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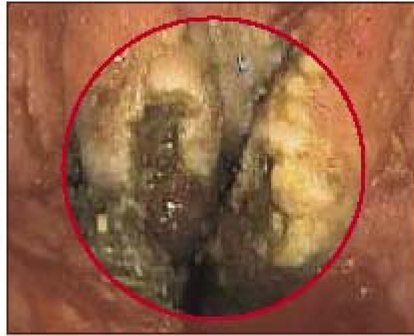

Equine Gastric Ulcer Syndrome (EGUS)

what you DON'T see is what you get!

The renowned equine veterinarian Dr Percy Sykes reiterated these famous words… "What the eye does not see, the heart does not grieve for" and unfortunately this is the case for a large number of horses who cannot ask you to have a look.

The acronym EGUS describes various types of lining erosion or ulceration of the oesophagus, stomach and small intestine and can be a painful condition with severe welfare implications. This syndrome involves stomach acid build-up and irritation, leading to ulceration of the stomach lining where the predominantly aggressive factor influencing the micro-environment of the stomach's gastric mucosa is hydrochloric acid. The acid secretion stimulated by ingestion of feed is not the primary concern, as it is largely neutralized by the buffering capacity of the ingested feed and bicarbonate rich salivary secretions stimulated by feeding. The concern is basal gastric acid secretion (horses secrete gastric acid constantly even in the absence of feed consumption), which has been implicated in the erosion of the squamous mucosa of the stomach. Anatomically, the stomach is divided into two functional areas namely, the glandular region and the squamous (non-glandular) region, which does not have the protective mechanisms such as bicarbonate rich mucus of the glandular region and is therefore predisposed to acid attack and subsequent injury. The areas around the upper portion of the stomach, particularly around the entrance from the oesophagus and the margin dividing glandular and non-glandular portions of the stomach are most commonly affected.

EGUS has long been suspected as a common performance limiting condition in racehorses however, the prevalence in a variety of other horse classes is just as concerning and it is important to consider that horses of all ages and all breeds have the potential to develop gastric ulcers. A recent Australian study ascertained EGUS has an 86% prevalence in Thoroughbred racehorses (Begg and O'Sullivan, 2003) however, alarmingly this is a syndrome not confined to race horses and also occurs in approximately 58% of show horses (McClure *et al*, 1999), 87% of Standardbreds (Rabuffo *et al*, 2002), 67% of endurance horses (Nieto *et al*, 2004), 51% of foals and 84% of Thoroughbred yearlings (Murray 1989, Murray *et al* 1987, 1990). While the adverse health effects in horses with gastric lesions remain undetermined, research suggests that the effects of gastric ulceration on condition and performance are much greater than has been recognized.

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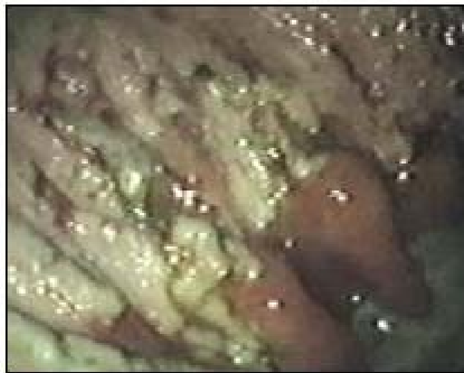
In the natural state, the horse is designed to be a continuous grazer of pasture and are not subjected to the many stresses of rigorous training programs or the confinement of stabling. Due to being designed as continuous grazers when left to their own devices, this means that the horse rarely has an empty stomach and conversely, never completely fills its stomach either. Additionally, under 'natural' conditions, horses consume a diet much higher in fibre and much lower in carbohydrates. A diet high in fibre requires adequate chewing, therefore forcing the horse to produce copious amounts of saliva manufactured during the intensive chewing process. Food intake is also slow and controlled with only small feed portions reaching the stomach at a time, yet the stomach remains particularly filled all of the time and is therefore assumed that assumed that the stomach would always have sufficient feed and bicarbonate rich saliva to buffer hydrochloric acid production. Furthermore, it must be considered that the size of the equine stomach is small (8-15 litres) and is therefore adapted for small, continuous meals as compared to stabling feeding regimens which only facilitate once or twice daily feeding in the majority of instances and these provided feeds are likely to be provided in large volumes in place of regular, smaller feeds. Unfortunately, horses in training or performing work require a more concentrated, high energy feed as opposed to bulk feed which is also contrary to the natural state. It is therefore not surprising, with the benefit of gastric endoscopy, to find such a high incidence in horses in training with these hidden gastric lesions.

In many cases, horses with gastric lesions remain asymptomatic (do not display clinical signs) and in the absence of correct diagnostic investigation, you may never know. However, there are certain aggressive factors which increase the risk of the development, severity and duration of gastric ulcers;

- Intense training and/or intense athletic performance
- Exercise on an empty stomach. *Horses that are exercised on an empty stomach have an increased risk of acid reflux when compared to those horses provided with a small roughage meal of hay or chaff prior to exercise.*
- High carbohydrate, energy dense grain based diets. *High concentrate diets are also high in digestible carbohydrates which are fermented by resident bacteria in the hindgut, resulting in the production of volatile fatty acids (VFA's), which in the presence of low stomach pH cause acid damage to the non-glandular squamous mucosa of the stomach.*
- Extended periods of training in the absence of spelling
- Extended periods without feed ingestion. *Horses grazing at pasture have a decreased incidence of EGUS due to the continuous flow of saliva and ingesta aiding to buffer stomach acid production. When feed is withheld from horses before performance or in managed stables, gastric pH drops rapidly and the non-glandular mucosa is exposed to an acid environment.*
- Insufficient provision of roughage (hay and pasture)
- Long distance transport, particularly if water and feed are restricted for prolonged periods of time
- Stress
- Stall confinement
- Non Steroidal Anti-Inflammatory drugs (NSAID's) such as phenylbutazone (bute). *This class of pharmaceuticals have been shown to increase the incidence of EGUS and are thought to cause more severe ulcers in the glandular stomach mucosa due to decreasing mucosal blood flow, decreasing mucus production and increasing hydrochloric acid secretion.*

