The association between gastric ulceration and clinical signs in adult horses

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Empirical study
Licentiate thesis in Veterinary Medicine
Helsinki University
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Equine Internal Medicine
2014

PREFACE

This study was focused on the statistical association between common clinical signs of gastric ulceration in the adult horse and the presence or severity of gastric ulceration as determined by gastroscopy. I hope the results might be of relevance to the everyday work done by veterinarians and students in the field.

I would really like to thank my supervisor Dr. Michael Hewetson for offering his support and knowledge. I'm grateful to Piia Maaria Sulku for letting me use the questionnaire piloted in her Master Thesis. I would also like to thank Dr. Marja Raekallio for helping me with the statistics done in this study.

March 2014, Cecilia Forsman

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ABSTRACT

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Työn nimi - Arbetets titel - Title
The association between gastric ulceration and clinical signs in adult horses

Oppiaine - Läroämne - Subject
Equine internal medicine

Työn laji - Arbetets art - Level	Aika - Datum - Month and year	Sivumäärä - Sidoantal - Number of pages
Empirical study	March 2014	47

Tiivistelmä - Referat - Abstract

Clinical signs associated with equine gastric ulceration are commonly reported in the literature, but are vague and often unsubstantiated. Clinical signs of gastric ulceration in yearlings and mature horses are less well recognized than in foals, but may be more important economically. There are no studies in the literature that have investigated the statistical association between clinical signs and gastric ulceration.

The aim of this study was to determine whether there is a statistical association between commonly reported clinical signs of gastric ulceration and gastric ulcer severity as determined by endoscopic examination of the stomach. The hypothesis of this study was that there is no association between the severity of gastric ulceration and the owners perception of clinical signs of gastric ulceration.

To achieve statistical significance, the study included 100 horses. A gastroscopic examination was performed on all the horses and documented on video. Owners were then asked to fill in a questionnaire documenting the clinical signs exhibited by their horses in the 3 months prior to the examination.

The ulcers where graded into four categories1) presence or absence of gastric ulcers; 2) presence or absence of clinical significant gastric ulcers (i.e. needing treatment or not); 3) presence or absence of glandular ulcers; and 4) presence or absence of non-glandular ulcers. The four categories where compared to the clinical signs using a Pearson Chi-Square or Mann-Whitney U-test. Significance was set at $p<0.05$.

A statistical association was found between clinical significant ulcers and losing weight (p=0,01) and between ulcer or no ulcer and losing weight (p=0,051). The results suggest that an owners perception of their horse losing weight could be associated with the presence of gastric ulcers and an increased severity of gastric ulcers, and can be used as an indication to perform gastroscopy on these individuals.

There was no association between gastric ulcer severity and the owners perception of colic, crib-biting, flank- biting, fussy eating, changes in behaviour, chronic diarrhoea, bruxism, poor body condition, poor coat condition and poor performance, and requests from owners to have gastroscopy performed on their horses based upon these clinical signs should be approached with caution.

Avainsanat - Nyckelord - Keywords
Equine gastric ulceration, clinical sign, statistics

Säilytyspaikka - Förvaringställe - Where deposited

Eläinlääke- ja elintarviketieteiden (EE) -talon Oppimiskeskus

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Tekijä - Författare - Author Cecilia Forsman		
Työn nimi - Arbetets titel - Title Samband mellan ulcus och kliniska symptom hos hästar		
Oppiaine - Läroämne - Subject Inre medicin, häst		
Työn laji - Arbetets art - Level Empririsk forskning	Aika - Datum - Month and year Mars 2014	Sivumäärä - Sidoantal - Number of pages 47

Tiivistelmä - Referat - Abstract

Kliniska symptom förknippade med ulcus hos hästar är ofta rapporterad i litterära studier. Kliniska symptom hos åringar och fullvuxna hästar är mindre kända men ekonomiskt mer relevanta än symptom förknippade med ulcus hos föl. Det finns inga studier gjorda inom litteraturen som undersöker en statistiskt relevant association mellan kliniska symptom och ulcus hos hästar.

Målet med denna studie var att fastställa ifall det fanns ett samband mellan vanligen rapporterade kliniska symptom för ulcus hos hästar och resultatet efter en gastroskopisk undersökning. Hypotesen löd att det inte fanns någon statistiskt relevant association mellan ulcus och kliniska symptom rapporterade av ägarna.

För att få ett statistiskt betydande resultat deltog 100 hästar i denna studie. En gastroskopisk undersökning gjordes på alla hästarna och resultatet dokumenterades på video. Ägarna ombads fylla i ett frågeformulär om kliniska symptom hos hästarna under perioden 3 månader före undersökningen.

Ulcusen graderades enligt fyra kategorier 1) ulcus eller inte 2) kliniskt betydande ulcus (behov av medicinering eller inte) 3) glandulär ulcus eller inte 4) icke-glandulär ulcus eller inte. Alla fyra kategorierna jämfördes med de kliniska symptomen genom Pearson Chi-Square eller Mann-Whitney U-testet. Ett betydande resultat var $p<0,05$.

Det fanns en statistisk association mellan kliniskt betydande ulcus och viktminskning ($p=0,01$) samt mellan ulcus eller inte och viktminskning ($p=0,051$). På basis av resultaten kan man dra slutsatsen att en ägares påstående att hästen magrat kan tas som en indikation för att utföra en gastroskopisk undersökning. Det fanns inga andra statistiskt relevanta associationer mellan ulcus och kliniska symptom i denna studie.

Avainsanat - Nyckelord - Keywords
Ulcus hos häst, kliniska symptom, statistik

Säilytyspaikka - Förvaringställe - Where deposited

Eläinlääke- ja elintarviketieteiden (EE) -husets Oppimiskeskus

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2 INTRODUCTION

Gastric ulceration is a common problem in equine veterinary medicine (Bell et al. 2007a). Clinical signs associated with equine gastric ulceration are common, but are often not specific. Furthermore, horses with clinical signs suggestive of gastric ulceration may have no endoscopic evidence of gastric ulceration. It is also commonly believed that gastric ulceration can occur without clinical signs, and the clinical relevance of gastric ulcers in these cases is therefore questionable. There may be subtle clinical signs in these cases however, but they are just being missed or ignored? For example, in a study by Murray et al. (1989), racehorses that were thought to be free of clinical signs according to the owners; but that were later found to have severe gastric ulceration; were treated with omepratzole and improved in appetite and performance. Suggesting that subclinical ulcus may be more important than previously thought.

There have been no studies that have investigated the statistical association between commonly reported clinical signs of gastric ulceration and the severity of gastric ulceration. A pilot study has already been carried out by Sulku et al (2011), who also created the questionnaire used in this study. She found an association between (1) glandular ulcers and inappetance; and (2) non-glandular ulcers and poor performance. However the population in this study was small (n=48), making statistical interpretation of the results difficult. The aim of this study was therefore to continue the work done by Sulku et al (2011) and attempt to determine whether there is a statistical association between commonly reported clinical signs of gastric ulceration and gastric ulcer severity (as determined by endoscopic examination of the stomach) using a large number of adult horses.

3 LITERATURE REVIEW

3.1 Gastric ulceration in horses

3.1.1 Introduction

Gastric ulceration is a clinically significant disorder both in adult horses and foals (Murray 1994b). In foals, gastric ulcers are potentially devastating and can result in death. In adults the economical

impact is more significant, with higher costs related to treatment and poor performance (Murray 1994b). The pathogenesis for gastric ulcers in critically ill foals may be different than in adult horses and is therefore not included in this study (Buchanan and Andrews 2003). Gastric ulcers can occur throughout the stomach, but the most commonly affected area is the non-glandular stratified squamous mucosa along the *margo plicatus* (Hammond et al. 1986, Murray et al. 1989, Murray et al. 1989).

3.2 Anatomy and pathophysiology

The equine stomach is divided into two different anatomical regions, the non-glandular stratified squamous mucosa and the glandular mucosa (Merritt 1999). There is a marked visible junction between these two regions, referred to as the *margo plicatus* (Merritt 1999). The majority of gastric ulcers (80%) are found in the proximal third of the non-glandular region of the stomach (Videla and Andrews 2009). Most commonly affected is the non-glandular stratified squamous mucosa along the *margo plicatus* (Hammond et al. 1986, Murray et al. 1989, Murray et al. 1989).

The anatomical distribution differs depending upon on the breed and use. Standardbred horses are affected by ulcers located mainly in the non-glandular region, whereas ulcers are most likely to be found in both regions (glandular and non-glandular) in Sport Ponies and Warmbloods (Luthersson et al. 2009a).

Horses are continuous HCl secretors and the pH of equine gastric contents can be less than 2,0 (Campbell-Thompson and Merritt 1990). Acid exposure is thought to be the primary cause of equine gastric ulcer syndrome (EGUS). The non-glandular squamous mucosa is predisposed to acid injury because of its lack of protective mucus and bicarbonate layer (Orlando 1991, Murray 1994b, Videla and Andrews 2009). The glandular region is protected by mucus and the bicarbonate layers (Videla and Andrews 2009). In this region hydrochloric acid (HCl) is secreted by parietal glands and pepsinogen (for digestion) is secreted by the zymogen (chief) cells (Murray 1992b, Merritt 1999). Gastric HCl secretion is stimulated by gastrin, histamine and acteylcholine (a neurotransmitter from the Vagus nerve) (Videla and Andrews 2009). Gastrin is produced by the G-cells, which are located in the pyloric glandular region (Merritt 1999). Histamine is secreted by the enterochromaffin-like (ECL) cells (Merritt 1999). Other acids found in the stomach like volatile fatty acids (VFAs), bile acids (BA), lactic acid (LA) and enzymes (pepsin) may also irritate the stomach mucosa (Videla and Andrews 2009). Ulceration in the glandular mucosa of the stomach is

considered to be a consequence of diminished mucosal protection (Murray 1994b) in combination with high acid exposure, however the pathophysiology is not fully understood (Murray 1992b). Prostaglandin E2 has an important role in gastric mucosal protection, although the precise mechanism is not fully understood. Prostaglandins promote mucosal blood flow, inhibit acid secretion, increase mucus and bicarbonate secretions; and support mucosal cell repair (Anonymous 1999). Epidermal growth factors (EGFs) also play an important role in gastric glandular mucosal protection (Anonymous 1999). EGFs are found in salivary gland secretions and promote DNA synthesis and proliferation of gastric mucosal cells (Anonymous 1999).

