



Hyperinsulinaemia

Chaired by Andy Durham

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09.00–09.25

The role of hyperinsulinaemia in the development of laminitis

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Laminitis usually occurs as a consequence of 3 broad categories of disorders: systemic inflammation, endocrine disturbances or trauma. By far the majority of research on laminitis has been associated with inflammatory models of laminitis including the carbohydrate overload with starch or oligofructose models and the inflammatory (Black Walnut) model.

Endocrinopathic laminitis has been defined as laminitis developing from hormonal influences and not pro-inflammatory or intestinal conditions (Johnson *et al.* 2004). Conditions associated with endocrinopathic laminitis include Equine Metabolic Syndrome (EMS), pasture-associated laminitis or prelaminitic equine metabolic syndrome, Equine Cushing's Syndrome (ECS) also called pituitary pars intermedia dysfunction (PPID) and iatrogenic corticosteroid administration. Common to all are disturbed glucose and insulin regulation resulting in the development of insulin resistance and hyperinsulinaemia which has been shown both in field studies (McGowan *et al.* 2004; Treiber *et al.* 2006) and in experimental studies (Asplin *et al.* 2007; de Laat *et al.* 2010).

A recent shift in opinion about laminitis has been the realisation that endocrinopathic is by far the most common form of laminitis. This has prompted increasing interest in endocrine events resulting in laminitis. Horses that suffer endocrinopathic laminitis differ from those with systemic inflammation in that they are not systemically unwell, are not pyrexia and do not have alterations in gastrointestinal function. Insulin has been shown to be a final triggering event in causing endocrinopathic laminitis in both ponies and horses. Laminitis could be induced in 100% of ponies or horses exposed to high concentrations of insulin (approximately 1000 μ iu/ml) while maintaining normal blood glucose concentrations within normal reference ranges using a modified euglycaemic-hyperinsulinaemic clamp technique (Asplin *et al.* 2007; de Laat *et al.* 2010). All ponies and horses were healthy, young and nonobese, with no history of laminitis and no evidence of endocrine or other abnormalities on blood tests.

These studies have shown the crucial role insulin plays as an important trigger of endocrinopathic laminitis. Of importance is that the induction of laminitis occurred independently of glucose or (direct) dietary factors, and also without any evidence of gastrointestinal disturbance. Horses and ponies in the study had routine blood tests performed both before and at the onset of laminitis and no changes were found, nor were there any clinical signs indicative of system illness or inflammation.

Insulin levels reached in these experiments were higher than typically seen in grazing horses with prelaminitic metabolic syndrome (Treiber *et al.* 2006). However, insulin values over 1000 μ iu/ml have been reported in naturally occurring cases of severe EMS (Reeves *et al.* 2001; McGowan and Riley 2004). Whether the effect is the same for longer term exposure to lower levels of insulin or not remains to be determined with further study.

Interestingly, despite the lack of systemic or gastrointestinal clinical illness, endocrinopathic laminitis may also involve some of the same mechanisms as those occurring in inflammatory laminitis. However, some important differences are emerging which have prompted a re-evaluation of what we had previously thought applied to all cases of laminitis.

1. Histological changes were milder and leucocyte emigration was significantly less in horses with insulin-induced laminitis than horses developing laminitis secondary to carbohydrate overload (de Laat *et al.* 2011a). Insulin-induced lesions at Obel grade 2 lacked widespread basement membrane degradation, especially in ponies. Instead there were cellular changes and stretching/elongation of secondary (but not primary) epidermal lamellae (Asplin *et al.* 2010).
2. Supporting the lack of widespread basement membrane degradation was the finding that matrix metalloproteinases (MMP-2, MT1-MMP, TIMP-3 and ADAMTS-4) were not upregulated in the insulin-induced model and that increases in MMP9 only were detected after 48 h corresponding to the neutrophil infiltration of the lamellae (i.e. a later event) (de Laat *et al.* 2011b).

For practitioners, the information from research into endocrinopathic causes of laminitis has direct implications on laminitis management. In all cases of laminitis where evidence of systemic inflammation/gastrointestinal disease or trauma has not been found, horses should be suspected as endocrinopathic and appropriate endocrine testing carried out. For those horses <15 years of age and with no evidence of ECS, this will predominantly involve tests for basal insulin concentration and insulin resistance. Basal insulin values are often all that is required for native British breeds, but practitioners should be prepared to perform dynamic endocrine testing to support a diagnosis where basal testing is unhelpful. Where endocrinopathic laminitis has been confirmed, treatment should also include monitoring and treatment of the underlying endocrinopathy.

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NOTES



09.25–09.50

Managing weight loss in the hyperinsulinaemic pony

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Hyperinsulinaemia is a cardinal feature of insulin resistance. Obesity (BCS $\geq 7/9$), is associated with an inflammatory state and insulin resistance, although not all obese animals are insulin resistant and not all insulin resistant animals are obese, e.g. those with PPID. However 40–50% of the domestic horses and ponies in the UK are now considered overweight or obese.

The consequences of insulin resistance and hyperinsulinaemia include endocrinopathic laminitis and a predisposition to pasture-associated laminitis. While the exact mechanisms of pasture-associated laminitis remain to be determined, insulin resistant animals display exaggerated glycaemic and insulinaemic responses to dietary starches and sugars.

For overweight, insulin resistant animals, weight reduction is initially managed by dietary restriction, combined with physical exercise when possible.

Weight reduction programmes

- The owner/carer must be committed for the long-term and must be aware that there is no quick fix. A detailed history of the feeding protocol and work level should be obtained.
- Feed analysis (hay/haylage) enables more accurate calculations of future, regulated intakes.
- The readily available carbohydrate content of the feed also requires careful consideration.
- Monitor at least monthly: BM (weighbridge or weight tapes [accurate to ± 50 kg]), BCS, and girth (neck, trunk).

What to feed

- Removal from pasture is preferable to enable good control over food intake, although limited turn-out on graze-poor paddocks may be all that is available. (Beware soil/sand intake on muddy/sandy pastures.)
 - Use of grazing muzzles reduces food intake, but ensure animals can drink water and beware of herd hierarchy changes.
- Offer a forage (low energy density)-based diet. Avoid high starch/sugar diets which have a high glycaemic/insulinaemic index and promote hyperinsulinaemia.
 - Each portion must be weighed, not eyeballed.
 - No tit-bits (carrots, apples) allowed.
 - Hay can be soaked to try to remove some of the soluble carbohydrates, but the effect of soaking may depend upon the maturity and fibre length of the forage, in addition to the soak volume, temperature and time. Valuable minerals may also be leached out, so that mineral supplementation is recommended. (In order to avoid over-restriction of protein/essential amino acids, a balancer with protein, vitamins and minerals is usually recommended).
 - Caloric dilution by addition of straw (relatively indigestible fibre) remains controversial because of the risks of gastrointestinal impaction colics and stomach ulcers. Straw bedding and faeces, however, will likely be ingested by food-restricted animals, as will wood-shavings.
- Feed little and often (if practical) to reduce boredom and try to extend total eating time.
 - Use multiple, doubled, small-hole haynets at different positions in the stable.
 - Use manger obstacles to slow down food intake and allay boredom.

- Stable toys may help reduce boredom.

- Use winter as the natural aid to weight loss: turn out as much as possible (as long as pasture is sparse); don't rug up, consider trace-clipping; but beware sudden cold stress as this may precipitate laminitic episodes.

How much to feed

For weight reduction, most researchers have suggested cautious food restriction because of the potential complications for trickle-feeders, i.e. stereotypies and stomach ulcers. Up until recently, based on the evidence that most grazing horses will eat around 2–2.5% of their body mass (BM) as dry matter per day, restriction to around 1.3–1.5% of BM as dry matter intake (DMI) has been suggested.

A 'safe' rate of weight loss is often advocated as 1% of BM per week. Several equine studies targeting such a rate suggest that dietary restriction can safely be initiated at 1.25% BM as daily DMI, with further restriction to 1% BM as daily DMI if weight loss is negligible after the first month. Some animals will likely eat bedding or display coprophagia but adverse effects following these behaviours or the development of overt stereotypies should be rare. Hyperlipaemia should also be avoided.

