Arthropod-Borne Encephalitis in 1956

E. RUSSELL ALEXANDER, M.D., and WALTER A. MURRAY, JR., M.D.

DURING 1956, the prevalence of arthropod-borne encephalitis varied considerably in the United States. Eastern equine encephalitis (EEE) returned in a small outbreak of human cases in southeastern Massachusetts, where 12 cases occurred, 8 of them fatal. Although the disease was widespread among horses and pheasants all along the Atlantic and Gulf Coasts from Massachusetts to Louisiana, only 3 other human cases were reported—2 from Maryland and 1 from Delaware. Western equine encephalitis (WEE) affected man and horses in a sporadic and sparse fashion over the western half of the country. St. Louis encephalitis (SLE) was the largest disease problem; there were several small foci in the West, with a large epidemic focus in the high plains of Texas, and an urban epidemic in Louisville, Ky., accounting for the majority of the cases.

The incidence of acute infectious encephalitis in man is reported routinely to the National Office of Vital Statistics, Public Health Service, by all States on a weekly basis. Because of the different criteria used by reporting States, this composite category embraces a crude collection of diseases of central nervous system infections including the arthropod-borne encephalitis group. The 1956 total of 2,624 cases listed according to State serves as a starting point for case appraisal (table 1). This number is the largest annual total reported for the disease category. Final totals have exceeded 2,200 cases only in 1954 and 1955. Marked annual variation in incidence for the years 1952 and 1956 is seen in figure 1, where sharp peaks in the even years reflect the known larger epidemic occurrence of the arthropod-borne encephalitides (*1*). Further statistical analysis of the 1956 report data has proved of limited usefulness in appraisal of the arthropod-borne encephalitides.

In 1956, encephalitis in horses produced 1,284 clinical cases, with 493 deaths, as reported to the U. S. Department of Agriculture (*2*). Cases and deaths listed by State in table 2 reflect reports from widely scattered endemic foci in large geographic areas of the West, as well as highly fatal cases concentrated on the eastern seaboard. Typical of the outbreaks were those in the Columbia River Basin in Washington, western Idaho, and adjoining counties of eastern Oregon. States reporting high mortality were localized in the Atlantic and Gulf Coast sections. That EEE was the cause of the high case fatality rate (80–90 percent) was proved by laboratories reporting isolations of the virus from these areas. In sections of known WEE prevalence, appraisal of reported cases is more difficult; this disease has a much lower mortality rate (15–35 percent), and therefore fewer brain specimens are submitted for laboratory diagnosis.

Eastern Equine Encephalitis

Appraisal of the 15 human cases of EEE reported in 1956 resulted in the classification of 13 as confirmed and 2 as presumptive (table 1). The death of 10 of the patients and incapacitating sequelae in three survivors testify to the severity of this disease. A study of geographic distribution of cases shows a concentration in Massachusetts, where there were 12 cases, the

Dr. Alexander, formerly chief of the Surveillance Section, Epidemiology Branch, Communicable Disease Center, Public Health Service, is now with the department of pediatrics at the University of Chicago. Dr. Murray serves as assistant chief of the Surveillance Section of the Center.

majority occurring in the southeastern part of the State in an area characterized by numerous fresh-water swamps. In 1938 the same area had 34 human cases of EEE, 25 of them fatal (*3*). The 1938 and 1956 outbreaks represent the largest and second largest, respectively, both for the State and for the Nation. One of the 1956 cases occurred in Delaware, and 2 in Maryland.

Seven laboratories within 10 Gulf and Atlantic Coast States reported a total of 42 isolations of EEE virus from horse brain specimens, the majority from New Jersey and Massachusetts (table 2). A wide range of seasonal occurrence is represented by isolations from Louisiana in May, and from New Jersey in early November. Although no virus isolations were reported from horses in Alabama and Florida, strong presumptive evidence of EEE is reflected in the reported mortality in excess of 90 percent (table 2).

Followup studies in 1956 on outbreaks of EEE in domestic pheasants resulted in 42 virus isolations from brain specimens submitted from Massachusetts, Rhode Island, Connecticut, and New Jersey.

EEE virus was isolated from 20 serum specimens collected from wild birds during ecologic studies in four States—Massachusetts, Rhode Island, New Jersey, and Louisiana (*4–8*). A few of the 20 virus isolations were from immature birds, and about equal numbers were from permanent-resident and migrant birds.

Natural isolations of EEE virus were obtained from 11 pools of arthropods collected in three States. From Massachusetts, five pools of unengorged *Culiseta melanura* were positive (*7*). Two pools of unengorged and one of engorged *C. melanura* collected in New Jersey, and two of engorged *Culex salinarius* collected in that State, yielded virus when tested in the Communicable Disease Center laboratory (*9, 10*).

