

EPIDEMIOLOGY OF EQUINE ENCEPHALOMYELITIS IN THE EASTERN UNITED STATES

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Although it has probably existed for many years in the United States, equine encephalomyelitis was not recognized as a separate entity until 1930 when Meyer, Haring, and Howitt (1) demonstrated a filtrable virus as the cause of the condition. Since then the disease has been found in various western states, and in the summer of 1933 a similar condition was recognized in Virginia, Delaware, New Jersey, and Maryland. Although the equine disease was much the same as that described in the West and the virus isolated was pathogenic for the same species of animals, it differed serologically (2) from the virus obtained by Meyer. We have therefore referred to the disease with which we have been working as the eastern type of encephalomyelitis, and it is our purpose here to record various facts that may have a bearing on its transmission as a background for experiments that are to follow.

Seasonal Distribution

Like the western disease the one found in the East has a seasonal distribution; in the last two years it has appeared in August, reached its height during September, and disappeared in October. We have had reports of winter cases but the diagnoses were clinical ones and in the few instances where we were able to get brain material for examination there has been no evidence of the virus disease either histologically or by animal inoculation. Forage poisoning (3) and leucoencephalitis (4) are often confused with the virus disease and may occur in the same regions. A positive diagnosis of equine encephalomyelitis in winter cases should therefore be substantiated by more than the clinical picture, preferably by the demonstration of

the virus. In the region to be described, where we have the cooperation of a very intelligent county agent, winter cases have not been detected although up till the time of frost many cases were found.

Geographic Distribution

Unlike the western disease the cases that we have encountered have been closely related to salt marsh areas. This is brought out in Text-fig. 1 which shows the distribution of infected farms on the Northern Neck of Virginia during the years 1933 and 1934. These data were secured for us by Dr. H. C. Givens, State Veterinarian, and by Mr. C. Carter Chase. It will be noted that the great majority of cases are within 2 miles of the coast line which has many inlets and much marshy ground. There were cases of the disease in the inland area where the horse population is greater than along the shore, but in several instances they were in horses that had been carting produce to the shore. In other instances no such history was obtained, so that we can say that the disease is not strictly limited to salt marsh areas. While we have been unable to secure data which would enable us to make similar maps for other regions, the information we have obtained from the Eastern Shore of Virginia, from Delaware, and from southern New Jersey shows that the disease incidence is far greater near the coast line than it is inland. In the summer of 1934 more cases were detected inland in New Jersey, but the great majority were in horses used in gathering hay from the salt marshes. Since the disease is not reportable figures on the distribution are only approximate.

Consideration of Contact Infection

Influenced by the work that has been done on the transmission of poliomyelitis our first thought was that equine encephalomyelitis was also spread by contact. The more conditions in the field were observed, the less likely it seemed that the two diseases were transmitted in the same way. As noted in the preceding paragraph, the cases were sharply localized to regions near the shore and there was very little tendency for it to spread inland. On some farms all horses developed the disease but on many others only a portion were infected. Often the disease appeared on farms a mile or more distant from those on which cases had occurred and there would be no known



TEXT-FIG. 1

contact between the horses on the two farms and no cases in the intervening areas.

The possibility that the disease might be widespread among horses of which only a few developed symptoms has been considered and serum from horses in areas where the disease had occurred has been tested for neutralizing antibodies. The results of these tests are given in Table I. With the exception of the control horses the animals were on farms in southern New Jersey where it is suspected the disease has existed for many years. It will be seen that many of the horses

TABLE I

Horses from farms	No. of horses tested	No. of sera showing neutralizing antibodies*	Percentage showing neutralizing antibodies
Where one or more cases of disease had occurred from a month to 6 weeks previously to bleeding	53	9	17
In the area where the disease was epidemic but on which no known cases had been found	14	2	14
Institute (control group)	10	0	0

* The tests for neutralizing antibodies were made by injection of mixtures of undiluted serum and known dilutions of virus into mice. In each test serum from a horse known to be negative was included as a control. While most of these tests were made by intracerebral injections we have since found the intraperitoneal route more satisfactory.

must have been infected at some time in their lives, but the figures do not support an hypothesis that all or nearly all have been infected.

