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A CLINICAL STUDY OF ACUTE POLIOMYELITIS

BY

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*(From the Hospital of The Rockefeller Institute for Medical Research,
New York.)*

PLATES I-13.

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The following study is a report of the work on acute poliomyelitis carried on at the Hospital of The Rockefeller Institute for Medical Research during the summer of 1911. The material consisted of one hundred and sixty-one cases which occurred during the present season and twenty-two cases from previous years. Seventy-one of these cases were admitted to the hospital and were

under observation for a period averaging from three to four weeks. The other cases were seen in the dispensary.

The study was chiefly of a biological nature, and, on account of lack of time, observations of the disturbances of the reaction of the muscles to electrical stimulation were used for diagnosis and prognosis to a limited extent only.

We have attempted to correlate our experience with that of other observers, but no effort has been made to give a complete review of the literature. For a complete bibliography we refer the reader to the articles and monographs on which we have drawn freely.¹

INTRODUCTION.

The recognition of poliomyelitis as an acute infectious disease was delayed largely owing to the strange relationship between the obscure, inconsequential features of the early stage and the flagrant character of the resultant paralyses. In most text-books, descriptions of the disease as an acute infection dismiss the fever and initial malaise in a few lines, while the diagnosis is declared easy because of the sudden and complete paralysis. The following words from Allbutt's *System of Medicine* (1905) are significant: "... and after two days, when the pain had passed away, the case was found to be one of infantile paralysis. The fever and general constitutional disturbances at the onset had obscured the diagnosis." Furthermore, as a result of the earlier pathological studies of infantile paralysis,

¹ Römer, P. H., *Die epidemische Kinderlähmung*, Berlin, 1911.

Wickman, Ivar, *Beiträge zur Kenntnis der Heine-Medinschen Krankheit*, Berlin, 1907; *Die akute Poliomyelitis, bzw., Heine-Medinsche Krankheit*, Berlin, 1911.

Zappert, von Wiesner, and Leiner, *Studien über die Heine-Medinsche Krankheit*, Leipzig and Vienna, 1911.

Müller, E., *Die spinale Kinderlähmung*, Berlin, 1910.

Harbitz and Scheel, *Pathologisch-anatomische Untersuchungen über akute Poliomyelitis und verwandte Krankheiten*, Christiania, 1907.

Flexner, *The Contribution of Experimental to Human Poliomyelitis*, *Jour. Am. Med. Assn.*, 1910, lv, 1105.

Massachusetts State Board of Health, *Infantile Paralysis in Massachusetts in 1909*, Boston, 1910.

Epidemic Poliomyelitis. Report on the New York Epidemic of 1907 of the Collective Investigation Committee, New York, 1910.

made chiefly upon cases that died later from other causes, the term atrophic was added to the obscuring nomenclature of the disease. The changes noted in the spinal cord were scar formations in the anterior horns, and no lesions were found elsewhere in the body. Consequently, attention was focussed on certain chronic changes in a limited portion of the spinal cord. From the practitioner's standpoint, the natural course of the malady is such that his efforts have been palliative rather than curative. Usually he is called to face, helplessly, a child recovering from a febrile disturbance, but stricken with an amazing paralysis. The crippled state of the patient becomes the all-engrossing feature of the case, and the physician's chief effort is to devise methods of support and artificial activity. The management of this disease, therefore, has become a problem for the orthopedic surgeon; the internist is too late.

Fortunately, however, the work of the past few years on the experimentally produced disease, and, above all, the fine accounts of the human affliction by Wickman, Müller, and others have been most illuminating. It has become clear that the first outward expression of the disease is the fever, and in order that the subsequent paralyses may surely be prevented, the infecting agent must be destroyed before it has called forth a damaging reaction in the body. In the event of possible means of treatment in the future, therefore, early diagnosis is imperative, and in view of the obscure and often insignificant mode of onset, the problem becomes one of great difficulty, which calls for the best qualities and equipment of the internist. In those very symptoms of onset, the fever and general constitutional disturbances, which have been said to obscure the diagnosis, lie the warnings which should arouse the physician at once to such special procedures as are known to be helpful in making the diagnosis.

Besides the importance to the affected individual, the recognition of acute poliomyelitis in its febrile stage has a serious bearing on the community at large. It has been definitely shown that there are many obscure cases that never develop paralysis, whose mild symptoms indicate no specific infection, but which may carry and spread contagion. Just what proportion of all cases these abortive cases form is still a doubtful point. In the large epidemics, where

physicians are expectant, the reported number of such cases is relatively larger than among a series of sporadic instances of the disease.

At all events, it seems evident that abortive cases play a most important part in the total incidence of the disease; and in connection with their great frequency, it must be borne in mind that the virus is probably present in these cases in as active a form as in the patients with paralysis. In view of these considerations, it is obviously a matter of great importance to determine the relative frequency of the abortive forms of the disease, and to recognize them, for it is these cases which, not being recognized, escape quarantine and spread the virus. In general, the cases that develop no paralysis are clinically similar as regards history, prodromata, and onset, to those that go on to disability. Early diagnosis, therefore, serves a double purpose. The individual may, with an effective therapy, be spared a paralytic visitation, and the community may be protected from the menace of an unrestricted virus carrier.

The purpose, then, of the following pages is to emphasize the fact that poliomyelitis is an acute disease, in which the often insignificant febrile stage is of high epidemiologic and therapeutic importance.

HISTORICAL.

During the past seventy years infantile paralysis, or acute poliomyelitis, has been gradually separated from a mass of heterogeneous palsies and established as a pathological and clinical entity. Although between forty and fifty epidemics have been described, it is a striking fact that the first three important milestones on the path of our advancing knowledge of the malady are separated by long intervals of time.

In 1840, Heine,² an orthopedic surgeon of Cannstadt in Germany, published a monograph on paralytic conditions of the lower extremities and their management. The chief stress of the work is laid upon the surgical aspect of the disease, but there is a short, clear cut description of the onset and acute stage. For etiology,

²Heine, Jacob, *Beobachtungen über Lähmungszustände der untern Extremitäten und deren Behandlungen*, Stuttgart, 1840; *Spinale Kinderlähmung*, Stuttgart, 1860.

a sudden serous exudation in the spinal cord is suggested, and a rough description of spinal cord changes indicates the author's idea of the site of the lesion. The work of Heine is a fine example of keen clinical observation and leaves no doubt of the correctness of his view that the disease is an entity. Except for an epidemic described by Colmer,³ there is little more of clinical importance in the literature of poliomyelitis for the next forty years. Charcot,⁴ in 1870, studied the pathology of poliomyelitis and declared a primary degeneration of the anterior horn cells to be the morbid process. This view has been the subject of much discussion. In 1884, Strümpell⁵ pointed out the resemblance between certain cases of acute encephalitis with spastic paralysis in children and cases of poliomyelitis. He concluded that these two types of disease of the central nervous system had a common etiological factor, and suggested for the first time the hypothesis that an external infectious agent was responsible for them. His recognition of the true cerebral form of the disease was a most important contribution to the knowledge of poliomyelitis. During the numerous epidemics reported in the following years, many instances of apparently direct contagion were observed, which added support to Strümpell's suggestion. There was still lacking, however, a description of the symptomatology of the disease in its complete course.

In 1890, Medin,⁶ basing his studies upon the extensive Swedish epidemics, published the first good clinical account of acute poliomyelitis. Medin's work is perhaps as much of a classic as Heine's, and his name has been coupled with the latter's in association with the disease. He likewise described the spastic types of poliomyelitis which had been recognized by Strümpell as occurring in association with the usual forms. Following this, almost each year saw reports of smaller epidemics in France, Italy, Germany, and the United States; but no important additions to the existing knowledge of the disease were made, until Wickman,⁷ in 1905, studying a vast amount

³ Colmer, *Am. Jour. Med. Sc.*, 1843, v, 248.

⁴ Charcot, quoted by Oppenheim, *Lehrbuch der Nervenkrankheiten*, Berlin, 1908.

⁵ Strümpell, *Jahrb. f. Kinderheilk.*, 1884, xxii, 173.

⁶ Medin, *Verhandl. d. X. internat. med. Cong.*, 1890, ii, 6 Abt., 37.

⁷ Wickman, *Beiträge zur Kenntnis der Heine-Medinschen Krankheit*, *loc. cit.*

of material in Sweden, developed the epidemiology of the disease and its pathology, and described for the first time the abortive types. The importance of this particular contribution will become more evident in the discussion of the epidemiology. Wickman's exhaustive studies demonstrated conclusively the contagious nature of the disease. There remained, therefore, the problem of finding the infecting agent.

Experimental production of the disease in monkeys was first accomplished in 1909 by Landsteiner and Popper,⁸ and a few months later by Flexner and Lewis⁹ and by Strauss¹⁰ in this country. Three observers in different cities independently succeeded in transferring the disease from one monkey to another. This was done in November, 1909, by Flexner and Lewis in New York, Leiner and von Wiesner¹¹ in Vienna, and Landsteiner and Levaditi¹² in Paris. The reports from the various sets of workers appeared within a period of two weeks.

As a result of the extensive work which has been done on the experimental disease, many important facts about the etiology have been acquired. A bacterial cause has been definitely ruled out and there is now undoubted evidence that the infecting agent belongs to the group of so-called filterable viruses.¹³ The virus of poliomyelitis is highly resistant to many destructive measures; thus, it withstands glycerination for long periods and is not injured by 0.5 per cent. carbolic acid. Furthermore, freezing at -2° to -4° C. for forty days does not affect it materially. To heat, it is less resistant and it can be destroyed by a temperature of 45° to 50° C. for half an hour. The virus is readily destroyed by hydrogen peroxide in 2 per cent. solution, by menthol, and by corrosive sublimate. In monkeys, one attack of the experimental disease prevents a second successful inoculation. Fairly satisfactory results in the production of active immunity, induced by repeated injections of small amounts of attenuated virus, have been obtained, but these

⁸ Landsteiner and Popper, *Ztschr. f. Immunitätsforsch., Orig.*, 1909, ii, 377.

⁹ Flexner and Lewis, *Jour. Am. Med. Assn.*, 1909, liii, 1639.

¹⁰ Strauss and Huntoon, *N. Y. Med. Jour.*, 1910, xci, 64.

¹¹ Leiner and von Wiesner, *Wien. klin. Wchnschr.*, 1909, xxii, 1698.

¹² Landsteiner and Levaditi, *Compt. rend. Soc. de biol.*, 1909, lxxvii, 592.

¹³ Landsteiner and Levaditi, *loc. cit.*; Flexner and Lewis, *Jour. Am. Med. Assn.*, 1909, liii, 2095.

have not warranted the application of this procedure in man. The serum of recovered monkeys, and also the serum of human patients that have recovered from this disease, have been shown to contain a substance that is capable of neutralizing the virus *in vitro*. If 0.1 of a cubic centimeter of the active virus¹⁴ and one cubic centimeter of the serum of a case of poliomyelitis be mixed in a test tube, incubated for two hours at 37° C., refrigerated over night and then injected intracerebrally into a monkey, the animal does not have any symptoms of the disease. This neutralizing substance has also been demonstrated in the blood of abortive human cases.

EPIDEMIOLOGY.

The study of the epidemiology or mode of transmission of epidemic poliomyelitis has been much promoted by the observations of Wickman in 1905, who first drew special attention to its contagious character. All subsequent studies have been influenced by the fundamental observations of Wickman, and have tended to support his deductions.

An entirely new light was thrown on the obscure question of dissemination by Wickman's recognition of the so-called abortive and meningitic forms or types of the affection. Until then, attention had been riveted on the paralysis, and the possibility of cases of epidemic poliomyelitis occurring in which paralysis is entirely absent seems not to have been entertained. It is at once clear that the existence of such cases would throw an entirely new light upon the mode of transmission of the infection.

That epidemic poliomyelitis must be an infective process follows from its epidemic character. We are unacquainted with any disease that flourishes in epidemics that is due to other causes than parasitic. That the infective cause finds a considerable degree of resistance naturally present in any community is proven by the strong tendency of the frankly paralyzed types of the disease to appear as isolated cases in a given family or household. But this is merely a tendency, since the occurrence of dual and even multiple infection in one household is by no means uncommon. Indeed, now that the abortive and meningitic forms of the affection, without paralysis,

¹⁴ The active virus, so-called, is merely a filtrate of a 5 per cent. salt solution suspension of the spinal cord of an infected and paralyzed monkey.

are acknowledged, the instances of multiple cases are not so infrequent as they were believed to be.

To demonstrate that more than one case may frequently occur in the same family, the following three instances are significant. Two of these involve bottle-fed infants, the other deals with older children.

Family I.—Belle and Clara T., twins, eight months old.

Belle T. Feverish, August 18; next day temperature 103° F., throat red and inflamed, slight patch on the right tonsil; patient nervous; breathing rapid; pneumonia suspected. August 20, child less nervous and began to seem dull. August 21, twitching of face and athetoid movements of foot; later in the day weakness in left arm and leg. August 23, involvement of respiratory muscles; death.

Clara T. August 21, child irritable, vomited several times, and coughed. August 22, temperature 102° F., vomited several times; examination of right leg caused unusually loud screaming; slight weakness in right thigh. August 23, paralysis definite.

Here is a situation where twin infants were infected so nearly synchronously that it is impossible to say whether one case contracted the disease from the other, or whether a common source provided the virus for both. The fact that both infants were bottle-fed makes infection by interchange of nipples conceivable. The next example of family contagion also presents a somewhat similar situation.

Family II.—Margaret W., age fourteen months, began on October 4 to have typical symptoms of onset and in three days developed incomplete paralysis of both lower extremities. Four days later her infant sister became feverish and looked sick. Two days later the latter was brought to the hospital with beginning paralyzes which advanced rapidly and terminated fatally by involvement of the respiratory muscles. The mother said that this child had frequently been put in the same bed with the older one, and that she had seen the children alternately suck the nipple of the common feeding bottle.

Family III.—Hannah T., age four years. On October 1, she was well in the morning but vomited in the afternoon and was feverish. The next day she was paralyzed. There are four other children in the family, one older and three younger than Hannah. One week before her illness the older brother had had tonsillitis, and a day or two after his attack began, the three younger children had what the mother called a "drowsy sickness," in one case with fever. A small brother, one of the three, was brought to the hospital with typical symptoms of the acute stage of the disease and significant changes in his spinal fluid. There were no definite paralyzes, but a distinct shakiness in walking was noticed.

These family infections speak for themselves in the matter of direct contagion. The last instance, although lacking definite proof,

is highly suggestive of the importance of abortive cases. One further example, however, where the evidence of a neutralization test made probable the correctness of the diagnosis of acute poliomyelitis in a case that had no muscular weakness, is of interest at this time.

Helen K., age three years and ten months. On August 16, she had headache and was feverish; on the 18th, paralysis of the legs began and ascended rapidly to the arms and terminated fatally by respiratory involvement on August 21. Her constant playmate, Helen B., age six years, had headache on September 1, that is about ten days after Helen K. was ill, and the following day was brought to the hospital. Examination of the spinal fluid showed changes similar to those seen in poliomyelitis. She never developed paralysis and after fifteen days went home apparently well. Her blood serum protected a monkey against 0.1 c.c. of the virus.

This child had no definite symptoms beyond some stiffness of the neck, headache, apathy, and anorexia. Except for her close association with the fatal case, her mother would have paid no attention to what might have passed as a slight summer ailment, and a diagnosis would never have been made. The child would have gone to school in a few days and perhaps have been the source of infection for other individuals.

Human beings are very unequally subject to the effects of different contagious affections. It is only smallpox and measles that can vegetate in any soil; all other direct infections are conditioned by degrees of susceptibility of the host that determine the extent of their prevalence. Scarlet fever, typhoid fever, and epidemic meningitis can claim far fewer victims. Indeed, the degree of susceptibility, as determined by age and other circumstances, is strikingly similar in epidemic meningitis and epidemic poliomyelitis.

Epidemic poliomyelitis has been clearly observed to follow the lines of human contact and travel. Wickman has placed the primary center of infection and transmission in the village school, and the secondary centers he has traced to contacts that arise out of circumstances of ordinary human intercourse. Through these, the infection is spread in a village and from one village to an adjacent one. Moreover, he has emphasized that the carrying of the infection is twofold, by the affected and the non-affected, or actively and passively, as one would now say. The most important active

carrier of the epidemic is the victim of an abortive attack of the disease, since he may not be confined during any period and is as capable, probably, of distributing the infection as a case attended by paralysis. This is equally true of some other contagious diseases, notably diphtheria and scarlet fever. For in these diseases which are not highly contagious, as is measles, the healthy carrier has never been given much consideration; indeed it has been largely overlooked. This omission has been due to the high natural resistance possessed by the community.

Attention is then to be fastened upon the human agency of transmission. This is all important because, if it is the true and most frequent agency, then preventive measures, effecting both limitation and final suppression, will be taken accordingly. The admission of this source of contagion will dispose of the vague notions regarding the existence of sources of infection among the lower animals.

The following extract from a paper of Flexner's¹⁵ on the control of epidemic poliomyelitis will serve to show the present state of knowledge and conjecture on this aspect of the problem:

"Paralytic diseases among domestic animals are known and not highly infrequent. They have been noted among dogs, horses, and fowl, but thus far it has not been found possible to correlate the paralytic diseases of the lower animals and those of man. Perhaps the most frequently observed coincidental paralytic diseases have been between hens and human beings. Undoubtedly since the wide prevalence of epidemic poliomyelitis, the existence of a paralytic disease among barnyard fowl has been more commonly noted. Possibly the condition has not actually become more frequent, but owing to the circumstance mentioned it has been oftener observed. It would appear that the paralysis among fowl is caused not by lesions of the central nervous system, but by lesions of the peripheral nerves and that it is due to a peripheral neuritis. It has not been found possible to transmit by direct inoculation the paralytic disease from chicken to chicken, or from chicken to monkey, or from paralytic monkey to chicken (Flexner and Lewis). However, it has been found possible to develop the paralysis in the laboratory by keeping the chickens in confinement for some time, and by supplying them

¹⁵ Flexner, *Am. Jour. Dis. Child.*, 1911, ii, 96.

an unusual and doubtless improper form of food (Flexner and Clark). It has proved as little possible to transfer the paralytic affection of dogs from one individual to another by direct inoculation or from dog to monkey or from paralyzed monkey to dog (Flexner and Lewis). These failures do not, of course, exclude the possibility that a reservoir for the virus may exist among domesticated animals that do not even respond to its presence by developing paralysis or other conditions which could be recognized as resembling poliomyelitis in man. The manner of the action of the virus of poliomyelitis in rabbits provides an illustration which shows how necessary it is to avoid general deductions in this field. At first it was strenuously denied that rabbits could be infected at all with the virus of poliomyelitis, and the examples of supposed successful inoculation reported were entirely disbelieved (Krause and Meinicke, Lentz and Huntemüller); but it must now be accepted that young rabbits occasionally, but by no means generally, are subject to inoculation with the virus of poliomyelitis, at least after it has passed through a long series of monkeys (Marks). Apparently a small percentage only of the inoculated rabbits develop any obvious symptoms, and these die, as a rule, during convulsive seizures which come on suddenly. A given virus has up to the present been sent through a series of six rabbits, after which it has failed to be further propagated. From the sixth series it has been reimplanted on the monkey, in which animal typical paralysis has been produced (Marks). It remains to add that the rabbits which succumb to the inoculation do not show any characteristic lesion of the central nervous system or other organs, as far as has been determined. The monkey, on the other hand, invariably shows the typical lesions of the central nervous system.

“Insect contamination with the virus would serve, were it proved not only to be an experimental possibility but to occur in nature, to clear away any present apparent discrepancies in the epidemiology of the disease. In this connection it should be stated that not only does epidemic poliomyelitis spread over a wide territory, but its spread is not promiscuous, but along the routes of human travel. Therefore insects that seek human habitations and routes of travel,

that possess the power to migrate over a considerable territory, that affect all classes of society, that abound during the period of greatest prevalence of the disease, and that do not wholly disappear at any season, should be the first to come under suspicion. Many, if not all, of these conditions are fulfilled by the common house-fly."

The effort to make out a case for insect transmission has, however, failed up to the present. The part that the house-fly may play in the transfer of the virus is strictly passive. That these insects can carry the virus in a living and actively infectious state for forty-eight hours or longer has been proven by the laboratory experiments of Flexner and Clark. They can, therefore, conceivably accomplish a similar transportation of the virus, as a contamination, in natural infection. Their exclusion from poliomyelitis patients should be zealously carried out.

But the main problem relates to the human carrier, active or passive. Where does he harbor the virus, and how is it taken up by the victims? The evidence is growing constantly stronger that the upper respiratory tract, as early pointed out by Flexner and Lewis, is the site both of ingress and of egress of the virus.

That the lymphatics of the nasal mucosa are in almost direct connection with the subarachnoid space has been clearly demonstrated. Flexner¹⁶ has shown in the case of *Diplococcus intracellularis* that when monkeys are infected by intraspinal injection of cultures, the diplococcus can be found subsequently both in leukocytes and free in the nasopharynx. This fact and the demonstration of the virus of poliomyelitis in the nasal mucosa of infected monkeys point clearly to the nasopharynx as a path of discharge for agents infecting the cerebrospinal space. Successful inoculations of the nasopharyngeal mucosa of monkeys with poliomyelitis virus have shown, furthermore, that the same path may serve to admit the infecting agent.

In addition to the nasopharyngeal mucosa as a source for the distribution of the virus to the outer world, Landsteiner, Levaditi, and Pastia,¹⁷ and Flexner and Clark¹⁸ have shown that tonsils from fatal human cases also contain the virus of poliomyelitis.

¹⁶ Flexner, *Jour. Am. Med. Assn.*, 1910, lv, 1105.

¹⁷ Landsteiner, Levaditi, and Pastia, *Sem. méd.*, 1911, xxi, 296.

¹⁸ Flexner and Clark, *Jour. Am. Med. Assn.*, 1911, lvii, 1685.

The virus of poliomyelitis is unknown apart from infected human beings and monkeys, and yet it possesses a high degree of resistance. It withstands low degrees of cold and ordinary degrees of heat for long periods of time. When enclosed in albuminous matter, it withstands drying for several weeks. It can therefore survive both in moist and dry conditions and thus is capable of being carried directly to the upper respiratory mucous membrane as spray produced by coughing and sneezing and even loud speaking of the infected, and as dust. A more detailed discussion of the conditions that affect the virus was given in the historical section.

From the evidence at present available, therefore, it is evident that epidemic poliomyelitis is a human-borne, contagious affection, the portal of entry for the virus of which is the upper respiratory tract, in particular, the nasopharyngeal mucous membrane. Other agencies than man play a part in distribution that is distinctly subordinate and inferior. The infection can be carried and implanted both by active and passive carriers; being resistant and having access to external nature with mucous secretions, it can become attached to dead objects, bedding, clothing, etc., and to domestic pets and domestic insects (fly); and it can be ground into dust and conceivably be disseminated by wind. Recent experiments of Neustaedter and Thro¹⁹ lend support to this latter hypothesis. These being the probable modes of transmission, preventive measures should be taken accordingly. They are such as are already in use in contagious diseases that have a similar mode of infection; namely, diphtheria and scarlet fever. A further consideration of methods of prevention is given in the section on treatment. Osgood and Lucas²⁰ have shown that the virus is present in the nasal mucosa of recovered monkeys five months after the acute attack of the disease. A similarly long period of survival of the virus in the mucous membrane of affected human beings has, however, not been demonstrated.

Epidemics rise and fall, and rarely disappear suddenly. The series of cases that form the basis of the present study constitute the instances still arising that owe their initial impulse to the epidemic wave that appeared in New York in 1909, and has since been slowly disappearing.

¹⁹ Neustaedter and Thro, *N. Y. Med. Jour.*, 1911, xciv, 813.

²⁰ Osgood and Lucas, *Jour. Am. Med. Assn.*, 1911, lvii, 495.

NEUTRALIZATION TEST.

As has been emphasized in the discussion of the epidemiology of poliomyelitis, abortive attacks are of common occurrence when the disease is epidemic. These instances of poliomyelitis are difficult to recognize clinically and a specific biological test would be of great assistance in the diagnosis of such atypical infections. Up to the present time no characteristic reaction has been demonstrated during the acute stage of the disease. Levaditi and Landsteiner, Römer and Joseph, and Flexner and Lewis have shown that the serum of recovered monkeys is able to protect normal animals against fatal doses of the virus. Netter and Levaditi, Flexner and Lewis, and Anderson and Frost have shown that the serum of recovered human cases possesses the same protective property. The last named observers demonstrated this quality in the sera of 66.7 per cent. of a series of abortive instances of poliomyelitis. They also proved that normal human serum has some neutralizing action, but are of the opinion that this action has quantitative limits which clearly differentiate it from the action of the serum of persons who have had poliomyelitis. While such protective bodies do not appear in the serum until about two weeks after the onset of the disease, the fact that the virus is known to survive in the throats of experimental animals for a much longer period of time may make it advisable in extensive epidemics to employ the test in order that the community may be protected from suspected individuals who may act as carriers.

Up to the time of the publication of the paper by Anderson and Frost, the reliability of the test was not questioned. These observers show that normal human serum possesses limited power to neutralize the virus of poliomyelitis. During the past year at the Hospital of The Rockefeller Institute, the serum of a number of individuals was tested for its ability to protect monkeys against infection with the virus of poliomyelitis. The material consisted of sera from normal persons, from those exposed to infection in the hospital ward, from individuals suspected of having an abortive attack of the disease, and from a certain number of typical cases. The test was performed in the following way: the serum was mixed with a fatal dose of a known active virus, incubated from one

to two hours at 37° C. and then allowed to stand for twenty-four hours on ice. The injections were made intracerebrally into monkeys (*Macacus rhesus*). The protocols of the experiments follow.

NORMAL SERA.

Experiment I.—The serum was obtained from a normal adult, one of the physicians in constant attendance on cases of poliomyelitis in the hospital ward. During this time he had presented no signs of illness. One c.c. of the fresh serum was mixed with 0.1 c.c. of virus and was incubated for two hours at 37° C. After standing over night on ice, the mixture was injected intracerebrally into a monkey. After seventeen days the animal began to show signs of weakness and was found dead on the following day. At autopsy the characteristic lesions of poliomyelitis were present in the spinal cord.

Experiment II.—In this instance the serum was obtained from a normal adult, a nurse in charge of cases of poliomyelitis in the hospital. She had no paralyses and gave no history of any illness resembling poliomyelitis, nor was she ill at any time during her service in the poliomyelitis ward. One c.c. of this serum was mixed with 0.1 c.c. of virus and neutralization tested in the manner described. The monkey inoculated became ill on the eighth day after injection. It was somewhat depressed and its movements were slow for several days. On the nineteenth day it developed diarrhea and eight days later it was found dead. At no time did the animal show definite paralyses. At autopsy the microscopic lesions were not characteristic.

Experiment III.—The serum for this experiment was obtained from a girl of eight years of age, a patient in the hospital suffering from chronic endocarditis. She had no paralyses and gave no history of any illness resembling poliomyelitis. Two tests were made with this serum. The first monkey was inoculated with a mixture of 1 c.c. of fresh serum and 0.1 c.c. of virus, prepared in the usual way. The animal survived and at no time developed any symptoms. There was no hyperexcitability and no paralysis. A second test was made with this serum, in which 1 c.c. of serum was mixed with 0.3 c.c. of virus. This animal likewise remained healthy and survived.

Experiment IV.—The serum for this experiment was obtained from one of the doctors in constant attendance on the ward. He gave no previous history of poliomyelitis, but some six weeks previously had a slight illness of about a week's duration, the chief symptoms of which were diarrhea, dizziness, and pain in the neck. There was no muscular weakness. One c.c. of this serum was mixed with 0.1 c.c. of virus and the neutralizing power tested in the usual manner. Seven days after injection, the animal's nutrition was poor; there were no paralyses and no hyperexcitability. On the fortieth day the monkey was still living, when it was again inoculated with 1 c.c. of serum mixed with 0.3 c.c. of virus. The animal developed diarrhea, became emaciated, and died on the eighteenth day after the second injection, without having developed any paralyses.

Experiment V.—The serum of a normal adult, a nurse in attendance on cases of poliomyelitis, was used for this experiment. Some weeks previously she had

a slight illness, lasting three days, the symptoms of which were dizziness and gastro-intestinal disturbance. There was no muscular weakness and no pain associated with the attack. One c.c. of this serum was mixed with 0.1 c.c. of virus and after allowing time for neutralization to occur was injected intracerebrally into a monkey. Forty days later the animal was living and had shown no symptoms. At this time another test was made, using 1 c.c. of serum and 0.3 c.c. of virus. Eleven days later the animal's movements became slow, it grew thin and weak and died on the fourteenth day after the second injection. At autopsy the grey matter of the cord looked somewhat edematous. There were no definite microscopic lesions of poliomyelitis.

Experiment VI.—The serum for this experiment was obtained from M. S., two and one half years old, a patient in the hospital suffering from cerebral hemorrhage. Two tests were made with this serum; in one, 1 c.c. of serum was mixed with 0.1 c.c. of virus; and in the other, 1 c.c. of serum with 0.3 c.c. of virus. The first animal died of poliomyelitis in seven days and the second in eight days.

SERA OF SUSPECTED CASES.

Experiment VII.—The serum was obtained from Patrick T., aged twenty-three months, a patient in the poliomyelitis ward. At the time of admission to the ward, the patient's sister was suffering from poliomyelitis with paralyses. The patient on admission was drowsy, showed the characteristic irritability on being disturbed, and had slight fever. The spinal fluid showed 94 cells per c.mm., practically all of which were of the mononuclear type. Although the mother noticed some weakness in the child when he got up from a sitting posture, the child showed no paralyses while in the hospital. One c.c. of the serum obtained from this patient seven days after the onset of the disease, was mixed with 0.1 c.c. of active virus and injected intracerebrally into a monkey. Nine days later the animal died of typical poliomyelitis.

Experiment VIII.—The serum for this test was obtained from Donald P., the brother of a patient who died of poliomyelitis in the hospital ward. He had shown suspicious symptoms at the time of the other child's illness. The serum was obtained about three weeks after the suspected attack and 1 c.c. of it was mixed with 0.1 c.c. of virus and injected intracerebrally into a monkey. Ten days later the animal died, having developed a characteristic poliomyelitis.

Experiment IX.—The serum used in this test was obtained from H. B., a patient in the hospital. A constant playmate had died of poliomyelitis some two weeks before the patient's admission. The patient, when studied in the ward, had slight fever and showed marked drowsiness. There were no paralyses but slight spasticity on walking and some rigidity of the neck. The maximum cell count in the spinal fluid was 62 cells per c.mm., chiefly mononuclear. The globulin content of the fluid was slightly increased. A monkey, injected intracerebrally with a mixture of 0.1 c.c. of active virus and 1 c.c. of the patient's serum, obtained three weeks after the onset of the illness, showed no signs of poliomyelitis.

SERA OF TYPICAL CASES OF POLIOMYELITIS.

Experiment X.—For this experiment, serum was obtained from C. T., an adult who had suffered from poliomyelitis thirty years previously. Two tests

were made with this serum. In one, 0.1 c.c. of active virus was mixed with 1 c.c. of serum; and in the other, 0.3 c.c. of the virus was mixed with 1 c.c. of serum. Two monkeys were injected with the material in the usual way. Both animals were protected, neither showing any signs of poliomyelitis.

Experiment XI.—In this instance the serum was obtained from an individual, E. R., sixteen years of age, who had had poliomyelitis fourteen years previously. One c.c. of serum was mixed with 0.1 c.c. of active virus and injected into a monkey. Twenty days later the animal developed a paralysis of one leg. The paralysis, however, did not progress and the condition of the monkey returned to normal, with the exception of a residual paralysis. Inasmuch as the dose of virus given has invariably proven fatal in unprotected monkeys, the serum of this patient must be considered as having some protective power.

Experiment XII.—The serum for this test was obtained from C. A., a child eleven years old who had had poliomyelitis ten years previously. One c.c. of serum was mixed with 0.1 cc. of active virus and injected into a monkey in the usual way. The animal was protected, showing no symptoms of poliomyelitis at any time.

Experiment XIII.—The serum used for this experiment was obtained from M. G., four years old, a patient in the poliomyelitis ward. On admission there was partial paralysis of the external ocular muscles, high fever, and some stiffness of the neck. The maximum cell count in the spinal fluid was 320 cells per c.mm. One c.c. of serum obtained thirty days after the onset of the disease was mixed with 0.1 c.c. of active virus and injected intracerebrally into a monkey. Protection was complete, the animal showing no evidence of poliomyelitis.