3.3 Predisposing factors and prevalence

3.3.1 Signalment

The prevalence for gastric ulcers varies widely depending upon breed and use. In racehorses in active training in New Zealand, the prevalence was as high as 88,3 % (Bell et al. 2007b). This is similar to results reported elsewhere for racehorses in active training (Bell et al. 2007b). Horses used for pleasure riding have a much lower prevalence (37%) and the degree of the ulceration is less severe (Feige et al. 2002). Differences between gender or age based on the prevalence of gastric ulceration has been shown in some studies but refuted in others (Rabuffo et al. 2009). In the most recent studies no association between sex and gastric ulceration has been found (Rabuffo et al. 2002, Bezdekova et al. 2007). The prevalence for EGUS increased with age in a study performed in Standardbred racehorses in training; the severity of EGUS was higher in horses ≥ 3 years of age (Rabuffo et al. 2002). This could, however have been because the older horses had been in race training for a longer period, and longer training means that the squamous mucosa is exposed to the acidic gastric contents for a longer period of time (Lorenzo-Figueras and Merritt 2002, Jonsson and Egenvall 2006).

3.3.2 Exercise

Training and exercise appears to predispose horses to gastric ulceration. In several reports the prevalence of gastric lesions in racehorses in active training was 70-90 % (Hammond et al. 1986, Murray et al. 1989). It is proven that horses exercised at maximal intensity (i.e. gallop) have significantly greater numbers and severity of gastric squamous mucosal lesions than horses

exercised only at a trot (Vastistas et al. 1999, Dionne et al. 2003). Increased intra-abdominal pressure during intense exercise in horses causes gastric compression, leading to acidic contents being pushed into the proximal, squamous-lined region of the stomach (Lorenzo-Figueras and Merritt 2002). Longer exposure of gastric mucosa to gastric acidity may be the reason that squamous lesions tend to develop or worsen when horses are in intensive training programs.

An increase in serum gastrin has been shown to occur in exercising horses (Furr et al. 1994). This elevated serum gastrin may increase glandular HCl secretion that may lead to acid damage. Another theory is that intense training might cause a diversion of blood flow to muscles, thereby decreasing mucosal blood flow and lead to a decrease in mucosal acid resistance (Murray et al. 1989, Andrews and Nadeau 1999, Lorenzo-Figueras and Merritt 2002). Horses that have been training for a longer period of time were thought to be more predisposed to gastric ulceration (Rabuffo et al. 2002).

A Swedish study found that horses in preparatory training had more severe ulcers than those who where fit for racing but had not raced during the last month; an interesting result since the former had been in training for a shorter time (Jonsson and Egenvall 2006). An explanation for this might be that horses in preparatory training often have longer training sessions than older horses that have reached a higher level of fitness (Jonsson and Egenvall 2006). The relationship between training, feed constituents, feeding management and eating behaviour on gastric peptic injuries has not yet been fully elucidated (Murray 1999).

3.3.3 Feeding

Horses are non-ruminant herbivores, well suited to a high fibre, low starch diet and would naturally spend up to 18 h / day foraging, and rarely fast voluntarily for more than 2-4 h at a time (Luthersson et al. 2009b). Horses are continuous HCl secretors and the pH of equine gastric contents can be less than 2,0 (Campbell-Thompson and Merritt 1990). The stomach of a mature horse secretes approximately 1,5 l gastric juice hourly and about 4-60 mmol of hydrochloric acid per hour (Campbell-Thompson and Merritt 1987). When horses have free access to hay, gastric pH measurements are often greater than 6 (Murray and Schusser 1993). The acids are neutralized by salivary bicarbonate and absorption of acidic gastric secretions by ingested hay (Murray and Schusser 1993). Horses grazing at pasture have a decreased prevalence of EGUS, most probably because there is a continuous flow of saliva and forage buffering the stomach (Reese and Andrews 2009). However a recent study found that pastured pregnant and non-pregnant mares had a high

prevalence of gastric ulcers (le Jeune et al. 2009). Another recent study found that the proximal stomach pH was lower in the early morning hours regardless of housing (Husted et al. 2008). This may be why pastured horses are also susceptible to EGUS, especially when these broodmares consumed less forage during the evening hours compared with the daytime hours, which may result in less saliva production and low pH environment in the proximal stomach (Husted et al. 2008, le Jeune et al. 2009, Videla and Andrews 2009).

Intermittent feeding has been demonstrated to cause and increase the severity of non-glandular ulcers in horses (Murray 1994a). Gastric ulceration was found in 75% of horses fed twice daily and in 57,9% horses fed 3 times a day (Feige et al. 2002). A recent study in Danish pleasure horses showed an increased risk in EGUS when forage feeding interval exceeded 6 hours (Luthersson et al. 2009b), suggesting that continuous forage feeding might be critical in preventing gastric ulceration.

3.3.4 High concentrate diet

Consumption of high concentrate diets causes a greater stimulus of gastric acid secretion than consumption of hay (Smyth et al. 1989). Serum gastrin concentrations are highest in horses fed a high concentrate diet. A grain diet is high in digestible carbohydrates, which are fermented by resident bacteria, resulting in the production of volatile fatty acids (VFA)) (Reese and Andrews 2009). In the presence of a low stomach pH, VFA can cause acid damage to the non-glandular squamous mucosa (Nadeau et al. 2003). Therefore a diet high in concentrates and low in fibre is thought to produce a poorly buffered, acidic environment in the stomach and predispose to gastric injury (Luthersson et al. 2009b). However, a recent study found that alfalfa hay and a grain diet had a higher stomach pH level and lower gastric ulcer scores than horses fed Brome grass without grains (Nadeau et al. 2000). The authors speculated that the high concentrations of calcium and protein in the alfalfa hay buffered stomach contents resulting in a protective effect of the non-glandular mucosa. Eating behaviour and the types of feed being consumed must be considered to have an influence on gastric acidity (Murray 1994b, Luthersson et al. 2009a).

3.3.5 Stall confinement

Stall confinement has been implicated as a risk factor for EGUS, however different studies show varying results (Murray and Eichorn 1996, Bell et al. 2007b). This might be because of other factors playing a role in stabled horse that increases the risk of EGUS (Videla and Andrews 2009). It is

important to remember that horses in stall confinement are also more often in training, they are also fed higher amounts of starch and the high prevalence of gastric ulceration may therefore be caused more by the diet (high in starch and therefore VLAs), than stall confinement itself (Radostits 2007). In a study by Bell et al (2007), there was no significant difference between stall confinement and pasture, as all the racehorses in this study had the same prevalence of gastric ulceration. Furthermore, differences in quality between pastures did not impact on the results in this study. However all the horses in this study where in training and were fed high amounts of grain, which might have confounded the results.

3.3.6 Non-steroidal anti-inflammatory drugs

Nonsteroidal anti-inflammatory drug (NSAIDs) administration can cause gastric ulcers (MacAllister et al. 1993). Both phenylbutazone and flunixin meglumine have been found to induce gastric ulcers in horses, but usually at higher-than-recommended doses (MacAllister et al. 1993). The cause is probably related to decreased blood flow causing blockage of prostaglandin synthesis, which causes deceased mucosal blood flow, stimulates gastric acid secretion and decreases bicarbonate secretion by glandular mucosa (Andrews and Nadeau 1999). Adequate mucosal blood flow is necessary to remove hydrogen ions that diffuse through the mucus layer (Videla and Andrews 2009). Ischemia of the gastric mucosa may lead to hypoxia-induced cellular acidosis and release of oxygen-free radicals, phospholipase, and proteases, which may damage the cell membrane leading to necrosis (Videla and Andrews 2009).

3.3.7 Bacteria

Helicobacter spp have been isolated from humans and animals affected by gastric ulceration (Fox 2002), but their role in equine gastric ulceration has not yet been proven (Videla and Andrews 2009). Once gastric ulceration is present, other bacteria have been implicated in inhibiting ulcer healing (Videla and Andrews 2009). The author speculates that bacterial colonization of gastric ulcers in the stomach may delay healing of the ulcers. This may be true, considering that bacteria such as *Escherichia coli* have been cultured from the stomach of horses (Al Jassim et al. 2005).

3.3.8 Stress

Physiological and psychological stress may be important determinants of gastric ulceration (Lester et al. 2008). Stress can induce gastric ulcers in horses due to an excessive release of endogenous corticosteroids that can inhibit prostaglandin synthesis. Decrease in prostaglandins leads to breakdown of mucosal protective factors and may be the cause of gastric ulceration in horses (Andrews and Nadeau 1999). Nervous temperament as a predisposing factor has been shown to be associated with gastric ulcers in different studies done with Standardbreds and Thoroughbreds (McClure et al. 1999, Dionne et al. 2003). One study hypothesised that anxiety could be associated with persistent tension of abdominal muscles which could mimic the reduction in gastric volume seen during exercise (*AM Merritt, personal communication)*(Lester et al. 2008). Physiologic stress (e.g. severe illness) has been proven to correlate with an increased incidence of peptic ulceration in foals (Furr et al. 1992).

3.4 Clinical signs

Clinical signs associated with equine gastric ulceration are commonly reported in the literature, but are vague and often unsubstantiated (Nadeau and Andrews 2009). Clinical signs of gastric ulceration in yearlings and mature horses are less well recognized than in foals, but may be more important economically (Murray 1994b, Andrews and Nadeau 1999). The most commonly reported clinical signs in the literature include poor performance, decreased appetite, poor body condition, poor hair coat, acute or recurrent colic, diarrhoea, bruxism and behavioural changes (Murray 1992a, Murray 1994b, Vastistas et al. 1999, Sandin et al. 2000). Early recognition of the clinical signs of gastric ulceration in horses would mean that treatment could be prescribed earlier and further complications prevented (Andrews and Nadeau 1999). Not all horses with equine gastric ulceration show clinical signs, and in the literature, horses with gastric ulcers can be divided into those with 'silent' or non-clinical ulcers and those with clinical ulcers (Andrews and Nadeau 1999).

It is suggested that gastric ulcers are in general more severe in horses with clinical signs compared to horses not showing clinical signs (Murray et al. 1989). In a previous study, horses with clinical signs had a prevalence of gastric ulceration of 86 % compared with 37% for horses without clinical signs (Murray et al. 1989). Few studies have been done to determine whether specific clinical signs are associated with the severity or location of gastric ulcers .

