

FUNDED BY LLOYD'S OF LONDON UNDERWRITERS AND BROKERS AND THEIR KENTUCKY AGENTS

 C O M M E N T A R Y  I N T E R N A T I O N A L

IN THIS ISSUE

Commentary

INTERNATIONAL 1

Second Quarter

NATIONAL 2

EVA

Guttural Pouches

Thyroid

KENTUCKY 4

EHV-1 Abortion

Gastric Ulcers

University of Kentucky

College of Agriculture

Department of
Veterinary Science

The report in the Second Quarter of 1997 of Japanese encephalitis occurring in Korea among recently imported horses re-emphasizes the serious consequences that can occur following the international movement of horses if appropriate preventive measures are not taken.

Japanese encephalitis is a mosquito borne viral disease affecting mainly pigs, horses and humans. It causes abortion in pigs and fever and encephalitis with deaths in horses and humans. The disease is widely distributed in Asia including countries such as Japan, Korea, Thailand, Malaysia and India (which in recent times have increased the number of horses they import from North America), Australasia and Europe. Horses imported from these countries have never been exposed to this virus and are therefore completely susceptible to the disease particularly during the "rainy summer season" when the risk of mosquito transmission is increased.

An inactivated vaccine against the disease used in Japan since 1948 has significantly reduced mortality among horses over the years, and horses in Hong Kong and Singapore are routinely vaccinated against Japanese encephalitis. The only source of equine vaccine is currently from Japan although a live vaccine is utilized in China. However it would seem prudent to advise those who import horses to Asian and Pacific Rim countries from outside that region to vaccinate all horses on arrival against Japanese encephalitis and to avoid importation during the rainy season. Foals born to imported mares should also receive the vaccine.

Second Quarter 1997

The International Collating Centre, Newmarket, reported the following disease outbreaks.

Two cases of Contagious Equine Metritis (CEM) were confirmed in Japan. Respiratory disease and abortion attributable to EVA were observed in Denmark and a Standardbred stallion in the United Kingdom was found to be a shedder of the virus in its semen. Abortions attributable to equine herpesvirus-1 (EHV-1) were recorded in Italy, France and Sweden. Respiratory disease due to EHV-4 was reported in France and Ireland with cases of EHV-4 abortion also identified in Ireland.

Equine protozoal myeloencephalitis (EPM) was diagnosed in a 3-year old Thoroughbred gelding in Hong Kong imported from California.

Influenza was reported from Denmark, France, Ireland, Norway, Sweden and the United Kingdom.

Eighteen fatalities were recorded in Korea during June attributable to Japanese encephalitis among horses imported from Australia, New Zealand and Ireland within the previous 12 months.

An increased number of cases of piroplasmiasis due to *Babesia caballi* were diagnosed in Switzerland during the first half of the year. Strangles was reported from Australia, Denmark, New Zealand, Sweden, United Arab Emirates and the United Kingdom.

Vesicular stomatitis (VS) was confirmed on premises in Arizona, New Mexico and Colorado from June through September. As of September 5, VS had been confirmed on 126 premises primarily in New Mexico (54) and Colorado (71).



The Carrier Stallion as a Reservoir of Equine Arteritis Virus

Two important findings to emerge from the 1984 epidemic of equine viral arteritis (EVA) on Thoroughbred breeding farms in Kentucky were: 1) the efficiency with which the causal agent, equine arteritis virus, could be venereally transmitted by the acutely infected stallion; and 2) the high carrier rate that supervened in stallions following natural infection with the virus.

Results of experimental and field studies in the United States and other countries conducted over the past 13 years have confirmed the significance of the carrier stallion in the epidemiology of this disease. Apart from the lateral transmission of equine arteritis virus by the respiratory route during outbreaks of EVA at racetracks, sales, horse shows and breeding farms, venereal transmission by the carrier stallion either by natural service or through the use of artificial insemination has been shown to be the most important means of persistence and dissemination of the virus in various horse populations throughout the world.

The risk of spread of equine arteritis virus on both a national and international level has been enhanced in recent years by the more widespread acceptance and use of artificial insemination and by the increasing number of stallions used for dual-hemisphere breeding. If uncontrolled, either practice could lead to the inadvertent introduction of this virus into previously susceptible populations of horses. Measures to circumvent this risk are currently included in the import policies of nearly all of the major horse breeding countries of the world.

