Human Equine Encephalomyelitis in Kern County, California 1938, 1939, and 1940*

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IN addition to the known neurotropic virus diseases that have been responsible for much human and animal sickness, medical science has recently added equine encephalomyelitis to those affecting man. In 1930, Meyer, Haring, and Howitt [1] first isolated a virus from the brain tissue of infected horses in California. Meyer [2] had suspected the occurrence of human encephalomyelitis in 1932, when the disease was reported in three men working with horses having encephalomyelitis. No virus, however, was recovered. In 1938 Fothergill, Dingle, Farber, and Connerly [3] reported obtaining the eastern strain of the equine virus from human cases in Massachusetts, while in the same year the relationship of the western strain to man was first established by Howitt [4] in California. Meyer [5] had suggested that, in the lower San Joaquin Valley, cases of encephalitis had been inaccurately reported as poliomyelitis or had been missed. Subsequently, in so far as could be determined by serum neutralization tests as reported by Howitt [6] in 1939 and by diagnostic information from other sources, a different viewpoint was obtained concerning neurotropic virus diseases in Kern County, Calif. Although poliomyelitis predominated, an unexpectedy high proportion of the cases proved to be of the equine encephalomyelitic type, and a small number of the St. Louis type of encephalitis, as demonstrated by means of the serum neutralization tests. A few remained not specifically diagnosed. Thus it was shown that more than one variety of neurotropic virus disease had been prevalent in Kern County.

Seventy-one patients in 1938, 160 in 1939, and 85 in 1940 were admitted to the Kern County General Hospital with a tentative diagnosis of neurotropic virus disease. A few cases were quarantined at home during each of the three years. On the basis of the neutralization tests, the epidemiological histories and the clinical and laboratory findings, 116 cases were segregated as equine encephalomyelitis. There were 22 cases in 1938, 46 in 1939, and 48 in 1940. The recovery of the western equine virus from the brains of 2 patients confirmed the presence of human cases in Kern County.

The neutralization tests employed for the differential diagnosis of the cases were performed according to methods previously described.[6] The strain of western equine virus isolated in Cali-

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fornia in 1938[4] was used throughout the studies, while the St. Louis virus was one originally sent by Dr. L. T. Webster of the Rockefeller Institute. Whenever material could be obtained, 10 per cent suspensions of brain or cord from autopsy cases were inoculated into mice, guinea pigs, and a monkey. Upon recovery of a virus from one or all of these animals, further serological and immunological tests were made to determine the type.

The following report deals with a neurotropic virus study in Kern County, Calif., conducted by the staff members of the Kern County General Hospital and the Kern County Department of Health in the years of 1938, 1939, and 1940, in collaboration with Miss Howitt.

EPIDEMIOLOGY

Unsolved problems concerning the epidemiology of equine encephalomyelitis are numerous, even though further knowledge of the disease has accumulated rapidly. In Kern County, the summer and fall occurrence parallels the seasonal incidence shown in other sections of the country. The reoccurrence of cases in successive years shows a spotty distribution in rural areas. In the farming districts the horse population has been repeatedly exposed to the virus; there are irrigation ditches or other breeding places for mosquitoes that might be incriminated as insect vectors, and there are the human cases. There was no epidemiological evidence to indicate that the virus of equine encephalomyelitis was carried through milk, water, or food. It seemed quite unlikely that secondary cases occurred by direct or indirect contact. For these reasons and because of the limitation of the disease to the warm season of the year, the possibility of its spread by an insect vector, particularly the mosquito, was seriously considered. Evidence to corroborate this theory can be seen in the following typical histories taken from the group of human cases of encephalomyelitis of 1939.

