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ACUTE ANTERIOR POLIOMYELITIS

(*Infantile paralysis*)

A PRÉCIS

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PREPARED BY DIRECTION OF THE SURGEON GENERAL

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IN THE HOUSE OF REPRESENTATIVES,
March 26, 1912.

Resolved, That there be printed for the use of the House of Representatives five thousand copies of Public Health Bulletin Numbered Forty-four, as issued for February, nineteen hundred and eleven, by the Public Health and Marine-Hospital Service of the United States, entitled "Acute Anterior Poliomyelitis," and all to be delivered to the Superintendent of the Document Room of the House of Representatives for distribution.

Attest: SOUTH TRIMBLE,
Clerk.

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(INFANTILE PARALYSIS.)

By WADE H. FROST,

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[From the Hygienic Laboratory.]

INTRODUCTION.

Few diseases present a more complex problem than does acute anterior poliomyelitis. Eminent specialists in diseases of the nervous system, general pathology, experimental biology, orthopedic surgery, pediatrics, and epidemiology have all found in this disease special problems in their respective fields of research. The record of their special researches makes an extensive and exhaustive literature, dealing with the problem from many points of view. This literature is, however, widely scattered at present, partly because much of it is too technical to be of interest except to those engaged in some special field, partly because the bulk of the best of it is still too recent to have found its way into reference books in general use.

The object of this paper is to present, in abstract, such essential facts of the disease as may be of use, especially to the busy general practitioner to whom much of the recent literature is unavailable, to give him a general idea of the problem, and more specifically to aid him in his all-important rôle in the campaign of prevention, viz, the early recognition of cases. For those who care to go more deeply into the subject a classified bibliography is appended, referring to such of the more important special articles on various aspects of the disease as are most easily available.

For obvious reasons no attempt is made here to give a review of all the literature on the subject. Only such articles have been selected as seemed best suited, by reason of their originality, accuracy, or comprehensiveness, to the purpose of this paper.

Free use has been made of the report of the Collective Investigation Committee of New York on the epidemic of 1907,[1] the report of special investigators to the Massachusetts State Board of Health,[2] the works of Flexner and Lewis, and various other articles. I am especially indebted to Wickman's[3] extensive work, which, although written before the experimental demonstration of the infectious nature of acute anterior poliomyelitis, remains in many respects, especially in its clinical descriptions, the most comprehensive and satisfactory treatise on the subject. This work has been freely quoted and given preference over other clinical works because it is the result of the most extensive study of the disease ever made by an individual. In fact, it is doubtful if anything essential to our clinical knowledge of the disease, except statistical data and occasional reports of rare cases, has been added to Wickman's observations. Most of the reports made since his writing have been confirmations of his accuracy and comprehensiveness. A translation of Wickman's work, especially the clinical section, would be of great benefit to the physicians of the country.

SYNONYMS.

The term "acute anterior poliomyelitis," indicating as it does an acute inflammation characteristically localized in the *anterior cornua of the gray matter* of the spinal cord, is not an altogether satisfactory designation for the disease under consideration, for it has been amply demonstrated that the lesions are not confined to that area of the cord, and not necessarily confined even to the spinal cord. It is desirable, however, for the sake of uniformity and precision in mortality and morbitity reports to have one term in general use. The Bureau of the Census has experienced considerable difficulty in the compilation of mortality statistics for this disease by reason of the numerous designations employed. The returns for 1909 were received under 24 different designations.[4] The Bureau of the Census urges, therefore, with good reason, the uniform adoption of the term "acute anterior poliomyelitis." The term "infantile paralysis" is objectionable, because it is hardly applicable to adult cases; also, because it is likely to be confused with other infantile paralyses of altogether different etiology. Other terms in general use are "acute

[1] Epidemic poliomyelitis: Report of the Collective Investigation Committee on the New York Epidemic of 1907. Nervous and Mental Diseases, monograph, series No. 6, New York, 1910.

[2] Lovett, Robt. W.: The Occurrence of Infantile Paralysis in Massachusetts in 1909. Boston Med. and Surg. Jour., 1910, vol. 163, pp. 37–55.

[3] Wickman, Ivar: Beiträge zur Kenntniss der Heine-Medinschen Krankheit (Poliomyelitis acuta und verwandter Erkrankungen). Berlin, 1907. S. Karger.

[4] Mortality statistics, 1909. Department of Commerce and Labor, Bureau of the Census, Bull. 108, Washington, 1910, p. 25.

poliomyelitis," "epidemic poliomyelitis," "acute spinal paralysis," "acute atrophic spinal paralysis," "essential paralysis," and a number of other variations.

HISTORICAL.

Acute anterior poliomyelitis is not, in any sense, a "new disease." Mitchell [1] found in the skeleton of an Egyptian mummy, dating back to 3700 B. C., evidence of probable poliomyelitis. Jacob von Heine [2] is generally credited with having given, in 1840, the first clear clinical description and differentiation of the affection. He was not, however, the first to mention the disease. Underwood,[3] in 1774, gave a recognizable, though by no means clear-cut, description; and doubtless still other references may be found in early medical literature.

EPIDEMIC PREVALENCE.

The epidemic occurrence of poliomyelitis has been recognized more recently. Colmer [4] records the occurrence of an epidemic of paralysis among teething infants in Louisiana in 1841. He did not identify the epidemic with the disease described by Von Heine. Bergenholz [5] is credited with being the first to recognize an epidemic of poliomyelitis, in 1881; his observations, however, were not published until 1890, when Medin made them known. Oxholm [6] and Cordier [7] had each in the meantime observed a small epidemic, but their publications attracted little attention until Medin [8] brought them to light in 1890, at the same time reporting his own observations on an epidemic of 43 cases which occured in and around Stockholm in 1887. Since that time epidemics have been observed with increasing frequency in various parts of the world. The largest epidemics recorded have been as follows: In Vermont, 1894, 126 cases; Norway and Sweden, 1905, about 1,500 cases; New York City and vicinity, 1907, about 2,500 cases.

From 1907 to 1910 outbreaks have occurred in the following States in this country: Connecticut, 1910; District of Columbia, 1910; Florida, 1907; Illinois, 1909; Iowa, 1908–1910; Kansas, 1909–10; Massachusetts, 1907, 1908, 1909, 1910; Michigan, 1907–8, Minnesota, 1908, 1909, 1910; Missouri (?), 1908; Nebraska, 1909; New York,

[1] Mitchell, J. K., Trans. Assn. Am. Physicians, vol. 15, 1900, pp. 134–136.

[2] Von Heine, Jacob, Spinale Kinderlähmung, II Aufl., Stuttgart, 1860, J. G. Cotta. (First ed. pub. in 1840.)

[3] Underwood, Michael: Treatise on the diseases of children. Phila., 1893, F. Dobson.

[4] Colmer, George: Medical Notes: Paralysis in teething children. Am. Jour. Med. Sci., 1843, vol. 5, p. 248.

[5] Cited by Medin. Verhandl. d. X. internat. med Kongr., Berlin, 1890. II Abt., VI, 1891.

[6] Cited by Leegard. Neurol. Centralblatt, 1890, p. 760.

[7] Lyon Méd., 1888, vol. 57, 5; 48.

[8] Medin. Verhandl. d. X. internat. med. Kongr., 1890. II Abt., VI, Berlin, 1891.

1907, 1910; Oregon, 1910; Pennsylvania, 1910; South Dakota, 1910; Virginia, 1908–1910; Washington, 1910; Wisconsin, 1908. Since 1907 epidemics have also occurred in Canada, in various parts of Germany and Austria, in Cuba, and in the island of Nauru, in Melanesia (1910).

Since the severe outbreak in Norway and Sweden in 1905, epidemic poliomyelitis has prevailed more widely than ever before. Whether or not this pandemic may be attributed to a spread of infection from Scandinavia is a question still open to dispute, the discussion of which is beyond the scope of this paper. Lovett's[1] compilation of the reported epidemics since 1881, is as follows:

Years.	Cases.	Outbreaks.	Average number of cases.
1880–1884	23	2	11.5
1885–1889	93	7	13
1890–1894	151	4	38
1895–1899	345	23	15
1900–1904	349	9	39
1905–1909	8,054	25	322

These figures show very strikingly the enormous increase in the epidemic prevalence of poliomyelitis from 1905 to 1909. There is good reason to believe that there has been a corresponding increase during the same period, in so-called "sporadic" cases—cases which are not recognized as occurring in connection with any well-defined epidemic. This is inferred from hospital reports, showing a large increase in the number of patients admitted for treatment for the resulting paralysis, in districts where no epidemic has been reported.

Lovett calls attention to the fact that the United States has suffered more severely than any other country, being credited with some 5,500 of the 8,000 cases reported from 1905 to 1909. It will be noted, too, that the great majority of epidemics in the United States have occurred in the northern States, east of the Dakotas. While reports for 1910 are as yet incomplete, it may safely be asserted that the prevalence of the disease has very considerably increased during this year, in respect both to the number of persons attacked and the area over which the epidemics have occurred.

According to the statistics of the Census Bureau,[2] there were 569 deaths from acute anterior poliomyelitis in the registration area of the United States in 1909. The population from which these statistics are collected represents 55.3 per cent of the total population of the United States. The 569 deaths reported are from 33 States, and do not include a considerable number of deaths in nonregistration States, notably in Minnesota and Nebraska.

[1] Lovett, Robt. W., Bost. Med. and Surg. Jour., July 14, 1910, vol. 163, No. 2, pp. 37–55.

[2] Mortality statistics, 1909. Department of Commerce and Labor, Bureau of the Census, Bull. 108, Washington, 1910, pp. 24–26.

It naturally follows that the increased prevalence of epidemic poliomyelitis, with the consequent stimulation of interest therein, should have resulted in many valuable additions to our knowledge of the subject.

Pathology.—Jacob von Heine,[1] as above stated, first gave a clear-cut clinical description of acute anterior poliomyelitis in 1840. Prevost and Vulpian,[2] in 1865, are credited with having given the first anatomical demonstration of the destruction of the ganglion cells of the anterior cornu in poliomyelitis. The clinical studies of Von Heine and the pathological studies of Prevost and Vulpian both dealt with the late manifestations of the disease as seen subsequent to the acute stage. Roger and Damaschino,[3] in 1871, reported the first pathological study of a cord from a case in the acute stage; and first recognized interstitial changes as the *primary* lesions of acute poliomyelitis. From that time ensued a running fight among pathologists in regard to pathogenesis, one school holding the essential acute lesion to be specific intoxication of the motor cells of the anterior cornu, the interstitial changes being of secondary importance. The other school deemed the primary essential lesion in the acute stage to be a diffuse myelitis, resulting secondarily in degenerative changes of the motor ganglion cells. With the increase of the epidemic prevalence of poliomyelitis and the increased opportunities for the study of material obtained from recent acute cases, it has become more and more evident that the views of the latter school were correct; and it is now generally recognized that the acute nervous lesions of poliomyelitis are interstitial and widespread throughout the cerebrospinal axis. While many pathologists have had a part in this demonstration, special credit should be given to Wickman[4] and to Harbitz and Scheel.[5]

Symptomatology.—Parallel with the development of knowledge of the pathology of the disease has been the development of the knowledge of its clinical manifestations. Medin,[6] in 1890, gave the first comprehensive account of the symptoms of the acute stage from observations of an epidemic in Stockholm in 1887. He was the first to call attention forcibly to the varied clinical manifestations of the acute stage, indicative of changes in all parts of the nervous system,

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[1] Von Heine, Jacob. Spinale Kinderlähmung.

[2] Prevost and Vulpian: Observation de la paralysis infantile. C. R. de la Soc. de Biol., 1865. Paris, 1866, 4 s., ii, pp. 215–218.

[3] Roger and Damaschino: Recherches anatomo-pathologiques sur la paralysie spinale de l'infance. C. R. de la Soc. de Biol., 1871. Paris, 1873, 5 s., iii, pt. 2, pp. 49–93.

[4] Wickman, I.: Studien über Poliomyelitis actua. Arb. aus d. Path. Inst. d. Univ. Helsingfors, 1905–1907. Berlin, 1907, S. Karger, pub.

[5] Harbitz u. Scheel: Pathologisch-anatomische Untersuchungen über akute Poliomyelitis u. verwandter Erkrankungen. Christiania, 1907, A. W. Broggers, pub.

[6] Medin, loc. cit.

although Strümpell[1] and Pierre-Marie[2] had previously noted the occurrence of encephalitis in acute poliomyelitis, and others had, in a less comprehensive manner than Medin, noted many of the facts which he first put together so clearly. In view of the pioneer services of von Heine in describing the later stages and of Medin in describing accurately the acute stage, Wickman has suggested the name "Heine-Medin disease" for acute poliomyelitis, since pathological designations (acute anterior poliomyelitis, acute poliomyelitis, epidemic poliomyelitis, epidemic spinal paralysis, infantile paralysis, poliomyelo-meningo-encephalitis) are either misnomers or overcumbersome. The most valuable clinical contribution since Medin's has been made by Wickman, whose special service in this respect has been the recognition of abortive forms of the disease, and the presentation of the whole subject from a broader, more comprehensive point of view than any of his predecessors.

Epidemiology.—The recognition of the epidemic occurrence of the disease has already been referred to as dating from the observations of Bergenholz, in 1881. While many observers since Bergenholz have reported epidemics of acute poliomyelitis, and in many cases have gone more or less into the question of the origin and transmission of the epidemics, the first systematic study from an epidemiologic point of view was undertaken by Wickman in Sweden in 1905–6. He personally investigated over 1,000 cases, attempting to trace the routes by which the disease spread. He found abundant evidence to show that it was contagious, though usually slightly so. He directed especial attention to several factors in its spread—routes of travel, public gatherings of children (schools), abortive (ambulant) cases, and healthy intermediate carriers. Since that time systematic epidemiologic investigations have been undertaken by the Governments of several foreign countries; and in this country by several States, notably Massachusetts, Minnesota, Nebraska, Kansas, and Iowa. A committee of the New York Neurological Society investigated and has recently reported upon the New York epidemic of 1907. The reports of the New York investigation committee and of the Massachusetts State Board of Health are especially valuable, on account of their comprehensiveness and thoroughness.

Experimental poliomyelitis.—The most recent development of knowledge concerning epidemic poliomyelitis is derived from the study of the disease experimentally produced in monkeys. It had been recognized for some years prior to 1909 that acute poliomyelitis must be due to some specific infection; and quite a number of observers had isolated from the nervous system of patients suffering from the disease bacteria which they believed to be the specific causa-

[1] Strümpell, Jahrb. f. Kinderheilk., 1885.
[2] Pierre-Marie, Progrès méd., 1885, II, s., II, 167–169.

tive agent. Some observers claimed to have reproduced the disease in lower animals by injections of pure cultures of such bacteria. However, the bacteria found by different observers belonged to different species; many competent bacteriologists constantly failed to find any bacteria whatsoever, and the lesions occasionally produced in lower animals did not correspond to the lesions of human poliomyelitis. Consequently, none of the above claims were generally accepted. In the spring of 1909, Landsteiner and Popper [1] succeeded in transmitting the disease to two monkeys by inoculation with the spinal cord of a child which had died of acute poliomyelitis. The lesions found in the cords of these monkeys were typical, but Landsteiner and Popper failed in their attempts to transmit the disease from these to other monkeys. Later in the year Flexner and Lewis [2] succeeded in confirming the work of Landsteiner and Popper, and further succeeded in transmitting the infection from monkey to monkey through an indefinite number of passages. Since then a great deal of very brilliant experimental work has been done upon this subject by Flexner and Lewis, Römer and Joseph, Landsteiner and Levaditi, Leiner and Weisner, Krause and Meinecke, R. Kraus, and others.[3]

ETIOLOGY.

