Equine Encephalomyelitis

BY L. T. GILTNER AND M. S. SHAHAN

THE SAME VIRUS that causes encephalomyelitis in horses can also cause sleeping sickness (encephalitis) in human beings. Both maladies belong in a group of serious diseases of the central nervous system that includes poliomyelitis (infantile paralysis) of human beings. The intensive research work on encephalomyelitis in recent years is both significant and unusually interesting.

It is estimated that since 1930 nearly a million horses and mules in the United States have been affected by infectious encephalomyelitis. The disease, known popularly as sleeping sickness and brain fever, is believed to have been present in this country for several decades, although the specific cause—a filtrable virus—was not discovered here until 1930 (40).

In 1930 and 1931, an estimated 6,000 horses and mules contracted the disease in California alone (39). During the next few years the malady gradually spread eastward through Arizona, Oregon, South Dakota, Colorado, and Nebraska, finally involving every section and practically every State in this country and some of the Canadian Provinces.

In 1933 the disease appeared along the Atlantic seaboard, in Delaware, Maryland, New Jersey, and Virginia. From these cases a type of virus different from that found in the West was identified (41, 21, 57, 67). This so-called eastern type of virus was found to be more deadly than the western type, and in addition it was determined that

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2 Italic numbers in parentheses refer to Literature Cited, p. 388.
recovery from infection with one type did not immunize an animal against the other. The two types are thus what is known as immunologically distinct, even though both produce essentially the same clinical disease. During the years 1930 to 1941 the western type of the disease spread from California to Kentucky and Alabama, being especially prevalent in the watershed of the Mississippi River. The eastern type in the same period spread chiefly along the Atlantic coast into Florida, Georgia, Massachusetts, North Carolina, and South Carolina, inland into Alabama, and along the Gulf coast to Mexico (46, 71). So far as is known, the two types of virus have up to the time of writing remained geographically separated, except in Alabama (59) and Texas (46, 71).

SYMPTOMS

There are certain indications of infectious equine encephalomyelitis and clinically similar diseases that should be known by everyone handling horses, mules, or asses. Nearly every animal that has sleeping sickness develops fever, sleepiness, grinding of the teeth, wobbly gait, and more or less difficulty in chewing and swallowing.

The first indication, fever, is present in practically all cases. The temperature varies from 102° to 107° F. During the early part of the febrile stage, nothing more indicative of illness than lack of spirit or slightly peculiar actions may be noticed, but it is at this time especially that the virus of the disease is present in the blood (19, 48, 56) and multiplying rapidly. The disease may then terminate, if the infection is of the so-called inapparent or occult type; or it may progress, causing distinct symptoms of involvement of the central nervous system (brain and spinal cord) and constituting what is known as the frank case. Both types of attack constitute true infections, and a considerable degree of immunity results from either. These observations have been made in both experimental and field cases (15, 66). It is believed that during an outbreak many animals, interspersed among the frank cases and the apparently wholly normal animals, develop occult infections. In an epizootic area during an outbreak, animals with this type of the disease may be found in almost any stable.

Sluggishness and drowsiness are early symptoms in the developing case of infectious equine encephalomyelitis. The lips become loose and droopy or are tensed and wrinkled or drawn to one side. Groups of muscles about the head, shoulder, or flank may be seen to twitch spasmodically. With the progress of the disease, the animal stands dejectedly and moves reluctantly with an awkward stumbling or staggering gait (fig. 1). Some affected animals are inclined to back persistently or walk stumblingly in a circle in one direction, the tail switching as they move. Such animals may blunder blindly into obstructions in their path. Whinnying is a common symptom. In many cases there is extreme sensitiveness, as shown by flinching at the slightest touch or by jerking muscular contractions when the animal is excited by unusual sounds. In most cases the animals are
stupid and intractable, but a few become wild and unmanageable as in furious rabies. Sometimes sexual excitement occurs.

