Intragastric pH profiles in sick newborn foals often differ from those seen in healthy animals. Within the population of diseased foals, there exists wide variation in profile, related primarily to type and severity of illness. The increase in intragastric pH after H2 receptor antagonism (ranitidine, 2.0 mg/kg IV) was predictable in healthy animals but was inconsistent in many diseased foals. Ingestion of milk resulted in a substantial increase in intragastric pH in most foals. Author’s address: Island Whirl Equine Colic Research Laboratory, College of Veterinary Medicine, University of Florida, Box 100136, Gainesville, FL 32610. © 1999 AAEP.

1. Introduction

Peptic ulcer disease occurs in neonatal, juvenile, and adult horses. In contrast to older foals, critically ill newborn animals may develop perforating gastric lesions without overt clinical signs. Consequently, many foals are prescribed prophylactic acid-blocking agents when diseased. The intragastric pH profile and response to histamine type 2 (H2) receptor antagonism have been well described in healthy foals, but similar data have not been reported in sick animals. Critically ill human neonates appear to have different profiles and responses to acid suppressing drugs than healthy babies. The aim of the current study was to characterize intragastric pH profiles and the response to H2 receptor blockade in critically ill foals. These data will be contrasted to that collected in healthy newborn foals using identical methodology.

2. Materials and Methods

The study was approved by the University of Florida Institutional Animal Care and Use Committee. Twenty-three sick foals ≤ 7 days of age that required placement of an indwelling nasogastric tube for therapeutic reasons were included in the study, subject to owner consent. Therapeutic management was not altered due to inclusion in the study and therefore varied widely between foals. All were medicated with ranitidine hydrochloride (2 mg/kg IV q 8 h).

Intragastric pH was measured using a disposable, calibrated antimony pH electrode placed through the lumen of the indwelling nasogastric tube. Electrode placement was confirmed by abdominal radiography, free aspiration of gastric contents, and/or observation of a low pH values (<4.0). The electrode was connected to an ambulatory data record-
ing system, which automatically recorded pH every 4 seconds for the duration of electrode placement, which lasted between 12 and 24 hours. At the conclusion of each session, data were downloaded onto a PC via custom software. Mean pH and percent time pH > 4 were calculated for each hour.

3. Results
Primary disease and gestational age (273–380 days) varied between foals (n = 23). The most common complaints included sepsis, prematurity, enteritis, and hypoxic ischemic encephalomyelopathy. The intragastric pH varied widely and was influenced by disease type, severity, and milk intake. Mean hourly pH prior to ranitidine (2 mg/kg IV, Q8h) ranged from 2.1 to 8. The alkaline pH profile was most frequently noted in severely ill, recumbent foals. These animals usually had clinical, radiographic, and ultrasonographic findings compatible with intestinal ileus. The response to ranitidine was variable and did not mimic that seen in healthy foals. The increase in pH was frequently short-lived or absent. In contrast, a strong, but short-lived, alkalinizing effect of milk on intragastric pH was noted in most foals.

4. Discussion
It is probable that enterogastric reflux of alkaline secretions was the principal contributing factor in the generation of the higher intragastric pH in the recumbent foals, although other factors may be involved. Some of the foals had concurrent prematurity contributing to their overall condition. The ontogeny of gastric acid production merits further investigation. In contrast to these severely ill neonates, foals with less severe disease that could ambulate with or without assistance had pH profiles that mimicked those seen in healthy cohorts.

Intravenous administration of ranitidine to normal foals significantly raises mean pH for 4 hours. The response is less predictable in sick neonatal foals and varied from absent to normal. When present the effect tended to be short-lived. Clearly, evaluating the effect in foals where the pre-ranitidine pH > 6.0 is not possible. The necessity of continual acid suppression in neonatal foals is controversial. Gastric acidification may provide a protective mechanism against intestinal pathogens. However, in foals where normal mucosal protection is impaired due to systemic illness, exposure to prolonged periods of acidified contents could contribute to deep ulceration and perforation. Failure of ranitidine to effectively raise the intragastric pH in all sick foals provides the rationale to investigate alternative anti-acid therapies in diseased newborn foals.

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References and Footnotes

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