SOUTHERN MEDICAL JOURNAL
JOURNAL OF THE SOUTHERN MEDICAL ASSOCIATION
PUBLISHED MONTHLY BY THE SOUTHERN MEDICAL ASSOCIATION AT BIRMINGHAM, ALA.

Volume 31 MAY 1938 Number 5

POLIOMYELITIS: FACTS AND FALLACIES*

By L. L. LUMSDEN, M.D.†
New Orleans, Louisiana

"What is truth?" asked Pontius Pilate, and did not tarry for an answer.

To the finite mind a sharp differentiation between fact and fallacy is often difficult and sometimes impossible. We accept as fact that which according to all available evidence appears so, and as fallacy that which according to some or all established evidence appears not so. A review of the exhaustive and exhausting literature on poliomyelitis leaves any open-minded, unbiased person in doubt as to what is so and what is not so among many of the statements therein presented "authoritatively" as positive conclusions or strong probabilities.

This paper has been prepared with constant effort to that thorough open-mindedness becoming a humble student of a great problem—and poliomyelitis is a great vital problem. Its main objective is to encourage further study of available data, to urge further practical work and to help open wide the door to reasonable thought and constructive speculation on what appear to some of us as the unsolved phases of the problem.

For purposes of discussion the following premises are submitted:

(1) We are not sure whether what we call poliomyelitis is one disease or a group of two or more diseases;

(2) We do not know whether it is an ancient or a comparatively new disease;

(3) We do not understand the nature of the causative factors;

(4) Our knowledge of the pathogenesis is incomplete and indefinite;

(5) The diagnosis of the disease as it is designated and reported is often uncertain and frequently erroneous;

(6) Our knowledge of the geographical distribution in the United States and other countries, even in recent years, is incomplete;

(7) We do not know the main source or sources of the infection nor, with certainty, whether the disease is infectious;

(8) We do not know definitely the usual mode or modes of spread, the portal or portals of entry into the human body nor whether it is directly or indirectly communicable from person to person;

(9) Various methods proposed for specific treatment are of uncertain value, and

(10) Many of the measures proposed and frequently adopted for prevention or control appear unreasonable, unwarranted, futile and, in some instances, dangerous.

ENTITY

The different behaviors of different outbreaks suggest at least the possibility that what we call poliomyelitis is a group of two or more diseases caused by different agents operating or spreading in different ways and as distinct from each other as typhus fever and typhoid fever now are recognized to be.[1] In this connection the recent designation[2] of benign lymphocytic choriomeningitis and of meningoencephalitis as disease entities are of interest. Laboratory experiments appear to have demonstrated that there are at least three or four immunologically distinct strains of the virus which generically is accepted by most of our leading laboratory workers as the cause of poliomyelitis.[3]

HISTORY

Some of the authors of published works on poliomyelitis favor the idea that the disease is of

*Read in Section on Public Health, Southern Medical Association, Thirty-First Annual Meeting, New Orleans, Louisiana, November 30–December 1-2-3, 1937.

†Medical Director, United States Public Health Service.

ancient origin, even suggesting that "its virus is as old as man, and has dwelt always in the mucous passages of his nose and throat."[4] Most of the authors appear to agree that it probably is comparatively new among diseases of epidemic potentialities. Some of the argument presented to indicate the antiquity of the disease appears flimsy. The much referenced carving on an Egyptian stele made about 1300 B.C. of Ruma, a Syrian priest, presents a picture of deformity resembling some of those which follow poliomyelitis. Among the expert interpreters of this classic some argue that the affliction was the result of poliomyelitis and others that it was more likely the result of congenital deformity or of tuberculosis.[5] Of course, it is possible that the affliction portrayed was merely symbolic and intended by the sculptor to indicate that Ruma did not do well in his profession while he walked the earth.

From the recorded history it seems probable that the malady occurred sporadically for many years before, but did not assume epidemic proportion anywhere until about 1905. Accounts of the reported incidence of the disease in different countries are published in Public Health Bulletin No. 91 for years prior to 1917, and in the League of Nations Epidemiological Reports for recent years. Between 1904 and 1916 endemics, outbreaks or epidemics with a total of over 1,000 cases within a year were reported in Sweden (in 1905 and 1911), in Norway (in 1911), in England (in 1911) and in New York City (in 1907). The largest epidemic ever reported in one locality was that in the New York City vicinity in 1916. Why this disease or group of diseases called poliomyelitis gathered terrific epidemic momentum about 1905 in a few regions or countries of the world is not known or even surmised.

CAUSATION AND PATHOGENESIS

The most generally promulgated and the most highly authenticated[6] view regarding the causation of poliomyelitis is (1) that the etiological agent is a specific filterable virus, whose only habitat is the human being, (2) that the invariable source of the infection is the nose and throat discharges of infected persons and of human carriers, (3) that the infection is usually conveyed through direct personal contact but rarely may have as its immediate source articles (including unpasteurized milk) recently soiled with the nose and throat discharges of persons harboring the virus, and (4) that when the virus (presumably from a carrier in most instances) is introduced into the nose or nasopharynx of a susceptible person, it enters the brain by way of the olfactory nerve and bulb and traveling through nerve fibers or tracts locates eventually in certain cells of the spinal cord or the brain, or both, which it damages and thereby produces the disease. This view rests fundamentally and entirely or almost entirely on findings from experiments in monkeys. To some of us it does not appear to square with the facts obtained by epidemiological studies of the disease among human beings. It seems too elastic, too restful.