The dejected "sleeper" when aroused may exhibit momentary interest in feed or water, only soon to lapse into a stupor with unchewed food in the mouth or water trickling from the lips or nostrils. The lips, tongue, or cheeks may be paralyzed, but in most cases the difficulty in swallowing appears to come from inability to coordinate the normal muscular activity; often if the water bucket is held up to the animal and he is repeatedly aroused, he may succeed in obtaining a considerable quantity of water, a small amount at a time.

During the periods of stupor, grinding of the teeth is common. Stretching of the neck and head in a yawning motion is frequently observed. There is usually a slight watery discharge from the nostrils, and the mouth becomes foul smelling owing to the accumulation and putrefaction of secretions and unswallowed food. If the eye is examined carefully, the membranes will invariably be found to be congested and of a dull yellowish or muddy color. Sometimes small hemorrhages are present.

With the progress of the illness, the animal becomes increasingly gaunt, and there is commonly a prominent ridge due to muscular tenseness along the lower abdominal wall. The digestive and excretory processes are retarded, and more or less constipation or sluggishness of the bowels results. Urine collects in the bladder, which may become greatly distended. Dribbling of urine without normal periodic voiding is common.
In about 1 of every 4 or 5 cases due to the western type of virus and 9 of every 10 cases caused by the eastern type, the animal gradually becomes weaker and weaker, finally collapsing on the ground. Very few of those that go down and are unable to regain their feet unaided recover, though it may be hours or days before the end. During this time they may lie quietly, breathing with a snoring sound, or they may thrash about considerably. Some animals literally bury themselves, digging up the earth with running movements of the legs in their delirium. Marked bruising and swelling develop on the parts of the body in contact with the ground.

If the animal lies quietly, the body temperature is usually normal or only slightly raised at this stage, but if there is much struggling or if complications such as pneumonia or other organic diseases occur there may be considerable fever. Among the animals that survive a typical attack of the disease, a few—the so-called dummies—retain evidence of more or less permanent injury to the brain and spinal cord.

**DEVELOPMENT AND SPREAD OF THE DISEASE**

Infectious equine encephalomyelitis is essentially an epizootic disease—that is, one occurring in outbreaks that correspond to epidemics among human beings. While scattered or sporadic cases may occur at any time during the warm months, the peak of an epizootic is usually reached during July, August, or September in most sections. The disease invariably disappears after the first sharp frosts of fall. There is only one proved winter case on record, and that occurred in Florida in January during a mild season (43).

Sleeping sickness is essentially a disease of pastured animals, occurring only rarely among those continuously stabled. This is considered to be one of the reasons why Army posts and Thoroughbred breeding establishments remain relatively free from it.

The malady strikes hardest in farm and ranch areas along or near a stream, lake, marsh, swamp, or the seashore. In the West, irrigated sections are especially affected, as well as arid regions when unusually heavy rains occur. In the East, the tidewater regions have the most cases. Scattered or sporadic cases may be observed early in the summer and throughout the warm months under conditions of drought. On the other hand, heavy rains that result in the flooding of pastures and waste land and the formation of pools are frequently followed in 2 or 3 weeks by large numbers of cases. Under the latter conditions, extended hot weather also appears to be a factor in promoting the spread of the disease. Both moisture and heat, of course, encourage the breeding and feeding activities of insects.

There is a tendency for the disease to occur sporadically for one or more seasons in a given locality and then, under conditions favorable to it, to become very severe and widespread for one or more seasons. Following a severe and extended epizootic period, the disease may practically or entirely disappear for several years, only to reassert itself later when circumstances again become favorable.

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3 Detailed clinical descriptions of the disease are presented in citations, 6, 10, 17, 39, 49.
During severe outbreaks, 10 percent or more of the horses and mules in an area may show unmistakable signs of the sickness. It is seldom that more than one or a few of the animals on a given farm sicken. Cases are often separated by several miles, and some premises appear to escape altogether. However, the virus probably attacks more animals than is superficially apparent, producing occult infections.