THE SPECIFIC ORGANISM.

The proof that the causative agent of epidemic poliomyelitis is a *living organism* is furnished by its demonstrable multiplication or *reproduction* in the body of an inoculated animal, reproduction being a property confined to living organisms. A comparatively small amount (0.5 c. c. of a 5 per cent emulsion) of the spinal cord of a person who has died of epidemic poliomyelitis, injected into the brain of a monkey, is sufficient to produce the disease in this animal after an incubation period of 5 to 46 days. This effect might be ascribed to the action of either a living organism, capable of multiplication or a chemical poison (toxin). In the latter case, the toxin must undergo great dilution, since portions of the central nervous system of this animal, remote from the site of inoculation, have been found capable of reproducing the disease in other monkeys. After a very few such passages the amount of toxin contained in, say, 1 gram of the spinal cord would be inconceivably small, and the potency of the cord in causing the disease proportionately diminished. As a matter of fact, Flexner and Lewis [4] have succeeded in transmit-

[1] Landsteiner, K., and Popper, E. Zeitschr., f. Immunitätsforsch, usw., Orig., 1909, vol. 2, p. 377.

[2] Flexner, S., and Lewis, Paul A., Jour. Am. Med. Assn., 1909, vol. 53, p. 1639.

[3] The more important works of all these authors are cited by Lovett in Boston Med. and Surg. Jour., vol. 163, 1910, pp. 37–55, and by Landsteiner and Levaditi in Ann. de l'Inst. Pasteur, vol. 24, 1910, pp. 833–876.

[4] Flexner, S., Journ. Am. Med. Assn., vol. 55, pp. 1105–1113.

ting the disease through a series of at least 25 monkeys, using at times quantities of the virus as small as 0.01 c. c. to 0.001 c. c.

From one monkey, inoculated with a minimal dose of the virus, enough virus can be obtained to reproduce the disease in *several hundred* monkeys. The multiplication of the virus in 25 passages is, therefore, enormous.

In spite of the fact that this organism can not be isolated in pure culture, as can the bacteria, a great deal of quite definite knowledge concerning it has already been acquired by the work of the above-mentioned observers.

It is known to be an exceedingly small organism, because emulsions of virulent spinal cord are still infective after filtration through very dense porcelain (Chamberland) filters, the pores of which are so small that even very minute *bacteria* can not pass through. The organism belongs, therefore, to the class of so-called "filterable viruses," other examples of which are the organisms of yellow fever, rabies, the foot-and-mouth disease of cattle, probably hog cholera, and others.

It is not visible in preparations made by the usual bacteriologic methods, but is thought to have been seen with high-power lenses in preparations very carefully stained; also unstained, by the use of dark-background illumination. It will not grow upon the ordinary culture media in general use for cultivating bacteria, but will probably multiply to a slight extent in ascitic bouillon. Such cultures have been successfully transplanted, but are not virulent and are of little practical value in experimental studies.

The virus of poliomyelitis is killed by a temperature of 45°–50° C. in half an hour; also, by comparatively weak disinfectants, such as 1:500 solution of permanganate of potash;[1] 1 per cent menthol in oil;[1] a powder containing menthol 0.5, salol 5, and boric acid 20;[1] a dilution of perhydrol (Merck) equivalent to 1 per cent of peroxide of Hydrogen.[2, 3]

It is not destroyed by very low temperatures nor by drying over caustic potash or in vacuo for a considerable period. A virulent cord has been kept for almost five months in pure glycerin without losing its virulence, resembling in this respect the virus of rabies, vaccine, etc., and differing from nonspore-bearing pathogenic *bacteria*, which are, for the most part, killed by pure glycerin in a short while.

As is to be expected, the organism is present in greatest abundance in the spinal cord of infected persons and animals, as shown by the extremely small amounts of these organs necessary to infect a monkey

[1] Landsteiner, K., and Levaditi, C., Ann. de l'Inst. Pasteur, 1910, vol. 24; pp. 833–876.
[2] Flexner, S., and Lewis, Paul A., Journ. Am. Med. Assn., vol. 56, 1910, p. 1782.
[3] The ordinary commercial preparation of hydrogen peroxide contains about 3 per cent of the peroxide (H_2O_2). Perhydrol (Merck) contains about 30 per cent H_2O_2.

by inoculation (0.001 c. c. to 0.01 c. c. of an emulsion of cord—Flexner). It is also, however, quite constantly present in the brain and has been demonstrated in various other organs of infected animals, viz, in the mucous membrane of the nose and pharynx, the salivary glands, the mesenteric glands, the regional (axillary or inguinal) lymph glands after subcutaneous inoculation, in the blood, and in the cerebro-spinal fluid. In the blood, however, the virus appears to be present in small quantity and only in the early stages of the disease, and the same may be said of the cerebro-spinal fluid. No one has yet succeeded in demonstrating the presence of the virus in the urine or feces, though it is suspected, from the pathology of the disease, that the feces may be infectious, and technical difficulties may explain the failure to demonstrate this.

The most uniformly successful method of inoculating monkeys is by injecting an emulsion of infectious material directly into the central nervous system, preferably into the brain. Monkeys may also be infected by subcutaneous, intraperitoneal and intravenous inoculation, by rubbing virulent material into the scarified mucous membrane of the nose, by transplantation of infectious tissue into the trachea, and by introducing the virus into the stomach along with an opiate to restrain peristalsis. Leiner and Weisner [1] have infected monkeys through the *uninjured* nasal mucous membrane. This is, however, an uncertain method of inoculation, as other observers have failed in their attempts to reproduce the disease in this way. Monkeys have so far never been known to contract the disease spontaneously from intimate association with infected monkeys.

Numerous attempts have been made to transmit the disease to lower animals other than the monkey, viz, to guinea pigs, rabbits, horses, sheep, hogs, dogs, cats, chickens, ducks, and pigeons. These animals have all been found insusceptible, with the exception of certain breeds of rabbits. Krause and Meinicke [2] were the first to report the successful inoculation of rabbits from human material, and subsequent transmission from rabbits to other rabbits and to monkeys. Their results have been confirmed to some extent by other workers [3] who have succeeded in producing in a comparatively small proportion of rabbits inoculated, a disease which has more or less clinical resemblance to acute poliomyelitis. Landsteiner and Levaditi [3] found in the cord of one of their rabbits lesions similar to those found in man and in the monkey. Others have failed to find such

[1] Leiner, C., and Weisner, R. v., Wien. klin. Wochenschr., 1910, vol. 23, p. 323.

[2] Krause, R., and Meinicke, E., Deutsche med. Wochenschr., 1910, vol. 35, p. 647; ibid., 1910, vol. 35, p. 1825.

[3] Beneke, Münch. med. Woch., 1910, vol. 57, pp. 176–178; Kraus, R., Med. Klin., 1910, vol. 6, pp. 470–472; Landsteiner, K. and Levaditi, C., Ann. de l'Inst. Past., 1910, vol. 24, pp. 833–876; Bonhof, Münch. med. Woch., 1910, vol. 57, p. 105; Dahm, Münch. med. Woch., 1909, vol. 56, p. 2553; Lentz and Hüntemuller, Tag. der fr. Verein. Mikrobiol., Berlin, 1910 (cited by Landsteiner and Levaditi, supra).

lesions; and indeed the great majority of attempts to inoculate rabbits have proved unsuccessful. The question of the susceptibility of rabbits to human infection has not been sufficiently cleared up to warrant very definite conclusions. It appears, however, that they are at least very much less susceptible than monkeys.

IMMUNITY.

Monkeys which have recovered from the infection of poliomyelitis show a definite immunity, demonstrable in two ways: (1) They are not susceptible to infection by reinoculation, and (2) their blood serum, when mixed in suitable proportions with an emulsion of virulent spinal cord and allowed to stand for several hours, renders the virus harmless. This property has also been demonstrated in the blood of persons who have recovered from poliomyelitis, but is not shown by the blood of normal persons and of normal monkeys.

The immunity is probably very lasting. The neutralizing power of the blood has been shown to last for 3 years after an attack of poliomyelitis, but was found absent in one case tested 11 years after the acute attack.[1] A second attack of the disease is rare, but a few instances have been reported of recurrence after the lapse of several years.

It has not been found possible as yet to obtain from immunized animals a serum which will arrest the progress of the disease after it has developed. While the hope of such a serum may still be held out as a possibility, its usefulness would necessarily be limited to the treatment of the early stages of the disease, before destruction of nerve centers had taken place. Attempts to actively immunize (vaccinate) monkeys have, in some instances, been successful, but no method has yet been developed which may safely be applied to human beings.

CONTAGIOUSNESS.

It has been experimentally demonstrated that the *mucous membrane* of the nose of infected monkeys is infectious, and in one case the salivary glands;[2] and it has been quite reasonably inferred therefrom that the *secretions* of the nose and mouth are infectious, although experiments aiming to demonstrate the latter have failed.[3] It has also been shown that infection may take place through the mucous membrane of the respiratory and digestive tracts.

These facts indicate very strongly that the disease is transmissible directly from person to person by direct contact, a conclusion which

[1] Netter, A., and Levaditi, C., Comp. rend. de la Soc. de Biol. 1910, vol. 68, pp. 855–857.
[2] Landsteiner, K., and Levaditi, C., Comp. rend. de la Soc. de Biol. 1909, vol. 67, p. 788.
[3] Landsteiner, K., and Levaditi, C., Comp. rend. de la Soc. de Biol. 1909, vol. 67, p. 788.

Wickman had already reached from his epidemiologic studies. Others have reached varying conclusions from the study of epidemics, some asserting that there was no evidence that contact with the sick played any rôle in the transmission of the disease, some considering its contagiousness established. While it is beyond the scope of this paper to discuss in detail the evidence for and against the contagiousness of epidemic poliomyelitis, it may be said that the best evidence at present available indicates that the disease is *transmissible* from person to person, probably by direct contact. It must be usually rather slightly transmissible, since only a small proportion of persons in intimate contact with cases contract the disease. Under some circumstances, however, it appears to be rather highly contagious, affecting a very considerable proportion of the population of a limited area. Examples of seemingly quite contagious epidemics are reported by Wickman, from Traestena, Sweden; by Shidler,[1] from Polk County, Nebr.; and by Armstrong,[2] from North St. Paul, Minn. The writer investigated a small, apparently highly contagious outbreak among the attendants of a rural school in Hancock County, Iowa, in the summer of 1910, details of which will be given in a later publication.

OTHER ETIOLOGICAL FACTORS.

It is evident that certain predisposing factors are operative in causing infection, either by increasing the susceptibility of persons exposed, by increasing the virulence of the infecting organism, or by facilitating its transmission.

Geographic distribution.—Epidemics of poliomyelitis have been most prevalent in the northern parts of Europe and of the United States, and, more recently, also in Canada. The disease has not been confined to these countries, however. Epidemics have occurred in southern Europe (Italy) and in the southern part of the United States (Alabama, South Carolina, and Virginia); a considerable epidemic occurred in Cuba in 1909. There have been several epidemics in Australia, and during January, 1910, a very remarkable epidemic on the little island of Nauru, situated near the Equator, north of Australia. There are, therefore, no well-defined geographic limits to the area within which acute poliomyelitis has been known to become epidemic. Sporadic cases, not known to be connected with epidemic outbreaks, have occurred over a still wider area.

The occurrence of epidemics in various places has not been proportional to their geographic proximity to recognized previous epidemic foci, nor has it apparently been proportional to the amount of travel from such foci. It appears, however, that sporadic cases

[1] Shidler, G. P., Pediatrics, vol. 22, 1910, pp. 539–543.
[2] Armstrong, J. M., Pediatrics, vol. 22, 1910, pp. 486–501.

are unusually prevalent along routes of travel leading from epidemic foci.

Density of population.—The occurrence and spread of epidemic poliomyelitis is not proportional to the density of population. On the contrary, epidemics have been more severe and the case rates have been higher in small towns and rural districts than in the more densely populated cities, and in cities the disease has not been found more prevalent in the crowded districts.

Climate.—The countries which have suffered most have been those with a cold climate, showing marked seasonal variations in temperature, but striking exceptions to this have been noted above.

Season.—Epidemics of acute poliomyelitis occur almost invariably in the warm, dry months—in the Northern Hemisphere from May to November; in the Southern Hemisphere from November to May. Sporadic cases occur, however, throughout the year, and warm weather need not therefore be considered a *necessary* factor in infection.

Age.—The proportion of cases decreases progressively in each decade after the first. The proportion of adult cases, however, may be considerable in epidemics. The great variations in the age incidence in different epidemics are illustrated in the following tables, the first of which is arranged in three-year periods, in order to include Wickman's statistics, while the second is arranged in the more usual five-year periods. The majority of cases are in children between 1 and 5 years of age.

THREE-YEAR PERIODS.

Ages.	Reported by—							
	Collective investigation committee, New York (1907).		Lovett, Massachusetts (1909).		Hill, Minnesota (1909).		Wickman, Sweden (1905).	
	No. of cases.	Per cent of total.	No. of cases.	Per cent of total.	No. of cases.	Per cent of total.	No. of cases.	Per cent of total.
0–2, inclusive	463	63.5	258	41.9	89	27.4	169	19.5
3–5, inclusive	197	27.0	182	29.6	90	27.7	181	21.1
6–8, inclusive	40	5.5	70	11.4	53	16.3	154	17.6
9–14, inclusive	21	2.9	57	9.3	47	14.5	165	19.0
15 and over	8	1.1	48	7.8	46	14.2	199	22.9
Total cases	729	.....	615	.....	325	.....	868	.....

FIVE-YEAR PERIODS.

Under 1 year	62	8.5	44	7.2	21	6.5	.....	.....
1–5 years	598	82.0	396	64.5	158	48.6	.....	.....
6–10 years	47	6.4	98	15.9	77	23.7	.....	.....
11–15 years	14	1.9	31	5.0	25	7.7	.....	.....
16–20 years	5	.68	15	2.4	21	6.5	.....	.....
Over 20 years	3	.40	31	5.0	23	7.0	[1] 87	[1] 10.0
Total cases	729	.....	615	.....	325	.....	868	.....

[1] Wickman's figures are given only in three-year periods and are therefore available only for the last column of this table.

The New York epidemic of 1907 is characterized by the large percentage of cases occurring in childhood (90.5 per cent in the first six years) while the Swedish epidemic of 1905 stands at the other extreme, with only 40.6 per cent of cases in the first six years, and 10 per cent among *adults*. The epidemics in Massachusetts and Minnesota are intermediate between these two extremes, the Minnesota statistics approximating more closely those from Sweden.

The generalization seems warranted that in epidemics affecting a comparatively large proportion of the population in a given area the proportion of adult cases is high. In the very remarkable epidemic of 1910 on the island of Nauru, where 700 cases occurred within a few weeks in a population of little over 2,500, the majority of cases were in adults.[1]

Sex.—More males are affected than females. In Massachusetts in 1909, the ratio was males, 363; females, 263; in Minnesota in 1909, males, 193; females, 139. As shown by Hill[2] in his analysis of the Minnesota cases, the proportion of males and females affected is more nearly equal in the first decade of life, while after 10 years of age males are affected in much greater proportion than females.