Exercise

Exercise combined with dietary restriction will promote weight loss and improvements in insulin sensitivity but may be contraindicated in some horses/ponies due to laminitis. Reported effects of exercise on equine insulin sensitivity, however, are often confusing, possibly due to different levels of exercise, different body condition scores of animals recruited and different tests of insulin sensitivity being used and, in this author's opinion, particularly whether animals were starved before testing (which can itself increase insulin resistance).

Other strategies

For laminitic animals unable to exercise and/or where weight loss is very slow despite severe dietary restriction, then additional treatments may be considered: thyroid hormone supplementation or metformin.

Further reading

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09.50–10.15

Inter-relationships of hyperinsulinaemia and PPID

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Pituitary pars intermedia dysfunction (PPID, Equine Cushing's disease) and insulin resistance (IR) are the commonest endocrinopathies seen in equine practice and are both associated with increased risk of laminitis. The 2 conditions may be seen independently of one another or may coexist which may cause diagnostic and therapeutic confusion. Coexistence might be a pure coincidence given that they are both common conditions. Alternatively there might be a causal inter-relationship of the 2 conditions such that IR might predispose to PPID and/or *vice versa*.

It is recognised that PPID arises as a result of oxidative damage to hypothalamic dopaminergic neurons. Years of obesity and IR in earlier life might possibly create chronic inflammatory and oxidative challenge adequate to lead to neuronal damage and PPID. Furthermore, long-term management of obese, insulin resistant, laminitis-prone ponies might also inadvertently lead to deficiencies that could also promote disease, for example, lack of antioxidants present in fresh grass or vitamin D from exposure to sunlight resulting from restricted turnout. Conversely, it is also possible that PPID might be a cause of IR as a result of endocrine factors that might promote IR (e.g. cortisol, ACTH) and hyperinsulinaemia (e.g. corticotrophin-like intermediate lobe peptide).

Data from 744 cases of PPID seen between January 2009 and December 2010 were examined to look for associations between several variables including signalment data (age, breed, gender, time of year) and results of blood tests (ACTH, insulin, glucose, triglycerides) with the aim of providing information that might help clarify putative inter-relationships between PPID and IR. Cases of PPID were defined as a combination of a clinical suspicion of PPID and a plasma ACTH concentration greater than the reference interval for that time of year (Nov–Jul 29 pg/ml; Aug–Oct 47 pg/ml).

More PPID cases were seen in ponies than horses (61% vs. 39%). Comparison of ponies vs. horses revealed that PPID horses were significantly older and had lower serum insulin concentrations than PPID ponies. It was also found that several other associations examined (below) were different in horses and ponies and therefore these 2 populations were examined separately.

Comparison of laboratory data derived at different times of year was performed. In both PPID horses and PPID ponies it was found that plasma ACTH was significantly greater between the months of August and October than the rest of the year. It was also found that, in horses only, plasma glucose was significantly greater between August and October than during the rest of the year.

When data were compared between genders, the only significant finding was that plasma glucose was significantly higher in PPID pony geldings than in PPID pony mares. No significant gender effects were found in PPID horses.

Using a cutoff of 20 years of age, older PPID horses were found to have significantly higher plasma ACTH and lower plasma glucose than younger PPID horses. Older PPID ponies were found to have significantly higher plasma ACTH and lower serum insulin than younger PPID ponies.

Hyperglycaemia was found in similar numbers of PPID horses and PPID ponies (7% vs. 9%). Hyperglycaemic PPID horses had significantly higher plasma ACTH, serum insulin and serum triglycerides than normo-glycaemic PPID horses; whereas hyperglycaemic PPID ponies only differed from normo-glycaemic PPID ponies in having higher serum insulin.

Hyperinsulinaemia was more prevalent in PPID ponies than PPID horses (49% vs. 36%). Hyperinsulinaemic PPID horses had significantly higher plasma ACTH, plasma glucose and serum triglycerides than normo-insulinaemic PPID horses; whereas no significant differences were found between hyperinsulinaemic and normo-insulinaemic PPID ponies.

Hypertriglyceridaemia was more prevalent in PPID ponies than PPID horses (53% vs. 28%). Hypertriglyceridaemic PPID horses had significantly higher plasma ACTH and serum insulin than normo-triglyceridaemic PPID horses; whereas no significant differences were found between hypertriglyceridaemic and normo-triglyceridaemic PPID ponies.

The median ACTH concentration of all PPID cases was 65 pg/ml (all cases had plasma ACTH > seasonal reference interval). This value was then used as a cutoff to define 'high-ACTH' (>65 pg/ml) and 'low ACTH' (<65 pg/ml) PPID cases. PPID horses with high ACTH were significantly older and had higher serum insulin and plasma glucose than PPID horses with low ACTH. PPID ponies with high ACTH only differed from those with low ACTH in being significantly older.

These data indicate several interesting epidemiologic and pathophysiological inter-relationships within 2 complex endocrinopathic conditions and are illustrated in **Figure 1**. Further targeted investigation is required to further elucidate inter-related factors although the following appear to be plausible theories consistent with the findings.

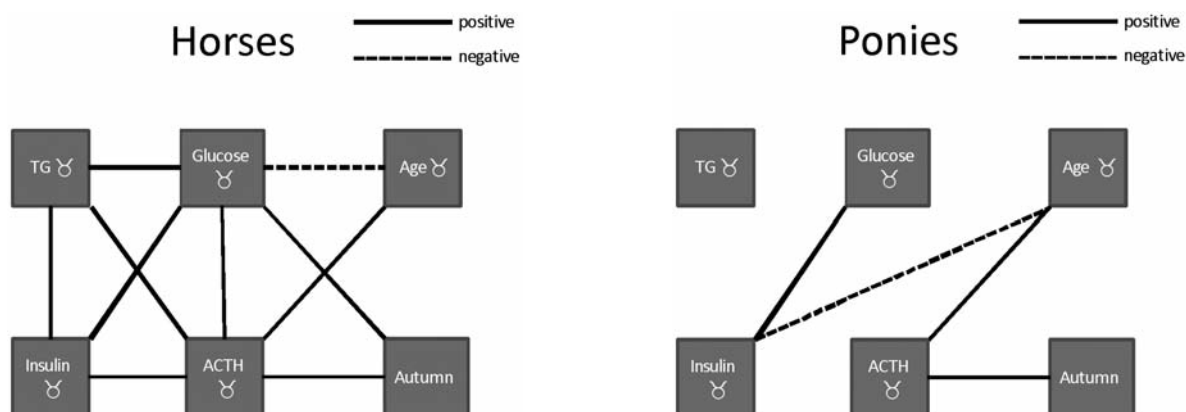


Fig 1: Illustrations of significant associations demonstrated in the study (gender effects not included for clarity).



Saturday 10th September ■ Hall 3B

PPID causes IR in horses?

In horses there appeared to be close associations between ACTH (a marker of PPID) and insulin, glucose and triglycerides (markers of IR) consistent with the possibility that PPID is a cause of IR in horses. In contrast, ponies had no significant relationship between ACTH and markers of IR casting doubt on PPID being a common cause of IR in ponies.

IR causes PPID in ponies?

The fact that ponies developed PPID at a younger age than horses is consistent with there being a predisposing factor or factors that are more common in ponies than horses. Possible candidates might include hyperinsulinaemia and IR. Interestingly insulin was higher in young vs. old PPID ponies which is consistent with them being IR at the onset of PPID with subsequent weight loss as a result of PPID gradually improving insulin sensitivity.

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Gastrointestinal Medicine

Chaired by Tim Greet

Sponsored by University of Liverpool



10.45–10.55

A tribute to Professor G. Barrie Edwards

Tim Greet

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The loss of Barrie Edwards leaves a massive hole in the veterinary profession in the United Kingdom. Barrie was known and loved by all. During a career spanning 5 decades, he acquired an international reputation; whilst for generations of veterinary students at Liverpool Veterinary School and the Royal Veterinary College, he was a legendary figure, the cornerstone of their undergraduate equine clinical education.