These isolations incriminate *C. melanura* as the most probable vector of EEE, since it is the only species thus far to yield multiple isolations of virus from unengorged mosquitoes. This species was in high prevalence in both the Massachusetts and the New Jersey areas. Studies have indicated that their blood meals were primarily from birds and rarely, if ever, from man or horses. Therefore, this species may be the prime vector between birds and the occasional vector to humans or horses. It may be, however, that another species less often infected transfers the disease to these mammalian hosts.

Isolations of EEE virus were made from arthropods collected in Georgia, in July 1956 (*11*). For the first time, the virus was isolated from *Aedes mitchellae* and a pool of mixed species of insects from the genus *Culicoides*, and, for the second time, from *Anopheles crucians*. The separation of engorged specimens from the processed pools was not reported.

Western Equine Encephalitis

The sporadic and endemic occurrence of WEE in the western half of the United States during 1956 resulted in reports of 47 cases, 37

Data Collection

For the past decade, the Communicable Disease Center of the Public Health Service has conducted intensive field studies on the arthropod-borne encephalitides. Beginning in 1955, a systematic effort was made to collect data on the occurrence of these infections throughout the country. Sources included reports from the National Office of Vital Statistics of the Public Health Service, State and local health departments, academic and many other medical and veterinary virus diagnostic laboratories, the Disease Eradication Branch of the Department of Agriculture, and the Walter Reed Army Institute of Research. A report summarizing all the material was then distributed to contributors and others concerned with the control of these diseases.

This nationwide surveillance and continued mutual exchange of data resulted, in 1956, in five concurrent seasonal reports and an annual summary report, which are available on request to those interested in encephalitis research. By defining the pattern of arthropod-borne encephalitis in 1956, it is hoped that the public health importance of these diseases will be further clarified.

The present paper summarizes the information on the occurrence of arthropod-borne infections in 1956 and includes a summary of data for past years.

Classification Standards

In order to compare encephalitis activity in the different States from which varying amounts of data were submitted, standards were adopted for the classification of reported cases. These standards are flexible in recognition of the inevitable minor differences in technique between laboratories. The "confirmed" category includes all cases fulfilling the following criteria:

1. Isolation of virus.
2. Fourfold rise in titer of antibodies from acute to convalescent blood specimens.
3. A fourfold fall in titer during the convalescent stage.
4. A single high titer of complement fixing antibodies in a single convalescent serum collected from an area of proved concurrent epidemic.

The "presumptive" case includes all cases demonstrating any of the following:

1. A single high complement fixing antibody titer in an individual with clinical illness compatible with arthropod-borne encephalitis.
2. Case history of clinical encephalitis without any laboratory diagnosis in an area of proved concurrent epidemic.

The only criterion accepted as evidence of current animal infection (horses, pheasants, wild birds, mosquitoes, and other mammals) was isolation of virus.

of them confirmed (table 1). These cases, occurring in 10 States, showed a restricted, early seasonal occurrence, with a peak in mid-August. Analysis of age distribution revealed a concentration in infants (table 3). No fatalities were reported among either confirmed or presumptive cases.

Western equine encephalitis in horses was confirmed by reports of 2 isolations of the virus from brain specimens, 1 in Oklahoma and 1 in Washington. In the California encephalitis surveillance reports diagnoses were confirmed in fourfold, or greater, rises in antibody titer in clinical cases in horses (*12*). Including serologic and histopathological diagnoses, the U. S. Department of Agriculture lists another 33 instances of confirmation of diagnoses in 10 States (*2*). It was possible to accept as confirmed cases only those with virus isolation because of the difficulty encountered in the interpretation of serologic results of single blood specimens submitted without knowledge of the immunity status. The extensive use of vaccines, the difficulty in obtaining adequate history of their use, and the relatively sparse horse populations discourage reliance on reported clinical cases of WEE in horses. Characteristic case fatality of 25 to 35 percent was recorded in the clinical reports of the 1956 epidemics in the Columbia River Basin.

Eight species of wild birds yielded WEE virus in Rhode Island, New Jersey, and Louisiana, as reported by research groups who are working primarily on the ecology of EEE (*4–6*, *8*). For Rhode Island, the isolation of WEE from a chukar partridge is the first demonstration of this virus in the State, but in the other two States there were previous isolations.

The encephalitis surveillance program of the California State Department of Health reported isolations in 1956 of WEE from brains of four grey squirrels.

During 1956, WEE virus was isolated from 307 pools of mosquitoes collected in New Jersey, North Dakota, Texas, Nevada, Colorado, Idaho, Washington, and California, with the most isolations from the last four States. All isolations were from pools of *Culex tarsalis* except 2 in Texas which were from *Culex quinquefasciatus*, and 1 from *C. melanura* in an engorged pool in New Jersey.