The above series of tests of the ability of normal, of suspected, and of certain poliomyelitic sera to protect monkeys against infection with the virus of poliomyelitis, needs very little comment. Judging from the results obtained, the test cannot be considered to give specific evidence as to whether a given individual has or has not suffered from a previous attack of poliomyelitis. Of the six normal sera tested, but two failed to protect animals against infection, whereas four gave complete protection, and three of the latter protected against such large doses of the virus as 0.3 of a cubic centimeter. It is, however, fair to say that in two of the instances in which protection was obtained, the individuals had been constantly exposed to infection with the virus of poliomyelitis, and during the time of exposure had suffered from an indefinite illness which might possibly be attributed to an abortive attack of poliomyelitis. In three instances serum was obtained from persons suspected of having poliomyelitis. Of these sera, one protected a monkey against experimental infection and two failed to protect. Two of these individuals were carefully observed in the hospital

and both presented fairly definite clinical evidence of the disease. The serum of one of the latter protected a monkey against infection, and the serum of the one that failed to protect was obtained too early (seven days after the onset of the disease) for the immune bodies to be present in the serum in sufficient concentration. Four sera obtained from individuals who had had a characteristic poliomyelitis protected animals against fatal infection in every instance. One serum which was obtained thirty years after the occurrence of the disease, protected against both 0.1 of a cubic centimeter and 0.3 of a cubic centimeter of active virus. From these results it would seem that in typical instances of poliomyelitis, the serum of the infected individual probably always contains, after a certain interval, protective bodies, and that these substances persist for a very long time. In spite of the irregular results obtained with normal sera and the sera of suspected individuals, one cannot as yet rule out the specificity of the test. In the case of apparently normal persons whose serum protects, the possibility must always be borne in mind that he may previously have suffered from an unrecognized, abortive attack of the disease. The accurate determination of the protective value of normal serum can probably be made only by a study of sera from very young individuals in whom the possibility of previous infection can certainly be excluded.

PATHOLOGY.

The earliest pathological studies in poliomyelitis were made on chronic cases of the disease, and the lesions described were the atrophic scars found in the anterior horns of the cord. With the development of a better clinical knowledge of the acute stage of the disease, a new pathology arose which laid especial stress on the earliest morphological changes in the cord, and explained their relation to the development of the chronic forms. The work of many investigators, notably Harbitz and Scheel,²¹ Wickman,²² and Strauss,²³ has produced an accurate picture of the anatomical lesions

²¹ Harbitz and Scheel, *loc. cit.*

²² Wickman, *Die akute Poliomyelitis, loc cit.*, p. 13.

²³ Strauss, *The Pathology of Acute Poliomyelitis, Report on the New York Epidemic of 1907 of the Collective Investigation Committee, New York, 1910.*

occurring in the brain and spinal cord. The attention of observers has hitherto been almost wholly centered on the lesions of the central nervous system, and in consideration of the fact that the most striking disturbances, both pathological and clinical, are associated with the nervous system, this is not remarkable. In view, however, of the very definite and constant changes which are found at autopsy in other viscera, it is rather strange that they should have been almost wholly disregarded, and that so little emphasis should have been put on the fact that acute poliomyelitis is essentially a general infection. Rissler,²⁴ Strauss, Harbitz and Scheel, and Wickman have all noted the presence of lesions outside the nervous system, but have passed them by as having little bearing on the disease. As a matter of fact, the recognition of acute poliomyelitis as a general infection has an important bearing both in explaining the clinical course of the disease and as affecting any possible means of treating it.

Inasmuch as the lesions of the central nervous system are of chief importance, they may be considered first. At autopsy the meninges are usually found to be somewhat edematous and injected. There is little increase of cerebrospinal fluid. The brain and cord, on section, have a moist, translucent, edematous appearance, and the gray matter of the cord is often swollen so that it projects above the level of the white matter. It is darker than normally in color and is typically of a grayish pink hue. Not infrequently minute hemorrhages can be distinguished in the gray or white matter.

The exact path by which the virus enters the body is at present not definitely known, but there is clinical and experimental evidence which makes it seem probable that infection frequently gains access from the upper respiratory tract. It has been shown both anatomically and experimentally (Flexner²⁵) that the upper nasal cavities are in direct communication with the meninges by means of the lymphatics which pass outward with the filaments of the olfactory nerve. The view that the virus may enter the body by means of those lymphatics and thus exert its first effect upon the meninges is

²⁴ Rissler, Zur Kenntniss der Veränderungen des Nervensystems bei Poliomyelitis anterior acuta, *Nord. med. Ark.*, 1888, xx, 1.

²⁵ Flexner, *Jour. Am. Med. Assn.*, loc. cit.

strengthened by the anatomical findings. The earliest change which has been described in the nervous system is hyperemia and the collection of numbers of small mononuclear cells, probably lymphocytes, in the perivascular lymph spaces of the blood-vessels of the leptomeninges. These lymphatic spaces surrounding the vessels are anatomically processes of the arachnoid spaces, and the lymph in them is in communication with the cerebrospinal fluid. This first change, then, is an acute interstitial meningitis, which is not associated with fibrin formation or with exudate on the surface of the meninges. It is usually most marked on the anterior surface of the spinal cord, and especially in the anterior fissure, from which the larger vessels enter the cord (figure 1); but smaller collections of cells are often found along the meningeal vessels which are situated over the lateral and posterior aspects of the cord. The blood supply of the cord is derived from the vessels of the meninges, and with the advance of the pathological process, this perivascular infiltration follows along the vessels as they enter the cord from the meninges. Thus the earliest change that is found in the cord itself, both in human beings and in the experimentally produced disease, is hyperemia and the collection of small round cells in the lymph spaces surrounding the vessels. This cellular exudate forms a sheath apparently completely surrounding the vessels for long stretches (figure 2), and in many places the cells are so numerous that they form thick collars which seem to press on the lumen, and thus exert a mechanical effect in obstructing the circulation. While the cellular exudate is in the outer part of the vessel wall, it is probable that there is often some effect, either toxic or mechanical, on the intimal lining of the vessels, for hemorrhages (figure 3), minute or extensive, are frequent findings, and one of the prominent features of most cases is the extensive edema. These three factors, cellular exudate, hemorrhages, and edema, all of them dependent on vascular changes, may perhaps be regarded as the primary reaction of the nervous system to the virus of poliomyelitis. The effects produced on the nerve cells themselves are probably either dependent on these vascular disturbances or they may be due to a direct action of the virus. This superior importance of the vascular system in determining the nervous lesions has for a

long time explained the fact that the cervical and lumbar enlargements of the cord are most affected, and that the anterior horns of the gray matter are more involved than the posterior horns or the white matter. These are, of course, the regions of the spinal cord to which the blood supply is most abundant. Moreover, the frequency with which lesions are asymmetrical probably depends on the irregularity with which the vessels supplying the cord are given off at different levels. It has been suggested that the process by means of which the vascular lesions affect the nerve cells may be essentially a mechanical one. It is quite impossible to exclude the fact that the virus may exert some directly toxic action on these cells, but in many ways, the clinical and anatomical pictures are readily explained by the presence of the circulatory disturbance and of the exudate. On such an hypothesis the damaging effects can be assumed to result in part from the direct pressure on the nerve cells of hemorrhages, edema, and exudate. There is also the additional factor of anemia following the constriction of the blood vessels by the same mechanism. On account of this pressure and anemia, the nerve cells degenerate. If the hemorrhage and exudate are absorbed soon enough, the cells may recover their function. If, on the other hand, the anemia and pressure have been prolonged or excessive, the nerve cells go on to complete necrosis. Histological examination shows nerve cells in all stages of degeneration, from those with the slightest changes in their protoplasm to others of which only a granular detritus remains. A most striking picture is formed by the entrance of polymorphonuclear neurophages into the necrotic nerve cells (figure 4). A single nerve cell may be invaded by a dozen of these phagocytes, and by means of them necrotic material is completely disposed of. In more severe lesions, one sees the hyperemia, the perivascular infiltration, hemorrhages, edema, and a diffuse cellular infiltration throughout the gray and white matter, but nerve cells may be completely absent from the picture. These changes, most prominent in the anterior horns of the gray matter, are not sharply circumscribed, but are scattered more or less diffusely through both the gray and white matter of the cord (figures 5, 6, and 7).

The same sequence of changes, vascular disturbance, and subse-

quent degeneration of the nervous elements, is found to a less degree in the brain, medulla (figure 8), and pons. Hyperemia and a moderate amount of cellular infiltration may be found in association with the vessels of the cerebrum and cerebellum, but lesions extensive enough to produce motor symptoms are exceedingly rare. The medulla and pons show some slight degree of involvement in most cases, and one frequently finds in them a marked cellular exudate and many hemorrhages. It is noteworthy, however, that it is often extremely difficult to reconcile the clinical symptoms, which are referable to pontine lesions, with the actual autopsy findings. Cases which have shown bulbar paralyses in life may fail to show adequate anatomical lesions to account for them, and other cases which have given clinical evidence of spinal involvement only may show changes through the pons and medulla.

Of practically constant occurrence are the lesions in the posterior root ganglia. The histological changes are similar to those that take place in the cord itself (figures 9 and 10). There is an infiltration of small round cells in the lymphatic spaces surrounding the vessels which enter the ganglia from the meninges. This has been shown experimentally to be the first step in the process. Then follows a more general, diffuse exudation of cells, degeneration and necrosis of the nerve cells, and finally the entrance of polymorphonuclear leucocytes into the necrotic cells and removal of the disintegrating cells by neurophages (figure 11). The suggestion has been made that these lesions in the sensory ganglia may in part account for the pain which is such a constant feature of the acute stage of the disease. Another element in the production of pain is the cellular infiltration which is found along the nerve roots.

The changes which are found in other organs in acute poliomyelitis are less striking than those in the nervous system, but they have been, in our experience, practically as constant. In all of the eleven acute cases which we have been able to examine, there has been more or less extensive involvement of the lymphoid tissue and of parenchymatous organs. The lymphoid tissue throughout the body appears to react to the virus. The Peyer's patches of the intestine and the mesenteric lymph glands show perhaps the most marked acute swelling. The mucosa over the Peyer's patches is,

however, unaffected. There is also definite, and sometimes pronounced enlargement of the substernal, bronchial, cervical, axillary, and inguinal lymph glands and of the tonsils. The spleen is frequently somewhat enlarged, and on section the Malpighian corpuscles stand up in raised, pale, obviously translucent nodules. The thymus shows changes identical with those in the lymphoid tissue elsewhere. On histological examination, one is struck by the resemblance of the lesions found to those described by Mallory²⁶ in typhoid fever. The reaction is, in general, the same throughout the lymphoid tissue, regardless of its location. On histological examination some of the lymphoid nodules may present a normal appearance, but the majority consist of a zone of lymphocytes surrounding a more or less sharply circumscribed pale center (figure 12). High magnification shows the center of the lobule to consist chiefly of large endothelial cells with oval vesicular nuclei. These cells are similar to the cells lining the lymph sinuses, but most of them are larger, more swollen, and take the stain very lightly. Sometimes the nuclei look like pale shadows, and the outline of the protoplasm is so faint that it can scarcely be distinguished. Where they are closely packed, the individual cells appear to be fused together to form compact masses. The better preserved of these cells are markedly phagocytic and frequently contain many particles of necrotic cells. These cell inclusions are surrounded by a lightly stained halo and are apparently situated in vacuoles in the protoplasm. Scattered throughout the center of the nodule are many broken down cells and granular fragments of necrotic nuclei. The cells which are going to pieces are for the most part lymphocytes, but the endothelial cells also seem to swell up and finally disintegrate. In areas with extensive necrosis there is often an invasion by polymorphonuclear leucocytes.

In the lymph sinuses there are also large numbers of the same phagocytic endothelial cells. Many of them are of great size and contain necrotic fragments of nuclei, whole lymphocytes, or numbers of red blood corpuscles. In the lymph sinuses, there is extensive proliferation of the endothelial cells, as is evidenced by the frequency with which mitotic figures are found. Numbers of ne-

²⁶ Mallory, *Jour. Exper. Med.*, 1898, iii, 611.

crotic cells are met with in the lymph sinuses, but, in general, necrosis is a more prominent feature in the centers of the lymphatic nodules, and proliferation in the lymph sinuses.

Among the parenchymatous organs, "cloudy swelling," such as has been frequently described, is usually met with. In the liver, however, there are other more striking changes, and these again resemble in character those that have been described by Mallory and others in typhoid fever. They are for the most part sharply circumscribed areas, ranging in size from lesions which consist of one or two cells to others which include nearly one eighth of a liver lobule, in which degeneration of liver cells and infiltration of lymphoid cells and polymorphonuclear cells have taken place (figure 13). The number of these necroses is very variable. They may be rather difficult to find, or there may be many of them in a single low power field. The lesions are apparently closely associated with the blood vessels, and while chiefly with the portal vein, they are also associated with central or sublobular vessels. The section is, however, sometimes cut so that the relation to the blood-vessels is not directly evident. The lesions are round or oval in cross section, but there may be long finger-like projections, extending out from the main area, and involving one or two columns of liver cells. It is striking that the liver cells directly adjacent to the infiltrated areas are usually completely spared, but occasionally one finds that some of them show a homogeneous quality of the protoplasm, and an exaggerated affinity for eosin, which suggest a beginning involvement through a peripheral spread of the lesion.

The character of the lesion varies, of course, with its size and age, but in general it is very constant. In the earlier lesions one finds one or two disintegrating liver cells, with homogeneous, hyaline, pink-staining protoplasm, and occasionally with irregular, distorted nuclei. In the older lesions, even more apparent than the necrotic liver cells is the evidence of an early and rapid proliferation. Scattered through practically all the lesions are small groups of liver cell nuclei, frequently a nest of three or four nuclei surrounded by infiltrated tissue, or several nuclei apparently beginning to proliferate within a degenerated liver cell body. The protoplasm around these dividing nuclei may appear as a faint pink halo, or it may be

quite impossible to make out any protoplasm at all. In older lesions, the protoplasm is better defined and the cells have frequently arranged themselves circularly, in acini, or in double columns. This arrangement of cells simulates that in normal bile ducts, but it is found in association with nearly all the lesions, irrespective of their position in the lobule, although most frequently adjacent to the portal spaces. According to the position of the infiltrative lesion, much or little fibrillated tissue appears among the degenerating and infiltrating cells. The greater amount of fibrillated tissue occurs in and adjacent to the portal spaces, where all the lesions of an early cirrhosis may be displayed. That this condition is of the nature of a beginning cirrhosis is further indicated by the microscopic depressions of the capsule of the organ, where the lesions extend toward and reach that structure. However, the process probably is not permanent, since it is so young and cellular that it can readily undergo retrogression; while the infiltrated nodules elsewhere in the lobules are surely easily subject to restoration. One case has indeed come to autopsy, the child dying of a laryngeal diphtheria two months after the onset of poliomyelitic paralysis affecting both legs. In the liver of this case were remains of the portal infiltrative lesions described and, in addition, younger necrotic and infiltrated lesions among the columns of liver cells. The former probably had been greater and were diminishing; it is uncertain whether the latter were residues of the poliomyelitic infection or the result of the recent diphtheritic process. That these changes in the lymphoid tissues and in the liver are, in fact, a part of the reaction of the body to the virus of poliomyelitis, would seem to be made certain by the fact that exactly similar lesions may be found in the organs of monkeys which have been experimentally infected with the disease (Flexner²⁷).

The demonstration of such a widespread reaction to the virus is wholly in line with recent clinical and epidemiological advances which tend to recognize acute poliomyelitis as a general infection. The disease must be regarded as a generalized process which affects parenchymatous organs, lymphoid tissue, and more especially the nervous system. It is possible that two distinct effects of the disease

²⁷ Flexner, *Folio serolog.*, 1911, vii, 1101.

on the organism should be differentiated. On the one hand is the general toxemic process which affects organs throughout the body, but which apparently acts mildly. On the other hand is the local process in the spinal cord, producing death by destruction of the nerve cells controlling respiration. The anatomical findings which point to an action of the virus of poliomyelitis on the lymphoid tissue throughout the body may well be correlated with the results obtained by the inoculation of emulsions of lymphoid organs into monkeys. Not long after the disease was first transferred to monkeys by the intracerebral inoculation of filtrates of the spinal cord, the virus was proved, in the same manner, to be present in a mesenteric lymph gland. Since then similar positive results have been obtained with still other lymphatic glands in the monkey, and with emulsions of the tonsils in man and the monkey.

These observations are not, however, of the same significance and importance. What they tend to indicate is that the living virus may come to rest for a time in organs outside the central nervous system, to which it is conveyed by the blood. It does not establish the fact that the visceral lesions outside the nervous system are caused directly by the virus, rather than by some secondary toxic substance produced in the course of its proliferation. Since the only means at present available to demonstrate the presence of the virus is the inoculation of monkeys, the tests made are too few to determine how widespread in the organs the virus really is. It is established that it is present in the central nervous system, even when it cannot be demonstrated in the viscera generally. But the finding of the virus in the tonsils and nasal mucosa with as great constancy as in the nervous system indicates that these organs play a part in the conveyance of the virus into and away from the central nervous system. The virus is regularly present in them in fatal and doubtless in non-fatal cases of poliomyelitis in children, and it has been demonstrated in the nasal mucosa, the tonsils, and even in the nasal mucous membrane in infected monkeys; it finds its way into the nasal mucous membrane, even when injected into the peritoneal cavity. The evidence, therefore, is strong that, as was first pointed out by Flexner, the upper respiratory mucous membrane provides for both the ingress and egress of the virus of the disease, through which infec-

tion is produced and the renewal of the cause of the disease maintained.

SYMPTOMATOLOGY.

CLINICAL CLASSIFICATION OF CASES.

The study of any disease is simplified if the various clinical forms in which it may appear can be grouped together into a few definite types. While no two cases of any disease are ever exactly similar, one probably finds variety of gross clinical manifestation most strikingly illustrated in the diseases of the nervous system, and a pathological process which may extend more or less continuously through brain, medulla, pons, spinal cord, and spinal ganglia, or which may be localized in any part of this system may produce signs and symptoms of very diverse character. Such are the possibilities that are present in poliomyelitis, and it is no wonder that the clinical classification of cases has been the subject of much discussion. The most generally accepted system has been that of Wickman, who recognizes eight forms: (1) the spinal, poliomyelitic form, (2) the cases simulating Landry's paralysis, (3) the bulbar or pontine form, (4) the encephalitic form, (5) the ataxic form, (6) the neuritic form, (7) the meningeal form, and (8) the abortive cases. While such a classification certainly covers all possible cases and makes it easy to place most cases in their appropriate groups, it does not appear to us to be wholly satisfactory. It is based neither on pathological anatomy, nor on clinical symptomatology, but on a mixture of the two. The spinal, the bulbar, and the encephalitic forms are anatomical forms; the neuritic, the meningeal, and the ataxic forms are essentially symptomatic forms. A more satisfactory classification would be either anatomical or clinical. Such a classification would simplify a confusion arising from the use of two systems, for it is impossible to draw any hard and fast line between some of the groups. Thus most neuritic forms are essentially of the spinal type, and most meningeal cases are either spinal or bulbar. The ataxic form Zappert regards as based on a single, not especially prominent symptom, rather than on an anatomical foundation. The cases simulating Landry's paralysis are in the main instances of the spinal type in which the process advances, usually to end in death

from involvement of the respiratory muscles. Cases that are classed among the meningeal or neuritic forms are so classed because they show some of the clinical manifestations of meningitis or of neuritis, rather than because they have a pathology which is essentially different from other bulbar or spinal cases. These types of the disease are, in fact, not true types at all. They merely represent some of the variations in symptomatology which may be found more or less developed in association with either the abortive, the bulbar, the spinal, or the cerebral types of the disease, and their enumeration as definite forms is complicating and at times confusing. As a result of this duplicating and overlapping of subdivisions, the picture of the disease, as a whole, is made much more complicated and obscure than is necessary.

More simple and, at the same time, more practical is the classification proposed by Müller. Adopting, as far as possible, an anatomical basis, he proposes the four following classes: (1) the spinal form, (2) the bulbar form, (3) the cerebral form, and (4) the abortive cases. In any system of classification, the abortive cases must, of course, form a distinct type. In considering the cases, however, which result in paralysis, one would seem to get a clearer outline of the disease process, if the cases are looked at from the point of view of pathological physiology and divided into two main groups: (1) those cases in which the upper motor neurone is primarily affected, and (2) the larger group of cases in which the lower motor neurone is involved. The first group is the cerebral form of Müller, and the second group is composed of his spinal and bulbar forms. The two latter forms it is wiser to consider together, as in both the lesion is essentially the same, a lower motor neurone lesion involving, on the one hand, the nuclei of the pons or medulla and, on the other hand, the anterior horn cells, the result being in either instance a flaccid paralysis with subsequent atrophy. The two types which are thus based on similar pathological lesions are also inseparable clinically, for a somewhat larger number of cases occurs with cranial nerve involvement, in association with spinal lesions, than occurs with cranial nerve involvement alone. The third group of cases in Müller's classification is the cerebral type, the same group to which Wickman gives the name encephalitic. This

is a fairly definite pathological entity, at least in so far as the most prominent lesion apparently involves the upper motor neurone. While the bulbospinal type is characterized by flaccid paralyses, this cerebral type is characterized by spastic paralyses or by other evidence of a lesion above the lower neurone. The exact location of the pathological processes in these cases is not wholly clear, but it seems probable that the upper neurone may be attacked in either or both of two places. Some few cases are probably due to lesions in the cortex with involvement of much larger areas than are commonly found. Other cases, the majority, seem to depend on involvement of the pyramidal tracts, either high up or in the cord. That this class of case, originally described by Strümpell as *polioencephalitis acuta* is very uncommon, but that it is in reality a form of infantile paralysis, has been made quite certain by epidemiological studies which show the spastic type occurring in the same epidemic and in the same house with the flaccid types, by the occasional occurrence of both spastic and flaccid paralyses in the same patient, as well as by the pathological studies of Harbitz and Scheel.

It would seem, then, that the best appreciation of acute poliomyelitis, from the clinical point of view, is obtained if one recognizes three groups of cases. The first group consists of the *abortive cases*, cases of infection which never become paralyzed. The second or *cerebral group* contains the rare cases in which involvement of the upper motor neurone with resulting spastic paralysis is the chief characteristic. The third or *bulbospinal group* is much larger and comprises all cases with lesions in the lower motor neurone and flaccid paralyses.

Such a classification is, of course, open to the objection that many cases are not purely of one type, either anatomically or clinically. Thus most bulbospinal cases show some pathological foci in the brain, and cerebral cases may show lesions extending into the cord (Harbitz and Scheel). Then from the clinical point of view a certain number of bulbospinal cases develop symptoms as, for instance, ataxia, which may well depend on an upper motor neurone lesion, either in the brain or cerebellum, in the pyramidal tracts, or in Clarke's columns. When one recognizes how diffusely the pathological process extends through the whole central nervous system,

it becomes evident that it is impossible to make any hard and fast classification which shall be strictly applicable to all cases. The best one can do is to attempt to reconcile the chief clinical symptoms with the predominant anatomical lesion. In the following sections, we shall consider the symptomatology and course of each of these types in detail.

PREDISPOSING CAUSES.

Acute poliomyelitis is a disease which shows very definite seasonal variations in its incidence. The records of epidemics in many countries show that it occurs during the summer and reaches its maximum in the late summer and early autumn. In the Swedish epidemic of 1905, 86 per cent. of the cases had their onset between July and October, and 35 per cent. during August. The New York epidemic of 1907 showed a very similar curve, reaching its maximum, however, in September. Other epidemics show some slight variations in duration and in the period at which the highest point is reached, but that the disease is distinctly one of summer and fall has been generally accepted. There are a few well substantiated epidemics which prove that poliomyelitis may also occur during cold weather. Wickman cites one epidemic in the north of Sweden, lasting through the winter, and reaching its maximum in April and May. He also reports three closely related epidemics, occurring in neighboring sections of the country, the first (eighteen cases) lasted from June to October, the second (twenty-seven cases), from July to December, and the third (sixty-two cases), from the end of September to February, with its maximum in November and December. These winter epidemics are of interest in relation to attempts that are being made to account for the spread of the disease by some intermediary insect host. Stress has been laid on the frequent occurrence of epidemics of poliomyelitis in unusually hot, dry years, but this relationship is certainly far from constant.

It has been generally noticed that acute poliomyelitis is a disease of open country, rather than of cities. Among our own cases, a relatively larger number of children came from the suburbs and surrounding country than from the densely populated tenement district in which the hospital stands. Moreover, a considerable

proportion came from families which are in comparatively well-to-do circumstances and in which the children enjoy every comfort and care. Pneumonia, measles, and other acute diseases are often mentioned as predisposing causes of poliomyelitis, but in our experience the absence of any history of previous illness has been much more noticeable. In general, they had been perfectly healthy children.

Childhood is essentially the age which is most susceptible to poliomyelitis, but that adults are by no means immune is shown by Wickman's statistics. More than one fifth of his cases were persons over fifteen years old. He mentions one case in a man of forty-six years, and instances of the disease occurring in even older persons are on record. In the New York epidemic of 1907, the youngest case was two weeks old. Three cases seen by us were three months old, and fifteen cases were between six and twelve months. As in the 1907 epidemic in New York, the majority of our cases were between the ages of one and three years. The figures obtained by Müller agree rather remarkably with ours in respect to age incidence. Of his cases 96 per cent., and of ours 97 per cent. were in the first decade, while 90 per cent. of his, and 89 per cent. of ours were below the age of five. Perhaps the age most liable to infection is the latter half of the second year. Both sexes are almost equally susceptible to the disease, the number of males being slightly greater than the number of females.

TABLE OF AGE INCIDENCE.

	Months.		Years.													Total.
	Under 6	6-12	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-15	15-20	20-25	Over 25	
Rockefeller In- stitute Hospi- tal, 1911...	3	15	62	29	18	13	6	4	0	2	1	3	0	1	0	157
New York Epidemic, 1907.....	62		221	180	106	63	28	18	11	11	7	14	5	1	2	729
Wickman, Sweden, 1905.....		183			214			179			229			220		1,025

THE PRODROMAL PERIOD.

In most typical cases of infantile paralysis, the course of the disease is fairly constant. A period of incubation is followed first by a period of prodromal symptoms, then by an acute stage with paralysis, and finally by a stage of retrogression. The duration of the incubation period is variable. In the experimental disease in monkeys, Flexner has found that the time elapsing between inoculation and the onset of paralysis is from three or four days to thirty-three days, the average being eight or nine days. Prodromal symptoms are of short duration in the monkey, rarely lasting over twenty-four hours. With a virus that has been passed through several generations of monkeys, and has thus become adapted to its new host, the period of incubation is much more constant than it is on the first transfer from man. Such a fixed virus causes paralysis with great regularity in seven or eight days. In the disease in man it is obviously much more difficult to determine the exact length of the incubation period. Thus, even when two cases appear in one family, it is often impossible to be certain at what time the second case became infected by the first, or even whether both were not infected at the same time from a common source. Wickman considers the incubation period as being from one to four days, his opinion being based on the interval between the dates of onset of the disease in two persons of the same family. Our own observations of families in which two cases have occurred would lead us to a nearly similar conclusion, but we believe that these are in all probability simultaneous infections from a common source. At any rate, previous contact had been so intimate that infection might have taken place at any time during the incubation period of the first case. In one family at least, two children became paralyzed on the same day. Müller has seen six instances in which the length of the incubation period could be determined more accurately, and he concluded that it varies between five and ten days. The average incubation period was about a week, thus practically the same as is found in the experimentally produced disease.

Following the incubation period and preceding the onset of paralysis, there is, in the vast majority of cases, a period marked by prodromal symptoms. These symptoms are at times of such

a mild and fleeting nature that they may be entirely overlooked, but except in the case of small babies, and with unusually unintelligent parents, careful history taking will almost always bring out evidence of some prodromata. There is, however, a small number of cases in which the acute stage with paralysis really seems to be the first outward manifestation of the disease. We have seen several cases occurring in intelligent families in which, in spite of careful questioning, it was impossible to obtain evidence of prodromal symptoms. The usual history in this rather exceptional type of onset is that the child goes to bed perfectly well and wakes up paralyzed. These represent but a very small fraction of the total number of cases; the others give a definite history of a prodromal stage. The prodromal period, which, until recently, received little attention at the hands of clinicians, has, in the light of our present knowledge of epidemiology and of our hopes for a therapeutic control of the disease, assumed an unexpected prominence. It is during the prodromal period that one must isolate and quarantine, if such measures are expected to be efficacious, and it is only in the prodromal period, before an extensive destruction of nerve cells takes place, that one can ever hope to make treatment efficient. This is, therefore, the most important stage in the course of the disease, for on its recognition depends the possibility of controlling the infection.

The duration of the prodromal symptoms is variable. In our series of cases they lasted, in most instances, from one to seven days, with one to three days as an average. The usual history is that the symptoms increase progressively in severity through the prodromal period, but occasionally, after being sick for two or three days, there is a distinct cessation of symptoms and the child apparently recovers. One or two days later, however, and perhaps without further warning, the child becomes paralyzed (case I, page 122).

The severity of the prodromal symptoms, also, varies greatly from case to case. While in one child they are so mild and transient that they are quite disregarded, in another they may be alarming. In a considerable percentage of our cases the children were sick enough during the prodromal period to make the mothers feel that a doctor should be sent for. It is a generally accepted fact that the severity of the prodromata bear no relation to the extent of the ensuing paralysis or to the subsequent course of the disease.

The prodromal symptoms are, on the whole, of a general nature. A few are more specific in character and shed some light on the nature of the disease. For the most part they are such as may appear at the onset of almost any acute infection or, indeed, such as occur in children with very little recognizable cause. While individual cases show considerable differences, there are certain groups of symptoms which are present in the majority of all cases. Other types of symptoms have been shown to have a tendency to vary from epidemic to epidemic. Thus in the epidemic which occurred in Hesse-Nassau and was reported by Müller, the majority of the cases had prodromal symptoms referable to the respiratory tract. In the neighboring province of Westphalia, during the same epidemic, Krause²⁸ found a preponderance of cases with gastrointestinal symptoms. Two thirds of his cases had a marked diarrhea. During the New York epidemic of 1907, intestinal symptoms were not marked, but gastric disturbances were common.

Probably the most constant feature of the prodromal period is *fever*. In most cases this is noted among the earliest symptoms, in fact the usual story is that the patient was perfectly well in the morning, but towards evening became feverish. Sometimes fever was apparently not noticed until the second or third day after the child became sick, but this may be due to the fact that a history of the presence or absence of fever is usually based on the appearance of the child and the temperature of the skin, rather than on actual temperature determinations with the thermometer. However, in a number of cases, actual measurements showed that a temperature of 103° F. or higher is of frequent occurrence. Onset with chill is comparatively rare. The febrile reaction generally lasts through the prodromal period, with slight morning remissions, and the temperature comes down to normal a few days after paralysis sets in. Occasionally the fever remits earlier, especially in those cases with a distinct interval, in which the child is without symptoms, between the prodromata and the acute stage.

Müller has laid special stress on profuse *sweating*, which he designates as one of the cardinal symptoms of the early stage of the disease. It was present in 75 per cent. of his cases. A definite

²⁸ Krause, *Deutsch. med. Wchnschr.*, 1909, xxxv, 1822.

explanation of the mechanism of its production he is unable to give, but he suggests that a pathological involvement of spinal sweat centers may play a part. The question of sweating was carefully gone into in our histories, but in only 25 per cent. of the cases had it been noticed at all. The number of cases with profuse, drenching sweats was very small. One naturally expects to find sweating a rather common accompaniment of fever, especially in children, and the comparatively slight transient sweats met with in most of our cases were no more than could be explained by the febrile reaction, and by the warm summer weather. On the whole, sweating was not a prominent symptom in this series of cases. The few cases in which sweating persisted after the temperature became normal will be discussed in the following section.

Associated with fever and perhaps dependent on it, is *drowsiness*, which was a very noticeable symptom in many cases. The children are apathetic and want to sleep most of the time. This drowsiness sometimes lasts only a day or two and then disappears, but often it increases and runs into a mildly stuporous condition with the onset of the acute stage. With flushed face and dulled, apathetic sensorium, the child may have a definitely typhoidal appearance. Frequently, the degree of apathy is out of all proportion to the height of the temperature. On being awakened, the patient is usually irritable. He cries and wants to be let alone to doze off again. Not infrequently drowsiness is less prominent, and *irritability* with a nervous, excited, complaining disposition replaces it. One baby, whom we watched in the prodromal period, was wide-eyed, alert, nervous, observant of all that went on around her, breathing rapidly, and crying when anyone approached her or attempted to touch her. This irritability is closely connected with what Müller considers as one of the three cardinal symptoms of the prodromal period, *hyperesthesia*. This symptom, which was present in three quarters of the cases in the Hesse-Nassau epidemic, was also very constantly seen in our cases. Müller mentions a hyperesthesia of the skin of which we found no instance in the preparalytic stage, but *pain on passive motion* was very frequently noticed. The mother would often say that the child cried when it was picked up, or when it was moved to change the bed linen. The movements which tend to produce pain

are essentially those which cause an anterior flexion of the spine; thus, flexion of the legs at the hip joint and flexion of the neck. At this early stage in the disease, retraction of the head is rarely met with, but stiffness of the neck and a definite resistance to flexion are very common. Similarly, the attempt to test for the Kernig sign often causes pain and evokes a resistant contracture of the hamstring muscles. This type of pain would seem to depend on the inflammatory involvement of the meninges, which has been shown in monkeys to occur in the preparalytic stage.