There are significant differences of opinion among recognized laboratory research workers on the problem regarding the nature of the etiological agent, the sources of the infection, the modes of spread, the portals of entry to the human body and the courses taken by the virus to reach its cells of election after it gains entrance to the human body. Some have presented what they think impressive evidence that the etiological agent is a bacterial micrococcus[7] and others contend vigorously and persistently that in one stage of its life cycle it is a microscopically visible streptococcus or a coccoid form of a streptococcus and in another stage an ultra-microscopic virus.[8] Some, after prolonged intensive laboratory work with monkeys and other animals, have reached the conclusion that the portal of entry of the virus to the human body is usually not through the olfactory nerves, but through the sympathetic nerves from the intestinal tract.[9]

Most of the authorities* appear convinced that when the virus is introduced into the blood or lymph circulation in any part of the body or into the gastro-intestinal tract in sufficient quantity to produce disease it must travel up to and enter afresh the olfactory nerve terminals in the roof of the nose in order to reach its cells of election in the spinal cord. This seems a most remarkable course for any living thing to take in its struggle for existence. Yet upon such conviction or upon the belief that the virus invariably enters the human body by way of the nose is based the advocacy of nose sprays as a prophylactic. The usual freedom from pronounced or even observable lesions of the olfactory bulbs of persons who have died from poliomyelitis,[10] the absence of such lesions also from the olfactory bulbs of monkeys in which what is regarded the characteristic disease has been produced by introduction of the virus into any part of the body other than the nasal fossa,[11] the reported production of the disease in monkeys by injecting

*An "authority" may be defined, for our present practical purposes, as one who has written or spoken much on a given subject.

the virus in small doses into an area near an injured sciatic, femoral or vagus nerve,[12] the distribution of the paralysis in some cases in children following the use of, and by some skilled observers definitely attributed to, an attenuated virus which, with prophylactic intent, had been injected subcutaneously into the arms of children[13] and the general and usual epidemiological features of the disease all appear opposed to the hypothesis that poliomyelitis is a contagious disease spread among human beings by nose-to-nose or any other direct personal contact.

It is evident that the different interpretations by different research workers of the findings from experimentation on monkeys furnish much interesting ground for debate. In view of the dosage of virus usually employed to produce the disease in monkeys, a practical student of the problem unlearned in high laboratory technic may wonder if any of the findings from experimentation on monkeys have any particular significance with respect to the spread or occurrence of the disease under usual and natural conditions in persons. Man and monkey do differ from each other in some respects.

On epidemiological grounds alone, it appears conceivable that poliomyelitis is not caused by a living micro-organism or a virus, but by a toxin somewhat comparable to that of tick paralysis.[14] Such concept might be reconciled with the production of a poliomyelitic disease in monkeys by the inoculation of them with spinal cord substance from persons who have died from poliomyelitis by hypothesizing that the toxin from the human being either activates or makes virulent or pathogenic a virus usually present in and harmless to the monkey or sensitizes the nerve cells of the monkey to pathogenic invasion by such virus and after the virus so has been made pathogenic it usually continues pathogenic, and, to increasing degree, in passage from monkey to monkey. On similar grounds, it seems also conceivable that pollen or rusts or food products from certain plants are causative factors. Such "wild" hypothesizing may lead to constructive speculation and constructive speculation may lead to work and work may lead to truth.

The seasonal incidence, the geographical distribution, the rarity of traceable connection between cases, the low rate of incidence, even in severe epidemics, compared to that of the diseases established as contagious, and all other evidence, except the laboratory monkey evidence, appear to support much more an hypothesis that poliomyelitis is spread to man by some wide-ranging biting insect from some lower wide-traveling or widely transported animal which serves as the main reservoir of the infection than they do the iterated and reiterated hypothesis—too often presented in the guise of fact or of definite conclusion—that the disease is spread only by persons, mainly carriers, and that only persons with some thymus, lymphoid, gonad, mongoloid or other peculiar condition fall victim to it.

It now seems that we might well give thought to taking out of the discard and carefully reconsidering the work of Rosenau and Brues[15] and of Frost and Anderson,[16] in which they appeared to succeed in producing poliomyelitis in monkeys by exposing them to bites of stable flies (*stomoxys calcitrans*) captured in nature. Their experience may have been comparable to that of Carlos Finlay in his transmission of yellow fever virus by mosquitoes.

The efforts to reconcile the contagion hypothesis with the geographical distribution, seasonal incidence and other factual features of the disease appear to some of us more and more to compose a structure comparable to a pyramid of straw with the big end up. The contagion hypothesis may be right, but proof of it is yet lacking, and for proof of this or of any of the other hypotheses applied to the causation of poliomyelitis work—much more work—is needed.

GEOGRAPHICAL DISTRIBUTION AND SEASONAL INCIDENCE

As the reported incidence of poliomyelitis in different countries is presented more or less fully for most of the previous years in works already published, the detailed discussion in this paper of geographical distribution is confined mostly to the incidence in the last four years in the United States and especially in the Southern states.

