letter to the editor

Dear Sir:

I regret having to write a letter of this nature to your fine publication. I am certain that you want only accurate articles that conform to your high journalistic standards.

I feel that it was unfortunate that my article, "Aplastic Anemia in a Cat," was published under that title in Vol. 33, No. 1 of the I.S.U. Veterinarian. The title was changed after the article was submitted and does not conform with the case as presented. A better title would have been: "A Possible Case of X-Radiation Toxicity in a Cat."

I would also like to point out a correction of my own manuscript. The term "granulocytic anemia" may have been more correctly stated as a leukopenia.

Sincerely,
Jeffrey L. Johnson

Editors' Note: We sincerely regret our error concerning the title of Mr. Johnson's article and assume full responsibility for the mistake. We would also like to thank Mr. Johnson for the correction to his article and also for helping us maintain a journal of high scientific quality.

Tetanus in the Horse

by K. D. Dibbern† and R. L. Lundvall, D.V.M., M.Sc.‡

Introduction

Tetanus is a neurotoxic disease of warmblooded animals characterized by hyperesthesia, tetany, and convulsions. Recognized since the days of Hippocrates, tetanus was described in horses by Aspyrtus in the fourth century.[5] A "modern" textbook of equine medicine written by an English veterinarian in 1883 quite accurately described the clinical syndrome, but the concept of the cause was given as realignment of the polarity of the nerves due to various forms of trauma. Although the causative organism was not isolated until 1896, the Englishman's philosophy of treatment has remained sound to this day.[4]

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Case Report

A three year old quarter horse filly was admitted to the Stange Memorial Clinic on Dec. 20, 1970, displaying classic signs of tetanus. The animal immediately was given 400 mg. Promazine HCl* I.V., 3,000 I.U. tetanus antitoxin I.M., and 8 million I.U. penicillin** I.M. A crusted, granulating wound was noted just distal and lateral to the right stifle. This was removed. The horse was given 400 mg. Promazine HCl I.V.* three times a day and 7 million I.U. penicillin I.M.** once a day from Dec. 21, 1970, until Jan. 5, 1971.

Clinical signs noted were the classic signs of tetanus. The neck and head were stiffly extended. Some difficulty was expe-

*Sparine ®, Wyeth Laboratories, 50 mg/cc
**Combiotic ®, Pfizer, 200,000 I.U. Procaine Penicillin G and 250 mg. Dihydrostreptomycin/cc.

rienced in swallowing due to the trismus present the first two days. The ears were erect, the nostrils flared, and the tail was elevated. Locomotion was quite stiff and unsteady. The response to tranquilization was quite rewarding, as the tonic spasms were controlled well enough that the animal was able to stand unaided throughout the course of the disease. Some improvement was noted after the third day of treatment. The appetite remained good throughout, but on Dec. 26 it was felt that dehydration was severe enough to warrant the administration of two gallons of water and electrolytes via stomach tube. This was repeated for three consecutive days with no complications being noted. Daily improvement was observed throughout the course of hospitalization. The horse was dismissed on Jan. 8, 1971, after a pregnancy exam revealed a nongravid uterus.

The filly was presented again to the Iowa State clinic on March 24, 1971. An exuberant granulation was again removed from the site of the original injury on the stifle. The horse appeared normal clinically. Complete hemogram and ophthalmic examinations were given with no abnormalities noted. The animal was dismissed the same day.

Discussion on Tetanus

Clinical cases of tetanus are more common in the areas of intense cultivation, with warmer climates being more often involved than colder. Mortality rates approach 80% according to several reports. Horses are the most susceptible animals, followed by hogs and sheep. Cattle and goats seldom contract the disease. Tetanus in small animals is rare. Dogs are known to be three hundred times more resistant to the effects of the toxin by weight than are horses.[5]

Although the disease is most often due to the entrance of *Clostridium tetani* spores into a wound, idiopathic tetanus has been reported.[5] This is quite rare as most cases result from contaminated wounds, observed or unobserved. Nail punctures, compound fractures, gunshot wounds, castration, tail docking, saddle gall, umbilical infections in foals, and foaling injuries in mares are a few of the commonly reported sites of infection.