The frequency with which *spontaneous pain* occurs is difficult to determine on account of the youth of most of the patients. Those that are old enough, however, frequently complain of it. Pain in the head, in the back of the neck, in the back, or in the legs, is often present, though usually of a transient nature. Not infrequently there is pain in those limbs which subsequently become paralyzed. It seems probable that such pain may be due to the inflammatory reaction in the intervertebral ganglia. Pain, both spontaneous and that produced by motion, tends to increase as the acute stage is approached.

Another not uncommon forerunner of paralysis is *weakness* of one or more limbs or groups of muscles. A child may be noticed to "favor" one arm more than the other, or he walks with a limp, or his legs "give way under him." Physical examination shows no paralysis, but only a more or less definite weakness of the part. Associated with this may be changes in the tendon reflexes. These changes are by no means constant and may consist either in an exaggeration, in a reduction, or in complete loss. Perhaps most cases show first an early exaggeration of reflexes during the "irritative" stage, followed by a loss of reflexes just before the onset of paralysis. Less common "irritative" phenomena are *muscular twitchings* and tremors, such as were present in several of our cases. As one mother put it, "the baby's leg quivered." In the same category belong the general *convulsions*, which are, in our experience, rather unusual prodromata. Occasionally, however, they play a prominent part in the early history of the illness and suggest a cerebral disturbance.

In some epidemics of poliomyelitis, the disease is frequently ushered in by symptoms involving the respiratory tract. This, as has been pointed out, was notably the case in the Hesse-Nassau epidemic of 1909, in which over 50 per cent. of the patients had such symptoms. The character of the symptoms varied from a persistent cold in the head or a conjunctivitis, to an angina, bronchitis, or even a bronchopneumonia. While this type of symptom has not been at all frequent in our cases, we have had a few which gave a history of nasal discharge, and others of a more or less troublesome cough in the prodromal period. Occasionally, one of the older patients has complained of sore throat. The tonsils, as we have seen them, usually, however, after the onset of the acute stage, have been larger than normally and often ragged. Reddening of the tonsils has been rare, and in no case was a true exudate present. On the whole, disturbances of the respiratory tract have been rather remarkable for their absence.

Gastric symptoms, on the other hand, have been very frequently observed in our cases, as they were during the New York epidemic of 1907. Loss of appetite and nausea were fairly constant symptoms, and vomiting occurred in a large percentage of the cases. The vomiting was usually an early symptom and was in many instances the first evidence of sickness. The great majority of the children vomited only once or at most twice, and frequently it was noted that the vomiting followed immediately the taking of food. Many writers have called attention to the difference between this type and the recurring, projectile vomiting which occurs early in meningitis. Rarely, however, is persistent vomiting met with.

In the Westphalian epidemic described by Krause, over two thirds of the cases had a diarrhea in the prodromal period. Apparently it is more common to have a moderate grade of constipation. Very few of our cases showed any tendency to diarrhea. In almost all, the bowel movements had been normal, or there had been a degree of constipation such as one usually finds when a patient is put to bed. There has been some attempt to associate diarrhea with the pathological lesions of the intestine found at autopsy. Our experience makes such a relationship seem improbable, for all our autopsies

sies showed well marked intestinal lesions, but in none of the cases had there been any diarrhea.

The clinical picture of the prodromal period of infantile paralysis shows very little that is of specific diagnostic value. In general, the history is that of a previously healthy child taken suddenly sick during the summer or early fall with fever, a moderate gastro-intestinal disturbance, a sore throat, or a slight cough, pain in the head, back, or legs, and often becoming quickly very drowsy. On physical examination there is stiffness of the neck with resistance to flexion; the Kernig manipulation of the legs and other passive motions are painful; the child is irritable when handled and wants to be left alone. Hyperesthesia is the most characteristic symptom, if one exists, but obviously there is little on which to base a diagnosis unless the relationship is close to other cases in an epidemic. The vagueness of the clinical picture is illustrated by a glance at the diagnosis made in some of our cases before the onset of paralysis: teething, tonsillitis, malaria, gastro-intestinal disturbance, and pneumonia. These show how far at sea one can be in the face of these prodromata. Have we, then, other methods of diagnosis that may be summoned to give assistance?

Müller has laid great emphasis on the fact that the blood picture is of value in the differential diagnosis of infantile paralysis. Both in the experimentally produced disease in monkeys and in the disease in man, he was struck with the tendency towards a leukopenia in the febrile stage. In his examination of the blood in fifteen cases, he found a low leukocyte count (3,000-5,000) in the majority, and a normal count in the remainder. Associated with this was a relative lymphocytosis. What proportion of these cases were in the pre-paralytic period and what proportion were in the early acute stage with a persisting fever, he does not state. These results, however, were not confirmed by La Fetra,²⁹ who found, in what Müller terms an "atypical epidemic" in New York, leukocyte counts running from 13,400 to 20,600. In four cases between the second and fifth day after the acute onset, all "still in the hypersensitive state with commencing paralysis," Gay and Lucas found leukocyte counts of 17,400, 10,000, 12,000, and 13,400. Our own experience with

²⁹ La Fetra, *Arch. Pediat.*, 1909, xxvi, 328.

blood examinations during the prodromal period has been limited, but those cases which we have seen lead us to believe that leukopenia is not a sign to be relied on. One case gave on the fourth day after the onset a white count of 7,000, and three days later, on the day preceding the paralysis, a count of 3,000. The other cases seen in the preparalytic stage had white counts of 8,800, 17,000, 15,000, 17,600, 15,000 and 35,000. The last was in a baby seven months old.

Of much more importance have been the results obtained by examination of the spinal fluid. In six instances we have done lumbar puncture in the prodromal period, and in all of them we have obtained pathological fluids. The character of these fluids will be discussed in detail in a later section; but it may be said that the general change consists in a moderate increase in pressure, an often striking increase in the number of cells per cubic millimeter, and in some cases an increase in the globulin content. The type of cells present may be either mononuclear or polynuclear. Pathological fluids at this stage of the disease have also been observed by Frissell (one case) and Lucas (nine cases). While the spinal fluid shows, according to our present knowledge, no changes that are specific for poliomyelitis, it often gives evidence very suggestive of meningeal involvement, and the absence of bacteria that are associated with the usual types of meningitis clears the field of many confusing elements. It would seem then that where one can correlate the clinical picture with the findings in the spinal fluid, a correct diagnosis should be frequently made in the prodromal period of poliomyelitis.

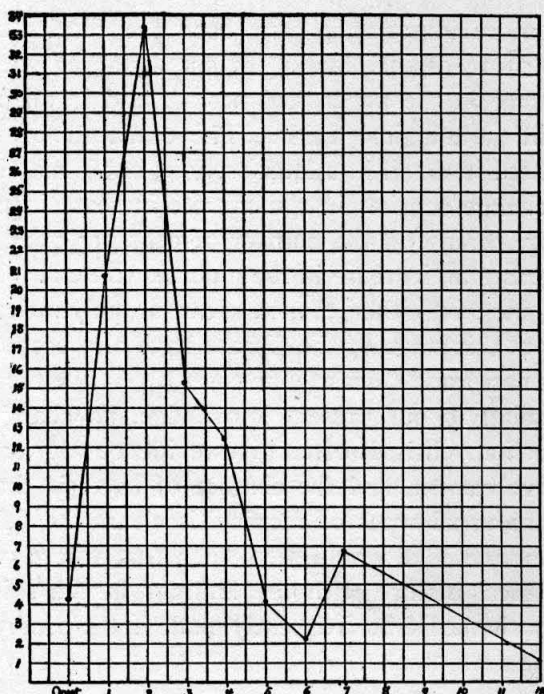
BULBOSPINAL TYPE.

ACUTE STAGE.

It is impossible to draw very definite lines about the stage of the disease to be called acute. The temperature is not a definite criterion of the stage of the pathological process, for it is often gone before paralysis appears. Frequently, also, a child may become more prostrated and more stuporous several days after the temperature has fallen to normal. A better though still somewhat unsatisfactory signal of the fully developed disease is the *advent of*

paralysis. Any limits, therefore, put upon an acute stage in poliomyelitis are arbitrary and necessarily loose; but we have considered the acute stage to include the days between the onset of the paralysis and the disappearance of tenderness on spinal flexion.

The clinical picture of the fastigium of acute poliomyelitis, like that of the period of onset, is as varied as its pathological mechanism would indicate. It is convenient, however, to describe this part of



TEXT-FIG. 1. Chart showing the relation of the onset of paralysis to the day of disease.

the disease under four groupings: (I) the usual form; (II) the cases which have no prodromata and whose first symptom is paralysis; (III) the cases with a remission of symptoms and delayed paralysis; and (IV) the cases with deep stupor.

I. Usual Form.—In the commonest form of the disease, the paralysis appears on the first or second day after onset (text-figure 1). At this time the child may be found lying on its back,

with thighs slightly flexed and everted in a frog-like manner, and the head usually rotated to one side. The eyes are partly or wholly closed and there is a peculiar tired, wilted expression. Not infrequently, the chin is pointed upward a little, indicating a small degree of retraction. From this drowsy or almost sleeping condition, the child can be roused suddenly, often by the gentlest touch or manipulation of an extremity. Very frequently when the leg is lifted only a few inches from the bed, an expression of annoyance, rather than distress, crosses the face, and if the leg be the paralyzed one, the child often tries to free it from the examiner's hands by twisting the trunk and shoulders. This procedure is a surprisingly common one and is usually accompanied by a pettish, fretful, rather bored look and whine. But when the examiner stands back from the bed, the patient lapses almost at once into the drowsy state. In contrast to these cases are those of a more sthenic nature with evidently greater meningeal irritation. These children are almost always found lying on their sides with their heads drawn well back and their knees and thighs flexed, a typical meningitis posture. Occasionally a true opisthotonos appears. One such patient could not be made to lie on his back, evidently because the flat line of the mattress prevented the slight degree of opisthotonos which made him comfortable; but when a pillow was doubled up under the lower dorsal and lumbar region so that an opisthotonos was induced by gravity acting on hips and shoulders, the child promptly went to sleep in the dorsal position. Such cases also are apt to have the drowsy, wilted look. Much more rarely, the child is wide-eyed and has an anxious, apprehensive, rather frightened expression. Many times these patients do not wait to be touched before objecting, but cry out even when the nurse or doctor approaches the bed; and it is surprising how keenly the little patients seem to determine whether or not an approaching person is coming to perform some service which necessitates manipulation. In these cases one is amazed at the ingenuity and activity with which the child, using what muscles he has, braces and turns and twists to escape painful positions. Indeed, in two instances where both legs, both arms, back, anterior, and posterior neck muscles were paralyzed, the hopeless attempt at defence was limited to wagging the head from side to side and feebly whining.

In sharp contradistinction to these types are the few individuals who with limited or extensive paralysis do not seem to be at all sick.

As a rule, unless definite ocular palsies are present, the eyes present no abnormalities. Sometimes photophobia is marked. The ear drums are normal in the great majority of cases. An occasional slight reddening along the handle of the malleus or about the margin of attachment has been seen. The throats, on the other hand, have usually shown a considerable degree of congestion. The tonsils have been, as a rule, enlarged but not reddened. In many cases they have appeared ragged and swollen. The tongue, in most instances, is covered with a delicate pale gray coating and sometimes heavily furred. Some of the younger children have been cutting teeth. Herpes labialis appeared only once or twice. The superficial lymph nodes were always palpable and often enlarged. It is, of course, not justifiable to lay too much stress upon glandular enlargement in children, but the finding has been very constant, and it is of interest that recent pathological studies have shown that a general adenopathy belongs to the disease.

Two interesting features of poliomyelitis associated with the neck have attracted our attention. As is generally described, rigidity of the posterior muscles has been almost a constant finding. It has not always been possible, however, to be sure whether this rigidity was of reflex origin or due to a calculated effort on the child's part to prevent anterior flexion. This question will be more fully discussed subsequently. The cervical rigidity has varied in degree from sufficient stiffness to permit lifting the whole trunk by the head to an almost normal flexibility. Most cases are markedly stiff and occasionally rigidity and retraction of the neck is as well developed as in a true meningitis (cases 2 and 3, pages 123 and 125). The other neck phenomenon has been a peculiar weakness of the anterior neck muscles, apparently not associated with a definite paralysis or even paresis. Very many of the children have shown it in the first few days of the disease. If the patient, lying in the dorsal position, is lifted by the shoulders, the head hangs back. Apparently no effort is made by the patient to raise the head so that it may follow the shoulders and trunk. Sometimes a slight effort may be made, but soon the head is allowed to drop back. This may depend on weakness of the muscles, but it is also possible that the

child gets some momentary sense of comfort, of which he takes full advantage, from the hyperextension of the neck which the manipulation permits. When the sign has persisted after the first or second week, we have usually thought that a definite paresis or paralysis was present.

Except in the cases where some part of the respiratory mechanism is disabled, the thorax has shown no abnormality caused by the disease. But the picture of the respiratory paralyses has been very striking. A description of them, however, does not belong here and will be given in the section covering the paralyses in general. The lung findings also have not been abnormal save in the cases with weakness of diaphragm or intercostals; but in those instances there have occasionally been found extensive areas of moist rales resembling in considerable detail the sounds of an acute pulmonary edema. These patients have had no variation in temperature at the time when the rales were most conspicuous. The significance of these sounds with a consideration of certain other vasomotor phenomena will also be discussed in the section on paralysis. Frank bronchopneumonia appeared in two of the thirteen cases with weakness of the respiratory muscles. In both of these the temperature continued high to the end.

The abdomen, like the thorax, is rarely the site of abnormality. There is often a slight distension, due perhaps to the same cause that underlies the constipation so commonly met with. The belly is soft and not tender. When the recti or lateral muscles are paralyzed there is sometimes a peculiarly lax, inelastic feeling to the abdominal wall. In these cases, also, a startling protrusion of part of the parietes may appear when the child cries. In the younger children, the liver is palpable, as becomes their age, but it is never tender nor pathologically enlarged. The spleen likewise shows no constant clinical abnormalities.

A curious thing is the unheralded advent of the paralysis. One may observe in the morning that a child moves its arms easily; a few hours later, on going to the bedside for some other purpose, the patient is found lying quietly as before, but when he rolls over, one arm falls back limp. The child seems unaware of the loss of power. In a few cases patients have complained of pain in an

extremity shortly before paralysis supervened. While the presence of paralysis is easily detected in older children, it is sometimes most difficult to find in infants. In any case, the most satisfactory method of beginning a search for muscular weakness is to sit down by the bed and watch the child for many minutes. Of course, in some instances, the patient lies a limp, inert form, the subtle expression of vitality gone. In such cases a glance is enough to determine the presence of paralysis, and more careful examination is needed only to learn its extent. If the pain or tenderness is not too intense, gentle handling of the extremities soon discloses the muscle groups that still have some power left in them. Older children will usually move the arms or legs upon suggestions calculated to demonstrate faulty motion. Often, however, when there is great pain, it is hard to tell whether the children will not or cannot move. In such cases the physician must decide whether the demonstration of the presence of a paralysis is of sufficient importance to justify his causing the patient the pain involved in such demonstration. In such cases the only way in which the presence of a paralysis may be accurately determined is by pricking the skin and determining whether or not the child draws the part away from the source of discomfort. For instance, if it be suspected that the deltoid is weak, the skin should be pricked on the posterior and inner aspect of the arm, when, if the deltoid is not involved, the arm will be drawn outward and upward. The extremity should be so placed at first that the suspected muscle will have to work against gravity; but it is surprising how quickly and skillfully the smallest infants turn and twist to make use of this natural force. Occasionally the presence of tone in a muscle can be shown by putting it suddenly on the stretch. For example, if the flexed forearm be sharply drawn down by the examiner toward extension, definite resistance will be noticed when the biceps is normal.

There is one group of cases in which it is almost impossible to locate definitely the paralysis. These patients present all the other features of the disease but no paralyzed muscles can be found. If such children be stood on their feet, however, they suddenly buckle at the hips and fall in a heap on the floor. Probably weakness of

the gluteal muscles in some cases is accountable for this form; in others, weakness of the quadriceps.

Müller states that paralysis of the intrinsic back muscles is of frequent occurrence. This is a difficult palsy to demonstrate in the acute stage of the disease; for in the great majority of instances, the children will not or cannot sit up at this time. In some cases pain makes the patient antagonize the effort to sit up; in others there is apparently such general weakness of trunk and neck that the head drops and the spine bends like a reed. Yet in a week or two these children may be sitting up straight in bed and playing actively.

The superficial and deep reflexes have shown considerable variation, but, on the whole, they have had a tendency to be present in the acute stage of the disease. This applies, of course, to the muscles where paralysis does not prevent response. The following table is made from a group of thirty-seven cases in which the power of response to patellar tendon reflex existed in one or both lower extremities.

TABLE OF KNEE JERKS.

Paralysis.	Number of cases.	Right knee jerk.		Left knee jerk.		Both knee jerks.	
		Present.	Absent.	Present.	Absent.	Present.	Absent.
Upper extremity and respiration alone	11					5	6
Bulbar	5					4	1
One lower extremity, either with or without upper . .	21	10		4		3	4
Total	37						

Knee jerk (one or both) present in 26 cases.

Knee jerks (both) absent in 11 cases.

Knee jerks (both) exaggerated in 3 preparalytic cases.

Knee jerk (right) present, knee jerk (left) absent in 1 preparalytic case.

It is evident that the knee jerk on the unaffected limb is more often present than absent. In eleven cases where paralysis was limited to the upper half of the body, the knee reflex was positive on both sides five times and negative six times. In eighteen cases of lower monoplegia, the knee jerk appeared in the strong leg fourteen times and was absent in four instances. Three cases with paralysis in muscles below the knee only had retained knee jerks. Of the four preparalytic cases, three presented bilateral exaggera-

tion; in one case the right reflex was present and the left absent. In a few cases the disappearance of a knee jerk, which had previously been active, has preceded paralysis of the extremity. However, absence of a knee jerk has not always meant loss of muscle power. Perhaps of more significance from a prognostic standpoint than the disappearing knee reflex at the beginning of the disease, is its reappearance in the period of improvement. Often the earliest sign of returning power is a slight jump of part of the quadriceps femoris when the patellar tendon is tapped. Occasional cases have shown greatly increased knee jerk on the strong side, when the reflex in the paralyzed leg is absent. This exaggeration has usually been more striking in the period of improvement, and sometimes has been associated with a crossed adductor reflex.

The appearance of a cutaneous eruption, which has been described as part of the disease, we have not seen. There have been several cases having more or less extensive erythematous rashes about the neck and chest, but these have looked like prickly heat and have had no constant distribution. Some of the children had been badly bitten by mosquitoes and many had pediculosis. Many cases have shown a pinkish purple mottling of the paralyzed extremities, but none the extreme degrees of cold purple skin with edema that have been described. In one instance, giant urticaria appeared, but there has been no example of zoster.

Sweating has not been a constant feature in the acute stage, but numerous cases showed localized areas which were continuously covered by perspiration. Thus one child's hands and feet sweat profusely, but the arms and legs were dry. Several of the patients had sweating heads; in one case it was sharply limited to the left half of the face. A few of the children sweat generally and fairly profusely for a week or more. There was no constant relationship between sweating areas and paralyzed muscles. Several tests for sweating by local hot air baths failed to give any satisfactory results.

A few cases have had retention of urine so that catheterization was necessary. This condition never persisted more than a few days. In some instances, true retention did not exist but there was difficulty in starting the stream. Hot applications over the dis-

tended bladder, or placing the child on a chamber containing hot water usually sufficed to induce micturition. In one case with stupor there was incontinence.

Constipation has been the rule. In only one patient was there any indication of loss of rectal control. This was in the case of an intelligent eight year old boy who, when constipated, had no difficulty; but when, as the result of catharsis, the stools were very loose, he had involuntary movements on several occasions and did not know it.

Pain in some form is a constant feature of the acute stage of poliomyelitis. In general, three types are found: spontaneous pain, pain caused by manipulation, and tenderness to pressure of the muscles and nerve trunks. These are not all equally common. Pain caused by passive motion is most frequent and seems to depend primarily upon anterior flexion of the spine. The clearest demonstration of this fact occurs when a child's trunk is bent ventrally, shoulders toward hips, to throw the spinous processes apart in preparation for lumbar puncture. Such a procedure brings about immediately a marked degree of anterior spinal flexion and is strenuously objected to by the patient. The entrance of the needle is often unnoticed. There are several other manipulations, like the test for stiff neck and Kernig's sign, which necessitate more or less bending of the spine anteriorly. The ingenious and active efforts of the children to thwart any motion which involves the least bending forward of the spine, or indeed diminishes a slight protective opisthotonos, have been very striking, so much so that we have been led to believe that the stiff neck of poliomyelitis differed from that of meningitis in being voluntary rather than reflex. With the Kernig's sign also, the voluntary element, where retained muscle power permits, is even more definite. If flaccid paralysis of a lower extremity makes resistance impossible, there is always complaint of pain when extension is carried until the buttocks begin to rotate forward and upward; but when the muscles have power to act, resistance to hyperextension is definitely voluntary and has not the feeling of reflex spasm. This painful bending of the spine is also often responsible for the unwillingness of children to begin sitting up in bed. The symptom occasionally persists for sev-

eral weeks and, in these instances, is the only thing that keeps a happy, healthy looking child flat in bed. It is interesting to watch these patients on their backs, playing cheerfully and actively with arms and hands, suddenly look glum, apprehensive, and suspicious when a move is made toward them that may mean raising their shoulders and heads from the pillow. Often early improvement in the paralysis is masked by this painful symptom, or the fear of it (cases 4 and 5, pages 127 and 129).

Spontaneous pain sometimes occurs in poliomyelitis. This is much less frequent than pain on passive motion. Usually it follows the course of the nerves like a true neuritis. It may be very severe. Young children cannot definitely recognize limitation of pain to the course of a nerve and they complain, therefore, of distress in the whole leg or foot. Such pain may perhaps be more often present than is generally supposed, for it frequently requires much urging and even sharp prodding to make a child move an extremity which seems to be paralyzed, when in reality the muscles have power but are painful. In some cases a child will cry out with pain which seems to come in stabs and paroxysms. The duration of pain of this sort varies like most of the symptoms of the disease. As a rule, it rarely lasts more than a week. In the case of one adult, the pain was so severe that morphine was necessary on several occasions. With young children we have used codeine. The third painful feature of acute poliomyelitis is the tenderness of muscles to pressure. Sometimes merely a touch suffices, but usually the muscles must be seized between thumb and fingers and a little pressure made to produce pain. There is little doubt that this tenderness is in the muscles and is not a hyperesthesia of the skin, for rubbing the skin without pressing on the underlying muscles, or even pinching, causes no painful sensation. A few cases also have definite tenderness over the nerve trunks like a neuritis (case 6, page 131). In one adult with complete flaccid paralysis of the right lower extremity, who had also great spontaneous pain, pressure over the anterior crural trunk caused intense suffering. Headache is more a symptom of onset than of the acute stage.

From the discussion of fever in the section on prodromata, it is evident that an elevation of temperature in poliomyelitis is not only

a constant feature, but also of short duration. The day of disease has been calculated from the date either of vomiting or onset of feverishness. The following table is based on the temperature findings of fifty-four cases in the acute stage of the disease.

TEMPERATURE TABLE NO. 1.

Day.	105° F.	104° F.	103.5° F.	103° F.	102.5° F.	102° F.	101.5° F.	101° F.	100.5° F.	100° F.	99.5° F.	99° F.	98.5° F.	98.10° F.
1														
2			1		2				1					1
3		1			2		2							
4				1	2			2		1		1		1
5						2		3				5		1
6						1	1					3		2
7				1					2			2		3
8	1											2		1
9												1		3
10												1		1

TEMPERATURE TABLE NO. 2.

2d Day.		3d-6th Days (Inclusive).		7th-10th Days (Inclusive).	
Temperature.	Number of cases.	Temperature.	Number of cases.	Temperature.	Number of cases.
98° F.	1	98° F.	4	97° F.	1
100.6° F.	1	99° F.	9	98° F.	8
102.6° F.	2	100° F.	1	99° F.	6
103.8° F.	1	101° F.	8	100° F.	2
		102° F.	7	103.3° F.	1
		103° F.	1	105° F.	1
		104° F.	1		

Total number of cases = 54.

It is obvious that after the fourth day there are only eleven cases with temperature over 99° F., whereas there are twenty-five cases with temperature of 99° F. or less. Before and including the fourth day, fifteen cases had temperatures above 99° F., and only three 99° F. or less. Consequently, since most of the patients have come under our observation after the acute stage has begun, the charts in the hospital have shown only the end of the temperature curve. Many of the cases have had continued low temperature, varying from just above normal to 99° F. or 99.5° F. for several weeks. Possibly there is a slight relationship, at least in point of time, between the onset of paralysis and the fall of temperature. This is not especially definite, nor does there seem to be any particular reason

why it should be. A somewhat remarkable feature of the temperature in many cases is the fact that its presence is shown only by the thermometer. The patients do not seem feverish. The typical end of the temperature curve is a sharp lysis, lasting usually twelve to twenty-four hours. Some cases have a much slower fall, and the temperature abates very gradually through two, three, or more days. In these cases it occasionally runs on for two or three weeks at just above normal. All the temperatures were taken by rectum, however, so that a curve in the smaller children continuing between 99°, 99.5°, or 100° F. is hardly to be considered an abnormal elevation.

II. Cases without Prodromata in Which Paralysis is the First Symptom.—A glance at text-figure 1 will show that in the seventy-two cases analyzed, only $4\frac{1}{5}$ per cent. began with paralysis. In these instances the children have been brought to us on the first or second day of the disease. Usually they have the aspect of patients who have begun in the ordinary way, for in almost every case the child has been found in a drowsy, irritable state, and sometimes has vomited within twenty-four or forty-eight hours after the appearance of the muscular weakness. In one instance, the typical tenderness on spinal flexion developed after a day or two and persisted rather longer than usual. In general, therefore, these cases do not differ essentially from the more common type of the disease. They are a most significant group, however, from the therapeutic point of view. With them there has been no warning, and checking a possible advance of paralysis is the only hope (case 7, page 133).

III. Cases with Remission of Symptoms and Delayed Paralysis.—In the prodromal period described above, the occurrence of remission in the symptoms was discussed. A somewhat similar phenomenon sometimes appears in the acute stage of the disease, but very rarely. Thus a child will become less drowsy rather suddenly and, after seeming to be better for a day or two, again become irritable and sicker than before (case 8, page 134). In one case in which the spinal fluid had shown marked improvement, and the clinical symptoms were distinctly better, there was a sudden change late in the disease. The globulin content of the spinal fluid rose to double plus and at the same time the patient became highly nervous,

trembled, and had almost a spastic condition of the muscles of the arms. A Babinski reaction was also present. Subsequently the case improved again and left the hospital with only a slight residual paralysis.

IV. Cases with Deep Stupor.—An important clinical type of the disease, about which a good deal of confusion in nomenclature exists, still remains to be considered. The term "cerebral" or "encephalitic" was applied by Strümpell, Wickman, Müller, and others to describe those few rare cases which have had spastic paralysees, dependent upon upper motor neurone lesions. Recently, however, there has crept into the literature a confusing use of the words cerebral and encephalitic. They have been applied rather loosely to cases of poliomyelitis which have had marked disturbance of the sensorium. In view of the accompanying paralysis, these cases properly belong to the bulbospinal group. The only clinical evidence to indicate that cases of the type about to be described are poliomyelitis is the paralysis, which is usually of the flaccid or lower motor neurone variety. Ultimately it may be proper to consider that profound disturbances of the sensorium in this disease are due to lesions of the silent brain areas, but we have no anatomical evidence as yet for this assumption. Consequently, if an anatomical classification is accepted for the disease as a whole, these cases must be grouped primarily according to the anatomical lesion indicated by their paralysees. Usually this is bulbar, but it may also be spinal. Profound stupor, however, is such a striking feature that it must be recognized in any system of classification; but in relation to a primarily anatomical grouping, it can merely be placed as the sub-heading of a clinical variety. Therefore it is simplest to consider these as cases of bulbospinal poliomyelitis with profound stupor.

In its manner of onset, this clinical sub-group of the disease does not differ materially from the usual type. If there is any difference, it is that drowsiness preponderates in the early days and gradually deepens into stupor. In one case this process occupied nearly a week. The patients, of whom we had four, were brought to the hospital in varying degrees of stupor. They may lie in a sort of coma vigil, the head and eyes drawn to one side, and the

eyes wide open and expressionless. The face has a peculiar waxy mask-like immobility, although no seventh nerve paralysis exists. The head is drawn back a little, and from time to time an expression of annoyance, almost distress, crosses the features. Now and then a faint twitching or tremor passes over an extremity or the whole side. Yet, despite the coma-like condition, the patient can be rather easily roused by handling or prodding. There is almost immediate response and objection in the manner which has been so often seen in the other cases of this extraordinary disease, a displeased, irritated whine, and a vexed shrugging movement of the shoulder forward and upward, conveying quite distinctly the child's wish to be let alone (figure 15). Other cases of this kind may show a more stuporous condition with partly closed eyelids beneath which moves a slowly rolling eyeball. These individuals may or may not have retraction of the head, and lie prostrated and somnolent. They bear a strangely similar resemblance to patients with tuberculous meningitis. Like the coma vigil cases, however, they can also be rather easily roused by manipulation or prodding, and lapse as quickly again into stupor when undisturbed. Still other patients behave as though heavily drugged, and carry out sharp commands by slow, lazy, intensely apathetic motions.

An elevation of temperature is usually present in these cases. It seems to bear little relation, however, to the degree of stupor. Thus, one individual with a temperature of 99.6° F. was far more stuporous than another with a temperature of 104.2° F. Furthermore, the first case had a rise of temperature during the period of awakening from the stuporous condition.

As a rule, the stupor clears with considerable rapidity, after a duration of from three to six days. In one instance the patient after four days awoke as though from sleep, looked about in a bewildered fashion and then said she wanted to go home. The other cases regained normal mental condition more slowly, but the process occupied only a few hours.

To differentiate these cases from tuberculous meningitis is sometimes almost impossible. If paralysis exists when the case is first seen, one inclines more perhaps to the diagnosis of poliomyelitis, but in the preparalytic stage there is often nothing to give a differ-

ential clue. It is in these cases that the want of a specific reaction for poliomyelitis in the spinal fluid is most keenly felt, especially if the first search for tubercle bacilli is negative. The negative von Pirquet reaction, which occurred in our cases, is also a distinct help towards ruling out tuberculosis. All of our cases of stupor have had lesions in the cord at high levels. Three had palsies of the facial nerve, and one had upper extremity paralysis. This case and one of the bulbar cases also had slight, transient lower extremity involvement. One individual had lost only the power to look down and to converge. The extraordinary thing about the inability to converge was that both internal recti acted well in conjugate lateral deviation of the eyes.

From these various general clinical expressions of the acute stage of the disease, the patients recover more or less in the same way. As the acute symptoms pass, and the child begins again to take note of its surroundings and becomes cheerful, the paralyses, although previously observed, assume greater significance. Some cases show improvement more slowly than others. The children, as a rule, first become less drowsy and irritable. They will put out a hand into an offered palm or answer faintly in monosyllables. With some individuals, this change of interest comes toward the end of the first week; with others, not for two, three, or sometimes four weeks. This awakening, as it may be termed, is usually the beginning of improvement. From this time on, the convalescence is steady. Some cases seem entirely well in a day or two, while others gain in health and cheerfulness more slowly for a week or longer.

It is in this period that certain interesting psychic phenomena have been observed. Some children have been sullen and unwilling to play or be played with. In many cases fretfulness and irritability have persisted, and these children cry on the slightest provocation and sob for a long while afterwards. Still other individuals, fewer in number, present a peculiar emotional instability. Often such children begin to cry for no apparent reason and a moment later, if their attention is diverted by some trivial occurrence, begin as suddenly to laugh and giggle in a typically hysterical manner (case 9, page 137).

In general, however, the children rapidly regain normal psychic

poise, although in some cases there is a nervous apprehension which lasts for weeks. Furthermore, they soon lose the tired, wilted look and become rosy and fat. It is often surprising to see how quickly a very sick child looks well again, and sits up in bed smiling and happy. In such cases, only when the coverlet is drawn back and the helpless extremities appear, does one fully realize what a damaging blow the infection has dealt.

THE PARALYSES.

We have endeavored to show that with the advance in the knowledge of poliomyelitis, there has been a gradual shifting of the point of view from which the disease is approached. While not many years ago paralysis was regarded as the essential feature, the one important element of the picture, and the accompanying general symptoms were more or less completely disregarded, the tendency of present medical thought is towards the conception of poliomyelitis as an acute infectious disease which often leaves in its wake a variety of disabling end results.