3.4.1 Abdominal pain

Abdominal pain usually leads to inappetance and consequently diminished feed intake (Sandin et al., 2000). Feed deprivation generally results in increased stomach acidity (Murray and Schusser 1993)(Murray 1994b).Therefore any condition that diminished food intake might hypothetically contribute to development of gastric ulceration (Sandin et al., 2000). Therefore, colic can be a primary cause of gastric ulcers or gastric ulcers may be secondary to problems in the gastrointestinal tract (Murray 1994a). A connection between intestinal lesions and gastric ulceration might also suggest that gastric ulceration is often part of a larger gastrointestinal disease complex (Sandin et al., 2000).

An association between colic and gastric ulceration has been demonstrated in a recent post-mortem study of Swedish horses (Sandin et al. 2000), however Rabuffo et al (2002) found little significance between colic and gastric ulceration when comparing horses with signs of colic to horses without. This was similar to the findings of Vastistas et al. (1999), who demonstrated only 6/167 (3.5%) of horses with gastric ulceration had signs of colic over the preceding month, leading them to suggest that colic might be of less significant as a clinical sign for gastric ulceration.

Results from a study by Dukti et al (2006) indicated that fewer horses that had surgically managed colic had gastric ulcers than horses that had been medically-managed gastric ulcers. The reason for this may be that medically treated horses are generally hospitalized for longer and may be starved for longer periods of time than horses that are surgically managed, although this is only speculation. The same study found a connection between duodenitis proximal jejunitis and gastric ulceration when compared with other gastric lesions. This might be because horses with duodenitis proximal jejunitis are more likely to have gastric ulcerations because they are not eating, often having a nasogastric tube or the disease itself may cause a gastritis.

3.4.2 Inappetance

Signs of inappetance may vary from mild to severe, which might be why it is often unrecognized by owners. In one study the owners where usually referring to decreased appetite as "fussy" eating, not considering it a clinical sign for EGUS (Luthersson et al. 2009a). Poor appetite was associated with poor body weight in one study (Lester et al. 2008). There were also some findings of diminished appetite correlating with excessive amounts of work. These problems where accentuated with the length of time in work when caloric demands was greater (Lester et al. 2008). Apparently ulceration

might limit feed intake and the horse will lose weight as a consequence, this might also contribute to reduced athletic performance (Lester et al. 2008). If the diagnosis is correct, signs of inappetance usually resolve within a few days after initiating treatment, and the horse is expected to gain weight within 2 to 3 weeks (Murray et al. 1989, Murray 1991).

3.4.3 Poor body condition and weight loss

Poor body condition has been reported to be associated with a high prevalence of gastric lesions in one study (Dionne et al. 2003). In some cases the owners had reported no signs of weight loss, but the authors had the impression that the horses had some weight loss and loss of appetite (Luthersson et al. 2009a, Luthersson et al. 2009b). In this same study there was no significant relationship between body scoring and EGUS. Another study of Thoroughbred racehorses found a positive relationship between difficulties of maintaining body weight and squamous mucosal ulceration (Lester et al. 2008). In a recent study, two broodmares with poor body condition had gastric glandular lesions, but due to the low number of horses there was no statistical significance in the results (le Jeune et al. 2009).

3.4.4 Changes in behaviour

Horses that demonstrated stereotypic or altered behaviour, specifically crib biting or wind sucking, were more likely to have ulcers than those that did not in a study done in Australia (Lester et al. 2008). Behavioural changes such as nervousness or aggression where often reported in this study, and often improved with antacid therapy. These results indicate that both physiological and psychological stress may be important determinants of equine gastric ulceration (Lester et al. 2008). An association between cribbing and gastric ulcers has also been reported but the mechanism is still not truly proven (Nicol et al. 2002). In a study done with nineteen young horses, the stomachs of the cribbing foals where significantly more ulcerated than the stomachs of the normal foals (Nicol et al. 2002). Antacid treatment resulted in significant improvement in the foals' stomachs and a decrease in the cribbing behaviour. In another study, horses without clinical signs but with endoscopic findings of gastric ulceration, showed great improvement behaviourally following treatment (Murray et al. 1989).

3.4.5 Diarrhoea

Diarrhoea is described as a vague clinical sign for gastric ulceration in foals (Becht and Byars 1986). Foals with gastrointestinal symptoms for example diarrhoea had a higher prevalence of having gastric ulceration than foals with other primary diseases (Elfenbein and Sanchez 2012). There is no evidence that gastric ulceration can cause diarrhoea in adult horses and is anatomically and physiologically implausible, except in the situation where gastric ulceration is part of a wider disease process involving the large colon of the horse.

3.4.6 Poor coat condition

Poor coat condition is usually listed as a vague clinical sign of gastric ulceration, but in a cross-sectional study done in Thoroughbred horses, there was a statistical association between gastric ulceration and rough hair coat (Vastistas et al. 1999). In contrast, numerous other studies have been done with no reports about rough hair coat as a clinical sign (Dionne et al. 2003, le Jeune et al. 2009). The mechanism of poor hair coat as a symptom of EGUS, could be due to decreased uptake of nutrition associated with EGUS.

3.4.7 Poor performance

Decreased performance has been suggested to be linked with gastric ulceration in many studies (Vastistas et al. 1999, Buchanan and Andrews 2003), and was recently proven in a study done on four Thoroughbred race horses with poor performance (Franklin et al. 2008). The horses had no abnormal findings other than EGUS found during a thorough investigation of all body systems (Franklin et al. 2008). After treatment with omeprazole, all horses showed considerable improvement in performance. This is the first report in which evidence is presented suggesting a direct link between EGUS and decreased performance, where other causes of poor performance have been excluded. However further investigations are needed because of the small study population. Excluding other factors that might influence poor performance is a challenge as is the determination of how much and at what grade gastric ulceration impacts on performance.

In the literature there are several reports about horses that where performing normally according to the owner, but with endoscopic evidence of gastric ulceration. These horses received treatment and according to the owners, improved in performance, illustrating how difficult it is to determine the normal level of performance for a horse and the influence that the ‘placebo effect’ may have on

interpretation (Murray et al. 1989, Buchanan and Andrews 2003). This shows how challenging it is to assess what level of performance is normal for a particular horse and how to measure and monitor it. How can we link poor performance to EGUS if we cannot truly determine the fact that a horse has poor performance? Clearly more research is needed in this area.

3.5 Diagnosis and treatment

3.5.1 Diagnosis

Gastroscopic examination is currently the only method for defining gastric ulceration, and assessing their severity and extent (Bell et al. 2007a). The procedure requires an approximately 2,5-3 m endoscope to allow visualization also of the pylorus (Buchanan and Andrews 2003, Bell 2007). The lesions should be scored, however there is no universal scoring system. Generally the lesions are classified both according to location and severity (Bell et al. 2007a). The Equine Gastric Ulceration Council has developed a scoring system ranging from 0-4 (Anonymous 1999).

Currently there are no haematological or biomechanical markers available to aid in diagnosis of gastric ulceration (Vastistas et al. 1999). There are studies published about evaluating sucrose concentrations in urine (O'Conner et al. 2004) and in blood (Hewetson et al. 2006) after administration via nasogastric sucrose. The method is based on the fact that sucrose penetrate through damaged gastric or intestinal mucosal and its possible to detect sucrose in serum or urine that correlates with the severity of the damaged gastric mucosa (Bell et al. 2007a). The urine sucrose test was recently reported reliable but due to technical difficulties with collecting urine the practical value of the test is less valuable for general practitioner (Hewetson et al. 2006, Bell et al. 2007a). The use of sucrose concentrations in serum has the advantages of being easier to collect, and according to the pilot study done correlates well with the severity of gastric ulceration (Hewetson et al. 2006). Empiric treatment may be an option if current diagnostic tools are not available, however treatment is expensive and if the horse doesn't respond to treatment, referral to a facility with possibilities to perform a gastroscopic examination is indicated (Buchanan and Andrews 2003).

To determine whether gastric ulceration is the primary cause and not a response to something else, four criteria have to be fulfilled: (1) first endoscopic confirmation that there is gastric ulceration; (2) there are no other health abnormalities; (3) the horse needs to respond to treatment that effectively suppresses or neutralizes gastric acidity; and (4) there needs to be confirmed that the healing of the gastric ulcers is improving or completely healed (Radostits 2007).

3.5.2 Treatment

The goals of gastric ulcer treatment are to relieve pain, promote healing, eliminate clinical signs, prevent secondary complications and prevent recurrence (Buchanan and Andrews 2003). Some ulcers heal spontaneously, but more severe ones usually need treatment, especially if the horse is to remain in athletic training (Rabuffo et al. 2002). There are many different ways of treating gastric ulcers but the primary one is acid suppressive therapy, which changes the stomach's pH and allows ulcer healing (Buchanan and Andrews 2003). There are many different pharmacological agents available on the market but only a few have shown to be effective in treatment of gastric ulcers.

3.5.2.1 Proton pump inhibitors

The most effective proton pump inhibitor on the equine pharmaceutical market is omeprazole, a substituted benzimidazole (Johnson et al. 2001). In an acid environment, omeprazole is activated to a sulfonamide derivative and binds irreversibly to the parietal cells inhibiting the transport of hydrogen ions into the stomach (Vatistas et al. 1999). A consequence of this irreversible binding is that the inhibiting effects are prolonged, making once- daily dosing possible (Vatistas et al. 1999). A recent study done about the efficiency of this drug reported an improvement in 94 % of the horses and complete healing in 65 % of the horses after 28 days of oral omeprazole treatment at 4 mg/kg/day (Johnson et al. 2001).

3.5.2.2 H2 receptor antagonists

These drugs act by blocking the interaction of histamine with H2- receptors on the parietal cells and thus decreases the basal secretion of HCl. H2-receptor antagonists available for use in horses include ranitidine, cimetidine, nizatidine and famotidine. Ranitidine is the most frequently used but has to be administered PO every 8 hours. In a study done comparing omeprazole with ranitidine, the improvement after omeprazole treatment was significantly greater than with ranitidine (Lester et al. 2005).

3.5.2.3 Coating and binding agents

Sucralfate is a compound that binds to stomach ulcers and promote healing. Sucralfate increases the bicarbonate secretion, stimulates the prostaglandin production and therefore helps buffering HCL. In a clinical trial in horses oral sucralfate did not improve subclinical ulcers healing in 6- and 7-month-old foals. Sucralfate used alone for treatment of EGUS might not be effective but can be used in conjuction with acid-suppressive theraphy (Borne and MacAllister 1993).

Many other drugs and methods have been tested and suggested as a treatment for gastric ulceration, however none has proven to be effective or practical enough (Buchanan and Andrews 2003, Bell et al. 2007a).