Case 13—S.C.L., male, 5 months of age, was admitted to the Kern County General Hospital on July 15, 1939, because of irritability, stiffness of the neck, convulsions, and fever. Onset of illness was July 8, 1939. Physical findings were normal except for stiff neck, bulging anterior fontanel, and spasticity of the extremities. X-ray of the chest was negative. Spinal fluid showed increased pressure, ground glass appearance, 115 cells, 56 per cent polymorphonuclears and 44 per cent lymphocytes, globulin 4+. The child made a complete recovery. The neutralization test was positive for the western equine virus. This child was from the Buttonwillow district where there had been horses sick with equine encephalomyelitis within a half mile of the home. The child was covered with old and new insect bites which the parents stated were from mosquitoes.

Case 19—G.G., 7 weeks old Mexican female, a neighbor of Case 13, became ill July 28, 1939, and was admitted to the Kern County General Hospital on August 1, 1939, because of chills, fever, convulsions, and neck stiffness. These findings were noted in the hospital stay as well as cyanosis, bulging fontanels, irritability, and difficulty in taking food. Spinal fluid examination on August 5, showed 36 cells, 10 per cent polymorphonuclears and 90 per cent lymphocytes, with 4+ globulin. The child died on August 9, 1939. The gross post-mortem findings were negative. Microscopic pathology was consistent with encephalomyelitis as the anatomical diagnosis. The serum neutralization test was also positive for equine encephalomyelitis. The child had been covered with old and new mosquito bites.

Case 26—C.F., a white male, age 5½ weeks, became ill on August 14, 1939, and was admitted to the Kern County General Hospital on August 19, 1939, because of fever, vomiting, convulsions, neck stiffness, and head cold. Physical findings were normal except for stiffness of the neck, spasticity of the extremities, and slight bulging of the fontanels. Spinal fluid examination of August 20, 1939, showed increased pressure, ground glass appearance, 938 cells, 50 per cent polymorphonuclears and 50 per cent lymphocytes, with 4+ globulin. The serum neutralization test was positive for equine encephalomyelitis. This child had been bitten frequently by mosquitoes, and made a complete recovery.

Case 30—R.B., white male, 30 years of age, farm laborer, resident of McFarland for 2

years. Onset of illness was on August 23, 1939, at which time he had drowsiness, occipital headache, stiffness of the neck, chills, fever, loss of appetite, nausea and vomiting, and weakness of the left arm and left shoulder. Delirium and then unconsciousness began about August 29. A flat chest plate taken August 29, 1939, showed a bronchopneumonia of both upper lobes that cleared uneventfully. A complete recovery was made after 3 months of physical therapy treatment for the left arm and shoulder.

Prior to his illness this man had been working with a sick horse which the veterinarian diagnosed as having equine encephalomyelitis. Neutralization tests on the sera of the patient and the unvaccinated horse were positive for this disease. The man had been sleeping on a haystack near the horse and had been bitten repeatedly by mosquitoes.

CLINICAL PICTURE AND DIFFERENTIAL DIAGNOSIS

Acute anterior poliomyelitis and equine encephalomyelitis were given the most consideration among the neuroptropic virus diseases seen in Kern County during 1938, 1939, and 1940. The diagnosis was clear-cut only in the instances of true paralytic poliomyelitis and in the age group under 1 year for the equine disease. The symptoms and clinical findings for human equine encephalomyelitis were particularly characteristic for cases under 1 year of age. Fever was common with temperatures to 107° F. in fatal and non-fatal cases; convulsions with muscle twitchings and spasms were observed; cyanosis, irritability, drowsiness, tremors, vomiting, muscle weakness, and a residual spastic type of paralysis were noteworthy. The spinal fluid findings usually showed a ground glass appearance with increased globulin to 4+, slight increase in pressure, and white cell counts of 50 to 1,500. The differential white cell count seemed to vary considerably. In 1938, 52.6 per cent of the cases showed a predominance of lymphocytes over polymorphonuclears; in 1939, there were 78.5 per cent, while in 1940, the ratio had changed to 54.7 per cent of the cases with more lymphocytes to 45.3 per cent with more polymorphonuclears.

Because of the high incidence of tuberculosis and coccidioidomycosis in Kern County, Mantoux and coccidiodin skin tests were done routinely on every case in this series. Those having positive reactions to either skin test and considered as possibly having pulmonary forms of either of these diseases were given routine chest x-ray examinations.