Self-effacing, incredibly talented and with seemingly endless time for everyone, Barrie was the ideal undergraduate teacher.

Originally a general large animal surgeon, it was as an equine surgeon and more particularly as the doyen of equine abdominal surgery in the UK that Barrie's name became synonymous with excellence and compassion. Others have demonstrated great skill, but very few could match him for the hours devoted to the care of his patients. Operating often in the dead of night, he retained the enthusiasm even at the end of a long procedure, to give a master class to his adoring undergraduate and post graduate students.

Barrie was the President of the British Equine Veterinary Association in 1995 and although not particularly forceful in committee he chaired Council meetings with a quiet assurance. He frequently presented lectures at BEVA meetings, particularly on abdominal surgery.

The CBE, RCVS Fellowship, the Dalrymple-Champneys Cup from the BVA for outstanding contributions to veterinary science, the Richard Hartley Award (twice), the EVJ Open Award, the Victory Medal of the Central Veterinary Society, the Animal Health Trust Award for outstanding scientific achievement, the Bridge Award of the BCVA, the BBC Veterinary Award, all reflect the appreciation and respect of a broad sector of our community.

A private family man, he was an accomplished artist and passionate about opera, cricket, golf and unsurprisingly for a Welshman, rugby football. However, even in retirement, Barrie regularly donned his operating gown and continued to demonstrate the skill which saved countless equine lives over the years.

We are much the poorer having lost a role model for any aspiring veterinary surgeon.

A gentleman and a gentle man of the old school; always prepared to embrace new ideas whilst retaining a passion for encouraging the young. I, like many others in the profession, have lost a good friend whose gentle and honest advice over the years has been of inestimable value.

Our sympathies are very much with Sue and the family at this time.



10.55–11.40

The contribution of Barrie Edwards to the treatment of colic in the horse

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When Professor Barrie Edwards passed away on May 16, 2011, our profession lost a giant in this soft spoken and humble man. I have no doubt that many of the great advances that have been made in colic surgery would have failed to reach fruition without the work of Professor Edwards and his colleagues at the University of Liverpool. I write from the perspective of one who could view his accomplishments from a distance, and thereby feel the great impact that they had on the international veterinary community. Professor Edwards will be missed beyond measure.

Surgeons that specialise in equine colic have access to information from many sources, such as bench type research, live animal studies and clinical cases. Clinical case material can prove to be the most valuable, because it allows study of the disease process and the response to treatment in a real world setting. However, this material takes time to develop to that point, and a review of Barrie Edwards' career makes it abundantly evident that he was able to establish a very active and strong colic caseload wherever he worked. From great success in building confidence in his wonderful skills as a veterinarian and as a surgeon, he and his colleagues were able to develop one of the largest databases for colic surgery in the world. This database has spawned some of the most comprehensive and meaningful publications on the epidemiology of colic, its causes, treatments and complications. Examples are effects of different diseases, types of anastomoses, and adhesions on survival, and risk factors for complications. These studies are all the more credible because they are supported by such a high volume of case material.

Barrie Edwards' legacy in the treatment of colic is improved survival rates for the unfortunate animals afflicted by this cruel disease. Many of the published works that bear his name are the results of teamwork and collaboration and the development of a very strong research group that will continue his work at the University of Liverpool. A first step in achieving success is in identifying failures, and this was accomplished in an early study on the incidence, diagnosis and treatment of post operative complications in colic cases. At a time when colic surgery was practiced by few and the case numbers were small, he and his co-workers were able to review a then staggering 259 surgical colic cases and demonstrated that post operative ileus and circulatory/endotoxaemic shock accounted for most post operative deaths. This laid the groundwork that underlies current treatments against these all too common complications.

Because of my own strong interest in small intestinal diseases that cause colic, I was easily attracted to Professor Edwards' practical and insightful approach to surgical management of these diseases. He has contributed to our understanding of some of the more common and dangerous diseases of this organ, specifically intussusceptions, strangulating lipomas and epiploic foramen entrapment. He recognised the unique challenges posed by the

equine ileum and these were accurately described in a 1981 publication that is highly relevant to this day. His manuscript on resection and anastomosis of equine small intestine is a must read for all colic surgeons. In a straightforward manner and clearly illustrated, it depicts some of the more common and complex procedures, supported by a balance between the observations of others and a wealth of personal experience.

Barrie Edwards' wonderful accomplishments in treatment of colic in the horse have earned him prestigious awards and recognitions, such as a doctorate, RCVS Fellowship, presidency of the British Equine Veterinary Association, the Dalrymple-Champneys Cup and Medal, and a CBE. He has received praise from students and colleagues and the highest national and international recognition for his work on colic surgery. Thanks to Barrie Edwards, horses with surgical colic are now referred earlier, receive better surgical care, and are more likely to survive longer than would be possible without his efforts. Perhaps the greatest tribute to his improvements in surgical techniques, aftercare and disease processes related to colic are his very visible contributions to improved survival rates that are evident today. As we expand on this success, we should continue to recognise the role of such pioneers as Barrie Edwards and how he made it all possible.

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Hall 3B ■ Saturday 10th September

11.40–12.10

Clinical use of epidemiology in the management of colic

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Epidemiology has greatly increased our understanding of equine intestinal disease (colic) over the past 20 years. This presentation will review how this knowledge can be applied to clinical situations and assist our management of colic cases.

Diagnosis

Many associations have been reported between specific types of colic and animal or management characteristics. Knowledge of these risk factors can be very helpful in compiling a list of differential diagnoses for an individual case before even examining the horse.

- *Season.* Recent work has highlighted the season pattern of occurrence of some types of colic. Colon displacements are more common in the Spring and Autumn, epiploic foramen entrapment most common between December and February. Equine grass sickness is a highly seasonal disease with peak prevalence between April and June.
- *Signalment.* Simply knowing the age, breed and sex of a colic patient helps to refine the differential diagnosis list. Pedunculated lipoma strangulation is a condition that is most common in geldings in their teens and older; large colon volvulus is more prevalent in post parturient mares; young horses are more susceptible to parasite-associated colic and also to intussusceptions.
- *History.* A history of recent change in diet is commonly encountered in colonic distension and displacement colics and in equine grass sickness. A poor worm control history increases the likelihood of parasite-associated colic. Horses demonstrating crib-biting/windsucking behaviour are at increased risk of several types of colic, most notably epiploic foramen entrapment.

Epidemiological clues can't give you a specific diagnosis, but they can help you to structure a list of differential diagnoses in order of likelihood.

Treatment

Epidemiology has confirmed what most vets knew anyway: that the majority of colic cases will resolve with medical treatment. It has also highlighted the importance of making the decision to advise surgical treatment as early in the course of disease as possible in order to maximise prognosis.

There exists reasonable literature documenting the patterns of post operative survival for horses after colic surgery. Epidemiology allows us to give clients realistic expectations of

what surgical treatment can offer:

- *Post operative survival.* This has been reported for many different horse populations and from several different hospitals. Global estimates of short-term survival are around 80%, with about 70% of horses surviving surgery living to at least one year post operatively. Vets should seek clinic-specific figures from their own hospitals or those that they refer to. Age does not affect survival of small intestinal cases until the horse is older than around 25 years.
- *Post operative complications.* A number of complications (e.g. wound infection, jugular thrombosis, post operative ileus) are more likely to occur in horses that are endotoxaemic. Horses recovering from pedunculated lipoma strangulation are at increased risk of post operative ileus.
- *Return to athletic performance.* This is a common question raised by owners around the time of colic surgery. The small amount of evidence available suggests that if horses survive colic surgery their probability of making a full return to athletic performance is high.

Prevention

Perhaps the most important clinical application of epidemiology is in the prevention of colic. We understand some of the risk factors for intestinal disease and some of these are modifiable through appropriate management:

- *Diet.* The importance of fibre and the potential dangers of high carbohydrate diets are increasingly recognised. Dietary change is also associated with increased risk of colic.
- *Parasites.* Good worm control has been demonstrated to be highly effective in reducing the risk of colic.
- *Dental care.* This risk factor for colic is only starting to be understood but there is clear evidence that poor dental health increases the risk of colic.
- *Equine grass sickness.* Many risk factors for this disease have been identified and although there is uncertainty about the pathways leading to the disease, we also recognise interventions that will decrease risk. Specifically, avoiding dietary change, avoiding pasture disturbance and tolerating low levels of strongyle infection are recommended.