The prevention and dietary management are equally important prior to treatment. Providing constant access to alfalfa or good-quality hay helps to raise the gastric pH (Murray and Eichorn 1996).

4 STUDY OBJECTIVE

The objective of this study was to determine if there is a statistical association between commonly reported clinical signs of gastric ulceration (as perceived by owners) and gastric ulcer severity (as determined by endoscopic examination of the stomach).

Hypothesis: There is no association between the severity of gastric ulceration and the owners' perception of clinical signs.

5 MATERIALS AND METHODS

5.1 Descriptive/Patient data

One hundred adult horses were included in this study (42 mares, 54 geldings and 4 stallions) with an age range from 1,5-21 years. Breeds included 44/100 Warmbloods, 21/100 Finnhorses, 18/100 Standardbreds, 5/100 ponies, 2/100 Oldenburg horses, 2/100 Tori horses, 2/100 Trakhener horses, 1/100 Arab, 1/100 Cold blooded horse, 1/100 Islandic horse, 1/100 Shetland pony, 1/100 Thouroughbred, 1/100 Quarter horse Islandic horses and 1/100 Quarter horse.

The horses in this study where used for various reasons, including racing, breeding, showjumping, dressage, riding school and pleasure riding. Five horses where owned by Ypäjä Equine college. The other horses were privately owned. Most horses where examined by request of the owner, some horses where examined by request from the treating veterinarian, for example as a follow up for colic.

For each horse, the person filling in the questionnaire had been the primary caretaker of the horse for at least 3 months prior to the endoscopic examination. There was a large variation in these respondents; professional race trainers, students or employees of the equestrian college, high-level competition riders and pleasure horse owners.

During the endoscopic examination, the study horses were given a body condition score (0-5) by the examining clinician (see appendix 2).

5.2 Questionnaire

The questionnaire was based on potential clinical signs of gastric ulceration that have been described in the literature. These included colic, inappetance, crib-biting, flank-biting, selective or ”fussy” eating, body condition and weight loss, changes in behaviour, chronic diarrhoea, bruxism, poor coat condition and decreased performance. Each question had a "Yes" or "No" answer, and based on the answer, there were then additional questions regarding the subject (see appendix 1). The owners were asked to fill out the questionnaire during the gastroscopic examination. If that was not possible the questionnaires where sent to the owners shortly afterwards by post. All questionnaires where filled in within a two year period (2012-2013) from when the data was collected.

5.3 Endoscopic examination

The owners where asked to starve the horses at home for 16 hours and the water should have been withdrawn for 6 hours before the gastroscopic examination. The horses where confined in stocks and all horses where sedated using a combination of detomidine and butorphanol. A nasal twitch was used as a restraint. A lubricated plastic tube was passed into the ventral meatus of the nasal cavity, into the oesophagus and secured with tape to the halter. The endoscope could then safely be

passed into the oesophagus and through the cardiac sphincter into the stomach. In some cases a mouth gag was used instead of a lubricated plastic tube to prevent the horse from being able to chew the endoscope in the event of retroflexion of the endoscope into the oral cavity as it was passed through the nasopharynx. The stomach was insufflated with air until the stomach was distended. Gastric contents were rinsed off the mucosa by flushing water through the endoscopy biopsy channel. The fundus was visualised, then the margo plicatus was viewed along the greater and lesser curvatures of the stomach. Then the pylorus was viewed and the endoscope was then advanced through pylorus and the duodenum was visualized. Video footage was recorded for each gastroscopic examination performed and was later used to document the presence and grade the severity of gastric ulceration in each horse. The gastroscopic examinations were performed by experienced clinicians at the Helsinki University Equine Hospital or at Ypäjä Equine Clinic between 2012-2013.

5.4 Grading the gastric ulcers

Gastric ulcers where graded into four categories; 1) presence or absence of gastric ulcers; 2) presence or absence of clinical significant gastric ulcers (i.e. needing treatment or not); 3) presence or absence of glandular ulcers; and 4) presence or absence of non-glandular ulcers.

The aim of this study was to determine the significance of the ulcers and therefor grading ulcers according to the perceived need for treatment seemed to be the most logical and descriptive. Clinical significance was determined by an experienced clinician who assessed each video and determined whether in his opinion, the horse would require treatment or not. Horses that required treatment were scored as having clinically significant ulcers whereas horses that did not require treatment were scored as having clinically insignificant ulcers. We also wanted to differentiate between glandular ulcers and non-glandular ulcers, which meant that there were four categories being compared to the clinical signs. This was done because with the current EGUS scoring system, there is no means of differentiating between glandular and non-glandular lesions, which may have a bearing on the results of the study.

The anatomical regions of the stomach that where assessed included the dorsal part of the fundus, the squamous epithelium from the right side of the stomach along the margo plicatus (MPRT), the greater curvature along the margo plicatus (MPGC), the lesser curvature along the margo plicatus (MPLC), the pylorus, and the duodenum (Fig 2).

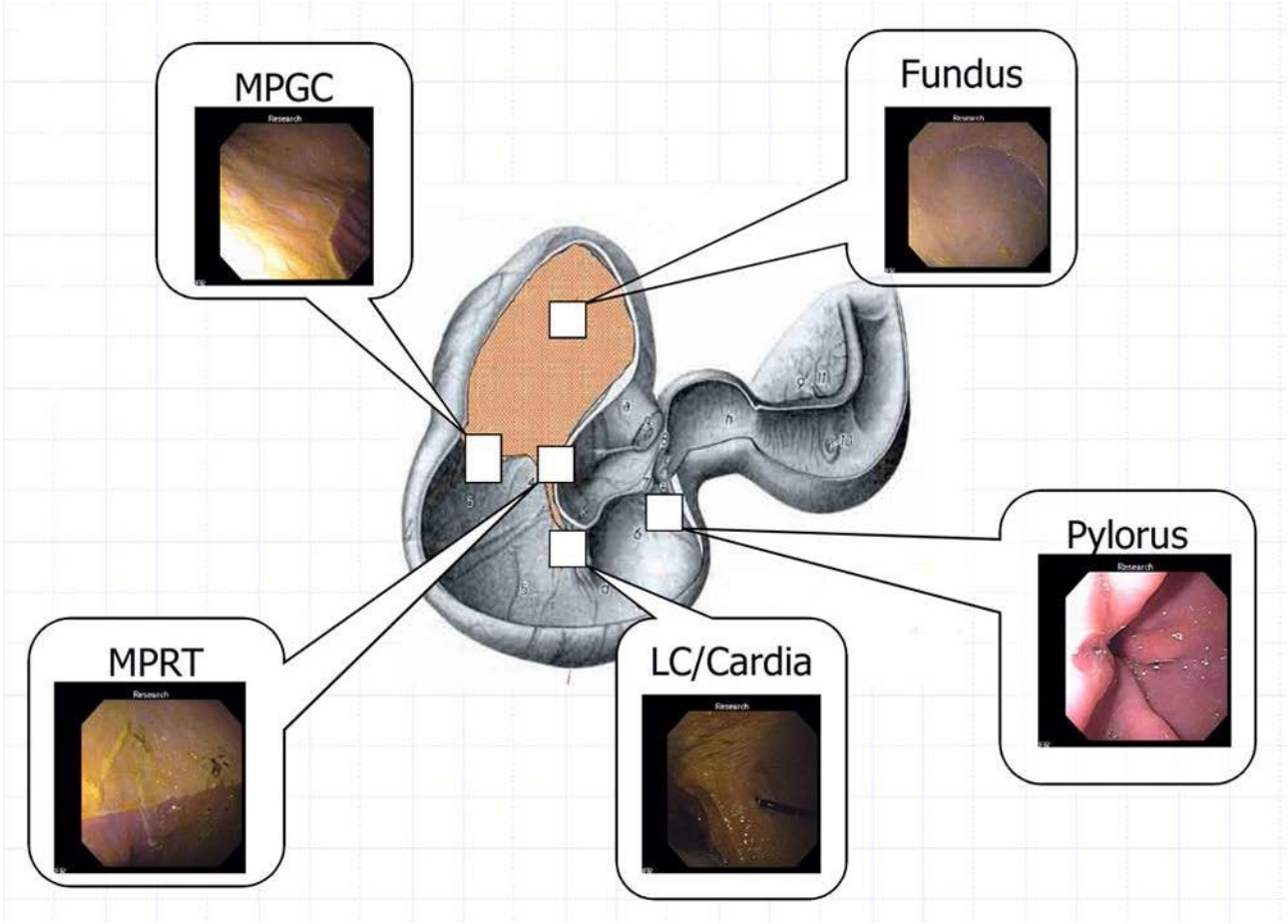


Figure 2: Anatomical regions of the stomach used for ulcer grading.

5.5 Statistical analyses

The Mann-Whitney U-test was used to determine if there was a statistically significant difference between 1) presence or absence of gastric ulcers; 2) presence or absence of clinical significant gastric ulcers (i.e. needing treatment or not); 3) presence or absence of glandular ulcers; and 4) presence or absence of non-glandular ulcers when compared to body score from 0-5. All the other clinical signs where compared to these four categories using crosstabs and the p-value of the Pearson Chi-Square. Six horses where left out from the glandular ulcer category because the pylorus was not visible in the video.