Poliomyelitis, then, is not synonymous with paralysis, and the rôle played by this secondary symptom in the clinical history of the disease is becoming relatively less important. The new knowledge of abortive cases and of the probable infectivity of the prodromal stage teaches us that satisfactory control of poliomyelitis must, in the end, depend on the recognition of cases wholly apart from the paralysis. Where, formerly, paralysis was the one diagnostic criterion, its onset may now be only confirmatory evidence of a previously suspected diagnosis. If, however, paralysis is no longer considered to be the whole disease, it is unquestionably the greatest part of it. Now, and until the complex problems of diagnosis and therapeutics are finally solved, the feature of the disease which will form the center of the picture will be the paralysis, its prominence being dependent in large part on our imperfect methods.

The paralyzes, while not considered of such primary diagnostic importance as they used to be, throw a good deal of light on the extent of the pathological process in the individual case; they offer valuable diagnostic information in atypical cases; and they so frequently dominate the clinical picture and constitute the whole of the

problem for the attending physician that they deserve a somewhat detailed study. The relation between the clinical signs and the pathological process in the nervous system is not an especially close one. The clinical signs depend, of course, on the pathological lesions, but they do not represent with any accuracy the extent of the lesions. Post-mortem examination shows a pathological involvement of the gray matter over an area which is usually much more extensive than that represented by the muscular paralysis, and an involvement of the white matter which has usually called forth no symptoms at all. That large areas of gray matter can be affected without producing clinical symptoms may depend on too slight involvement, or on the fact that most muscles derive nerve fibres from several levels of the cord, and their functional capacity is thus endowed with a large factor of safety. The absence of clinical symptoms depending on lesions in the white matter, even when these are extensive, is a necessary result of the coëxistence of lesions of the anterior horn cells, for in the presence of a widespread lower motor neurone disturbance, evidence of upper neurone involvement will usually be masked. Thus, even when the typical anterior horn lesions of poliomyelitis are associated with a diffuse inflammatory reaction through the white matter of the cord, the predominating part of the clinical picture will be the flaccid paralysis of lower neurone destruction.

One of the most characteristic of the general features of the paralyzes in poliomyelitis is their peculiarly unsystematic distribution. Thus, in one case both legs are paralyzed, in another one leg and one arm, in a third case there is a peroneal associated with a deltoid or perhaps a facial paralysis, bizarre combinations with no apparent anatomical or physiological basis. There is, of course, a definite tendency for certain parts of the cord to be involved more frequently than others. Lesions in the lumbar enlargement are by far the most common, and next in frequency are lesions in the cervical enlargement with resultant leg and arm paralyzes. The fact that certain parts of the cord are more apt to be affected than others probably depends largely on the blood supply. The primary reaction to the virus being a perivascular infiltration, the extent of the inflammation will vary more or less with the size and number

of the blood-vessels. That the lesion is most marked in the anterior horns of the gray matter is due to the fact that here, about the large motor nerve cells, the circulation is more abundant than in the posterior horns or in the white matter. Again, that extensive lesions are more frequent in the cervical and lumbar enlargements than at other levels of the cord is explained on an anatomical basis by the fact that the blood-vessels are largest and most numerous at the levels from which the great nerve plexuses arise. Other characteristics of the distribution of the paralysis, the affection of individual muscles, the involvement of various groups of muscles, as well as the sparing of others, depend on the pathological lesion being located in the cord itself and not in the peripheral nerves.

In other ways, too, the clinical picture and the pathological anatomy of poliomyelitis may be harmonized. Thus some paralyses are complete and permanent. In these there must have been an actual destruction of all or nearly all the nerve cells from which the affected muscles received nerve fibres. At other times one sees what is a weakness rather than a paralysis. This may be due either to an interference with nerve cell function by the pressure of exudate and edema without actual destruction of cells, or, it may be due to the destruction of a limited number of cells. As Bing³⁰ says, "Anterior root lesions, . . . unless very extensive, merely weaken and do not completely paralyze the muscle, owing to the fact that, as a rule, the muscle is innervated from several roots." Another class of paralyses is characterized by its transient nature. Either they last only a day or two, or they may persist for several weeks and eventually disappear. These are apparently the result, not of a destruction of nerve cells, but of a profound disturbance of function by toxic influences, or by the pressure of exudate, edema, and hemorrhage. When the disease ceases to progress, and the reparative stage with absorption of exudate begins, there is a relief of pressure, and the cells are enabled to resume their normal function.

The general manner of the onset of paralysis has already been considered. Some cases pass through a short period of muscular weakness corresponding to the beginning of the pathological

³⁰ Bing, *Compendium of Regional Diagnosis in Affections of the Brain and Spinal Cord*, translated by Arnold, New York, 1911.

changes, but the onset is usually sudden and swift, and the damage is quickly complete. The history is apt to state that paralysis came on over night, but occasionally one obtains more accurate evidence as to the possible rapidity of onset. One of our patients, an adult twenty years old, after several days of prodromata, went to sleep at half past twelve in the afternoon. Until then she had moved her legs normally. At two o'clock, one and one half hours later, she awoke and found her right leg completely paralyzed. There was no progression of symptoms. The whole damage was done at one stroke. Other instances are on record of even more rapid onset. Not infrequently, however, the first paralysis is followed at an interval of hours or days by further advances, and in one typical class of cases the process, as we shall see, sweeps like a wave over the whole spinal cord.

A discussion of the relative frequency with which different muscle groups are affected is of value only when a very large series of cases is analyzed. We, therefore, quote the following table from Wickman. It represents 868 cases seen by him in 1905.

1. One or both legs	353
2. One or both arms	75
3. Combination of arms and legs	152
4. Combination of legs and trunk muscles	85
5. Combination of arms and trunk muscles.....	10
6. Trunk muscles alone	9
7. Paralysis of "the whole body".....	23
8. Ascending paralysis	32
9. Descending paralysis	13
10. Combination of spinal and cranial nerves.....	34
11. Cranial nerves alone	22
12. Localization of paralyzes not given.....	60

In 43.69 per cent. of the cases, the paralysis was limited to the legs. One or both legs were affected in 85.64 per cent. of all the cases. A second table prepared by Lovett and Lucas³¹ gives similar information (see page 58).

In the following more detailed consideration of the various clinical types of lower motor neurone paralysis, we shall take them up from the anatomical point of view: first those dependent on cord

³¹ Lovett and Lucas, *Jour. Am. Med. Assn.*, 1908, li, 1677.

	Lovett and Lucas.	Duchenne, Seligmüller, Sinkler, Starr.
1. Both legs	130	84
2. Right leg	216	134
3. Left leg	239	133
4. Right arm	5	30
5. Left arm	5	23
6. Both arms alone	0	9
7. All four extremities	3	51
8. Arm and leg, same side	15	48
9. Arm and leg, opposite side	7	10
10. One arm, both legs	2	15
11. Abdomen with other paralysis.....	6	

lesions, next those with combined cord and bulbar lesions, and then those of purely bulbar origin. Finally, we shall describe the rapidly progressive cases as a class by themselves.

Spinal Paralyses.

Statistical studies show that in the great majority of cases of poliomyelitis, muscles of one or both legs are paralyzed, and that in nearly one half the cases the paralysis is limited to the legs. This corresponds to a pathological lesion in the lumbar enlargement of the spinal cord, and especially between the first lumbar and second sacral segments. Either one or both legs or any individual muscle may be affected. In the upper leg the quadriceps femoris is most often paralyzed, and in the lower leg the anterior group of muscles, the peroneals, the flexors of the foot, and the extensors of the toes, are most commonly involved. Extensive leg paralyses are of very frequent occurrence, but complete and permanent paralysis is not the rule. More often the flexors of the toes are spared, or if they are paralyzed immediately after the onset, they are usually the first muscles to show a return of function. On account of this relative immunity of the extensors of the foot and flexors of the toes, toe drop and contractures of the foot due to the unopposed pull of one group of muscles form a complication difficult to overcome. The paralyzed limbs are often cool to the touch and there may be a red-dish purple mottling of the skin. The feet especially are apt to be cold, and they may be covered with a cold sweat. In the paralyses of leg muscles, one sometimes sees, perhaps more often than else-

where, a remarkably late restoration of function after such a long period has elapsed that one has begun to fear that the paralysis was permanent. In our experience this has been especially noticeable in the flexors and adductors of the thigh.

Sphincter paralyses, dependent on lower cord lesions, are probably rare. Many cases have bladder disturbances, usually retention of urine, but the facts that this is transient, that it appears during the febrile period, and may begin before the onset of other paralysis, make it seem probable that this symptom is more often analogous to what is seen in other acute febrile diseases, rather than a result of cord involvement. The reports by other observers of more severe and more persistent sphincter disturbances make it almost certain that paralyses of the vesical sphincters do exist. A few of our cases have had to be catheterized, one during a period of several days. Bowel disturbances suggesting paralysis of the sphincters are much more unusual. In one case of an intelligent boy, it seemed that such a condition might be the cause of an apparent inability to control the bowels.

Next in frequency to paralyses of the legs are paralyses of the arms, depending on lesions in the cervical enlargement of the cord from the fifth cervical to the first thoracic segment. In our series of seventy-one ward patients, thirty had involvement of the arms, but, as is usually the case, the great majority of them had a leg paralysis also. In only four instances the legs were not affected. Two of these showed an arm paralysis only; one had, in addition, a transient facial, and one a transient diaphragmatic paralysis. Except where arm lesions form a part of a very general paralysis, they are usually unilateral (case 10, page 138). We saw but one instance of both arms being affected in the absence of any leg disturbance (case 9, page 137). There is a tendency, greater even in the arms than in the legs, for the paralyses to be limited to muscle groups. The shoulder muscles and more especially the deltoids are most apt to be involved. It is a generally recognized fact that the proximal muscle groups of the limbs, especially the upper limbs, are more apt to be paralyzed than the distal muscles, and that after paralysis, recovery is quicker and more certain in the distal than in the proximal muscles. The shoulders are paralyzed more

often and their recovery is less certain. Elbow, hand, and finger muscles are less frequently affected and show a greater tendency to regain their power. Here, in analogy with the legs, one finds the flexors of the fingers less often involved and more completely recovering than the extensors. Complete and permanent flaccid paralysis of the whole limb is unusual. A recovery of the flexors of the fingers almost always occurs. Transient weakness or a temporary paralysis of one or both shoulders, occurring as a part of a more general process, is often followed by complete return of function, but if the paralysis persists and becomes well established, it appears to have an unusually bad prognosis. No muscle seems to atrophy as fast as the deltoid. One rather peculiar symptom complex was observed in association with an arm paralysis. The child had at first a complete flaccid paralysis of the left arm, but it gradually abated, with return of power in the triceps and extensors of the fingers, and to some extent in the flexors of the fingers. With this was noted failure of the left pupil to dilate, narrowing of the left eye slit, hemicranial sweating, the left side of the face being nearly dry, and hemicranial vasomotor phenomena. These symptoms depended apparently on a lesion of the ciliospinal ganglion which is situated in the cord at the level of the eighth cervical and first thoracic segments. The flexor muscles which showed such a delayed return of power derive their nerves principally from the seventh and eighth cervical, and first thoracic segments (case 15, page 147).

When the pathological process is fairly definitely limited to the cervical cord, and the legs are not paralyzed, one often observes a tendency to hold the legs stiffly, an exaggeration of the knee jerks and ankle reflexes, and a peculiar transient spastic ataxia of the legs. These symptoms certainly suggest an upper neurone disturbance and it seems probable that they are due to an involvement of the pyramidal tracts or of Clarke's columns, where these pass through the cervical region.

Paralysis of the Diaphragm.—In the upper part of the cervical cord, in the third, fourth, and fifth cervical segments, and in general above the area from which the brachial plexus arises, lie the cells whose axones form the phrenic nerve and innervate the dia-

phragm. Owing to the fact that the phrenic nerve has a large number of roots, or to some peculiarly fortunate circulatory condition, comparatively rarely is this area so severely affected that the action of the diaphragm is interfered with. Among fatal cases, however, this is not true, for practically all cases that die of poliomyelitis, without complication, die of respiratory failure, and respiratory failure means paralysis of the intercostal muscles and the diaphragm. It is noteworthy that among our fatal cases the diaphragm was usually the last to be affected, paralysis of the intercostals often preceding it by many hours. Owing to the vital importance of the diaphragm, its paralysis makes a serious prognosis, but recovery not infrequently takes place. We have seen two instances of paralysis of the diaphragm lasting for over a week and ending in complete return to normal. In one case it was associated with a paralysis of both arms, and in the other with an extensive paralysis of neck, arms, abdomen, and legs. In neither of these children was there any marked respiratory disturbance, but the picture was perfectly typical. Respiration is wholly thoracic, and sometimes the accessory muscles of the neck come into play. The abdominal wall moves with each respiration, but instead of its normal inspiratory protrusion, there is the typical inspiratory retraction depending on the lax diaphragm. Firm pressure on the sides of the thorax fails to induce any abdominal breathing and causes rapid, labored respiration. A condition which may be temporarily confounded with diaphragmatic paralysis is the type of breathing sometimes seen in crying or sobbing children; respiration is usually irregular, thoracic, and associated with an inspiratory retraction of the abdomen. This, of course, passes off as soon as the child becomes quiet again (case 13, page 143; see also case 9, page 137).

Paralysis of the Intercostal Muscles.—The thoracic portion of the spinal cord, probably owing to its less abundant blood supply, is also comparatively rarely attacked, except in the overwhelming infections terminating fatally. In such cases there is usually a progression upward from the lumbar region or downward from the cervical region, and the paralysis of the intercostal muscles which follows the invasion of the dorsal cord is one of the factors that cause respiratory failure and death. While the majority of cases with

paralysis of the intercostals end fatally, we have seen three instances of recovery. In two there was an associated paralysis of both arms and legs, and in one other of legs and abdominal muscles. The two former still showed an intercostal paralysis on discharge from the hospital, several weeks after the onset of the disease. In the latter there was return of function. In a fourth, seen in the dispensary, the associated leg paralysis cleared up almost entirely, but a complete intercostal paralysis persisted. Atrophy of the chest muscles, narrowing of the chest, and protrusion of the abdomen make a peculiarly characteristic late clinical picture (see figure 16). In some cases there has been an association of intercostal paralysis with edema of the lungs. The presence of coarse, moist rales in both lungs in the absence of fever and of cardiac weakness, and in the absence of evidence of a bronchopneumonia at autopsy have made us feel that the process may possibly be one of vasomotor origin. The diagnosis of paralysis of the intercostal muscles is less simple than that of the diaphragm, for in many children the normal type of respiration is almost wholly diaphragmatic and suggestive of weakness of the intercostals. In typical paralysis of the intercostal muscles, respiration is wholly abdominal, the thorax moves very slightly, its movement on inspiration being downward and backward instead of forward and upward. There is sometimes a sucking inwards of the intercostal spaces and, in children with flexible chest walls, of the lower part of the chest on inspiration (figures 16 and 17). Pressure over the abdomen does not induce a thoracic type of respiration, but it causes rapid, labored breathing, an effect not produced by compression of the chest. Both intercostal and diaphragmatic paralyses predispose to the development of bronchopneumonia (cases 14, 15, and 16, pages 145, 147, and 149).

Paralysis of the Abdominal Muscles.—The statements of various observers show a great divergence of opinion as to the frequency of paralysis of the abdominal muscles. From a study of our own cases, we have felt that an accurate estimation of its occurrence is extremely difficult, for the diagnosis in an acutely sick child of a transient weakness or paralysis of the abdominal wall is by no means easy. It is readily simulated by soft, atonic abdominal muscles. Apart from these transient paralyses, which undoubtedly

occur frequently, and from the involvements in rapidly fatal cases, there is an interesting class of abdominal paralyses which usually persist. These consist in either local areas of weakness or general paralyses of the abdominal muscles on one or both sides. The external and internal oblique muscles and the transversalis are usually involved together. The rectus abdominis is usually spared. Sometimes all the muscular sheet outside the rectus is weak, and there is a bulging of the sides of the belly, but more often the paralysis is localized, and there is a circumscribed protrusion of the wall resembling an abdominal hernia. These paralyses are brought out more definitely by coughing, by crying, or by attempting to raise the body in bed (figures 18 and 19). Paralysis of the abdominal muscles may be the only symptom that persists.

Paralyses of the Neck and Back Muscles.—These have been most common in our patients in severe cases with extensive paralyses. In most of them there has been involvement of both arms and legs, associated with that of the back and neck. In one case, however, the only paralysis ever noticed was a transient affection of the neck (case 17, page 151). Müller calls especial attention to the great frequency with which paresis of the trunk muscles occurs early in the disease. We have found the back muscles very difficult to test early in the acute stage, for the children are often either so sick or have so much pain that they will not try to support the spine. Moreover, it is hard to distinguish whether weakness of the back is caused by a spinal cord lesion, or is merely a general symptom. In paralysis of the neck muscles, the child cannot support its head, which falls helplessly forward, backward, or to one side. Paralysis of the back muscles is either unilateral or bilateral. In the former case the child may be able to sit up, but the spine bends with its convexity towards the weak side. If the lesion is bilateral, the patient cannot support the trunk and it falls backward, sideways, or forward, so that the body doubles up on top of the legs. Except in a few very extensive cases, paralyses of the neck and back muscles tend to disappear early.

Bulbospinal Paralyzes.

It is probable that in most cases of poliomyelitis the pathological changes in the central nervous system are much more extensive than the clinical picture would lead one to suspect, and that scattered areas of exudate or hemorrhage are often present in the medulla and pons. In a smaller proportion of cases, but not infrequently, these lesions are large enough and so situated as to produce definite symptoms by involvement of the nuclei of the cranial nerves. Most commonly the cranial nerve affections form part of a general process in which the cord is simultaneously affected, but in a small proportion of cases the lesions of the cord are so slight as not to cause paralyzes, and the clinical picture is that of an acute bulbar paralysis. Most instances of combined spinal and bulbar paralyzes have extensive lesions involving legs, arms, and regions supplied from the bulb. Localized processes in the bulb and upper cord are much less frequent. Both where the cranial nerve affections occur alone, and where they are associated with spinal cord lesions, they are almost always unilateral. Bilateral bulbar involvements are extremely rare. Pathologically, as we have seen, the spinal and bulbar paralyzes have the same basis. Clinically, too, though they may present quite different pictures, it is impossible to separate them, as one type merges gradually into the other. The greater number of cases form the intermediate type with both spinal and bulbar paralyzes. In the Swedish epidemic of 1905, in which there were 685 cases, Wickman saw forty-two instances of combined spinal and bulbar paralysis. In the series of seventy-one cases which we have observed in the hospital there were, excluding the fatal cases, twelve of combined paralysis. The fact that our figures are proportionately nearly three times as large as Wickman's probably depends on the possibility of closer observation afforded in a hospital. Many of the cranial nerve paralyzes were comparatively slight and transient, so that constant watching of the children was necessary for their detection. It was quite noticeable that the bulbar paralyzes occurring in association with spinal paralyzes were much less severe and less likely to be permanent than when they occurred alone. As has been the experience of other clinicians, we have found the

facial nerve most often affected. A facial paralysis occurred in eight of the twelve cases with both cord and bulb lesions, but in almost all it was slight and disappeared quickly. In three of the four cases of purely bulbar paralysis, facial paralysis was present and improved very slowly. Either the whole facial nerve or one branch may be affected (figures 20, 21, and 22). Ocular palsies were present four times in the hospital cases. In agreement with the findings of Wickman and Müller, we have found an abducens paralysis to be the commonest ocular palsy. Paralysis of the ocular muscles was sometimes transient, but often apparently permanent. One case seen in the dispensary had at first a paralysis of the right leg and of the right external rectus. Four weeks after the acute onset of the disease the leg had almost completely recovered, and the only evidence of the disease was a well marked internal squint. It is perhaps worth considering whether slight attacks of poliomyelitis may not be a more frequent cause of strabismus than is usually recognized. More extensive palsies and even complete ophthalmoplegia externa have been reported by Wickman. Nystagmus was noted several times in the early part of the acute stage of the disease. The eye grounds were examined in many of our cases, but we found no abnormalities of the optic nerve. While Wickman has reported one instance of optic neuritis in an acute case, and Tedeschi (quoted by Wickman³²) found complete blindness and optic atrophy of the left eye in a chronic case, Müller failed to find any evidence of optic neuritis or choked disc in a large number of acute cases, and he believes that if either is found, the case is almost certainly not one of poliomyelitis. Disturbances of speech and phonation are quite frequently met with; the latter may be of all degrees from hoarseness or slight weakness to complete aphonia. They usually occur very early in the course of the disease and are transient, though we have seen them become progressively worse during the second week, and in one case the mother said that the child could not talk for nine days. Difficulties of deglutition are sometimes seen at the acute onset of the disease. This may be a transient symptom which lasts a day or two and consists only in an inability to swallow solids. In other patients, as

³² Wickman, *Die akute Poliomyelitis*, *loc. cit.*

in two purely bulbar cases to be described, there may be total inability to swallow, lasting for many days and necessitating feeding by gavage. While the situation appears at the time to be most serious, the tendency to recovery, according to our experience, seems to be much better than one would expect, and return of function may take place even after ten days of paralysis. In progressive fatal cases, a paralysis of swallowing is frequently seen. Disturbances of speech and disturbances of swallowing often occur together. With paralysis of deglutition may be associated a paralysis of the hypoglossal nerve. This is usually a unilateral lesion causing inability to protrude the tongue straight, but cases of bilateral involvement with complete inability to protrude the tongue are reported. In the two of our cases which had prolonged difficulty in swallowing, but which finally became normal in this respect, a persistent hypoglossal paralysis remained as a residual lesion. In a dispensary case, the only bulbar symptom remaining several weeks after the onset was a deviation of the tongue with an atrophy of one half of it. Atrophy of the intrinsic muscles of the tongue occurs quite rapidly after hypoglossal paralysis, but no interference with function is apparent. Evidence of involvement of the vagus nerve in poliomyelitis will be shown in the consideration of the progressive fatal cases. Two fatal cases showed spinal fluids which had an exceptionally high power of reducing Fehling's solution. One of these had also a glycosuria. It is possible that an inflammatory lesion between the nuclei of the eighth and tenth nerves had the same effect as the "sugar puncture" of Claude Bernard and caused a hyperglycemia.

One of the most interesting clinical forms in which poliomyelitis appears is that of the purely bulbar paralysis. Until Medin's publication, the relation of cases with affections limited to the cranial nerves to the typical poliomyelitic cases had not been definitely brought out. There is now, however, abundant evidence, both pathological and epidemiological, to show that many cases formerly called acute bulbar paralysis are, in fact, poliomyelitis with a localized lesion in the pons and medulla. This type of case is not especially uncommon. We have had four instances under observation in the hospital and have seen other acute cases in the dispensary.

We have already called attention to the fact that the paralysis in these cases tends to be more severe and more lasting, than are the bulbar paralyzes when they occur in association with general cord lesions. Except for the residual paralysis, however, the prognosis seems to be good. One might expect to see death from involvement of some of the higher centers, but this has not occurred in our experience, nor have we noted any tendency for a bulbar paralysis, once definitely established, to spread to the phrenic or upper cervical centers. Any of the cranial motor nerves may be affected, and the combinations in which various nerves are involved are numerous. As usual, the facial nerve is most frequently paralyzed, and a facial palsy may be the only evidence of the disease. In one case we found a paralysis of the sixth, seventh, and twelfth nerves, and complete inability to swallow. After being fed by gavage for five days, the patient regained the power of deglutition, and later the strabismus disappeared. The facial and hypoglossal paralyzes persisted. In another bulbar case the only evidences of paralysis were inability to swallow, deviation of the tongue, and hoarseness of the voice. Feeding by gavage was continued for ten days. During this time the patient's general appearance became worse and his hoarseness was suddenly succeeded by almost complete aphonia. At this time, when he was very weak, sixteen days after the onset of the disease, he suddenly showed some return of the ability to swallow, and small amounts of food were given by mouth. Recovery of voice and deglutition proceeded uninterruptedly and the child was discharged perfectly well except for a residual paralysis of the right hypoglossus nerve. Quite characteristic of these high lesions has been an ataxia which has become evident when the children were well enough to begin to use their arms and legs. Knee jerks and Achilles reflexes are greatly exaggerated, and the legs may be held stiffly. The gait suggests somewhat a spastic ataxia. The children have difficulty in balancing themselves, a well marked Romberg sign may be present, and they walk with rather stiff legs and feet spread wide apart. This is a temporary condition and, after they have been on their feet for a few days, the gait becomes normal, but the exaggeration of reflexes often persists much longer. The cause of this phenomenon is difficult to determine with accuracy.

It may be due to lesions in the cerebellum, in Clarke's columns, or in the pyramidal tracts as they pass through the bulb or upper cord (cases 18, 19, 20 and 21, pages 152, 154, 155, and 158).

Rapidly Progressive Cases.

From the pathological point of view, there is no reason for considering separately those cases of poliomyelitis in which there is a progressive involvement of the nervous system, for the lesions are in themselves similar to those found in other cases. Clinically, however, they have formed such a characteristic group in our experience that it is convenient to discuss them by themselves. They constitute the greater number of fatal cases. Many, if not all cases showing the symptom-complex known as Landry's paralysis, belong to this group of poliomyelitis. There are in general two ways in which death occurs in poliomyelitis. Either it is due to a complication, for the most part bronchopneumonia, arising in the already injured system, or it is due to paralysis of the muscles of respiration. We have seen that a patient can live and can even recover after a paralysis of either the diaphragm or the intercostal muscles. When both become paralyzed, respiration ceases and death ensues. The fatal issue depends not on the nature of the pathological process, but on its location. If certain vital parts are spared, the extent of the disease seems to have little effect on the general health. The essential feature of death in poliomyelitis is that it is a respiratory and, in a way, a mechanical death. In most other acute infections we regard death as being due to a toxemia. The body becomes overwhelmed by a poison which interferes with its functions, until finally some organ, usually the heart, weakens and fails. That a toxic substance plays some rôle in the destruction of the nerve cells in poliomyelitis cannot at present be disproved, but that anemia and pressure play a very striking part is certain. The typical clinical picture, moreover, as we have seen it in the severe, fatal cases, is not that of a patient dulled by a general toxemia, but of one with a clear, alert sensorium, fighting for every breath until he is literally suffocated. This does not mean, of course, that there are not other cases of poliomyelitis which pass from somnolence to stupor and die without regaining consciousness. Whether, in such

cases, the mental condition is a toxic affect, or whether it, too, may not also depend in large measure on the mechanical action of the poliomyelitic process involving the centers of consciousness, is a question which we feel cannot be answered at present, but there is much to make the latter suggestion seem not improbable.

The average mortality for different epidemics of poliomyelitis varies between 10 and 20 per cent. Of the cases which we have followed in the hospital ten, or about 14 per cent., died. In three of these death was due to bronchopneumonia supervening on an extensive paralysis which included either the intercostal muscles or the diaphragm. In seven cases there was no evidence of any complication, and death was the result of poliomyelitis with paralysis of the respiratory muscles. We have seen that in most cases of poliomyelitis the paralysis is sudden and severe, and reaches its maximum in a comparatively short time. The same may occur in fatal cases. In one case seen outside the hospital, there was a sudden overwhelming paralysis which caused death within five hours after its onset. In the majority of cases, however, after the acute onset of a more or less widespread paralysis which usually involves either the intercostals or the diaphragm, there is an interval of a few days with a pause or a slower progression in the paralysis until there is a weakening and a final cessation of respiration.

The typical Landry's paralysis is an ascending paralysis involving first the legs, then the intercostals, arms, neck, and diaphragm. In most cases the picture is not quite so clear cut, for the primary paralysis is more extensive and involves both arms and legs, but in six of the seven uncomplicated, fatal cases, the intercostals were first affected, and death followed the failure of the diaphragm. In the three cases which had a terminal bronchopneumonia, however, there was a primary paralysis of the diaphragm. The majority of the uncomplicated cases conformed closely to the ascending type of Landry's paralysis. One only was of the descending type with early paralysis of the diaphragm and terminal involvement of the intercostals.

In the fatal cases, then, as well as in those that recover, there is usually a pause after the acute onset of the paralysis. There may be one or two days without any definite increase in paralysis, but it

is frequently noticeable that the children are not doing so well as those who will eventually recover. Often the respiration is more rapid and a trifle more difficult than the degree of paralysis warrants. They are frequently unusually nervous, excitable, and irritable. Then the paralysis may begin to increase. A laryngeal disturbance with hoarseness, aphonia, or difficulty in swallowing may be the first evidence of the spreading lesion. A weakening of the remaining muscles of respiration soon follows. If the intercostals are still active, the movement of the chest becomes less marked. If the diaphragm has hitherto been intact, its movement, as represented by the abdominal wall, becomes weaker, or there is an asymmetrical movement suggesting a paralysis of one side of the diaphragm. Respiration becomes more rapid, more labored, and perhaps irregular. Instead of the normal smooth, rhythmic cycle, there may be a sharp, jerky, forcible movement of the abdominal wall on expiration. Pressure over the abdomen causes distress and interference with breathing. The alæ nasi dilate with inspiration, and the accessory muscles of respiration in the neck begin to come into play. As the diaphragm weakens, the neck muscles become more and more prominent until it seems as if the whole work of breathing depended on them. The sternomastoids pull on the clavicle, and in one case they caused a partial dislocation of the sternoclavicular joint with each inspiratory effort. The head is thrown back, and with every breath the lower jaw is pushed forward and downward in a gasping attempt to get air. Occasionally, there is typical Cheyne-Stokes respiration, probably dependent on a lesion of the nucleus of the pneumogastric nerve. Meanwhile the lungs may have remained perfectly clear until the very end, or a few hours before death coarse, moist rales may accumulate, an edema suggesting vasomotor paralysis. The heart is sometimes strong and regular until it stops. Usually it continues beating for a considerable period after the cessation of respiration. Heart sounds have been audible for as much as five minutes after breathing stopped, and electrocardiographic records showed that stimulus formation continued for twenty-three minutes after cessation of the sounds. Several times a characteristic arrhythmia has set in for the last few hours of life. The heart beats perfectly

regularly and then suddenly changes its rate; jumps from perhaps one hundred per minute to one hundred and forty, remains regular at this rate for a few beats and then, possibly with a few interpolated irregular beats, drops back again to its former slow rate and regular rhythm (figure 14). In one instance the rate dropped from eighty-four to thirty-six and then varied between these two. This rhythmic arrhythmia may persist or it may give way to a regular rhythm and return later. It is apparently a so-called sinus-arrhythmia, and probably represents a vagus nerve disturbance. Records taken with the electrocardiograph show a condition which is quite similar to that described by Luciani as occurring in the heart of the asphyxiated frog. It is interesting that in one case, in association with the institution of artificial respiration and a lessening of cyanosis, the irregularity of heart action completely disappeared.

Fever was present in all of the fatal cases when they were admitted to the hospital. In most of them it varied between 101.5° and 103.5° F. In two cases it did not run over 100° F. The course of the temperature, however, was not unlike that seen in cases ending in recovery. In general, it was highest on the first day of paralysis, lower on the next day, falling approximately to normal by the day of death. There has been no tendency for the usual fever curve to be prolonged in the fatal cases.

The most remarkable feature of our fatal cases was the condition of the sensorium. Three of our patients were so young that observations on their mental state were not of value, but four, between the ages of three and a half and ten years, showed a very interesting and comparatively constant picture. We have already called attention to the apparent absence of toxic effects in many cases of poliomyelitis, and this is nowhere more strikingly illustrated than in these severe, fatal cases. During the prodromata, and often during the acute onset of the early paralysis, the children may be sleepy and drowsy in the manner which is characteristic of so many cases. This condition is, however, apt to be mild and transient and it is often soon replaced by a clear mental state. With the onset of respiratory difficulty, it seems almost as if the children were suddenly awakened and made to realize the struggle before them. Little children seem to age in a few hours. One sees a heedless,

careless, sleepy baby become all at once wide awake, high strung, alert to the matter in hand, and this is, breathing. The whole mind and body appear to be concentrated on respiration. Respiration becomes an active, voluntary process, and every breath represents hard work. The child gives the impression of one who has a fight on his hands, and who knows perfectly how to manage it. All he wants is to be left alone, not to be interfered with, to be allowed to carry out his fight on his own lines. Instinctively he husbands his strength, refuses food, and speaks, when speech is necessary, quietly and with few words. One little child of four, so helplessly paralyzed that she was unable to move, but with a mind that seemed to take in the whole situation, said to the nurse clearly but rather abruptly, between her hard-taken breaths, "My arm hurts"; "Turn me over"; "Scratch my nostril"; and then when the doctor approached, "Let me alone, doctor!"; "Don't touch my chest." Pressure on the chest, tight neck bands, anything that obstructs easy respiration is immediately resented. The child demands constant attention, is irritated unless everything is done exactly as he wishes it, and often shows an instinctive appreciation for some especially efficient nurse. He is nervous, fearful, and dreads being left alone. The mouth becomes filled with frothy saliva which the child is unable to swallow, so he collects it between his lips and waits for the nurse to wipe it away. He likes to have his lips wet with cold water, but rarely attempts to take it into his mouth, for he knows he cannot swallow it. During the whole course it is remarkable that cyanosis is absent. There is a little bluish tingeing of the lips and tongue, but much more distinctive is the pallor, which is sometimes striking. Sweating is profuse. Then, as respiration gets weaker, the mind becomes dull, and with the occasional return of a lucid interval, he gradually drifts into unconsciousness. An hour or more later respiration ceases. This peculiarly alert, keen mental state has been much less noticeable in small babies. They tend to be dull and drowsy most of the time; but in the older children this alertness has been such a characteristic feature of the fatal cases, that we almost preferred to have a child brought to us in a stuporous condition, rather than with a mind whose nervous acuity seemed due to a perception of impending danger (cases 22, 23, 24, 25, 26, 27, and 28, pages 160, 161, 163, 165, 168, 171, and 173).