Some measure of comparison with previous years is available from reports of the California State Department of Health. Mosquitoes collected in similar fashion from year to year in four county study areas reveal the following:

	1954	*1955*	*1956*
Pools tested	989	1,113	1,047
Pools positive	238	82	145
WEE virus	151	68	143
SLE virus	87	14	2

In terms of WEE virus recovery from mosquitoes, 1956 was not a light year, yet few human cases occurred.

St. Louis Encephalitis

The occurrence of 562 cases of SLE, 227 confirmed, makes this disease paramount among

Table 1. Encephalitis by State during 1956

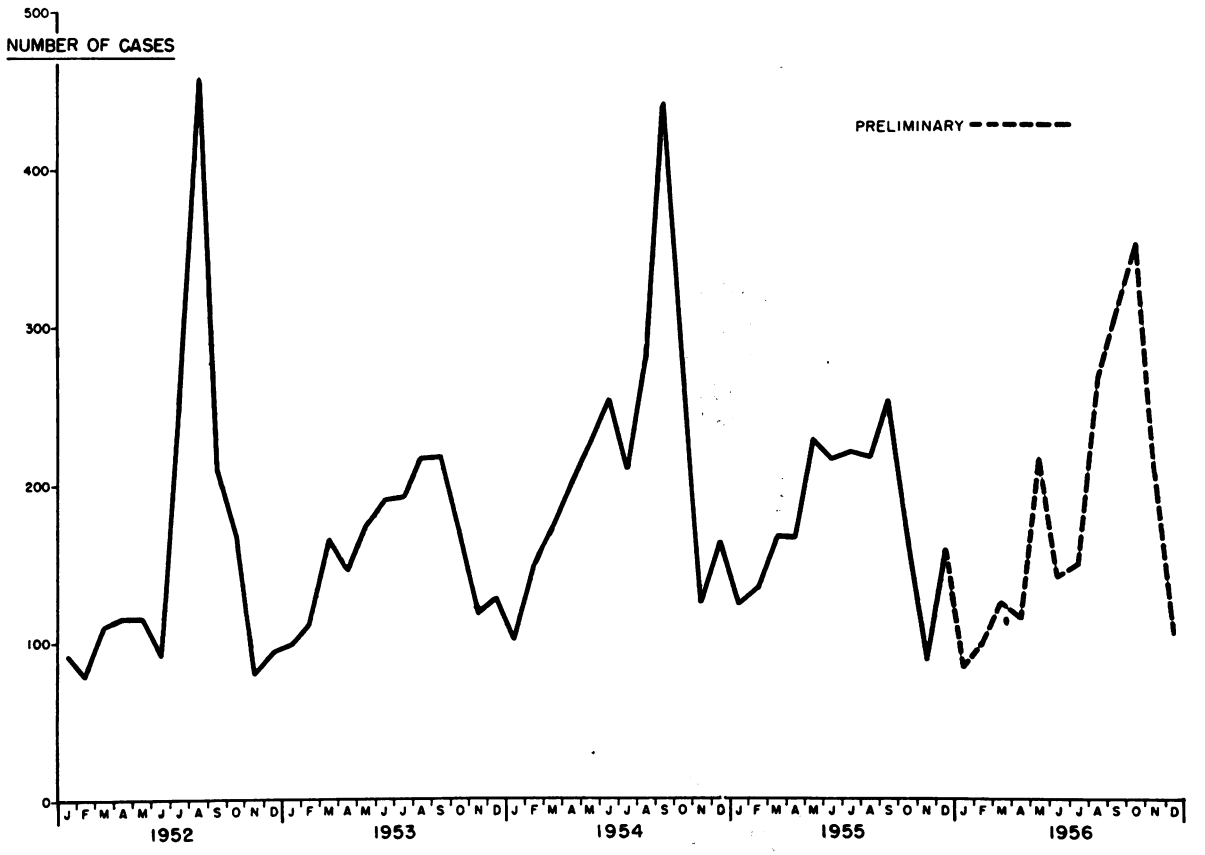
State	Report data [1]	Cases appraised as arthropod-borne encephalitis						Fatal cases
		EEE		WEE		SLE		
		Confirmed	Presumptive	Confirmed	Presumptive	Confirmed	Presumptive	
Maine	5							
New Hampshire	1							
Massachusetts	35	11	1					[2] 8
Rhode Island	5							
Connecticut	10							
New York	386							
New Jersey	34							
Pennsylvania	1							
Ohio	125							
Indiana	74					20	4	
Illinois	146							
Michigan	85							
Wisconsin	13							
Minnesota	8			2		2		
Iowa	24							
Missouri	15						2	
North Dakota	16							
South Dakota	14			1		2		
Nebraska	19							
Kansas	136				5	12	42	[3] 3
Delaware		1						
Maryland	27	1	1					[2] 2
District of Columbia	10							
Virginia	27							
West Virginia	4							
North Carolina	34							
South Carolina	21							
Georgia	17							
Florida	15							
Kentucky	145					64	56	[3] 14
Tennessee	27					1	3	[3] 1
Alabama	27							
Mississippi	19							
Arkansas	19							
Louisiana	4							
Oklahoma	16							
Texas	346			15	2	89	193	[3] 9
Montana	4							
Idaho	7			2	1	1	1	
Wyoming	3			2				
Colorado	66				1	24	33	[3] 3
New Mexico	7				1			
Arizona	7							
Utah	6					1		
Nevada	2						1	
Washington	29			1		3		[3] 1
Oregon	24					1		
California	559			14		7		
Total	2,624	13	2	37	10	227	335	41