Race.—There is little to indicate that nationality has any influence upon susceptibility. In the New York epidemic of 1907, there were proportionately fewer cases among the negroes than among white races. Upon the island of Nauru the population and attack rate are given as follows:

	Number of persons.	Number of cases.	Number of cases per 1,000.
Natives	1,250	470	376
Imported laborers[1]	1,000	220	220
Whites	80	3	37.5

[1] Partly Chinese and partly Caroline Islanders.

The death rate among the natives was 7.8 per cent, while among the imported laborers it was 0.45 per cent, and among the whites (3 cases) there was no mortality. There was, therefore, in this instance, apparently a very marked difference in racial susceptibility; but this epidemic was so remarkable in many other respects that it can not be taken as illustrative of epidemic poliomyelitis in general.

The high death rate and large proportion of adult cases in Norway and Sweden arouse a suspicion that the Scandinavians may be peculiarly susceptible. In this connection it may be noted that in the

[1] Müller, A., Arch. f. Schiffs-u. Trop.-Hyg., 1910, vol. 14, No. 17.

[2] Hill, H. W.: Epidemiologic study of anterior poliomyelitis in Minnesota, Trans. Section on Preventive Med., Amer. Med. Assn., 1910.

United States the most fatal and most infective epidemics have occurred in the Middle West and Northwest, where a large proportion of the population is of Scandinavian descent. On the other hand, it has not been shown that an undue proportion of Scandinavians have been attacked, even in the Northwest. The question of difference in racial susceptibility is therefore still an open one.

Social and hygienic conditions.—Poverty and insanitary conditions of life seem to have little, if any, influence in determining infection. All classes are affected in about equal proportions.

PATHOLOGY.

A widespread misunderstanding of the pathologic anatomy of acute poliomyelitis has contributed largely to a very prevalent misconception of its symptoms. The earlier pathologic studies were made chiefly on cases that had long since passed the acute stage. The most characteristic lesion observed in such cases is degeneration of the motor cells in the anterior cornua of the cord, and this was therefore naturally regarded as the essential *primary* lesion of the disease. Although for the past 20 years there has been ample evidence, both clinical and anatomical, that in the acute stage the characteristic lesion is an *interstitial inflammation, not confined to the motor area of the cord*, and although these facts have been published in many reference books, still the conception of the disease as essentially an inflammatory degeneration of the motor cells of the cord, has remained fixed in the minds of many of the medical profession, and statements to this effect remain in a considerable proportion of recent textbooks on the practice of medicine. The result is that those who are without experience in epidemics of the disease are unprepared to recognize as characteristic of it such nervous symptoms as are not referable to damage of the *motor cells* of the cord. In order to understand the symptoms observed, it is necessary to have at least a general knowledge of the anatomic lesions to which the symptoms are due.

Acute anterior poliomyelitis must be recognized as a general infection producing characteristic lesions in the central nervous symptoms, viz, *congestion, infiltration and edema of the cord, brain, and leptomeninges.*

MENINGES.

The dura mater is practically unchanged. The pia-arachnoid throughout the cerebrospinal axis shows quite constant and characteristic changes in the acute stage. The vessels are congested and their sheaths infiltrated with round cells. Between the vessels the infiltration is more diffuse. The round cells are mostly mononuclear.

No exudate is found on the surface of the meninges, which to the naked eye appear normal or simply congested. The infiltration of the pia mater varies at different levels; it is most intense in the lumbar region, where it is equally marked around the whole circumference of the cord. In the higher segments the infiltration of the pia mater is most marked over the anterior surface of the cord, and may be quite irregular in its distribution.

It is believed that the meninges are the seat of the earliest changes in the central nervous system; that the virus first invades the lepto-meninges, and extends thence, following the vascular prolongations of the pia, into the substance of the brain and cord.

CEREBRO-SPINAL FLUID.

Corresponding to the changes in the meninges, there are characteristic changes in the cerebro-spinal fluid. It is increased in quantity quite early, before the onset of definite symptoms. At this time the fluid is opalescent, due to an increase in the number of cells; the protein is increased, and the fluid may coagulate spontaneously. The cellular elements are chiefly lymphocytes, although in the earlier stages there may be a large proportion of polymorphonuclear leucocytes. By the time paralysis has developed the cerebro-spinal fluid has returned more nearly to the normal; it is now clear, but still more or less increased in quantity, and still contains an abnormal number of lymphocytes.

CORD.

The early changes in the cord are congestion, round-cell infiltration, and edema. All *the vessels of the cord* are congested, and their sheaths show an infiltration of round cells similar to and continuous with the perivascular infiltration of the pia mater. In the *gray matter*, more especially in the anterior cornua, at the level of the cervical and dorsal enlargements, in addition to the infiltration immediately around the vessels, there is a more diffuse infiltration, with here and there foci of closely packed round cells. Hemorrhages, due to the rupture of small blood vessels, are quite commonly found in the gray matter and less frequently in the white substance. The infiltration is all of vascular origin, and its distribution in various areas of the cord is proportional to the vascularity of these areas. Because of a richer blood supply, the gray matter is more affected than the white, the anterior cornua more than the posterior, and the cervical and lumbar enlargements more than the other segments of the cord. The irregular distribution of the paralysis probably depends to some extent upon irregularities in the blood supply of the cord.

The ganglion cells of the cord suffer more or less secondary damage, which is generally proportionate to the infiltration around them.

The most common and extensive damage occurs in the motor nerve cells situated in the anterior cornua, especially in the lumbar and cervical enlargements, where, as already noted, the infiltration is most intense; but considerable damage, and even destruction, of some of the ganglion cells in the posterior horn, especially in Clark's column, is not uncommon. In the lumbar cord, according to Wickman, the posterior cornua show quite as extensive lesions as the anterior cornua. The damage to the ganglion cells may result in temporary loss of function, perhaps due to edema, without demonstrable changes in the cell; in partial but not permanent degeneration of the cells, or in the complete destruction of a part or all of the cells in a given area of the cord. Complete destruction of the ganglion cells of the anterior cornua in a segment of the cord results in permanent motor paralysis and atrophy of the muscles *supplied* by these neurons. The less severe cell changes result in paresis or temporary paralysis of the corresponding muscles.

The *white substance* of the cord undergoes less severe changes than the gray matter, but constantly shows infiltration of the vessel walls, edema, occasional small hemorrhages and rarely definite foci of round-cell infiltration. The acute inflammatory lesions of the white substance of the cord are important to bear in mind as explanatory of some of the symptoms in the acute stage. The rarer, more permanent lesions found here may explain some unusual motor disturbances (ataxia, exaggerated reflexes, spasticity) occasionally encountered.

SPINAL GANGLIA.

The intervertebral ganglia have been found to show changes similar to those in the cord, viz., infiltration, and degeneration of ganglion cells and nerve fibers (Straus, Flexner).

PERIPHERAL NERVES.

The peripheral nerves have not been examined as carefully as the spinal cord, but it is believed that they show no acute inflammation, except perhaps near their emergence from the spinal cord.

MEDULLA.

In the medulla oblongata and pons cerebri there are found edema and perivascular infiltration as in the spinal cord; and also, not infrequently, foci of round cells. Infiltration around the nuclei of the cranial nerves explains the cranial-nerve paralysis often noted in acute poliomyelitis. The ganglion cells in the foci of infiltration are more or less damaged; but as the foci are usually small, the complete destruction of a bulbar nerve center is rather rare.

CEREBRUM.

Changes in the brain similar to those in the cord, but less intense, are a constant characteristic of acute poliomyelitis. Severe lesions of the cerebral cortex or the conducting paths therefrom are rare, but may occur.

OTHER ORGANS.

The lesions outside the nervous system are not characteristic and are apparently not constant. In quite a number of autopsies, however, lesions have been found indicative of acute general infection, affecting especially the digestive and respiratory tracts and the lymph glands, viz., congestion of the mucous membrane of the small intestine and sometimes of the stomach; congestion and enlargement of the solitary follicles, Peyer's patches and mesenteric glands; less commonly a more general glandular enlargement; occasional pneumonic foci in the lungs; congestion of the liver and spleen, with occasional enlargement of the latter; congestion and sometimes beginning parenchymatous degeneration of the kidneys.

THE BLOOD.

The characteristic blood changes in the acute stage are diminution of the total number of white cells (leucopenia) with relative increase in the proportion of *lymphocytes*. More observations are needed to fully establish the constancy and degree of these changes.

SYMPTOMATOLOGY.

As is to be expected from its patholgy the symptoms of acute poliomyelitis are most diverse. Broadly speaking, they are the symptoms which may be expected from—

(1) An acute general infection.

(2) An acute inflammation affecting the leptomeninges and medullary substance of the brain and cord. The nervous symptoms are first those of irritation, followed in more severe cases by symptoms of depression, and in typical cases by loss of function of certain areas of the central nervous system. The irritative symptoms, arising from inflammatory lesions of comparatively mild degree but wide extent, are usually both sensory and motor; predominantly spinal, but to some extent bulbar and cerebral. The depression and loss of function resulting from more intense inflammatory changes, characteristically localized in the anterior cornua of the cord, are motor.

Of a disease embracing such a variety of symptoms it is impossible to give a single, clear-cut, clinical picture. Wickman has differentiated eight clinical types, and as later observations have confirmed

his classification, it will be adopted and followed as closely as consistent with the scope and purpose of this paper. Wickman's types of acute poliomyelitis are as follows:

I. The *spinal poliomyelitic* type, characterized by onset with fever, gastro-intestinal disturbances or angina, headache, pain, often rigidity of the neck and spine, and pains in the extremities of varying intensity and distribution. From one to six days after the onset of the febrile symptoms there develops a paralysis which has certain distinctive characteristics, viz, it is a flaccid, motor paralysis. It is sudden in its onset, reaching its height within a few days, after which it shows a regression in extent, the final result being usually a permanent paralysis of considerably less extent than in the acute stage, although complete recovery may take place. This is the most common and easily recognized form of acute anterior poliomyelitis.

II. The *ascending* or *descending* type of paralysis runs the clinical course of a *Landry's paralysis.* Beginning usually in the lower extremities, the paralysis ascends until it involves, in some instances, the whole of the body; such cases usually terminate fatally from respiratory paralysis. In rare cases the paralysis is descending instead of ascending.

III. The *bulbar* or *pontine* form is characterized by paralysis of muscles supplied by cranial nerves (having their nuclei in the medulla or pons).

IV. The *encephalitic* type is distinguished by paralysis due to lesions in the motor area of the brain, resulting in a spastic monoplegia or hemiplegia. This is the rarest type.

V. In the *ataxic* type the characteristic motor disturbance is an acute ataxia, with or without paralysis.

VI. The *polyneuritic* type gives a clinical picture closely resembling multiple neuritis.

VII. The *meningeal* type includes (1) cases which in their onset are characterized by marked symptoms of meningitis, but which result in spinal or bulbar paralysis; and (2) cases in which the symptoms of meningitis are not followed by paralysis.

VIII. Abortive types include cases showing the initial symptoms of acute poliomyelitis, but not followed by paralysis.

The differentiation of these types depends upon the localization of the lesions in the central nervous system. The initial symptoms, before definite localization has taken place, are in a general way similar in all the types.

INCUBATION.

The incubation period of acute poliomyelitis has been found to vary from 3 to 46 days in monkeys experimentally inoculated, the more common period being from 7 to 15 days. The length of incu-

bation in persons has not been definitely determined. Approximately 2 to 10 days has been generally accepted as the most usual period, but apparently great variations occur, the period being sometimes prolonged to 3 weeks or more.

INITIAL SYMPTOMS.

Definite prodromal symptoms are relatively rare. In some cases malaise, weakness, digestive disturbances (nausea, constipation, or diarrhea), angina or restlessness precede by several days the definite onset of the acute febrile stage. Bronchitis and coryza more rarely precede an attack of acute poliomyelitis—probably not more often than may be ascribed to coincidence.

Sometimes the disease develops in two stages. After slight, indefinite prodromal symptoms, the patient apparently recovers completely in a few days, and with no suspicion of a serious illness, returns to the usual routine of life; but a few days later is stricken down by an acute attack of poliomyelitis. Wickman expresses the suspicion that these cases may be relapses, brought on by exertion, and may illustrate the therapeutic value of *rest*, after even a very mild attack of this disease.

Numerous cases are reported in which no constitutional disturbance was noted prior to paralysis. As such cases have most commonly occurred in children, it is probable that in many instances a mild febrile stage has been overlooked. It nevertheless appears to be certain that cases of poliomyelitis may develop with constitutional symptoms which are negligible.

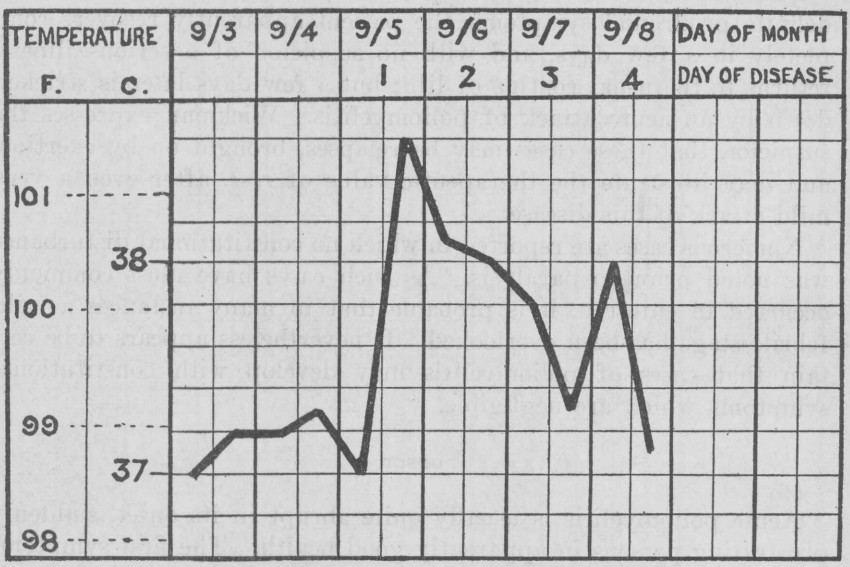
ONSET.

Acute poliomyelitis is usually quite abrupt in its onset, suddenly prostrating persons in apparently good health. The first symptoms observed vary a good deal. A sudden, sharp rise of temperature is probably the most common feature; a definite chill is rare. In its onset acute poliomyelitis may present the clinical picture of mild meningitis, acute neuritis, gastroenteritis, or tonsillitis. In some epidemics gastroenteritis has been the most prominent symptom of onset; in others, meningitis, neuritis, or tonsillitis. In a certain proportion of cases the onset is gradual and insidious.

GENERAL SYMPTOMS.

Fever is perhaps the most constant single symptom, although it seems fairly well established that some cases run their course without fever. The rise of temperature is usually sudden and sharp, often reaching its maximum in the first day of illness.

