trial lead-poisoning in this country in comparison with countries where there is proper legislative regulation of the dangerous trades. This contrast was brought vividly home to me by a description which I found in T. Weyl's "Handbuch der Arbeiter Krankheiten." He is drawing what he considers a shocking picture of lead-poisoning as it used to be found years ago. He says:

Lead tabes is the severest form of lead cachexia, now almost never seen, thanks to prophylactic measures. Here we find more or less all the phenomena of plumbism in one individual. Pallor like that of chlorosis, a predominance of the flexors over the extensors, as shown in the hanging head, the bowed shoulder, the hands held in pronation; muscular tremors, causing a shambling gait and trembling movements of all the muscles of the body; disappearance of all possible fat; general emaciation.

From my studies so far I can unfortunately testify to the fact that, thanks to the lack of prophylactic measures, Weyl's lead tabes is far from being a rare condition in our country; that instances of it can be found in every town where there are lead industries of a dangerous character, and that it is not even a vanishing condition, for new instances of lead tabes are being added to the number every year.

Surely there is every reason why we should devote to this disease the same intelligence and energy that we devote to other preventable diseases. There are many problems waiting solution. We need research into the question of sex susceptibility, of race susceptibility, of the influence of summer heat, of the influence of muscular fatigue; we need, above all, research into the different lead trades of our country and the different forms of lead-poisoning that they produce, the localization of palsies, the frequency of encephalopathies. As it is now, it would be hard to point to any disease equally prevalent, equally serious, equally controllable, which has been so neglected by the medical profession in America as industrial lead-poisoning.

EPIDEMIC POLIOMYELITIS IN NORWAY

ITS ETIOLOGY AND THE POSSIBILITIES OF ITS PREVENTION

FRANCIS HARBITZ, M.D.

Professor of Pathology in the University of Christiania
CHRISTIANIA, NORWAY

I. HISTORY

Acute poliomyelitis has been recognized in Norway as a distinct disease ever since 1820. Cases were exhibited in the Medical Society of Christiania in 1842 and in 1853. In 1863, discussing the case from 1853, Voss pointed out that, judging from the resulting paralysis, the disease must have its seat in the spinal cord or in the anterior nerve-roots.

The first epidemic of acute poliomyelitis broke out in 1868 in Dr. A. Christian Bull's district and was reported by him to the medical board of the state. This epidemic lasted from June to August. Fourteen persons were stricken, of whom twelve were children, with five deaths. The symptoms were so characteristic that any doubt of the nature of the disease can be excluded. Bull also reported two abortive cases—those of two children who were attacked by brief fever and indistinct paresis. The possibility of a contagious virus as the cause of the disease was discussed.

In 1886, Dr. Oxholm described an epidemic in his district; it also prevailed during the months of July and August. Nine persons were attacked. In the

eighties several new cases were reported to the state medical board; these cases were regarded partly as cerebrospinal meningitis. In 1898, Bülow-Hanson and Harbitz¹ reported three cases of acute poliomyelitis; two of the patients died, while the third, who had the abortive form, recovered. The anatomic study gave the now well-known result: acute, diffuse infiltration of the spinal cord and its membranes and of the medulla and pons. Based on the anatomic conditions, the theory was advanced that acute poliomyelitis was an acute infectious disease and that the infection entered the system through the alimentary tract and throat.

In 1889, another epidemic broke out in a country district (Bratsberg) during the months of July to October. There were fifty-four cases, mostly in children. This epidemic was thoroughly studied and described by C. B. Leegaard² who pointed out that the epidemic spread along the main roads of communication, branching out to the sides with small groups of cases. The invasion of the infection was supposed to occur through the alimentary tract, and the disease was pronounced contagious with a short period of incubation, hardly more than twenty-four hours. Leegaard proposed that the disease be subjected to public control and be made reportable; he also proposed isolation for three weeks and subsequent disinfection. Professor Johannesen,³ in 1911, published a synopsis of twenty-three cases of infirmities resulting from acute poliomyelitis, observed at the pediatric clinic during the years 1893-1899.

In 1903 and 1904, new, small epidemics occurred but, in 1905 and 1906, the disease broke out simultaneously in different and large tracts of the country. In 1905, 952 cases were reported of which 358 were of the abortive type.

The state physician for epidemic diseases, Dr. Geirsvold, gave a preliminary report of the epidemic of 1905; he maintained that acute poliomyelitis was a specific infectious and contagious disease. He further pointed out that great stress should be laid on the abortive cases and that the disease often was disseminated through the medium of healthy persons. The disease left immunity; stricken districts were the next year exempt from its ravages. It would spread from place to place, in the beginning often with influenza-like symptoms, later on developing into characteristic poliomyelitis, now and then accompanied by meningitis, abortive forms, Landry's paralysis, etc.