Should any symptoms appear, they are fairly non-specific and include varying degrees of the following;

- Loss of appetite
- Gradual loss of condition/inability to maintain condition
- Temperament changes and loss of performance
- Dull/dry coat
- Picky eating behaviour
- Stereotypies such as windsucking
- Eating faeces, sand and/or dirt
- Anaemia
- Mild to severe colic is seen on rare occasions



ULCER - Pre Treatment

ULCER - Post Treatment

Under the un-natural conditions of imposed feeding and training regimens, it is difficult to find some means for the prevention of gastric ulceration and fortunately, horses respond well to similar human pharmaceutical medications. Currently, the most definitive method of diagnosis is gastric endoscopy, which allows direct visualization of all areas of the stomach. Ranvet were the first company to introduce the use of a video gastric endoscope, as we believe "Seeing is believing". The procedure involves the introduction of a 3 metre video endoscope through the nasal passages which passes down the oesophagus and into the stomach. The horse is required to have feed withheld for 12 hours prior to the procedure as in some cases is administered minimal sedation with the procedure generally being completed within 10 minutes. During this time, all areas of the stomach are visualized allowing ulcers (if present) to be isolated and graded according to severity and distribution via the use of a universally used 5 point scoring system. Due to the lack of additional laboratory diagnosis, in situations where ulcers are strongly suspected but gastric endoscopy is not available, it may be advisable to commence treatment and observe the horse for resolution of clinical signs. The duration of treatment can be difficult to predict and is closely associated with the severity of ulceration and treatment should be tailored for each horse and discussed with your veterinarian. Generally, a treatment phase of 2-3 weeks is recommended followed by a maintenance phase of less frequent administrations although it must be considered that clinical signs may resolve before healing is complete and horses remaining in training need to be maintained on treatment to prevent recurrence. Additionally, improvements in body condition and temperament may be seen shortly after commencement of therapy with ulcer healing having been shown to occur in 14-40 days.

Recent ulcer therapy clinical trials conducted by Ranvet established efficacy of Ranvet's oral paste with once daily administration. This oral paste works by being absorbed into the bloodstream following ingestion and acting on acid secreting glands to inhibit acid secretion, providing an environment conducive to wound healing.

The goals of anti-ulcer therapy are to relieve pain and discomfort, eliminate clinical signs, promote healing, prevent secondary complications and prevent recurrence. In very few instances, ulcers may heal spontaneously however, the vast majority require pharmacologic therapy to achieve healing, particularly while horses remain in athletic training. Current therapeutic strategies rely on acid suppressive therapy and the blocking of gastric acid secretion and raising stomach pH. This will instate a permissive environment in the stomach to allow ulcer healing however, dietary manipulation and environmental management are also encouraged to accompany pharmacologic intervention.

Due to the high recurrence rate, effective acid control should be accompanied by altered management strategies and/or long term treatment to prevent recurrence. The implementation of the following dietary, management and environmental strategies is advised to complement anti-acid pharmacologic therapy;

- Pasture turnout is ideal however, management of stalled horses can also be modified to decrease the risk of ulceration.
- The provision of adequate *ad lib* roughage (hay) may help to minimise the occurrence and severity of gastric ulcers by buffering gastric acidity due to increased chewing and hence increased saliva production. *Studies indicate that horses chew or 'jaw sweep' approximately 1,000 times to consume 1kg of hay and only 200 times to grind 1kg of oats. Horses eating 'sweet' or highly processed feeds which are highly palatable, may only chew 500 times per kg.*
- Providing constant access to high quality protein hay such as Lucerne hay will aid to raise gastric pH
- Sweet feed should be kept to a minimum and grains such as barley and oats may be substituted to increase the chewing mechanism
- Ensure teeth are checked regularly
- Ensure the implementation of a regular worming program and rotational drenching
- Feed type is important. *It must be considered that twice as much saliva is produced when horses eat roughage based feeds and pasture as compared with grain and other concentrates. Therefore, high grain, low fibre diets decrease saliva flow and result in lower gastric acidity which is a primary risk factor for the development of gastric ulcers. Alternatively, high roughage diets tend to stimulate production of bicarbonate rich saliva which aids to buffer gastric acid. It must be considered that the consumption of 1kg of hay make take 40 minutes in contrast to the consumption of 1kg of grains or concentrate may take 10 minutes, which significantly reduces the total feeding time*.
- Meal size is important. *Larger meals pass through the digestive tract more quickly than smaller meals due to stomach emptying being controlled by meal volume and unfortunately practices such as once or twice daily feeding of stabled horses force the consumption of large volumes of feed in short periods of time, which do not remain in the stomach for the required period of time. In addition, the high grain or concentrate component of most stabled horse rations tend to reduce the actual time spent eating and chewing, resulting in less saliva production and additional gastric acid action on the stomach.*

If you would like further information on Ranvet's ulcer treatment or information regarding Ranvet's video gastric endoscopy service and nutritional advisory services, please contact Ranvet toll free on 1800 727 217.

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