Epidemiology is the study of disease in populations. There are dangers in extrapolation between populations and extrapolation from population level to individuals. However, used with common sense, epidemiological knowledge provides clinicians with an additional, very valuable tool in the clinical setting.

NOTES



12.10–12.45

Latest concepts in the management and treatment of colic

David Freeman

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Long-term survival rates after colic surgery have improved, which might reflect earlier referral and improvements in surgical techniques and post operative care. Although primary disease and type of anastomosis could affect survival in the long term, short-term survival rate does not appear to be affected by the horse's age. Most deaths occur during the first 10 post operative days, with 69% during the first 100 days after surgery and then a slower decline in death rate.

Epiploic foramen entrapment

The apparent predisposition to EFE in older horses has been refuted. Some studies found Thoroughbreds and Thoroughbred crosses and males to be at a high risk, mainly between October and March, suggesting that stabling played a role in its cause. Also there is strong evidence that cribbing is associated with this disease. The long-term survival after completed surgery for EFE is lower than for other small intestinal diseases.

Diagnosis

In one study, peritoneal fluid lactate was a useful predictor of early intestinal ischaemia from a strangulating obstruction. Another study demonstrated that peritoneal fluid lactate is a more useful and sensitive prognostic indicator than plasma lactate in horses with colic. However, plasma lactate concentration can be highly accurate in predicting survival after large colon volvulus.

Ultrasonography is useful for diagnosis of intestinal strangulation, peritonitis, intussusceptions, displacements, renosplenic entrapment, diaphragmatic hernia, cholelithiasis, ruptured bladder, ascarid impactions, inguinal hernias and abdominal neoplasia. Large colon torsions can also be diagnosed with a high degree of accuracy.

Nephrosplenic ligament entrapment of the colon

Almost all horses with large colon trapped over the nephrosplenic or renosplenic ligament can be successfully treated by nonsurgical methods, such as rolling the horse while anaesthetised with short acting intravenous anaesthetic. Alternatively, the spleen can be reduced in size by phenylephrine at 3 µg/kg bwt/min for 15 min and the colon dislodged by lungeing the horse for 10–15 min. Phenylephrine carries a risk of fatal haemorrhage in old horses. Open, laparoscopic, or hand-assisted laparoscopic closure of the renosplenic space can be used selectively to prevent recurrence of this disease.

Large colon resection

Large colon volvulus has a high rate of recurrence, especially in broodmares. This and other diseases of the colon can be treated effectively by resection. Colopexy of the ventral colon to the body wall is an alternative to prevent recurrence of colon displacements, but is not recommended for a compromised colon. A technique for large colon resection with complete bypass of the right dorsal colon was designed for those horses with extensive and advanced colonic disease.

Incisional hernia prevention and treatment

An incisional hernia can occur in 7–10% of cases after colic surgery and is typically preceded by surgical site infection in the first week after surgery. Short surgery times, attention to proper technique

and asepsis, short incisions, application of a protective adhesive barrier over gauze sponges before placement in the recovery stall, and routine use of an abdominal bandage after surgery can prevent infection. Skin staples can increase the risk of incisional infection and horses that develop incisional infections usually culture multi-resistant bacteria from the incision within 12 h of surgery. Once a hernia has developed, an abdominal Hernia Belt can be used effectively as a conservative treatment method to reduce the hernia, especially during the early stages of healing and fibrosis. Large hernias that do not respond to bandaging and exercise can be closed with sutures alone or be repaired with synthetic mesh.

Adhesion prevention

Intestinal adhesions develop in 6–13% of horses after small intestinal surgery. They can involve any region of the intestine without predilection for anastomosis or enterotomy sites, so pan-abdominal methods for adhesion prevention are indicated. Although the primary method of prevention is careful surgical technique, studies have found some benefit from i.v. dimethyl sulfoxide (20 mg/kg bwt), potassium penicillin (22,000 iu/kg bwt), and flunixin meglumine (Banamine, 1.1 mg/kg bwt), and for intraoperative treatments, such as intraperitoneal unfractionated heparin (20,000 iu), omentectomy and sodium carboxymethylcellulose (SCMC 7 ml/kg bwt). Post operative peritoneal lavage can decrease adhesion formation in horses, but can be labour-intensive and complicated by catheter occlusion.

Pharmacological management of gastrointestinal tract diseases and complications

Recent interest in lidocaine has focused on its ability to prevent post operative ileus (POI). For this purpose, 2% lidocaine is given as a bolus of 1.3 mg/kg i.v. slowly over 5 min followed by 0.05 mg/kg bwt/min in Normosol®-R as a CRI for 24 h. Lidocaine can provide somatic analgesia, but had no effect on visceral pain in one study. Although initially assumed that lidocaine was a prokinetic agent, it might inhibit normal motility, which could be an argument against its use after colic surgery. The perceived clinical benefit of lidocaine CRI in horses after gastrointestinal tract surgery is currently attributed to novel anti-inflammatory effects on neutrophils, which require a concentration that is less than the concentration necessary to block sodium channels. Intestinal inflammation appears to be an important cause of POI in laboratory animals after surgical manipulation. However, lidocaine does not reduce inflammation in some nongastrointestinal inflammatory models in the horse.

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How To Deal With:

Chaired by Chris Pearce

14.00–14.25

How to deal with: Skin wounds of the upper limb

Patrick J. Pollock

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Wounds are one of the most commonly encountered injuries in equine practice. Indeed it seems that of all the species dealt with by veterinary surgeons, the horse is particularly prone to wounds. The conditions in which horses are kept, the type of work that they are involved in, and the potentially 'flighty' equine temperament probably all contribute to the high incidence of such injuries.

Healing is an orderly biological process of repair that restores continuity to injured tissue. Healing occurs by first intention when union or restoration of continuity of tissue occurs directly without the production of granulation tissue. Wounds are sutured with the intent that they will heal by first intention. Wounds left unsutured heal by second intention. Third-intention healing occurs when closure is delayed to allow treatment (delayed closure). Wounds move predictably down a common pathway of healing, and knowledge of events of healing can be used to decide steps in wound management. The sequence of events progresses through 4 overlapping phases:

1. Inflammatory phase
2. Debridement phase
3. Repair phase
4. Maturation phase

Upper limb wounds are commonly encountered in equine practice and generally heal rapidly with fewer complications than those of the distal limb. There are a number of reasons for this including greater soft tissue protection for underlying synovial and neurovascular structures, greater collateral blood supply and an increased rate of wound contraction.

Wound contraction is a major contributor to the healing process, and the rate of contraction of wounds has been calculated from experimental studies involving experimental wounds of a standard size and shape. It has been shown that wound contraction not only speeds up the healing process but enhances the tensile strength and cosmetic appearance of the healed wound. Even very large wounds involving the upper portion of the limb can heal with a good cosmetic and functional result.

Despite this a number of problems are encountered with upper limb wounds, indeed severe wounds to the upper limb can result in tremendous tissue loss and exposure of underlying structures that can present challenges to the clinician.

Wounds involving the upper limbs are often sustained during high impact collisions and there may be a great deal of co-lateral damage, tissue loss and underlying trauma.

Veterinary evaluation of a wound to the upper portion of the limb should follow a standard approach. Assessment of the status of the entire patient to rule out immediately life threatening complications is followed by a systematic examination of the wound. The integrity of underlying structures should then be carefully investigated and appropriate management instigated.

A myriad of impediments to successful wound healing have been identified in horses, and many of these apply to wounds

affecting the upper portion of the limbs.

These include: involvement of sensitive underlying structures such as joints or tendon sheaths; excessive movement; production of exuberant granulation tissue; heavy contamination with soil, debris and faeces, and the potentially uncooperative nature of the animal. Further challenges include the potential for wound treatment to become very expensive for the client.