The differential diagnosis of the neurotropic virus disease was frequently difficult, so that positive neutralization tests were most helpful. The atypical cases of equine encephalomyelitis and cases of non-paralytic poliomyelitis, particularly in the older age group, caused most concern in differentiation.

In the series of cases studied in Kern County in 1938, 1939, and 1940, the following diseases were considered in the differential diagnosis: the St. Louis encephalitis, lymphocytic chorio-meningitis, infectious mononucleosis, tubercular pachymeningitis associated with generalized tuberculosis of childhood, coccidioidal meningitis, purulent types of meningitis, cerebral accident, luetic meningitis, postvaccinal encephalitis,

TABLE 1

Summary of Neutralization Tests for Equine Encephalomyelitis Among Neurotropic Virus Cases

	Cases of Encephalomyelitic Infection				*Cases of Poliomyelitic Infection*			
Year	*No. of Cases*	*No. Tested*	*No. Positive*	*Per cent Positive*	*No. of Cases*	*No. Tested*	*No. Positive*	*Pre cent Positive*
1938	22	19	15	78.9	27	2	0	0
1939	46	46	42	91.3	73	65	5	7.6
1940	48	47	40	85.1	18	15	0	0
Total	116	112	97	86.6	118	82	5	6.0

encephalitis following measles, and one case of transverse myelitis.

Neutralization tests against the St. Louis encephalitic virus were done on all sera for the first 2 years but were discontinued during 1940. In 1938, 3 sera out of 19 (15.7 per cent) were positive to this virus alone, while in 1939 the numbers had dropped to 4 out of 46 (8.6 per cent). Four sera in 1938, and 5 in 1939 gave positive tests to both the St. Louis and the equine viruses.

Table 1 summarizes the results of the neutralization tests against the equine virus on sera of patients showing symptoms of either encephalitis or poliomyelitis during 1938, 1939, and 1940. Of 112 sera from cases diagnosed as encephalitis, 97 (86.6 per cent) were positive and of 82 sera from cases of poliomyelitis, only 5 (6 per cent) neutralized the equine virus.

During this 3 year period blood was obtained from different groups of so-called normal individuals, mainly relatives or friends who had been in contact with frank cases of equine encephalomyelitis. Certain of these people had manifested symptoms suggestive of a mild attack of a neurotropic virus disease and were tabulated as " sick contacts " as shown in Tables 2 and 3.

TABLE 2

Summary of Neutralization Tests for Equine Encephalomyelitis on Sera of Contacts

	Sick Contacts (Mild Cases of Infection)			*Well Contacts*		
Year	*No. Tested*	*No. Positive*	*Per cent Positive*	*No. Tested*	*No. Positive*	*Per cent Positive*
1938	6	6	100	8	0	0
1939	17	7	41.1	14	2±	14.3
					3±	
1940	18	16	88.9	60	1+	6.7
					5±	
					1+	
Total	41	29	70.7	82		7.3

From a total of 82 well contacts, only 6 sera (7.3 per cent) showed evidence of neutralizing ability. Five of these were weakly positive and only 1 strongly positive. The latter serum came from a man of 29 years who was the father of an infant showing typical symptoms of the disease. The serum of the mother was negative. In fact, it was interesting that of 9 sets of both parents and 5 single parents tested for antibodies to the equine virus, only this man showed a positive test. Most of these parents had infants under 1 year of age who manifested typical symptoms of equine encephalomyelitis and gave positive serum neutralization tests. Their antibodies were therefore not acquired from the mothers.