Cases 29 and 30 (pages 174 and 176) died of a terminal bronchopneumonia.

Cerebral Type.

In a masterly article, Strümpell,³³ in 1885, first called attention to the analogy between certain forms of cerebral paralysis in children and poliomyelitis. For this group of cases he suggested the name polioencephalitis. In the twenty-four instances analyzed by him, the disease occurred in nineteen cases below the age of four. None of the children were over six years old. In general the onset was sudden, and there was an initial stage with fever, vomiting, and convulsions. The last was the most frequent initial symptom. A prodromal period seemed to be wholly wanting in certain cases; in others it lasted two or three days, and in some instances it extended over several weeks, during which there were constant convulsive seizures. After the prodromal period, the child was found to have a hemiplegia. The paralysis usually began to improve shortly, but a limp and, more often, disturbed function of the arm usually persisted. The face was less often and less severely affected than the arm or leg. Strabismus occurred in several cases. Monoplegic paralysis of arm or leg, or even an ataxia without actual paralysis, was occasionally the only disturbance. There was no atrophy of the muscles or reaction of degeneration. The reflexes were usually exaggerated. Athetosis, epilepsy, disturbances of speech and intelligence sometimes occurred as sequelæ. In his discussion of the analogy between this type of case and poliomyelitis, Strümpell says it is noteworthy that, "in both diseases the chief seat of the lesion is the gray matter, in one case in the gray matter of the anterior horns, and in the other in the corresponding portion of the cerebrum, the cortex."

When, in 1898, Medin³⁴ published the first comprehensive description of the epidemic form of poliomyelitis, he was able to include the reports of three cases with fever, somnolence, convulsions, spastic hemiplegia, and exaggerated reflexes, and of a fourth in which apparently both cerebral hemispheres were affected. In

³³ Strümpell, *loc. cit.*

³⁴ Medin, *Arch. méd. des enfants*, 1898, i, 257, 321.

two instances the sixth cranial nerve was also paralyzed, and this bulbar affection formed a connecting link between the spinal and encephalitic forms. This, and the occurrence of both spinal and cerebral paralyses in the same epidemic convinced him that they had a common cause.

Harbitz and Scheel described a case of acute encephalitis in a man thirty-nine years old. After an onset with headache, fever, and sweating, stiffness of the neck, vomiting, and convulsions set in. The patient was unconscious. All the limbs could be moved, but they were rigid, and the patellar reflexes were exaggerated. The tongue deviated to the left. The man died, and at autopsy macroscopic circumscribed encephalitic or meningoencephalitic lesions were found in the right temporal lobe and in the gyrus fornicatus on both sides. Microscopically the lesion was more widespread and involved the central ganglia and medulla. In the spinal cord there was meningeal infiltration, but there was no definite inflammation of the anterior horns except in the cervical region. "Histologically, the inflammatory process resembled acute poliomyelitis in all its details."

These three sets of observations, the first two of which at least are of historical importance, give a fairly definite outline of what is to be considered as the cerebral form of poliomyelitis. The classification of this type rests, as far as possible, on an anatomical basis. These are cases in which the predominating pathological lesion is in the brain itself, and in which the clinical manifestations suggest a disturbance of the upper motor neurone. The identity of the cerebral form of the disease has, as we have already stated, been greatly obscured by the tendency of many authors, especially English and American, to use inaccurately the terms cerebral or encephalitic. The frequent application of these terms to cases which show certain general symptoms, such as stupor, delirium, or meningism, has led to much confusion. That the term cerebral as applied to this disease has reference to the supposed anatomical seat of the lesion, and that the cases form a fairly definite clinical group, is shown by a glance at the historical basis on which the classification has developed. It will often, however, be difficult to draw a hard and fast line between what shall be called spinal and what

cerebral cases. Clinically as well as pathologically, the types must merge. It would seem best to be governed by the most prominent symptoms. A case with a spastic hemiplegia would be classed as cerebral, even if it were associated with a slight nuclear paralysis of the sixth nerve; and a case with flaccid facial and arm paralysis would certainly be of the bulbospinal type in spite of showing a somewhat ataxic gait and exaggerated knee jerks, which might point to upper neurone lesions. After all, classification is purely artificial, and the real reason for making a separate class of these cases is to call attention to the fact that the virus of poliomyelitis may cause symptoms of cerebral rather than of spinal origin.

In spite of the acknowledged fact that small scattered lesions are not uncommonly encountered as autopsy findings in the brain in poliomyelitis, the occurrence of extensive cerebral involvement has rarely been demonstrated. Moreover, the final proof that typical cerebral paralyzes may have the same etiology as poliomyelitis can be obtained only experimentally. As yet the virus has not been shown to exist in typical polioencephalitis by transference of the disease to monkeys. Anderson and Frost,³⁵ however, report that the blood serum of a patient who had a paraplegia of the legs, at first flaccid, but in a few days becoming spastic, was able to neutralize active virus. It is rather remarkable, too, that, in spite of the fact that monkeys are usually inoculated intracerebrally, the paralyzes are always spinal and not cerebral. The evidence, however, of the identity of the two diseases is still largely clinical and consists in the finding of cerebral cases during the course of typical epidemics, and of finding both flaccid and spastic paralyzes in the same patient. Thus both Möbius³⁶ and Hoffmann³⁷ saw instances of flaccid and spastic paralysis occurring in members of the same family. Much more striking, however, than that a number of such suggestive instances have been observed, is the fact that, with the tremendous increase in the incidence of poliomyelitis during the past few years, and with its more general recognition, there has not been a corresponding increase in the number of true cerebral cases reported.

³⁵ Anderson and Frost, *Jour. Am. Med. Assn.*, 1911, lvi, 663.

³⁶ Möbius, *Schmidt's Jahrb. d. ges. Med.*, 1884, cciv, 135.

³⁷ Hoffmann, *München. med. Wchnschr.*, 1904, li, 225.

The majority of those reported as cerebral or encephalitic cases do not conform with the anatomical standard that has been laid down. Wickman, who has probably had a wider experience than anyone, states that during the great Swedish epidemic of 1905 he did not see any definite cerebral cases. Krause, in the Westphalian epidemic of 1909, saw one case with encephalitic features. Zappert, whose studies include 555 cases, saw only five instances of cerebral hemiplegia. Müller reports four cases with spastic paralyzes, but states that in none of them was there conclusive evidence that the condition was due to a polioencephalitis of the cerebrum. He believes that the symptoms depend rather on an involvement of the pyramidal tracts in the bulb or cord. One case only might have been due to a disseminated encephalitis,—a boy who developed a complete right-sided hemiplegia, and, in addition, a few days later, a left-sided spastic paralysis. Such a case would fall in line with the fact that the lesions of the brain as observed at autopsy are always bilateral.

In our own series of cases we have seen no example of a typical polioencephalitis. One case, however, showed a picture which was so suggestive of cerebral involvement that we are inclined to believe that it belongs to this group. A boy of four years and three quarters was taken sick with fever and vomiting. He became somnolent and when first seen was in an almost stuporous condition. The eyes showed a slight external strabismus on the left, and there was an inability to converge or to look downwards, which, according to our ophthalmological consultant, Dr. Schirmer, depended not on a lesion of the eye muscle nuclei, but on a higher coördinating center. There were no other paralyzes. The Oppenheim sign was positive on both sides, and the Babinski reaction was suggestive. As the child came out of his stupor, the knee jerks became exaggerated, and a marked ataxia of arms and legs was observed. The diagnosis in this case was made more certain by the fact that the child's infant cousin, who had been in bed with him during his sickness, later developed a typical poliomyelitis (case 31, page 177).

During the past season, we have seen but one case of cerebral hemiplegia in a child. The conformity of the history and of the course of the process in this instance to the cases cited by Strümpell

were quite striking, but the chief interest in the case lies in the fact that, while occurring at a time when there was much poliomyelitis in the vicinity, we have good evidence in support of a different etiology. The child, a little girl of three years, hitherto perfectly well, was noticed to be feverish and out of sorts on the morning of October 4th. She played during the day but in the evening she had high fever, reaching 105.2° F., and after taking some milk, vomited. Shortly afterwards she was taken with convulsions which were so prolonged and so severe that the attending physician gave her some chloroform. When she quieted down, her left arm was found to be limp. The next day a left-sided facial paralysis, with paralysis of the left arm and leg, was observed. Improvement began quickly, especially in the leg. When seen by us, thirteen days after the onset, her left leg was practically well, but a well marked facial paralysis persisted, and there was ataxia and incoördination of the left arm. The knee jerks and Achilles tendon reflexes were active on both sides; there was no Babinski reflex and no Oppenheim sign. The eye grounds were negative. Lumbar puncture was done and the spinal fluid found to be absolutely normal. After a little over three weeks, a specimen of blood was taken, the serum mixed with active virus of poliomyelitis (0.1 of a cubic centimeter and 0.3 of a cubic centimeter in two experiments), the mixture incubated and injected into two monkeys. Both monkeys became sick at about the same time as the controls, and at autopsy showed lesions characteristic of poliomyelitis. The serum had no power of neutralizing the virus, and the case was, therefore, probably not one of poliomyelitis. The many features which this case has in common with those described as polioencephalitis, and its occurrence in association with other typical cases of poliomyelitis, made the test of considerable interest.

ABORTIVE TYPE.

The term "abortive" was applied by Wickman to those instances of poliomyelitis which do not develop paralysis. Certain border line cases with slight weakness but no true paralysis, Müller has called "*lavierte*" or "embryo" types. This seems perhaps an unnecessary distinction, especially in view of the new conception of the disease as a general systemic infection. As long as the cerebro-

spinal tract was held to be the only seat of the malady, and paralysis was considered its usual manifestation, the word "abortive" for unparalyzed cases was entirely satisfactory; but since recent developments in the pathology of poliomyelitis have demonstrated a general disease affecting the lymphatic apparatus and many of the parenchymatous organs as well, there is some chance of a misunderstanding of the term. The case which fortunately escapes paralysis is as much a true example of acute poliomyelitis as the paralytic, and as a source of contagion it is more dangerous. Indeed there are certain scattered facts gleaned during the past decade by clinicians and laboratory workers which, when brought together to bear upon the question, indicate that the non-paralyzed cases are possibly the more usual, and that the paralyzed ones are less frequent forms of the disease. Wickman, for example, found in his large epidemiological studies that the abortive cases represented from 25 to 56 per cent. of the total incidence of the disease, but he states that these figures are probably too low. Müller also believes such figures to be too low. He considers that the unparalyzed cases considerably outnumber the paralyzed. This view receives striking support from the recent demonstration of typical visceral lesions indicating that there is a general systemic infection. With such pathological findings, symptoms such as fever and malaise are reasonably to be expected. It may be that the abortive cases are those in which this general process is present, but in which the nervous system has been spared. Netter and Levaditi³⁸ have demonstrated that the serum of abortive cases neutralizes the virus *in vitro*, just as does the serum of the patients that develop paralysis. It is quite probable that in the past many of the abortive cases have been unrecognized and in certain cases at least, the apparent immunity of adults may be dependent upon such a previous, unrecognized attack. That the neutralizing substance in the blood may persist for a long period following an attack of the disease, and probably immunity be present as well, is shown by the case of a man who had been paralyzed thirty years before and whose serum still protected a monkey from the virus.

Since there is no essential difference, except in degree, between the

³⁸ Netter and Levaditi, *Compt. rend. Soc. de biol.*, 1910, lxviii, 617.

severe and abortive cases, a description of the symptomatology of the abortive type as a clinically separate group seems unnecessary. Wickman has attempted to classify the various forms of the abortive type, and groups them under four headings, thus:

1. Cases which run the course of a general infection.
2. Cases in which meningeal irritation is especially marked (meningism-like).
3. Cases in which pain is very marked (influenza-like).
4. Cases with gastro-intestinal disturbance.

Such a classification, based purely on symptoms, is perhaps as good as any other, but it is a grouping into which all cases of the disease can well be put during the time when diagnosis is especially difficult and really important. Actually there is little difference between the symptoms of the abortive cases and the prodromal symptoms of cases which become paralyzed, so that in solving the problem of diagnosis during the preparalytic stage, the possibility of recognition of the abortive case will be coincidentally accomplished.

Our experience with cases which did not develop paralysis has been small numerically. Certain significant points, however, concerning the unparalyzed group have come to our notice. There seems to be in many cases some degree of muscular weakness. This may be transient. Two of our cases showed some indefinite weakness about the pelvis which made it difficult for the children to stand. They appeared to buckle at the hips and fall, following a sudden change of position or balance (case 32, page 180). Such cases belong perhaps more properly to Müller's group of "embryo" forms and serve, therefore, to emphasize the number of grades of severity that shade almost imperceptibly one into the other. The record of an abortive case (case 33, page 181) is given completely because the patient was under observation continuously from within twenty-four hours of the first symptom throughout the whole course of the disease. History of exposure, clinical picture, blood and spinal fluid examinations, and finally neutralization of virus M.A., all supported the diagnosis of poliomyelitis without paralysis. In this instance diagnosis of acute poliomyelitis without paralysis was fairly justified. Besides the history of exposure, the spinal fluid showed changes that were most suggestive. Furthermore, the clin-

ical picture was very striking, apathy and drowsiness being most obvious. However, it is difficult to select any one feature of the case as pathognomonic; a history of exposure is in itself extremely unreliable evidence. The following example is illustrative of this fact. Donald P., aged three years, suffered from malaise, anorexia, and drowsiness a day or two before his brother fell sick with a fatal attack of poliomyelitis, but his blood serum failed to neutralize the virus. While this is perhaps not absolute proof, still it should make one a little less ready to diagnose abortive poliomyelitis in every child who is a little indisposed in the summer. Two other examples of the abortive type that came to our notice were interesting because of the very slight muscular weakness that each displayed. If it had not been for the history of exposure, it is quite possible that both of these cases would have been overlooked. The weakness was so slight and consequently so difficult to locate, and above all so transient, that it very nearly escaped observation.

Of the other clinical features, it may be said that irritability and drowsiness are frequently present. The knee jerks are most variable. Wickman reports instances of disappearance of the reflex on one or both sides, and also cases where the reflex is exaggerated. Pain is a very common symptom, just as it is in the paralyzed cases. The pain, as described in the section on the acute stage, may be muscular or neuritic, often in the neck or back, or in the form of headache. The disease which, perhaps, the abortive cases most frequently resemble is influenza. Consequently, such cases occurring during the summer months, especially in the neighborhood of a patient with paralysis, should be viewed with suspicion and quarantined.

THE BLOOD.

The complete symptomatology of filterable virus diseases in man is not yet established. Certain clinical features of the two examples most studied, hydrophobia and poliomyelitis, have been well described, but the blood picture, which has been so thoroughly determined in most infectious diseases and is of such value in differential diagnosis, is little discussed. Owing to the resemblance of numerous features of rabies and poliomyelitis, it seemed that a comparative study of the blood changes in the two diseases might throw

some further light on the nature of the human body's reaction to invasion by infecting agents of this type. Unfortunately, however, despite the great amount of information that has been recorded about each of these diseases, only a few meagre and often conflicting statements are to be found on the subject of blood. The Systems of Medicine make no mention of blood counts in either malady. In one exhaustive report of thirty cases of rabies by Bain and Maloney³⁹ the blood of one case is reported to have had a "slight polymorphonuclear leukocytosis."

Through the courtesy of the attending physicians at the Presbyterian Hospital, New York, the following blood counts from cases of hydrophobia are available:

Patient I. Male, age 35 years. Temperature 99.3° F.

	Per cent.	Per cent.
Leukocyte count 9,400.	Polymorphonuclears ... 76.0	Transitionals 3.8
	Lymphocytes 10.5	Basophiles 0.0
	Large mononuclears... 9.8	Eosinophiles 0.0

Patient II. Male, age 27 years. Temperature 100-103° F.

	Per cent.	Per cent.
Leukocyte count 13,000.	Polymorphonuclears ... 76.5	Transitionals 2.5
	Lymphocytes 11.0	Basophiles 0.0
	Large mononuclears... 10.0	Eosinophiles 0.0

Patient III. Male, age 27 years. Temperature 103° F. (Nov. 26.)

	Per cent.	Per cent.
Leukocyte count 34,450.	Polymorphonuclears .. 85.0	Transitionals 3.0
	Lymphocytes 12.0	Basophiles 0.0
	Large mononuclears... 0.0	Eosinophiles 0.0

Patient III. Temperature 103° F. (Nov. 27.)

	Per cent.	Per cent.
Leukocyte count 30,200.	Polymorphonuclears .. 87.0	Transitionals 2.0
	Lymphocytes 10.5	Basophiles 0.0
	Large mononuclears... 0.5	Eosinophiles 0.0

³⁹ Bain and Maloney, *Lancet*, 1909, ii, 772.

Obviously, except in the first case where the total count was normal, there was a leukocytosis. In all the cases a definite polymorphonucleosis existed with a low lymphocyte percentage.

In the literature of poliomyelitis there are conflicting statements about the leukocyte count. La Fetra,⁴⁰ in New York, and Müller, in Germany, have made the most extensive observations on the blood. The former reported in six cases a leukocytosis, running from 13,400 to 20,600. He says nothing about the differential counts. Müller, on the other hand, reporting fifteen cases, says that a leukopenia of from 3,000 to 5,000 was always present in the acute stage. He gives no figures but states that there was a slight increase of the lymphocytes. Gay and Lucas made a comparative study of the blood in monkeys suffering from the experimentally produced disease, and of children with poliomyelitis. In monkeys they report a leukopenia during the acute stage with a lymphocytosis ranging from 10 to 20 per cent. above normal. The protocols of the human cases, however, do not support very convincingly their statements that a similar blood picture occurs in the human cases. The lowest count, that reported from a nine months' old baby, (case II) is 7,800 to 12,200. The other three cases showed the following leukocyte counts:

Case I, 20 months, 11,600-17,400.

Case III, 2 years, 12,000.

Case IV, 5 years, 13,400.

These can hardly be considered leukopenias. In two of their four cases, a lymphocytosis, "slight" for one, and "62 per cent." for the other, is given. Of the other two cases, one, a child of five years, had a normal differential count, and the other a polymorphonuclear cytosis of 75.5 per cent.

The following tables and the ensuing statements are based upon the study of the blood of our seventy-one hospital patients. The actual figures given represent the counts from only fifty-nine cases. The records of the other twelve cases were omitted because those patients suffered from other conditions which might have affected the blood count. Care was taken to avoid collecting the blood at

⁴⁰ La Fetra, *loc. cit.*

times when a digestive leukocytosis might have been present. The total white count was made in the usual way, the blood and 0.5 per cent. acetic acid solution being mixed in a 1:10 pipette and the drop counted on a 0.1 of a millimeter cell. For differential counts, Wright's modification of the Romanowski stain was used.

The first series of tables has been arranged simply to record the counts by weeks of the disease and age of patients. The second series is designed to show minimal, maximal, and average counts for groups of cases. The composite table (page 95) shows minimal, maximal, and average total leukocyte counts of all ages together by weeks. Such a compilation seems justifiable, because the age variation is much less in respect to the total than the differential leukocyte count.

SERIES I.

Age 6 to 12 Months.

First week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms,†
1	$\frac{25}{30}$ * 25,300	59.0	23.5	2.5	13.5	0.5	0.0	1.0
2	$\frac{35}{20}$ 35,600	32.0	55.5	1.0	9.0	0.0	1.0	1.5
3	$\frac{23}{10}$ 23,900	25.0	57.0	2.0	15.0	0.0	1.0	1.5
4	$\frac{23}{10}$ 23,800	65.0	16.5	18.0	0.5	0.0	0.0	0.0

Age 1 to 2 Years.

First Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	$\frac{15}{10}$ 15,300	36.0	50.0	2.0	7.5	0.0	5.5	0.0
2	$\frac{16}{10}$ 16,300	26.5	42.0	7.5	13.5	1.0	9.5	0.0
3	$\frac{15}{10}$ 15,000	52.5	31.5	13.5	1.5	0.5	0.5	0.0
4	$\frac{15}{10}$ 15,200	44.0	33.5	12.5	6.0	0.0	3.0	1.0
8	$\frac{15}{10}$ 15,700	53.5	28.5	12.5	4.5	1.0	0.0	0.0
9	$\frac{26}{10}$ 26,000	27.0	39.0	10.0	8.0	0.0	15.5	0.5
10	$\frac{18}{10}$ 18,200	51.5	29.0	2.0	9.0	0.5	8.0	0.0
12	$\frac{23}{10}$ 23,300	70.0	16.5	3.0	9.0	0.5	0.5	1.0
14	$\frac{17}{10}$ 17,500	42.5	47.0	1.5	5.0	0.0	3.5	0.5
17	$\frac{12}{10}$ 12,200	47.0	40.0	10.0	2.5	0.0	0.5	0.0
18	$\frac{17}{10}$ 17,100	38.0	49.0	1.5	11.0	0.0	0.0	0.5
20	$\frac{13}{10}$ 13,400	67.5	28.0	7.0	5.0	0.0	2.0	0.5

* The numerator of the fraction signifies the day of disease, the denominator the day of paralysis.

† "Stimulation forms" of Türk.

SERIES I (CONTINUED).

Age 1 to 2 Years.

Second Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms
5	$\frac{12}{10}$ 14,800	44.0	56.0	—	—	—	—	—
6	$\frac{12}{10}$ 14,200	43.5	42.0	7.5	6.5	0.0	0.5	0.0
8	$\frac{12}{10}$ 22,000	46.0	38.5	0.5	11.0	0.5	3.5	0.0
10	$\frac{12}{10}$ 30,400	73.5	9.0	8.5	7.0	6.0	2.0	0.0
11	$\frac{12}{10}$ 12,300	37.0	56.0	1.0	4.0	0.5	1.0	0.5
13	$\frac{12}{10}$ 16,000	45.5	21.5	23.0	6.0	2.5	1.0	0.0
14	$\frac{12}{10}$ 16,100	47.0	38.0	1.0	7.0	1.0	5.5	0.5
15	$\frac{12}{10}$ 16,200	51.0	30.5	9.0	5.0	4.5	0.0	0.0
17	$\frac{12}{10}$ 17,400	64.0	23.0	0.5	8.0	2.0	2.0	0.5
19	$\frac{12}{10}$ 20,000	45.0	40.5	0.0	9.5	1.0	3.5	0.5
20	$\frac{12}{10}$ 16,200	53.5	31.5	4.5	10.0	0.0	0.0	0.5

Third Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms
1	$\frac{15}{10}$ 14,600	47.0	43.0	4.0	2.0	0.0	3.5	0.5
2	$\frac{15}{10}$ 17,600	41.0	38.5	3.0	4.0	0.0	10.5	2.5
4	$\frac{15}{10}$ 14,800	43.0	39.5	1.5	13.5	1.0	1.5	0.0
5	$\frac{15}{10}$ 8,300	61.5	30.5	5.0	0.5	0.0	4.0	0.5
6	$\frac{15}{10}$ 19,100	50.0	40.5	2.0	6.5	0.5	0.5	0.0
7	$\frac{15}{10}$ 12,200	67.0	22.0	7.5	2.5	0.5	0.0	0.5
8	$\frac{15}{10}$ 15,900	50.0	35.0	4.0	8.5	0.5	1.5	0.5
9	$\frac{15}{10}$ 26,100	30.5	58.5	3.0	5.5	0.0	2.5	0.0
13	$\frac{15}{10}$ 38.4	55.2	5.6	0.4	0.0	0.0	0.4	
15	$\frac{15}{10}$ 22,500	64.5	29.5	3.0	1.5	1.5	2.0	0.0
16	$\frac{15}{10}$ 13,400	42.0	43.5	1.0	10.0	1.0	2.5	1.0
18	$\frac{15}{10}$ 17,200	49.5	39.0	6.5	3.5	0.0	1.0	0.5
19	$\frac{15}{10}$ 25,600	59.0	33.5	0.0	7.5	0.0	0.0	0.0

Fourth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	$\frac{27}{20}$ 13,500	33.0	44.0	9.5	9.0	0.0	4.0	0.5
4	$\frac{27}{20}$ 20,100	33.0	52.5	3.5	7.5	0.0	2.0	1.5
5	$\frac{27}{20}$ 17,800	60.0	34.5	3.0	1.5	0.0	1.0	0.0
6	$\frac{27}{20}$ 24,700	55.5	38.0	5.5	2.0	0.0	1.5	0.0
7	$\frac{27}{20}$ 10,100	41.0	50.0	6.0	2.0	0.5	0.0	0.5
11	$\frac{27}{20}$ 15,800	42.0	37.0	4.0	17.5	1.0	1.0	2.5
13	$\frac{27}{20}$ 16,000	29.0	63.5	4.5	1.5	0.0	1.5	0.0
14	$\frac{27}{20}$ 18,400	34.0	52.0	7.0	5.5	0.0	1.5	0.0
15	$\frac{27}{20}$ 21,500	46.5	28.0	22.0	3.5	0.0	0.0	0.0
16	$\frac{27}{20}$ 14,200	26.5	23.0	44.0	3.5	0.0	2.5	0.0
19	$\frac{27}{20}$ 15,200	51.0	21.5	20.5	3.5	1.0	2.5	0.0

SERIES I (CONTINUED).

Age 1 to 2 Years.

Fifth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	20,300	60.5	20.5	10.0	2.0	0.5	6.0	0.5
2	23,600	42.5	26.0	13.5	10.0	0.0	6.5	1.0
4	9,100	40.0	42.0	7.5	7.5	0.0	2.5	0.5
6	21,600	38.0	56.0	1.0	3.5	0.0	2.0	0.0
7	13,100	38.5	43.7	8.0	8.5	0.0	2.0	0.0
8	16,400	43.0	42.0	8.0	3.5	0.0	2.5	0.5

Sixth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	12,600	37.5	49.0	0.0	9.5	0.5	2.5	1.0
7	9,600	40.0	32.0	10.0	15.0	0.5	0.5	2.0
11	26,700	23.0	69.0	6.0	0.5	0.0	1.5	0.0

Seventh Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
8	26,700	51.0	34.5	10.0	4.0	0.0	0.5	0.0
11	23,700	31.5	45.0	9.0	5.0	2.0	6.5	0.5

Age 2 to 3 Years.

First Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	15,300	56.0	30.5	8.5	3.0	1.5	0.5	0.0
2	23,000	56.0	20.0	10.5	12.0	0.5	0.5	0.5
3	18,400	32.5	43.0	14.0	9.5	0.0	0.5	0.0
11	13,800	47.5	31.0	1.5	7.0	0.5	7.0	1.0
12	9,800	64.5	21.5	7.0	3.5	1.0	1.0	1.5
14	14,200	58.5	31.5	7.0	3.0	0.0	0.0	0.0
15	10,600	49.0	28.0	2.0	18.0	0.5	2.0	0.5

Second Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
5	16,400	38.0	41.0	5.0	10.5	0.5	4.5	0.5
6	15,900	60.0	10.0	23.5	5.5	0.0	1.0	0.0
7	9,800	61.5	21.5	2.0	7.0	0.0	7.5	0.5
9	20,000	57.0	23.5	8.5	4.5	0.0	6.5	0.0
10	12,400	54.0	28.0	7.0	5.0	0.0	5.0	0.0
11	15,200	47.6	31.3	14.0	2.0	0.0	0.5	0.0
13	16,600	53.0	34.0	4.0	3.5	5.0	0.0	0.5
14	11,200	52.0	35.5	7.5	3.0	0.5	1.5	0.0

SERIES I (CONTINUED).

Age 2 to 3 Years.

Third Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	15 23,700	63.5	24.5	0.0	9.5	0.0	2.0	0.5
5	13 13,600	45.0	42.5	5.0	6.0	0.0	1.0	0.5
6	12 12,800	55.0	16.0	24.5	2.5	0.5	1.5	0.0
8	14 14,100	52.0	27.5	14.5	2.5	0.0	2.5	0.5
15	18 18,700	47.5	35.5	7.0	7.0	0.0	3.0	0.0

Fourth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	23 23,600	65.0	23.0	4.0	7.0	0.0	1.0	0.0
6	18 18,800	43.5	46.0	0.5	10.0	0.0	0.0	0.0
8	14 14,600	26.5	50.0	10.5	7.0	0.0	6.0	0.0
10	19 19,800	61.0	23.5	9.5	4.5	0.5	0.5	0.0
11	23 23,000	57.0	32.0	0.0	7.5	0.0	3.0	0.0

Fifth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	19 19,400	46.0	33.5	4.5	6.0	0.0	9.5	0.5
6	21 21,700	57.5	36.0	2.0	5.0	0.0	2.5	0.0
7	16 16,400	40.0	38.5	9.5	14.0	0.0	8.0	0.0
8	15 15,000	43.0	44.5	5.0	3.5	0.0	3.5	0.0
9	18 18,100	58.0	21.5	5.5	11.5	0.0	3.5	0.0
11	27 27,000	46.0	41.0	0.0	5.0	0.0	7.5	0.0

Sixth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	22 22,800	57.0	29.0	4.5	4.5	0.5	2.5	2.0
6	14 14,800	51.5	44.0	3.0	0.5	0.0	0.5	0.5
9	10 10,200	45.0	39.0	2.0	9.0	0.0	3.5	1.0

Seventh Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	15 15,800	70.0	16.0	9.5	3.5	0.0	1.0	0.0
6	22 22,300	42.5	43.6	0.5	7.5	0.5	1.0	0.0
9	16 16,800	51.5	25.5	11.0	6.5	0.5	5.0	0.0

SERIES I (CONTINUED).

Age 3 to 4 Years.

First Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	6,900	56.5	29.0	—	13.0	0.5	0.0	1.0
2	8,300	72.0	21.5	1.0	5.0	0.0	0.0	0.5
3	15,400	63.0	17.0	5.0	8.5	0.0	6.0	0.5
4	10,100	61.0	26.5	6.5	5.0	0.0	0.5	0.5
5	15,100	55.0	26.0	8.5	1.5	0.0	9.0	0.0

Second Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	14,600	57.5	21.5	13.5	6.5	0.0	0.5	0.5
5	19,700	84.0	6.5	6.5	3.0	0.0	0.0	0.0

Third Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	10,300	59.5	20.0	6.5	14.0	0.0	0.5	0.0
4	15,700	45.0	37.5	2.0	5.5	1.0	6.5	—

Fourth Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	19,800	51.0	37.5	0.0	3.5	0.0	8.0	0.0
5	11,000	40.5	46.5	1.5	8.5	0.5	2.0	0.5

Fifth Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	12,800	54.0	30.0	6.0	6.5	0.0	2.5	0.5
4	16,700	55.0	30.0	10.5	2.5	0.0	2.0	0.0
5	15,000	60.5	25.0	6.5	3.0	1.0	4.0	0.0

Seventh Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	9,800	49.5	30.5	4.0	11.5	0.5	4.0	0.0

SERIES I (CONTINUED).

Age 4 to 5 Years.

First Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	33,000	—	—	—	—	—	—	—
4	19,300	73.0	18.6	2.6	4.6	0.0	0.0	0.6
5	17,600	44.5	37.5	3.0	14.5	0.0	0.0	0.5
6	20,000	49.0	25.0	26.5	8.0	1.5	0.0	0.0
7	13,000	51.5	37.0	7.0	4.5	0.0	0.0	0.0
8	12,400	53.0	24.0	9.5	12.0	0.0	0.0	0.0
Second Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	28,000	—	—	—	—	—	—	—
Third Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
3	20,800	—	—	—	—	—	—	—
Fourth Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	10,500	55.0	30.0	10.5	4.0	0.0	0.5	0.0
2	12,000	58.5	27.9	9.0	2.0	0.0	3.5	0.0
3	22,300	42.0	33.8	6.4	2.1	0.6	2.7	0.3
Fifth Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
2	14,800	85.0	—	—	—	—	—	—
	6,300	62.5	19.0	12.0	3.5	1.0	2.0	0.0
3	20,000	65.0	24.0	0.5	7.0	0.0	2.0	1.0
Sixth Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	13,100	64.0	23.5	8.5	3.0	0.0	1.0	0.0
3	28,600	57.5	34.0	7.0	1.5	0.0	0.0	0.0
Seventh Week.								
Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
1	12,400	39.5	54.5	0.5	4.0	0.5	0.5	0.5
2	11,000	62.0	16.0	10.5	0.5	9.5	1.0	0.5

SERIES I (CONTINUED).