Time elapsed between wounding and treatment should be given only modest consideration. Veterinary surgeons have traditionally held to the principle that wounds should be closed during the 'golden period', which is the time necessary for contaminating bacteria to multiply to the concentration of 10⁵ organisms per gram of tissue. This period is generally considered to be 6–8 h from the time of wounding, but this period is arbitrary and may differ among areas of the body.

Whether the wound is being closed or left open, the wound and surrounding skin should be cleansed to remove microfauna, gross contaminants and blood clots.

Hair in a closed wound acts as a foreign body, therefore care should be exercised to avoid contaminating the wound when removing hair. Hair surrounding the wound should be dampened with water or coated with K-Y water soluble jelly.

The skin surrounding the wound should be cleansed with antiseptic solutions, but these should not be applied to the wound itself or they may interfere with healing.

Contaminating bacteria on wounds can be reduced to noninfective concentrations by using high-pressure lavage, debridement, and antimicrobial drugs administered systemically or topically.

Pressure of 8 pounds per square inch or higher is classified as 'high-pressure', and pressure below this level, as obtained with a bulb syringe, is designated as 'low-pressure'. A common concern about high-pressure lavage is that it may disseminate foreign material and bacteria from the surface of the wound into the depths of the tissue, but this concern is not valid. High-pressure lavage can damage tissue, however, and should not be used indiscriminately.

When assessing a wound, the following information may be important: when and how did the wound occur; how big it is and which part of the body is affected; is there any discharge; could there be involvement of underlying structures; whether the horse is lame; if the wound is recent, whether there is significant haemorrhage, and, if the wound is old, is there evidence of infection or severe inflammation?

It is the expectation of many horse owners that the wound will or should be closed immediately. This is often NOT in the horse's best interests particularly where there is a substantial loss of skin and/or a great deal of contamination.

A great many options have been advocated for dealing with equine wounds and some of these will be discussed in the course of this presentation.



14.25–14.50

How to deal with: Skin wounds of the lower limb

David Lloyd

The Liphook Equine Hospital, Forest Mere, Liphook, Hampshire GU30 7JG, UK.

Wounds involving the lower limbs of horses are very common and their significance can vary immensely. The lower limb is susceptible to many forms of trauma, causing sharp laceration of the skin, foreign body penetration or loss of skin through blunt trauma or abrasion. Injuries often include a high level of bacterial and fungal contamination by organic debris. The lower limb has limited soft tissue protecting the vital structures beneath, thus injuries involving a tendon, ligament or synovial structure need to be identified as early as possible. It is sometimes difficult to fully assess the extent of the injury, due to a distressed horse or owner, or profuse haemorrhage for example, and sometimes poor conditions or facilities make thorough examination difficult. Under such circumstances the horse may require sedation, control of haemorrhage and bandaging so the wound can be examined later, once the horse has been moved to better facilities.

The principal aim is restoration of the tissues and the accurate assessment of the wound, at whatever its stage of healing, can help optimise repair. Compared to other areas of the body, there are several factors affecting healing of the lower limb which require consideration and this will help decide whether a wound is closed primarily, whether primary closure is delayed or if the wound is left to heal by secondary intention. In chronic cases there may be little option but to continue secondary intention healing and options regarding the wide variety of treatments to optimise healing will be discussed during this presentation.

Some of the factors to consider when dealing with a lower limb wound:

- *The nature of the injury:* how the wound(s) occurred and the possibility of foreign body penetration? Blunt trauma is more likely to compromise the tissues further away from the wound margins with swelling causing increased tension on any primary closure.
- *Anatomical location:* there is extensive movement of the skin, particularly over the carpus/tarsus and digits causing instability

and excessive granulation tissue formation. Adequate measures to limit movement can improve healing and minimise dehiscence of wound closures. Wounds involving the coronary band should be repaired whenever possible to limit abnormalities in hoof wall growth.

- *Injury to underlying structures:* careful examination can help identify injuries requiring immediate attention. Skin lacerations or wounds are not very painful and severe lameness may indicate injury to the underlying structures. Diagnostic imaging, hospitalisation or referral may be required to fully assess deeper injuries.
- *Contamination/infection:* there are many factors determining whether microorganisms become established within the tissues surrounding a wound. Several measures, including those which deliver local treatment to the area affected, can help minimise colonisation or persistent infection. In many cases, the presence of excessive numbers of microorganisms is likely to be the single most important factor limiting wound healing and effective treatment can help optimise wound healing.
- *The phase of wound healing:* Inflammatory>Debridement>Repair>Maturation. Although the different phases are not distinct, recognition of the stage of healing can help identify what needs to be done to optimise healing.
- *Treatment costs:* Treatment can extend over long periods, regular re-assessment is often required and dressing materials all cumulate in significant costs for the owner. It is better that the client is aware of potential cost from the outset.

With careful consideration of the different factors affecting healing of distal limb wounds we can formulate treatment to enhance the healing process. It is important to regularly re-assess healing, particularly during the early stages, as complications such as infection or excessive granulation can rapidly reverse any progress made!

NOTES



Hall 3B ■ Saturday 10th September

14.50–15.15

How to deal with: Penetrating injuries to the foot

Matthew R.W. Smith

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Clinical evaluation

In acute injuries, diagnosis may be straightforward, as the site of penetration and injuring object are often known. Once the penetrating object is removed, the soft horn of the frog tends to collapse around tracts making them less obvious, and in this situation, hoof tester examination and careful paring of the foot will aid identification of the site of penetration. Involvement of the distal interphalangeal (DIP) joint or digital flexor tendon sheath (DFTS) results in distension of either respective structure. Following penetration of the navicular bursa, pain can often be elicited upon palpation of the distal deep digital flexor tendon (DDFT) from the palmar aspect of the pastern.

Regional anaesthesia is infrequently necessary to identify the site of lameness, but will aid radiographic examination of severely lame horses, and facilitate both exploration of puncture wounds, and synoviocentesis and positive contrast radiography of all 3 potentially involved synovial structures.

Diagnostic imaging

If the penetrating object is still *in situ*, a lateral and dorsopalmar/plantar radiograph should initially be obtained to ascertain the depth and direction of the penetrating object. Complete radiographic examination of the foot should then always be performed, to identify the presence of fractures, radiopaque foreign material, and in chronic cases osteitis/osteomyelitis. In the acute phase gas may also be identified within one or more synovial cavities confirming penetration of that structure. Following cleaning and paring of the foot, positive contrast radiography with a malleable metallic probe placed in the tract can be used as a means to define the direction and extent of penetration. Communication of the penetrating wound with any of the navicular bursa, DIP joint or DFTS can also be demonstrated by infusion and distension of each respective synovial cavity with a radiopaque contrast agent. This is evident radiographically as contrast material leaking from the synovial cavity along the penetrating tract. Ultrasonographic examination may yield additional information, and most usefully is performed from the palmar aspect of the pastern, allowing imaging of the proximal aspects of the podotrochlear apparatus.

Synoviocentesis

In horses with a solar penetration, the navicular bursa should be sampled first; if the synovial fluid from the navicular bursa is normal, then infection of the DIP joint or DFTS is unlikely (Smith 2011a). The navicular bursa is most easily sampled with the limb held raised, by insertion of a 3.5 inch, 18 gauge spinal needle between the heel bulbs, just proximal to the coronary band. The needle should be gently advanced in a sagittal plane aiming towards a point 1 cm distal to the coronary band, and halfway between the most dorsal and most palmar aspects of the coronary band. When significant resistance is encountered, the needle tip is usually located adjacent to the navicular bone within the bursa.

Advanced imaging

Scintigraphy and/or MRI are usually unnecessary for evaluation of acute penetrations, but can provide useful information in chronically lame horses following penetration.

General treatment

Antimicrobial therapy

Antimicrobial selection should be rationalised by the likely contaminating organisms, and their pattern of antimicrobial sensitivity. The horse's environment is often contaminated with soil and faeces, and *Enterobacteriaceae* and anaerobes are frequently implicated. A combination of penicillin G and gentamicin is usually appropriate in the first instance. Addition of metronidazole is also logical to extend coverage against anaerobes resistant to penicillin.