[1] Reported Incidence of Notifiable Diseases in the United States, 1956, Morbidity and Mortality Weekly Report, Annual Supplement, vol. 5, No. 53, National Office of Vital Statistics, Public Health Service. No reported or appraised cases from Vermont.

[2] Eastern equine encephalitis.

[3] St. Louis encephalitis.

Figure 1. Reported infectious encephalitis cases in the United States, 1952–56.



the arthropod-borne encephalitides in 1956 (table 1). Two large epidemics accounted for the majority of these cases. In the high plains area of the Texas Panhandle, a rural epidemic included over 250 cases of clinical encephalitis. Of these, 89 were confirmed in the laboratory as SLE and 15 as WEE, while the remainder were classified as presumptive. During an urban epidemic in Louisville, Ky., 110 people were affected. Other small epidemic foci were in Colorado, Kansas, Indiana, and southern Kentucky (at the Tennessee border), and sporadic cases were reported from 11 other States. Seven of 31 deaths were confirmed, 2 by virus isolation from brain tissue (table 1). Following the epidemic in Louisville, a 6 percent prevalence of complement fixing antibodies was found in serums from over 700 persons. From this, a crude estimate shows that the ratio of inapparent to apparent infections was more than 200 to 1.

There were 26 isolations of SLE virus from pools of mosquitoes reported in 1956, still sparse in comparison with reported isolations of WEE. All were made from pools of *C. tarsalis* collected from widely distributed foci in North Dakota, Kansas, Idaho, Colorado, Utah, Washington, and California. Considerable numbers of mosquitoes were collected in Texas and in the Louisville area but no SLE virus was obtained. No SLE virus isolations were reported from wild birds or other animals during that year.

Discussion

A composite of the total virus activity in 1956 for each of the three arthropod-borne encephalitides, as shown in figure 2, represents the sum of all evidence for activity of each specified virus in certain mammals, wild or domestic birds, and mosquitoes.

In some States the only evidence of EEE virus activity in an area was the excess mortality

in horses, but this evidence, although presumptive in nature, has proved remarkably reliable in the past. In addition, the report data on horses more clearly reflect seasonal occurrence than do data on human cases.

In 1956, the farthest extension southward of human cases of EEE was in Maryland, and the southernmost outbreaks in pheasants occurred in New Jersey. In contrast, during 1955, human cases of EEE were reported from Texas and Louisiana, and pheasant outbreaks from North Carolina and Florida. The seasonal occurrence of human cases was restricted to August and September in 1956; during 1955, onsets in an identical number of reported cases ranged from April to December. The 1956 epidemic in Massachusetts reached a peak during the first week of September, while the four cases reported from Massachusetts in 1955 had onset dates in late September and early October. This may have resulted from a late seasonal increase in mosquito prevalence attributed to the mid-September hurricane Diane. The epidemic peak in 1938 was reached shortly before a mid-September hurricane. Also in contrast with previous years, increased interest and improved laboratory techniques in 1956 have generated an impressive number of EEE virus isolations from wild birds and mosquitoes.

The distribution of WEE virus in 1956 followed expected patterns except for an extension into Rhode Island, as represented by a single isolation from a chukar partridge (fig. 3). For the second year this virus was active in New Jersey, where an impressive number of isolations were made from wild birds and from one pool of mosquitoes (*4–6,9,10*). These isolations demonstrate the extension of WEE virus activity to eastern shores and give fair warning signals for careful surveillance in these areas for both human and equine cases of this disease.

Again in 1956, as in recent years, an even more restricted interepidemic reporting of human and horse cases occurred, but substantial virus recovery from mosquitoes in several areas indicated that natural reservoirs were maintained and became widely dispersed.

Throughout much of the West, an abundance of the most efficient WEE vector, *C. tarsalis*, and the indications of highly susceptible human populations makes the disease a threat of considerable magnitude. This is especially true in rapidly developing irrigated areas, which may result in the introduction of a greater number of susceptible humans, more wild birds, and new breeding sites for *C. tarsalis*.