Geirsvold also called attention to the fact that exposure to cold and damp weather with subsequent catarrhal conditions or physical overexertion would predispose to the disease. He pronounced acute poliomyelitis infectious, with its point of invasion as a rule through the throat; the time of incubation, three to five days. At the time of the epidemics he had not observed similar diseases among the domestic or other animals. This short article by Dr. Geirsvold on the epidemic of 1905 contains in brief the main features of what has been observed during the different subsequent epidemics. In 1906, there were 466 cases with fifty deaths.

Francis Harbitz and Olaf Scheel,⁴ in 1907, presented a review of the epidemic in Norway in the years 1903-1906, inclusive. They communicated the results of the

1. Bülow-Hanson and Harbitz: *Beitr. z. pathol. Anat. u. allgem. Pathol.* (Ziegler's), 1899, xxvi, 517.

2. Leegaard, C. B.: *Epidemisk poliomyelitis; Bakteriologiske undersøgelser*, Norsk Mag. f. Lægevidensk., 1901, lxi, 377.

3. "Festschrift" in Honor of Abraham Jacob, 1900.

4. Harbitz and Scheel: *Epidemic Acute Poliomyelitis in Norway in the Years 1903-1906*, *THE JOURNAL A. M. A.*, Oct. 20, 1907, p. 1420.

careful anatomic examinations of nineteen cases. Of the patients, thirteen, most of them children, died during the acute stage of the disease. Harbitz and Scheel demonstrated a diffuse inflammation of the spinal pia-arachnoid, of the gray matter of the spinal cord and the medulla oblongata and the surface of the brain. They again pronounced the disease to be specific, infectious and contagious, and entirely different from epidemic cerebrospinal meningitis, because it occurs at a different season, and because the virus occurs in the cerebrospinal fluid and membranes whence the cord and medulla probably are infected; further, that the infection enters the system through the digestive apparatus (the nose and alimentary tract) and that the central nervous system is infected either by way of the lymph-vessels, along the nerve-tracts, or still more plausibly through the blood. Cases of neuritis or transverse myelitis were not observed in connection with the examinations.

Leegaard, in a more extensive work,⁵ has thoroughly described the epidemic of 1905. Here also the majority of patients were children; 50 per cent. of the cases occurred in August and September, 25 per cent. in July and October, and the remainder were distributed over the other months. In the genuine poliomyelitis cases 14.5 per cent. of the patients died and 58.5 per cent. became invalids. The disease this time also was disseminated along the main road of communication, along the coast by way of steamers, in through the country by the railroads. The disease was contagious, especially during the first three days but also the following three weeks. The time of incubation was found to be from two to three days as a rule. The contagion was carried from person to person and also through the intermediary of healthy persons (infection carriers) and its mode of entrance was found as a rule to be through the nose and throat.

During the four years following, the disease was on the retreat: in 1907, 204 cases with 15.2 per cent. mortality, in 1908, fifty-nine; in 1909, fifty-one, and in 1910, thirty-two cases. Real epidemics did not occur; only sporadic cases or small endemics. In 1911, however, the disease broke out with about 1,250 cases with about 10 per cent. mortality. Most cases occurred in July, August and September.⁶ The disease this time also was characterized by its wide dissemination, apparently without connecting links. As a rule, though, districts which previously had been ravaged were now comparatively exempted. The majority of cases were also now found in the rural districts, but several cases occurred in the larger cities — in Christiania there were sixty-two cases with twelve deaths. During the first months of 1912 sporadic cases of acute poliomyelitis — 117 cases with five deaths — occurred. The same year an extensive epidemic of several hundred cases of cerebrospinal meningitis infested Christiania and neighboring districts.

II. GENERAL EPIDEMIOLOGY

From the foregoing history it will be evident that acute poliomyelitis has been prevalent in Norway for almost a century, further, that small epidemics have been observed and described for about forty-five years. Consequently, we must assume that its virus has been constantly present in our country and if not active in the form of smaller or larger epidemics at least latent

in many and different districts which again under favorable circumstances have been the starting-points for dissemination of the disease. Only during the last twenty or twenty-five years have the Norwegian physicians collectively given much attention to this scourge and considered it to be of an infectious nature. This was first pointed out by Bull in 1868, openly pronounced by Leegaard in 1889 and strongly advocated by Bülow-Hanson and Harbitz in 1898, culminating in the State Board of Health circulars of 1904 and 1905 with a view of checking or preventing the occurrence of the disease.