Sero-epidemiological surveys carried out over the past 10 to 20 years have revealed that the prevalence of equine arteritis virus infection can vary widely between various horse breeds, with the highest infection rate usually found in adult Standardbreds. The carrier rate in naturally infected stallions was initially reported in the late 1980s to range from 20 to 33%, with no significant difference observed between Thoroughbred and Standardbred breeds.

Table 1

Breed Distribution of Stallions Confirmed Semen Shedders of EVA 1984 - 1997

BREED	Number
Standardbred	165
Thoroughbred	27
Arabian	19
Dutch Warmblood	7
Tennessee Walking Horse	4
Hanovarian	3
Trakehner	2
American Saddlebred	1
Pony	1

Extensive screening of stallions carried out at the Gluck Equine Research Center has confirmed occurrence of the carrier state in a wide range of horse breeds (Table 1). Frequency of the carrier state varies from 22% in non-EVA vaccinated Thoroughbred stallions to 55% in Standardbred stallions, with the percentage in other breeds falling somewhere in between. Approximately 71% of a total of 747 stallions of different breeds tested were found to be seropositive for antibodies to equine arteritis virus. The mean carrier rate among the seropositive subtotal was 43.4%. It should be noted that the carrier state has never yet been detected either in a seronegative stallion or in a stallion vaccinated with the modified live virus vaccine against EVA (ARVAC[®]; Ft. Dodge Animal Health, Ft. Dodge, Iowa, USA).

Current laboratory findings confirm the widespread occurrence of carriers among non-vaccinated naturally infected stallions and the wisdom of implementing an appropriate vaccination policy to prevent establishment of the carrier state in the stallion and sexually mature colt.

CONTACT:

Dr. Peter J. Timoney, ptimoney@ca.uky.edu
 Dr. William H. McCollum, (606) 257-4757
 Maxwell H. Gluck Equine Research Center; or
 Dr. Mary Lynne Vickers, (606) 253-0571
mrvickers@ca.uky.edu
 Livestock Disease Diagnostic Center



Equine Disease Quarterly

Editors

Roberta Dwyer
 Lenn Harrison
 David Powell

Staff

Deborah William
 Diane Furry
 Linda Millercox

Correspondence should be addressed to the editors, Department of Veterinary Science, Gluck Equine Research Center, University of Kentucky, Lexington, KY 40546-0099. Telephone (606) 257-4757. Fax (606) 257-8542.

Internet address:
gopher.ca.uky.edu

or
<http://www.uky.edu/Agriculture/VetScience/gluck1.htm>

Material published in the Quarterly is not subject to copyright. Permission is therefore granted to reproduce articles although acknowledgement of the source is requested.

The University of Kentucky is an Equal Opportunity Organization.



Printed on recycled paper

Why the Guttural Pouches

The guttural pouches are outpouchings of the eustachian tube and are peculiar to the *Perissodactyla* (horse, tapir, rhinoceros). They are the site of several pathological conditions including mycosis, empyema, and tympany in modern horses.

One can say that the modern horse has been predisposed to disease of the guttural pouches by the evolution of high-crowned *hypsodont* teeth and the narrow deep skull configuration needed to accommodate those teeth. That requires some explanation.

During the Miocene period, about 20 million years ago, the evolving horses began to change from browsers—eating leaves, bushes, and twigs like modern deer—to grazers as we know them today. That occurred because the environment in which the horses found themselves changed, gradually but markedly, from forest and swamp to savannah and open grasslands.

With the change (and remember we are talking thousands and millions of years) the short, low-crowned *brachydont* teeth of the browser were replaced by long high-crowned teeth with greater abrasion resistance. That was necessary, of course, because grasses contain silica—a very abrasive material—and the longer tooth was needed to last the lifetime of the horse.

In order to accommodate those longer teeth, the skull of the horse became deeper and narrower. The pharynx became deeper and narrower, as a result of which swallowing was compromised. Skoda, an Austrian anatomist, showed in 1911 that the narrow pharynx of the horse was not attached firmly to bone as in other species and moved considerably during swallowing. This movement was necessitated by the long, narrow pharynx so that a bolus of food could pass from mouth to esophagus without entering the larynx.

Skoda further showed that movement could be accomplished because the wall of the pharynx was attached to the flexible walls of the guttural pouches. He proposed, and his theory has not been refuted, that the guttural pouches developed primarily as a necessary aid to swallowing. Abnormal swallowing does occur with tympany, empyema, or inflammatory disease of the guttural pouches supporting but not proving Skoda's hypothesis.