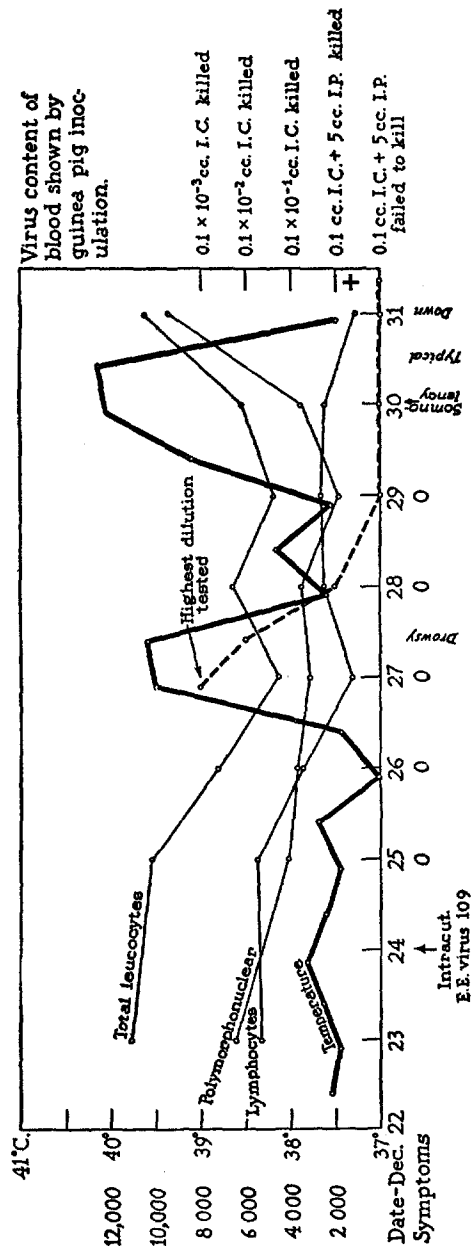
We have made no experiments to determine whether horses could be infected by contact, but Records and Vawter (5) were unable to secure transmission of the western disease by exposure to infected animals. In a subsequent paper (6) they report a possible contact infection but the diagnosis was a clinical one, there being no isolation of virus from the animal nor a study of the neutralizing antibodies in the blood. They further state that, "repeated attempts to demonstrate virus in the Berkefeld filtrates and nasal washings have been negative." Following this they report the isolation of the virus from the nasal washings of a horse 72 and 96 hours after 5 cc. of a 2 per cent suspension of brain from an infected animal was introduced into the nose. In another horse they report virus in the nasal washings 72 and 96 hours after intracerebral inoculation. Since the washings

were not filtered, and since the tests were made by the intracerebral inoculation of guinea pigs, the criteria they used for judging the presence of virus, "high fever, loss of weight, and paralysis," are hardly sufficient. Granted, however, that virus was present in the two inoculated cases, it does not follow that it would be found in the natural disease.

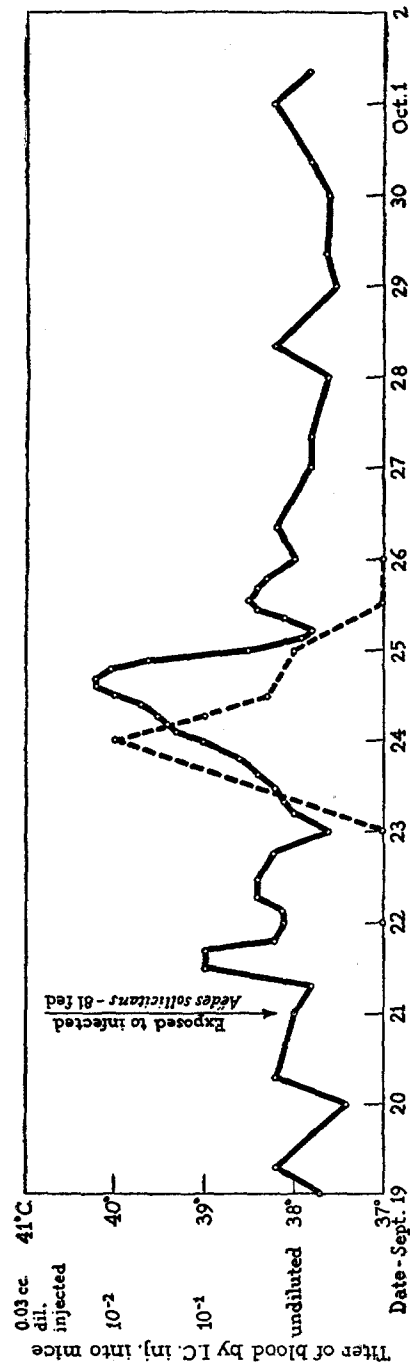
We have, in a limited number of cases, tested for virus the urine, nasal washings, and nasal mucosae from horses killed in the acute stages of the disease. In not a single instance has virus been obtained from such material. It should be noted, however, that Vawter and Records (7) have succeeded in infecting two horses by the intranasal route but that they used relatively large amounts of inocula. Small animals can be readily infected by intranasal inoculation but animals in contact with them do not develop the disease.