The incubation period of the naturally acquired disease (the time elapsing between entrance of the virus into the body and the appearance of the first symptoms) is believed to vary in length, as does this period in artificially infected animals. In the latter, the first symptoms usually appear 3 to 10 days after the introduction of the virus, depending on the dose and route of injection and on the type of virus. In some unusual cases as long as 3 weeks may elapse.

Although it is generally believed that horses of all ages and breeds are susceptible, there is some evidence to indicate that certain breeds may be less resistant than others (30). Certainly animals that have been exposed to the infection through the course of one or more outbreaks may be reasonably assumed to be more resistant as a class than others, such as colts, which have never before encountered the disease. Heredity, age, and nutrition are all suspected of playing a part in the artificially produced disease in mice, and these may be factors in the case of horses also.

Tests with guinea pigs indicate that young animals nursing immune mothers are highly resistant to the disease (20). Foals and young horses are believed to be more likely to recover than older horses. There is a widespread opinion among practicing veterinarians, to some extent supported by available data, that mules are less susceptible than horses (51).

It is generally believed that the disease is transmitted by blood-sucking insects. It has been found experimentally that at least 11 species of mosquitoes, all of the genus *Aedes*, can be infected, and 10 of these are capable of transmitting the virus (9, 16, 26, 27, 33, 34, 36, 61). Some species have been found that are practically incapable of transmitting one type of virus but that infect with the other type with a single bite (15, 37). Other species transmit both types with equal facility. Some of these mosquitoes are known to be widely distributed in the United States, others are somewhat restricted in distribution, but all except one species are present to some extent in one or more of the sections where the disease has occurred (5).

Mosquitoes (*Culex tarsalis*) naturally infected with the western type of virus were discovered in the Yakima Valley, Wash., in 1941 (16). Numerous fruitless attempts had been made to find the virus in mosquitoes and other insects (including horseflies, houseflies, stable-flies, horn flies, and spinose ear ticks) in nature (44, 56, 70) before this important finding was reported.

The virus has been reported as present in so-called assassin bugs (*Triatoma sanguisuga*) (29) collected in Kansas where the disease has been prevalent. This bug is relatively infrequently found in comparison with other insects that might be suspected as vectors (carriers) and probably cannot be held solely responsible for the dis-
semination of the equine disease. The chief importance of finding infection in this species would seem to lie in the probable existence of a reservoir for the virus somewhere among the many kinds of victims of its bite.

The tick that transmits Rocky Mountain spotted fever (*Dermacentor andersoni*) has been found to be capable of transmitting equine encephalomyelitis virus under experimental conditions (63). Moreover, it has been shown to transmit the virus from one generation to the next through the eggs and larvae (64). Like other ticks, these are relatively long-lived, and it is easy to conceive of their carrying the virus through the winter, or even through several years, and then transmitting it to susceptible species for further propagation, as has been suggested (62). The fact that the disease can readily be transmitted under certain conditions by several species of arthropods, particularly mosquitoes, must be accepted. However, that they actually do transmit it under natural conditions, or that they are the sole means of transmission, remains unproved despite very strong circumstantial evidence supporting the theory.

What becomes of the virus with the seasonal subsidence of the disease in horses is unknown. No evidence has yet been presented which proves that horses harbor the virus from one epizootic season to the next. Inasmuch as the virus is capable of surviving for only a relatively short time outside the animal body, it is assumed that it is perpetuated in some species other than the horse, mule, or ass.