Management of the solar/frog wound

The solar wound should be debrided to remove contaminated and devitalised tissue, until healthy margins are achieved. This usually requires quite limited paring of puncture wounds. Initial lavage should be performed and then the wound bandaged. Puncture wounds usually require bandaging only until sealed. For larger wounds, systemic antimicrobials should be maintained until a confluent bed of granulation tissue fills the wound. Once epithelialisation has occurred, bandaging is often replaced by application of a hospital plate incorporated into a shoe. Once the wound has completely epithelialised, topical astringents such as bismuth subnitrate or 2% iodine may be applied cautiously to harden the newly formed stratum corneum.

Specific conditions

Infected osteitis of the distal phalanx

Infected osteitis with or without fracture and sequestration of a portion of the distal phalanx are potential sequelae of deep solar penetrations. Effective treatment almost invariably requires a combination of surgical debridement and antimicrobial therapy.

Synovial sepsis

Puncture wounds to the central third region of the frog are the most likely to result in penetration of the navicular bursa. Less commonly, punctures extend through the bursa to involve also the DIP joint and/or the DFTS. Until 1999, the primary treatment employed was surgical exploration, debridement and lavage using the so-called 'streetnail' procedure (Richardson *et al.* 1986). This has now been superseded by endoscopy of the navicular bursa (Wright *et al.* 1999). The procedure is performed under general anaesthesia with a standard 25 or 30° inclined arthroscope. The solar wound often can be utilised as an instrument portal. Advantages of bursoscopy over the streetnail procedure include more comprehensive evaluation of the navicular bursa, reduced periods of hospitalisation (and associated costs), shorter periods of convalescence, and greatly reduced patient morbidity and mortality.

Prior to routine use of endoscopic surgery, prognosis was considered poor. However, this has improved considerably and of 57 cases of penetrating wounds involving the navicular bursa, managed endoscopically at Newmarket Equine Hospital, 48 survived. Of these, 30 returned to their pre-injury use, 9 were able to work at a lower level of performance and 4 horses were useable following neurectomy of the palmar digital nerves. Five horses were retired.



DDFT injuries

Injury to the DDFT is inevitable following a solar puncture and infection of the navicular bursa. The DDFT may also be damaged by a penetrating object without synovial infection. These horses are typically lammer than would be expected following an uncomplicated penetration, and remain lame for longer. Diagnosis can be difficult, and confirmation usually requires MRI. If infected tendinitis of the DDFT is suspected, exploration and debridement of the solar penetration is indicated. The surgical technique is similar to the aforementioned streetnail procedure, but without fenestration of the DDFT. Involvement of the DDFT dictates a prolonged convalescence, usually 6–12 months. Rest and controlled exercise are paramount in modulating tendon healing.

Necrosis of the cartilages of the foot (quittor)

Penetrating injuries to the coronary band may traumatise the medial or lateral cartilage of the foot. This may in turn lead to

infection and subsequent necrosis of the affected cartilage, resulting in a chronic discharging sinus (or sinuses) proximal to the coronary band. Radiographic examination should be performed to rule out osseous involvement or radiopaque foreign bodies. Ultrasonographic examination is useful to confirm involvement of the underlying cartilage. Medical treatment alone is frequently unrewarding, and surgical debridement of the infected cartilage is usually necessary (Smith 2011b).

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NOTES



Hall 3B ■ Saturday 10th September

15.15–15.40

How to manage suspected synovial sepsis

Bruce Bladon

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Synovial sepsis is a common condition in equine practice. The condition is important as it represents one of the few opportunities for a veterinary surgeon to make a difference to a horse's future; manage the condition well and the horse will return to full athletic activity with the benefit of a large bill, manage it badly and the horse will remain lame for ever, and the initial veterinary profitability will be reduced.

The cause of synovial sepsis in the adult horse is almost invariably a penetrating wound. This wound can be iatrogenic, i.e. secondary to joint injection, or can be traumatic. In the foal the cause of joint sepsis is usually haematogenous spread, and this is rarely seen in the adult horse.

The diagnosis of synovial sepsis is apparently straightforward. A sample of synovial fluid is obtained and analysed for total white cell count and total protein. A white cell count of $>30 \times 10^9/l$ is considered diagnostic of synovial sepsis, and a total protein of $>40 \text{ g/l}$ is strongly suggestive. EDTA always results in artificially elevated total protein levels when measured by refractometry. This is seldom significant in blood samples but if a small volume of fluid is obtained and placed into EDTA anti-coagulant then a markedly elevated protein level will be recorded. Intra-articular fractures and other trauma can raise the white cell count, so care must be taken to fully evaluate the joint. Acetic acid based diluents (Turk's solution) used for manual cell counts are unreliable as they precipitate hyaluronan so 0.9% saline should be used as the diluent.

In a recent onset case, $<6 \text{ h}$, it is possible to sample the joint prior to the elevation in the white cell count. At this stage the joint is contaminated, with viable bacteria present, but not infected, with the bacteria multiplying in the synovial membrane. The simplest way to confirm a diagnosis of joint contamination is to demonstrate that a wound is continuous with the joint. This is easily achieved at the same time as synoviocentesis. Following obtaining synovial fluid, the joint should be injected with Hartmann's solution, to distend the cavity. If the wound is continuous with the joint the fluid will usually be observed to flow clearly from the wound. Inability to retrieve fluid is one of the commonest complications of the diagnosis of synovial sepsis. This is usually the result of small joint spaces with limited amounts of fluid and large wounds, which result in all the fluid falling out.

Radiography and ultrasonography should not be ignored. Some causes of sepsis may be evident on radiographs, such as axial osteitis of the proximal sesamoid bones. Some cases of synovial sepsis are associated with further damage, typically lacerations of the digital flexor tendon sheath, often with laceration of the enclosed tendons, and ultrasonography may help to quantify such damage. I would not condemn anyone for operating without radiographs or ultrasonography, but both the veterinary surgeon and the client should be aware that a 'shortcut' is being taken.

In recent years there has been a trend with the management of synovial sepsis, back towards killing the bacteria, as well as

flushing them out. This approach should begin during diagnosis. The joint should be treated concurrently by injection with antibiotics. A concentration dependent antibiotic should be chosen, which is not irritant. In most situations this is amikacin. Gentamicin can be used but is painful and should be used with local anaesthetic.

The preferred treatment for synovial sepsis is lavage of the joint with large volumes of polyionic fluids. I recommend that this should be under arthroscopic control, and I do not advocate needle lavage. There is universal agreement from all authorities that surgery to lavage the joint should be undertaken rapidly. Expert opinion is the lowest level of evidence based medicine, and there have been very few case series which back up this opinion. One of the few was Fraser and Bladon (2004), who showed a higher success rate for horses treated within 36 h of injury. We continue to rely on this data. Thus we will operate on a horse admitted to Donnington Grove Veterinary Surgery the same day as admission in many cases, but for a simple joint laceration sustained that day and admitted that evening, we will ensure that the joint has been medicated with antibiotics and operate the following day.

Post operative treatment has also evolved to place more emphasis on local antibacterial treatment and less on repeat surgery. We follow a set protocol in most cases. The day following surgery, the horse is treated by i.v. regional perfusion with antibiotics. The choice of antibiotic is usually 500 mg amikacin, as culture and sensitivity results are seldom available yet. However, if culture results are known other agents may be more appropriate. Enrofloxacin is irritant and should be avoided. Ceftiofur is more active following hepatic metabolism, so is not ideal, particularly as cefquinome is a good substitute. We always dilute the selected dose of antibiotics to 60 ml with saline. A tourniquet made of a mountain bike inner tube is wrapped around the limb proximal to the injury. If the veins are not easily palpated, it can help to wrap the tourniquet downwards, to increase the blood volume in the lower limb, like a reversed Esmarch bandage. A vein is then injected - we prefer the use of a 23 gauge butterfly needle. We have had poor success with indwelling catheters, which have rapidly resulted in thrombosis. The tourniquet is maintained for 20 min after injection and then released.

The following day synoviocentesis is repeated and the joint is medicated with antibiotics. If the white cell count has returned to $<10 \times 10^9/l$ then no further specific management is necessary, other than completing a course of systemic antibiotics and routine bandaging. If the white cell count remains elevated then the cycle of i.v. regional perfusion followed by synoviocentesis is repeated.