Age 4 to 5 Years.

Eighth Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1	$\frac{52}{100}$ 12,000	47.0	37.5	8.5	5.5	0.0	1.5	0.0
2	$\frac{54}{100}$ 10,700	52.5	35.5	9.0	2.5	0.0	0.5	0.0

Tenth Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1	$\frac{66}{83}$ 10,800	61.5	23.5	4.5	9.0	0.0	0.0	0.5

Twelfth Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1	$\frac{72}{78}$ 16,000	54.0	38.0	5.5	2.0	0.0	1.5	0.0

Age 5 to 7 Years.

First Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1	$\frac{6}{8}$ 19,400	62.2	16.7	11.7	1.0	0.2	8.0	0.0
2	$\frac{5}{10}$ 23,200	76.0	17.0	3.0	2.5	1.0	0.0	0.5
3	$\frac{2}{10}$ 8,800	77.0	17.5	0.5	4.0	0.0	0.0	1.0
4	$\frac{6}{14}$ 20,800	66.0	24.0	1.0	5.5	1.5	2.0	0.0

Second Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
3	$\frac{13}{16}$ 11,800	76.5	14.5	4.5	3.5	0.0	0.5	0.5

Third Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
4	$\frac{15}{18}$ 28,800	72.0	9.0	2.0	10.0	0.0	2.0	0.0

Fourth Week.

Number of case.	Total leukocyte count.	Polymor-phonuclears.	Lympho-cytes.	Large mono-nuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1	$\frac{26}{33}$ 14,200	49.5	36.0	12.5	1.5	0.0	0.0	0.0
2	$\frac{33}{44}$ 21,100	64.5	25.0	7.5	2.5	0.5	0.0	0.0

SERIES I (CONCLUDED).

Age 5 to 7 Years.

Sixth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
I	$\frac{87}{15,600}$	50.0	32.5	4.0	11.0	0.0	1.5	1.0

Seventh Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
I	$\frac{44}{16,000}$	59.5	22.0	7.5	8.5	0.5	2.0	0.0

Age 8 to 12 Years.

First Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
2	$\frac{7}{16,700}$	75.0	15.0	0.5	9.5	0.0	0.0	0.0
3	$\frac{12}{12,000}$	75.0	13.0	3.5	8.0	0.0	1.0	0.0
4*	$\frac{13}{13,000}$	59.0	17.5	17.0	4.5	0.5	1.5	0.0

Second Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	$\frac{12}{12,000}$	62.0	17.5	16.0	3.5	0.0	1.0	0.0

Third Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
I	$\frac{18}{7,800}$	63.0	28.0	4.5	2.0	0.0	2.5	0.0
4	$\frac{18}{18,000}$	67.5	19.5	3.5	9.0	0.0	1.0	0.0

Fourth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
I	$\frac{9}{9,400}$	64.5	26.0	0.0	4.5	0.5	4.5	0.0
4	$\frac{10}{10,700}$	62.5	26.0	2.5	8.0	0.0	0.5	0.5

Sixth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
I	$\frac{9}{9,400}$	47.0	37.5	5.5	4.0	0.0	3.5	2.5

Eighth Week.

Number of case.	Total leukocyte count.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Basophiles.	Eosinophiles.	Stimulation forms.
4	$\frac{16}{16,000}$	58.0	32.5	1.0	6.0	0.0	2.0	0.5

* Age, 21 years.

SERIES II.

First Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Eosinophiles.	Eosino-philic.	Stimulation forms.
6-12 mos.	4	23,900-35,600	{ Maximum 59.0 Minimum 25.0 Average 42.0	57.0 16.5 36.7	18.0 1.0 9.5	15.0 0.5 7.7	0.5 0.0	1.0 0.0	1.5 0.0
1-2 yrs.	12	12,200-26,000	{ Maximum 70.0 Minimum 26.5 Average 48.2	50.0 16.5 33.2	13.5 1.5 7.5	13.5 2.5 8.0	1.0 0.0	15.5 0.0	1.0 0.0
2-3 yrs.	5	9,000-23,000	{ Maximum 64.5 Minimum 32.5 Average 48.5	43.0 20.0 31.5	14.0 1.5 7.7	18.0 3.0 10.5	1.5 0.0	7.0 0.0	1.5 0.0
3-4 yrs.	4	6,900-15,000	{ Maximum 72.0 Minimum 55.0 Average 68.5	29.0 17.0 23.0	8.5 1.0 4.7	13.0 1.5 7.2	0.5 0.0	9.0 0.0	1.0 0.0
4-5 yrs.	6	12,400-33,000	{ Maximum 81.0 Minimum 44.5 Average 62.7	37.5 18.6 28.0	26.5 2.6 14.5	14.5 4.5 9.5	1.5 0.0	0.0 0.0	0.5 0.0
5-7 yrs.	4	8,800-23,300	{ Maximum 77.0 Minimum 62.2 Average 69.6	24.0 16.7 20.3	11.7 0.5 6.5	5.5 1.0 3.2	1.0 0.0	8.0 0.0	1.0 0.0
8-21 yrs.	3	12,000-16,700	{ Maximum 75.0 Minimum 59.0 Average 67.0	17.5 13.0 15.2	17.0 0.5 8.7	9.5 4.5 7.0	0.05 0.0	1.5 0.0	0.0 0.0
Total	38								

Second Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Eosinophiles.	Eosino-philic.	Stimulation forms.
1-2 yrs.	11	12,300-30,400	{ Maximum 73.0 Minimum 37.0 Average 55.0	56.0 9.0 32.5	23.0 0.5 11.7	11.0 4.0 7.7	6.0 0.0	5.5 0.0	0.05 0.0
2-3 yrs.	8	9,800-20,000	{ Maximum 61.5 Minimum 38.0 Average 49.7	41.0 10.0 25.5	23.5 2.0 12.7	10.5 2.0 6.2	5.0 0.0	7.5 0.0	0.5 0.0
3-4 yrs.	2	14,600-19,700	{ Maximum 84.0 Minimum 57.5 Average 70.7	21.5 6.5 14.0	13.5 6.5 10.0	6.5 3.0 4.7	0.0 0.0	0.5 0.0	0.5 0.0
4-5 yrs.	1	28,000							
5-7 yrs.	1	11,800	76.5	14.5	4.5	3.5	0.0	0.5	0.5
8-12 yrs.	1	12,000	62.0	17.5	16.0	3.5	0.0	1.0	0.0
Total	24								

SERIES II (CONTINUED).

Third Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononu-clears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1-2 yrs.	13	8,300-26,100	{ Maximum 67.0 Minimum 30.5 Average 48.7	58.5 22.0 40.2	7.5 0.0 3.7	13.5 0.5 7.0	1.5 0.0 0.0	10.5 0.0 0.0	1.0 0.0 0.0
2-3 yrs.	5	12,800-23,700	{ Maximum 63.5 Minimum 45.0 Average 54.7	42.5 16.0 29.2	24.5 0.0 12.2	9.5 2.5 6.0	0.5 0.0 0.0	3.0 1.0 0.0	0.5 0.0 0.0
3-4 yrs.	2	10,300-15,700	{ Maximum 59.5 Minimum 45.0 Average 52.2	37.5 20.0 28.7	6.5 2.0 4.2	14.0 5.5 9.7	1.0 0.0 0.0	6.5 0.5 0.0	0.0 0.0 0.0
4-5 yrs.	1	28,000							
5-7 yrs.	1	28,000	72.0	9.0	2.0	10.0	0.0	2.0	0.0
8-12 yrs.	2	7,800-18,000	{ Maximum 67.5 Minimum 63.0 Average 65.2	28.0 19.5 23.7	4.5 3.5 4.0	9.0 2.0 0.0	0.0 0.0 0.0	2.5 1.0 0.0	0.0 0.0 0.0
Total	24								

Fourth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononu-clears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1-2 yrs.	11	10,100-24,700	{ Maximum 60.0 Minimum 26.5 Average 43.2	63.5 21.5 42.5	41.0 35.0 23.7	17.0 1.5 9.5	1.0 0.0 0.0	4.0 0.0 0.0	2.5 0.0 0.0
2-3 yrs.	5	14,600-23,600	{ Maximum 65.0 Minimum 26.5 Average 45.7	46.0 23.5 34.7	10.5 0.5 5.5	10.0 4.5 7.2	0.5 0.0 0.0	6.0 0.0 0.0	0.0 0.0 0.0
3-4 yrs.	2	11,000-19,800	{ Maximum 51.0 Minimum 40.5 Average 45.7	46.5 37.5 42.0	1.5 0.0 0.7	8.5 3.5 6.0	0.5 0.0 0.0	8.0 2.0 0.0	0.5 0.0 0.0
4-5 yrs.	3	10,500-22,300	{ Maximum 58.5 Minimum 42.0 Average 50.2	33.8 27.9 30.8	10.5 6.4 8.4	4.0 2.0 3.0	0.6 0.0 0.0	2.7 0.5 0.0	0.3 0.0 0.0
5-7 yrs.	2	14,200-21,000	{ Maximum 64.5 Minimum 49.5 Average 57.0	36.0 25.0 30.0	12.5 7.5 10.0	2.5 1.5 2.0	0.5 0.0 0.0	0.0 0.0 0.0	0.0 0.0 0.0
8-12 yrs.	2	9,400-10,700	{ Maximum 64.5 Minimum 62.5 Average 63.5	26.0 0.0 1.7	2.5 0.0 1.7	8.0 4.5 6.2	0.5 0.0 0.0	4.5 0.5 0.0	0.5 0.0 0.0
Total	25								

SERIES II (CONTINUED).

Fifth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononu-clears.	Transi-tionals.	Baso-philcs.	Eosino-philcs.	Stimula-tion forms.
1-2 yrs.	6	9,100-23,600	{ Maximum 60.5 Minimum 38.0 Average 49.2	56.0 20.5 38.2	13.5 1.0 7.2	10.0 2.0 6.0	0.5 0.0 0.0	6.5 1.5 0.0	1.0 0.0 0.0
2-3 yrs.	6	15,000-27,000	{ Maximum 58.0 Minimum 40.0 Average 49.0	44.5 21.5 33.0	9.5 0.0 4.2	14.0 5.0 9.5	0.0 0.0 0.0	9.5 3.5 0.0	0.5 0.0 0.0
3-4 yrs.	3	12,800-16,700	{ Maximum 60.5 Minimum 54.5 Average 57.5	30.0 25.0 27.5	10.5 6.0 8.2	6.5 2.5 4.5	1.0 0.0 0.0	4.0 2.0 0.0	0.5 0.0 0.0
4-5 yrs.	2	6,300-20,000	{ Maximum 85.0 Minimum 62.5 Average 73.7	24.0 19.0 21.5	12.0 0.5 6.2	7.0 3.5 5.2	1.0 0.0 0.0	2.0 0.0 0.0	1.0 0.0 0.0
Total	17								

Sixth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononu-clears.	Transi-tionals.	Baso-philcs.	Eosino-philcs.	Stimula-tion forms.
1-2 yrs.	3	9,600-26,700	{ Maximum 40.0 Minimum 23.0 Average 31.5	69.0 32.0 50.5	10.0 0.0 5.0	15.0 0.5 7.7	0.5 0.0 0.0	2.5 0.5 0.0	2.0 0.0 0.0
2-3 yrs.	2	10,200-22,800	{ Maximum 57.0 Minimum 45.0 Average 51.0	44.0 29.0 36.5	4.5 2.0 3.2	9.0 0.5 4.7	0.5 0.0 0.0	3.5 0.5 0.0	2.0 0.5 0.0
4-5 yrs.	2	13,100-28,600	{ Maximum 64.0 Minimum 57.5 Average 60.7	34.0 23.5 28.7	8.5 7.0 7.7	3.0 1.5 2.2	0.0 0.0 0.0	1.0 0.0 0.0	0.0 0.0 0.0
5-7 yrs.	1	15,000	50.0	32.5	4.0	11.0	0.0	1.5	1.0
8-12 yrs.	1	9,400	47.0	37.5	5.5	4.0	0.0	3.5	2.5
Total	9								

SERIES II (CONCLUDED).

Seventh Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
1-2 yrs.	2	23,700-26,700	{ Maximum 51.0 Minimum 31.5 Average 41.2	45.0 34.5 39.7	10.0 9.5 9.7	5.0 4.0	2.0 0.0	6.5 0.5	0.5 0.0
2-3 yrs.	3	15,800-22,300	{ Maximum 70.0 Minimum 42.5 Average 56.2	43.6 16.0 29.8	11.0 0.5 5.7	7.5 3.5 5.5	0.5 0.0	5.0 1.0	0.0 0.0
3-4 yrs.	1	9,800	49.0	30.5	4.0	11.5	0.5	4.0	0.0
4-5 yrs.	2	11,000-12,400	{ Maximum 62.0 Minimum 39.5 Average 50.7	54.5 16.0 35.2	10.5 0.5 5.5	4.0 0.5 2.2	9.5 0.5	1.0 0.5	0.5
5-7 yrs.	1	16,000	59.5	22.0	7.5	8.5	0.5	2.0	0.0
Total	9								

Eighth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
4-5 yrs.	2	10,700-12,000	{ Maximum 52.5 Minimum 47.0 Average 49.7	37.5 35.5 36.5	9.0 8.5 8.7	5.5 2.5 4.0	0.0 0.0	1.5 0.5	0.0
8-12 yrs.	1	16,000	58.0	32.5	1.0	6.0	0.0	2.0	0.5
Total	3								

Tenth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
4-5 yrs.	1	10,800	61.5	23.5	4.5	9.0	0.0	0.0	0.5

Twelfth Week.

Age.	Number of cases.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononuclears.	Transi-tionals.	Baso-philic.	Eosino-philic.	Stimula-tion forms.
4-5 yrs.	1	16,000	54.0	38.0	5.5	2.0	0.0	1.5	0.0

ALL AGES.

	Number of cases.	Leukocyte count.		
		Maximum.	Minimum.	Average.
1st week	38	24,600	12,200	18,400
2d week	24	24,500	12,000	18,250
3d week	24	23,300	10,400	16,800
4th week	25	20,300	11,600	15,900
5th week	17	21,800	10,800	16,300
6th week	9	23,200	10,500	16,800
7th week	9	19,500	15,000	17,250
8th week	3	14,000	10,700	12,400
10th week	1			10,800
12th week	1			16,000
Total	151			

The following table comprises separately the counts from pre-paralytic and abortive cases.

	Age.	Total leukocyte count.	Poly-morpho-nuclears.	Lympho-cytes.	Large mononu-clears.	Transi-tionals.	Baso-philes.	Eosino-philes.	Stimula-tion forms.
Breed's case		15,000	—	—	—	—	—	—	—
		7,000	81.0	—	—	—	—	—	—
J. S.	2½ yrs.	14,200	58.5	31.5	7.0	3.0	0.0	0.0	0.0
C. T.	8 mos.	35,600	32.0	55.0	1.0	9.0	0.0	1.0	1.5
E. N.	6 yrs.	23,200	76.0	17.0	3.0	2.5	0.0	1.0	0.5
H. B.	6 yrs.	8,800	77.0	17.5	0.5	4.0	0.0	0.0	1.0
		11,200	79.5	14.5	0.5	4.5	0.0	0.5	0.5
		5,600	68.5	22.0	1.5	6.0	0.0	1.0	1.0
		11,800	76.5	14.5	4.5	3.5	0.0	0.5	0.5
P. T.	2 yrs.	15,000	52.5	31.5	13.5	1.5	0.5	0.5	0.0

It is obvious from these figures that while in most cases in the preparalytic stage the total leukocyte count varies within the normal, there is a tendency toward the upper limits. More cases showed counts well above than below the usual extremes. In the differential counts there is a definite polymorphonucleosis with the one exception of a count in an infant eight months old, where the relation of polymorphonuclears and lymphocytes is normal. Furthermore, the lymphocyte percentages in all the other cases are distinctly below the usual figure.

In one instance where blood counts were made by the physician before the patient came to the hospital, a low total count appeared;

7,000, two days before the paralysis, and 3,000 on the day after paralysis. The differential count, however, showed a polymorphonuclear increase each time (81 per cent. and 58 per cent.). Except for this one case which subsequently became one of our seventy-one, we have not seen a true leukopenia. One abortive case in a little girl six years of age showed counts of 5,600 to 11,800. In the youngest children the leukocyte counts have been highest, and the polymorphonuclear increase both relatively and actually has been more marked than in the older patients. A glance at the tables, however, will show that all ages have a high white cell count. Furthermore, the leukocytosis persists for a surprising length of time. In nine cases, for example, observed during the seventh week, the leukocytes numbered 15,000 to 19,500, with an average of 17,250.

The differential count presents a fairly constant picture. For comparison, a table of differential counts from normal individuals prepared by Schloss⁴¹ is printed. There is obviously in poliomyelitis an increase in polymorphonuclear cells of 10 to 15 per cent. above the normal. This increase appears irrespective of the patient's age, though the youngest children have shown perhaps the highest polymorphonuclear counts. Contrary to the observations of Müller, and Gay and Lucas, we have found a diminution of lymphocytes. In general, they have been from 15 to 20 per cent. below the normal percentages. Notwithstanding the great variation which these cells present in relation to age, the finding of low lymphocyte percentages has been constant in all our cases of poliomyelitis.

The transitional and large mononuclear cells have reached no abnormal figures. In many cases there has been an increase in eosinophiles. This finding, however, has not been sufficiently constant to justify further comment. No counts were made of the red cells, nor were any hemoglobin estimations recorded. There has been nothing in the appearance of the patients to suggest an anemia resulting from the disease; nor did the stained erythrocytes look abnormal. No actual counts of the blood platelets were made, but the impression was gained in the course of many differential counts that these elements were somewhat increased.

⁴¹ Schloss, *Arch. Int. Med.*, 1910, vi, 638.

DIFFERENTIAL BLOOD COUNTS IN NORMAL INDIVIDUALS.

Age.		Polymorpho- nuclears.	Lympho- cytes.	Large mono- nuclears.	Eosino- philes.	Basophiles.
6-12 mos.	Maximum	35.9	58.5	12.2	4.5	0.8
	Minimum	24.6	50.5	7.3	0.0	0.1
	Average	30.4	55.9	9.6	2.6	0.4
1-2 yrs.	Maximum	39.7	58.8	11.7	6.0	0.5
	Minimum	27.5	45.3	6.7	1.6	0.0
	Average	36.3	51.2	8.5	3.2	0.2
2-3 yrs.	Maximum	44.3	55.0	11.3	6.0	1.2
	Minimum	33.2	43.5	5.0	0.5	0.0
	Average	38.7	49.0	8.2	3.1	0.4
3-4 yrs.	Maximum	54.1	47.6	16.2	4.2	0.9
	Minimum	36.2	32.2	5.0	1.5	0.0
	Average	44.7	39.1	11.2	2.8	0.5
4-5 yrs.	Maximum	51.7	49.5	6.7	4.0	0.6
	Minimum	42.2	38.4	3.4	1.6	0.3
	Average	48.5	42.1	6.0	2.6	0.3
5-6 yrs.	Maximum	61.8	36.7	16.0	4.7	1.0
	Minimum	52.6	21.2	6.5	0.7	0.3
	Average	56.5	29.9	10.0	2.5	0.6
6-7 yrs.	Maximum	61.3	34.1	15.7	4.7	0.6
	Minimum	52.3	24.5	8.1	0.1	0.0
	Average	56.0	30.4	10.8	2.2	0.2
7-8 yrs.	Maximum	72.0	39.1	15.2	3.5	0.2
	Minimum	45.2	21.1	6.7	0.0	0.0
	Average	54.4	32.5	11.6	0.1	0.06

To sum up, we have found in the blood of patients with poliomyelitis a constant and marked leukocytosis. In several instances the count has been as high as 30,000. In only one case has there been a definite leukopenia. Besides the increase in the total number of cells, there has been an equally constant increase of polymorphonuclears of 10 to 15 per cent., and diminution of lymphocytes of 15 to 20 per cent. The other forms of leukocytes have shown no abnormalities.

The recent demonstration of the visceral lesions of poliomyelitis, showing the active part of phagocytosis played by the polymorphonuclear cells is perhaps sufficient cause for the polymorphonuclear increase in the circulating blood. In view of the fact, however, that the brunt of the body's attempt at defense falls upon the

lymphatic system, and that the cells which are assembled at the point of attack seem to be lymphocytes, one might be led at first to expect a lymphocytosis in the blood. On the other hand, the great destruction of lymphocytes, which is evident in the visceral lesions, suggests that these cells are relatively few in the circulation because they are constantly being withdrawn to meet the invasion of the virus at its various points of attack. It will require a more fundamental knowledge of the kind of reaction the body develops to this type of infecting agent before a correct explanation of the blood picture can be made. At all events, while the blood picture in poliomyelitis is perhaps not any more specific than is the spinal fluid, it is helpful. If taken in connection with other available evidence, a leukocytosis of 15,000 to 30,000 is distinctly suggestive of the disease in question, especially if the polymorphonuclear cells are increased at the expense of the lymphocytes.

THE CEREBROSPINAL FLUID.

There are two ways in which a careful study of the cerebrospinal fluid in acute poliomyelitis may help to throw light on the disease. On the one hand, the changes observed may facilitate early diagnosis and thus enhance the value of any method of treatment which may be discovered in the future, and, on the other hand, the variations in the character of the fluid, as observed during the progress of the disease in each case, may give some information as to the usual course of the process and thus be an aid in determining how far any given remedy is effective or to what extent the natural course of the disease may be influenced. The following study was thus made with the twofold object of determining whether the examination of the spinal fluid may be an aid in early diagnosis, and what changes occur in the fluid during the course of the disease.

Previous investigations along this line have not been extensive. Most writers report only a few cases, and those who have apparently made a large number of lumbar punctures have not submitted the fluids to careful examination. The results obtained are somewhat variable, but in general the fluid is said to contain an abnormal amount of albumen and a sediment consisting of lymphocytes, large mononuclear cells, and, occasionally, polymorphonuclear cells. Im-

portant work has been done by Gay and Lucas,⁴² whose contributions are the most valuable in this field. Their studies have included the spinal fluids from monkeys in the incubation period, the prodromal stage, and the acute stage, and from eleven human cases in the preparalytic stage. Besides noting the character of the cells, they have estimated the number of cells per cubic millimeter. In the eleven early cases, the cell count varied from 55 to 580 per cubic millimeter, and the percentage of mononuclears from 75 to 100. Chemical examinations were omitted. Sophian⁴³ reports that the globulin test is positive in the early stages.

Inasmuch as previous work by Wollstein,⁴⁴ Römer and Joseph,⁴⁵ and Gay and Lucas failed to demonstrate the presence of any specific antibody in the spinal fluid in infantile paralysis, we have confined our study to simple chemical and cytological examinations.

It has been our object to see the cases as early as possible after the onset of the disease, to make a lumbar puncture on admission to the hospital, to repeat the puncture every two to four days in the early stages, and in the later stages to make a puncture every five to ten days while the case was under observation, or until the fluid became normal. We have examined in all 233 fluids from sixty-nine cases. The number of cases seen in the first week of the disease (dating from onset of symptoms, not of paralysis) was forty-three, in the second week, forty-five, in the third week, forty, and in the fourth week, thirty. The number of cases seen later in the course was much smaller. It is to be regretted that the number of preparalytic and abortive cases was so small; of the former, four, and of the latter only two were seen in the active stage. It was, however, rare that cases were referred to the hospital until a positive diagnosis, based on the appearance of paralysis, had been made by the attending physician.

The methods used for the examination of the cerebrospinal fluid have included estimation of the pressure, determination of the number of cells per cubic millimeter and the types of cells present,

⁴² Gay and Lucas, *Arch. Int. Med.*, 1910, vi, 330; Lucas, *Am. Jour. Dis. Child.*, 1911, i, 230.

⁴³ Sophian, *Arch. Pediat.*, 1911, xxviii, 201.

⁴⁴ Wollstein, *Jour. Exper. Med.*, 1908, x, 476.

⁴⁵ Römer and Joseph, *München. med. Wchnschr.*, 1910, lvii, 568.

estimation of the chlorides, and observations of the power of reducing Fehling's solution, and the globulin content. Globulin was tested by the butyric acid method of Noguchi, and the relative strength of the reaction noted. Thus very slightly plus (v. s. +) indicates a faint precipitate, which is, however, definitely more than that obtained with normal spinal fluids; slightly plus (s. +) is the term applied to reactions giving a well marked cloud or a slightly flocculent precipitate; plus (+) signifies a precipitate coming down in large flocculi; and double plus (++), a heavy flocculent precipitate. The reaction was performed with accurately measured amounts of spinal fluid and of reagents, and the reading was made after five to ten minutes' standing.⁴⁶ The gross appearance of the fluids was very constant. Almost all were clear, colorless, and watery. Only a few showed the slightest opalescence. On standing, a delicate web-like clot formed in a small proportion of cases. Clot formation was most frequently seen in the early stages of the disease. It bore no apparent relation to the globulin content of the fluid.

The results obtained by several of these methods may be dismissed briefly. Pressure determinations were made in a number of cases and readings were usually above normal. The pressure was read in millimeters of spinal fluid in a tube of one millimeter bore. No extremely high readings were obtained, and in some instances normal pressures were seen during the early stages of the disease. On the whole, the estimations of pressure were unsatisfactory, as the patients were usually crying children.

The chlorides were estimated in a series of cases with the idea that they might throw light on the inorganic substances in the fluid. No marked deviations from the normal were observed. Every fluid examined showed a power of reducing Fehling's solution which was, roughly speaking, normal. This reaction serves as a point of differentiation from many fluids in cases of meningitis, in which the reducing substance may be absent.

The type of cell present in the fluid was almost always the

⁴⁶ To 0.2 c.c. of spinal fluid is added 0.5 c.c. of butyric acid solution (10 per cent. butyric acid in 0.85 per cent. solution of sodium chloride). The mixture is boiled, 0.1 c.c. of normal sodium hydrate solution added, and then boiled again.

mononuclear. A large number of differential counts showed that the relative number of the different types of mononuclear cells was of comparatively little importance, so in later observations we were content with noting merely the relative number of mononuclear and polymorphonuclear cells. In several instances numbers of very large phagocytic cells containing vacuoles and broken down cells were seen. Once this occurred several weeks after the onset of the disease in association with a sudden rise in the cell count resembling that seen in acute meningitis. Polymorphonuclear cells, except in small number and probably due for the most part to a slight admixture of blood, were, except in a very few instances, absent after the first week of the disease. Early in the disease, however, and in the preparalytic period, the polymorphonuclears sometimes outnumbered the mononuclears and made up 80 to 90 per cent. of the total.

The results described by Flexner and Lewis⁴⁷ of a study of the cerebrospinal fluid of a monkey after inoculation with the virus of poliomyelitis are interesting. Twenty-four hours after inoculation the fluid contained "a considerable number of small cells, hardly exceeding a lymphocyte in size but showing a polyform nucleus, a few lymphocytes and some red corpuscles. At the expiration of forty-eight hours, the white cells have increased in numbers, but the cells with polyform nuclei still predominate. At the expiration of seventy-two hours, a large number of mononuclear cells have appeared and the fluid presents a striking opalescent appearance. On the day of paralysis the fluid tends to be only slightly cloudy and contains a mixture of large and smaller (lymphoid) mononuclear cells and a few cells with polymorphous nuclei." Lucas, too, found in the monkey during the prodromal stage "a marked increase in the cells, often reaching 1,000 per cubic millimeter. In this stage, also, polynuclears are still present, in some cases as high as 60 per cent., though the large mononuclears and lymphocytes were very evident. In the early acute stage the increase in cells is very marked. The cells are now, however, mostly of the lymphocytic or very early form of cells, and sometimes very hard to place, as they are apparently undifferentiated cells." Of his eleven preparalytic

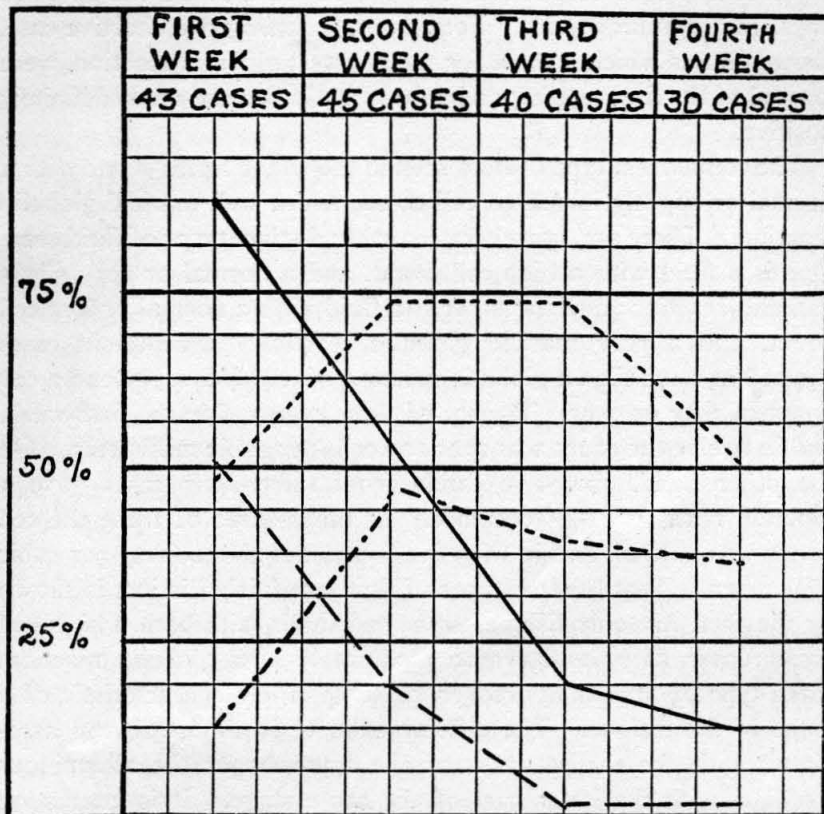
⁴⁷Flexner and Lewis, *Jour. Am. Med. Assn.*, 1910, liv, 1140.

human cases, however, only one showed less than 87 per cent. of mononuclears, and that had 75 per cent. of mononuclears. A return of polymorphonuclears later in the disease, as was reported by Lucas to occur in monkeys, was not noticeable in our human cases.

The cell count was made immediately after the fluid was obtained. The first portion of fluid obtained was used, but controls made by comparing counts of early and late portions of the fluid showed no important discrepancy. While the normal number of cells per cubic millimeter of spinal fluid is usually given as 3 to 5, we have considered the upper normal limit as 10 to 12. In general, it may be said that the highest cell counts are found in the early days of the disease, and that there is a progressive falling off as time advances. Thus of forty-three cases in the first week, twenty-three showed fluids with over fifty cells per cubic millimeter, and thirty-eight with cell counts above normal. Two cases showed in the prodromal period 990 and 650 cells respectively. Of forty-five cases in the second week, in eight the counts were over fifty, and in twenty-three cases they were above normal; of forty cases in the third week in only one was the count over fifty, and in only eight above normal. On the other hand, six cases in the first week, twenty-two cases in the second week, and thirty-two of the forty cases in the third week showed normal counts.

In contrast to the cell count which thus tends to be high in the early part of the disease, the globulin content is usually low in the first part of the acute stage, especially as compared with the cell count. It rises during the second and third weeks and then gradually falls, though frequently globulin is present long after all acute symptoms have passed (text-figure 2). We have seen a strong + globulin reaction on the forty-sixth day of the disease. During the first week, only six of forty-three cases showed at any time a globulin reaction of + (see above) or more. During the second week, twenty out of forty-five cases showed a + reaction; in the third week sixteen out of forty, and in the fourth week nine out of thirty. On the other hand, sixteen cases in the first week showed an absolutely normal globulin reaction. Five cases in the second week and only three in the third week were negative for globulin. Most cases, even on discharge, gave a globulin reaction which varied from

slightly to markedly above the normal. In eleven cases the fluids were followed until both cell count and globulin reaction became normal. The return to normal occurred in the third week in two cases; in the fourth, in two cases; in the fifth, in one case; in the



TEXT-FIG. 2. Chart showing variations in cell count and in globulin content of cerebrospinal fluid in infantile paralysis. — Percentage of cases with cell count above normal. — — — Percentage of cases with cell count above 50 per c.mm. Percentage of cases with globulin s. +. — · — · — Percentage of cases with globulin + or over.

sixth, in two cases; in the seventh, in three cases; and in the tenth week, in one case.

Of the sixty-nine cases studied, only two never showed any abnormal fluids. In each of these cases the fluids were examined but

once, on the twenty-fifth and thirty-third days of the disease respectively. One case showed, in five fluids taken between the ninth and forty-first days of the disease, no evidence of abnormality other than the slightest positive globulin reaction (v. s. +), and another, in three fluids obtained on the ninth, sixteenth, and twenty-third days, gave similar results. Four cases, including one abortive case, showed fluids which, except for the slightest globulin reaction, were normal at the first tapping and subsequently became more definitely positive.