The range of temperature is quite variable. Wickman states that it is usually from 38° to 39° C. (99° to 102.2° F.). The investigation of the New York epidemic of 1907 showed the most usual range of fever to be from 101° to 104° F. Higher temperatures (105°–106° F.) have occasionally been noted. The height of the temperature is, according to Wickman, no index to the severity of the infection. It has been noted, however, by Lovett and Lucas[1] that cases in which the onset is accompanied by severe symptoms are more apt to result in extensive paralysis than cases with mild symptoms. Abortive cases, which recover in a few days, may, however, run quite as high temperatures as cases resulting in extensive paralysis or death.



C. R. D. Boy, 4 months old. Taken sick in General Foundling Asylum, Stockholm, Sept. 5, 1899. Extensive paralysis. Died Sept. 9, 1899. (Wickman, 1907.)

Exact records of the course of the fever are scarce. The following charts, adopted from Wickman, illustrate the irregularities in the development and course of the fever.

The fever may be expected to continue from one to seven days, usually falling to normal about the time paralysis develops. Cases are cited, however, in which the fever has continued for several weeks.

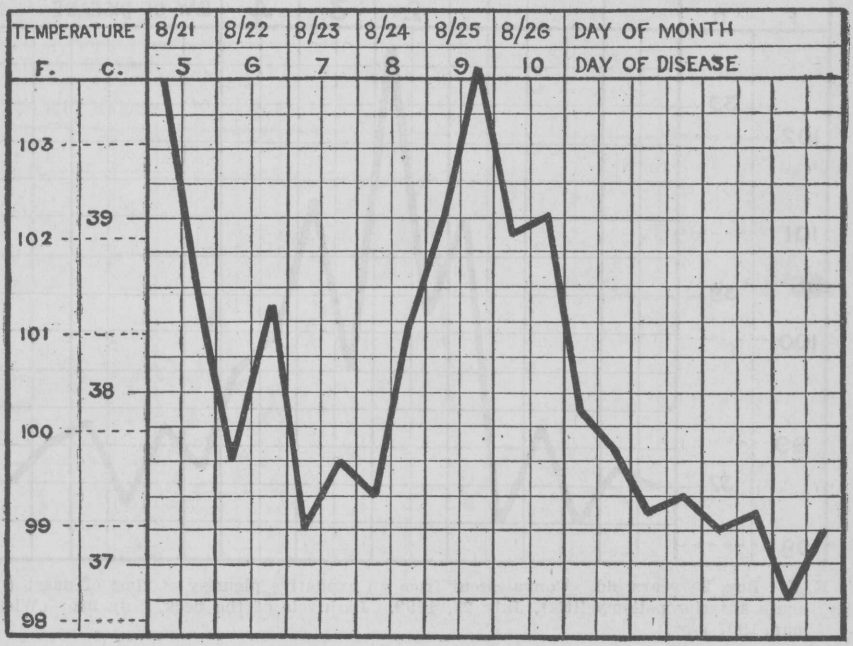
Headache is complained of by a large proportion of those who are old enough to give a clear account of their sensations. According to Wickman's observations the headache is usually occipital, but the committee which investigated the New York epidemic found it

[1] Lovett, R. W., and Lucas, W. P., Jour. Am. Med. Assn., 1908, vol. 51, pp. 1677–1684.

more commonly general or frontal. The headache is usually of moderate severity, such as is to be expected in almost any acute general infection, but is occasionally intense, constituting the most prominent symptom.

Prostration.—When the onset is sudden and acute there is marked prostration from the first. Even in cases with mild constitutional symptoms the prostration is often much greater than would be expected. Extreme weariness and muscular weakness are characteristic features of many mild, abortive cases.

Digestive system.—Some disturbance of digestion is among the most common early symptoms. Constipation is perhaps the most



J. P. Girl, 17 years old. Taken sick Aug. 17, 1905. Quite extensive paralysis of arms and legs. (Wickman, 1907.)

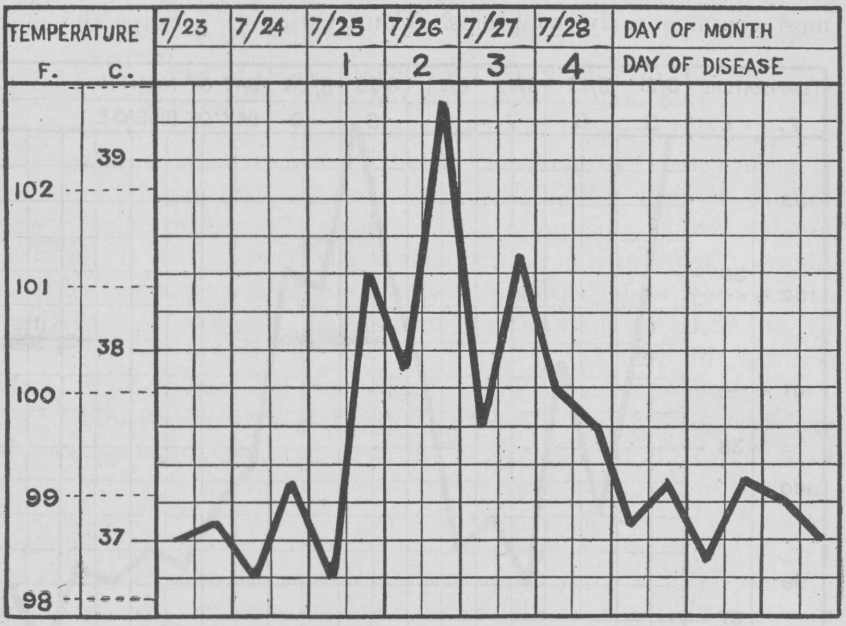
usual derangement. Diarrhea has been very common in certain epidemics, notably one in Westphalia studied by Krause,[1] but in recent American epidemics has been found to be much less usual than constipation. More serious disturbances of the bowels, such as obstipation or incontinence are rather rare. Vomiting is very common; it usually occurs early in the illness and subsides in a few days. When constipation is marked there is apt to be abdominal distention.

Respiratory system.—Catarrhal conditions of the respiratory system are rare, a point emphasized by Wickman as an aid in the early differentiation of acute poliomyelitis from influenza, which it often

[1] Krause, P., Deutsch. med. Wochenschr., 1909, vol. 35, p. 1822.

resembles in many respects. Sore throat is fairly common in some epidemics but is not in general a prominent symptom. More serious respiratory troubles, especially broncho-pneumonia, may develop later in the disease, due to paresis or paralysis of respiratory muscles.

Urinary system.—Although, post-mortem, the kidneys are sometimes found congested, with incipient parenchymatous degeneration, there is very seldom any clinical evidence of damage to the urinary organs. Albuminuria is rare. Retention of the urine occurs occasionally. Incontinence of urine is more rare.



K. J. Boy, 2½ years old. Convalescent from an exudative pleurisy at time of onset (of acute anterior poliomyelitis), July 25, 1899. Paralysis of the neck, July 26. (Wickman, 1907.)

Circulatory system.—The heart's action suffers the derangement common to acute infections. More serious disturbances—arrhythmia, tachycardia, bradycardia—may result from early involvement of the vagus center. Vaso-motor disturbances are sometimes observed, but are not characteristic.

The skin.—Excessive sweating has been noted by Müller[1] as a characteristic early symptom. While this has been noted occasionally in the epidemics studied elsewhere, it has not been found a constant distinctive feature. Skin eruptions were noted in less than 10 per cent of the cases investigated in the New York epidemic of 1907, and in 6 out of 150 cases studied in Massachusetts in 1909. The char-

[1] Müller, Eduard, Münch. med. Wochenschr., 1909, vol. 56, pp. 2460–2462.

acter of the skin lesions varied greatly. No skin eruption can be said to be at all characteristic of acute poliomyelitis. The rarity of herpes has been noted by Wickman.

NERVOUS SYMPTOMS.

Restlessness or irritability is a very common and marked symptom. In children very often the first symptom to attract attention is their irritability. Older persons frequently show an early stage of excitement, characterized by extreme restlessness, vague anxiety, and mental perturbation out of proportion to the severity of their other symptoms.

An *apathetic, drowsy state* frequently follows, especially in children. A child may lie sometimes for a day or more, asleep or half asleep, unless aroused; but when aroused, the faculties are clear, or at most, somewhat confused. *Coma* is rare, it being a noteworthy feature of acute poliomyelitis that even in fatal cases the patient usually retains consciousness throughout the greater part of the illness. *Delirium* is not uncommon, but is usually of short duration, occurs early, and is often a confused state rather than a wild delirium such as characterizes meningitis. *Convulsions* sometimes occur in children.

Pain and tenderness.—Pain of some sort is a very constant early symptom, giving clinical confirmation to the anatomical studies which have shown the involvement of the meninges and spinal ganglia of the cord in the acute stage. The most characteristic pain is in the back of the neck and spine; the whole of the spinal column may be painful, especially on motion, and very tender. It is Wickman's opinion that the pain which young children evidently feel on being moved is due rather to the tenderness and consequent rigidity of the spine than to general cutaneous hyperesthesia. The pain in the neck and back is sometimes extremely severe. Pains are also very common in the arms and legs, sometimes in the face, and less commonly in the trunk. The pains in the limbs may resemble a myalgia, without cutaneous hyperesthesia or tenderness over the nerve trunks. In other cases, however, the clinical picture is that of neuritis, with marked hyperesthesia and tenderness over nerve trunks. The pains may be of short duration, subsiding with or before the onset of paralysis, but in some cases tenderness over the nerve trunks persists for weeks.

Sensory disturbances other than pain have been observed occasionally. *Paræsthesias* are not uncommon; numbness is noted in a fair proportion of cases in older children and adults. Wickman describes a case observed by him, with motor paralysis of both lower limbs, great *diminution of the sense of pain* from the hips down, and

diminution of temperature-sense in the feet. He cites a similar case described by Wernicke, and also cites from reference other cases, in one of which there was complete *anesthesia*, and in several others *loss of electrocutaneous sensation.* Wickman thinks it probable from the pathology of the disease that closer observation of cases would show disturbances of temperature, pain, and electro-cutaneous sensation to be more common than heretofore reported.

Meningitic symptoms.—A train of symptoms indicative of meningeal irritation is frequently observed; these symptoms are—

Pain in the neck, which in varying degrees of intensity is quite common; *stiffness of the neck*, usually of moderate severity, rendering it painful or even impossible to bend the head forward; less frequently *retraction of the head*, due to actual contraction of the posterior neck muscles. While marked retraction of the head, such as is characteristic of cerebro-spinal meningitis is rare, a great many cases are found in which the head is held farther back than normal. *Stiffness of the spine* is usually due to the pain which motion of the spine causes, but in rare cases is due to actual *contracture of the spinal muscles.* A modified *Kernig's sign*—that is, inability to completely extend the leg when the thigh is flexed at a right angle, is sometimes present in cases showing other meningitic symptoms.

Some of the above symptoms are seen in the majority of cases with acute onset, as is to be expected from the fact that the pia mater is always found to be congested and infiltrated in the acute stage. Meningitic symptoms of moderate severity, when present, constitute the most characteristic symptom-complex of the early stage of acute poliomyelitis. When the meningitic symptoms are severe, the differentiation from cerebro-spinal meningitis may be impossible without examination of the cerebro-spinal fluid.

Motor symptoms.—Prior to the onset of paralysis, as well as in cases which do not result in paralysis, disturbance of the motor centers may be indicated by muscular twitching, jerking of the limbs, or tremor. The jerking of the limbs is usually slight and irregular. It may be most noticeable when the patient is asleep or may be brought on by the disturbance incident to making a physical examination. In one case which came under the writer's observation a slight disturbance of the patient set up clonic movements of one leg, continuing for several minutes. The other leg was flaccid, almost completely paralyzed.

Reflexes.—The patellar reflex is quite commonly exaggerated in the early stages, but is almost always diminished or abolished prior to the onset of paralysis; it may be abolished on one side and exaggerated on the other. In rare cases a persistently exaggerated patellar reflex may be associated with bulbar or cervical-segment paralysis (Wickman).

Reflexes other than the patellar have not been studied closely or extensively enough to warrant definite generalizations. Krause[1] found the skin, plantar, abdominal, and cremasteric reflexes normal in most cases. The New York investigating committee states that "A study of the reflexes established the general doctrine that in poliomyelitis the deep reflexes in the parts paralyzed are absent and that they are often absent in nonparalyzed parts."

Disturbances of the ocular reflexes are not characteristic, but need cause no surprise if noted in occasional cases.

Summarizing briefly the symptoms above enumerated, the characteristic features of acute anterior poliomyelitis in the early stage are sudden onset with fever, gastro-enteric disturbances (vomiting, diarrhea, constipation), occasionally sore throat, headache, restlessness followed often by apathy, pains in the neck, back, and limbs; muscular twitchings, exaggeration or abolition of tendon reflexes. Symptoms of a rather mild meningitis are present in a varying proportion of cases, and when present are rather characteristic.

The clinical picture prior to the onset of paralysis may be that of an indefinite general infection or toxemia, gastro-enteritis, tonsillitis, multiple neuritis, meningitis, or encephalitis.

TYPES OF ACUTE POLIOMYELITIS.

I. THE SPINAL POLIOMYELITIC TYPE.

The characteristic of this, the most typical and common form of poliomyelitis, is the development of a *flaccid motor* paralysis of parts supplied by nerves of *spinal* origin.

The paralysis develops, in the great majority of cases, within a week after the onset of acute symptoms, most commonly from the second to the fourth day. In exceptional instances it may develop more than a week after the onset.

In small children it is difficult to follow accurately the development of paralysis, which has given rise to the common statement that complete paralysis develops quite suddenly. Observations upon older persons have shown that the paralysis is often somewhat more gradual in its onset, beginning as a paresis, which may not be noted even by the patient until some effort is made which requires considerable strength. For instance, an adult, able to move his legs in bed, may not suspect a loss of power in them until he attempts to stand. Or the first indication of a beginning paralysis may be intention tremor and incoordination. In some cases the weakness never progresses to actual paralysis, receding sometimes to complete recovery in a few days. Cases of this kind are undoubtedly often overlooked in children. Usually, however, the paresis progresses rapidly to

[1] Münch. med. Wochenschr., 1910, vol. 57, p. 47.

complete paralysis of the affected part, reaching its maximum, both in degree and extent, within one or two days. It is quite the general rule that the extent of the paralysis is greater at first than it is later. The paralysis is primarily due to edema and infiltration of the cord. As the acute inflammation in the cord subsides, improvement takes place in some of the affected limbs. This improvement may be quite rapid, so that a limb which was completely paralyzed may return to normal in a few weeks, or even in a few days. Where the acute process has resulted in the destruction of the ganglion cells there is, of course, no return of function in the corresponding muscle. The parts supplied by nerves from these centers are permanently paralyzed. Between edema and infiltration, with transitory disturbance of the function of the ganglion cells in an area, and complete destruction of the ganglion cells, there are many gradations. The cells may be damaged so severely as to return slowly to normal, or only a part of the cells in an area may be destroyed, leaving the rest capable of performing their function. The rate and degree of improvement is correspondingly variable, and there is no certain means of telling, when the paralysis is at its maximum, how extensive it will finally be.

A sufficient number of statistics have been collected within the past few years to warrant some generalizations as to the parts most commonly paralyzed. The lower limbs are affected more than twice as frequently as the upper. Any combination may occur, as both legs, one leg and one arm of the same or opposite sides, both legs and one arm, both legs and both arms, both arms and one leg, both arms alone, etc. Combinations of parts supplied by the same spinal segment are more common than combinations of parts supplied by different segments—for example, it is more common to have paralysis of both legs than of one leg and one arm.