The individuals listed as sick con-

TABLE 3

Neutralization Tests Among Contacts to Cases of Equine Encephalomyelitis in 1940 According to Age *

	No. of People Tested	*Under 1 Year*		*1–9 Years*		*10–19 Years*		*20 Years and Over*	
		0	+	*0*	+	*0*	+	*0*	+
Sick Contacts	18	0	0	2 (11.1%)	3 (16.6%)	0	7 (38.8%)	0	6 (33.3%)
Well Contacts	60	0	0	8 (13.3%)	1 (1.6%)	15 (25.0%)	1 (1.6%)	33 (55.0%)	2 (3.3%)

* *0* = No neutralization
\+ = Neutralized

tacts should probably have been placed with the group of real cases, since they all showed a mild or abortive attack of some illness, although few of them were hospitalized. However, because they were all either friends or relatives of someone having a more severe form of the equine disease, they were put in a separate group. During 1938, 1939, and 1940, sera from 41 such cases were tested for neutralizing ability and 29 (70.7 per cent) were positive to the virus of equine encephalomyelitis. The figures for 1940 are probably more reliable than for the other 2 years and also included the largest number of sera tested from apparently healthy individuals. That so many of the sick contacts were really mild cases of the same disease, affecting one or more other members in a family, is suggestive of a common source of infection or exposure to similar environmental conditions. That many of these people lived on farms, in labor camps, in box cars, or in small towns with an abundance of mosquitoes in common lends itself to this idea. Of one group of 9 children and adults tested from a migratory camp, 1 had the typical disease, 1 an abortive form with positive antibodies in the serum, while 1 other remained well but gave a weakly positive serum neutralization test. All 3 children were under 5 years of age.

Table 3 summarizes the age distribution for both the sick and well contacts of 1940 and shows the number having neutralizing antibodies for the equine virus in each period.

AGE AND SEX

Among the cases diagnosed as encephalitis in 1938, the range in age varied from 3 weeks to 41 years with 5 (22.7 per cent) of the group under 1 year of age and 50 per cent under 10 years. There were 10 females and 12 males (Table 4) with a mortality of 1 (4.5 per cent).

Among the poliomyelitic cases for the same year, the ages varied from 17½ months to 66 years, with none under 1 year. However, there were 12 cases (44 per cent) under 10 years of age. The youngest of 17½ months had negative neutralization tests for both the equine and the St. Louis viruses. There were 8 females, 19 males, and a mortality of 6 (22.2 per cent).

In 1939 the encephalitic cases ranged in age from 6 weeks to 62 years with 27 (58.6 per cent) under 10 years. Among the group of 8 cases (17.3 per cent) under 1 year of age, the diagnosis was substantiated both by clinical symptoms and the presence of neutralizing antibodies against the equine virus. There were 15 females and 31 males with a mortality of 3 (6.5 per cent). Two deaths occurred among those under 1 year of age.

For the poliomyelitic patients of 1939, the age varied from 10 months to 39 years, with 38 (52 per cent) of the 73 cases under 10 years. There were 27 females and 48 males and a mortality of 3 (4.1 per cent).

In 1940 the cases of encephalitis varied in age from 3 weeks to 63 years, with 16 (33.3 per cent) under 1 year. Twenty-nine (60.4 per cent) were in the group under 10 years. There were 15 females and 33 males with 1 (14.5 per cent) mortality. Only 18 cases of poliomyelitis occurred in 1940 (Table 4).

One may observe especially in Table 4, (1) that males predominated over females in both groups diagnosed either as poliomyelitis or encephalitis, with twice as many males in the years 1938 and 1939 for the latter disease, and (2) the largest percentage of cases diagnosed as encephalomyelitis in all 3 years occurred in the group under 10 years. There were over twice as many cases among the latter group as among adults 20 years or over. The