That might not be the only reason for the presence of guttural pouches, but it is a good reason and the only one for which we have both experimental and theoretical evidence.

CONTACT:
Dr. James R. Rooney, (410) 827-8085

Thyroid Hormone

Thyroxine (T_4) is the predominant thyroid hormone in horses but is believed to have less biological activity than triiodothyronine (T_3). T_3 is important for proper fuel metabolism (glucose absorption, protein synthesis and lipid metabolism), temperature regulation and muscle function. Thyroid hormone concentrations may be influenced by age, season, pregnancy, drugs, stress, stage of training, exercise and nutrition.

Because thyroid hormone plays a role in the regulation of fuel metabolism and muscle function, a study was conducted to assess the effects of diet on thyroid hormone concentrations in regularly exercised horses. The effects of short-term feed restriction and the calorie source (roughage vs. concentrate) on T_4 and T_3 concentrations and metabolic responses to feeding a small meal and to exercise were studied. Feed restriction was used to simulate the effects of horses going "off feed" for a brief period during a training program.

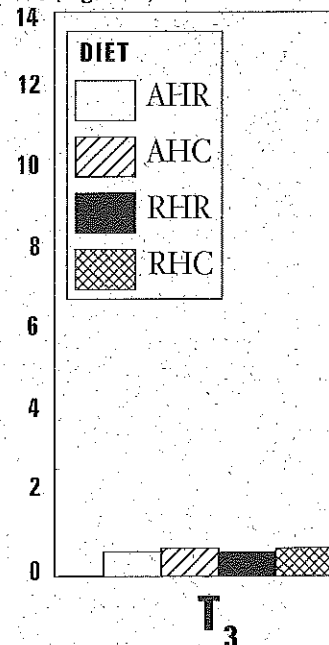
Four mature conditioned Thoroughbred geldings were assigned to four treatments in a 4x4 Latin square design, so that every horse received every treatment for 11 or 12 days. The treatments consisted of 1) a nutritionally adequate high roughage ration (70:30% roughage:concentrate) [AHR]; 2) a nutritionally adequate high concentrate ration (40:60% roughage:concentrate) [AHC]; 3) a diet restricted to 70% of 1 [RHR]; 4) a diet restricted to 70% of 2 [RHC].

On day 9 of each treatment period, each horse was fed 1.0 kg of oats in order to determine thyroid hormone response following a meal. When horses received the AHC diet, serum T_4 concentrations were lower compared to when they received the other diets, as shown in Figure 1. This result suggests that diet composition can affect the concentration of T_4 occurring in serum. Studies in other species have shown that diet can affect the conversion of T_4 to T_3 , thus it is possible that the AHC diet enhanced this conversion. However, average serum T_3 concentrations were not affected by diet.

Thyroid hormone response to exercise was evaluated on day 11 or 12 by each horse performing a 25 minute exercise test on a high speed treadmill. Horses performed a standardized exercise test on day 11 or 12 of each period.

When the horses received the RHR diet, T_4 concentrations were elevated during the exercise test

Figure 1
Mean serum T_4 and
following a meal
(ng/mL)



and recovery periods. This response may again indicate an effect of diet on regulation of conversion of T_4 to T_3 . However, T_3 concentrations were not affected by diet but tended to increase with exercise. The performance variables of lactate concentration, heart rate and rectal-temperature were not affected by diet. Further study is underway to evaluate the effects of longer term diet manipulation on T_4 and T_3 concentrations.

From the above results, T_4 concentration was lower after a meal when horses received the AHC diet, which may suggest that the high concentrate diet increased T_4 to T_3 conversion thus resulting in a lower serum T_4 concentration. When the horses received the RHR diet, T_4 concentration was elevated throughout exercise which may imply decreased conversion of T_4 to T_3 thus resulting in a higher serum T_4 concentration.

In practice, blood samples are often collected at the most convenient time without regard to feeding or exercise condition. From the above study, feed restriction and calorie source appear to affect total serum T_4 concentrations but have little influence on total serum T_3 concentrations in healthy adult geldings undergoing conditioning. These results indicate that exercise, diet and feeding state should be considered when total T_4 concentration in a serum sample is being used to assess thyroid states in horses.