The resulting permanent paralysis, while it may involve one or more limbs in their entirety, is more usually limited to certain muscle-groups. In the lower limbs the groups most often affected are the peroneal and the quadriceps femoris. In the upper extremity the scapular muscles, deltoid, and upper-arm muscles are more frequently affected than the muscles of the forearm and hand.

Paralysis of the extremities is the most common and most serious feature of poliomyelitis; but the muscles of the trunk are affected more often than is generally supposed. In the acute stage the muscles of the back are very often paralyzed. As this usually occurs while the patient is confined to bed and in connection with paralysis of one or more extremities and is often of comparatively short duration, it may easily be overlooked. Paralysis of the neck muscles, rendering the patient unable to raise the head, is less common, occurs most frequently in combination with paralysis of the upper extremities, and can hardly be overlooked. Wickman lays emphasis upon

the comparative frequency of paralysis of the abdominal muscles, usually associated with rather extensive paralysis of the extremities. When abdominal paralysis is bilateral, distention is apt to result; the abdomen is flaccid and can not be contracted in expiratory efforts; the patient is unable to raise his body from the recumbent posture without support. Unilateral or more localized abdominal paralysis may give rise to hernia-like protrusions and retraction of the navel toward the unaffected side.

Paralysis of the bladder and rectum is very rare as compared with paralysis of the lower limbs. In cases with extensive paralysis of the extremities, retention of urine may occur, indicating a paralysis of the bladder, but this condition is almost without exception of short duration. Paralysis of the sphincters, with incontinence of feces or urine, is also rare.

Paralysis of the muscles of respiration seldom occurs except in cases of extensive paralysis. It may occur, however, with less extensive paralysis of parts supplied by the cervical segment of the cord. If the intercostal muscles are paralyzed, the chest is immobile, and respiration is of the abdominal type. Paralysis of the diaphragm causes a reversal of the usual *abdominal* movements in respiration; the abdomen is retracted in inspiration and protruded in expiration. Diaphragmatic paralysis is more serious than intercostal paralysis. Paralysis of both diaphragm and intercostals causes death by respiratory failure. Respiratory paralysis is the gravest symptom of acute poliomyelitis. Occasionally, however, the disturbance of function of these muscles does not reach total paralysis, and in such cases may be quite transient. If the patient does not die of the respiratory paralysis or of pneumonia secondary thereto, these muscles are likely to return to their normal function.

The tendon reflexes in paralyzed limbs are totally abolished in the great majority of cases. Prior to the onset of paralysis, however, the reflexes, especially the patellar, are quite commonly exaggerated. The exaggeration of any reflex, however, need not cause astonishment. Even after the development of complete *flaccid* paralysis of a limb, exaggeration of the patellar reflex may persist. This, according to Wickman's view, may be accounted for by a lesion in the pyramidal tract of the cord, overcompensating the diminution of the reflex caused by the partial destruction of the ganglion cells of the anterior cornua.

The eye reflexes have been found disturbed in various ways. The pupils may react sluggishly or unevenly. Wickman has noted in rare cases signs of involvement of the cilio-spinal center in the cervical cord, viz, narrowing of the aperture between the lids of one side, with contraction of the pupil.

The subsidence of the symptoms which characterize the preparalytic stage is usually about coincident with the development of paralysis. The temperature may, however, in rather exceptional cases persist for a week or more after this time. The persistence of other constitutional symptoms is variable. While the acute pains in the limbs usually subside about this time or even before, there is often a persistent tenderness of the muscles and nerve trunks, and considerable pain in the joints on attempted passive motion.

Stage of regression.—As already stated, following the development of paralysis, there is a more or less indefinite stage of improvement. Up to a certain point this improvement is rapid, being noticeable from week to week, perhaps from day to day, until a part or in some cases all the paralyzed muscles are restored to their normal function. After the first few weeks improvement is much slower; the parts which remain paralyzed now show atrophy, and quite frequently a lowering of surface temperature. This indicates a severe degree of damage to the spinal motor centers, but not necessarily complete destruction. Even after several months, improvement may continue to take place in muscles that appeared completely paralyzed.

Contractures of the muscles and deformities of the limbs due to such contractures or to overaction of healthy muscles opposing paralyzed muscles are likely to occur in this stage unless care is exercised in their prevention.

The electrical reactions of the paralyzed limbs usually show alteration by the second week. The alteration may consist of a diminution of the normal electrical excitability of the muscles or more profound changes—the reaction of degeneration. The development of the reaction of degeneration, denoting destruction of the spinal center of a muscle, is given by Church and Petersen[1] as follows:

"First. The *muscle* responds weakly, sluggishly, and deliberately to faradism, and shows a tendency to maintain the contraction after the current is withdrawn. This is the *modal change.*

Second. The *nerve trunk* loses progressively and equally its responsiveness to both galvanism and faradism—a quantitative change.

Third. The *muscle* becomes much more excitable by galvanism and much less excitable by faradism, which latter reaction, with the nerve trunk responses, is completely lost after two or three weeks. This is the *qualitative change.*

Fourth. A *polar change* appears in the *muscle* about the second week, when directly stimulated by galvanism. The anodic closing contraction now equals or exceeds the cathodal closing contraction.

If there has been complete destruction of the nerve elements, with complete degeneration, *all electrical response* is gradually lost."

[1] Church, H., and Petersen, F.: Nervous and Mental Diseases. Phila., 1907, W. B. Saunders Co., 5th ed., p. 46.

II. THE ASCENDING OR DESCENDING TYPE OF PARALYSIS.

In the more common spinal type of poliomyelitis the paralysis is pretty definitely limited, reaching its maximum in a few hours or a few days. In some cases, however, the paralysis progresses from the part first affected, either ascending or descending until nearly the whole of the body is paralyzed, or until death ensues from paralysis of the respiratory muscles. Usually the progress of the paralysis is *upward*, affecting in order the legs, abdomen, back, intercostals, arms, neck, and diaphragm. The progress may be very rapid; in one case which came under my observation, death ensued from respiratory paralysis 48 hours after the onset of the illness and less than 24 hours after paralysis was first noted. In some epidemics cases of this type have been observed more frequently in young adults and older children than in infants. The clinical course of a case of ascending or descending acute poliomyelitis is identical with that of a Landry's paralysis. Wickman has shown that a number of cases diagnosed as Landry's paralysis were in reality acute ascending poliomyelitis. He also doubts whether this type is more common in adults than in children, ascribing the general impression to that effect to the greater accuracy with which the course of the paralysis can be followed in an adult.

When the paralysis is of the descending type, appearing first in the upper extremities or in the muscles supplied by the cranial nerves, death from respiratory failure is likely to occur before the lower limbs are affected.

Respiratory failure may be due either to paralysis of the respiratory muscles (intercostals and diaphragm), as already described, or to a lesion affecting the respiratory center in the medulla. In the latter case the onset of dyspnœa is more sudden. Cheyne-Stokes respiration and acceleration of the heart's action may be noted in such cases, due to interference with the vagus center.

It is typical of such cases as the above that the patient retains consciousness to the end.

III. BULBAR (MEDULLARY) OR PONTINE TYPE.

This type embraces those cases in which there is paralysis of muscles supplied by *cranial* nerves whose nuclei are situated in the medulla oblongata or pons cerebri. It includes: (*a*) cases in which, in addition to the paralysis of the cranial nerves, there is paralysis of typical spinal type, and (*b*) cases in which the only paralysis is bulbar.

Wickman cites Medin (1890) as the first to call attention to the frequency of paralysis of cranial nerves in epidemic poliomyelitis. The unexpected frequency of this form of paralysis has been one of

the striking facts brought out by the recent studies of Wickman, the New York Investigation Committee, the Massachusetts State Board of Health, and others. In the report of the New York committee paralysis of the cranial nerves was found as follows: Facial, 27; eye muscles, 26; eyelids, 18; speech, 28; out of a total of 625 to 700 cases. The report of the Massachusetts State Board of Health for 1909 records 34 cases of facial paralysis in a total of 628 cases of poliomyelitis (4.7 per cent). In 150 cases studied more carefully double vision was noted in four cases, difficulty of deglutition in two, difficulty of speech in two. Medin [1] noted involvement of some of the bulbar centers in 17 out of 65 cases (=26 per cent). It would appear from the wide variations between these statistics that there must be differences in epidemics in regard to the frequency of the occurrence of cranial-nerve paralysis.

The most common paralysis of this type is facial, which is much more often unilateral than bilateral. Ocular paralyses are next in frequency. The external rectus is most frequently affected, causing the eye to turn inward. Paralysis of the oculo-motor may cause divergent squint, with or without ptosis; or, more rarely, ptosis may be the only indication of ocular paralysis. In very rare cases there is paralysis of all the muscles of the eye. Transient motor disturbances of the eye, either nystagmus, diplopia, or fixedness of the eyes may occur. Wickman cites two cases in which the optic nerve was affected, with resulting atrophy and blindness of one eye.

Sudden deafness, usually of short duration, has been noted in the course of acute poliomyelitis, but it is a very rare symptom.

Disturbances of deglutition, when they occur, are usually associated with rather extensive paralysis, although there are exceptions to this. Disturbances of speech were noted in 28 cases (not noted in 615 cases) collected by the New York investigation committee.

Paralysis affecting the respiratory center may also be included as a "bulbar" symptom. In a case reported by the writer sudden death was due apparently to this cause. The patient, a previously healthy girl of 12, after a few days of slight illness, died very suddenly, with no paralysis. There having been no distinctive symptoms of poliomyelitis in this case, the diagnosis was made only by post-mortem histological examination.

Paralysis of bulbar origin is often temporary, as is to be expected from the fact, already cited, that the damage to the ganglion cells in the bulb is usually less severe than in the cord.

Lesions in the white matter which forms the conducting tracts from the cerebellum may perhaps give rise to the acute ataxia noted in some cases. Similar lesions in the pyramidal tracts of the medulla,

[1] Cited by Wickman: Beiträge zur Kenntniss der Heine-Medin'schen Krankheit, p. 27.

interrupting the connection between the cerebral and spinal motor centers, may give rise to exaggeration of reflexes.

IV. CEREBRAL OR ENCEPHALITIC TYPE.

There has long been a discussion among clinicians as to the occurrence, in acute anterior poliomyelitis, of paralysis due to lesions in the motor area of the brain. Paralysis due to such a lesion would be monoplegic or hemiplegic, and spastic, resulting in contractions, but no atrophy. A similar paralysis might result from lesions in the conducting tracts of the upper motor segment.

Paralysis of this type is extremely rare in acute poliomyelitis. Wickman found no example of it among the 1,031 cases studied by him, and the collective investigation of the New York epidemic failed to reveal any case of this type. Wickman, however, considers the occurrence of the type established by the following evidence:

1. Lesions are commonly found in the cortex of the brain at autopsy, even when there has been no clinical evidence of the existence of such lesions.

2. A number of authors [1] have noted, in epidemics of acute poliomyelitis, the occurrence of cases presenting initial symptoms similar to acute poliomyelitis, but resulting in spastic hemiplegia. Pasteur, Buccelli, and Hoffman noted instances in which two or more children of a family were affected about the same time, with similar initial symptoms. One child developed spastic hemiplegia, and the others developed the typical spinal flaccid paralysis of acute poliomyelitis.

3. Others have reported the coexistence in the same patient of typical flaccid spinal poliomyelitic paralysis in one part of the body, and equally typical spastic cerebral paralysis in other parts. Pierre-Marie and Rossi [2] were able to demonstrate by autopsy on a case of this kind well-marked cortical and spinal lesions.

V. THE ATAXIC TYPE.

This type, like the preceding, was first differentiated by Medin, who noted, during the Stockholm epidemic, cases whose motor disturbance was an ataxia of cerebellar type, associated with exaggerated reflexes, not followed by atrophy, and terminating usually in recovery. According to the observations of Wickman and to more recent observations in the United States, cases in which a marked ataxia is the only motor disturbance are relatively rare. It is not rare, however, to find some degree of incoordination associated with

[1] Moebius, Schmidt's Jahrb., 1884; Pasteur, W. Trans. Clin. Soc., 1897; Buccelli, Policlinico, 1897; Strümpell, Beitr. z. path. Anat. u. klin. Med., Leipzig, 1897; Medin, cited by Wickman, Beiträge zur Kenntniss der Heine-Medinschen Krankheit, p. 72; Hoffman, cited by Wickman, ibid.

[2] Cited by Wickman. Beiträge zur Kenntniss der Heine-Medischen Krankheit, pp. 74–75.

paresis in the early stage of cases of acute poliomyelitis. Incoordination may be ascribed to several possible causes:

(1) Lesions of the cerebellum.

(2) Lesions in the conducting tracts leading from the cerebellum.

(3) Lesions in the posterior cornua of the cord, affecting muscle sense.

(4) Paresis of limbs, especially paresis of certain groups of muscles, disturbing the balance between these and their opposing (unaffected) muscles.

(5) Peripheral neuritis.

There is ground to believe that any of the above lesions may occur, in varying degrees, in cases of poliomyelitis, except, perhaps, peripheral neuritis, which is considered improbable.

VI. POLYNEURITIC TYPE.

Wickman describes three classes of cases *clinically* resembling multiple neuritis:

(1) Cases which in the stage of onset are characterized by marked pain and tenderness in the extremities, but which recover without paralysis or with only a transitory paresis; many of these cases could perhaps be better classed as abortive.

(2) Cases in which, after the subsidence of acute symptoms, there remains tenderness of the nerve trunks.

(3) Cases of acute ataxia associated with marked pain and tenderness; such cases may be considered as belonging to either the ataxic or the polyneuritic type, dependent upon the predominance of ataxia, or of pain and tenderness.

The frequent occurrence during epidemics of cases clinically resembling acute polyneuritis, and the occurrence of symptoms of neuritis in cases of undoubted poliomyelitis, are sufficient to establish the identity of causation between this type and the paralytic form of the disease. There has been some discussion as to whether the clinical picture in these cases is actually due to inflammation of the peripheral nerves or to lesions in the cord. Wickman concludes that the lesions are central, giving the following reasons:

(1) There is no loss of sensation in these cases, whereas in toxic, peripheral neuritis, loss of senation is usually more marked than loss of motion.

(2) Post-mortem examination has failed to reveal peripheral neuritis in cases of poliomyelitis, even where the involvement of the cord was very extensive.

(3) These symptoms may all be accounted for by lesions which are quite generally found in the spinal cord in cases of poliomyelitis,

viz, diffuse infiltration and edema of the whole cord, infiltration and edema of the pia mater.

Infiltration of the spinal ganglia has, however, been found at autopsy in cases of poliomyelitis, both human [1] and experimental.[2]

VII. THE MENINGITIC TYPE.

Symptoms indicative of a mild grade of meningitis have been noted (p. 28) as among the most characteristic manifestations of acute anterior poliomyelitis in the early stage. Such symptoms are not usually, however, predominant over the other symptoms. In some cases, the proportion of which seems to vary in different epidemics, the most striking symptom complex of the acute stage is as follows: Intense headache, ocular disturbances, pain in the neck and back, retraction of the head, contracture of the spinal muscles, spasticity of the limbs, Kernig's sign. No better idea can be given of this type than by citing a case of Wickman's:

Wickman's case No. 299.—Boy, 10 years old; taken sick August 18, 1905, after feeling badly for several days. Headache, fever, and vomiting; could be up and out of bed for the first few days, after which he was confined to bed; paresis noted later; stiffness of neck, pains in his whole spine; could not support himself upon his legs.