TABLE 4

Age and Sex Distribution and Mortality Rate for Neurotropic Virus Cases *

Year	Disease	Sex: Male	Sex: Female	Total No. of Cases	Under 1 Yr.	1–9 Yrs.	10–19 Yrs.	20 and Over	Mortality
1938	Polio.	19 (70.4%)	8 (29.6%)	27	—	12 (44.4%)	4 (14.8%)	11 (40.7%)	6 (22.2%)
	E. E.	12 (54.5%)	10 (45.5%)	22	5 (22.7%)	6 (27.2%)	4 (18.1%)	7 (31.8%)	1 (4.5%)
1939	Polio.	46 (63.0%)	27 (37.0%)	73	1 (1.3%)	37 (50.6%)	19 (26.0%)	16 (21.9%)	3 (4.1%)
	E. E.	31 (67.3%)	15 (32.7%)	46	8 (17.3%)	19 (41.3%)	7 (15.2%)	12 (26.0%)	3 (6.5%)
1940	Polio.	10 (55.5%)	8 (44.5%)	18	—	7 (38.8%)	4 (22.2%)	7 (38.8%)	2 (11.1%)
	E. E.	33 (69.1%)	15 (30.9%)	48	16 (33.3%)	13 (27.0%)	7 (14.5%)	12 (25.0%)	7 (14.5%)

* Polio = Poliomyelitis
E. E. = Equine encephalomyelitis

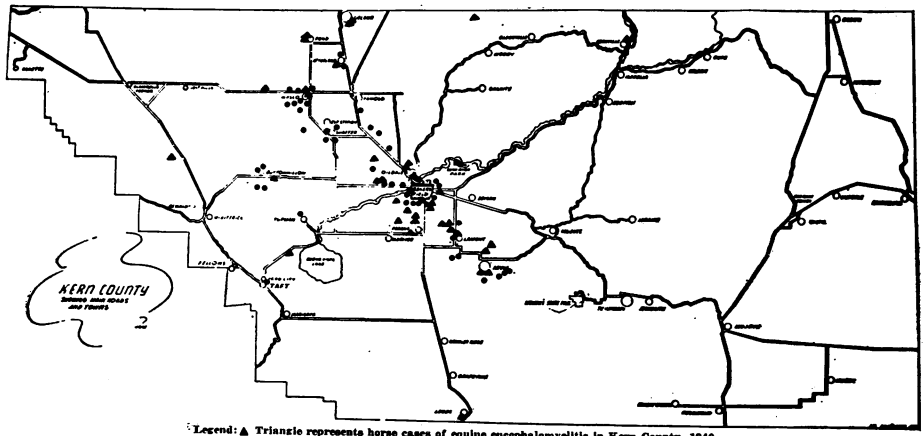
percentage is also high among infants under 1 year.

RACE AND GEOGRAPHICAL DISTRIBUTION

During the 3 years there was nothing significant about the racial distribution of the patients diagnosed as either poliomyelitis or encephalomyelitis. In 1939 there were 4 Negroes and 2 Mexicans among the cases of poliomyelitis and 4 Negroes, 1 Mexican, and 1 Japanese among those of encephalomyelitis. In 1940 there was 1 Negro.

The geographical distribution among the encephalitic groups occurred in definite endemic localities. In 1939 there were 6 cases in the Buttonwillow district. Three of them were 5 months old or under, and all of them were under 5 years of age. This age group and the locality seem significant because of the presence of numerous mosquitoes and of many cases of encephalomyelitis reported among the horses. There were 11 human cases in the Wasco-Shafter area, 6 around Delano-McFarland Pond, and 3 in the Arvin-Lamont-Weedpatch districts. The remainder of the 46 patients were from rural localities outside of Bakersfield. In 1940 the same areas contributed most of the cases of human equine encephalomyelitis as shown in the map, in which a comparison is made of the distribution of human and equine cases.

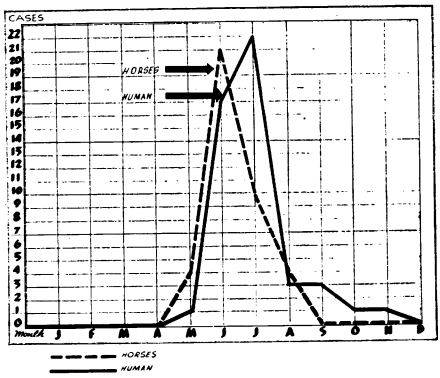
The distribution of poliomyelitis was



Legend: ▲ Triangle represents horse cases of equine encephalomyelitis in Kern County, 1940.
● Dot represents human cases of equine encephalomyelitis in Kern County, 1940.

