localities where the situation, with respect to climatic conditions for the breeding of arthropod vectors, permits the cyclic chain of events to be completed.

Similarities of the clinical and epidemiologic aspects of some of these diseases and the multiplicity of types and strains of their etiologic agents make it necessary to study elaborately the characteristics of the causal agents, all their possible reservoirs and vectors, and their laboratory diagnosis. The physician and health officer must depend almost entirely on the laboratory for accurate diagnosis, and upon the collaborative efforts of the virologist, epidemiologist, entomologist, and clinician for a complete understanding of these diseases in order to formulate means for their control and prevention.

At present an absolute diagnosis can be made only in one of the following ways:

1. Isolation of the virus from the central nervous system of fatal human cases or horses (in equine encephalomyelitis) involved in the locality. Occasionally virus may be isolated from the blood or spinal fluid of patients if obtained very early in the disease, but this is not dependable. In order to preserve the viability of the virus, material for virus isolation must be frozen (using dry ice) immediately and kept that way until it reaches the laboratory for animal inoculation.
2. Demonstration of the formation of specific antibodies against one of the viruses in the blood of the suspected patient during the course of illness. For this, two blood specimens must be obtained. The first (acute phase specimen) should be obtained as early as possible after onset, preferably within the first 4 days of the disease; the second (convalescent phase specimen), 3 or 4 weeks after onset. At least 20 cubic centimeters of blood should be drawn asceptically; the serum should be separated and saved in the refrigerator in a sterile, sealed container until the second specimen is obtained and the paired specimens, together with appropriate clinical and epidemiologic data, forwarded to the laboratory. No refrigeration is necessary in transit for blood or serum specimens to be tested for antibodies, provided there is no unusual delay. The tests for antibodies are made by complement fixation or neutralization tests. A significant rise in titer between the first and second specimens is accepted as presumptive evidence of infection.

There is no specific therapeutic agent as yet available for any of these diseases. Preventive measures have not been fully formulated but, on the basis of our present knowledge, would consist of vector control and vaccination. Effective vaccines have been prepared for some of these diseases, but their general use is not advocated except in special situations. Studies are in progress with the hope of filling in the many remaining gaps in our understanding of the epidemiology of the encephalitides and ultimately of their complete eradication.

Eastern Equine Encephalomyelitis

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Eastern equine encephalomyelitis is an acute and highly fatal infectious disease caused by a virus which exhibits a wide host range, including horses and mules, pigeons, pheasants, monkeys, and man. It is closely related to, but immunologically distinct from, the viruses of St. Louis encephalitis and Western and Venezuelan equine encephalomyelitis. The virus has a widespread distribution in the tissues of the infected host, but the symptoms of the disease are referable to inva-

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sion of the brain and spinal cord. Differentiation from infection by the related viruses or from other infections of the central nervous system depends upon isolation and identification of the virus or observance of a rise in specific serum antibodies during convalescence.

The typical equine case begins with a short period of high temperature (up to 106° F.) and inappetence, followed by 1 or 2 days of apparent well-being. At the end of this time, a second rise in temperature occurs which usually reaches its peak at about 103° - 104° F. The nervous symptoms appear at this stage. The animal at first appears dazed and will wander aimlessly through fences and into objects or walk in circles. Occasionally it becomes vicious and will attack persons coming near it. Within a few hours facial and pharyngeal paralysis and motor incoordination appear, the animal leaning his head or hindquarters against objects for support, or standing with the feet widely spaced and weaving back and forth. Within 6 to 12 hours the horse falls to the ground, has repeated convulsions, exhibits alternate spasticity and flaccid paralysis of the limbs, and becomes comatose. Death usually occurs within 12 to 72 hours after the onset of nervous symptoms. An occasional animal will recover, but usually shows residual effects such as blindness, partial paralysis, severely impaired "intelligence," or an unstable temperament.

Epizootics of the disease possess certain rather characteristic features. With one exception (Michigan) the disease has appeared only in the coastal areas along the Gulf of Mexico and the Atlantic seaboard. It is a disease of summer and autumn, with a sharp drop in the number of cases with the advent of cooler weather. It usually appears in epizootic form among the equine population. The mortality approaches 90 percent. Accurate morbidity rates are impossible to determine due to inadequacies in reporting; but during one epidemic period, there were approximately 100 cases per 1,000 population among the equines of southwestern Louisiana. The morbidity is much lower in the human population, and there is an apparent predilection for children. Human epidemics may occur in conjunction with an epizootic among the equines and follow the peak of the equine cases by an interval of about 2 weeks or longer. Experimental mosquito transmission has been accomplished, and workers in the virus laboratory at Montgomery, Ala., have demonstrated the virus in nature in *Mansonia perturbans* from Georgia.