The irrigated areas of California contained a considerable number of pools of *C. tarsalis* with WEE virus in 1956. The fact that there were few human cases has been attributed by some to the effectiveness of the mosquito control program in the State, which, although not

Table 2. Reported encephalitis in horses, by State, in 1956

State	Reported cases[1]	Total deaths[1]	Number confirmed by virus isolation
Massachusetts	46	46	[2] 8
Rhode Island	9	9	[2] 2
Connecticut	1	1	[2] 1
New Jersey	48	46	[2] 11
Indiana	10	2	----
Illinois	7	----	----
Michigan	19	1	----
Minnesota	14	----	----
Iowa	15	10	----
Missouri	28	----	----
North Dakota	8	----	----
South Dakota	50	4	----
Nebraska	20	4	----
Kansas	24	4	----
Delaware	25	17	[2] 3
Maryland	33	33	[2] 3
Virginia	28	----	----
North Carolina	34	19	[2] 1
South Carolina	27	25	[2] 3
Georgia	44	32	[2] 8
Florida	107	87	----
Tennessee	11	11	----
Alabama	33	32	----
Mississippi	9	9	----
Louisiana	37	35	[2] 2
Oklahoma	36	14	[3] 1
Texas	133	----	----
Montana	35	----	----
Idaho	103	11	----
Wyoming	29	9	----
Colorado	25	5	----
New Mexico	5	----	----
Arizona	16	----	----
Utah	16	2	----
Nevada	8	2	----
Washington	69	21	[3] 1
Oregon	65	1	----
California	57	1	----
Total	1,284	493	[2] 42 [3] 2

[1] Data from Animal Disease Eradication Division, U. S. Department of Agriculture.
[2] Eastern equine encephalitis.
[3] Western equine encephalitis.

Table 3. Age distribution of encephalitis cases under age 10

Age group (years)	Eastern equine encephalitis			Western equine encephalitis			St. Louis encephalitis								
							Mountain and Pacific			East Central			West Central		
	C	P	Total	C	P	Total	C	P	Total	C	P	Total	C	P	Total
0–1	2		2	9		9		1	1				1	19	20
1–2	1		1	2		2								4	4
2–3	1		1								2	2	1	6	7
3–4	1		1				1	1	2		1	1	1	11	12
4–5	1	1	2	2		2	1		1	1		1	1	6	7
5–6				1		1	3	3	6	2		2		3	3
6–7	1		1	3	1	4								6	6
7–8	1		1				1		1	1		1		4	4
8–9	1		1	2		2	1		1				2	3	5
9–10	1		1	1		1	3		3				1	1	2
Total	10	1	11	20	1	21	10	5	15	4	3	7	7	63	70

C=confirmed; P=presumptive.

reducing the reservoir of the virus in nature, is effective in preventing transmission to man. Another point of view holds that the incidence in humans is not directly related to the virus infection rate in mosquitoes, that it has direct relation to the immunity status of the exposed population. Further evidence will be needed to clarify this point.

Although sizable widespread outbreaks of SLE were recorded in 3 States and sporadic cases in 11 others, activity of this virus was not described outside previously known areas (fig. 3). For an analysis of SLE characteristics in different areas, cases were arranged into the following geographic divisions: East Central States (Indiana, Kentucky, and Tennessee), West Central States (Minnesota, Missouri, South Dakota, Kansas, and Texas), and Mountain and Pacific States (Idaho, Colorado, Utah, Nevada, Washington, Oregon, and California).

A study of the sex distribution of cases in these areas reveals markedly equal incidence in males and females in West Central and Mountain-Pacific States (males, 221; females, 220), but a notable predominance of cases in females in the East Central States (males, 49; females, 99). This is a reflection of the data from Louisville, but this same predominance among females was not found in a recent serologic survey.

Marked contrasts occur in age distribution of cases of SLE in these areas (fig. 4). In the East Central States there is a paucity of cases among children and young people, with peak incidence in the 40- to 70-year age groups. This pattern is typical for 1933 and for the past 2 years. In this area the epidemiological characteristic most likely to suggest SLE is the initial report of seasonal encephalitis in older persons. In sharp contrast, epidemiologists in western States report a typical preponderance of SLE cases in the under-10-year age group, but with a weighting toward the upper level of this age bracket. It also contrasts sharply with the age pattern for WEE and EEE, both of which most frequently affect infants (table 3).

The West Central States have a larger proportion of presumptive SLE cases, as reported from extensive occurrences in Texas and Kansas (fig. 4). In the light of WEE virus activity in these areas, some cases classified as presumptive on clinical grounds in these major SLE epidemic areas actually may have been WEE. This might account for some of the cases in the under-10 age group, and particularly those in infants (table 3). To ascertain the true distribution of SLE, incidence figures should include only confirmed cases. To determine whether the age distribution pattern for SLE in the West Central States adheres to the pattern in the Mountain and Pacific States or to that in the East Central States, or a mixture of the two, requires more exact data.