Clostridium tetani is a long slender gram positive rod which forms spores after twenty-four to forty-eight hours in culture. The terminal spore has a "drumstick" appearance and usually stains gram negative. After inoculation into a wound, dead tissue and anaerobic conditions must be present for growth of the organism. Washed spores, free of the toxin, will not germinate in healthy living tissue, and are ingested and destroyed by phagocytes.[8] During an incubation period of one week to several months, two distinct toxins are produced. Tetanolysin, which hemolyses blood, is of no real significance. Tetanospasmin has a specific effect on nervous tissue. It spreads from the focal bacterial growth area up the nerve sheath axis cylinders and nerve lymphatic channels.[3] A local or tonic contraction results from the effect of the toxin on the muscle nerve endings; however, the central nervous signs are not observed until the toxin reaches the motor neurons in the ventral horn of the spinal cord. This causes reflex motor convulsions known as clonic spasms. Although some toxin is absorbed by the blood-vascular system, no general toxemia results. The observed clinical signs form too slowly to be the result of general toxemia. A bacteremia does not occur, and the bacterial growth usually remains focalized in the original wound area.

A variety of clinical signs may be observed in the clinical manifestation of the disease. First noted is a localized stiffness of the muscles of mastication or of the hind-limbs. Within twenty-four hours the stiffness is usually generalized. Hyperesthesia and clonic spasms soon follow due to the central neurotoxic effect. Consciousness remains unaffected, and the reflexes are increased until there is a systemic response to a point stimulus. Trismus becomes more pronounced. Other clinical signs frequently observed are an extended tail, erect ears, dilated nostrils, and a stiffly extended head and neck. Protrusion of the nictitating membrane results from any sudden stimulus or by simply lifting the

stiff head and neck above shoulder level. The protrusion is actually the result of the retraction of the globe by the ocular group of muscles, and is commonly noted as a very suggestive sign of tetanus. As the general muscular tone stiffens locomotion becomes more difficult and the limbs become fixed in extension causing a characteristic "sawhorse" stance. Constipation and urine retention are common. Respiratory difficulty may be noted along with sweating, increased heart rate, and congested mucous membranes. The temperature remains essentially normal until death approaches, when it may rise to 108° due to brain damage. Death usually is the result of exhaustion and respiratory paralysis. Most deaths occur within five to ten days after onset of clinical signs; if the animal recovers, the convalescent period may be three to six weeks.[7]

A differential diagnosis must be made between tetanus and rabies, strychnine poisoning, eclampsia, acute laminitis, and cerebral spinal meningitis. The most common signs observed by owners are respiratory distress, stiffness, reluctance to move, and "something wrong with the eyes" (probably the misunderstood protrusion of the nictatating membrane).

The methods of treatment have remained virtually unchanged for at least ninety years with the exception of the use of tranquilizers, antibiotics, and sedatives. If a wound or probable point of infection is found, it should be debrided and drainage established. Large doses of systemic antibiotics are used by many practitioners. Others feel that after clinical signs appear, antibiotics have little effect on the eventual outcome. At Stange Memorial Clinic large doses of appropriate antibiotics are used, but seldom are large doses of tetanus antitoxin administered. If tetanus antitoxin is used, at least 100,000 to 200,000 I.U. are recommended. The antitoxin must be administered very early in the course of the disease to have any effect, once the toxin has adhered to the nervous tissue, it is not affected by the antitoxin. Tranquilizers (such as chlorpromazine, promazine, or acepromazine) may be used to reduce the severity of the spasms and may allow the horse to eat or drink. Food and water should be at head level so the animal does not have to bend the stiffly extended neck. Chloral hydrate has been used as a sedative but may be contraindicated because it tends to lower the body temperature, does not aid the animal in eating or drinking, and may precipitate spasms due to manipulation when passing a stomach tube.[2] Curariform drugs may be used, but overdosage may paralyze the respiratory muscles and necessitate the use of a respirator. If an animal is unable to eat or drink, it may be maintained by passing one to two gallons of water, normal electrolytes, and dextrose via stomach tube. If the animal is unable to stand without aid, the prognosis is very guarded. Some sources recommend the use of a sling to keep the horse on its feet.