The commonest type of fluid seen in the whole series is one with a normal or slightly increased cell count, and a well marked globulin reaction. There are, however, two other distinct types of fluid seen. One is a fluid with a high cell count, and a normal or very slight globulin reaction; and the other is a fluid with a normal or low cell count, and a very marked globulin reaction. Twenty-six cases showed at some time a globulin reaction of not over s. +, and a cell count of fifty or over. Twenty of these occurred in the first week, and in twelve the count was over 100 cells per cubic millimeter. On the other hand, sixty-two fluids from thirty-four cases gave a globulin reaction of + or more. In thirty-three of these the cell count was normal, and in fifty-five it was not above forty per cubic millimeter. That the two types of fluid are fairly distinct is shown by the fact that only five cases showed fluids with both a high cell count (over fifty) and even a moderately high globulin reaction. Each type of fluid is, moreover, quite definitely characteristic of a stage in the disease. The cellular exudate is almost always associated with the earliest days of the acute stage; the albuminous exudate, with the latter part of the acute stage. It is interesting that of ten cases seen within the first three days after the onset of symptoms, all but one had a cell count of over fifty (one was thirty-seven), and six were over 100. On the other hand, six gave a negative globulin reaction. In four the reaction was s. +.

As regards early diagnosis, the cases examined before the onset of paralysis are of the greatest interest. Six cases were seen in what may be termed the prodromal period. Four of these developed paralysis later and two proved to be of the abortive type. In one case a single lumbar puncture was made on the fourth day

after the onset. The fluid gave a cell count of 650 per cubic millimeter, 90 per cent. polynuclears, and a + globulin reaction. Three days later the child suddenly developed paralysis and in five hours died of respiratory failure. A second case, the sister of a child already in the hospital, was seen on the day after the onset of symptoms. The spinal fluid contained 990 cells per cubic millimeter, 90 per cent. of which were polymorphonuclears, and the globulin reaction was s. +. On the following day one leg was paralyzed and the cell count of the spinal fluid was 627 per cubic millimeter with 80 per cent. mononuclears. On the next day both legs were paralyzed and the cell count was 1,221 per cubic millimeter with 92 per cent.* mononuclears, including many large phagocytic cells. The globulin reaction was + (case 34, page 184). Thus two cases seen very early in the course of the disease showed fluids with unusually high cell counts, and with a marked predominance of polymorphonuclear cells. In one of them the change of the cell picture from the polynuclear type to the mononuclear type could be followed coincidentally with the onset of the paralysis. Three other cases in the prodromal period, one an abortive case and two becoming paralyzed on the day of the first lumbar puncture, gave cell counts of 37, 94, and 113 per cubic millimeter, with 100 per cent., 93 per cent., and 83 per cent. of mononuclear cells respectively, and globulin reactions which fell within normal limits. Finally, in one abortive case lumbar puncture on the second day after the onset of symptoms gave a wholly normal fluid. On the third day the cell count was sixty-two per cubic millimeter with 89 per cent. mononuclears and a s. + globulin reaction. On the sixth day the cell count was fifty-nine per cubic millimeter, all mononuclears, and a globulin reaction of s. +. On the eleventh day the spinal fluid was again normal. That this was, indeed, an abortive case of poliomyelitis was later rendered most probable by a test showing that the patient's blood serum was capable of neutralizing active virus when mixed with it and injected intracranially into a monkey.

In these six cases examination of the spinal fluid gave evidence which was helpful in making a diagnosis of poliomyelitis before the onset of paralysis. Similar evidence was afforded by eleven cases of Lucas and by one of Frissell.⁴⁸ In none of these twelve cases,

⁴⁸ Frissell, *Jour. Am. Med. Assn.*, 1911, lvi, 661.

however, was a large percentage of polynuclear cells found. But it seems of importance to call attention to the fact that a fluid whose cytology resembles that found in cerebrospinal meningitis and other types of purulent meningitis may also occur in the early stages of poliomyelitis.

The question arises as to whether there is any relation between the situation and extent of the paralysis or the severity of the disease and the character of the spinal fluid. Conclusions on this point cannot be drawn from the number of cases seen by us. It would seem, however, that in cases in which the paralysis is limited to arms or to cranial nerves, the lumbar puncture fluid is rather apt to show less deviation from the normal than where the legs are involved. This is, however, far from being a constant finding. Several cases which had acute symptoms (pain, irritability) lasting for an unusually prolonged period, gave an unusually large and persistent globulin reaction. Moreover, the disappearance of the acute symptoms was frequently coincident with a diminution in the globulin in the spinal fluid. The fatal cases showed nothing in the fluid on which to base an unfavorable prognosis.

Since the infecting organism in poliomyelitis is too small to admit of its being seen, even if it is present in the cerebrospinal fluid, and since the more complicated biological tests have failed to prove the presence of antibodies in the fluid, the diagnostic value of examinations of the spinal fluid by simple chemical and microscopic methods must necessarily be merely relative. While in cerebrospinal meningitis, in tuberculous meningitis, in pneumococcus, influenza, and similar types of meningitis, one obtains specific information and is usually enabled to make a positive diagnosis from an examination of the spinal fluid, the value of the examination in poliomyelitis is necessarily less direct. Thus, in a fluid containing a high percentage of polymorphonuclear cells, the failure to find any organisms would certainly be suggestive of poliomyelitis. The difficulty of differential diagnosis is perhaps greater when one has a fluid with a lymphocytic cytology. Lucas has discussed at length the non-specificity of the cytological findings in various meningeal conditions, and finds a similar cell picture in encephalitis, poliomyelitis, meningism, tuberculous meningitis, and syphilis of the

central nervous system. Because, however, the examination of the spinal fluid in poliomyelitis lacks specific diagnostic value, it would be wrong to conclude that it was not an important aid in diagnosis, or that lumbar puncture should not be performed in suspicious cases. Routine blood examinations and the usual urinary analyses have comparatively rarely a specific diagnostic value. Their greatest usefulness consists in helping to rule out certain possibilities, and in focusing the attention on a narrower group. Within such a group the blood or urine examination, partly by itself, and partly when considered with clinical experience, may, by bringing either negative or positive evidence, be the determining factor which points towards the correct diagnosis. The value of positive findings in spinal fluid examinations is undoubted. The value of negative results in the examination approaches it in importance. Thus in the case of poliomyelitis, the failure to demonstrate specific changes in the spinal fluid should rule out several serious meningeal conditions, the differential diagnosis of which from poliomyelitis in the preparalytic stage may be most confusing. On the other hand, by the demonstration of non-specific changes,—for we have seen that by far the greater number of fluids in poliomyelitis are in one way or another abnormal,—a host of other conditions which may simulate incipient poliomyelitis are removed from further consideration. Among the small group of remaining possibilities, certain characteristic types of fluid, as, for instance, one with a high cell count and a normal globulin reaction, may be of some value in deciding the diagnosis. More careful studies of the fluids in conditions other than poliomyelitis may throw further light on the differential value of non-specific fluids. At any rate, when even the non-specific fluid of poliomyelitis is considered in association with the clinical features of the case, the diagnosis can probably be made in the great majority of cases even in the preparalytic stage. The ease and safety with which lumbar puncture can be performed and the simplicity of the examination of spinal fluids should make the method much more widely used than it is at present. The value of any future method of treatment of poliomyelitis must depend on the possibility of early diagnosis, for where the nerve cells have been destroyed, the results from any therapeutic measures will be comparatively small.

Conclusions.—The spinal fluid from cases of acute poliomyelitis during the first few weeks after the onset of symptoms shows, in the great majority of instances, deviations from the normal.

Fluids taken during the early days of the disease and especially before the onset of paralysis tend to show an increased cell count with a low or normal globulin content. At this early stage the polymorphonuclear cells may amount to 90 per cent. of the total. Most fluids, however, show almost exclusively lymphocytes and large mononuclear cells.

After the first two weeks the cell count usually drops to normal, or nearly normal, and there is frequently an increase in the globulin content. A slight increase in globulin may persist for seven weeks or longer.

Analogous changes may be found in the spinal fluid of abortive cases.

All fluids examined reduced Fehling's solution.

The examination of the cerebrospinal fluid in acute poliomyelitis, while giving, as far as is yet known, no specific diagnostic criteria, is of the utmost value as an aid to diagnosis both in preparalytic and in abortive cases (see tables, pages 109, 110, 111, and 112).

PROGNOSIS.

Prognosis in poliomyelitis is a complicated problem. If death and recovery were the only results to be foretold, the matter would be no more difficult than in other infectious diseases, but there is the added uncertainty of the paralyses. In a given case seen in the preparalytic stage, for example, the question at once arises as to whether or not paralysis will appear at all. In another case seen on the first or second day, when there are marked general symptoms and when paralysis of a leg or an arm already exists, it is important to decide whether the process in the spinal cord will probably advance or not. Finally the question of residual paralysis must be considered.

The literature of poliomyelitis is fairly rich in mortality statistics, which form, perhaps, the best basis for prognosis as to life or death. Wickman's series of 868 cases showed a death rate of 16.7 per cent. The figures of numerous other observers are approxi-

TABLE OF EXAMINATIONS OF CEREBROSPINAL FLUIDS.

The numerator of the fraction signifies the day of disease; the denominator is the day of the paralysis. The figure following is the number of cells per c.mm. of spinal fluid. o=normal globulin test; v.s. += faint precipitate (above normal); s. += well marked cloud or slightly flocculent precipitate; += precipitate of large flocculi; and ++= heavy flocculent precipitate with the butyric acid test.

Number of case.	1st week.	2d week.	3d week.	4th week.	5th week.	6th-10th week.
1	$\frac{6}{2}$ 31 v.s. +	$\frac{12}{8}$ 7 v.s. +	$\frac{17}{13}$ 6 v.s. +			
2	$\frac{2}{0}$ 880 + $\frac{1}{1}$ 627 $\frac{2}{2}$ 1,221 +					
3	$\frac{6}{8}$ 7++	$\frac{12}{12}$ 5+				
4	$\frac{7}{3}$ 10 s. +	$\frac{11}{7}$ 46 s. +	$\frac{20}{15}$ 5 s. +	$\frac{27}{12}$ 4 v.s. +		
5					$\frac{31}{2}$ 6 s. +	
6		$\frac{15}{15}$ 4 ++ $\frac{15}{15}$ 4 +++		$\frac{22}{15}$ 6 s. + $\frac{22}{15}$ 7 s. +		$\frac{37}{2}$ 2 s. +
7		$\frac{8}{10}$ 13 ++ $\frac{10}{10}$ 19 s. +	$\frac{16}{14}$ 7 s. +	$\frac{21}{11}$ 6 v.s. +		
8	$\frac{3}{2}$ 37 s. +	$\frac{8}{8}$ 12 s. +	$\frac{16}{15}$ 9 o			
9						$\frac{12}{3}$ 30 + $\frac{12}{3}$ 23 s. +
10			$\frac{16}{14}$ 7 s. +	$\frac{21}{11}$ 3 v.s. +		
11	$\frac{2}{1}$ 120 o					
12				$\frac{21}{11}$ 1 o		
13	$\frac{6}{5}$ 30 s. +					
14		$\frac{10}{3}$ 21 s. +				
15	$\frac{5}{7}$ 60 o $\frac{6}{7}$ 64 s. +					
16	$\frac{5}{4}$ 39 s. +	$\frac{13}{12}$ 13 +		$\frac{21}{11}$ 7 v.s. +		
17			$\frac{18}{15}$ 29 +			$\frac{38}{10}$ 3 s. +
18			$\frac{19}{12}$ 16+++	$\frac{27}{10}$ 20+++		$\frac{39}{10}$ 16 +
19					$\frac{35}{12}$ 165 s. +	$\frac{41}{10}$ 979 s. + $\frac{41}{10}$ 154 s. + $\frac{41}{10}$ 31 v.s. + $\frac{41}{10}$ 5 o

TABLE OF EXAMINATIONS OF CEREBROSPINAL FLUIDS (CONTINUED).

Number of case.	1st week.	2d week.	3d week.	4th week.	5th week.	6th-roth week.
20		$\frac{4}{10}$ 36 + $\frac{1}{10}$ 154 s. +			$\frac{32}{27}$ 12 s. +	$\frac{30}{31}$ 20 + $\frac{47}{42}$ 5 0
21		$\frac{10}{9}$ 10 + $\frac{13}{13}$ 6 +	$\frac{20}{19}$ 6 +	$\frac{27}{26}$ 2 s. +	$\frac{44}{33}$ 11 s. +	
22	$\frac{5}{8}$ 174 0	$\frac{8}{8}$ 44 0 $\frac{10}{8}$ 119 s. +				
23		$\frac{12}{11}$ 11 +	$\frac{20}{19}$ 12 +			
24	$\frac{20}{20}$ 6 0 $\frac{0}{0}$ 62 s. + $\frac{0}{0}$ 59 s. +	$\frac{11}{0}$ 5 0				
25		$\frac{9}{8}$ 12 v.s. +	$\frac{16}{18}$ 8 v.s. +	$\frac{23}{28}$ 10 v.s. +	$\frac{34}{34}$ 4 0	$\frac{41}{41}$ 4 0
26	$\frac{5}{10}$ 16 s. + $\frac{10}{7}$ 10 v.s. +	$\frac{14}{14}$ 10 0				
27		$\frac{11}{10}$ 1 + + $\frac{10}{10}$ 6 +	$\frac{21}{18}$ 2 s. +	$\frac{28}{25}$ 2 v.s. +		
28			$\frac{16}{4}$ 6 + +	$\frac{22}{21}$ 11 s. + $\frac{28}{14}$ 4 0	$\frac{35}{21}$ 10 0	$\frac{44}{40}$ 4 0
29	$\frac{5}{8}$ 10 s. +	$\frac{8}{12}$ 14 + + $\frac{14}{12}$ 3 + +	$\frac{19}{17}$ 2 + +	$\frac{27}{25}$ 5 + +	$\frac{34}{32}$ 5 +	$\frac{43}{40}$ 2 + $\frac{48}{48}$ 3 v.s. +
30	$\frac{4}{7}$ 55 v.s. + $\frac{7}{7}$ 295 s. +					
31	$\frac{4}{0}$ 650 +					
32	$\frac{4}{8}$ 61 0 $\frac{0}{8}$ 147 v.s. +	$\frac{12}{9}$ 7 +	$\frac{19}{18}$ 1 s. +			
33	$\frac{4}{1}$ 160 +					
34	$\frac{7}{5}$ 58 s. +	$\frac{13}{11}$ 23 s. +	$\frac{21}{19}$ 18 s. +			
35	$\frac{5}{8}$ 297 +	$\frac{14}{14}$ 38 + +	$\frac{19}{17}$ 27 + +	$\frac{24}{24}$ 8 +		$\frac{38}{38}$ 13 s. +
36		$\frac{12}{7}$ 26 s. +	$\frac{16}{11}$ 12 +	$\frac{23}{18}$ 7 s. +	$\frac{30}{25}$ 4 v.s. +	
37	$\frac{5}{2}$ 69 s. +	$\frac{13}{10}$ 9 s. +		$\frac{27}{24}$ 8 v.s. +	$\frac{34}{31}$ 3 0	
38			$\frac{15}{21}$ 12 s. + $\frac{18}{18}$ 11 s. +	$\frac{23}{23}$ 14 0		
39		$\frac{12}{11}$ 8 v.s. +	$\frac{17}{16}$ 3 +	$\frac{23}{22}$ 4 v.s. +	$\frac{30}{29}$ 2 v.s. +	
40		$\frac{8}{11}$ 13 s. + $\frac{11}{11}$ 18 +	$\frac{17}{14}$ 7 +	$\frac{22}{19}$ 24 s. +	$\frac{29}{28}$ 3 s. +	

TABLE OF EXAMINATIONS OF CEREBROSPINAL FLUIDS (CONTINUED).

Number of case.	1st week.	2d week.	3d week.	4th week.	5th week.	6th-roth week.
41	$\frac{7}{9}$ 22 0					
42			$\frac{17}{2}$ 4 0	$\frac{27}{7}$ 9 +	$\frac{29}{7}$ 3 v.s. +	
43	$\frac{7}{8}$ 12 v.s. +	$\frac{12}{11}$ 3 +	$\frac{18}{18}$ 11 s. +	$\frac{26}{23}$ 155 +	$\frac{30}{38}$ 29 s. + $\frac{34}{38}$ 49 +	$\frac{47}{46}$ 10 0
44	$\frac{7}{8}$ 39 s. +	$\frac{12}{10}$ 29 +	$\frac{17}{15}$ 53 v.s. +		$\frac{33}{31}$ 12 0	
45	$\frac{6}{4}$ 33 v.s. +		$\frac{15}{13}$ 1 s. +	$\frac{22}{20}$ 3 0		
46	$\frac{6}{0}$ 37 0	$\frac{8}{10}$ 44 v.s. + 10 57 0	$\frac{16}{10}$ 11 v.s. +	$\frac{22}{16}$ 12 v.s. +		
47	$\frac{7}{8}$ 15 +	$\frac{12}{8}$ 12 +	$\frac{19}{15}$ 25 +		$\frac{30}{26}$ 2 v.s. +	
48	$\frac{5}{8}$ 55 0	$\frac{9}{13}$ 15 + 22 +	$\frac{18}{18}$ 2 +	$\frac{27}{26}$ 3 +	$\frac{34}{32}$ 2 s. +	$\frac{38}{47}$ 4 s. + $\frac{45}{45}$ 3 v.s. +
49		$\frac{9}{4}$ 8 v.s. +	$\frac{16}{11}$ 4 v.s. +	$\frac{23}{18}$ 4 0		
50	$\frac{6}{4}$ 42 s. +	$\frac{10}{12}$ 6 + 30 +		$\frac{23}{21}$ 7 +		$\frac{36}{46}$ 4 s. + $\frac{40}{46}$ 6 s. +
51		$\frac{8}{13}$ 83 s. + 23 v.s. +	$\frac{17}{18}$ 11 s. +			
52	$\frac{5}{4}$ 58 s. +	$\frac{9}{8}$ 5 s. +	$\frac{17}{16}$ 6 s. +	$\frac{24}{23}$ 3 +	$\frac{31}{30}$ 4 s. +	$\frac{47}{46}$ 1 v.s. +
53	$\frac{20}{20}$ 113 0 $\frac{239}{239}$ v.s. + $\frac{112}{112}$ v.s. +	$\frac{11}{11}$ 5 s. +	$\frac{15}{13}$ 36 v.s. +	$\frac{23}{20}$ 12 +		$\frac{38}{38}$ 4 v.s. +
54	$\frac{8}{8}$ 133 0		$\frac{15}{14}$ 18 +	$\frac{22}{21}$ 4 s. +		$\frac{40}{36}$ 3 0
55	$\frac{4}{2}$ 20 v.s. +	$\frac{8}{8}$ 55 v.s. +	$\frac{13}{13}$ 10 +			
56		$\frac{8}{16}$ 40 + 35 v.s. +	$\frac{17}{16}$ 9 +		$\frac{31}{21}$ 8 s. +	$\frac{47}{46}$ 6 v.s. +
57	$\frac{3}{2}$ 196 0	$\frac{12}{11}$ 8 s. +	$\frac{20}{19}$ 5 s. +			
58	$\frac{2}{0}$ 94 0					
59	$\frac{6}{8}$ 12 v.s. +	$\frac{11}{11}$ 8 s. +		$\frac{28}{20}$ 8 s. +		
60	$\frac{4}{2}$ 18 0	$\frac{8}{8}$ 90 v.s. +	$\frac{16}{16}$ 3 s. +	$\frac{27}{24}$ 3 0		
61	$\frac{4}{2}$ 203 0	$\frac{12}{10}$ 18 +	$\frac{16}{14}$ 6 +			
62					$\frac{22}{20}$ 2 s. +	
63	$\frac{2}{1}$ 473 s. +					

TABLE OF EXAMINATIONS OF CEREBROSPINAL FLUIDS (CONTINUED).

Number of case.	1st week.	2d week.	3d week.	4th week.	5th week.	6th-roth week
64	$\frac{3}{4}$ 61 0 $\frac{4}{4}$ 320 v.s. +	$\frac{3}{10}$ 60 0 $\frac{1}{10}$ 25 0	$\frac{16}{15}$ 11 v.s. +			
65	$\frac{6}{4}$ 52 s. +	$\frac{13}{11}$ 14 s. +			$\frac{30}{28}$ 7 v.s. +	
66	$\frac{6}{4}$ 22 v.s. +	$\frac{3}{8}$ 114 v.s. +				
67					$\frac{33}{32}$ 4 0	
68	$\frac{6}{2}$ 26 0	$\frac{10}{8}$ 5 +	$\frac{16}{12}$ 5 v.s. +			
69	$\frac{4}{38}$ 90 0	$\frac{10}{9}$ 14 0	$\frac{20}{19}$ 5 0			

mately the same, the usual extremes being 10.8 per cent., reported by Zappert from the epidemic in Northern Austria of 1908, and 22.5 per cent. reported by Lindner and Mally⁴⁹ from the epidemic in East Austria in the same year. The mortality, furthermore, varies somewhat with the age of the patients. In younger children the outlook is better than in older ones and adults. Thus, Wickman found the death rate per cent. in patients below eleven years of age to be 11.9, and in cases between the ages of twelve and thirty-two, 27.6 per cent. Our series of seventy-one hospital cases showed a mortality of about 14 per cent. The figure, however, becomes much lower if the total number of cases applying for admission be used. In this total the death rate is 6.8 per cent. The hospital cases represent, in general, the sickest patients that applied.

While such statistics are of great value in giving a general conception of the fatality of the disease, they are of little help in the presence of a given case of poliomyelitis. This, of course, is true in some measure of all fatal diseases, but with acute poliomyelitis there is a peculiar element of chance not present in other general infections; namely, the accident of the lesion destroying simultaneously the phrenic and intercostal centers, an accident which is invariably fatal. Unfortunately, there is no way of knowing where the lesion will occur, or if an existing lesion will advance. Flaccid legs, arms, or facial muscles point only to the cord segment most seriously injured when the case is first observed, and form no cri-

⁴⁹Lindner and Mally, *Deutsch. Ztschr. f. Nervenh.*, 1910, xxxviii, 343.

terion of the extent or subsequent behavior of the lesion. Anterior horn cells that lie immediately outside the zone involved by the pathological process may continue to functionate properly, so that the proximity of the lesion to the phrenic and intercostal centers does not necessarily make the outlook worse. This and the fact that in most cases the initial lesion in the cord is the final one, and rarely advances, is perhaps the most encouraging knowledge that we command in attempting to make a prognosis as to life; for, as was pointed out in the section on paralysis, death in uncomplicated poliomyelitis invariably results from failure of the muscles of respiration. In other infectious diseases where death has been considered to depend upon toxemia, mortality statistics represent more nearly an average failure of human resistance, affected less, perhaps, by the element of chance. Consequently, at the bedside of a patient acutely ill with poliomyelitis, where the outward signs of the state of the disease are, from the nature of the malady, so unsatisfactory, the physician experiences an unpleasant sense of obscured vision. There are certain clinical features, however, which may help us in some measure to form an idea upon which to base prognosis as to life. Death, according to Wickman, occurs most often on the fourth day of the paralysis, the third to the seventh being the limit. In our cases, death occurred once on the second, four times on the third, once on the fourth, and once on the fifth day of the paralysis. Figuring, however, not in days of paralysis, but rather from the onset of the disease, the fifth day has been the most fatal, with limits from the fourth to the eighth days of the disease. We therefore made it a rule not to declare children out of danger until after the eighth day from the first appearance of muscular weakness.

Usually, the fatal cases are very ill in the first two or three days, and in our series all had paralysis of one or both deltoids; that is, an involvement of the cervical cord. The extreme prostration and the upper extremity paralysis, unless the case was of the rapidly ascending type (Landry), have been the only tangible prognostic features. An impression, however, derived largely from the patient's psychic state, has helped materially. In practically all the fatal cases the peculiarly alert cerebration, described under the sec-

tion on the fatal cases, has been present. None of the profoundly stuporous or highly irritable cases have died. Consequently, we have been glad to see patients in the early days either irritable or drowsy. One case, in particular, recovered, whose rapidly ascending paralysis, first involving both legs, then one arm, then the back and neck, had led us to give a bad prognosis. This patient was very drowsy, and was irritable if disturbed.

Paralysis of either diaphragm or thoracic musculature alone is not necessarily of bad prognostic significance. Children with such involvement, however, are rather prone to develop bronchopneumonia, which is then almost always fatal. Nevertheless, we saw one instance in a boy of twenty-one months, who reached the hospital with a paralyzed diaphragm and a resolving consolidation of lobar pneumonia. He made a good recovery.

Another equally difficult question in the prognosis in poliomyelitis is that which arises during the preparalytic stage; namely, will paralysis occur, or not? As in the rapidly advancing cases, where it is impossible to know whether or not the respiratory muscles are about to fail, so here the same sense of obscured vision baffles the physician. We have found absolutely no certain way of anticipating paralysis. Occasionally the patient may complain of pain in a member which is subsequently lamed. This is unsatisfactory, however, because pain in general is such a common feature of the disease. Furthermore, the disappearance or absence of a knee jerk is no infallible signal of approaching weakness. One abortive case which we observed practically from the day of onset throughout the course, was the source of hourly apprehension for eight days. At one time, on the third day of the disease, the knee jerks, which had been exaggerated, became much less easy to obtain. Three days later the left knee jerk reacted on reënfacement only, but no paralysis developed. In a similar way it is impossible to determine whether or not an advancing paralysis is about to stop. As a rule, although figures on this point are inaccurate and unconvincing, our experience has been that the initial paralysis is final. Nevertheless, there are enough examples of late involvements to make the anticipation of further paralyzes quite justified at any time until the seventh or eighth day.

A discussion of the prognosis in poliomyelitis necessarily involves a consideration of the ultimate disability caused by the disease. It was formerly supposed that the paralysis was always permanent. Wickman's extensive studies, however, and the report of the Massachusetts State Board of Inquiry show that this fortunately is far from the truth. For example, of the 530 cases, one to one and a half years after the acute attack, which were analyzed by Wickman, 56 per cent. were paralyzed, and 44 per cent. were cured. The records from Massachusetts indicate a considerably lower percentage of complete recoveries,—16.7 per cent. Complete return of power is more apt to occur in children than in adults. It is still too early to be able to report figures of value from our hospital cases. We have, however, been much struck by the surprising return of power which may occur after a few weeks in limbs that seemed hopelessly paralyzed, and we have been even more impressed with the marked improvements occurring after several months of a stationary condition of the paralyses.

TREATMENT.

At the present time there is no specific form of therapy by which the paralyses in acute poliomyelitis may be prevented, or by means of which resolution of the inflammatory process and, consequently, return of function may be hastened. The problem of treatment, therefore, consists in preventing the spread of the disease to other persons, in applying general symptomatic procedures, and in attempting the restoration of muscular efficiency and the prevention of deformities.

Inasmuch as the direct contagiousness of poliomyelitis and its dissemination by healthy intermediaries are now definitely established facts, the maintenance of strict quarantine is essential for the public health. In this disease, which in its sporadic form has been with us so long, and towards which there is apparently a high individual immunity, it is peculiarly difficult to convince many persons, both lay and medical, that a quarantine is not somewhat superfluous. Recent investigations, however, which show that in some epidemics the number of families or houses with more than one case may reach 40 per cent.; that "persons have been attacked by poliomyelitis

several days after a short and single contact with a patient";⁵⁰ that markets, fairs, schools, and public gatherings may be the means of spreading the disease; that transmission by fomites probably occurs; that the virus may exist in the saliva and nasal mucous membrane in monkeys and in the tonsils in man; and that it may also be found in the dust from the rooms of patients, have convinced public health authorities that poliomyelitis should be treated like any other definitely contagious disease. In Sweden, Norway, Germany, many provinces of Austria, and in a number of states in this country, poliomyelitis is one of the diseases in which notification, quarantine, and disinfection are required by law. We have already mentioned the outbreak in Nebraska in 1909, which was apparently checked by imposing an absolute quarantine for three months on all members of a patient's family with the exception of the bread winner. The Paris Academy of Medicine recommends the passage of a law which would "allow the interdiction of school attendance for three months, and would apply equally to patients and convalescents." It is probable, however, that the general enforcement of such prolonged quarantine would work hardships which would scarcely be compensated for by the results obtained. The question as to how long the disease ought to be considered contagious is a most difficult one. That the disease is contagious during the prodromal stage and perhaps also during the incubation period is, unfortunately, extremely probable. Some authorities believe that isolation should be continued through the acute stage of the disease, until the subsidence of pain, hyperesthesia, and all acute symptoms. In our own work we have drawn a rather arbitrary line based on the fact that in the experimental disease in monkeys, the virus rarely persists after three or four weeks. That it may persist longer in monkeys, and probably also in human beings, is of course certain, but we have felt that this gave the best practical rule for hospital purposes. We have endeavored, therefore, to isolate the children in the hospital until about four weeks after the onset of the disease. We have then allowed them to go home, but have urged that, when possible, they be kept apart from other small children for several weeks more.

⁵⁰ Notification of Poliomyelitis—Translation of a Report Presented to the Paris Academy of Medicine on "Obligatory Notification of Poliomyelitis (Infantile Paralysis)," Public Health Reports, 1911, xxvi, 1602.

During their stay in the hospital, the patients with poliomyelitis were kept in an isolation ward apart from all other patients. Suspected cases with uncertain diagnosis, or cases proving not to be poliomyelitis were placed in separate rooms and completely isolated. Nurses on duty in the ward never came in contact with patients in other parts of the hospital, and the same was usually true of doctors. All doctors and nurses wore caps and long gowns when working with the patients. When leaving the ward, the hands were thoroughly scrubbed with soap and a nail brush, and soaked in corrosive sublimate. The occasional use of a hydrogen peroxide gargle or nasal spray (1 per cent.) was recommended. Visitors were admitted on two days in the week, but only one visitor to a patient. All visitors were compelled to wear gowns while in the ward, to wash carefully and disinfect the hands in corrosive sublimate, and use a hydrogen peroxide mouth wash on leaving the ward. The clothes worn by patients on admission were disinfected by steam under pressure. Bedclothes and nightgowns were passed through a sterilizing washer. Thermometers were kept in corrosive sublimate. Bedpans and urinals were boiled after using. All urine and feces were sterilized by heat in a steam sterilizing hopper. After being used, the rooms were disinfected with formaldehyde gas, and the walls and floors were thoroughly scrubbed with soap and water.

The general treatment of the acute stage of the disease as regards diet, bathing, catharsis, etc., should be carried on exactly as in other infections. As soon as the children showed a desire for a liberal diet, it has been given to them. After they have passed the early acute stage, they are apt to have unusually large appetites. During the acute stage absolute rest in bed is, of course, a necessity in most cases, but even when the children feel well, it is probably wise to keep them quiet for a week or two after the onset of paralysis. At this early period, the most prominent indication for treatment is usually pain. In some instances, as we have seen, pain may be spontaneous, but much more frequently it is associated with movement of the limbs, and this necessitates the utmost gentleness and care in moving or turning the child and changing the bed linen. When lifting or moving a patient with marked hyperesthesia, the nurse should try to avoid flexing the neck or the hips, or bend-

ing the spine forward, as these are the most painful motions. Not infrequently, this pain is so great that the children develop a dread of being touched, and they cry out long before they are actually hurt. The nurse who realizes that their pain may be intensely severe, whose touch is gentle, and whose voice is reassuring, may do much towards preventing this unhappy state of mind. In patients with spontaneous pain or with tender muscles, the weight of the bedclothes may be sufficient to cause discomfort, and a cradle to raise them off the limbs is often a relief. In other cases a light, well padded splint seems to steady the limb and to take the strain from unaffected muscles. One of our children used to beg to have the splint replaced when it was taken off. In general, the most effective simple agent for overcoming pain seems to be heat. This is especially the case in paralyzed limbs which are often very cold and clammy. Wrapping the limb in cotton wool, blankets, hot packs, and hot water bags gives great comfort. We have frequently had occasion to notice the effect of cold weather and dampness on these patients, when a sudden change from warm summer weather to a cold rainy day would be accompanied by definite exacerbations of pain. The ward was, therefore, kept at a warm, equable temperature. A certain proportion of cases suffer so intensely that simple measures are not sufficient to control the pain, and drugs have to be resorted to. Bromides we have found helpful in little children. In older patients phenacetine, aspirine, codeine, and occasionally hypodermic injections of morphine are necessary. We have never felt convinced that lumbar puncture was of any therapeutic value. In one fatal case life was apparently prolonged for a short time by means of artificial respiration. Landolt⁵¹ reports a case in which artificial respiration was carried on almost constantly for seventy-two hours. During this period there was slight return of power in the toes and fingers, but the patient finally died. Theoretically, it would seem that one might possibly tide a person over by artificial respiration until an absorption of exudate allowed the nerve cells controlling the respiratory muscles to resume their function.