6 RESULTS

6.1 Table of results

Clinical signs	Number of reported cases		1.Presence or absence of gastric ulceration		2. Clinical significant ulcer		3. Presence or absence of glandular lesions		4. Presence or absence of non-glandular lesions		P-value (Crosstabs, Pearson Chi-square)			
	yes	no	yes	no	yes	no	yes	no	yes	no	1	2	3	4
1. Abdominal pain	28	72	21	7	17	11	20	8	11	17	0,64	0,184	0,441	0,64
2. Inappetance	46	54	38	8	25	21	33	9	22	24	0,621	0,923	0,684	0,368
3. Cribbing	10	90	8	2	5	5	6	4	6	4	0,637	0,79	0,19	0,252
4. Flank-biting	13	87	12	1	9	4	10	3	4	9	0,34	0,338	0,976	0,34
5. Fussy eating	40	60	35	5	26	14	30	8	18	22	0,187	0,328	0,657	0,742
6. Losing weight	45	55	41	4	32	13	36	7	23	22	0,01	0,051	0,134	0,138
7. Behavioural changes	58	42	48	10	34	24	41	16	22	36	0,7	0,94	0,185	0,229
7a. Tired	34	66	27	7	20	14	24	10	10	24	0,791	0,493	0,3	0,49
7b. Laying more	6	94	5	1	4	2	4	2	3	3	0,622	0,982	0,553	0,721
7c. More aggressive against humans	34	66	28	6	19	15	25	9	15	19	0,871	0,902	0,597	0,871
7d.Aggressive against horses	12	88	9	3	6	6	8	4	4	8	0,602	0,432	0,384	0,471
7e. More nervous	32	68	5	27	22	10	25	6	13	19	0,103	0,802	0,515	0,742
8. Diarrhoea	32	68	25	7	17	15	22	10	12	20	0,591	0,373	0,197	0,446
9a. Bruxism	7	93	6	1	5	2	6	1	3	4	0,424	0,843	0,554	0,994
9b.Bruxism	16	84	13	3	10	6	13	3	6	10	0,628	0,839	0,629	0,628
10a. Poor coat Condition (shedding)	27	73	24	3	14	13	20	5	9	18	0,527	0,34	0,639	0,235
10b. Poor coat Condition (losing hair)	10	90	9	1	8	2	7	2	5	5	0,121	0,539	0,93	0,637
11. Poor performance	47	53	41	6	28	19	36	10	19	28	0,624	0,288	0,709	0,624

Mann-Whitney U-test:
Poor body condition: P1=0,678; P2=0,868; P3=0,920; P4=0,201

6.2 Poor body condition and weight loss

Weight loss was listed as a clinical sign for 45/100 horses. Of these, 32/100 had clinical significant ulcers. There was a significant association between having a clinical significant gastric ulcer and weight loss (p=0,01). This result supports previous reports about losing weight as a clinical sign of gastric ulceration in the literature (Anonymous 1999, Bell et al. 2007a). There was also an

association between the presence of a gastric ulcer and weight loss (p= 0,51). There was no significant association between weight loss and 1) presence or absence of glandular lesions (p=0,134); and 2) presence or absence of non-glandular lesions (p=0,138).

There was no association found between poor body score and 1) presence or absence of gastric ulceration (p=0,678); 2) clinical significant ulcer (p=0,868); 3) presence or absence of glandular lesions (p=0,920); and 4) presence or absence of non-glandular lesions (p=0,201).

6.3 Abdominal pain

Of the horses in this study 28/100 had demonstrated abdominal pain as a clinical sign for the 3 months prior to the gastroscopic examination. Fourteen out of one hundred of these 28 horses had had more than one episode of colic.
Seventeen out of one hundred of these horses had clinical significant gastric ulcer scores. There was no significant association between abdominal pain and 1) presence or absence of gastric ulcers (p=0,184); 2) clinically significant gastric ulceration (p=0,64); 3) presence or absence of glandular ulcers (p=0,441); and 4) presence or absence of non-glandular ulcers (p=0,64).

6.4 Inappetance

Inappetance was reported as a clinical sign in 46/100 of the horses by owners. Of these, 25/100 horses had clinical significant ulcers. There was no significant association between inappetance and 1) presence or absence of gastric lesions (p=0,923); 2) clinically significant gastric ulceration (p=0,621); 3) presence or absence of glandular ulcers (p=0,684); and 4) presence or absence of non-glandular ulcers (p=0,368).

6.5 Changes in behaviour

Fifty-eight out of one hundred horses where reported to have changes in behaviour of these 34/100 where found to have clinical significant ulcerations. This was the most reported of all the clinical signs listed in this study. There was no significant association between behavioural changes and 1) presence or absence of gastric ulcers (p=0,94) ; 2) clinically significant gastric ulceration (p=0,7); 3) presence or absence of glandular ulcers (p=0,185); and 4) presence or absence of non-glandular ulcers (p=0,229).

6.6 *Diarrhoea*

Thirty-two out of one hundred horses were reported to have diarrhoea as a clinical sign, of these 17/100 horses had clinical significant gastric ulceration. There was no association found between diarrhoea and 1) presence or absence of gastric ulcers (p=0,373); 2) clinically significant gastric ulceration (p=0,598); 3) presence or absence of glandular ulcers (p=0,197); and 4) presence or absence of non-glandular ulcers (p=0,446).

6.7 *Poor coat condition*

Twenty-seven out of one hundred horses had reported clinical signs such as problem shedding or rough hair coat, of these 14/100 had clinical significant changes found in the gastroscopy. There was no association found between poor hair coat and 1) presence or absence of gastric ulcers (p(a/b)=0,34/0,539); 2) clinically significant gastric ulceration (p(a/b)=0,527/0,121)); 3) presence or absence of glandular ulcers (p(a/b)=0,639/0,93); and 4) presence or absence of non-glandular ulcers (p(a/b)=0,235/0,637).

6.8 *Poor performance*

The second largest group of clinical signs reported after changes in behaviour was poor performance with 47/100 horses, of these 28/100 had clinical significant findings. There was no association found between poor performance and 1) presence or absence of gastric ulcers (p=0,288); 2) clinically significant gastric ulceration (p=0,624); 3) presence or absence of glandular ulcers (p=0,709); and 4) presence or absence of non-glandular ulcers (p=0,624).

6.9 *Other clinical signs*

Ten out of one hundred horses showed signs of cribbing, 5/100 had clinical significant ulcers. There was no association found between cribbing and 1) presence or absence of gastric ulcers (p=0,79); 2) clinically significant gastric ulceration (p=0,637); 3) presence or absence of glandular ulcers (p=0,19); and 4) presence or absence of non-glandular ulcers (p=0,252).

Thirteen out of one hundred horses in this study showed signs of flank-biting, 9/100 had clinical significant ulcers. There was no association found between flank biting and 1) presence or absence of gastric ulcers (p=0,338); 2) clinically significant gastric ulceration (p=0,34); 3) presence or absence of glandular ulcers (p=0,976); and 4) presence or absence of non-glandular ulcers (p=0,34).

Fussy eating as in dropping food, taking pauses during eating and eating slowly was reported as a clinical sign for 40/100 horses, of these 26/100 had clinical significant ulcers. There was no association found between fussy eating and 1) presence or absence of gastric ulcers (p=0,328); 2) clinically significant gastric ulceration (p=0,187); 3) presence or absence of glandular ulcers (p=0,657); and 4) presence or absence of non-glandular ulcers (p=0,742).

Seven out of one hundred horses had been listed for bruxism, of these 5/100 had clinical significant findings. There was no association found between bruxism and 1) presence or absence of gastric ulcers (p(a/b)=0,424/0,839); 2) clinically significant gastric ulceration (p(a/b)=0,843/0,628); 3) presence or absence of glandular ulcers (p(a/b)=0,554/0,629); and 4) presence or absence of non-glandular ulcers (p(a/b)=0,994/0,628).

7 DISCUSSION

This study relies completely on the ability of the owners to assess their horses behaviour and presence of clinical signs. These owners were not trained professionals, and this should be kept in mind when comparing the results of this study with the results of studies done in which veterinarians have assessed the clinical signs.

7.1 Clinical signs

7.1.1 Poor body condition and weight loss

In this study there was a strong association between the presence of a clinical significant ulcer and weight loss. There was also an association found between the presence of an ulcer and weight loss. There was no association found between poor body condition and gastric.

In the pilot study (Sulku 2012) there was a correlation between nonglandular gastric ulceration and poor body score which is also supported by previous studies (Anonymous 1999, Buchanan and Andrews 2003, Dionne et al. 2003, Bell et al. 2007a). In this study the body condition score was compared to all four categories of scoring but no association was found. The study horses were given a body score (0-5) by the examining clinician during the endoscopic examination. The owners were also asked to evaluate the body condition of their horse

in the questionnaire. The results given by the examining clinician were used in this study. Body scoring is challenging for a non-professional and they tended to slightly underestimate their horses body condition. This might explain the different result compared to the pilot study.

One can debate about how specific weight loss as a sign for one specific disease is, as there are a lot of factors affecting weight loss. Also interesting as a side comment is that this weight loss tended to be fast and distinctive especially in Standardbred racehorses in training. This might be explained with the fact that these horses lose a lot of energy during training and the weight loss will be faster in these cases. It has to be kept in mind that there was no record of the horses previous weight 3 months prior to the gastroscopic examination, therefore the fact that the horse had lost weight was completely based on the owners subjective opinion.

7.1.2 Abdominal pain

There was no association found between colic and gastric ulceration in this study. Colic as a sign of gastric ulcerationis reported in many studies (Sandin et al. 2000) but there have also been some that have found no connection between them (Rabuffo et al. 2002). Gastric ulceration as a secondary cause of colic due to food deprivation has been reported (Sandin et al. 2000). Because of the questionnaire design, mild signs of colic such as stretching, flehmen and standing in a urination position might have gone unnoticed, as they where not listed.

7.1.3 Inappetance

There was no clinical significant association found between inappetance and gastric ulceration in this study. This is a clinical sign that is hard to monitor as there are various different feeding routines in every stable, therefore inappetance could be under or over reported. Some of the horses in this study had food available all the time while others were only given food three times a day. Furthermore, some of the owners reported that there was a single episode of inappetance.This was associated with the colic episode for which the horse had been brought to the hospital. The results relating to inappetance should therefore be interpreted with caution.

7.1.4 Changes in behaviour

There was no association found between change in behaviour and gastric ulceration, even though this was the single most commonly reported clinical sign. This was a little surprising considering previous reports (Anonymous 1999, Buchanan and Andrews 2003, Sulku 2012).
Various changes in behaviour such as aggressiveness (especially when tightening the girth), bucking, depression and altered behaviour as cribbing and wind sucking have been described as clinical signs for gastric ulceration, and in many cases, these behavioural changes have improved with antacid treatments (Murray et al. 1989, Nicol et al. 2002, Lester et al. 2008).

Behavioural change was the most commonly reported clinical sign by the owners in this study. These horses where mostly behaving differently during riding, refusing to jump, spooking and showing reluctance to move. The majority of these horses where later referred forward for lameness examination after no ulcers were found during gastroscopy. Most of these horses where later found to have either back or leg problems. The owners likelihood for suspecting gastric ulceration as an explanation for the changes in behaviour is a common phenomena at the clinic. This might be as a consequence of the reported studies associating behavioural changes with gastric ulceration. Furthermore, owners seem to prefer the diagnosis of gastric ulceration rather than lameness, which is why they request the gastric examination prior to having the horse examined for lameness. Looking at the results of this study as a professional, it might be logical to consider an orthopaedic examination first in horses that present with these behavioural changes prior to considering gastroscopy.

7.1.5 Diarrhoea

Diarrhoea was reported quite frequently in this study (32/100 horses), however no association was found between diarrhoea and gastric ulceration. Most of these horses had mild intermitted diarrhoea.