Man was suspected as early as 1932 of being susceptible to the virus of sleeping sickness of American horses (38), and the possibility that he might act as a reservoir of the virus was considered in 1935 (66). The eastern type of equine virus was first actually demonstrated in cases of disease of the brain in man in 1938 (12, 72). Other cases, some due to the western type of virus, were subsequently identified (11, 32), and it is now an accepted fact that man is susceptible to the disease, which, as far as is known at present, is not immunologically related to the somewhat similar diseases, infantile paralysis (poliomyelitis) and epidemic encephalitis (St. Louis type). In many if not most cases of the equine virus disease in human beings there has been no evidence of contact with horses, and it is assumed that people generally contract the infection from the same source as horses, though a few cases have occurred among laboratory workers and some among persons closely associated with sick horses. Children have been especially affected in some outbreaks. The virus recovered from the brain of a child has produced typical equine encephalomyelitis when injected into susceptible horses. Encephalitis in man due to the equine virus is a very serious disease at any age, but there is a particularly high mortality rate among children affected with the eastern type, and troublesome complications are reported to be common in those surviving. The disease caused by the western type of virus is relatively much less severe than that due to the eastern type. On the other hand, the former has been more widespread. Over 2,000 cases, mostly attributed to the equine virus (western type), were reported from North Dakota, South Dakota, Minnesota, Montana, and Nebraska in 1941.
It has been determined that horses are susceptible to human encephalitis (St. Louis type) virus when artificially inoculated, and in areas where sleeping sickness has been epizootic in horses, people and animals have been found the serum from which was capable of neutralizing the virus of that disease as well as the virus of infectious equine encephalomyelitis (8, 23, 24, 45). The assumption is that both viruses may produce the disease in horses and other animals as well as in man and that the two have existed simultaneously in some outbreaks of both the human and the equine disease, although St. Louis encephalitis virus has never been found naturally occurring in equines. The viruses of St. Louis encephalitis and equine encephalomyelitis have both been found in mosquitoes (Culex tarsalis) (16).

A disease of the central nervous system due to equine encephalomyelitis virus has been found to occur naturally in ring-necked pheasants, pigeons, and prairie chickens (3, 7). Thus, it is definitely known that birds are sometimes attacked by the virus of so-called brain fever of horses. This disease has no known connection with the virus disease of chickens known as chick encephalomyelitis or epidemic tremors (42).

A disease in ducks in Montana suspected of being encephalomyelitis was investigated in 1938, but the presence of the equine virus was not actually demonstrated (60). This outbreak, as well as the proved cases in pigeons, pheasants, and prairie chickens, occurred in an area where sleeping sickness of horses had been prevalent.

It is now known that over 20 species of birds are susceptible to the virus of sleeping sickness when artificially injected. Following inoculation by certain methods, hens and turkeys have been found to develop transitory inapparent infections, with virus circulating in the bloodstream (65). A similar condition has been shown with certain species of blackbirds and the mourning dove, cowbird, and grackle (9). Such birds, without showing obvious indications of infection, may play roles in the spread of the disease as important as or even more important than those of others which, like pigeons and pheasants, develop frank symptoms of encephalomyelitis and have a high death rate from the disease. The list of susceptible birds includes ducks, geese, a species of hawk, blackbirds, the tawny vulture, the white stork, guinea fowl, English sparrow, Gambel sparrow, quail, LeConte thrasher, the junco, the western burrowing owl, the American egret, red-shafted flicker, killdeer, and robin, besides those already mentioned.

Equine encephalomyelitis virus when artificially injected is capable of infecting more species of animals than perhaps any other known virus. About as many species of mammals as of birds have been so infected. In addition to the commonly used laboratory mammals—guinea pigs, white mice, white rats, rabbits, and monkeys—cattle, swine, sheep, goats, dogs, cats, several species of wild mice, rats, and other rodents, including rabbits, gophers, hedgehogs, and woodchucks may be infected. The list also includes weasels, ferrets, and hamsters (ratlike rodents of eastern Europe). Some species appear to be susceptible to both types of virus, while some can be infected with one type but not the other.
Although horses, mules, and human beings are the only mammalian species now known to contract the disease naturally, many others equally susceptible to artificial exposure may eventually be found naturally affected. In fact, the virus has recently been isolated from a wild deer in Montana. The studies being made by several agencies will probably lead to the identification of other naturally infected animals and eventually to important biological reservoirs of the virus.