The conclusions of the Norwegian physicians in regard to acute poliomyelitis are in the main that epidemic poliomyelitis is an acute infectious disease which, as indicated by several observations, must also be considered contagious.

The disease as a rule has started in widely separated smaller epidemics; somehow in an explosive manner in one or more places simultaneously, and without any apparent connection. It has been observed regularly that cases of genuine poliomyelitis (with paralysis), Landry's paralysis with subsequent deaths and abortive cases have occurred simultaneously and in the same localities, but no cases of neuritis, transverse myelitis or encephalitis.

The abortive cases were characterized by a transient fever, pains in the extremities, back and neck, nausea and vomiting, increased reflexes, a train of symptoms which often were mistaken for those of a heavy cold or "influenza," and so much more easily, as the symptoms quite strongly reminded one of the first severe epidemics of influenza or morbilli in previously uninfected districts.

For the contagiousness of the disease there are different theories. Even where the disease was greatly disseminated and single cases occurred simultaneously it could be determined by careful investigation that the disease had spread along the lines of communication, along the highways, railroads and steamship-routes, along the coast and into the fjords by smaller vessels from one place to another, but with different grades of velocity. This would be particularly manifest in sparsely inhabited districts with poor communications. Several reports pointed to small epidemics having started in schools, country stores, dairies, public meetings, weddings, funerals, or they could be traced to gypsies and other wanderers. These obstructions strengthened the theory of a direct contagion. In the study of virulent house-epidemics it was also found that one member of the family after the other became infected with a few days' intermission, and that they would exhibit the disease in its different forms. As an example the following may serve:

A physician treated some sporadic cases in his district in the latter part of June and the beginning of July. July 15, he himself became ill with fever, pains in the back, great exhaustion, diarrhea and vomiting. He recovered in three days. July 16, his son, aged 3, was seized with the same symptoms. July 18, his son, aged 5, was stricken in the same manner. He also recovered in three days. July 22, his oldest son was taken with well-defined gastro-intestinal symptoms, stiffness of the neck and paresis of the extremities and facialis. He recovered in eight days without permanent paralysis. The following days his 1-year-old son, the nursemaid, his wife and her sister were taken sick with acute angina, stiffness of the neck and fever; and finally a neighbor's child, who had been a playmate of the physician's children, was stricken with symptoms of a genuine poliomyelitis and with subsequent permanent paralysis.

5. Leegaard, C. B.: Videnskabselsk. Skr. I. Mathem-Naturvid, Kl., 1908, No. 11, Kliniske og epidemiologiske Undersøgelser over den acute Poliomyelitt i Norge meddelte til det norske Forekomst i Aaret, 1905.

6. Gram: Tidsskr. f. d. norske Lægefor., 1911, p. 850.

When these cases are studied in connection with each other it seems reasonable to assume that here was a virus which easily and quickly spread from person to person.

As an argument against the contagiousness of poliomyelitis it has been advanced that nurses who take care of these patients escape the disease. This argument does not hold good; in a separate pavilion of the hospital for epidemic diseases at Christiania were several poliomyelitis patients during July, August and September, 1911. Two of their nurses, aged 28 and 29, became ill in the latter part of August and died with acute poliomyelitis. They had served in this hospital-division for about one month. The virus must, however, have been quite widely disseminated in this hospital, because in August there occurred among the children in the diphtheria pavilion four cases of acute poliomyelitis of which three were in the same ward. In the scarlet-fever pavilion one case of acute poliomyelitis occurred in a child who previously had been taken care of by one of the two nurses who died from poliomyelitis, and finally a nurse who served in one of the medical sections of the same hospital took the disease and died. In this last case it was impossible to trace the source of infection. These cases which occurred almost simultaneously can be explained only by the existence of a direct infection and wide dissemination of the virus among adults and children.

The time of incubation, according to Norwegian experience, has been quite short—two, three or four days, but at times longer. Cases were observed in which a latent condition of the virus must be assumed. An example of this is the case of a young woman who had been in solitary confinement for three months and had contracted acute poliomyelitis. Hers was the only case inside the prison, and it is reasonable to conclude that she came to the institution already infected.

The infection is as a rule carried from one person to another. Of great interest are the abortive cases, which have been found to be just as contagious as the cases of well developed poliomyelitis. It must be supposed also that the infection can be spread through the intermediary of healthy bacillus carriers who have had a latent infection not infrequently for longer periods. Through this assumption is explained the extended time of incubation sometimes noted.