There are no evidence in the literature that gastric ulceration can cause diarrhoea in adult horses and is anatomically and physiologically implausible, except perhaps in the situation where gastric ulceration is part of a wider disease process involving the large colon of the horse. This might explain the results in a study done on foals where there was an association found between diarrhoea and gastric ulceration (Elfenbein and Sanchez 2012).

7.1.6 Poor coat condition

There was no association found between gastric ulceration and poor coat condition. This have been reported as a clinical sign of gastric ulceration in previous studies (Anonymous 1999, Vastistas et al. 1999). But also numerous other studies have been done with no reports about rough hair coat as a clinical sign (Dionne et al. 2003, le Jeune et al. 2009).
Poor coat condition might be hard to assess in the cold climate in Finland where most horses are clipped during the winter season to be able to still train them. This might have resulted in fewer complainst about poor hair coat, leading to an underrepresentation of this clinical sign in the results.

7.1.7 Poor performance

There was no association found between gastric ulceration and poor performance. Several studies have found evidence of poor performance being associated with gastric ulceration (Anonymous 1999, Vastistas et al. 1999, Buchanan and Andrews 2003, Jonsson and Egenvall 2006, Franklin et al. 2008). Therefore there could have been an expectation to find an association between poor performance and gastric ulceration. Excluding other factors that might influence on poor performance is a challenge as is the determination of how much and at what grade gastric ulceration impacts on the performance, this might be one of the hardest clinical signs to assess, especially for a non-professional person.

The most realistic results have most likely been received from racehorse trainers, as these people have the chance of comparing their horses to other horses of the same breed, age and use. Also the staff at Ypäjä equine college have the knowledge and possibilities to compare their horses to horses of similar age and training. Owners of show jumping and dressage horses seldom have the opportunities to compare their horses with other horses the same age at the same level. They might also have false expectations on how much the horse is able to perform, and therefore might misjudge the level of performance.

7.1.8 Other clinical signs

There was no association found between any of the other clinical signs reported and gastric ulceration. Various clinical signs such as flank-biting, cribbing and bruixism where also asked in the questionnaire. This study was not focusing on these signs but they where not left out completely, there where still statistics performed on these. These results conflicts with previous

results, where there had been associations found between cribbing and gastric ulceration in young horses and foals (Nicol et al. 2002). A possible explanation for the different results might be that the study was done using young horses and foals whereas this study only focused on adult horses.

It also seemed that a lot of the owners had a hard time assessing the severity of for example flank-biting. None of the horses in this study where biting so hard that they actually caused any damage to the skin. Fussy eating has often been reported in the literature as a sign of gastric ulceration (Murray et al. 1989, Buchanan and Andrews 2003, Bell et al. 2007a). However most of the owners do not spend enough time with their horse daily to truly be able to assess the eating behaviour. Most of the horses had had regular dental work done but the teeth where not checked during the examinations.

7.2 Limitations of the study

7.2.1 The respondents to the questionnaire

The owners where asked to fill in the questionnaire either before the gastroscopic examination or if not possible then after. None of the owners received the questionnaire later than 2 months after the examination. The fact that some of the owners knew the results and some didn't might affect their ability to answer the questionnaire.

It should also be kept in mind that most of the owners where not professionals. There was a wide variety of respondents, from professional trainers of racing horses to people owning their first horse. The most reliable answers where probably from the staff at Ypäjä horse college, where the horses are being watched by professionals daily. The trainers of the racing horses might be considered highly reliable, as the horses held at big training facilities are being closely monitored daily and they are being compared frequently with horses similar to age and breed. However most owners owning a show jumping, dressage or pleasure horse might only see the horse a few hours a day if even that, which might make it hard to assess signs as for example fussy eating. These owners are likely to have the most variation in experience of the owners, ranging from people owning their first horse to experience riders. Therefore it is possible to expect an underreporting of clinical signs from these owners. However, this situation mirrors the reality of practice, and therefore the results are still valid when taken into context of what we expect as clinicians in a general equine practice setting.

7.2.2 Study population

The study population consisted of a large variety of breeds, representing the horse population in Finland well. The horses were used for the main equestrian sports found in Finland: Standardbred racing; Coldblood racing; dressage; show jumping; pleasure riding; riding school; breeding and training young horses. What sets this population apart from most populations in which similar studies have been done is the lack of Thoroughbreds. In this study there was only one. Also stallions and ponies where underrepresented in this study. It is also good to keep in mind that Coldblood-type horses react different to pain than Warmblood-type horse which might make it more difficult to assess for example signs of colic in a Coldblood-type horse. This might lead to the fact that some signs are being underreported.

7.2.3 Questionnaire

The questionnaire was used in the pilot study and the questions where formed according to what seemed to be the easiest way for the owners to assess their horses (Sulku 2012). The questionnaire included questions about: abdominal pain; inappetance; crib-biting; flank-biting; fussy eating; body condition and weight loss; behaviour changes; diarrhoea; bruxism; poor coat condition and poor performance. In this study we decided to focus on the signs that where more frequently showed in the pilot study: colic; inappetance; fussy eating; body condition and weight loss; behaviour changes; diarrhoea; poor coat condition and poor performance. However the other ones where not left out, the statistical analyzes where still performed on these signs. The results are found under the section "Other clinical signs".

7.2.4 Grading the ulcers

The grading of the gastric ulcerations was done by a single experienced veterinarian. In order to get the right grading of what is a clinical significant gastric ulceration it would have been good to have a few more experienced veterinarians grading the ulcers and then assess the inter- and intra-observer agreement to validate the scoring system. That way the subjective assessment would have been minimized. However we got a positive p-value both in the category clinical significant ulcer and having an ulcer when compared to losing weight using crosstab. Therefore this is unlikely to have had an impact on the results of this study.

8 CONCLUSION

The results of this study suggest that there is a strong association between clinical significant gastric ulceration and weight loss (p=0,01) and the presence of a gastric ulcer and weight loss (p=0,051).This might be of great value for veterinarians in their every day work at the clinic. In conclusion it appears that an owners perception of the horse losing weight could be associated with the presence of a gastric ulcer and an increased severity of gastric ulceration and can be used as an indication to perform gastroscopy.

There was no association between gastric ulcer severity and the owners perception of colic, crib-biting, flank-biting, fussy eating, changes in behaviour, chronic diarrhoea, bruxism, poor body condition, poor coat condition and poor performance in their horses and requests from owners to have gastroscopy performed on their horses based upon these clinical signs should be approached with some caution.

The results of this study should however be approached with caution due to the fact that it is all based on the oweners ability to assess clinical signs. However this study is a good marker of circumstances out in the field, where we as veterinarians are often required to make a health assessment based on the owners observation skills .

9 REFERENCES

Al Jassim, R.A., Scott, P.T., Trebbin, A.L., Trott, D. & Pollitt, C.C. 2005. The genetic diversity of lactic acid producing bacteria in the equine gastrointestinal tract. *FEMS Microbiology Letters* 248: 75-81.

Andrews, F.M. & Nadeau, J.A. 1999. Clinical syndromes of gastric ulceration in foals and mature horses. *Equine Veterinary Journal* 31: 30-33.

Anonymous, 1999. Recommendations for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS): The Equine Gastric Ulcer Council. *Equine Veterinary Education* 11: 262-272.

Becht, J.L. & Byars, T.D. 1986. Gastroduodenal ulceration in foals. *Equine Veterinary Journal* 18: 307-312.

Bell, R.J. 2007. A comparison of two scoring systems for endoscopic grading of gastric ulceration in horses. 55: 19-22.

Bell, R.J., Mogg, T.D. & Kingston, J.K. 2007a. Equine gastric ulcer syndrome in adult horses: a review. *New Zealand Veterinary Journal* 55: 1-12.

Bell, R.J.W., Kingston, J.K., Mogg, T.D. & Perkins, N.R. 2007b. The prevalence of gastric ulceration in racehorses in New Zealand. *New Zealand Veterinary Journal* 55: 13-18.

Bezdekova, B., Jahn, P. & Vyskocil, M. 2007. Pathomorphological study on gastroduodenal ulceration in horses: localisation of lesions. *Acta Veterinaria Hungarica* 55: 241-249.

Borne, A.T. & MacAllister, C.G. 1993. Effect of sucralfate on healing of subclinical gastric ulcers in foals. *Journal of the American Veterinary Medical Association* 202: 1465-1468.

Buchanan, B.R. & Andrews, F.M. 2003. Treatment and prevention of equine gastric ulcer syndrome. *The Veterinary Clinics of North America.Equine Practice* 19: 575-597.

Campbell-Thompson, M.L. & Merritt, A.M. 1987. Effect of ranitidine on gastric acid secretion in young male horses. *American Journal of Veterinary Research* 48: 1511-1515.

Campbell-Thompson, M.L. & Merritt, A.M. 1990. Basal and pentagastrin-stimulated gastric secretion in young horses. *The American Journal of Physiology* 259: R1259-66.

Dionne, R.M., Vrins, A., Doucet, M.Y. & Pare, J. 2003. Gastric ulcers in standardbred racehorses: prevalence, lesion description, and risk factors. *Journal of Veterinary Internal Medicine / American College of Veterinary Internal Medicine* 17: 218-222.

Elfenbein, J.R. & Sanchez, L.C. 2012. Prevalence of gastric and duodenal ulceration in 691 nonsurviving foals (1995-2006). *Equine Veterinary Journal.Supplement* (41): 76-79.

Feige, K., Furst, A. & Eser, M.W. 2002. Effects of housing, feeding and use on equine health with emphasis on respiratory and gastrointestinal diseases. *Schweizer Archiv Fur Tierheilkunde* 144: 348-355.

Fox, J.G. 2002. The non-H pylori helicobacters: their expanding role in gastrointestinal and systemic diseases. *Gut* 50: 273-283.

Franklin, S.H., Brazil, T.J. & Allen, K.J. 2008. Poor performance associated with equine gastric ulceration syndrome in four Thoroughbred racehorses. *Equine Veterinary Education* 20: 119-124.

Furr, M., Taylor, L. & Kronfeld, D. 1994. The effects of exercise training on serum gastrin responses in the horse. *The Cornell Veterinarian* 84: 41-45.

Furr, M.O., Murray, M.J. & Ferguson, D.C. 1992. The effects of stress on gastric ulceration, T3, T4, reverse T3 and cortisol in neonatal foals. *Equine Veterinary Journal* 24: 37-40.