Highly efficient vaccines are available for veterinary use. This vaccine may be given to persons who may have special contact with this disease, such as laboratory workers or others unduly exposed.

The U. S. Public Health Service was invited to investigate the medical, veterinary, and entomological aspects of two severe epizootics of Eastern equine encephalomyelitis which affected the horse and mule populations of Louisiana in the summers of 1947 and 1949. During the latter year, the disease extended into central Arkansas where further studies were made. The earliest cases in both instances appeared in late spring (May and June) on the Gulf coast at points separated by approximately 70 miles, the area between consisting of relatively uninhabited swamp and marsh land. Except at the points of origin, the geographical areas involved in these epizootics showed very little overlapping. In 1947 the disease fanned out westward and northward and exhibited a rather sharp northernmost boundary which was approximately 70 miles from the Gulf of Mexico. The western extent involved Texas. In 1949 the disease progressed northward and, to a slight extent, toward the east. It followed the Mississippi Delta region as far north as the middle of Arkansas. Both areas possess a dense horse and mule population as compared to the average of the country. Wild horses, prevalent in both regions, would hinder an adequate immunization program.

Estimates, from various sources, of livestock losses in the earlier epidemic ranged from 3,713 to 11,727. Information from the latter source would indicate that the mortality was about 82 percent. A census taken 2 years previously shows 175,364 horses and mules in the parishes involved in this outbreak. If these figures can be accepted as expressing the equine population in 1947, the morbidity was about 12 percent. These figures, gathered by sending questionnaires to the practicing veterinarians, would not include cases in which a veterinarian was not consulted or those among the wild horses. The general impression was that horses were more susceptible than mules. From six farms on which Eastern equine encephalomyelitis appeared, 13 of 18 horses were affected while only 1 of 27 mules developed the disease. There was no apparent age or sex preference.

The area involved in the 1947 outbreak is flat coastal plain traversed by numerous bayous and streams. The land bordering the Gulf of Mexico is principally salt marsh with an occasional salt dome

or "island" rising above the surrounding plain. The arable land is devoted almost exclusively to the raising of sugarcane. Farther inland the epizootic area consisted of large farms on which the principal crop was rice. The northernmost limit of the epizootic was in the region where the rolling "piney woods" begin. The weather had been exceedingly dry except in the immediate vicinity of the Gulf where short daily thundershowers were the rule. Due to the impassability of the roads in the winter for automobiles, most of the families had one or more riding or driving horses, and horses and buggies were almost as common as automobiles along the roads.

The first cases of encephalomyelitis were observed in May in Iberia Parish (one of the coastal parishes). From here it radiated in a northwesterly manner at a rate which progressed approximately 25 miles every 2 weeks. As the epizootic continued, the interval between the appearance of the first case in an area and the attainment of the peak of infection in that area became shorter, requiring approximately 1 month at the outset and shortening to 2 weeks toward the end of the epizootic. Losses from the disease stopped rather abruptly during the first week of October.

Despite the heavy importation of horses into the area following the epizootic, to replace those lost, the ensuing 2 years revealed few cases of equine encephalomyelitis.

During the second week of June 1949, the Louisiana State Health Department was notified of the existence of 18 cases of encephalomyelitis in horses and mules in Lafourche Parish, an area not too far distant from the point of origin of the 1947 outbreak. By the last of June, cases had been reported from almost every parish in the southeastern part of the State, having progressed as far north as the Mississippi border. The total number of reported cases had reached 202 on July 25, the date of arrival of the CDC epidemiology team. This figure did not include a possible 400 unreported cases among wild horses in the swamps in one parish. On this date the disease had progressed northward to a point almost 200 miles from its origin.

At the time of arrival of the team, the disease seemed to be currently most active in the northern section, and it was decided to conduct the major investigation here. Seven farms on which cases of encephalomyelitis had occurred were surveyed, and serum specimens were obtained from healthy and recovered horses and other domestic animals. Brains removed from sacrificed, acutely ill animals or those recently dead from encephalomyelitis, were prepared for laboratory examination.