CHART I

Cases of Equine Encephalomyelitis in Kern County by month of onset in humans and in horses · 1940 ·



noticeably in the more urban districts, with a few cases in the western end of the county.

Of most significance in the geographical distribution was the apparent absence of neurotropic virus diseases in the eastern end of the county. No encephalomyelitis was reported from the dry desert portions lacking in water, while all the known cases were found in the farming regions where water was plentiful. It is noteworthy that for each season the human cases of this disease have occurred in the same endemic localities where there have been both many mosquitoes and cases among the horses.

From the incomplete reports of Kern County veterinarians, approximately 40 cases of encephalomyelitis occurred among the horses in 1938, with fewer numbers in 1939, and again 40 in 1940. Chart 1 compares the incidence of human and equine disease for 1940. It has been exceedingly difficult to correlate specifically the human with equine cases in certain areas, although in the Wasco district in 1940 5 human cases were found within one to two miles of 2 acute cases among horses.

SEASONAL INCIDENCE

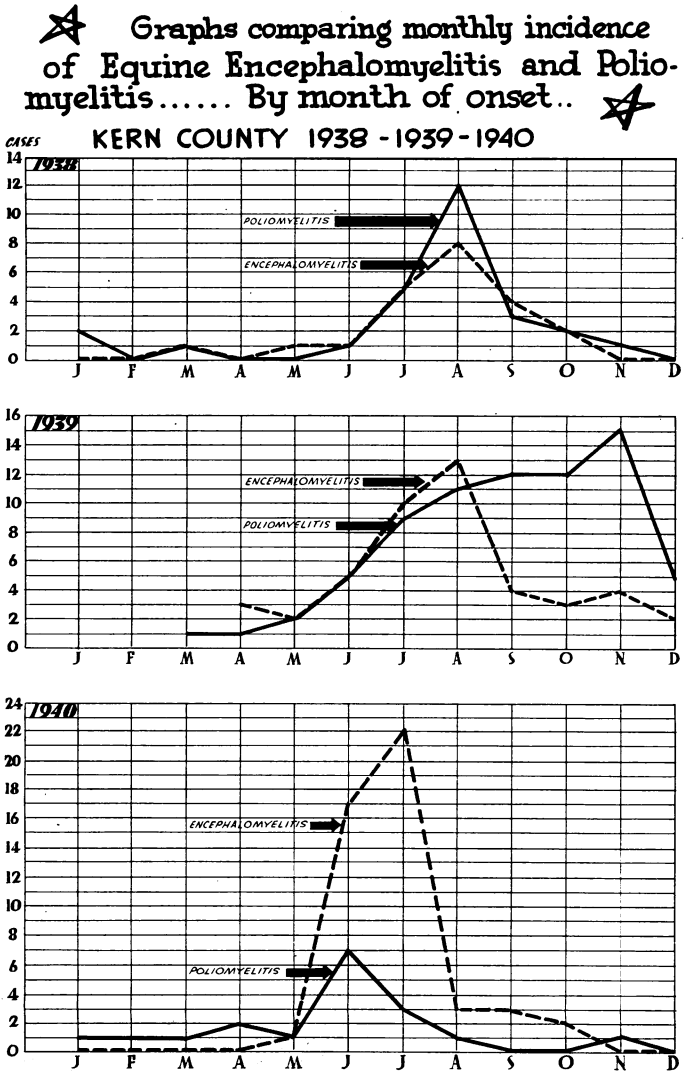
There is a definite seasonal incidence of neurotropic virus diseases in Kern County. In 1938 the number of cases diagnosed as poliomyelitis reached a peak in August, as did also those of the human equine disease as shown in Chart 2. In 1939 there were reported 119 cases of neurotropic virus disease. Compared with the expected number of cases of poliomyelitis based on the mean of reported incidence in 1934–1938, there seemed to be a minor epidemic trend in 1939, but when the cases of poliomyelitis were separated from those of equine encephalomyelitis there was no evidence of an epidemic. The peak of encephalomyelitis in 1939 was in August, and that of poliomyelitis in November. This unexpectedly late incidence for the latter disease had been noted also for 1934 and 1935 when the summer heat was prolonged into the autumn months.