The seasonal occurrence in these three geo-

Figure 2. Geographic distribution of arthropod-borne encephalitides in man and animals in the United States, 1956.

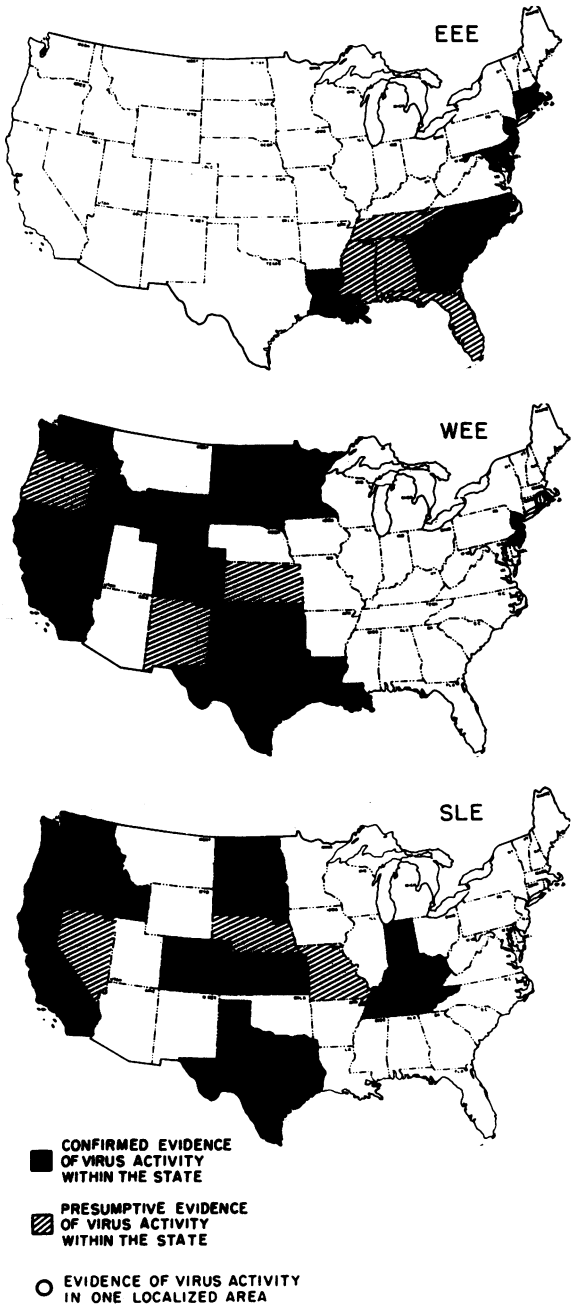
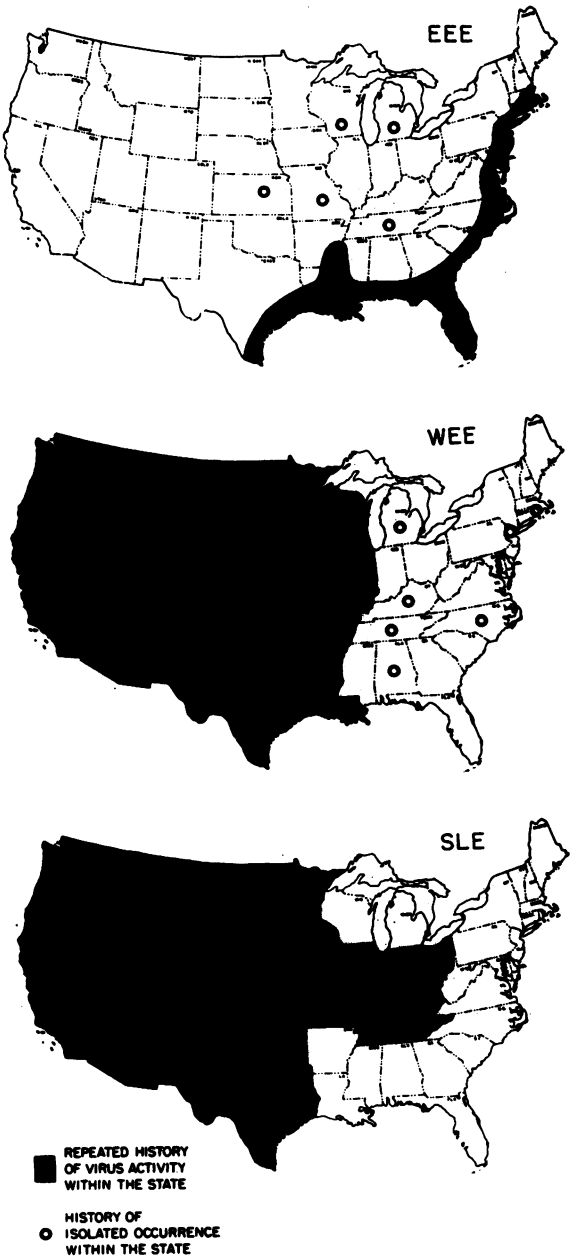