According to all the records since poliomyelitis was recognized, the high incidence generally and all of the major epidemics have been in the temperate and subarctic countries. The United States, Sweden and Norway have been the most severely afflicted countries among those from which reports are recorded. Both the endemicity and the epidemicity appear to have been singularly and consistently low in the tropics. The considerably exploited outbreak on a small isolated island (Nauru) located on the equator in the Pacific Ocean[17] appears of doubtful identity. The physician who was there at the time and who reported the outbreak expressed the opinion that the disease was not poliomyelitis.[18]

The reported incidence of the disease in the United States during the last four years is inter-

esting. In the four years 1934 to 1937, inclusive, there was reported for the 41-week periods ended about October 15 a very marked excess of cases over the four-year average in different groups of contiguous or nearly contiguous states as follows:

In 1934, in Montana, Idaho, Washington, California, Nevada and Arizona; in 1935, in Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Virginia, North Carolina and Kentucky; in 1936, in Tennessee and Alabama; in 1937, in Pennsylvania, Ohio, Indiana, Illinois, Wisconsin, Minnesota, Iowa, Missouri, Nebraska, Kansas, Colorado, Mississippi, Louisiana, Arkansas, Oklahoma and Texas. Thus the distribution of excess incidence in 1934 and 1935 was latitudinous, in 1937 both latitudinous and longitudinous and in 1936 remarkably localized (see *Table 1* and *Map 1*).*

In both the North Temperate Zone and the South Temperate Zone regions of comparatively high incidence, poliomyelitis is very largely a disease of the warm weather months. Of the very small proportion of cases reported in the cold weather months, a good many, perhaps, can be explained by erroneous diagnosis. There appears, however, no strong reason to doubt that a few local cases and even small localized outbreaks do occur at times in cold weather. The outbreak reported in the winter of 1916-17 in Elkins, West Virginia,[19] is of special interest, being, perhaps, even out of line with the hypothesis of "autarcesis" of Aycock.[20]

The usual and general seasonal incidence and the geographical distribution are among the most striking features of the epidemiology of the disease. The higher incidence in the regions with greater seasonal fluctuations in temperature and the higher incidence in those regions in warm weather seasons have been explained "authoritatively," and often quite glibly, as due to an increase in community susceptibility as the weather changes from cold to warm. The lower incidence in the tropical and semi-tropical regions and in the more temperate regions adjacent to them has been explained, similarly, as due to warm weather being favorable to the life and spread of the virus with a consequent greater constant degree of saturation with and immunization against the infection among the populations in those regions. These explanations were applied especially to the United States to account for the regularly higher incidence in the

*Annual incidence rates of reported cases, by states, for the five years 1931-1935 are shown graphically in the League of Nations Epidemiological Reports (R. E. 150) of 1935, page 212.

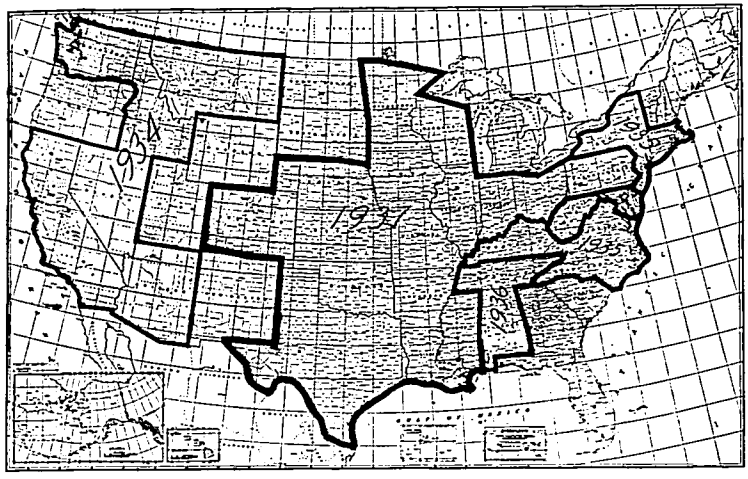
Table 1

POLIOMYELITIS CASES REPORTED IN EACH STATE*

Division and State	Forty-one Weeks Ended— Oct. 13, 1934	Oct. 12, 1935	Oct. 10, 1936	Oct. 16, 1937
All states†	6292	9296	3111	8449
New England:				
Maine	15	119	35	125
New Hampshire	5	52	3	21
Vermont	5	33	9	26
Massachusetts	66	1240	50	347
Rhode Island	1	304	2	14
Connecticut	14	351	12	95
Middle Atlantic:				
New York	203	2615	171	606
New Jersey	62	416	26	144
Pennsylvania	105	160	55	307
East North Central:				
Ohio	221	59	150	524
Indiana	48	30	39	138
Illinois	176	201	483	743
Michigan	155	530	98	417
Wisconsin	59	55	35	252
West North Central:				
Minnesota	67	58	22	267
Iowa	26	44	47	204
Missouri	29	30	36	330
North Dakota	10	10	12	6
South Dakota	35	7	10	26
Nebraska	13	13	16	188
Kansas	59	24	48	234
South Atlantic:				
Delaware	3	5	1	8
Maryland	20	57	24	81
District of Columbia	5	78	7	23
Virginia	62	661	47	57
West Virginia	75	37	40	63
North Carolina	38	634	39	95
South Carolina	10	28	16	21
Georgia	17	18	87	68
Florida	14	15	27	30
East South Central:				
Kentucky	97	273	56	121
Tennessee	50	71	303	107
Alabama	42	50	359	66
Mississippi	20	12	110	278
West South Central:				
Arkansas	11	21	9	316
Louisiana	13	86	22	108
Oklahoma	11	10	11	414
Texas	102	64	53	615
Mountain:†				
Montana	255	5	15	26
Idaho	115	1	13	11
Wyoming	7	2	6	38
Colorado	15	9	44	196
New Mexico	16	6	18	20
Arizona	102	15	5	19
Utah	11	6	3	23
Pacific:				
Washington	617	26	57	58
Oregon	61	13	25	43
California	3030	662	282	525

*Data from table in Public Health Reports 52:1520 (Oct. 29) 1937.