CONTACT:

Debra M. Powell, (606) 257-7510 or
Dr. Laurie Lawrence, (606) 257-7509
lmlawr01@ukcc.uky.edu
Department of Animal Sciences, University of Kentucky



K E N T U C K Y

Increased Incidence of EHV-1 Abortion

During the 1996 and 1997 foaling seasons a total of 85 abortions or neonatal deaths attributable to equine herpesvirus-1 (EHV-1) were confirmed from material submitted to the Livestock Disease Diagnostic Center (LDDC) in Lexington. The number is almost three times the cases which were diagnosed

during 1994 and 1995. The increased numbers are in part due to several farms which experienced multiple abortions including two farms with eight and five EHV-1 abortions during 1997.

The vast majority of cases occurred in Thoroughbred mares although cases were confirmed among Standardbreds, Quarter Horses, Appaloosa and mares of mixed breed. The vaccination status of the majority of mares for which histories were available indicated they had received an inactivated vaccine at 5, 7 and 9 months. A selection of virus isolates provided by Dr. Mary Lynne Vickers, virologist at the LDDC, and typed by Dr. George Allen at the Gluck Center indicated they were either the IP or IB strains, suggesting no new strain had emerged.

Although the size of the Thoroughbred pregnant mare population in Central Kentucky has expanded in recent years from approximately 10,000 in 1994 to 11,800 in 1996 and higher in 1997, it does not account for the increased incidence of EHV-1 abortions. Investigations on farms where multiple cases had occurred indicated that the traditional management procedures to prevent EHV-1 abortion had not been initiated. Examples included outside mares from other farms or sales and fillies from the racetrack being introduced into groups of pregnant mares during the last 3 months of gestation as well as mixing of pregnant mares during this critical period.

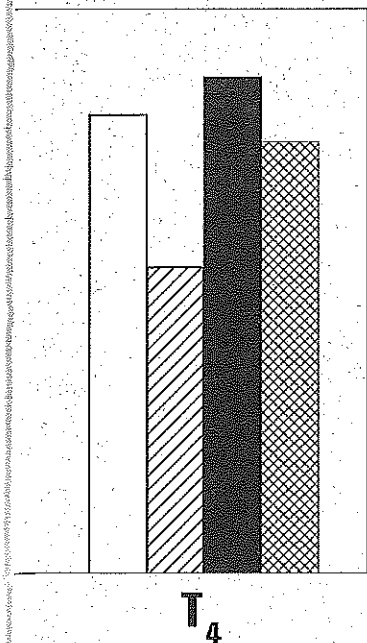
Guidelines for the prevention of EHV-1 abortion:

- Divide pregnant mares into small groups and maintain as an isolated group until foaling.
- Isolate all new animals for at least 14-21 days.
- Do not mix mares carrying their first foal with older mares.
- If mares are removed and are subsequently returned, do not add to the original group.
- Maintain all pregnant mares on a vaccination program as advised by the farm veterinarian.
- Pregnant mares should be completely isolated from weanlings and all other horses.
- Foster mares should not be in contact with pregnant mares.

If an abortion occurs:

- The aborted fetus should be placed immediately in a leak-proof container and submitted to the diagnostic laboratory.
- Notify the farm veterinarian.
- If the fetus is found in the stall, bedding should be sprayed liberally with a phenolic disinfectant, removed and burned. The stall should then be thor-

T_3 concentrations



KENTUCKY CONTINUED.

oroughly cleaned and disinfected with a phenolic compound.

■ The aborted mare should be washed and cleaned and placed in isolation until the results of the post-mortem examination are known. The mare may be covered on the second heat cycle after abortion. Other in-contact mares should not be moved.

■ If results confirm EHV-1 then the in-contact mares should be split into smaller groups.

CONTACT:

Dr. David G. Powell, (606) 257-2756
Maxwell H. Gluck Equine Research Center; or
Dr. Mary Lynne Vickers, (606) 253-0571
mvickers@ca.uky.edu
Livestock Disease Diagnostic Center

Gastric Ulceration— An Encouraging Trend

Like humans and several species of animals, horses are susceptible to ulcers in the lining of the stomach.

Gastric ulceration is the loss of a portion of the mucosal layer of the stomach with secondary inflammation and necrosis and, possibly, extension of the ulcer into the deeper portions of the stomach wall. If severe enough, the ulcer can extend full-thickness through the stomach wall resulting in perforation, especially in foals (as illustrated in **Figure 2**), and fatal peritonitis.