August 29: Lies with head retracted and stiff neck; cries when attempt is made to raise his head from pillow; can move head from side to side, but can not raise it; is tender all over spine and can hardly bear to be lifted up; the back is bowed (opisthonos) so that the patient can not lie flat upon the mattress; lying upon his back, he is supported upon his shoulders and hips, and a hand may be passed between his back and the bed; patient keeps his knees flexed—can extend them, but only with considerable pain; the feet hang limp and can neither be flexed nor extended; belly retracted and walls tense and tender; abdominal muscles paretic; patellar reflex abolished; cremaster and abdominal reflexes marked.

After about seven weeks in bed he was able to crawl upon the floor and eventually to walk.

October 22, 1905 (examination by Wickman): Gait "wabbly," paretic; patient drags the toes; is said to fall often; leg muscles atrophic and flaccid; thigh muscles feel flaccid, but not noticeably atrophied; diminished dorsal flexion of left foot; otherwise movements of legs all possible but weak; glutei of both sides weak, but can be contracted; has difficulty in raising himself after bending over, as to pick up something from the floor, helping himself up with his hands upon his legs like a person with progressive muscular atrophy. On raising the leg, extended on the thigh, patient feels pain at an angle of about 45°; no pain if knee is flexed; no contractions of muscles; patellar reflex on both sides exaggerated; no ankle clonus.

The diagnosis of poliomyelitis in this case is confirmed by the subsequent development of flaccid paralysis.

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[1] Strauss: Epidemic Poliomyelitis, Report of the Collective Investigation Committee on the New York Epidemic of 1907, p. 87.

[2] Flexner, Jour. A. M. A., 1910, vol. 55, pp. 1105–1113.

In other cases, however, there may be no paralysis, or what may be even more confusing, an ocular or facial paralysis. A fatal case of this kind, reported by Wickman, is abstracted for illustration:

H. K., female, age 27, married; taken sick suddenly August 19, 1905, with fever, headache, pains in back; next day vomiting so violently as to dislocate the jaw; tenderness and stiffness of neck, increasing until head was moderately retracted; violent tonic contraction of the shoulder muscles, throwing the arms up to the head; tonic contractions, flexing elbows, flexing fingers, and adducting thumb; cramp in muscles of the lower jaw, drawing it downward; no ocular paralysis; cramps so painful as to require chloroform; evening temperature 37° C. (99.6° F.). Patient fully conscious; during night cramps continued, and later affected muscles of back, causing opisthotonus.

August 21: Morning temperature 38.8° C. (101.8° F.); patient being six months pregnant, eclampsia was suspected, and forced delivery undertaken successfully; cramps continued, extending to legs; inability to swallow and difficulty of speech developed later in the same day; condition continued until death, at 6 a. m., August 22. Patient conscious throughout.

An autopsy was performed, revealing typical histologic lesions of acute poliomyelitis. The cerebrospinal fluid was found greatly increased in quantity and quite clear.

It will be noted that in neither of the above cases was there loss of consciousness. This can not, however, be taken as a constant point of differentiation between the meningitic form of acute poliomyelitis and cerebrospinal meningitis, for cases of poliomyelitis are cited, both by Wickman and by others, in which there was delirium followed by coma.

While there should be no great difficulty in distinguishing between an epidemic of poliomyelitis and an epidemic of cerebrospinal meningitis, there may be great difficulty in making the diagnosis in a particular case, especially if not closely associated with an epidemic of either disease. Lumbar puncture, with examination of the cerebrospinal fluid, is the only certain means of differentiation in such cases.

VIII. ABORTIVE FORMS.

Wickman[1] cites Breiglieb, Pasteur, and Leegard as having noted in intimate association with cases of undoubted poliomyelitis other cases of illness with strikingly similar initial symptoms, but terminating in rapid and complete recovery without paralysis. Caverly[2] also noted during an epidemic of poliomyelitis around Rutland, Vt., in 1894, that the prevalent diseases of children were accompanied by unusual nervous manifestations. It was Wickman, however, who first clearly pointed out the frequent association between cases of undoubted poliomyelitis and cases of a similar illness not followed by paralysis. It was he who recognized the latter as mild or abor-

[1] Wickman: Beiträge zur Kenntniss der Heine-Medinischen Krankheit, p. 132.
[2] Caverly, S. C., Med. Record, 1894, vol. 46, p. 673.

tive forms of acute anterior poliomyelitis, and called attention to the importance of including them in studies of the epidemiology.

In regard to the cases of illness without paralysis, considered by Wickman and others to be abortive forms of poliomyelitis, there are two chief points to be considered; first, whether such cases are due to the same infection as the paralytic forms of poliomyelitis, and second, whether they are clinically distinguishable from other infections.

(*a*) *Etiologic identity of abortive and paralytic forms.*—It has already been noted that the symptoms of acute poliomyelitis are due to *general infection*, *diffuse inflammation* of the central nervous system, and more severe *localized lesions* of the cord and brain. The localization of the nervous lesions gives to this disease its characteristic features and distinguishes the various types from one another. An abortive case of poliomyelitis may be considered as a case presenting only the symptoms of general infection and perhaps some diffuse inflammation of the cerebrospinal axis. The symptoms referable to these causes would be the same as the early symptoms in cases which later develop characteristic paralysis. There has been no pathologic evidence brought forward to prove the anatomic changes assumed as occurring in abortive cases, but the clinical evidence is very strong:

(1) Many observers in many parts of the world have noted during epidemics of poliomyelitis *cases presenting the same initial symptoms as paralytic cases*, but recovering in a short while without paralysis.

(2) Almost every closely studied epidemic shows a *gradation in severity of nervous symptoms*—extensive permanent paralysis; slight transient paralysis; partial paralysis (paresis); ataxia without paralysis; meningitic or neuritic symptoms without motor disturbances; general infection without distinctive nervous symptoms of any kind. A group of cases showing all these gradations, occurring in a circumscribed area within a short time, all presenting somewhat similar initial symptoms, differing to some extent from the symptoms of more usual infections, seldom fails to convince the observer of the existence of abortive cases of poliomyelitis.

(3) The occasional occurrence of such cases during an epidemic of poliomyelitis might be put down to merely coincident prevalence of two or more distinct infections; the *frequent, almost constant, occurrence* of such cases in intimate association with frank cases of poliomyelitis can not be ascribed to fortuitous coincidence.

(4) Experiments have demonstrated that monkeys inoculated with poliomyelitis occasionally develop an abortive form of the infection, characterized by rather mild and indefinite symptoms. Roemer and Joseph[1] have demonstrated in monkeys an immunity following such abortive attacks.

[1] Roemer, P., and Joseph, K., Münch. med. Mochenschr., 1910, vol. 57, pp. 520–522.

(5) Netter and Levaditi [2] have shown that the serum of a child recently recovered from an abortive attack was capable of neutralizing the virus of poliomyelitis. This property had been previously demonstrated in the serum of persons and monkeys who had recovered from frank attacks of poliomyelitis and had been shown to be absent from the serum of normal persons and monkeys. It may be taken as convincing evidence of infection with the virus of poliomyelitis.

It is therefore well established by clinical and experimental evidence that the infection of acute anterior poliomyelitis may cause slight illness without definite motor disturbances.

(*b*) *The recognition of abortive cases.*—Granting the occurrence of abortive cases, their recognition remains a difficult problem. In the obsence of any specific diagnostic test it is necessary to recognize, by clinical observations alone, cases which do not present clear-cut clinical characteristics.

The symptoms vary greatly in kind and degree. There is usually some fever, often of very short duration, sometimes less than a day. Headache is one of the most constant features. Many cases exhibit an unusual degree of physical weakness and indisposition to exertion. Nausea and vomiting, associated with either diarrhea or constipation, are the most prominent symptoms in some cases. Restlessness and mental anxiety may be marked in older persons; irritability or drowsiness in children. Pain of some kind is a very common symptom. It may be a neuritic pain of the extremities, with hyperæsthesia, or it may be a myalgic pain of the neck and back. The most characteristic, though perhaps not the most common, pain is in the back of the neck, sometimes extending down the spine. Tenderness over the spine is, in some groups of cases, a common and characteristic symptom. Slight motor disturbances, such as slight paresis, ataxia, or diplopia, may be noted. If definite disturbance of motion can be made out, the case should be classed as frank rather than abortive poliomyelitis; but in children the only manifestation may be a rather indefinite clumsiness or indisposition to use the legs. Disturbance of the patellar reflex, either exaggeration, diminution, or abolition, is often noted. Convulsions or muscular twitchings may be observed in children.

Wickman distinguishes four clinical types of abortive cases:

(1) With symptoms of general infection.

(2) With gastroenteritis.

(3) With pain and hyperesthesia (like neuritic influenza).

(4) With meningitic symptoms (severe occipital headache, pain and tenderness in neck and back, and rigidity of neck).

[2] Netter and Levaditi, Compt. Rend. Soc. Biol., 1910, Vol. LXVIII, No. 18, pp. 855–857.

Cases of the same type quite commonly occur in groups. In one epidemic most of the abortive cases may be of the gastroenteric type, in another of the meningitic type. The meningitic and neuritic types are apparently the more common forms. This may, however, be due to the fact that these forms are more *distinctive*, having less resemblance to the common epidemic diseases of summer, and are therefore more often recognized. There is, of course, no sharp line of differentiation between these several types of cases, which have been classified only according to the most prominent symptoms.

Frequency of abortive cases.—Wickman found among 1,025 cases of poliomyelitis studied in Sweden in 1905, 868 frank cases with paralysis, and 157 (= 15 per cent) abortive cases. He is of the opinion, however, that the proportion of abortive cases is greater than this. In Trästena, a small community where abortive as well as frank cases could be traced, he found 23 abortive cases (= 46 per cent) among a total of 49; in Atvidaberg, 11 abortive cases out of 31 (= 35 per cent), and in Smedjeback, 28 out of 50 (= 56 per cent).

Müller,[1] reporting an epidemic of 700 cases in the island of Nauru in January, 1910, states that many cases recovered without paralysis, and that many others had only slight paresis of two weeks or less duration. Only 50 cases had paralysis remaining after three months.

Anderson [2] observed in Polk County, Nebr., in the summer of 1909, 86 cases, of which 39 (= 44 per cent) had no definite paralysis.

The intensive study of 150 cases by the Massachusetts State Board of Health revealed 49 cases of illness, possibly abortive cases of poliomyelitis, occurring in the same houses with the 150 frank cases.

Mention has already been made of an epidemic which occurred in May, 1910, in a rural school district in Hancock County, Iowa, investigated later by the writer. Within a period of three weeks 30 cases of illness of the same general type occurred among 8 of the 12 families in attendance at this school. Five cases, resulting in typical, definite paralysis, were undoubtedly frank poliomyelitis. The remaining 25 may be considered in all probability abortive attacks of the same infection. The most common symptoms in this group were severe headache, pains in the limbs and back, stiffness of neck and spine, and gastrointestinal disturbances (nausea and constipation).

The proportion of abortive cases reported in various epidemics varies greatly, as is to be expected from the different circumstances under which the epidemics have been studied. In practically every epidemic studied in the light of Wickman's observations some abortive cases have been noted, and it may be stated pretty generally that the closer the observation the greater has been the proportion of abortive cases. From a review of the literature and from personal

[1] Müller, A., Arch. f. Schiffs u. Tropen. Hyg., 1910, vol. 14, No. 17.
[2] Anderson, C. A., Pediatrics, 1910, vol. 22, pp. 543–558.

observations in several localities where poliomyelitis was epidemic I am of the opinion that abortive cases are probably as numerous, and very possibly more numerous, than frank cases.

DIAGNOSIS.

Except in rare cases the diagnosis offers no difficulties after the onset of paralysis. The sudden onset of flaccid paralysis of one or more extremities, without loss of sensation, during or immediately following an acute febrile disturbance, is sufficiently characteristic. The rapid regression of paralysis of some of the parts, the reaction of degeneration, and atrophy of those muscles which remain paralyzed, complete the diagnosis. Even when the paralysis is transitory the diagnosis should offer no great difficulty if a satisfactory history can be obtained. Those cases in which cranial-nerve paralysis occurs without spinal paralysis have doubtless been frequently overlooked or wrongly diagnosed, and require more care in excluding local causes. The diagnosis in cases where the paralysis is of the cerebral, hemiplegic type will be very doubtful unless the early symptoms are quite typical and examinations of cerebro-spinal fluid and blood are made, or unless the association with undoubted cases of poliomyelitis has been striking. The combination of a flaccid paralysis with spastic, hemiplegic, paralysis would make the diagnosis of the cerebral type quite probable. While it is the general rule that the tendon reflexes are diminished or abolished in paralyzed extremities, the finding of persistent exaggerated reflexes does not exclude the diagnosis of poliomyelitis.

It is important to make the diagnosis, wherever possible, before the onset of paralysis, as well as in cases where no paralysis develops. This is obviously of great importance for the success of prophylactic measures. There is no evidence at present that failure to make an early correct diagnosis prejudices the patient's chance of recovery, but in the event that any effective specific treatment should be developed it will be necessary to employ it early. Before the use of a specific remedy it would be advisable to confirm the diagnosis by examinations of cerebrospinal fluid and blood, but the physician must at least suspect poliomyelitis from clinical evidence.

An acute febrile illness, with sudden onset, probably gastrointestinal disturbances, and symptoms of a mild meningitic inflammation, or other nervous symptoms, such as hyperesthesia, pains in the limbs, exaggerated or abolished tendon reflexes, ataxia, tremor, etc., warrants the suspicion of poliomyelitis. The diagnosis in such cases may be reasonably certain if the disease is known to be prevalent in the community. In the vicinity of Mason City, Iowa, the local physicians found it possible to make a fairly definite diagnosis prior to the development of paralysis in a considerable proportion of cases, prob-

ably 50 per cent. In other localities the proportion of cases showing distinctive initial symptoms may be smaller. There is always a certain proportion of cases in which the symptoms prior to paralysis are so slight or so indefinite as to arouse no suspicion of poliomyelitis, even in the presence of an epidemic.

The diseases with which poliomyelitis is most likely to be confounded are influenza, multiple neuritis, muscular rheumatism, acute articular rheumatism, gastroenteritis, and cerebrospinal meningitis. Certain forms of influenza may cause any or all of the symptoms seen in the early stage of poliomyelitis. Important considerations in the differential diagnosis are the frequency of catarrhal conditions of the respiratory tract in influenza and their rarity in poliomyelitis; the common occurrence of ear troubles in influenza; the greater prevalence of influenza in the winter months and of poliomyelitis in the summer months.