According to our experience, the mode of entrance must be the throat, nose and alimentary tract.

The degree of infectiousness as a rule may be designated as not so great. This is proved by the fact that only one case might occur in one household or among a large family of children, several of these even sharing beds; further, by the fact that only sporadic cases would occur in a well-populated country district or even in a large city. But here again we must take into consideration the fact that latent infection and lighter abortive cases have been much more widely spread; this has been proved by the fact that a district infected with sporadic cases or small epidemics the next year would be exempt from the disease.

In Norway there is no safe foundation for the belief that foods might be the source of infection nor any support to the theory of a coexistent infection of animals and a conveyance of the infection through them. Simultaneously with the poliomyelitis epidemics a great many deaths have occurred among animals, caused by epidemic diseases. These have been prevalent among chickens, reindeer, dogs and cats. By further examination into these conditions any connection between the

etiologic factors of the diseases of these animals and acute poliomyelitis has been disproved. Experiments made under control have given the same results. During the epidemics of 1911 with its unusually dry summer, masses of flies and other insects (fleas) were in evidence. The possibility of an indirect conveyance of the infection by these insects (see Flexner and Lewis' experiments) may be admitted, but there is no proof or convincing examples of such occurrences.

In the epidemiology of acute poliomyelitis are several unexplained points: (1) its breaking out in great epidemics in certain years without reference to changed climatic conditions; (2) that most cases occur during the summer and fall and only sporadic cases during winter and spring; (3) that the disease is most prevalent in the rural districts, and, finally, (4) that the different epidemics vary in the degree of contagiousness and malignancy in the different years and in different localities.

Such, however, is also the case with other diseases with well-known contagiousness as, for instance, epidemic cerebrospinal meningitis. This disease, which also assumes an epidemic nature, has been known in Norway for a great many years, is found preferably during the cold season and has, according to our experience, clinically and anatomically nothing in common with acute poliomyelitis, and they must be considered as two specific, entirely independent diseases.

III. MEASURES AGAINST AND TREATMENT OF THE DISEASE

Recognizing acute poliomyelitis as an infectious, contagious disease, the chief medical officer of the National Board of Health on May 26, 1904, sent to all physicians of our country a circular directing that acute poliomyelitis should be classed with the epidemic diseases subject to public control. In a circular of May 28, the same year, he further specified the measures to be taken:

1. Immediate notification to the board of every case, abortive cases included, with a short history of the disease and its etiology.

2. Isolation of the sick during the acute stage for three weeks.

3. Careful disposition of the patient's excretions and secretions, particularly from the nose and throat. The patient and everybody who came in contact with him were recommended to use gargles at regular intervals, consisting of solutions of potassium permanganate or hydrogen peroxid. The sick should be furnished with separate dishes, handkerchiefs, pillowcases, etc., and anything soiled with secretions or excretions from the patient should immediately be boiled.

4. Disinfection of everything in contact with the patient for a time of three weeks from the beginning of the disease.

5. Particular attention given to the lighter abortive cases and patients with symptoms of angina; also to the possibility of the disease being carried through the intermediary of healthy persons.

6. Attention paid to the possibility of infection through schoolchildren. In much-infected districts it was recommended to close the schools, and healthy as well as sick children from infected homes were prohibited from appearing at school for some time.

These measures received active cooperation from the local health boards of the infected districts. The local board at times took even more drastic measures by prohibiting public meetings and vaccination during the prevalence of an epidemic. As a rule those measures

were undertaken too late when the epidemic was in full outbreak, but reports from districts, especially those more sparsely inhabited, and those where the measures had been early instituted, show that these precautions helped to limit and stop the disease. This again seems to point to the contagious nature of acute poliomyelitis.

The treatment of individual cases has been mainly symptomatic, and possibly not of much avail. Dr. Sinding-Larsen⁷ recommends that treatment begin with laxatives, for instance, calomel followed with urotropin (hexamethylenamin) in doses of 0.30 gm. three times a day, and good results have been given in some cases. Spinal puncture and aspiration of spinal fluid were recommended and have seemed to act favorably by mechanically lowering the cerebrospinal pressure. Prophylaxis in the way of careful frequent antiseptic gargles and sprays of nose and throat have been found useful.

Persons with abortive cases and supposedly infected persons are admonished to exercise particular care in avoiding exposures to cold and overexertion, which experience has taught can excite the disease, with subsequent paralysis. For the resulting pareses and paralysis massage, persisted in if necessary for weeks to years, has seemed to be of great value.