In 1940 the peak of the 48 human equine cases occurred in July, with 22 reported, followed by an abrupt decline. In each year the cases began to appear about May, increasing each month to a peak in July or August, and then falling off in September, but continuing with a few sporadic cases through October and November if the weather remained warm.

TREATMENT

Active treatment during the acute and following stages of the diseases was entirely nonspecific. For the severe convulsions a variety of sedatives were used, the most beneficial being chloral hydrate by rectal instillation and soluble phenobarbital given intravenously in doses of 1½ to 2 grains. During 1938 sulfanilamide was given routinely, either parenterally or by gavage, during the period of illness. Later its use was limited to the first few days of illness until the possibility of bacterial meningitis had been excluded. During the comatose state parenteral fluids and gavage feedings were administered ac-

CHART II



cording to necessity. Small transfusions and intramuscular blood were given routinely. A thorough muscle check was given in the physiotherapy department to those cases recovering at the end of 21 days, and treatment instituted when needed. Later spasticity or weakness of the extremities was treated by physiotherapy or application of casts. A periodic check followed discharge from the hospital in every case with apparent normal recovery.

DISCUSSION

It is evident that the disease of equine encephalomyelitis has been present among the human population of Kern County, Calif., for a number of years, as demonstrated by finding neutralizing substances in the sera of a few individuals with onset of symptoms in 1934 or 1935.[6]

While the results of this study are based largely upon the outcome of the neutralization tests, yet the presence of

the equine virus was proved in 1940 by recovery of this western strain from the brain material of 2 patients, one an infant of 7 months (F.J.M.), and the other an adult of 63 years (T.F.). The disease in the infant ran a typical course with fever, irritability, retraction of the head, lethargy, tremors and convulsions, the child becoming comatose and dying in 5 days after the onset. The adult showed headache, drowsiness, fever, coma, and death in 12 days after the onset of the disease. Suspensions of the brain stem of the 2 cases yielded the virus of western equine encephalomyelitis when inoculated into mice, guinea pigs, and monkeys.

Although the eastern virus of equine encephalomyelitis was the first to be reported in man,[3, 7] it is apparent that the western strain has been infecting people over a larger area of the United States, as shown by reports based mainly on the results of serum neutralization tests or of clinical findings alone.[8-12] Fulton, however, has reported obtaining the virus from human brain and serum in Canada.[13] That there is more of this human disease along the Pacific slope than surmised is evidenced by reports from the State of Washington where cases have been found in the Yakima Valley.[14] In California the disease in man has become prevalent since 1937,[6] a total of 43 cases having been reported in the literature so far; 3 by Meyer,[2] 32 by Howitt,[6] 2 by Davis,[15] and 6 by Cope and Maytum.[16] These cases of human equine encephalomyelitis have occurred mainly in the two large central California valleys, with Kern County as a principle focus at the lower end. Since 97 encephalitic cases in this county have given positive neutralization tests against the western equine virus during the past 3 years, the evidence is strongly suggestive of a permanent endemic area unless some control measures are instituted.

SUMMARY

It has been found that the western virus of equine encephalomyelitis is endemic among the human as well as the horse population of Kern County, Calif. Although cases of poliomyelitis predominated among those having neurotropic virus diseases, by means of the serum neutralization test it has been shown that for the years 1938, 1939, and 1940, respectively, of 112 encephalitic cases that were tested, the sera of 97 (86.6 per cent) were positive for the virus of equine encephalomyelitis. Of 82 cases diagnosed as poliomyelitis, 5 (6 per cent) were also positive for the equine virus.

The sera of 6 out of 82 (7.3 per cent) well contacts and those of 29 of 41 (70.7 per cent) sick contacts (mild cases of infection) had neutralizing antibodies to the same virus.