Reference

Available on request from the author.

NOTES



How To Deal With:

Chaired by Deidre Carson

16.15–16.40

How to manage a difficult foaling

Andrew J. McGladdery

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Fortunately, dystocia is a relatively uncommon condition with the majority of mares foaling without difficulty. However, dystocia is a genuine emergency where prompt action can save the life of not only the foal but in some cases also the mare. The reported incidence of dystocia is about 4% in Thoroughbreds to about 10% in some draught breeds and Shetlands. It is important to have a basic kit of suitable obstetric equipment which should include sterile obstetric lubricant (with a sterile pump and tubing), obstetric ropes or chains and handles, and a foal emergency resuscitation kit and ideally an oxygen tank. A fetotomy kit is not essential as fetotomy is the remit of experienced obstetricians and should be avoided unless well trained. Calving jacks are not suitable for foaling mares and should not be used. Advice to the owner or attendant whilst responding to the emergency call can help improve outcome. This advice may include keeping the mare walking which will help reduce straining and pain and reduce the possibility of pelvic impaction of the foal. Allowing the mare to get up and down may help rotate a foal that is upside down and if a 'red bag' is presented then immediate rupture and delivery of the foal should be advised.

As time is critical, on arrival at a dystocia a rapid inspection of the mare's physical condition is usually the norm, the most important clinical condition to be aware of is if the mare has signs of haemorrhagic shock. A full examination of the mare can be performed once the dystocia has been resolved. Examination of the fetal extremities and mare's perineum will help determine how long the foaling has been in progress with moist fetal extremities and vulval tissues indicating a short duration and dry swollen tissues and fetus a more protracted condition. Important questions to ask, at the time, are when was the mare due? How long has second stage labour been underway? What manual intervention has been performed and what discovered? The mare should be examined after the perineum has been rapidly cleansed and the tail bandaged, hopefully in advance. Ideally, any vaginal palpation should be performed with the mare standing and appropriately restrained and consideration should always be given to the safety of both the examining veterinarian and the personnel handling the mare. Foaling mares can behave quite violently and in an unpredictable way. Stocks are generally not appropriate because many foaling mares will collapse without warning. Vaginal examinations may be made using rectal sleeves and surgical gloves however, many obstetricians find this reduces touch sensation and this can hinder attempts at correction. Repeated vaginal examinations will traumatise the mare's tissues and this should be borne in mind. Initial examination should assess any obvious injuries to the mare's tract and if there are any pelvic abnormalities that may affect delivery. The presentation, posture and position of the foal should be determined and if the possibility of twins or fetal deformity may be a complication. If the uterus is very contracted about the foal additional lubrication may be indicated which can be administered by sterile stomach

tube and pump. Proper placement of a head rope may be appropriate to ensure control of the head and the forelimbs may have ropes applied above the fetlocks. Obvious fetal movement may indicate a live fetus and or stimulation of fetal extremities may elicit a suitably positive response. It may be possible to palpate the fetal thorax for an apex heart beat. It also may be possible to intubate the fetus so that it can be ventilated whilst the dystocia is resolved. This can be quite difficult to achieve but if successful does ease the pressure of trying to achieve a very rapid resolution.

Once assessment has determined the cause of the dystocia manipulation usually involving repulsion and then replacing the extremities to their proper position for vaginal delivery can be attempted. It is important to be aware of time and unless it is certain the foal is dead one normally attempts to resolve the dystocia within 15 min or so before considering alternatives. It is important especially if the owner's wishes are uncertain to discuss the possible options as one performs the assessment. The economic factors associated with the mare and foal may have a bearing on how one proceeds and the options considered. In valuable breeding mares it is especially important not to risk major injury to the cervix through over enthusiastic vaginal delivery when caesarian section may be more appropriate. The use of sedation and tocolytics (e.g. clenbuterol) can be helpful. Remember that sedatives may have a detrimental effect on the foal and if large doses are used inappropriately there is also a risk that the mare may become recumbent and impossible to make stand such that it is difficult to move her to a hospital facility should that become necessary.

It is usually not practical to administer an epidural anaesthetic when the foal is still alive due to the time involved. However, short duration anaesthesia may help correct a dystocia especially if repulsion is impossible in the standing mare. Usually, the mare's hindquarters will be hoisted, once anaesthetised, to aid repulsion and correction of the dystocia. Large quantities of sterile lubricant can be infused at this time to aid the procedure before vaginal delivery is completed in a laterally recumbent position. During delivery the force required for extraction should be no more than the strength of 2 reasonably strong persons. If extraction is ineffective, consideration to ensuring the dystocia has been properly corrected should be made as often some degree of rotation along the foal's long axis may be helpful. If the dystocia cannot be corrected a caesarian section is often the ultimate option although, if the foal is dead and an experienced obstetrician present, a fetotomy may be considered.

Further reading

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- Munroe, G., Campbell, M., Munroe, Z. and Hanks, M. (2011) Dystocia. In: *Equine Clinical Medicine, Surgery and Reproduction*, Eds: G.A. Munroe and J.S. Weese, Manson Publishing, London. p 283.



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16.40–17.05

How to deal with colic

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Colic is something that most horse owners dread as they are aware of its potential seriousness. Whilst the majority of colic cases seen in practice resolve with medical therapy, it is important to remember that around 1 in 10 colic cases will require surgery. The practitioner plays a key role in identifying those cases and ensuring that where surgical treatment is an option, horses are referred for surgery promptly. Multiple studies have demonstrated that early referral, prior to the development of a marked deterioration in horses' cardiovascular parameters, remains one of the most significant factors in maximising their chances of survival following colic surgery.

History

It is important to obtain details about ongoing medical problems, the horse's usual management and any recent changes in this. Questioning about the colic episode should include when these signs started or when the horse was last seen to be normal and the signs of colic observed. If the horse is in violent pain when you arrive, it may be more appropriate to obtain some of these details once you have performed a clinical examination and have provided analgesia. However, it is important to obtain these facts as they may provide clues as to the most likely cause of the colic episode.

Observation - is it colic?

Most horse owners can recognise signs of severe colic but may wrongly identify a horse that spends more time lying down than normal as having mild colic, e.g. laminitis.

Baseline clinical examination

Clinical information that should be obtained include heart rate (prior to administration of medication), assessment of mucous membranes, respiratory rate and rectal temperature. Abdominal distension should be noted and auscultation performed to determine whether intestinal borborygmi are normal, absent or reduced. Where a colic case does not respond to medical therapy, repeat assessment of these clinical parameters is vital in determining whether the horse is showing evidence of systemic deterioration indicating the need for potential surgical intervention.

Additional tests

Rectal examination

This provides valuable additional information and may be diagnostic, e.g. pelvic flexure impaction. It is important to consider the potential risks such as rectal tears or injury to yourself, particularly in very small ponies and fractious horses. Sedation may be required in some cases (xylazine is very useful) and where the horse is straining, administration of butylscopolamine can help.

Nasogastric intubation

This enables the presence of any gastric reflux to be determined (net >2 l is significant) and should always be performed in a horse that has not responded to administration of analgesia. In the case of pelvic flexure impactions, it has a therapeutic benefit enabling fluids to be administered at the same time.

Systemic packed cell volume, total protein and lactate

In a hospital setting, these values can be obtained easily and quickly but out in the field, these become of less immediate practical benefit apart from lactate measurement which will be discussed in the lecture. However, in chronic colic cases, these can

give a good indicator of whether the horse's systemic parameters are improving or not.

Abdominocentesis

This is an easy to perform test that can provide valuable information. Normal peritoneal fluid should be straw coloured and clear and should have a total protein of <25 g/l, a lactate of <2.0 mmol/l and white blood cell count <5 x 10⁹/l.

What next?