Figure 3. Historical pattern of arthropod-borne encephalitis virus activity, by State, through 1956.



graphic areas reveals a situation similar to that in the age distribution study (fig. 5). Peak occurrence was in August in the Mountain-Pacific States following the August peak of WEE. Also consistent with previous reports was the September peak for SLE in East Central States, with abrupt cessation of activity even before killing frosts. In confirmed cases from West Central States, SLE activity is faithfully reflected in the late August peak, but the addition of presumptive cases shifts the curve to a later period. As with the age distribution data, this again is a dilution of the analysis with presumptive cases, which in-

Figure 4. Age distribution of St. Louis encephalitis in 1956, by region.

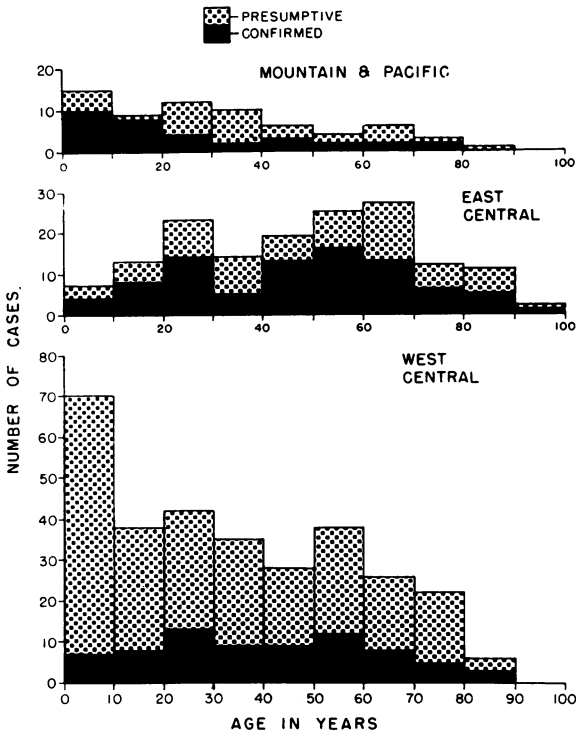
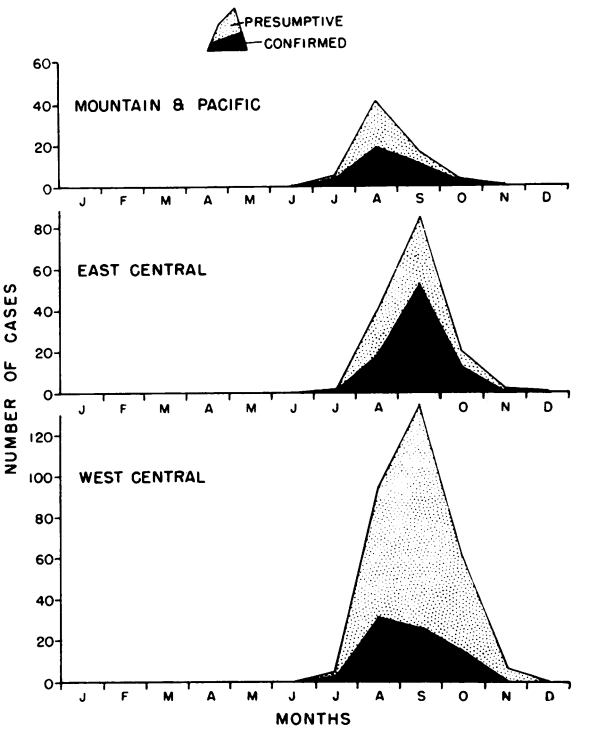


Figure 5. Monthly case occurrence of St. Louis encephalitis in 1956, by region.



cluded many with date of report rather than date of onset.

A problem concerned with arthropod-borne encephalitis illnesses is the severity of sequelae after apparent and, possibly, inapparent infections. In certain States, information on sequelae is being obtained as a part of research studies. A longitudinal clinical followup study in California has reported that permanent sequelae are mainly confined to those persons who were in the under-10 group at the time of infection, and that the frequency of severe sequelae appears to be greatest in infants, particularly in infants under 3 months of age (*13*).