While it is possible that horses may occasionally contract the disease by inhaling or swallowing virus from affected animals, judging from the observed facts, transmission by such means must be exceedingly rare. Several deliberate attempts to induce the disease in normal horses by putting them in contact with affected horses have failed (68, 69). In fact, in all the literature on the disease not one proved instance is mentioned of infection of normal animals by contact with infected animals of any species or with an environment that might have been contaminated. The tentative conclusion, based on the available information, that the disease is chiefly arthropod-borne, is inescapable, although more work remains to be done to prove it.

DIAGNOSIS

The diagnosis of infectious equine encephalomyelitis is based on a study of the symptoms in each case and such features as seasonal occurrence and distribution in the locality. However, because atypical cases do occur and unusual conditions are not infrequently encountered, a specific laboratory diagnosis (59) may be required for confirmation of clinical diagnosis or the determination of the type of virus involved.

If virus is sought in the living animal, repeated samplings of the blood very early in the course of the ailment, preferably before symptoms of disease of the nervous system appear, are usually required. The virus is present in the bloodstream for only a comparatively short time and then becomes fixed in the brain and spinal cord, only rarely being found elsewhere after that. It is shown to be present by the inoculation of the finely emulsified nervous tissue from the suspected case into any of the available susceptible animals, usually guinea pigs or white mice; or the tissue may be inoculated into hen’s eggs containing 9- to 13-day-old living embryos. Symptoms like those seen in horses develop in the guinea pig or mouse in a few days, and death usually results. The inoculated embryo succumbs to the virus in 15 to 24 hours (18). The virus is similarly demonstrable in the body of the inoculated animal and in the embryo, and it may be transmitted by inoculation from the original guinea pig, mouse, or embryo to others in long, continuous series.

If a horse or mule has been dead for some time or even prostrate for several days, it may be impossible to demonstrate virus in the tissue of the central nervous system. A few known artificially produced cases also fail to yield virus. It is then necessary to resort to microscopic examination of the tissues (4, 25, 28, 31). In the brain and spinal cord, the blood vessels are found to be engorged or swollen, and small hemorrhages, edema, and abnormal accumulations of various types of cells in the spaces surrounding the blood vessels or in other parts of the tissues are present. Varying degrees of degeneration of the nerve cells are to be observed. Somewhat similar but less characteristic changes may be found in the liver.

These changes are not apparent to the naked eye, although very minute hemorrhagic points may be seen in certain parts of the brain. The cerebrospinal fluid (surrounding the brain and spinal cord) is clear but greatly increased in amount and chemically and microscopically abnormal. The throat and nasal passages may be inflamed, and a putrid odor may be present. Usually the lungs are essentially normal, although there may be congestion or even pneumonia, especially in
animals that have been down for some time. The stomach usually contains a considerable amount of watery, foul-smelling food material. Evidence of catarrhal inflammation and sluggishness of the bowels are common. The bladder is usually distended with clear, amber-colored, sirupy urine. The liver, instead of being slate-colored as normally, is slightly enlarged and of a dull, yellowish-gray color. The various membranes of the body also are usually dull yellowish, owing to staining with bile. The muscles appear dry and lighter colored than normal as a result of great loss of water (dehydration) by the sick animal. These changes are not necessarily characteristic of sleeping sickness, and unqualified diagnosis of the disease by this means alone is rarely if ever justified, but a thorough post mortem examination is nevertheless often a great assistance to the veterinarian in identifying other diseases that may be responsible in suspected cases.

Experimental studies have been made of the complement-fixation test (involving the use of blood serum, virus, and red blood cells) as applied to sleeping sickness, but it has not yet been generally used in diagnosis (22, 41). The blood serum of a convalescing or recovered animal may be tested to determine its power to inactivate or neutralize encephalomyelitis virus (in vitro neutralization test) (14, 15, 19, 38, 66, 67). A positive reaction to this test is generally accepted as evidence of past or present infection, the neutralizing substances constituting a form of antibody.

**SIMILAR DISEASES AND CONDITIONS**

Not all cases of illness in horses and mules in which there is evidence of disturbance of the central nervous system are infectious encephalomyelitis.