Appropriate analgesia

You need to decide whether the horse can be treated medically initially or whether the signs are severe enough to warrant potential surgical intervention. You then need to determine the most appropriate analgesic - my preference would be phenylbutazone or butylscopolamine/metamizole for mild analgesia or an α_2 agonist (detomidine/romifidine) and butorphanol combination for more potent analgesia. There is always some controversy about the use of flunixin in colic cases. If surgery is not an option and more potent analgesia is required, flunixin is perfectly acceptable and if colic signs persist despite its administration euthanasia may then be the only option. In the case of a horse showing only mild/moderate colic signs and where surgery would be an option, use of flunixin should be considered very carefully (particularly for the less experienced practitioner). Its potent analgesic and anti-endotoxic properties do delay the signs of recurrence of abdominal pain and the increase in heart rate and PCV associated with endotoxaemia before it becomes obvious that a horse has a strangulating intestinal lesion (and is a common reason for delay in referral of surgical cases). A couple of hours makes a big difference in the case of a horse with strangulating lesion of the small intestine regarding whether intestinal resection is required - this has important consequences for its long-term survival.

Monitoring the horse and providing advice about colic prevention

Where possible the horse should be kept stabled and food removed. Water can be left in and the owner should be asked to update you again in 2 h, sooner if the horse shows any signs of pain. Some horse owners are reluctant to call the vet again if mild signs of colic recur and delay this until a few hours later when signs of pain are becoming more severe. This is a not uncommon reason for a delay in horses being referred for surgery and so it is important to give owners explicit instructions about when they should call you. In the case of horses that recover following medical management, it is important to rule out common causes of colic (e.g. dental disease and parasites) to make future episodes less likely.

Referring a horse for surgery

Indicators of the possible need for surgery will be discussed in the lecture. The decision to refer can sometimes be difficult to make, but referral centres will be happy to provide advice about management of cases including appropriate analgesia and likely costs. Owners should be provided with the relevant information (which will be discussed in the lecture) so they can make an informed decision about referral for surgery.

Further reading

Archer, D.C. (2004) Decision making in the management of the colicky horse. *In Pract.* 26, 378-385.



17.05–17.30

How to approach the horse with choke

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Oesophageal structure and function

The oesophagus has no serosa but 2 muscularis layers: *muscularis mucosa*: smooth muscle, progressively thicker proximal to distal; *muscularis externa*: striated muscle to mid thorax then smooth muscle to cardia.

Peristalsis is propagated proximal → distal from the pharynx with secondary waves generated at the level of a bolus. Hence why there are repeated oesophageal spasms when there is an obstruction.

Causes

1. Intrinsic (luminal) obstruction: FEED IMPACTION, foreign body, stricture, diverticulum, neoplasia, muscular hypertrophy.
2. Extrinsic compression: abscessation, lymphadenopathy, neoplasia, intramural inclusion cysts, persistent right aortic arch, hydatidosis.
3. Motility disorders: oesophagitis (post choke), oesophageal spasm (?), idiopathic megaesophagus, achalasia (Branchial arch defects ?), Grass Sickness, tetanus, myopathies, rabies.

History

Access to high risk feeds, especially inadequately soaked sugar beet pulp and proprietary cubes or nuts in greedy animals that eat rapidly. There may be a history of recent choke, sedation or nasogastric intubation (<24 h).

Clinical signs of 'choke'

Signs develop acutely with head and neck extension, muscle spasms, retching, swallowing, coughing and dysphagia with expulsion of food, water and saliva from the mouth and nares. Rarely is oesophageal distension palpable. Choke can be secondary, e.g. tetanus. If the animal has been choked for some time a thorough clinical examination is required. Some complicated or long standing cases provide greater cause for concern presenting with distress/colic, dyspnoea, fever, subcutaneous emphysema, circulatory compromise and/or endotoxaemia.

Diagnosis

Usually straightforward and may be a 'telephone' diagnosis based on a description of the typical signs or feeding/management history. Animals that have been obstructed for more than 1 h may be much more relaxed, standing quietly but remain dysphagic. Diagnosis is confirmed by failure to pass nasogastric tube to the stomach with reflux of air. Always stomach tube if at all possible, not least to confirm that the obstruction has cleared spontaneously. If still obviously obstructed sedate first. Intubation complications are rare. In a referral hospital based study of horses with colic, complications occurred in 9/580 horses (1.5%). Six out of 9 were repeatedly intubated for ≥4 days. Pharyngeal (n = 3) and oesophageal (n = 6) trauma was reported with perforation in 5/6 of the oesophageal trauma cases of which 4/5 were subjected to euthanasia.

Management and treatment

There are strongly held yet widely disparate views among practitioners as to the best treatment. Many cases will respond to mild sedation and spasmolytic therapy combined with starvation.

1. **Spontaneous relief** will occur in many cases due to profuse salivation, coughing, retching and primary and secondary peristalsis. I advise owners to observe the animal for 20 min then report back by telephone as inexperienced owners tend to panic as soon as an animal chokes. If the signs resolve spontaneously within a few minutes most animals recover uneventfully.
2. **Starvation:** If the choke fails to clear spontaneously then the animal should be starved and water withdrawn until the obstruction has been successfully cleared.
3. **Sedation and muscle relaxation:** Sedation with at least ACP but preferably detomidine or romifidine + butorphanol will achieve a number of aims:
 - a. Facilitate nasogastric intubation in fractious or distressed animals.
 - b. Animal will lower head aiding drainage of food and saliva.
 - c. Reduces risk of aspiration. Sedation MUST be adequate to lower head below shoulder if lavage of the oesophagus is planned.
 - d. Provides some degree of (probably) centrally mediated clinically apparent proximal striated muscle relaxation and changes in smooth muscle peristaltic activity.
4. **Spasmolytic therapy:** Buscopan may assist smooth muscle relaxation. Oxytocin (0.11–0.22 iu/kg bwt) causes a paradoxical relaxation of the smooth muscle portion of the oesophagus and is useful in some refractory cases. 5% of animals treated with oxytocin will show signs of colic. Although difficult to evaluate clinically many animals benefit from NSAID therapy.
5. **Nasogastric intubation:** will identify proximal extent of obstruction but NO attempt should be made to push the obstructing material distally.
6. **Standing oesophageal lavage:** Gentle gravity administration of water down a stomach tube at the obstruction followed by allowing the water/slurry to reflux back up the stomach tube will be successful in some cases. If the obstruction is not cleared in 20–30 min leave and try again in 4–8 h. Ensure that the animal keeps its head down to avoid aspiration. A cuffed oesophageal tube with a secondary narrow gauge tube attached to the interior of its lumen through which water can be pumped under pressure is available from Kruse UK.
7. **Lavage under general anaesthesia:** Rare nonresponsive cases may warrant lavage on a tilted table under GA. A securely cuffed endotracheal tube must be placed and water can be pumped in a controlled fashion down the stomach tube. Endoscopy afterwards.
8. **Oesophagotomy:** Used in cases in which a focal foreign body has been identified endoscopically.
9. **Fluid therapy:** Prolonged obstruction can result in dehydration and a hyponatraemic, hypochloraemic metabolic acidosis due to ongoing salivary losses. Theoretically 0.9% saline is the intravenous fluid of choice. In reality even after several days of obstruction many horses have surprisingly normal acid/base and electrolyte parameters and lactated Ringer's (Isolec) is satisfactory.
10. **Antibacterial and anti-inflammatory therapy:** All except the most simple of cases should receive broad spectrum antibiotics and NSAIDs because of the risk of aspiration pneumonia.



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Management following relief

1. Animals should be starved offering water and oral electrolytes only for 12 h.
2. Antibacterials and NSAIDs should be continued as appropriate.
3. Animals choked for >24 h and those that rapidly re-obstruct when feeding is resumed should have oesophagoscopy to check for mucosal damage.
4. Dietary management: gradual return to normal diet. Wet mash → grass → haylage → hay → pelleted feeds

Complications of 'choke'

1. *Aspiration pneumonia/pleuropneumonia*: all horses with choke will have some degree of tracheal food contamination. 10–15% will develop some degree of aspiration pneumonia. Severely affected cases have a grave prognosis.
2. *Oesophageal dysfunction*.
3. Mucosal injury/ulceration.
4. Circumferential ulceration which may result in stricture formation.
5. Diverticulum formation.
6. Oesophageal perforation.

NOTES