Virus in the Blood Stream

From the facts as we know them the disease appears to be transmitted by biting insects rather than by contact infection. Against the theory of insect transmission is the fact that virus has never been demonstrated in the blood of horses showing symptoms of the disease, either by us or by Meyer and his coworkers in the West. Howitt (8) found the virus in the blood of intracerebrally inoculated guinea pigs, monkeys, and a horse during the febrile period. Records and Vawter (6) recovered the virus from the blood of one horse inoculated intracerebrally and of another inoculated intranasally. In Text-fig. 2 are essential data on a horse that was inoculated intracutaneously with virus. It will be seen that there was a diphasic temperature and that symptoms appeared relatively late in the disease. Determinations of the virus in the blood stream showed that the virus content was highest at the first examination which was made during the first febrile reaction and 3 days before the first definite symptoms. Furthermore it will be seen that the virus content decreased rapidly and that it was absent 24 hours before the symptoms appeared and thereafter. Our belief that this is typical of field cases is strengthened by the fact that we have encountered two horses in regions where the disease was occurring that had temperatures but no symptoms. In both instances the blood drawn at this time was shown to contain virus, and in both instances the animals subsequently developed



TEXT-FIG. 2. Horse 194. I. C. = intracerebral injection. I. P. = intraperitoneal injection.



TEXT-FIG. 3. Horse 249.

characteristic symptoms of the disease. Moreover this diphasic temperature with virus in the blood stream during the first rise is found regularly in guinea pigs that are inoculated subcutaneously. It appears to us to be a regular feature of the disease.

In Text-fig. 3 is a chart of another horse which gives us some information about transmission. We shall not discuss the inoculation of this animal as it will be referred to in a subsequent paper. Two days after exposure the temperature began to rise and, as will be seen, at the same time the virus appeared in the blood stream. When the temperature came down virus was no longer demonstrable. There was only this one temperature rise and the animal showed absolutely no central nervous system or other symptoms. Whereas the blood before the exposure contained no virus-neutralizing antibodies, after the temperature rise they were demonstrated to be present. Furthermore some 5 weeks after the temperature rise the animal was inoculated intracerebrally with virus. It showed no temperature rise and no symptoms of the disease and was subsequently disposed of. A control animal inoculated at the same time with the same material developed encephalomyelitis and died. This, then, is an abortive case of the disease and we believe that such are not uncommon, for, as stated earlier, sera from animals that are believed never to have been sick but kept in districts where the disease has occurred, frequently show immune bodies. Guinea pigs inoculated subcutaneously with virus often develop only one temperature rise and show no central nervous system symptoms. When after 2 to 3 weeks they are tested for immunity by the intracerebral route they are found to resist the virus whereas control animals come down. It is obvious that abortive cases in horses are just as much a source of virus for biting insects as animals showing symptoms.

Possible Reservoir Host

The virus curve shown in Text-fig. 2 may not be typical of the natural infection but it probably closely approximates it. It will be seen that the amount of virus falls off rapidly and that the time when biting insects could infect themselves is a relatively short one. If the horse is the only source of virus the transmitters must feed frequently or be present in enormous numbers.

It is possible, however, that the horse is a secondary host, to which the virus is transmitted from another species. Little is known about reservoir hosts in virus diseases but they may be of great importance. The wart hog carries a hog cholera-like virus (9) that it transmits to domestic swine, and Shope (10) has good evidence that pseudorabies is a very mild but contagious disease of swine which is rarely recognized, and that the highly fatal disease in cattle is contracted from infected pigs.

Various animals are susceptible to equine encephalomyelitis virus when it is injected intracerebrally, and rabbits, guinea pigs, and mice can be infected by subcutaneous inoculation. Giltner (11) found the pigeon susceptible to intracerebral inoculation and suggested that it might be involved in the transmission of the disease. We know practically nothing about the susceptibility of the great variety of wild animals and birds that are so closely associated with our domestic animals.

If there is a reservoir host it must be one that covers a large amount of territory, for the disease appears at approximately the same time in areas that are separated from one another by barriers that can be traversed by only a few forms. For example, the Northern Neck of Virginia and the Eastern Shore are separated by the Chesapeake Bay which is from 20 to 25 miles wide, yet the disease has appeared in these two regions at about the same time the past 2 years.

Man is the most widely traveled mammal and must be considered as a possible reservoir host. Meyer (12) suggested that man was susceptible to the virus, for he learned of three human cases of encephalitis that had been in contact with infected horses. No virus was secured from these cases and no tests for neutralizing antibodies were made on the sera from the two cases that recovered. We have made inquiries from doctors practicing in the regions where the disease was prevalent and have been unable to get a history of any human infections that resembled encephalitis. Our experience in the laboratory leads us to believe that man is not very susceptible to the virus. Six of us have been working with the disease for over 2 years, and although precautions have been taken, accidental contacts with infected material have frequently occurred; yet not one of us has developed a disease resembling encephalomyelitis, and the sera of all are free from neutralizing antibodies.

From the above it seems hardly probable that man is a reservoir host for the virus. Birds should be considered as possible hosts and are made suspect by the epidemiological findings. We have, however, no facts that support such an hypothesis and will not consider it further at the present time.

SUMMARY

Equine encephalomyelitis of the eastern type is a disease of the late summer and fall and cases are found in greatest numbers near salt marshes. The epidemiological findings are against its transmission by contact and favor the view that it is insect borne. Although virus can be demonstrated in the blood of infected horses it is present for a relatively short time, and the possibility that the disease is not primarily an infection of horses but that it is transmitted to them from another host is considered.

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