy an incredibly useful imaging modality for equine lameness diagnostics.

Case Study – “Coming up against a brick wall”

‘Leo’, a 9 year old Warmblood gelding was referred in September 2012, with a history of earlier in the day colliding with and demolishing a breeze block wall. With the exception of moderate epistaxis and occasional skin abrasions, Leo appeared relatively unchanged. Fortunately, and somewhat miraculously, Leo’s rider sustained no injuries at all. Despite Leo’s initial appearance, he was found recumbent in the field later that day and, when encouraged to rise, demonstrated an ataxic gait. Examination revealed mucous membrane pallor and a significant tachycardia (104 beats/minute) which prompted immediate referral.

On arrival at the hospital, Leo was quiet and responsive. Clinical examination confirmed the mucous membrane pallor and revealed a tachycardia (105 beats/minute) and tachypnoea (20 breaths/minute). Although he did not appear ataxic upon arrival, his gait was slow and laboured. A tentative diagnosis of haemorrhage into a 3rd space was made, with resultant acute hypovolaemia. Despite a relatively normal packed cell volume (0.36L/L), a raised blood lactate concentration (4.6 mmol/L) supported the presence of significant hypovolaemia. Circulatory support was instigated in the form of intravenous colloid administered during which attempts were made to identify the site of haemorrhage. Ultrasound examination of the abdominal cavity was unremarkable; however, thoracic ultrasound revealed a significant volume of fluid ventrally within both the left and right hemithorax. The ultrasonographic appearance of the fluid was highly consistent with haemothorax. The volume of haemothorax appeared identical on both sides of the thorax, reflecting communication between the two sides via a fenestrated pleural membrane. No rib fractures were detectable ultrasonographically.

Leo was hospitalised overnight on intravenous fluid therapy, prophylactic antibiosis and analgesia. The circulatory volume replacement resulted in a reduction in heart rate to 80 beats/minute overnight and his PCV gradually reduced to 0.18L/L, due to haemodilution resulting initially from fluid shifts from the intravascular space into the extravascular and latterly from intravenous fluid administration. In light of the fact that the PCV stabilised at 0.18L/L and the heart rate continued to gradually reduce, no blood transfusion was performed. Following a further 24h, intravenous colloidal support was ceased and Leo resumed oral fluid and feed intake. Daily ultrasonographic examination of the thorax showed a gradual reduction in fluid accumulation on both sides of the chest; however, a small left sided pneumothorax was noted, consistent with lung parenchymal damage. Under such circumstances, the unilateral nature of the pneumothorax was expected due to accumulation of free air within the pleural space in a dorsal position and therefore distant from the communication between the left and right sides.

Continued monitoring of Leo’s clinical parameters revealed a return of both respiratory and heart rate to within normal limits. His PCV gradually increased to 24% by day 8 of hospitalisation, a rate of increase consistent with a degree of autotransfusion. He was discharged following 2 weeks of hospitalisation with instructions for box and paddock rest for the next 6 weeks.

This case highlights a few interesting areas of consideration: namely, (a) the traumatic incident itself is testimony to the power of the horse and was a key part of the history, (b) the clinical presentation of tachycardia with associated mucous membrane pallor was highly consistent with haemorrhage, the degree of which was not consistent with the epistaxis noted immediately following the event; (c) the absence of severe respiratory compromise caused by pleural fluid was not unexpected in this scenario, considering that the volume of blood necessary to cause a significant interference with lung expansion would exceed the volume of acute loss that was compatible with life; (d) in acute blood loss, PCV remains within or near normal limits until sufficient time has elapsed for fluid shifts to occur and result in haemodilution; therefore the blood lactate and heart rate were more accurate indicators of the degree of blood loss; (e) under clinical situations consistent with acute blood loss, in the absence of overt external haemorrhage, there are a limited number of sites which require evaluation and ultrasonography.
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| BVSc, PhD, DVA, DipECVAA, MRCVS |

| Neurology | Dr Caroline Hahn  
| DVM, MSc, PhD, DipECIEM, DipECV, MRCVS |

| Our Residents | Lucinda Meehan  
| BVSc, MRCVS | Justine Kane-Smyth  
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