†Exclusive of Nevada, from which state no report was received.



Map 1
Markedly excessive reported incidence of poliomyelitis by years and states in the four-year period 1934-37.

Northern than in the Southern states. The outbreaks in North Carolina, Virginia and Kentucky in 1935, those in Alabama, Tennessee and Mississippi in 1936 and those in Mississippi, Arkansas, Oklahoma and Texas in 1937 upset these "explanations," and, thus, observed obstinate fact caused favored hypothesis to pass into the realm of fallacy.

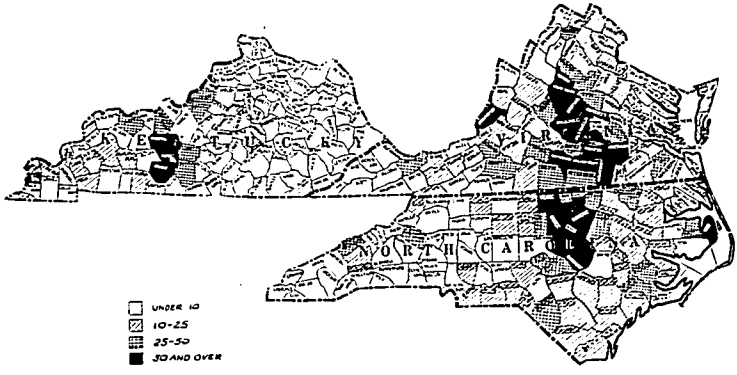
The periods of occurrence, the total numbers of cases reported and the incidence rates per 100,000 population in these outbreaks were as follows:

State	Period	Number of Cases	Incidence Rate
North Carolina	May 1 to Nov. 30, 1935	648	21.2
Virginia	May 1 to Nov. 30, 1935	674	27.6
Kentucky	July 1 to Nov. 30, 1935	316	12.1
Alabama	May 1 to Nov. 30, 1936	374	14.1
Tennessee	May 1 to Nov. 30, 1936	362	13.8
Mississippi	May 1 to Nov. 30, 1936	129	6.4
Mississippi	May 1 to Oct. 31, 1937	345	17.1
Arkansas	May 1 to Oct. 31, 1937	316	17.0
Oklahoma	May 1 to Oct. 31, 1937	411	16.2
Texas	May 1 to Oct. 31, 1937	584	10.0

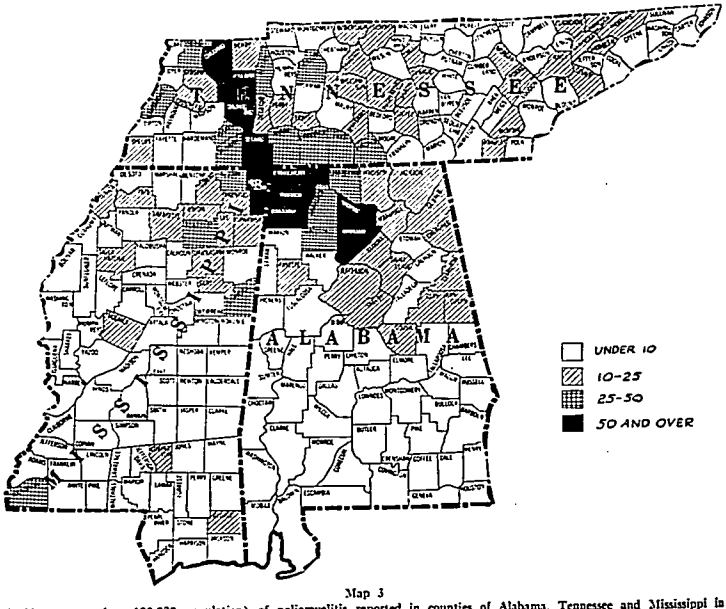
These outbreaks or epidemics in our Southern states were remarkably regional in their distribution (see *Maps 2, 3, 4* and *5*).

In the outbreak of the summer of 1935 in North Carolina and Virginia the high, or epidemic, incidence was in a region comprising two adjacent groups of counties, one in the north central part of North Carolina and one in the south central part of Virginia. The outbreak in Kentucky that same summer was in the west central part of the State. The outbreak in North Carolina, beginning about May 15, anteceded that in Virginia by about a month, and the outbreak in Kentucky followed that in Virginia by about a month. To say that in these outbreaks or in those in the other Southern states in the summers of 1936 and 1937 the disease "spread along lines of human traffic" is to say something which means nothing and which essentially is fallacious. In these days, such traffic goes in all directions and there was much human traffic between the affected regions and the non-affected regions.[21]