The medical authorities have demanded reports on the frequency and nature of disabilities resulting from the disease with a view of considering how steps can be taken to give these patients massage, surgical or orthopedic treatment at public expense. The preliminary report shows that following the epidemic of 1911 about 400 cases (32 per cent.) of permanent disability were recorded. Measures will now be taken to examine further into the degree of disability and to inaugurate suitable treatment of such cases. The chief medical officer of the State Board of Health on petition has been granted funds for the purpose of: (1) systematic examinations into all the epidemics of late years, and (2) experimental research on apes to secure if possible a more thorough understanding of the modes of infection of acute poliomyelitis.

These investigations are conducted by the bacteriologist and sanitary inspector of the State Board of Health, Dr. Gram; Dr. Aaser, the chief physician of the municipal hospital for infectious diseases, and E. U. Hansteen, the prosecutor. No conclusive results have as yet been reached.

A CONTRIBUTION TO THE ETIOLOGY OF POLIOMYELITIS *

M. NEUSTAEDTER, M.D., PH.D.

Instructor in Neurology, University and Bellevue Hospital Medical College; Attending Neurologist, University and Bellevue Hospital Medical College Clinic; Bellevue Hospital Outpatient Department; St. Mark's Hospital Outpatient Department

NEW YORK

The lesion of poliomyelitis confines itself mainly to the cerebrospinal system and produces focal clinical symptoms. This part of the human organism is rather remote from the external surface of the body and very tightly boxed in, as it were, and well protected from external influences. An infection, therefore, must primarily reach these tissues either through the circulatory or through the lymphatic system.

7. Larsen: Tidsskr. f. d. norske Lægefor., 1911.

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

Experimentally, the disease was produced by injecting monkeys with the infected spinal cord and brain intracerebrally, intraperitoneally, by rubbing it into an abraded surface and an unabraded one of the nasal mucosa, into the nerve-sheath and finally by rubbing it into an abraded surface of the skin. It was further shown that the nasal mucosa contains the virus quite early in the incubation period of the disease—three days after injection of the virus—and that the tonsils, the salivary glands, the retroperitoneal glands and lung tissue are infected as well. There is unmistakable evidence that the pia of the anterior longitudinal fissure of the cord is the primary seat of the lesion, and in my own two cases the pia surrounding the cord contained marked lesions while the vessels and gray matter were intact and the cases were of the abortive type. It is an established fact that, while a very small quantity of an infected-cord emulsion—0.1 c.c.—of a high strain is enough to produce the disease, it would take a comparatively large amount of the blood-serum of the same animal—20 c.c.—to be effective. That the intravenous route of injection did not yield any good results so far is a very striking contrast to the uniform results obtained with the above-enumerated methods of experimentation.

From these facts we must conclude that the lymphatic circulation is the primary route and the circulatory system the secondary and, with the gray matter of the cord and brain, the coincident seat of the lesion. For this route of infection three avenues are obviously open, namely, the nasopharynx, the lungs and the alimentary canal.

It is true that in about 33 per cent. of the cases there are gastro-intestinal disturbances, but we must not lose sight of the fact that the disease is an infantile one and is coming on during the summer months so that, aside from the fact that an infection of any kind is apt to produce a gastro-intestinal disturbance in any infant, there might be quite another cause for these disturbances, such as bad food or the swallowing down of the irritating nasopharyngeal discharge containing the virus. Experimentally, the disease was produced in one instance through the alimentary canal in the monkey after an opiate was administered to the animal so as to paralyze the peristaltic movements of the intestine, and in no instance was the disease produced by feeding the animals with the feces of poliomyelitic patients. Pathologic findings in the gastro-intestinal tract in my cases so far reported were negative. Thus the alimentary canal may be excluded as a focus of infection.

In some instances characteristic lesions of poliomyelitis were reported to have been found in the lungs,¹ but these were rather few and since the nasopharynx, according to my theory, is the point of entry for the virus, it would seem to be a striking coincidence, which after all might be accepted as a further proof of my theory.

The nasopharynx is then the only avenue left and now we are facing the problem, the first phase of the paper, the mode of infection.

For a period of eleven years I have had the opportunity to examine schoolchildren of ages from 6 to 14 years. I have noticed that between the ages of 5 and 8 years there are present hypertrophied tonsils in 85 per cent. of the children examined, but as we go up in the age-scale, say at 14, we find only about 15 per cent. of the children with hypertrophied tonsils. The question

1. Flexner, Peabody and Draper: THE JOURNAL A. M. A., Jan. 13, 1912, p. 100.