Hammond, C.J., Mason, D.K. & Watkins, K.L. 1986. Gastric ulceration in mature thoroughbred horses. *Equine Veterinary Journal* 18: 284-287.

Hewetson, M., Cohen, N.D., Love, S., Buddington, R.K., Holmes, W., Innocent, G.T. & Roussel, A.J. 2006. Sucrose concentration in blood: a new method for assessment of gastric permeability in horses with gastric ulceration. *Journal of Veterinary Internal Medicine / American College of Veterinary Internal Medicine* 20: 388-394.

Husted, L., Sanchez, L.C., Olsen, S.N., Baptiste, K.E. & Merritt, A.M. 2008. Effect of paddock vs. stall housing on 24 hour gastric pH within the proximal and ventral equine stomach. *Equine Veterinary Journal* 40: 337-341.

Johnson, J.H., Vatistas, N., Castro, L., Fischer, T., Pipers, F.S. & Maye, D. 2001. Field survey of the prevalence of gastric ulcers in Thoroughbred racehorses and on response to treatment of affected horses with omeprazole paste. *Equine Veterinary Education* 13: 221-224.

Jonsson, H. & Egenvall, A. 2006. Prevalence of gastric ulceration in Swedish Standardbreds in race training. *Equine Veterinary Journal* 38: 209-213.

le Jeune, S.S., Nieto, J.E., Dechant, J.E. & Snyder, J.R. 2009. Prevalence of gastric ulcers in Thoroughbred broodmares in pasture: a preliminary report. *Veterinary Journal (London, England : 1997)* 181: 251-255.

Lester, G.D., Robertson, I. & Secombe, C. 2008. Risk Factors for Gastric Ulceration in Thouroughbred Racehorses. *Risk Factors for Gastric Ulceration in Thoroughbred Racehorses* .

Lester, G.D., Smith, R.L. & Robertson, I.D. 2005. Effects of treatment with omeprazole or ranitidine on gastric squamous ulceration in racing Thoroughbreds. *Journal of the American Veterinary Medical Association* 227: 1636-1639.

Lorenzo-Figueras, M. & Merritt, A.M. 2002. Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses. *American Journal of Veterinary Research* 63: 1481-1487.

Luthersson, N., Nielsen, K.H., Harris, P. & Parkin, T.D. 2009a. The prevalence and anatomical distribution of equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine Veterinary Journal* 41: 619-624.

Luthersson, N., Nielsen, K.H., Harris, P. & Parkin, T.D. 2009b. Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine Veterinary Journal* 41: 625-630.

MacAllister, C.G., Morgan, S.J., Borne, A.T. & Pollet, R.A. 1993. Comparison of adverse effects of phenylbutazone, flunixin meglumine, and ketoprofen in horses. *Journal of the American Veterinary Medical Association* 202: 71-77.

McClure, S.R., Glickman, L.T. & Glickman, N.W. 1999. Prevalence of gastric ulcers in show horses. *Journal of the American Veterinary Medical Association* 215: 1130-1133.

Merritt, A.M. 1999. Normal equine gastroduodenal secretion and motility. *Equine Veterinary Journal* 31: 7-13.

Murray, M. 1991. Diagnosing and treating gastric ulcers in foals and horses. Diagnosing and treating gastric ulcers in foals and horses. *Veterinary Medicine.* p. 820-827.

Murray, M.J. 1992a. Gastric ulceration in horses: 91 cases (1987-1990). *Journal of the American Veterinary Medical Association* 201: 117-120.

Murray, M.J. 1992b. Review Article: Aetiopathogenesis and treatment of peptic ulcer in the horse: a comparative review. *Equine Veterinary Journal* 24: 63-74.

Murray, M.J. 1994a. Equine model of inducing ulceration in alimentary squamous epithelial mucosa. *Digestive Diseases and Sciences* 39: 2530-2535.

Murray, M.J. 1994b. Gastric ulcers in adult horses. The compendium June 1994: 792-793, 794.

Murray, M.J. 1999. Pathophysiology of peptic disorders in foals and horses: a review. *Equine Veterinary Journal* 31: 14-18.

Murray, M.J. & Eichorn, E.S. 1996. Effects of intermittent feed deprivation, intermittent feed deprivation with ranitidine administration, and stall confinement with ad libitum access to hay on gastric ulceration in horses. *American Journal of Veterinary Research* 57: 1599-1603.

Murray, M.J., Grodinsky, C., Anderson, C.W., Radue, P.F. & Schmidt, G.R. 1989. Gastric ulcers in horses: a comparison of endoscopic findings in horses with and without clinical signs. *Equine Veterinary Journal* 21: 68-72.

Murray, M.J. & Schusser, G.F. 1993. Measurement of 24-h gastric pH using an indwelling pH electrode in horses unfed, fed and treated with ranitidine. *Equine Veterinary Journal* 25: 417-421.

Nadeau, J.A. & Andrews, F.M. 2009. Equine gastric ulcer syndrome: The continuing conundrum. *Equine Veterinary Journal* 41: 611-615.

Nadeau, J.A., Andrews, F.M., Mathew, A.G., Argenzio, R.A., Blackford, J.T., Sohtell, M. & Saxton, A.M. 2000. Evaluation of diet as a cause of gastric ulcers in horses. *American Journal of Veterinary Research* 61: 784-790.

Nadeau, J.A., Andrews, F.M., Patton, C.S., Argenzio, R.A., Mathew, A.G. & Saxton, A.M. 2003. Effects of hydrochloric, acetic, butyric, and propionic acids on pathogenesis of ulcers in the nonglandular portion of the stomach of horses. *American Journal of Veterinary Research* 64: 404-412.

Nicol, C.J., Davidson, H.P., Harris, P.A., Waters, A.J. & Wilson, A.D. 2002. Study of crib-biting and gastric inflammation and ulceration in young horses. *The Veterinary Record* 151: 658-662.

O'Conner, M.S., Steiner, J.M., Roussel, A.J., Williams, D.A., Meddings, J.B., Pipers, F. & Cohen, N.D. 2004. Evaluation of urine sucrose concentration for detection of gastric ulcers in horses. *American Journal of Veterinary Research* 65: 31-39.

Orlando, R.C. 1991. Esophageal epithelial defense against acid injury. *Journal of Clinical Gastroenterology* 13 Suppl 2: S1-5.

Rabuffo, T.S., Hackett, E.S., Grenager, N., Boston, R. & Orsini, J.A. 2009. Prevalence of Gastric Ulcerations in Horses with Colic. *Journal of Equine Veterinary Science* 29: 540-546.

Rabuffo, T.S., Orsini, J.A., Sullivan, E., Engiles, J., Norman, T. & Boston, R. 2002. Associations between age or sex and prevalence of gastric ulceration in Standardbred racehorses in training. *Journal of the American Veterinary Medical Association* 221: 1156-1159.

Radostits, O. 2007. Veterinary Medicine: ATextbook of the Diseases of Cattle, horses, sheep, pigs, and goats. : 237-241.

Reese, R.E. & Andrews, F.M. 2009. Nutrition and dietary management of equine gastric ulcer syndrome. *The Veterinary Clinics of North America.Equine Practice* 25: 79-92, vi-vii.

Sandin, A., Skidell, J., Häggström, J. & Nilsson, G. 2000. Postmortem findings of gastric ulcers in Swedish horses older than age one year: a retrospective study of 3715 horses (1924?1996). *Equine Veterinary Journal* 32: 36-42.

Smyth, G.B., Young, D.W. & Hammond, L.S. 1989. Effects of diet and feeding on postprandial serum gastrin and insulin concentration in adult horses. *Equine Veterinary Journal.Supplement* (7): 56-59.

Sulku, P.M. 2012. *The association between endoscopic evaluation of equine gastric ulceration and clinical signs as perceived by the owner.* University of Copenhagen Faculty of Life Sciences Department of Large Animals Sciences.

Vastistas, N.J., Snyder, J.R., Carlson, G., Johnson, B., Arthu, R.M., Thurmond, M., Zhou, H. & Lloyd, K.L.K. 1999. Cross-sectional study of gastric ulcers of the squamous mucosa in Thoroughbred racehorses. *Equine Veterinary Journal* 31: 34-39.

Vatistas, N.J., Snyder, J.R., Nieto, J., Thompson, D., Pollmeier, M. & Holste, J. 1999. Acceptability of a paste formulation and efficacy of high dose omeprazole in healing gastric ulcers in horses maintained in race training. *Equine Veterinary Journal.Supplement* (29): 71-76.

Videla, R. & Andrews, F.M. 2009. New perspectives in equine gastric ulcer syndrome. *The Veterinary Clinics of North America.Equine Practice* 25: 283-301.

10 APPENDIXES

APPENDIX 1: QUESTIONNAIRE

1. COLIC			
Has lhe horse had colic episodes during lhe lasi 3 monlhs before lhe gaslroscopy?	No	Yes	
If you answered "Yes", how many colic episodes has lhe horse had?	One	Two ormore	
If you answered "One", please answer lo queslions 1a, 1b and 1c. If you answered "Two or more", please answer lo queslions 1d, 1e and 1f.			
One colic episode:			
1a. Whal symploms did lhe horse show?	Decreased faecal oulput. The horse was walching ils flanks and/or pawed, did nol lry lo lie down	Decreased faecal oulput. The horse was walching ils flanks and/or pawed and lried lo lie down bul gol up when chased up	Decreased faecal oulput. The horse was walching ils flanks and/or pawed, lay down and/or rolled even when chased up
1b. How long did lhe colic episode lasi for?	Less lhan 2 h	Over 2 h bul less lhan 24 h	Over24 h
1c. Whal kind of lrealmenl did lhe horse receive?	Colic resolved wilhoul velerinary assislance	Treared by a velerinarian al lhe hom	The horse was eilher referred by a velerinarian or laken direclly lo a clinic
Two or more colic episodes			
1d. How many colic episodes has lhe horse had?	2 Three lo six		Over6
1e. How long does one episode generally lasi for?	Less lhan 2 h	2 h- 24 h	Over24 h
1f. Whal kind of lrealmenl did lhe horse receive?	Ali episodes have resolved wilhoul velerinary assislance	The horse has never been laken lo a clinic bul al leasl one episode has required velerinary assislance	The horse has been laken lo a clinic al leasl once