Three types of prophylaxis are employed. First, any wound should be cleansed, debrided, and drained. Second, tetanus antitoxin is quite effective if given before toxins are produced by the bacilli. The passive immunity lasts up to twelve days. The 1,500 I.U. dose usually recommended was first established for man and was designed to be high enough to prevent tetanus but low enough to avoid anaphylactoid reactions due to the heterologous horse serum. This situation does not apply in the horse. While the 1,500 I.U. dose may be sufficient, one author recommends that at least 5,000 I.U. should be used if a wound is found which is not debrideable, badly contaminated, or necrotic.[1] Tetanus antitoxin reaches peak efficiency in two to three days and begins to disappear by the twelfth day.[1]

The most satisfactory method of prophylaxis is the routine use of toxoid. The animal is then protected from tetanus resulting from unobserved injuries. The possibility of an error is also eliminated in deciding when and if tetanus antitoxin should be used. The patient is maintained in a state of immune responsiveness. The recommended immunization procedure is to administer two doses of toxoid four to eight weeks apart, a third dose in six to twelve months, and a yearly booster thereafter. Once this immunity is established,

the circulating antitoxin titer may be elevated by the use of a booster dose of toxoid much more than by the use of tetanus antitoxin. Foals have been shown to develop a higher circulating antitoxin titer as a result of receiving normal colostrum at birth from a toxoid immunized mare than they would get from the usual prophylactic dose of tetanus antitoxin. One large breeding farm in Kentucky gives all mares a booster dose of toxoid in January just before foaling. The antibodies in the colostrum are the only protection against tetanus which the foals receive until eight days of age when they receive the first toxoid injection. The second injection is given at forty-five days.[6] The toxoid and antitoxin act independently and may be given simultaneously at different sites.

Summary

Tetanus is a neurotoxic disease most commonly seen in the horse following penetrating injuries. Early clinical signs reported by owners are labored respirations, prolapsed nictitating membrane, a general stiffness and reluctance to move, and difficulty in eating or drinking. Accepted treatment includes at least 100,000 units of tetanus antitoxin if very early in the course of the disease, large doses of an appropriate antibiotic, tranquilization to control tetanic spasms, a darkened, quiet stall to avoid stimuli from noise and light, and possibly maintenance with fluids and electrolytes administered by stomach tube if the animal is unable to eat or drink. Prophylaxis is best achieved by the regular use of a tetanus toxoid immunization program, or the routine use of tetanus antitoxin after any injury or surgical procedure. Foals are best protected for the first week of life by antitoxins received from the colostrum of toxoid immunized mares.

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Morris Animal Foundation Report

A study, seeking data to make possible artificial insemination in dogs, is underway at the University of Oregon Medical School at Portland.

The study is headed by Stephen W. J. Seager, M.V.B., an instructor at the school who discovered techniques for preserving dog semen.

The American Kennel Club is financially supporting the project, indicating that organization's interest in the work and in canine health matters in general. Morris Animal Foundation of Denver will monitor the work.

Artificial insemination procedures have been used successfuly for many years with cattle and swine, but scientists were baffled by canine semen which is more fragile than that of many animals.

Dr. Seager, working at Oregon Medical School, announced in 1970 that he had been able to preserve dog semen and to produce litters with stored semen. This, for the first time, offered the possibility of artificial insemination in dogs.

Through artificial insemination, it might be possible for the semen of a valuable sire to be used for breeding after his death. Artificial insemination also would permit the breeding of two valuable dogs across country simply by shipping of a vial of semen, offering very selective breeding possibilities. In cattle, it is possible to dilute the semen so that one valuable sire can service many more females than in normal breeding.

Morris Animal Foundation
Denver, Colorado