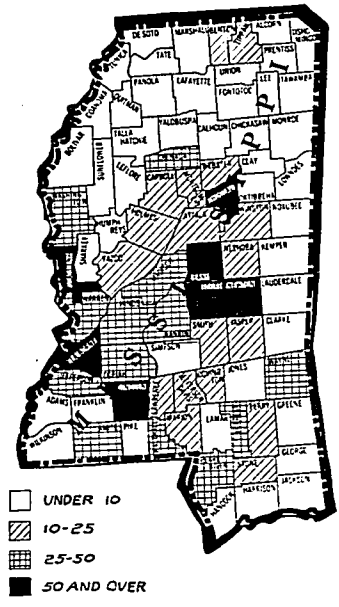
In the outbreaks in the summer of 1936 in the South, the high, or epidemic, incidence was mainly in a region comprising three adjacent groups of counties, one group in the northwestern part of Alabama, one in the west central part of Tennessee, and one in the northeastern part of Mississippi. The 7 counties in Alabama, the 10 in Tennessee and the 2 in Mississippi with



Map 2
Incidence rates (per 100,000 population) of poliomyelitis reported in counties of North Carolina, Virginia and Kentucky in the outbreak periods of 1935.



Map 3
Incidence rates (per 100,000 population) of poliomyelitis reported in counties of Alabama, Tennessee and Mississippi in the period May 1 to November 30, 1936.



Map 4

Incidence rates (per 100,000 population) of poliomyelitis reported in counties of Mississippi in the period May 1 to October 31, 1937.

the highest ranks in incidence (with rates over 30 per 100,000 population) are all in this one region.

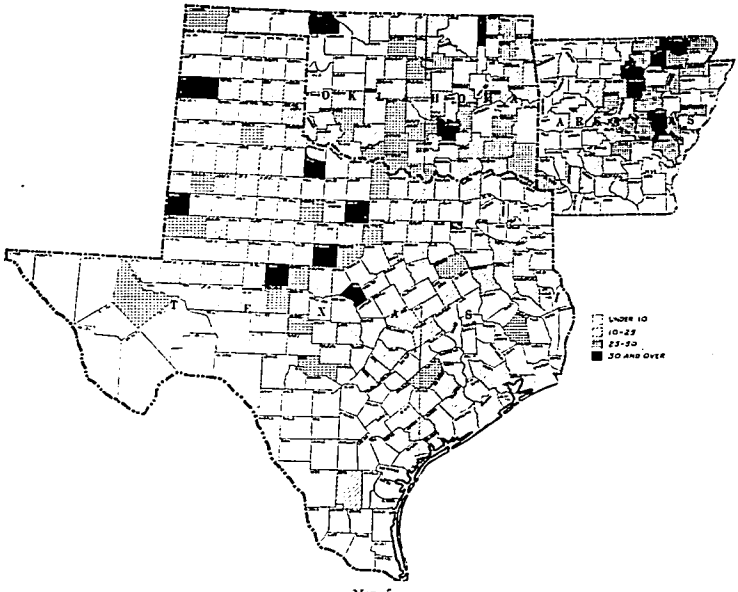
In Mississippi, exclusive of the three counties with the highest incidence rates, all in the northeastern part of the State, Tishomingo with 12 cases and an incidence rate of 73.1, Lowndes with 11 cases and an incidence rate of 36.6 and Alcorn with 7 cases and an incidence rate of 29.5, in no county were more than 6 cases reported and the incidence rate for the whole State was only 5.1 per 100,000 population.

In Tennessee, exclusive of the ten counties with the highest rates, all in the west central part of the State, Hardin, Carroll, Henderson, Weakley, Obion, Chester, Benton, Lincoln, Lawrence and Wayne with incidence rates of 120.9, 107.2, 85.0, 61.5, 37.8, 37.7, 35.6, 35.4, 33.6 and 33.1, respectively, no county had an incidence rate as high as 30 and the incidence rate for the whole State was only 9.9 per 100,000 population.

In Alabama, the rate of incidence per 100,000 population in a group of 8 contiguous counties in the northwest corner of the State was 72, in the group of 7 counties immediately to the south of the epidemic area the rate was 14 and in the group of 7 counties immediately to the east of the epidemic area the rate was 14.6. In the 8 contiguous counties comprised in the epidemic area the incidence rates were 112.5 in Lauderdale, 93.8 in Colbert, 79 in Morgan, 75.6 in Franklin, 55.1 in Cullman, 46.8 in Lawrence, 44.4 in Limestone and 25 in Winston. The rate in no other county of the State was as high as 25. In Jefferson County, with the largest and most congested county population of the State, the incidence rate was 16.3. In the 37 counties completely to the south of Jefferson County, in which is located the large industrial city of Birmingham, the rate was 2.5. In 23 of the total of 67 counties in Alabama not a case was reported. Of those 23 counties with an aggregate population of over 670,000 which escaped, all but one (Marion) are in the southern half of the State. Yet human traffic from North to South and West to East throughout the State is considerable. The area of high incidence of poliomyelitis in Alabama in the summer of 1936 was as sharply defined and as clear-cut as is that of the area of incidence of typhus fever in the southeastern part of that State.[22]

What is the reason for such regional distribution of the disease we call poliomyelitis? We simply do not know. None of the usual hypotheses of spread—the contagion or other—appear to apply to it to a completely satisfactory degree. The field pressingly invites constructive speculation and work, especially work. It does not seem reasonable to suppose, if the infection was widespread by human carriers all over Alabama in the summer of 1936, that most of those with peculiar constitutional diatheses making them susceptible to the disease were residing at that time in the one limited area in the northwestern part of the State nor that the meteorological conditions, in themselves, in that section were sufficiently different from those in the eastern part of northern Alabama to cause a marked difference in the distribution of susceptibles.