Toxic encephalitis, as contrasted with infectious encephalomyelitis, may occur at any time of the year—in the dead of winter as well as in midsummer. However, the commonest form, that known as moldy corn poisoning (55), usually makes its appearance in the late fall or the winter in horses or mules that have been fed on moldy, stunted, or otherwise deteriorated or inferior corn. This malady has been responsible for the death of thousands of valuable horses from time to time in several of the Corn Belt States, usually in the months of November to April after an unusually dry summer and heavy fall rains.

The symptoms of moldy corn poisoning are scarcely distinguishable from those of other clinically similar ailments by any but the trained observer. Usually, however, there is no yellowness of the membranes of the body and no great rise in body temperature. At autopsy, areas of softening and degeneration, sometimes microscopic only but more often plainly evident to the naked eye, are usually found in the brain.

Prevention consists in avoiding the feeding of corn or corn fodder about the absolute soundness of which there is the slightest doubt. Other species of animals may tolerate poor corn, but is dangerous to horses in this and other ways. It may be desirable in some instances to muzzle horses being worked in the corn harvest in order to prevent their eating potentially dangerous corn. Although moldiness is considered as an important indication of danger, unfortunately it is not always possible to determine by examination whether a certain lot of corn can be fed with safety. The toxin in bad corn, whatever its nature, appears to be cumulative in effect, and symptoms do not ordinarily develop in less than a month, even though the corn is fed continuously.
There is no specific remedy for the disease, and the medicinal aid given by the veterinarian varies somewhat with the conditions found in each case.

Other toxins or poisons in some deteriorated feeds other than corn, as well as some mineral poisons such as barium, lead, arsenic, phosphorus, and selenium, may cause symptoms attributable to or suggestive of encephalitis. Among the plants that may induce more or less similar symptoms are whorled milkweed, yellow tarweed, poisonhemlock, ragwort, locoweed, ergot-infested paspalum grass, and horsetail. Unripe and green as well as rotten or sprouted potatoes are very dangerous feed for horses or other animals and often produce symptoms suggestive of encephalitis. Certain ticks engorging on various animals, including the horse, sometimes cause a peculiar form of paralysis, which is usually promptly remedied by the removal of the ticks.

Such diseases as anthrax and influenza (shipping fever) sometimes cause encephalitis. Some cases of acute infectious anemia (swamp fever) show symptoms that resemble those of infectious encephalomyelitis. Rabies, a virus disease transmitted by the bite of an infected animal, usually a dog, is a form of encephalitis. Still other infectious diseases may cause symptoms of nervous disease. Even tetanus, or lockjaw, has been diagnosed as infectious encephalomyelitis by untrained persons. Diagnosis and treatment vary according to the nature of the primary disease in question.

Some animals affected by heat stroke or lightning develop spasms, convulsions, or residual paralysis, which must be differentiated from true encephalitis. These, of course, are usually isolated cases.

Tumors of various types and abscesses in the brain arising from bacterial infection may cause symptoms suggesting infectious encephalomyelitis. Close clinical examination and a study of the history of these comparatively rare cases will usually enable the experienced veterinarian to identify the condition. The same may be said of mechanical injuries to the head.

Botulism (13) is a disease caused by the ingestion of a toxin produced in feeds under certain conditions by a widely distributed germ known as Clostridium botulinum. The chief symptom consists of progressive paralysis, usually beginning in the eyes, tongue, and throat, finally involving the entire body, and resulting in death.

Azoturia, sometimes referred to as Monday morning disease because it commonly occurs in well-fed and underexercised horses after a week end of rest in the stable, has been confused with infectious encephalomyelitis. The disease is discussed in the section on Miscellaneous Diseases of Equines, page 452.