The largest percentage of cases diagnosed as having equine encephalomyelitis during all 3 years came in the group under 10 years of age, with a noticeable number below 1 year. Males predominated over females among the encephalitic patients as well as among those having poliomyelitis.

The western virus of equine encephalomyelitis was recovered from the brain material of 1 infant and 1 adult during 1940.

TABLE 5

Sex Distribution of Neurotropic Virus Cases

	Equine Encephalomyelitis			*Poliomyelitis*		
Year	*No. of Cases*	*Male*	*Female*	*No. of Cases*	*Male*	*Female*
1938	22	12 (54.5%)	10 (45.5%)	27	19 (70.4%)	8 (29.6%
1939	46	31 (67.3%)	15 (32.7%)	73	46 (63%)	27 (37%)
1940	48	33 (69.1%)	15 (30.9%)	18	10 (55.5%)	8 (44.5%)

The cases of human equine encephalomyelitis in Kern County began about May, increasing to a peak in August for the years 1938 and 1939, and in July for 1940, dropping off abruptly, with only a few through November. All cases were centered in the farming and irrigated areas in the central and western portions of the county, with none reported from the mountains or the eastern desert districts.

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REFERENCES

1. Meyer, K. F., Haring, C. M., and Howitt, B. F. The Etiology of Epizootic Encephalomyelitis of Horses in the San Joaquin Valley, 1930. *Science,* 74:227, 1931.

2. Meyer, K. F. A Summary of Recent Studies on Equine Encephalomyelitis. *Ann. Int. Med.,* 6:645, 1932.

3. Fothergill, LeR., Dingle, J. H., Farber, S., and Connerly, M. L. Human Encephalitis Caused by the Eastern Variety of Equine Encephalomyelitis. *New England J. Med.,* 219:411, 1938.

Webster, L. T., and Wright, F. H. Recovery of the Eastern Equine Encephalomyelitis Virus from Brain Tissue of Human Cases of Encephalitis in Massachusetts. *Science,* 88:305, 1938.

4. Howitt, B. F. Recovery of the Virus of Equine Encephalomyelitis from the Brain of a Child. *Science,* 88:455, 1938.

5. Meyer, K. F. Personal communication.

6. Howitt, B. F. Viruses of Equine and of St. Louis Encephalitis in Relationship to Human Infections in California, 1937–38. *A.J.P.H.,* 29:1083., 1939.

7. Feemster, R. F. Outbreak of Encephalitis in Man Due to the Eastern Virus of Equine Encephalomyelitis. *A.J.P.H.,* 28:1403, 1938.

8. Wesselhoeft, C., Smith. E. C., and Branch, C. F. Human Encephalitis; Eight Fatal Cases with Four Due to the Virus of Equine Encephalomyelitis. *J.A.M.A.,* 111:1735, 1938.

9. Ecklund, C. M., and Blumstein, A. The Relation of Human Encephalitis to Encephalitis in Horses. *J.A.M.A.,* 111:1734, 1938.

10. Breslich, P. J., Rowe, P. H., and Lehman, W. L. Epidemic Encephalitis in North Dakota. *J.A.M.A.*. 113:1722, 1939.

11. Larimer, R. N., and Wiesser, E. G. Human Equine Encephalomyelitis. *J. Iowa M. Soc.,* 29, 287, 1939.

12. Platou, R. V. Equine Encephalomyelitis in Infancy. *Am. J. Dis. Child.,* 60:1155, 1940.

13. Fulton, J. S. Relation of Equine Encephalomyelitis to the Epidemic of Human Encephalitis in Saskatchewan in 1938. *Canad. Pub. Health J.,* 32:6, 1941.

14. Hammon, W. McD., and Howitt, B. F. Unpublished report.

15. Davis, J. H. Equine Encephalomyelitis (western type) in Children. *J. Pediat.,* 16:591, 1940.

16. Cope, J. H., and Maytum, H. Equine Encephalomyelitis. *California & West. Med.,* 53:82, 1940.