The ways of insects and of many of the other lower animals are not well understood. In the past they should have been and in the future should be studied intensively and extensively in connection with the distribution of poliomyelitis.



Map 5

Incidence rates (per 100,000 population) of poliomyelitis reported in counties of Arkansas, Oklahoma and Texas in the period May 1 to October 31, 1937.

In the summer of 1937 the outbreaks in the far South were mainly in the southwestern part of Mississippi, the northeastern part of Arkansas, the north central part of Texas and pretty well scattered over Oklahoma. In Texas the incidence rate was comparatively low generally in the counties with large urban populations and was remarkably low throughout the large stretch of country along the Rio Grande through the port cities of which there is much cosmopolitan travel and traffic. In El Paso County 5 cases were reported and the incidence rate was 3.6 per 100,000 population. In an area two counties wide and extending east and southeast along the Rio Grande from El Paso County to the Gulf of Mexico and comprising 30 counties, only 4 cases were reported, 2 in Duval, 1 in Willacy and 1 in Cameron County.

There was a very striking difference between the distribution of the outbreak in Oklahoma this year and that of the outbreaks in Virginia, North Carolina, Alabama, Tennessee and Mississippi in the two previous years. We do not know why. If thorough studies, epidemiological, entomological, zoological and meteorological, had been made of those situations we might have a clue. But, alas, such studies were not made. In Mississippi there was, also, a striking difference in the regional distribution of the outbreak of 1937 from that of 1936 (see *Maps 3* and *4*). The experience of Mississippi was similar to that observed previously in other sections of the United States. There have been instances of high incidence rates in one region or locality for two or more successive years, but, as a rule, communities after having a severe outbreak have low incidence rates for one or two or more years thereafter. Why this is the case, we do not know. The contagion hypothesists hold that it is due to community immunization following the thoroughly spread infection among both susceptibles and non-susceptibles in the population. Yet, in New York City, one of the most congested and most visited population areas of the globe,

there is a considerable number of cases of poliomyelitis reported every year and in that city occurred epidemics in 1907, 1916 and 1931 which are recorded among the most severe in history. In Canada this year (1937) there has been a large epidemic in the Province of Ontario centering in the city of Toronto, which appeared not to "spread" to much, if any, extent to the adjacent provinces nor to Buffalo, New York. Along lines of heavy traffic by land and water from Toronto, where 756 cases were reported, there were reported 112 cases in Ottawa, only 70 in Montreal and none in Quebec in the period July 1 to November 6, 1937.[23]

An interesting suggestion which has been made[24] to account for the lapse in incidence after an outbreak in a community is that the infection has its sole or main permanent harborage in bovine animals which after harboring the infectious agent for a while become resistant against subsequent invasion: and, therefore, harborage for the infection is absent from the community until other cattle in large proportion of the total in the community are raised or imported.

The distribution of the disease in different states or large regions is no more mysterious than the distribution often observed in one locality. It is quite usual in small outbreaks in rural counties for individual cases to develop in separate homes three or four or more miles apart without there being any evidence of direct or indirect personal contact having operated between the persons afflicted.[25] This and other epidemiological observations suggest that whatever operates to spread or cause the disease must operate in high dilution or in a rare combination of the essential factors.

The immunization of the population during an outbreak as the factor of protection against a subsequent outbreak in the same community seems open to question, especially in view of a recent report from one of our leading laboratory research workers on the problem[26] that monkeys which have recovered from an attack (even severe) of experimental poliomyelitis are subject to reinfection. Furthermore, second attacks in persons are authentically reported. Even if one attack confers no immunity whatever in persons, it takes, in view of the low rate of incidence of the disease generally, but a simple mathematical calculation to indicate why second attacks are rare. If poliomyelitis is a contagious disease spread entirely or almost entirely by direct personal contact, it is different in its seasonal incidence from every disease which has been definitely established as a contagious disease spread in the secretions or discharges from the noses or throats of persons.[27] Its seasonal incidence is different from that of measles, whooping cough, scarlet fever, mumps, diphtheria, influenza, pneumonia and smallpox, but is much the same as that of typhoid fever, the dysenteries, yellow fever, dengue, typhus fever (caused by rat-harbored infection), undulant fever, infectious encephalitis (Type B), malaria and rabies.

THEORY

Not with the advocacy of the author, but merely for consideration, the following hypothesis is submitted:

Poliomyelitis is caused by a filterable virus, of two or more distinct strains. The infection very seldom, if ever, is communicable from person to person, and, if so, it, like psittacosis,[28] after one or two such passages ceases to be communicable from person to person. The virus has its essential and usual habitat in the body of a warm blooded (lower) animal, either a beast or a bird, among which animals it is spread with a high rate of incidence in different localities from time to time. Such animals exist, but in varying proportions, in most parts of the populated world. The virus to a minor degree, if any, and seldom, is pathogenic to its usual host and is present in the circulating blood of the host for a short period, perhaps for only a few hours, at some time after it invades its host. The virus is conveyed from animal to animal and occasionally from animal to man by a blood-sucking insect which may or may not enrich the virus in its body. The invasion of man is entirely incidental in the life history of the virus and is of no importance to the virus in its struggle for existence. This blood-sucking insect feeds upon both man and its proper animal host, but much more commonly upon the latter. It is quite selective in its attack on persons. It is capable of long flights or, along with its usual animal host, it is frequently transported considerable distances, perhaps a number of miles within a few days. It exists in most parts of the world, but is most abundant in countries of the temperate and subarctic regions. It varies in prevalence from year to year or in other chronological periods in different regions of any large area of country, showing to some degree migratory tendencies. In the temperate zones it is active in all seasons of the year, but seldom, and to a very limited degree and only under exceptional circumstances, in cold weather. In four-season regions, it usually becomes very active with the