In the last decade, in several States in areas where infectious encephalomyelitis had previously prevailed, a second illness, now called toxic hepatogenous icterus, in some ways resembling sleeping sickness and in others quite different, has appeared during the late fall and early winter 2 weeks to 2 months after epizootics of sleeping

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*The term "toxic hepatogenous icterus" has been proposed to replace the previously used terms "secondary disease" or "X disease." See Mohler, J. R. Report on Infectious Equine Encephalomyelitis in the United States in 1939. U. S. Bur. Anim. Indus., 4 pp., Illus. 1940. [Mimeographed.]
sickness (35, 58). Usually horses affected with this disease have no fever, develop an obstinate constipation and extreme icterus (jaundice), and become very unmanageable, pushing violently against any objects in their path. The affection is extremely acute and the death rate high. Studies conducted to date do not prove that it is infectious, and its true nature is not yet definitely known. Although many of the animals that have developed this affliction had been treated earlier with antiencephalomyelitis serum, some had not.

The chief abnormality found at autopsy is a damaged liver, which shows characteristic changes under the microscope. Success in treatment appears to depend chiefly on early diagnosis and prompt stimulation of the intestinal tract.

CONTROL AND PREVENTION

Although not everything is known about the spread of sleeping sickness, as has been pointed out, nevertheless there are certain justifiable, practical, and apparently effective means of prevention. The selection and application of these measures, singly or collectively, must properly be considered as technical matters. Accordingly, when control of the disease is to be attempted, a licensed veterinary practitioner or authorized veterinary official should be consulted. In the past, much money has been spent needlessly by frantic horse owners for worthless and in some cases even harmful so-called preventives or cures for infectious encephalomyelitis.

(1) Among the means of prevention, vaccination is of first importance. The approved, commercially available vaccine of today consists of artificially infected chick-embryo tissue, in which the power of the virus to produce the disease has been destroyed by the addition of a solution of formaldehyde (2). Other products have been tried with more of less success, but all have been displaced by the present one of chick-embryo origin known as encephalomyelitis vaccine. The vaccine is prepared from embryos infected with western, eastern, or both types of virus. Which should be used depends upon the type of virus known to prevail in the locality.

Since the virus is rendered harmless by the presence of formaldehyde in the vaccine, there is no danger of producing the disease in animals injected with the product. It is thus unlike some other products used for the control of animal diseases, but it is not so foolproof that it can be safely and effectively used by everyone. Its proper administration requires the services of a veterinarian who understands that the product deteriorates and becomes less effective, if not worthless, when not properly handled. Then, too, severe reactions and even death have been known to occur following vaccination (52, 53). These undesirable results can be largely if not wholly prevented by injecting the vaccine into the skin (54) rather than under it or into the muscles, as was once practiced.

While one dose of the vaccine results in an appreciable degree of resistance, it lasts for only a comparatively short time. Accordingly two doses at 7- to 10-day intervals should be given. Unfortunately many farmers do not have their animals vaccinated until the disease
has already made its appearance on their farms or in their vicinity, and under these conditions the method is not fully effective. Although vaccination during an outbreak appears to be safe and effective as a last resort, such a procedure cannot be expected to be as advantageous as vaccination before the epizootic season, which extends from July to October in most sections but is somewhat earlier in others. To secure the maximum protection during these months, the two-dose vaccination should preferably be completed not later than early July in most localities. In some areas vaccination in the early spring or even late winter may be advisable. Experimental tests indicate that most vaccinated animals develop immunity within 10 days to 2 weeks after the administration of the second dose of vaccine and retain a sufficient degree of immunity to resist even the severest artificial exposure for 6 months and sometimes even longer (50, 60). In order to assure protection, it appears at present that animals should be revaccinated each year.

(2) Second among preventive procedures is the prevention or limitation of bites by bloodsucking insects, particularly mosquitoes. Prevention or control of breeding of the insects, screening of stables, stabling of horses and mules as much as possible, the use of nets, etc., on working animals, and the frequent application to the animals and the premises of repellent compounds are all useful.