2. INAPPETANCE			
Has lhe horse been leaving ils feed (hay/concenlrales) during lhe lasi 3 monlhs before lhe Qaslroscopy?	No	Yes	
lf you answered "Yes", please answer lo queslions 2a and 2b.			
2a. How often has lhe horse been leaving ils feed (hay/concenlrales)?	There are more days when lhe horse does nol leave feed lhan days when ildoes leave feed	There are more days when lhe horse does leave feed lhan days when il does nol leave feed, bul nol every day	Leaves feed every day
2b. How much of lhe feed does lhe horse approximalely leave?	Less lhan 1/6	1/6- y,	Over Y,

3. CRIB-BITING (both when the horse bites on an object or sucks wind without biting on anything)			
Has the horse been cribbing during the last 3 months before the gastroscopy?	No	Yes	
If you answered "Yes", please answer to 3a, 3b and 3c.			
3a. How often does the horse crib?	There are more days when the horse does not crib than days when the horse cribs	There are more days when the horse cribs than days when does not crib, but still not every day	Every day
3b. When the horse does crib, which option best describes your observation?	The horse cribs a few times but stops on its own even when not interrupted	The horse stops cribbing when something else disturbs it (fex, people commanding it to stop)	Even major disturbances (fex. People) do not make the horse stop cribbing
3c. Where does the horse crib?	In the stable	When turned out to paddock/pasture	In the stable and when turned out to paddock/pasture

4. FLANK BITING			
Has the horse shown signs of flank biting during the last 3 months before the gastroscopy?	No	Yes	
If you answered "Yes", please answer to 4a, 4b, 4c, 4d and 4e.			
4a. What is the most severe grade the horse has shown signs of flank biting?	Rubs its flanks with the muzzle/teeth, but does not damage the coat or the skin	Damages the coat with its teeth but does not damage the skin	Has damaged the skin over the ftanks with teeth so that bleeding has been seen, at least once
4b. How often has the horse shown signs of ftank biting during the last 3 months before the gastroscopy?	There are more days when the horse has behaved normally than days when it has been showing signs of flank biting	There are more days when the horse has been showing signs of flank biting than normal days, but still not every day	Every day
4c. When the horse starts flank biting, how long do these bouts last for?	Less than 1 minute	1-15 minutes	Over 15 minutes
4d. Does anything in the environment ever trigger a bout?	No	Sometimes but not always	Always

4e. What kind of a factor? Please describe with own words:

5. FUSSY EATING				
Has lhe horse shown signs of fussy ealing during lhe lasi 3 monlhs before lhe gaslroscopy?	No	Yes		
If you answered "Yes", please answer lo 5a, 5b and 5c.				
5a. Has lhe horse been chewing slower Ihan normal during lhe lasi 3 monlhs before lhe gaslroscopy?	No	Yes bul lhere are more normal Ihan abnormal days	There are more days when lhe horse chews slowly Ihan normal days, bul siili nol every day	Every day
5b. Has lhe horse been dropping feed from ils moulh during lhe lasi 3 monlhs before lhe gaslroscopy?	No	Yes bul lhere are more normal Ihan abnormal days	There are more days when lhe horse drops feed Ihan normal days, bul siili nol every day	Every day
5c. Has lhe horse laken breaks while ealing during lhe lasi 3 monlhs before lhe gaslroscopy?	No	Yes bul lhere are more normal Ihan abnormal days	There are more days when lhe horse lakes breaks Ihan days wilhoul keeping breaks, bul siili nol every day	Every day

6. WEIGHT LOSS (Below there are pictures of six horses. Pic.nr. 0 is of a horse with BCS of 0, pic.nr. 1 of BCS 1,pic.nr. 2 of BCS 2, pic.nr. 3 of BCS 3, pic.nr. 4 of BCS 4 and pic.nr. 5 of BCS 5.)			
Has the horse lost weight during the last 3 months before the gastroscopy? If the horse has gained weight please answer "No".	No	Yes	
If you answered "Yes", please answer to 6a and 6b.			
6a. How many grades has the horse approximately changed? For example: If the horse went from Ieoking like the horse in picture 5 to Ieoking like the horse in picture 3, it has changed two grades. If the horse went from Ieoking like the horse in picture 3 to Ieoking like the horse in picture 0, it has changed three grades.	One grade	Two grades	Three grades or more
6b. What it the estimated time interval for the weight loss?	4 weeks or less	5-8 weeks	9 weeks or more

7. CHANGES IN BEHAVIOUR		
Have there been changes in the horses behaviour during the last 3 months before the gastroscopy?	No	Yes
If you answered "Yes", please answet to 7a, 7b, 7c, 7d and 7e.		
7a. Has the horse been more tired than usually?	No	Yes
7b. Has the horse been lying down more than it normally does (in the stable and/or when turned out)?	No	Yes
7c. Has the horse been more aggressive than normally towards people when handled (fex. During brushing, saddling or leading the horse)?	No	Yes
7d. Has the horse been more aggressive than normally towards other horses?	No	Yes
7e. Has the horse been more nervous than normally?	No	Yes

8. CHRONIC DIARRHOEA (Meaning diarrhoea that has been going on in some form for over a month.)				
Has the horse continuously had loose faeces during the last 3 months before the gastroscopy?	No	Yes		
If you answered "Yes", please answer to 8a and 8b.				
Ba. Which option best describes the type of diarrhoea?	Slightly looser than normal faeces	Cowpile-like faeces	Watery diarrhoea together with faeces of firmer concistency	Watery diarrhoea
8b. How etten has the horse had loose faeces?	There are more normal than abnormal days	There are more abnormal than normal days, but still not every day	Every day	

9. TEETH GRINDING (Here it means that the horse grinds its teeth together, producing a sound. The horse does not have a bit in its mouth while doing this.)			
9a. Has the horse ever been observed to drool when it is not eating or wearing a bit, during the last 3 months before the gastroscopy?	No	Yes	
9b. Has the horse been grinding its teeth together during the last 3 months before the gastroscopy?	No	Yes	
If you answered "Yes" to 9b, please answer to 9c.			
9c. How often does the horse grind its teeth together (may or may not drool while doing this)?	There are more days when the horse does not grind its teeth than days when it does	There are more days when the horse does grind its teeth than does not, but still not every day	Every day

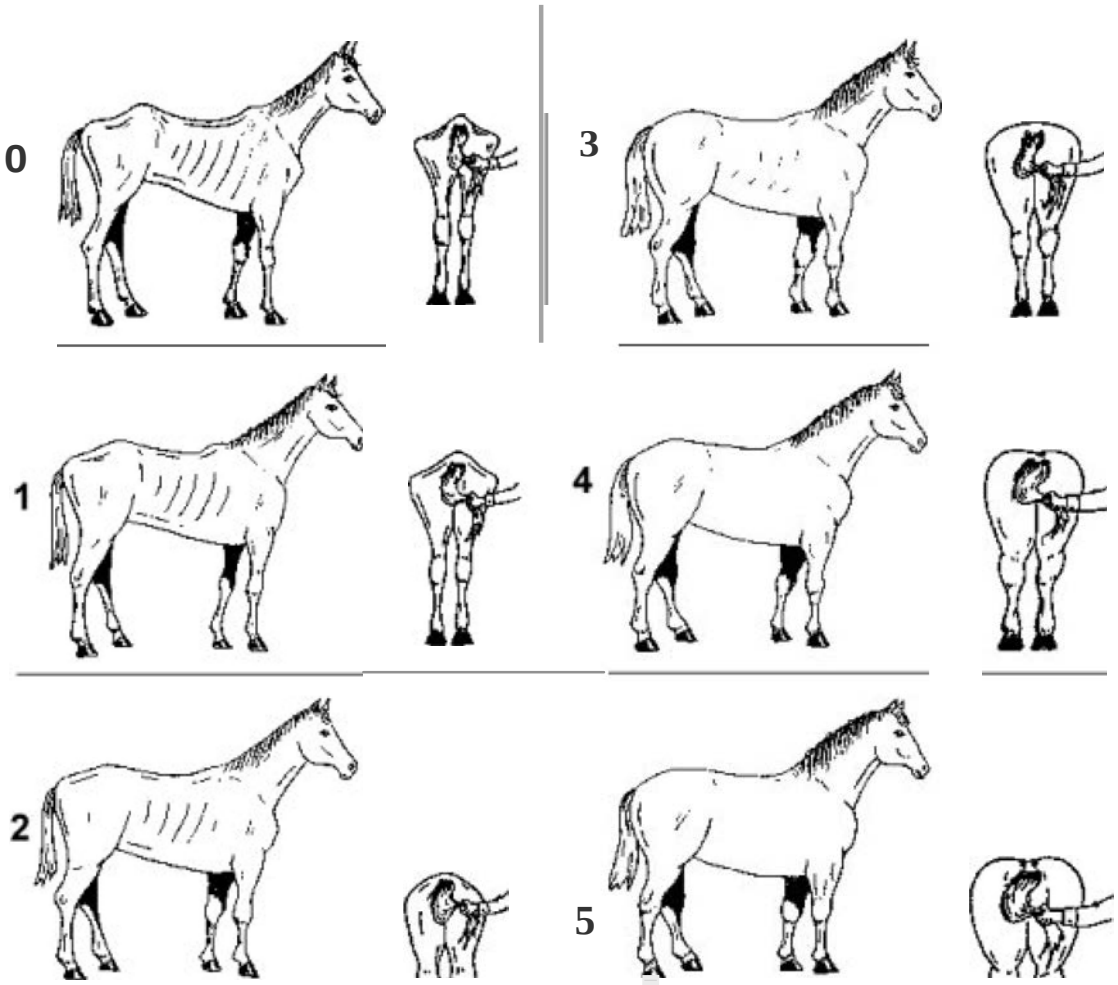
10. POOR COAT CONDITION		
Has the horse had any trouble (mild or severe) to shed the winter coat this year?	No	Yes
Has the horse been loosing abnormally much hair during the last 3 months before the gastroscopy?	No	Yes

11. POOR PERFORMANCE		
During the last 3 months before the gastroscopy, has the horse generally been performing under the expected level when exercised?	No	Yes

If you have extra comments, please write them under this section:

APPENDIX2

BODY CONDITION SCORING



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References

Johnson, J.H., Vatistas, N., Castro, L., Fischer, T., Pipers, F.S. & Maye, D. 2001. Field survey of the prevalence of gastric ulcers in Thoroughbred racehorses and on response to treatment of affected horses with omeprazole paste. *Equine Veterinary Education* 13: 221-224.

Sulku, P.M. 2012. *The association between endoscopic evaluation of equine gastric ulceration and clinical signs as perceived by the owner.* University of Copenhagen Faculty of Life Sciences Department of Large Animals Sciences.