It is interesting to speculate on the possible explanations of the differing epidemiological characteristics in these two areas. Two contributing factors may be operative. First, SLE in Mountain and Pacific States probably is transmitted primarily by *C. tarsalis*, whereas in Eastern Central States the vector is probably *Culex pipiens* or *C. quinquefasciatus*. The characteristics of these species regarding field or house environment may be important, and it is possible that the continued passage of the same virus through species with different incubation periods may lead to minor strain variations. Some laboratory investigations would suggest that such strain differences can be seen in virus isolated in those two geographic areas, although there is no antigenic differentiation.

The second factor to consider is the accumulating knowledge on the immunity of resident populations in the two areas. In certain endemic locations in the West there are significantly higher neutralizing antibody rates to SLE, with little history of clinical disease, than in comparable populations in the East Central States. Although the explanation for the differences in age distribution is not clear, and it is probably more complex than it appears, the fact that there is a significant difference between the two areas is important to record and study.

Studies of the hidden public health problems which may be presented by these diseases are in progress. Increased emphasis on diagnosis of aseptic meningitis may help elucidate cases that are often reported as viral encephalitis.

In many laboratories, work is advancing on the problems of the unidentified viruses, of isolated viruses whose potential for causing disease is unknown, and of disease agents that react with serums of patients from past epidemics of "unidentified encephalitis." Likewise, the problem of encephalitis of unknown etiology might be attacked by better analyses of the diseases reported as encephalitis, particularly the parainfectious encephalitides.

A real problem today resides in arthropod-borne encephalitides that are not now seen in the United States but might be imported. For example, in 1956 an illness in an individual, who became sick on arrival in California from Okinawa, was later diagnosed as Japanese B encephalitis, and in 1955 a case of Venezuelan encephalitis was reported in a laboratory worker in Washington, D. C. The public health hazard in such instances is the possibility of mosquitoes becoming infected, permitting survival and propagation of the virus locally. This, plus the possible introduction of infected mosquitoes, points up the need for increased knowledge and surveillance of arthropod-borne viruses by those responsible for the public health.

Summary

By charting the pattern of arthropod-borne encephalitis during 1956 through surveillance activities, it is hoped the public health importance of these diseases will be further defined.

The increased incidence of the arthropod-borne encephalitides in 1956 was characterized by:

The sporadic occurrence of EEE in horses throughout the Atlantic and Gulf States, with restriction in humans to 12 cases in southeastern Massachusetts, 1 in Delaware, and 2 in Maryland. Substantial virus isolations were recorded from mosquitoes and wild birds in Massachusetts and New Jersey.

Western equine encephalitis occurred diffusely in nature, as reflected by recovery of virus from mosquitoes. Although it failed to spill over into man and horses in epidemic fashion, it still remained a severe disease threat in irrigated areas of the West.

St. Louis encephalitis was the main contributor to the high incidence of reported acute infectious encephalitis. Although the virus was difficult to recover from participants in the natural cycle, the disease was readily apparent in a large rural epidemic in Texas and in three smaller foci in Kansas, Colorado, and Indiana. SLE reappeared in an urban epidemic in Louisville, Ky., for the first time since 1937.

Numerous public health problems, those stemming from encephalitis of unknown etiology, for example, and the possible importation of foreign encephalitides require further study.

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WHO Tenth Anniversary Publications

On the occasion of the tenth anniversary of the World Health Organization, the agency has prepared a variety of information materials, described briefly in the following list. Unless otherwise indicated, all items are available at the Office of Public Information, Pan American Sanitary Bureau, Regional Office for the Americas, 1501 New Hampshire Ave., NW., Washington, D. C.

- A series of basic fact sheets on WHO structure and programs, issued in quantity about every 2 weeks.
- A folder entitled "WHO, What it is, What it does." The last page presents in tabloid form, "Ten Years of World Health."
- An illustrated brochure, "Ten Steps Forward." It contains 40 pages of text written by a well-known English science writer and 24 pages of photographs. The publication is sold for 50 cents through the Columbia University Press, International Document Service, 2960 Broadway, New York 27, N. Y. Glossy prints may be obtained from the Pan American Sanitary Bureau.
- A 500-page book in commemoration of the tenth anniversary, covering all phases of WHO. At the end of May 1958 the book will also be available at the Columbia University Press.
- A book on WHO and the public health problem in the Americas and Western Africa. Written by Murray Morgan, a reporter from the State of Washington, it is published by Viking Press under the title "Doctors to the World." Available in May 1958 at regular bookstores.
- *World Health* magazine, formerly the WHO newsletter, special anniversary edition including 80,000 copies in English. The publication will be distributed in May and June 1958.
- A tenth anniversary film, in black and white, running for about 55 minutes, and especially tailored for television showing.
- A tenth anniversary set of 12 photographs entitled "World Health Advances." Each is supplied with an explanatory caption and packed in a large envelope.
- "Pictures of Health," a small booklet using the photographs which appear in the picture set. It is designed for use as a throwaway complementary to the WHO folder.