As already stated, the polyneuritic type of poliomyelitis gives at first a clinical picture identical with that of acute neuritis, from which it can be distinguished only by the subsequent developments. The swelling of the joints in acute articular rheumatism and the usual absence of severe constitutional symptoms in myalgia readily differentiate these diseases. Occasionally there is in acute anterior poliomyelitis tenderness and swelling of the joints, making the differentiation from acute articular rheumatism exceedingly difficult. Cerebrospinal meningitis can, in the great majority of cases, be excluded without lumbar puncture. Quantitative and differential leucocyte counts may be of aid, for in cerebrospinal meningitis the leucocyte count is high and the polymorphoneuclear leucocytes relatively increased, while in poliomyelitis there is more apt to be a leucopenia with relative increase in lymphocytes. In cases of the severe meningitic type, however, lumbar puncture is the only certain means of diagnosis.

TREATMENT.

No specific treatment has been developed.

The treatment in the acute stage must therefore be symptomatic, directed along the same general lines as in other acute infectious diseases.

Rest is important, and even in very mild cases should be enforced for a while after the subsidence of acute symptoms.

Moderate purgation is recommended, and, if necessary, enemata. Diuresis should be promoted by the free administration of water, or by saline enemata, in cases requiring them. Hot packs are useful to promote diaphoresis and to relieve the restlessness and the pains.

The diet during the acute stage should be liquid, easily digestible, and nutritious.

The administration of urotropin (hexamethylene-tetramine) is advised on the ground that formalin is excreted into the cerebrospinal fluid. While there is no proof as yet that this drug has any effect in modifying the course of the disease, its use is free from any valid objection and is quite generally recommended.

Except when absolutely necessary to allay severe pain, drugs which have for their purpose the relief of nervous symptoms should be avoided, especially the antipyretics and analgesics. Morphine or codeine may be given when necessary to allay pain.

When symptoms of pressure are present a lumbar puncture may be made, with the hope of relieving the condition. This should be done, however, with the strictest aseptic precautions, should not be undertaken by the inexperienced, and is not advised as an indiscriminate therapeutic measure.

The effect of any treatment in the acute stage is extremely difficult to ascertain. Quite independently of any treatment, cases which have severe early symptoms may recover in a few days with no paralysis at all, while other cases, with less severe initial symptoms, may result in extensive paralysis or death. The proportion of abortive and paralytic cases also varies greatly. In some epidemics half of the cases have been of the abortive type. It is therefore evident that even the complete recovery of a large proportion of apparently severe cases does not indicate that there was any specific virtue in the treatment which they received.

It is, however, reasonable to suppose that intelligent treatment along the lines indicated above will aid the patient in combating the infection, and will have some effect, however slight, in modifying the course of the disease.

The objects of treatment after the subsidence of the acute stage are to promote the comfort and general nutrition of the patient, prevent contractures and deformities of paralyzed parts, and to maintain the nutrition of paralyzed muscles. The general health of the patient is usually good, and intelligent supervision of diet, ventilation, etc., is all that is necessary to maintain it. Tonics may be given, but the administration of strychnine in doses larger than are usually given in tonics, with the idea of its exerting a specific restorative action upon the spinal motor centers is to be avoided.

For a considerable time after the onset of paralysis there is often pain in the paralyzed parts, due to sensitiveness of the nerve trunks. Motion may be very painful, and the limbs often become quite rigidly flexed. Hot baths, with gentle exercise, will do much toward relieving this pain and preventing or lessening contractures. Massage and electricity are contraindicated while pain and tenderness persist. Deformities must be prevented by suitable mechanical appliances, designed to keep the limbs in proper anatomic position.

For the treatment of the residual paralysis, after the subsidence of all acute symptoms, the reader is referred to articles cited in the appended bibliography and to the standard textbooks on orthopedic surgery.

PROPHYLAXIS.

While there are differences of opinion as to the contagiousness of epidemic poliomyelitis, its probability has been sufficiently demonstrated to render preventive measures imperative.

The patient should be isolated as completely as possible in a clean, bare room, well screened to keep out insects. The members of the family, other than the necessary attendant, should not be allowed to come into contact with the patient. All discharges, including sputum, nasal secretions, urine, and feces, as well as all articles (linen, eating and drinking utensils, etc.) which may be soiled by such discharges, should be thoroughly disinfected before they leave the sick room. The nurse and physician should observe the same precautions regarding their hands and clothing as in attending a case of scarlet fever.

The rest of the family should, so far as possible, be kept out of contact with the neighbors, at least to the extent of excluding the rest of the family from school and prohibiting all unnecessary visiting. The period during which isolation should be maintained is as yet indeterminate. Three weeks would seem to be a reasonable minimum for exclusion from school; but in some cases it may prove more practicable to fumigate the premises earlier than this.

Since the virus can be killed experimentally by a 1 per cent solution of peroxide of hydrogen, an antiseptic gargle of this solution is recommended to be used by the patient and other members of the family. The mentholated powder above mentioned (page 12) might perhaps be substituted for or used in conjunction with this solution.

As soon as practicable after the recovery of the patient the house should be fumigated with formaldehyd. If vermin are present, it would be advisable to use sulphur instead of formaldehyd.

In the presence of an epidemic it would be advisable to keep down the dust by sprinkling streets and yards. This is recommended because dry-weather conditions have seemed generally more favorable to the spread of epidemic poliomyelitis and because in several instances the abatement of dust has been followed by the cessation of an epidemic. It would also be advisable during an epidemic to keep children off the streets and away from public gatherings, to prohibit their using public drinking cups, to pay careful attention to their diet, to prevent gastrointestinal disorders, and to protect them from overheating and overexertion. which might lower vital resistance.

It is beyond the scope of this paper to enter into a discussion of State and municipal preventive measures. There is, however, practically unanimous agreement on the following points:

(1) That the disease should be required to be reported to the health authorities.

(2) That patients should be isolated.

(3) That members of their family should be excluded from schools for at least three weeks.

The degree of effectiveness of prophylactic measures is very problematic. A very apparent obstacle is the difficulty of recognizing cases early before the onset of paralysis and the difficulty, perhaps impossibility, of recognizing abortive forms.

PROGNOSIS.

The mortality from epidemic poliomyelitis varies greatly in different epidemics, as shown by the following figures, collected from several sources. Only cases showing *paralysis* are included in these figures.

Reported by—	Place.	Year.	Total cases.	Deaths.	Percentage mortality.
Caverly	Connecticut	1894	126	18	14
Wickman	Sweden (general)	1905	868	145	16.7
Do	Trastena, Sweden	1905	26	11	42.3
Do	Atudaberg, Sweden	1905	41	4	10
Do	Smedjeback, Sweden	1906	22	5	22.7
Committee of Investigation	New York	1907	[1] 2,000	[1] 100	[1] 5
Hill	Minnesota	1909	283	68	24
Lovett	Massachusetts	1909	628	51	8

[1] Estimated.

The mortality also shows variations according to the age of the persons affected; Wickman gives the mortality at different ages in 842 cases as follows: 0–11 years, 592 cases, 71 deaths, =12.2 per cent; 12–32 years, 250 cases, 69 deaths, =27.9 per cent.

The Massachusetts State Board of Health found the mortality in 628 cases, in 1909, to be as follows: Less than 1 year, 16 per cent; 1–10 years, 4 per cent; over 10 years, 20 per cent.

These figures agree in showing that the disease is relatively more fatal in older persons than in young children.

According to Wickman's statistics, death occurs most frequently within the first week of illness. Paralysis of respiration is the most frequent cause of death. Broncho-pneumonia, secondary to partial respiratory paralysis, may result fatally. The severity of the early symptoms bears no constant relation to the extent of supervening paralysis. Extensive paralysis, progressing after the first 24 hours, is usually a serious prognostic sign.

The chance of complete recovery—restoration of paralyzed parts to their normal function—is greater than has been commonly supposed. In Massachusetts in 1909, 62 of the 628 paralyzed cases (=10 per cent) were reported as recovered within a year. Out of 150 cases closely studied, 25 (=16.7 per cent) were found completely recovered after 3 days to 12 weeks. The extent of paralysis in these cases is given as follows: One thigh and leg, 4; both thighs and legs, 8; both thighs, 1; one leg, 2; one arm, 1; one leg, arm, and back, 1; one leg and back, 1; one thigh, leg, arm, and forearm, 1; one arm, forearm, and cervical region, 1; cervical region, 4; indefinite staggering gait, 1. It is shown by the above that some of these cases had been quite extensively paralyzed.

Complete recovery was recorded in 40 out of 754 cases (=5.3 per cent) investigated in the New York epidemic of 1907; and "almost complete disappearance of paralysis" in 13 (=1.8 per cent), giving a total of 53 cases (=7.1 per cent) making a practically complete recovery.

Hill[1] states that 15 per cent of 283 cases occurring in Minnesota during 1909 completely recovered within a year. Since 24 per cent of these 283 cases died, the 15 per cent of recoveries is equivalent to 19.2 per cent of recoveries among those who survived.

NOTE.—Since this paper went to press, Osgood and Lucas (Journ. Am. Med. Assn., Feb. 18, 1911, vol. 56, p. 495) have reported experiments demonstrating that the nasal mucous membrane of two monkeys, experimentally inoculated with poliomyelitis, remained infectious for six weeks and five and one-half months, respectively. This very important observation strengthens the suspicion of the existence of human "carriers," who may play an important part in the epidemiology of poliomyelitis. It raises still further doubts as to the efficiency of prophylactic measures directed only to persons in the acute stage.

[1] Hill, H. W.: Epidemiological Study of Anterior Poliomyelitis in Minnesota. Trans. Section on Preventive Medicine, Amer. Med. Assn., 1910.

REFERENCES.

The references here cited do not constitute a complete bibliography of the subject. Only such articles are cited as are considered most useful in presenting a comprehensive view of the subject and most likely to be generally available. Articles marked (*) are of special value and articles marked (†) have extensive bibliographies appended. Many articles highly important from a historical point of view have been purposely omitted.

GENERAL.

* Epidemic poliomyelitis. Report of the Collective Investigation Committee on the New York Epidemic of 1907. Nervous and mental disease monograph series, No. 6. New York, 1910.

I. Epidemiology. Bolduan, Ager, Terriberry.

II. Conditions preceding onset and the early symptoms of the disease. LaFetra and Schwartz.

III. Symptomatology. Clark, Hunt, Jelliffe, Sachs, Zabriskie.

IV. Pathology and pathological anatomy. Flexner, Lewis, and Strauss.

V. Treatment. LaFetra, Jelliffe, Ager, Taylor, and Sachs.

Illinois State Board of Health Bulletin, Sept., 1910, vol. 6, no. 9. Reprint of a number of articles from Pediatrics of August, 1910.

MÜLLER, EDUARD. Die spinale Kinderlähmung. Berlin, 1910.

* Pediatrics, August, 1910, vol. 22, No. 8. Special poliomyelitis number. Etiology, epidemiology, pathology, symptomatology, treatment. Articles by C. A. Anderson, J. M. Armstrong, F. E. Coulter, Simon Flexner, J. S. Fowler, Maximilian Herzog, Colin K. Russell, George P. Shidler, I. Strauss, editorial and reviews.

* WICKMAN, IVAR. Beiträge zur Kenntniss der Heine-Medinschen Krankheit. Berlin, 1907, S. Karger. 8°. 300 p. 7 charts. Symptomatology and epidemiology.

——— Ueber die akute Poliomyelitis und verwandte Erkrankungen (Heine-Medinsche Krankheit). Jahrb. f. Kinderh., Berlin, 1909, vol. 67, p. 182–196. Epidemiology, symptoms.

EXPERIMENTAL POLIOMYELITIS.

* FLEXNER, SIMON. The contribution of experimental to human poliomyelitis. Jour. Am. Med. Assn., vol. 55, Sept. 24, 1910, p. 1105–1113.

KRAUSE P., and MEINICKE, E. Zur Aetiologie der akuten epidemischen Kinderlähmung. Deut. med. Woch., vol. 35, Oct. 21, 1910, no. 42, p. 1825–1827.

LEVADITI, C. L'étude expérimentale de la poliomyélite aiguë. Presse méd., vol. 18, 1910, p. 297–299.

LEVADITI, C. Le virus de la poliomyélite aiguë. Presse méd., vol. 18, 1910, p. 378–380.

* LANDSTEINER, K., and LEVADITI, C. Étude expérimentale de la poliomyélite aiguë. Ann. de l'Inst. Pasteur, vol. 24, no. 11, Nov. 25, 1910, p. 833–876.

EPIDEMIOLOGY.

* CAVERLY, C. S. Notes of an epidemic of acute anterior poliomyelitis. Journ. Am. Med. Assn., vol. 26, 1896, p. 1–5. Med. Record, vol. 46, 1894, p. 673.

EMERSON, HERBERT C. An epidemic of infantile paralysis in Western Massachusetts in 1908. Rep. Mass. State Board of Health, 1908, p. 764. Abst. in Boston Med. & Surg. Journ., July 22, 1909.

FROST, W. H. The field investigation of epidemic poliomyelitis. Pub. Health Reports, vol. 25, no. 46, Nov. 18, 1910.

HARBITZ, F., and SCHEEL, O. Epidemic acute poliomyelitis in Norway and Sweden in the years 1903 to 1906. Journ. Am. Med. Assn., vol. 49, 1907, p. 1420–1425.

HILL, H. W. The contagiousness of poliomyelitis. Journ. Minn. Med. Assn., March 15, 1910.

* ———. Epidemiologic study of anterior poliomyelitis in Minnesota. Trans. Sect. Prev. Med. of Am. Med. Assn., 1910.

* † HOLT, L. E., and BARTLETT, F. H. The epidemiology of acute poliomyelitis. Am. Jour. Med. Sci., Phila., vol. 135, 1908, p. 647–662.

JOB and FROMENT. Le poliomyélite aigue. Étude epidemiologique. Rev. de méd., vol. 30, 1910, p. 162, 378, 481, 651.

KRAUSE, P. [Ueber die westfälische Epidemic von Kinderlähmung]. Deutsch. med. Woch., vol. 35, 1909, pp. 1822–1825.

LOVETT, R. W. The occurrence of infantile paralysis in Massachusetts in 1907 [with special reference to etiology]. Boston Med. & Surg. Journ., vol. 159, 1908, p. 131–139.

———. A study of poliomyelitis in Massachusetts in 1907 and 1908. Journ. Nerv. & Ment. Dis., vol. 36, 1909, p. 681–684.

———. The occurrence of infantile paralysis in Massachusetts in 1908. Boston Med. & Surg. Journ., July 22, 1909. Rep. Mass. State Board of Health, 1908, p. 756.

* † ———. The occurrence of infantile paralysis in Massachusetts in 1909. Boston Med. & Surg. Journ., vol. 163, 1910, p. 37–55.

———. Infantile Paralysis. Am. Journ. Pub. Hygiene, vol. 20, Nov., 1910, p. 875–887.

* MÜLLER, A. Eine epidemisch auftretende Erkrankung des Nervensystems auf Nauru. Arch. f. Schiffs.-u. Tropen-Hyg., vol. 14, Sept. 1, 1910.

NETTER, A. Fréquence insolite des poliomyélites en France pendant l'été dernier; leur identité avec les cas observés à l'étranger sous forme épidémique; relations entre la poliomyélite et la méningite cérébro-spinale épidémique. Bull. et mém. soc. méd. de hôp. de Par., 1909, 3 s., vol. 28, p. 515–528. Ibid., p. 553. [A new case], ibid., p. 629. Ibid., p. 746–761.