(3) All animals affected with the disease should be stabled in screened quarters or kept thoroughly sprayed with an effective repellent, especially in the early stage of the disease when the blood is infectious. If possible, a separate caretaker should be provided for them. Although it is probable that man does not contract the disease from horses or other affected animals by direct contact, those engaged in handling or treating such animals should avoid unnecessary risks, especially from soiling the hands or person with secretions or excretions from the sick animals.

(4) Extermination of rats, mice, and other vermin and exclusion from the places where the horses are kept of pigeons and stray or wild animals are advisable on general principles.

(5) Although rigid general quarantine measures have not been found practical in dealing with encephalomyelitis, the movement of horses or mules from an epizootic area into a clean area should be discouraged. When it is necessary, the animals might well be vaccinated at least 15 days before being moved. Animals going into an infected area from one where the disease has not been prevalent should be similarly treated.

(6) Animals dead of the disease should be promptly disposed of by thorough rendering or burning or deep burial with the addition of quick lime. The stables, sheds, corrals, etc., used by the affected animals should be thoroughly cleaned, and feed boxes, stalls, watering troughs, etc., should be disinfected with either a 2-percent aqueous solution of lye or a 1-percent solution of formalin. The formalin will in time evaporate from the treated surfaces, but lye should be thoroughly removed after a few hours by repeated washing with clean water.

(7) A specific antencephalomyelitis serum (19, 20, 38, 47) is com-
commercially available, and some experimental data indicate that it has some preventive value. Practical considerations, however, considerably restrict its use in the prevention of the disease, and vaccine is preferred in most instances.

Vaccination properly done does not interfere with working or breeding the animals. A few instances of abortion have been reported in pregnant mares vaccinated by the older method, but none following intradermic vaccination. A small, hard swelling may remain at the site of injection for as long as a few weeks, but this does no harm unless it is situated where harness or saddle will rub it, and it is eventually completely resorbed.

Laboratory workers exposed to unusual danger of contracting the infection have been safely vaccinated with a product of the same general type as that used for horses (1), but it has not been generally used in epidemic areas.

TREATMENT

If despite well-advised and diligently applied control measures, sleeping sickness or anything resembling it develops in a stable, a veterinarian should be called immediately. Except for antiencephalomyelitis serum, which if used at all must be given very early in the course of the disease and in large, frequently repeated doses, there is no specific treatment. Whether or not serum should be used is strictly a matter for the veterinarian's decision.

Reliance must be placed chiefly on good nursing, with such additional treatment as is recommended by the veterinarian for each individual case. Quiet, cool, comfortable, well-bedded quarters and protection of the animal from the sun and from self-injury are advisable.

If the animal has difficulty in standing, he may be placed in a leaning position against a padded wall or supported in a specially constructed padded frame.

Unnecessary fussing about the animal should be avoided, although almost constant attendance may be required to assist it in supporting itself. Many animals affected with sleeping sickness will not or cannot eat or drink unless the feed and water are held up to them and they are frequently aroused from their stupor. Water in small quantities, frequently given, is most desirable, and if it is not or cannot be swallowed it may be given by stomach tube, by way of the rectum, or into the blood stream in the form of various solutions. Feed is not nearly so important as water. However, when the animal will take small amounts of it, succulent feed may be allowed freely.

On the supposition that horses affected with encephalomyelitis, like humans with encephalitis, have a headache, cold compresses or ice packs are sometimes placed on the poll. This should do no harm and may be of benefit. Although very few animals that have been prostrate for hours or days can be expected to recover, they should be humanely treated if they are not destroyed.

Some cases may be benefited by stimulants; others may require laxatives, evacuation of the bladder by catheterization, or quieting
drugs. Such drugs as are given are usually administered either through a stomach tube or hypodermically. No one system of treatment is applicable in all cases. Above all, unguided home treatment, promiscuous drenching, or other administrations which may be suggested by unqualified advisers, should not be attempted. Unwise or injudicious treatment may easily result in the needless loss of the animal. On the other hand, many horses die despite the best efforts of the most experienced and highly qualified veterinarians.

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