The best available information on mortality in this area indicated that more than 90 percent of the affected animals died within 3 days after onset of symptoms, implicating the Eastern type of equine encephalomyelitis, on a clinical basis. This was later confirmed by laboratory investigations. Although there was a heavy mule population in the area, it was affected little by the disease – a fact concurring with observations in previous epidemics. There was no apparent overlapping between the two epidemics. The greatest concentration of cases in the parish surveyed (Avoyelles) existed in an area made up of large farms with long narrow pastures bordered on one side by a bayou and on the other by a large cypress swamp, approximately 4 miles wide and 10 miles long. A substantial horse and cattle population grazed here and had access to the swamp. Another severely affected area, about 15-20 miles west of the area of greatest concentration, consisted of many rice and cotton farms, averaging 30-40 acres each, with a dense human and horse population. There was no body of water within 2 miles, but the rice fields had been irrigated earlier and served as an excellent breeding place for mosquitoes. Egrets and blackbirds were the only wild birds known to be common to both areas.

This epizootic later extended to the Arkansas border and also appeared in central Arkansas, giving the opportunity for further study. The two epizootic areas apparently were not contiguous since no cases were reported in southern Arkansas. The Arkansas outbreak followed the character of that in Louisiana in that each affected region was near a great waterway – the Mississippi River in Louisiana and the Arkansas River and bayous in Arkansas. The preponderance of cases in Arkansas occurred in an area of typical, flat Mississippi Delta country, around England, Ark. A rice and cotton agriculture prevailed, with a heavy mosquito population. This region was separated by the Arkansas River on the west from a wooded, hilly area south of Little Rock, in which a moderate number of cases (35-40) had existed. The reporting in Arkansas was very irregular and no accurate estimate could be made of the total number of cases, nor could any reliable data be obtained on wild bird movements.

One thousand four hundred eighty-nine cases had been reported in the horses and mules of Louisiana as of the first of September. DDT spraying operations for reduction of insect populations in farm buildings, which were proceeding during the outbreak of the disease in Avoyelles Parish, did not appear to reduce the number of cases reported in this area, which rose from 40-42 on July 25 to 650 on September 1. Too few horses were vaccinated with Eastern equine encephalomyelitis vaccine to affect the course of the epidemic in this parish. Whether or not spraying may have had some effect on the incidence of human cases is a matter of speculation. It is interesting that in both Louisiana and Arkansas human cases remained relatively few or unrecognized. Certainly, there were no reports to indicate significant human involvement.

Various species of mosquitoes were collected for virus studies which have not yet been completed. The outstanding feature of the 1949 epidemic was its apparent tendency to follow water courses northward.

Surveys of serum antibodies have led toward the incrimination of wild and domestic birds as a possible reservoir for the virus of Western equine encephalomyelitis. Information in this regard has been lacking for Eastern equine encephalomyelitis. One hundred sixty domestic fowl were sampled in the area involved in the 1947 epizootic in Louisiana and tested for neutralizing antibodies at the Montgomery, Ala., virus laboratory. Only 2 chickens of 109 gallinaceous birds (chickens and turkeys) gave positive titers for neutralizing antibodies against Eastern equine encephalomyelitis. No positive sera were observed in 13 anserines (ducks and geese) or 38 Columbiformes (pigeons). Two cormorants (diving sea birds) showed neutralizing antibodies.

Six of eighteen unvaccinated horses with no history of encephalitic symptoms gave positive neutralization indices. Ten of eleven recovered equines were positive and all of four horses bled in the terminal stages gave positive titers.

SUMMARY

CDC investigated epizootics of Eastern equine encephalomyelitis in Louisiana and Arkansas during the summers of 1947 and 1949. These two epizootics possessed certain things in common. Both started on the Gulf coast within a few miles of each other in the late spring and spread at a rather regular rate covering approximately 25 miles every 2 weeks. Both occurred in districts which have large areas covered by still water such as swamps, bayous, and rice fields. The equine population was dense in each case, the morbidity high, and the mortality about 90 percent. Under apparently equal conditions for exposure, mules were more resistant to the disease than horses. Serum surveys revealed very few domestic fowl with significant antibody titers. Wild fowl, such as wading birds and blackbirds, were common in the epizootic areas and the mosquito population was high. Further investigation, with emphasis on the role of birds and associated arthropod vectors, has been planned by the Virus and Rickettsia Section of the Communicable Disease Center. This will be carried out in the near future in cooperation with Epidemiologic and Veterinary Public Health Services.

Western Equine Encephalomyelitis

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History bears out the fact that Western equine encephalomyelitis has been with us for a long time. As early as the middle of the nineteenth century, western veterinary practitioners recognized and reported outbreaks of a febrile disease of horses, with symptoms comparable to those observed in equine encephalomyelitis today. The literature contains information on about 10 major epizootics

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