———. Apparition sous forme épidémique de la paralysis infantile à Paris et sa banlieue en 1909; Notions fournies par l'étude des épidémies des autres pays et par la pathologie expérimentale. Bull. Acad. de Med., vol. 63, 3 ser., 1910, p. 458–481.

SHIDLER, GEORGE P. The epidemic of spinal disease in Nebraska. Journ. Am. Med. Assn., vol. 54, 1910, p. 277–281.

* † STARR, M. ALLEN. Epidemic infantile paralysis. Journ. Am. Med. Assn., vol. 51, 1908, p. 112–120.

PATHOLOGY.

* HARBITZ, F., and SCHEEL, O. Pathologische-anatomische Untersuchungen über akute Poliomyelitis und verwandte Erkrankungen. Christiania, A. W. Broggers, 1907.

HOUGH, WILLIAM H., and LAFORA, G. The cerebrospinal fluid in acute anterior poliomyelitis. N. Y. Med. Journ., vol. 92, no. 19, Nov. 5, 1910, p. 924.

LEWIS, PAUL A. The pathology of experimental anterior poliomyelitis in the monkey. Proc. N. Y. Path. Soc., vol. 10, n. s., 1910, p. 47.

ROBERTSON, H. E., and CHESLEY, A. J. Pathology and bacteriology of acute anterior poliomyelitis. Journ. Am. Med. Assn., vol. 55, Sept. 17, 1910, p. 1013.

STRAUSS, I. Studies on the pathology of poliomyelitis anterior acute based on the autopsy findings in five cases. Journ. Nerv. & Ment. Dis., vol. 36, 1909, p. 288.

*† WICKMAN, IVAR. Studien über poliomyelitis acuta. Arb. a. d. pathologisch. Inst. der Univ. Helsingfors, 1905–07. Berlin, 1907, S. Karger, p. 109–292.

——— Weitere Studien über Poliomyelitis acuta; ein Beitrag zur Kenntniss der Neuronphagen und Körnchenzellen. Deut. Zeitschr. f. Nervenh., Leipz., vol. 38, 1910, p. 396–436. 6 pl.

SYMPTOMS.

BUZZARD, E. F. Acute poliomyelitis. Mod. Med. (Osler), vol. 7, 1910, p. 258–268.

DAKIN, C. E. Acute epidemic poliomyelitis; symptomatology, diagnosis, treatment. Iowa Med. Journ., vol. 17, no. 5, Nov., 1910, p. 229–236.

FOERSTER, O. Zur Symptomatologie der Poliomyelitis anterior acuta. Berl. klin. Woch., vol. 46, 1909, p. 2180–2184.

HAMILTON, A. S. Epidemic anterior poliomyelitis. Journ. Minn. Med. Assn., vol. 29, 1909, p. 423–428.

KRAUSE, P. Zur Kenntniss der westfälischen Epidemie von akuter Kinderlähmung. Deut. med. Woch., vol. 35, 1909, p. 1822–1825.

LAFETRA, L. E. Early symptoms in 63 cases of the recent epidemic of anterior poliomyelitis. Arch. Pediat., N. Y., vol. 26, 1909, p. 328–340.

LUCAS, W. P. The diagnosis of infantile paralysis in the prodromal and early acute stage, as found in the experimental study of acute poliomyelitis in monkeys. Boston Med. & Surg. Journ., 1910, vol. 163, pp. 245–249.

MEDIN, O. Arch. des Medicin d'Infants, vol. 1, p. 259, 321.

——— Verhandl. d. X internat. Kong. Berlin, II Abt. VI, 1891.

MÜLLER, E. Ueber die Frühstadien der spinalen Kinderlähmung. Münch. med. Woch., vol. 56, p. 2460–2462.

VOGT, H. Poliomyelitis und Encephalitis; die Encephalitis der Kinder. Jaresb. f. ärztl. Fortbild., Münch., 1910, no. 5, p. 19–39.

TREATMENT.

*BRADFORD, E. H., et al. Methods of treatment in infantile paralysis; summarized by the department of orthopedic surgery of the Harvard Medical School. Boston Med. & Surg. Journal, vol. 162, 1910, p. 881–893.

LOVETT, R. W., and LUCAS, W. P. Infantile paralysis; a study of 635 cases from the Children's Hospital, Boston, with especial reference to treatment. [Abst.] Journ. Am. Med. Assn., vol. 51, 1908, p. 1677–1684.

MCCLANAHAN, H. M., SACHS, B., and RIDLON, JOHN. Symposium on the treatment of acute anterior poliomyelitis. Journ. Am. Med. Assn., vol. 55, Oct. 22, 1910, p. 1464–1468.

SILVER, DAVID. The surgical treatment of infantile spinal paralysis. Journ. Am. Med. Assn., vol 55, Sept. 17, 1910, p. 1014–1021.

SKOOG, ANDREW L. Treatment of acute anterior poliomyelitis. Journ. Am. Med. Assn., vol. 55, Nov. 19, 1910, p. 1804.

LIST OF PUBLIC HEALTH BULLETINS.

[Those marked (*) are out of print. Those marked (†) can be purchased from the Superintendent of Documents, Government Printing Office, Washington, D. C., at the cost marked after each. Copies of those still available for distribution may be had by request from the Surgeon General, Public Health and Marine-Hospital Service, Washington, D. C.]

*1. Report on Trichinæ and Trichinosis. By W. C. W. Glazier. 1881. 212 pages. 87 il. 1 map. Paper. Senate Executive Document No. 9, Forty-sixth Congress, third session. Out of print.

*2. Report of the Etiology and Prevention of Yellow Fever. By George M. Sternberg. 1890. 271 pages. 21 pl. 20 il. Cloth. Out of print.

*3. Mortality Statistics in the United States for the year ending December 31, 1897. From Annual Report Marine-Hospital Service, 1898. 24 pages. Paper. Out of print.

4. Yellow Fever: Its Nature, Diagnosis, Treatment, and Prophylaxis and Quarantine Regulations Relating Thereto. By officers of the Marine-Hospital Service. Reprint from Annual Report Marine-Hospital Service, 1898. 176 pages. 1 il. Paper.

*5. Shipment of Merchandise from a Town Infected with Yellow Fever. By H. R. Carter. 1899. 15 pages. Paper. Out of print.

†6. Report of Commission of Medical Officers Detailed by Authority of the President to Investigate the Cause of Yellow Fever. By Eugene Wasdin and H. D. Geddings. July, 1899. 98 pages. 26 charts. 2 il. Paper. 5 cents.

*7. The Bubonic Plague. By Walter Wyman. January, 1900. 50 pages. Paper. Superintendent of Documents. 5 cents.

†8. Report of Commission Appointed by the Secretary of the Treasury for the Investigation of Plague in San Francisco. By Prof. Simon Flexner, Prof. F. G. Novy, and Prof. L. F. Barker. January 23, 1901. 23 pages. 1 map. Paper. 5 cents.

*9. Report Relating to the Origin and Prevalence of Leprosy in the United States. By a Commission of Medical Officers of the United States Marine-Hospital Service. 1902. 119 pages. 25 il. Paper. Senate Document No. 269, Fifty-seventh Congress, first session. Superintendent of Documents. Cloth. $1.00.

*10. Plague Conference. Containing a copy of the address of the chairman, and resolutions passed by a conference called in accordance with requests from a number of State Boards of Health, and under authority of section 7, act of Congress approved July 1, 1902, to consider the plague situation. Reprint from P. H. R. No. 4, Vol. XVIII, January 23, 1903. 9 pages. And February 6, 1903. 41 pages. Paper. Out of print.

†11. Transactions of the First Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. June, 1903. 120 pages. Cloth. 20 cents.

†12. Transactions of the Second Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. June, 1904. 95 pages. Cloth. 20 cents.

†13. The Public Health and Marine-Hospital Service of the United States. A Brief History. Prepared for the Louisiana Purchase Exposition. December, 1904. 16 pages. Paper. 5 cents.

†14. Sanatorium for Consumptives, Fort Stanton, N. Mex. By P. M. Carrington. Reprint from Annual Report Public Health and Marine-Hospital Service, 1904. 19 pages. Paper. 5 cents.

†15. Transactions of the Third Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. May, 1905. 52 pages. Cloth. 30 cents.

16. How to Prevent Yellow Fever—No Mosquitoes, No Yellow Fever. By Walter Wyman. July 31, 1905. 3 pages. Circular.

†17. Transactions of the Fourth Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. May, 1906. 75 pages. Cloth. 35 cents.

†18. Transactions of the Fifth Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. May, 1907. 47 pages. Cloth. 25 cents.

19. Trachoma, Its Character and Effects. By Taliaferro Clark and J. W. Schereschewsky. 1907. 34 pages. 6 il. Paper. (Second edition.)

*20. The Public Health and Marine-Hospital Service of the United States. A Brief History. Prepared for the Jamestown Ter-Centennial Exposition, 1907. 12 pages. Paper. Out of print.

†21. Transactions of the Sixth Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. April, 1908. 79 pages. Cloth. 50 cents.

†22. The Present Pandemic of Plague. By J. M. Eager. 1908. 30 pages. Paper. 5 cents.

*23. Pellagra—A Précis. By C. H. Lavinder. July 24, 1908. 22 pages. 1 il. Paper. Revised. See Public Health Bulletin no. 48.

24. The Marine-Hospital Sanatorium, Fort Stanton, N. Mex. Prepared for the International Congress on Tuberculosis, held in Washington, September, 1908. 32 il. 56 pages. Paper.

*25. Hookworm Disease. Reprint from Annual Report P. H. and M. H. S., 1908. 5 pages. Paper. Out of print.

†26. Studies upon Leprosy.

I. The Present Status of the Leprosy Problem in Hawaii.

II. The Reaction of Lepers to Moro's "Percutaneous" Test.

III. A Note upon the Possibility of the Mosquito Acting in the Transmission of Leprosy. By W. R. Brinckerhoff. 1908. Investigations made in accordance with the act of Congress approved March 3, 1905. 25 pages. Paper. 10 cents.

27. Studies upon Leprosy.

IV. Upon the Utility of the Examination of the Nose and the Nasal Secretions for the Detection of Incipient Cases of Leprosy. By W. R. Brinckerhoff and W. L. Moore. 1909. Investigations made in accordance with the act of Congress approved March 3, 1905. 29 pages. Paper.

28. Studies upon Leprosy.

V. A Report upon the Treatment of Six Cases of Leprosy with Nastine (Deycke). By W. R. Brinckerhoff and J. T. Wayson, Honolulu, T. H.

VI. Leprosy in the United States of America in 1909. By W. R. Brinckerhoff. 1909. Investigations made in accordance with the act of Congress approved March 3, 1905. 25 pages. Paper.

†29. The Prevalence of Rabies in the United States. By J. W. Kerr and A. M. Stimson. 1909. 16 pages. Paper. 5 cents.

†30. The Rat and Its Relation to the Public Health. By various authors. 1910. 254. pages. 60 figs. 6 pl. Paper. 50 cents.

1. Introduction. By Walter Wyman.
2. Natural History of the Rat. By D. E. Lantz.
3. Plague Infection in Rats. By G. W. McCoy.
4. Rat Leprosy. By W. R. Brinckerhoff.
5. Bacterial Diseases of the Rat other than Plague. By D. H. Currie.
6. Organic Diseases of the Rat. By G. W. McCoy.
7. Ecto Parasites of the Rat. By N. Banks.
8. Intestinal Parasites of Rats and Mice in their Relation to Diseases of Man. By C. W. Stiles.
9. Rodents in Relation to the Transmission of Bubonic Plague. By Rupert Blue.
10. Rodent Extermination. Rats and Mice. By W. C. Rucker.
11. Natural Enemies of Rats. By D. E. Lantz.
12. Rat-Proofing as an Antiplague Measure. By R. H. Creel.
13. Inefficiency of Bacterial Viruses in the Extermination of Rats. By M. J. Rosenau.
14. Plague Eradication in Cities by Sectional Extermination of Rats and General Rat-Proofing. By Victor G. Heiser.
15. The Rat in Relation to Shipping. By W. C. Hobdy.
16. The Rat as an Economic Factor. By D. E. Lantz.
17. The Rat in Relation to International Sanitation. By J. W. Kerr.

†31. Transactions of the Seventh Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. June, 1909. 86 pages. Cloth. 35 cents.

32. Hookworm Disease (or Ground-Itch Anemia), its Nature, Treatment, and Prevention. By Prof. C. W. Stiles. 1910. 40 pages. Paper. (Second edition.)

33. Studies upon Leprosy. 1910. 25 pages. Paper.

VII. A Statistical Study of an Endemic Focus of Leprosy. By W. R. Brinckerhoff and A. C. Reinecke.

VIII. A Palliative Treatment for Leprous Rhinitis. By J. T. Wayson and A. C. Reinecke.

34. Maritime Quarantine. By L. E. Cofer. 1910. 25 figs. 64 pages. Paper. Appendix: Disinfectants Authorized by United States Quarantine Regulations and the Proper Method of Generating and Using Same.

35. The Relation of Climate to the Treatment of Pulmonary Tuberculosis. By F. C. Smith. 1910. 17 pages. Paper. (Second edition.)

36. Tuberculosis: Its Nature and Prevention. By F. C. Smith. 1910. 12 pages. 1 pl. Paper. (Second edition.)

37. The Sanitary Privy: Its Purpose and Construction. By Prof. C. W. Stiles. 1910. 24 pages. 12 figs. Paper. (Second edition.)

38. General Observations on the Bionomics of the Rodent and Human Fleas. By M. B. Mitzmain. 1910. 34 pages. Paper.

39. Studies upon Leprosy. September, 1910. 50 pages. Paper.

IX. Mosquitoes in Relation to the Transmission of Leprosy.

X. Flies in Relation to the Transmission of Leprosy. By D. H. Currie.

XI. Heredity Versus Environment in Leprosy. By H. T. Hollmann.

†40. Transactions of the Eighth Annual Conference of State and Territorial Health Officers with the United States Public Health and Marine-Hospital Service. November, 1910. 101 pages. Paper. 10 cents.

41. Studies upon Leprosy. November, 1910. 36 pages. Paper.
    XII. Notes on the Study of Histories of Lepers from the Standpoint of Transmission. By D. H. Curie.
    XIII. A Contribution to the Study of Rat Leprosy. By D. H. Currie and H. T. Hollmann.
42. Disinfectants: Their Use and Application in the Prevention of Communicable Diseases. By T. B. McClintic. December, 1910. 46 pages. Paper. (Second edition.)
43. Experimental Studies of Plague and a Plague-like Disease among Rodents. By George W. McCoy. April, 1911. 71 pages. 7 pl. Paper.
44. Acute Anterior Poliomyelitis (Infantile Paralysis). By Wade H. Frost. February, 1911. 52 pages. Paper. (Second edition.)
45. A Digest of the Laws and Regulations of the Various States relative to the Reporting of Cases of Sickness. By J. W. Trask.
46. Transactions of the Ninth Annual Conference of State and Territorial Health Officers with the Public Health and Marine-Hospital Service. September, 1911.
†47. Studies upon Leprosy. By D. H. Currie, M. T. Clegg, and H. T. Hollmann. September, 1911. 10 cents.
    XIV. The Artificial Cultivation of the Bacillus of Leprosy.
    XV. Attempts at Specific Therapy in Leprosy.
48. Pellagra. A Précis. (Revised edition.) By C. H. Lavinder. September, 1911. (Second edition.)

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