advent of the warm weather season and continues so until the cool season begins and then gradually lessens in activity. Most human beings have a considerable resistance against infection by the virus and perhaps persons who develop the disease are in considerable proportion those who have some peculiar diathesis or specific blood condition and/or those who receive the virus from the bite of the infected insect directly into or very near a nerve terminal or nerve fiber.

This hypothesis may appear to some persons as very wild speculation, but it has in some respects an analogy in the established doctrines of the modes of causation of a number of other diseases, such as bubonic plague, typhus fever, Rocky Mountain spotted fever, tularemia* and rabies. Not a definitely established fact about poliomyelitis is in conflict with this hypothesis. Therefore, to some very open-minded persons it may appear a case of hypothesis passing into theory.

If this theory is all wrong, work that would break it down might be of value. Among combinations which might be considered for experimental work are bovine, equine and other large domestic or domesticated animals, rats, fowls and birds, including especially English sparrows,† with their respective insect parasites or blood-sucking invaders. By such work a new route to India perhaps would not be found, but one or two Americas might be discovered on the way.

DIAGNOSIS AND REPORTING

We have not yet any specific method for diagnosis of poliomyelitis. Many other clinical conditions, especially in children, are confounded with it.[29] In times of outbreaks, attended with much publicity, cases of other diseases often in some localities are reported as poliomyelitis. It is even stated "authoritatively" that in New York City during the epidemic of 1916 "one would not have been far wrong to have considered that every child who fell ill during that fateful summer had poliomyelitis."[30]

In usual times the disease is under-reported in most affected localities. The records of the respective state health departments show that in some years during the last decade the number of deaths reported has exceeded the number of cases reported in some of our Southern states. Erroneous diagnosis and faulty reporting account for some of the apparent vagaries of geographical and seasonal distribution which have been epidemiologically misleading.[31]

The official recommendation[32] that only paralytic cases be counted officially as poliomyelitis and that the so-called abortive and preparalytic cases be considered and reported as suspected cases appears entirely sound.

TREATMENT

Specific measures such as the use of blood from persons who have recovered from the disease or pooled blood from adults regardless of the disease history and of serums from animals after inoculation of them with increasing doses of what are believed to be the causative organisms or viruses appear of very doubtful value. The common sense methods of putting the affected limbs of the patient at rest and after-treatment by the judicious and properly timed employment of massage and exercising appear beyond question of very great value.

PREVENTION

Due to lack of knowledge as to the factors of causation of the disease, we are unable to prescribe with any degree of satisfaction practical and practicable public health procedures for the prevention or control of the disease. If the contention of the contagionists (that the infection is spread mainly by apparently healthy, up and much-about human carriers and that by the time a few clinical cases have occurred in a locality the infection already has been widespread in that locality) is correct, such measures as quarantining persons with diagnosed cases promises little, if any, advantage. The closing or the postponement of the opening of public schools, the closing of churches and theaters and drastic restrictions upon the outdoor play and other wholesome activities of children, are, according to all of the accumulated evidence, of very doubtful value except, in some instances, as a placebo to public hysteria and demand.

The preponderance of evidence is that the various vaccines which have been proposed and used quite extensively are valueless and, in some instances, dangerous.

The nose sprays which in 1936 popularly followed in the wake of the vaccines of 1935 appear

*It has been noted in Montana that an epizootic of tularemia in rabbits seems to kill off most of them and that there is relative freedom from tularemia in persons for a few years thereafter until the disease develops in a new generation of rabbits (*Journal of American Medical Association* of July 24, 1937, p. 258).

†The English sparrow was introduced into the United States in 1550, when eight pairs were brought to Brooklyn, New York, and liberated the following spring.

as yet very definitely in the experimental stage. The extensive use of any of the nose sprays or nose drops proposed seems questionable in theory and inapplicable in general practicable public health procedure.

CONCLUSIONS

(1) Our knowledge as yet of the specificity, the etiology, the pathogenesis, the diagnosis, the therapeusis and the prevention of the disease which we call poliomyelitis is inadequate and indefinite.

(2) Much more work on the problem is urgently and critically needed, and the work should include epidemiological, laboratory, entomological, zoological, botanical, topographical and meteorological studies duly prolonged, intensive in character and wide in scope, extending from the Arctics to the Equator.