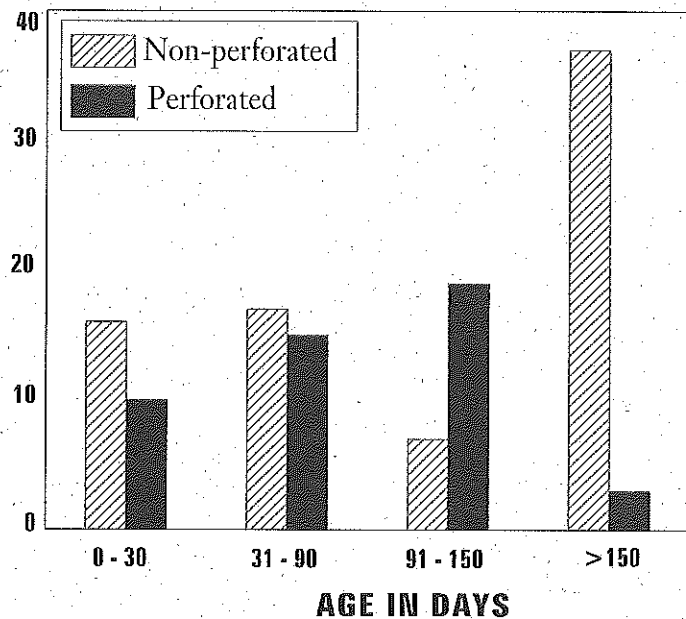
All horse age groups are susceptible to ulceration, and there are two basic types of ulceration of the stomach of horses. The more common ulceration occurs in the esophageal portion of the stomach along the margo plicatus, with a less common form occurring in the fundic and pyloric mucosa. Although the exact pathogenesis is not clear, several factors have been proposed as pos-

sible causes of equine gastric ulcers. These include stress (management practices and concurrent diseases), medication, infectious agents, parasites, type of diet, and feeding practices.

At the Livestock Disease Diagnostic Center, gastric ulcers are regularly diagnosed in horses submitted for necropsy examination. Over the last 4 years and the first half of 1997, there were a total of 130 horses diagnosed with gastric ulceration with 47 of the horses (36%) also having gastric perforation. The majority of the cases were foals less than 5 months old (52% of the horses with non-perforated ulcers and 94% of the cases with gastric perforation). Age was not provided in 6 cases. Although other reports have indicated no sex predilection, 67% of the horses with gastric ulcers in this report were females. Even when the sex distribution was determined for foals less than 5 months of age, fillies comprised 66% of the cases. Seventy-nine percent of the horses with ulcer disease were Thoroughbreds (consistent with the overall equine necropsy population).

Over the last 10-12 years, there has been a downward trend in the number of cases of both non-perforated and perforated gastric ulcers. From 1984-1988, an average of 53 cases of gastric ulceration per year was diagnosed with an average of 26 cases having perforation. By contrast, from 1993-1996 the yearly

Figure 2
Gastric ulcer cases by horse age,
1993 - June 1997



KENTUCKY CONTINUED

average was 26 cases of gastric ulceration and 9 cases with gastric perforation (Table 2). During the latter period, the total equine necropsy case-load was 15% less than the yearly average in the 1980s. The average yearly number of gastric ulcers in the 1990s represents a 50% reduction compared to the 1980s; confirming a real reduction in both number of cases and severity of gastric ulcers (perforation).

This decrease in both number and severity is believed to be largely the result of increased awareness of the condition, better medications with which to treat horses, and the increased usage of endoscopic examination by veterinarians to diagnose and monitor gastric ulcers.

CONTACT:
Dr. Neil Williams, (606) 253-0571
nmwillia@ca.uky.edu
Livestock Disease Diagnostic Center

Table 2
Gastric Ulcer Cases by Year

	Gastric Ulcer Cases	Perforated Ulcers
1984	58	34
1985	45	31
1986	34	24
1987	48	23
1988	81	16
1993	30	10
1994	29	8
1995	24	6
1996	24	13
1997	23	10

Equine Disease Quarterly Newsletter

**Department of Veterinary Science
Gluck Equine Research Center
University of Kentucky
Lexington, Kentucky 40546-0099**

Address Correction Requested

U.S. POSTAGE
PAID
BULK RATE
Lexington, Kentucky
Permit No. 51