REFERENCES

1. Lumsden, L. L.: Epidemiological Principles. Sou. Med. Jour., 29:303-309 (March) 1936.
2. Leake, J. P.: Poliomyelitis. J.A.M.A., 107:1095 (Oct. 3) 1936.
   Armstrong, Charles; and Wooley, Jerald G.: Benign Lymphocytic Choriomeningitis. J.A.M.A., 109:410 (Aug. 7) 1937.
   Dummer, Clyde M.; Lyon, Robert A.; and Stevenson, Frank E.: Aseptic Meningitis. J.A.M.A., 108:633 (Feb. 20) 1937.
3. Harmon, Paul H.: J.A.M.A., 107:1096 (Oct. 3) 1936, and 109:406 (Aug. 7) 1937.
4. Draper, George: Infantile Paralysis, p. 52. New York, 1935.
5. Landon, John F.; and Smith, Lawrence W.: Poliomyelitis. p. 1. New York, 1934.
6. Report of a Committee of the American Public Health Association: Control of Communicable Diseases: Public Health Reports, 50:1017-1018, and 1044-1045 (Aug. 9) 1935.
7. Mathers, G.: J.A.M.A., 67:1019, 1916; Nuzum, J. W.; and Willy, R. G.: J.A.M.A., 92:1725, 1929.
8. Rosenow, E. C.: Proc. Soc. Exp. Biol. & Med., 27:444. 1930.
9. Toomey, John A.: Active and Passive Immunity and Portal of Entry in Poliomyelitis. J.A.M.A., 109:402 (Aug. 7) 1937.
10. Landon and Smith: Poliomyelitis, p. 50. New York, 1934.
    Harmon, Paul H.: J.A.M.A., 109:406 (Aug. 7) 1937.
11. Sabin, Albert B.; and Olitsky, Peter K.: The Olfactory Bulbs in Experimental Poliomyelitis. J.A.M.A., 108:21 (Jan. 2) 1937.
12. Hudson, N. Paul; Lennette, Edwin H.; and Gordon, Francis B.: J.A.M.A., 106:2040 (June 13) 1936.
13. Leake, James P.: Amer. Jour. Public Health, 26:148 (Feb.) 1936, and J.A.M.A., p. 2152 (Dec. 28) 1935.
14. Barnett, E. J.: Wood Tick Paralysis in Children: J.A.M.A., 109:846 (Sept. 11) 1937.
15. Month. Bull. Bd. Health Mass., 7:314, 1912.
16. U. S. Public Health Reports, 28:1733, 1912.
17. J.A.M.A., 107:1094 (Oct. 3) 1936.
18. Muller, A.: Arch. f. Schiffs u. Tropen-Hyg., 14:535-543 (No. 17).
19. Leake, J. P.; Bolten, Joseph; and Smith, H. F.: Winter Outbreak of Poliomyelitis in Elkins, West Virginia. Public Health Reports, 32:1999 (Nov. 30) 1917.
20. Aycock, W. Lloyd: A Study of the Significance of Geographic and Season Variations in the Incidence of Poliomyelitis. Jour. Preventive Med., 2:245 (May) 1929.
21. Public Health Bulletin 228, p. 55.
22. Map in Office of Alabama State Health Department, Montgomery, Alabama, showing distribution of incidence of typhus fever.
23. Data from U. S. Consular Reports.
24. Robertson, H. McG., Medical Director, U. S. Public Health Service: Personal communication.
25. Public Health Bulletin 228, pp. 21, 27, 30 and 39.
26. Flexner, Simon: Reinfection (Second Attack) in Experimental Poliomyelitis. Jour. Exp. Med., 4:497 (April) 1937.
27. Olesen, Robert; and Hampton, Brock C.: Seasonal Patterns and Trends of Communicable Diseases. Public Health Reports, 52:609 (May 7) 1937.
28. J.A.M.A., 108:2053 (June 12) 1937.
29. Landon and Smith: Poliomyelitis, pp. 137-155. New York, 1934.
30. Draper, George: Infantile Paralysis, p. 88. New York, 1935.
31. Public Health Bull. 228, p. 37.
32. Public Health Reports, 50:1044 (Aug. 9) 1935.

OBSERVATIONS ON THE PATHOLOGY AND PATHOGENESIS OF ACUTE POLIOMYELITIS IN THE RECENT EPIDEMIC IN ARKANSAS*

By A. F. DEGROAT, M.D.
Little Rock, Arkansas

During the summer of 1937, acute anterior poliomyelitis appeared in Arkansas in mildly epidemic form. Twenty-eight cases from Little Rock and other parts of the State were segregated in the Little Rock City Hospital. There were fourteen deaths. Nine autopsies were performed.

The first cases to appear raised an acute problem. They occurred in a higher age group than is usual, and localizing symptoms, such as existed, were bulbar. The problem then was to determine to what extent we were dealing with virus diseases of the nervous system other than poliomyelitis, a situation that had already been encountered elsewhere,[1] [2] and has since been under investigation.[3] Moreover, the presence of poliomyelitis in states to the east, and of encephalitis and choriomeningitis in St. Louis, had caused us to anticipate a complicated outbreak for the previous three years. The second problem to arise had to do with nasal sprays. The health authorities were saddled with the heavy responsibility of deciding for or against the adoption of a method of prophylaxis which was largely founded upon animal experimentation rather than the pathologic findings in man. For our own part we endeavored to contribute something to the latter aspect of this problem.

Autopsies on 70 per cent of the fatal cases in the Little Rock City Hospital revealed the presence of poliomyelitis alone. Such clinical

*Read in Section on Pathology, Southern Medical Association, Thirty-First Annual Meeting, New Orleans, Louisiana, November 30–December 1-2-3, 1937.

*From the Department of Pathology, University of Arkansas School of Medicine and the Little Rock City Hospital.