During the early part of the disease the most important indica-

⁵¹ Landolt, *Cor.-Bl. f. Schweiz. Aerzte*, 1911, xli, 1144.

tion is for complete rest and quiet. When the acute symptoms pass, however, more active treatment directed towards the prevention of deformities and the restoration of muscular function should be instituted. The exact period at which it is best to begin treatment varies, but in general the pain seems to be a good guide. As soon after the first fortnight as the child can bear any movement or handling without pain, massage should be begun. It is important, although sometimes difficult, to distinguish between true pain and the fear of pain which may persist until the child's self-confidence is restored.

Contractures begin to develop early in poliomyelitis, and it is essential that they should be guarded against from the onset. The most common early deformity to be dealt with is the toe drop, occurring from the contraction of the flexors of the toes and extensors of the foot in the absence of normal opposing muscles. Such a deformity is hastened by the pressure of the bedclothes, so that a cradle is useful where it is feared. Sand bags may be used to support a limb; or light apparatus, such as a posterior wire splint with a foot-piece at right angles, may be sufficient to overcome the pull of the muscles. Such apparatus must be well padded and the straps applied loosely so that there is no obstruction to the circulation. The danger of apparatus is that it will keep the limb too quiet and prevent any attempt to use weakened muscles at precisely the period in which both active and passive motions are most to be encouraged. To obviate this, it has been our custom to have the apparatus applied at night only, and to leave the limbs free to move during the day time.

Massage is a most helpful aid to treatment. We have begun with light massage as soon as the pain would allow, and given it twice daily. Its chief usefulness probably consists in assisting the circulation by replacing the effect of lost muscular activity. Heat, and especially baking, also exert a good influence on the circulation. With massage, passive motion is used, the rhythmic performance of certain movements stimulating the patient to try to attempt them himself. Of all methods, however, by far the most valuable one is active movement, or muscle training. In attempting voluntarily to use a weakened or paralyzed muscle, the patient can accomplish

more for himself than can ever be done for him by anyone else. Muscle training is comparatively easy to carry out with adults or children old enough to do what they are told, but with little children, who by instinct avoid the use of any injured muscle, and try only to accomplish the same result by means of another healthy group of muscles, the problem is more difficult. In one way or another, the child must be made to attempt to use the weak muscles. It is here that the skill of the nurse shows itself. By merely telling the child to move an arm or leg, she accomplishes nothing, and as likely as not merely makes him suspicious and obstinate; but by playing with him, coaxing him, inventing ingenious games, which involve the use of muscles without his realizing it, and by showing infinite patience and tact, the end is obtained. Frequently, the children use their limbs better when they are in a warm bath than when in bed. The excitement of the tub bath and the fun of playing with floating toys makes them forget their disabilities, and they try new movements much more readily than in bed. Probably also, the heat and the buoyancy of the water make many muscular movements less difficult. Some children are ambitious and from the outset are continually anxious to progress, but with many each new step involves a struggle. One literally has to force them to try to sit up, to try to stand, and then to try to walk. Dr. Charles West⁵² discusses some methods of stimulating children to use their paralyzed limbs and then adds, "I need not say that much care and much patience are needed in carrying out any of these suggestions, and not a little of that intuitive love for children which teaches those who are its possessors how to extract fun and merriment from what might in other hands be a most irksome task."

In the therapeutic use of electricity, we have had little personal experience. To be of any value, electrical treatment must be given with considerable skill and over a long period of time. The amount of other work entailed by the large number of patients we have had in the hospital left us no opportunity to take up this method of treatment. The general methods which may be of service, however, are summed up in the following sentences from the report of the

⁵² West, *Lectures on the Diseases of Infancy and Childhood*, 3d edition, London, 1854.

Massachusetts State Board of Health⁵³ for June, 1909: "In the early stages, galvanism should be used on the nerve trunks and faradism on the muscles, so long as their irritability for contraction is maintained. When the irritability of contraction to the faradic is lost, galvanism should then be used, as having more influence on nutrition. With the returning muscle irritability, faradism should be used, and best by the use of the electrodes over the muscle points so as to obtain actual contraction of muscles rather than by the application of the electrical current to broad surfaces. This serves as a distinct exercise to the muscle during its early stage of weak contraction."

The results of treatment depend to a great extent on the faithfulness with which it is persisted in. Coincident with the absorption of the exudate, improvement is often quite rapid during the first weeks and months; then follows a long period which may be intensely discouraging in its apparent results, a period in which improvement is probably due to muscle training and to the assumption of new functions by the surviving nerve cells. During this period the patient learns many tricks by which surviving muscles may be made to assume in part the rôle formerly played by those which have become paralyzed. A slow recovery often continues for a very long time, and to be sure that the muscles have regained all the power that they are capable of, conscientious treatment ought to be continued for at least a year and a half or two years. We have been greatly impressed with the marked improvement which may come after the end of the first year. The problem as to when the case ceases to be a medical one and should be turned over to the orthopedic surgeon is difficult and much discussed. The danger of a too early resort to surgery and mechanical methods of support is that the patient will come to rely too much on these methods and cease developing his muscles to their utmost. Internists and neurologists thus tend to postpone calling in the orthopedic surgeon until they feel satisfied that they have done all in their power. The danger of this delay, on the other hand, is that deformities may set in which make the corrective problem much more difficult, so that the orthopedic surgeon receives the case at a distinct disadvantage.

⁵³ Report of the Massachusetts State Board of Health, *loc. cit.*

The best treatment would probably mean a coöperation in which the surgeon would foresee and correct deformities and the internist would protect the interest of the injured muscles.

When one member of a family is taken sick with poliomyelitis, the physician must take what measures he can to prevent the spread of the disease to other members of the family and to the community at large. Strict isolation of the individual case and isolation of those who have been in close contact with it during the fortnight preceding onset, are the most efficient measures. This is particularly important with regard to the prevention of contact with children. Attendance at school, church, or other places of public meeting should be forbidden for at least three weeks. As direct treatment for those exposed, urotropin, which by animal experimentation has been shown to have some slight action in destroying the virus, may be given for about a week. Longer administration is not advisable. Nasal and throat sprays or a gargle of 1 per cent. peroxide of hydrogen or of a 0.5 per cent. menthol solution should be used, but only for a few days, for their prolonged use is irritating and may make the throat and nasal passages more susceptible.

CASE HISTORIES.

CASE I.

Sherman R., age, 17 months. Ridgewood, L. I. Admitted, September 20, 1911. Discharged, November 8, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative.

Past History.—The child has never been sick before.

Present Illness.—Thursday morning, September 13, seven days ago, the patient was perfectly well. In the evening he vomited some chocolate which he had eaten. There was high fever that night and all the next day. The child was drowsy and wanted to be let alone. He had no convulsions, but is said to have been "twitchy." On Saturday his fever fell. The temperature was normal on Sunday, and the doctor thought the child would be well. He was drowsy and dull, however, all day. On Monday he seemed to have difficulty in swallowing and later in the day the doctor noticed that he could not use his legs. He has been constipated. The child is teething.

Physical Examination.—The patient is a well nourished, drowsy, small boy, who is fairly easily aroused and resents being handled. His head is usually held on one side and slightly retracted. *Eyes*.—Pupils are equal and react normally. Ocular movements are normal. *Ears*.—Normal. *Nose*.—No discharge. *Mouth*.—The tongue is heavily coated. Mucous membrane is clear. Pharynx is reddened;

tonsils are a little enlarged. *Superficial lymph nodes*.—The posterior cervical, axillary, and inguinal nodes are palpable and slightly enlarged. *Neck*.—There is a little tendency to retraction. The neck is so stiff that the body can be lifted by the head. This causes pain. *Thorax*.—Well formed, moves properly in respiration, though not widely. *Lungs*.—Clear. *Heart*.—Regular and not rapid. *Abdomen*.—A little full, but not distended, soft, tympanitic, no tenderness, and no masses. Abdomen moves properly in respiration. Liver and spleen are not felt. Abdominal reflex is very faint; cremasteric present. *Extremities*.—Upper apparently normal. Lower: there is complete flaccid paralysis of the right lower extremity, and complete flaccid paralysis of the left lower extremity, except for a little power of flexion of the toes and extension of the foot. Knee jerks are absent on both sides. Achilles reflex is present (?) on the right, absent on the left. Babinski reflex is absent on both sides. There is no resistance on testing for Kernig's sign and no spasm, but there is pain at the end of the manipulation. *Temperature*.—98.8° F.

September 20. Blood count: leukocytes, 13,400; differential count of 200 cells: polymorphonuclears, 67.5 per cent.; transitionals, 0; lymphocytes, 28 per cent.; basophiles, 0; large mononuclears, 7 per cent.; eosinophiles, 2 per cent.; stimulation form, 0.5 per cent. Spinal fluid: 30 c.c. of clear, limpid fluid; pressure slightly increased (child crying); cells, 15; globulin, +; sugar, +.

October 19. During the past two or three days there has been a striking improvement in the patient's general condition. He is bright and plays. He is also able to sit up alone in bed without the support of pillows. There is no return of power in the paralyzed muscles.

October 25. There is return of power in the flexors of the toes on the left and in the iliopsoas. The adductors are still paralyzed. The general condition is very good.

CASE 2.

William S., age, 2 years. New York City. Admitted, July 14, 1911. Discharged, September 13, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat*: the family lives in three rooms on the first floor in the rear. Air and light are good. The baby sleeps with the mother. No other children in the neighborhood are paralyzed.

Past History.—The baby has always been healthy.

Present Illness.—The child was taken sick July 7, one week ago. He seemed to have fever. The mother gave him castor oil and spirits of nitre, and the fever seemed to leave him. The baby went outdoors and played. Two days later he became feverish again. He was put to bed and given warm baths. The mother first noticed weakness in the legs two days ago. This came on in both legs at the same time. He limped on trying to walk, and said his foot hurt. It was first noticed this morning that the baby could not move his legs at all. There has been no nasal discharge, no eruption, no vomiting, and no chill. He has not cried, has slept most of the time, and has been dull. The bowels have been slightly constipated.

Physical Examination.—The child is a mulatto, lies quietly in bed, awake, with head turned to the left and slightly retracted. The skin is moist and warm. *Head*.—The head is well formed, fontanelle closed. Frontal eminences are

prominent. *Eyes*.—No ptosis. Scleræ and conjunctivæ are clear. Movements of the external ocular muscles are good. Pupils are equal, not dilated, react to light. *Ears*.—No discharge. No mastoid tenderness. *Mouth*.—Lips and mucous membranes are of good color. Teeth are well developed and in good condition. The tongue has a moist, white coat. Tonsils and pharynx cannot be seen. *Neck*.—The child keeps his head somewhat retracted and resists extension slightly. Other movements are easy. *Superficial lymph nodes*.—The cervical, axillary, and inguinal glands are all enlarged, easily palpable, somewhat shotty, and freely movable. *Thorax*.—The thorax is fairly well formed, symmetrical, and moves easily with respiration. The costal angle is very wide, with some flaring of the costal margin on both sides. There is some enlargement of the costochondral joints. Breathing is not rapid, rather shallow. Expiration is about equal on both sides. The lungs are everywhere resonant on percussion and are clear on auscultation. The breath sounds are easily heard and are vesicular in character. *Heart*.—Negative. *Abdomen*.—Protuberant and is everywhere soft; there is no resistance or tenderness, no masses, some rigidity in the region of the bladder which seems full. *Liver*.—Dullness extends from the fourth interspace to 2 cm. below the costal margin in the mammary line where the edge is easily palpable. *Spleen*.—Not felt. *Extremities*.—Upper: the movements of both arms seem normal, and there is no apparent weakness, the biceps reflex is easily obtained on both sides. Lower: both legs are perfectly limp and show a complete flaccid paralysis. On stimulating the soles of the foot, although the child cries and tries to draw the foot away, he is not able to do so. The patellar and Achilles reflexes are absent on both sides. Plantar stimulation gives plantar flexion of the great toes on both sides. *Sensorium*.—No areas of hyperesthesia can be made out. *Temperature*.—100.8° F.

July 15. Blood count: leukocytes, 20,000; differential count of 200 cells: polymorphonuclears, 57 per cent.; eosinophiles, 6.5 per cent.; small mononuclears, 23.5 per cent.; transitionals, 4.5 per cent.; large mononuclears, 8.5 per cent.; mast cells, 0; red cells, normal in size and shape, no nucleated forms seen.

July 16. Spinal fluid: 20 c.c. of clear, colorless fluid; pressure not especially increased; cell count, 36 per c.mm.; stained smear, mononuclears, 98 per cent.; polynuclears, 2 per cent.; globulin, +; albumin, +; sugar, +.

The child lies with great retraction of the neck and when turned on his back assumes a position of lordosis. He complains of much pain when handled, especially in the legs. No sign of involvement of the neck muscles, arms, back, or abdomen.

July 19. The neck still resists flexion. Von Pirquet reaction is negative.

July 30. The neck is still resistant, but less so. There is no retraction.

August 4. Back and neck muscles are weak; child cannot sit up well. The neck still resists flexion, there is no retraction. Abdominal muscles: on sitting there is bulging of the sides of the abdomen, as if the recti had their normal tone, and the other abdominal muscles were weak. Blood count: leukocytes, 18,100; differential count of 200 cells: polymorphonuclears, 58 per cent.; transitionals, 11.5 per cent.; lymphocytes, 21.5 per cent.; basophiles, 0; large mononuclears, 5.5 per cent.; eosinophiles, 3.5 per cent. Spinal fluid: 15 c.c. of perfectly clear, watery fluid; no blood; pressure not increased; cell count, 20 per c.mm.; globulin, ± (slight flocculent precipitate); sugar, +; smear, all small mononuclears; a few red cells.

September 13. There is very slight power of contraction in the left iliopsoas, also in the flexors of the toes; otherwise there is complete flaccid paralysis of the left leg. There is complete flaccid paralysis of the entire lower right extremity, the knee jerk is absent. The patient can sit up well and is very strong in the trunk and arms. The abdomen is large and prominent. The recti seem to be stronger than the lateral muscles. There is no more pain on spinal flexion. The patient's general condition is good. He is fat and his flesh is firm.

CASE 3.

Leo S., age, 4½ years. Port Richmond, S. I. Admitted, July 15, 1911. Discharged, August 22, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat*: the family lives in a tenement with four other families. The house is clean. The child has one cousin who had infantile paralysis eight years ago. This girl has seen the child once since it became sick, but previously had not seen the child for a long time and very rarely came in contact with it. There is no history of any other possible contact.

Past History.—Three years ago he had bloody dysentery followed by dropsy and swelling of the feet which lasted two weeks. He had whooping cough as an infant and measles at two years.

Present Illness.—Three weeks ago the child was taken with a headache and fever. The next day he fell on the floor and hurt his heel. Since then he has not been able to stand on his heel, but only on his toes. Two days later he "took a sleepy spell" and would not eat. That night both legs gave way when he was trying to stand. The next morning the doctor saw him and made a diagnosis of sprained ankle. The mother noticed that both legs were powerless. Since then he has been in bed. Two days after the onset of paralysis, he began to complain of pain in the legs. He has not been especially restless, he sweats a good deal after the onset. He has not vomited and has had no chill. On July 1 he was unable to pass urine and on July 2 was taken to the Smith Infirmary. While in the hospital he had to be catheterized for two or three days. Since July 2 he has been at the Smith Infirmary. The mother never noticed that the child was cross-eyed. The patient was transferred here from the Smith Infirmary.

Physical Examination.—The patient is a well developed and nourished boy. The skin is clear and warm, lips and conjunctivæ are of good color. *Eyes*.—The eyes move normally. There is an internal squint in the right eye. Pupils react normally. *Ears*.—Normal. There is no tenderness over the mastoids. *Nose*.—No discharge. *Mouth*.—The tongue is clean and protrudes straight. Teeth are in good condition. Tonsils are slightly enlarged but not ragged. *Neck*.—The neck resists flexion but there is no retraction. *Superficial lymph nodes*.—There are very small lymphatic glands felt in the neck, axillæ, and groins. *Chest*.—Symmetrical and well developed. *Heart*.—Normal. *Lungs*.—Negative. *Abdomen*.—Full, soft, symmetrical, and tympanitic, no masses or tenderness, abdominal reflexes are active. *Liver*.—Flatness extends from the sixth rib to the costal margin. *Spleen*.—Not felt. *Muscular system*.—Arms: muscular movements are normal. Legs: knee jerks, Achilles, Babinski, and Oppenheim reflexes not obtained on either side. Kernig's sign: there is no true limitation of motion, but flexion of the hip with extended leg is very painful. There is no pain on pressure except perhaps over the left calf. On extension of both legs he complains

of pain in the popliteal spaces. *Paralysis.*—There is apparently complete flaccid paralysis of both legs. Child is unable to move any of the muscles. He cannot wriggle his toes and cannot flex the thighs on the hips. There is double toe drop. The child is unable to sit up on account of the pain in his legs. There is apparently no weakness in the muscles of the back. *Temperature.*—99.2° F.

July 16. The temperature is normal, chest clear, and general condition very good. There is a slight internal squint of the right eye.

July 17. Blood count: leukocytes, 12,000; differential count of 200 cells: polymorphonuclears, 58.5 per cent.; small mononuclears, 27.9 per cent.; large mononuclears, 9 per cent.; eosinophiles, 3.5 per cent.; transitionals, 2 per cent. red cells normal; platelets normal.

Spinal fluid: 25 c.c. obtained; pressure slight; slightly bloody; specimen used for cell count was very opalescent; an almost turbid fluid with only a very slight pink tinge. Cell count, 529 per c.mm.; differential count of 200 cells; polymorphonuclears, 45 per cent.; mononuclears, 55 per cent.; globulin, ++; albumen, +; sugar, +.

July 20. Paralysis is unchanged, and is complete in the legs. There is marked pain in the back of the thigh on moving the right leg, and there is also pain on dorsal extension of both feet with legs extended.

Spinal fluid: 25 c.c. of opalescent fluid of slightly yellowish tinge; slight increase of pressure; no clot formed after 24 hours; cell count, 524; smear; differential count of 200 cells: polymorphonuclears, 60.5 per cent.; mononuclears, 26 per cent.; endothelial, 13.5 per cent.; globulin, +; albumen, + (faint cloud); sugar, +.

July 21. The temperature this evening is 101.8° F. The child has a headache and feels miserably. As soon as he is moved he complains of severe pain. This is localized, at times in the legs, at times in the back, and frequently in the abdomen in the region of the navel. It is difficult to make out the cause of the pain, but it is apparently associated with movements of the legs.

July 22. The temperature was normal this morning but has risen again to 103.4° F. The child is very uncomfortable when moved. The neck is quite stiff. The ears, eyes, and throat are negative. Heart and lungs (front) are negative. The abdomen is negative. Knee jerks are absent. It is difficult to localize the pain. He says it is worse at the inner side of the left knee, just below the inner tuberosity of the tibia, but when he is moved he has great pain in the lower part of the back, and back of the thighs. There is no especial tenderness. Leukocytes, 15,100 (82 per cent. polymorphonuclears—wet count).

July 23. The temperature has come down. The child cried a great deal during the night, partly from pain and partly not. The lungs are clear; the abdomen is soft with no tenderness.

July 24. Leukocytes, 14,800 (85 per cent. polymorphonuclears—wet count). The neck is very stiff and markedly retracted and is painful on flexion. Kernig's sign: there is much pain in the legs on an attempt to elicit the reaction; no spasm of muscles.

July 27. The fever is less, and the pain less. Extension of the legs is still painful, and the legs are somewhat tender; there is no retraction. Flexion is less painful, but complete flexion is distinctly painful and is resisted. Chest and abdomen are clear. There is marked atrophy of the legs.

July 28. Blood count: leukocytes, 6,300; differential count of 200 cells: polymorphonuclears, 62.5 per cent.; small mononuclears, 19 per cent.; large mononuclears, 12 per cent.; mast cells, 1 per cent.; transitionals, 3.5 per cent.; eosinophiles, 2 per cent.

July 30. The child looks much better. The head flexes more easily, but flexion is still painful.

August 9. Spinal fluid: 25 c.c. of perfectly clear watery fluid; pressure slightly increased; cell count 30 per c.mm.; smear: small mononuclears, 82 per cent.; large mononuclears, 18 per cent.; globulin, + (slight flocculent precipitate); sodium chloride, 0.74 per cent.

August 16. Spinal fluid: 20 c.c. of perfectly clear watery fluid; no blood; pressure slightly increased; cell count, 23 per c.mm.; mononuclears, + (occasional polymorphonuclears); globulin, slightly +; sugar, +.

August 18. Blood count: leukocytes, 10,700; differential count of 200 cells: polymorphonuclears, 52.5 per cent.; small mononuclears, 35.5 per cent.; large mononuclears, 9 per cent.; eosinophiles, 0.5 per cent.; transitionals, 2.5 per cent.

August 20. The child is much better and sits up very well. The chest is clear. There is no pain on flexion of the neck and no pain in the legs. The abdominal and back muscles are normal. He can flex the toes of the left foot, otherwise there is complete paralysis of both legs.

August 22. The general condition is much improved. The child has no pain and sits up well, but cannot stand. There has been practically no return of movement in the legs.

CASE 4.

Hannah T., age, 4 years. New York City. Admitted, October 3, 1911. Discharged, October 29, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Past History.—The patient has always been a strong healthy child.

Present Illness.—On Sunday, October 1, two days ago, the child was perfectly well in the morning. In the afternoon she was "kind of heavy and wanted to go to bed." That evening she vomited, slept poorly, and was restless. On Monday morning, when she got up and tried to walk, she fell. She tried again to walk, but again fell. Later in the day she said she felt well and wanted to get up, but on trying to walk, fell a third time. This morning she vomited. She has had no convulsions, has been constipated, and sweat heavily on Sunday night. She is said to have had fever.

Physical Examination.—The patient is a well nourished little girl who lies quietly on her back and tends to be drowsy. When she is aroused, she becomes greatly excited, frightened, and trembles. She objects to being handled. The breath has a heavy, sweetish odor. *Eyes*.—Pupils are equal, react normally. Ocular motions are normal. There is marked photophobia. *Ears*.—Normal. *Nose*.—There are a few, small, dried scabs on the inner margin of the external nares, no secretion. *Mouth*.—The lips are dry, the tongue heavily coated. Buccal mucous membrane is clear. Pharynx is slightly reddened. Tonsils are not very large. *Superficial lymph nodes*.—The posterior cervical, axillary, and inguinal nodes are palpable. *Neck*.—Anterior muscles seem a little weak. Posterior muscles resist attempt at flexion. There is no retraction. *Thorax*.—Well formed, moves properly in respiration. *Lungs*.—Clear. *Heart*.—A little rapid,

otherwise normal. *Abdomen*.—Soft, not distended, no tenderness or masses. *Liver*.—Edge felt about two fingers' breadth below costal margin. *Spleen*.—Edge just felt below costal margin in anterior axillary line. *Extremities*.—Upper: normal. Lower: there are no paralyses, but there is inability to stand. Both hip and knee joints buckle when weight is put on the lower extremities. There is apparently no definite weakness of the glutei, so that the trouble probably lies in weakness of the quadriceps extensors. Knee jerks and Achilles reflexes are absent on both sides. Babinski reflex is absent on the left and on the right. Kernig manipulation is not resisted and causes very little pain. *Temperature*.—101.2° F.

October 3. Blood count: leukocytes, 17,600; differential count: polymorphonuclears, 44.5 per cent.; lymphocytes, 37.5 per cent.; large mononuclears, 3 per cent.; transitionals, 14.5 per cent.; basophiles, 0; eosinophiles, 0; stimulation form, 0.5 per cent.

Spinal fluid: 20 c.c. of clear limpid fluid; pressure not increased; cells, 196; mononuclears, ++; globulin, slightly hazy; sugar, +.

October 5. The child is very irritable and complains of pain on being moved. The left ankle and foot are especially painful. The right quadriceps is apparently paralyzed, also the right peroneal group. The child lies on her side with marked retraction of the neck. There is great pain on flexion of the legs. Examination is difficult because of great irritability and the rigidity with which the body is held.

In the afternoon, the patient complains bitterly of pain in her feet. Complete paralysis is evident in both quadriceps.

October 6. The patient is lying quietly on her left side with head retracted. She is apparently not suffering from pain at this moment. Flexion of the neck is painful. The legs are not especially tender on pressure, but are very painful when she makes an attempt to move.

October 8. The pain seems to be somewhat less, but at times is so severe that she cries out from it. Both legs are very tender to pressure, apparently more so over the nerves. Movements of the legs are very painful, in fact, her legs have to be left in one position and motion cannot be tested.

October 9. The pain is perhaps a little less, but she cannot bear to have her legs moved.

October 10. There is pain when the muscles are squeezed.

October 12. The child still has much pain. She is very much afraid of being touched, as she has had so much pain, and it is hard to tell just how much is pain, and how much is nervousness. She has suffered so that she does not tell the nurse when she wishes to void, or when her bowels move, apparently dreading being put on the bed pan.

Spinal fluid: 15 c.c. of clear watery fluid, no increased pressure; cell count, 8 per c.mm.; globulin, slightly +; sugar, +.

October 14. The child is much better. For the first time she responds when spoken to and gives her hand. The arms move normally. Flexion of the neck is painful. Movement of the legs is still very painful, so that they cannot be tested for paralyses and reflexes.

October 18. The patient says she has not so much pain in the legs this morning, though she complained of it last night. If they are manipulated, however,

she cries out. The left knee jerk is present; the right, absent. The patient apparently has little power over the lower extremities. Flexion of the neck causes pain and the patient resists actively all attempts to make her sit up. She throws the head far back when this attempt is made.

October 20. Spinal fluid: cells, 5 per c.mm.; mononuclears, +; sugar, +; globulin, slightly +.

October 29. There is surprisingly little improvement in the tenderness of the spine and legs. The slightest movement towards flexion of the thigh causes intense pain, and the patient will not and probably also cannot move the lower extremities. The general condition is better. The patient is less moody.

October 29. The patient's mother insisted on taking her home. When the mother dressed her, the child did not complain of pain as she does when doctors or nurses touch her.

CASE 5.

* John O., age, 21 months. New York City. Admitted, July 25, 1911. Discharged, September 23, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in a tenement house. There are eleven other families in the house. They have five rooms on the ground floor. There is good light and fair ventilation. The only parasites that have been noted are bedbugs. The father states that these are worse in the children's room. The father is in good financial circumstances, is a fireman and has been working on a fireboat for the past eleven months. In his duties he has not been called into any house where there was infantile paralysis; in fact, he has been on the boat constantly.

Past History.—The child has grown well, and has always been in excellent health.

Present Illness.—Friday evening, July 21, four days ago, the child was slightly feverish and his face flushed. He took his supper as usual. The next morning he was still slightly feverish but took his breakfast. He complained of headache and pain in the stomach but could walk as well as usual. In the afternoon the mother took the child out in a carriage, and she noted that when he got out he did not want to walk but preferred to be carried. If supported, however, he could walk almost as well as usual. Two days ago, the fever was more marked, and the parents noted that there was some weakness of the legs. They noticed that when the physician examined him at that time, in stimulating the plantar surfaces of the feet he could wriggle the toes, although he could not flex the legs. He could, however, sit up in bed. Yesterday he could not sit up in bed, and absolutely no movements of the lower extremities could be obtained. Two days ago the fever was 103° F. Yesterday it was somewhat lower. The child has had but little appetite and has taken only milk and a little broth. Castor oil has been given each day, but an enema has been necessary to move the bowels. When the patient was quiet in bed, he did not seem to suffer much pain, but when moved, seemed to have a great deal of pain in the back and legs. This morning he seemed weaker than at any previous time. His cry to-day is noted to be rather weak. There has been no vomiting, and there have been no convulsions.

Physical Examination.—The child lies on his back with his head rotated

to the right. The skin is warm and moist. The child looks dull. *Head.*—Well formed, fontanelles closed. *Eyes.*—No ptosis, no strabismus. Ocular movements are good. Pupils are equal and react to light. Scleræ and conjunctivæ are clear. *Ears.*—No discharge, no tenderness. *Nose.*—No discharge. *Mouth.*—The lips and mucous membranes are of good color. Teeth are well formed, tongue has a moderate white coat. The mouth is drawn slightly to the right on crying. No exudate on the pharynx or tonsils. The right tonsil is enlarged, the left just visible. *Neck.*—The head is rotated to the right, resists rotation to the left or flexion towards the chest. Flexion causes pain. When the child is sitting up, the head drops back, although it can be brought forward. There is evident weakness of the muscles of the neck. *Superficial lymph nodes.*—The cervical and axillary nodes are palpable and slightly enlarged. *Chest.*—Well formed, costal angle wide. The chest moves with respiration slightly. There is more expansion on the left than on the right. Breathing is partly costal and partly diaphragmatic. *Lungs.*—Negative on auscultation and percussion. *Heart.*—No increase in size on percussion. Apex impulse is neither visible nor palpable. Heart sounds are clear and forceful. No murmurs are heard. *Abdomen.*—Well rounded, moves easily with respiration, not distended, no tenderness, no masses or rigidity. *Liver.*—Dullness extends from the fifth interspace to the costal margin in the right mammary line. Edge is not felt. *Spleen.*—Not felt. *External genitalia.*—Normal. *Extremities.*—Upper: movements of the arms seem normal. Brachial reflex is active on the left, not obtained on the right. There is no wrist drop. Lower: there is complete flaccid paralysis of all the muscles of both legs. No reflexes can be obtained, no Kernig's sign. There is no apparent muscular tenderness. *Temperature.*—100.4° F.

July 26. Spinal fluid: clear, colorless, not opalescent; pressure not increased; cell count, 10 per c.mm.; globulin, slightly +; differential count: mononuclears, 88 per cent.; polymorphonuclears, 8 per cent.; endothelial cells, 4 per cent.

July 27. Blood count: leukocytes, 15,700; differential count of 200 cells: polymorphonuclears, 53.5 per cent.; mast cells, 1 per cent.; small mononuclears, 28.5 per cent.; large mononuclears, 12.5 per cent.; transitionals, 4.5 per cent.; eosinophiles, 0.

July 29. Spinal fluid: 20 c.c., for the most part perfectly clear and watery; pressure apparently slightly increased; cell count, 14; smear contains a few red cells; mononuclears, 70 per cent.; endothelials, 20 per cent.; polymorphonuclears, 10 per cent.; globulin, ++; sugar, +.

August 1. Chest and abdomen are clear. The child's general condition improves very slowly. He does not get much brighter and cries or groans most of the time when not left alone. Physical examination does not show sufficient reason for his poor condition.

August 3. Von Pirquet reaction positive in 24 hours. When sitting up, the child can hold his head up for a short time, then it drops backward. The body is not supported from the hips. It falls forward so that the body and the thighs lie almost parallel, the body completely doubled up.

Spinal fluid: 15 c.c. of perfectly clear watery fluid; no increase of pressure; cell count, 3 per c.mm.; globulin, ++ (heavy flocculent precipitate).

August 4. Blood count: leukocytes, 22,000; differential count of 200 cells: polymorphonuclears, 46 per cent.; lymphocytes, 38.5 per cent.; large mono-

nuclears, 0.5 per cent.; transitionals, 11 per cent.; basophiles, 0.5 per cent.; eosinophiles, 3.5 per cent.; stimulation form, 0.

August 5. The patient continues to lie in a listless fashion without taking much notice of his surroundings. There is no change in paralysis. The neck is weak in supporting the head when the patient sits up.

August 9. Spinal fluid: 25 c.c. of clear colorless watery fluid; no increase of pressure; cell count, 2 per c.mm.; smear, almost all small mononuclears; an occasional polymorphonuclear; globulin, ++ (fairly heavy flocculent precipitate); sodium chloride, 0.72 per cent.

August 14. The child is still hypersensitive and timid. He begins to cry and sob as soon as any one approaches him and starts to examine him. The neck is resistant and flexion painful. When he sits up, the head drops over backwards. Sitting up is apparently very painful. He flexes and extends his hands and forearms, but when trying to raise the upper arms, he hunches his shoulders. There is paralysis of the shoulder muscles on both sides. Mentally, the child is hard to rouse and takes no interest in anything around him except in his food, which he takes well. He sweats very freely. There is a peculiar disagreeable odor to the skin.

August 25. The child seems much brighter and more interested in things. He plays a little. The nurse says he seems less tender when she handles him. He began to talk more yesterday. The paralysis is not changed.

September 3. The child seems rather bright. The head is held steadily on sitting up, though some weakness is still present in the neck muscles. All reflexes of the legs are still absent, paralysis of both legs being still complete. Movements of the right arm are good. The left forearm is weak. There is wrist-drop and the hand is held in a claw-like attitude.

CASE 6.

Jennie A., age, 20 years. New York City. Admitted, July 18, 1911. Discharged, October 5, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative.

Past History.—The patient has always been healthy.

Present Illness.—On July 14, four days ago, patient had pain in both legs, principally in the thighs. She rubbed her legs with alcohol, and the pain disappeared. The next morning she got up, cooked the breakfast, but afterwards felt feverish, had a very bad headache and vomited, so that she went to bed. Her legs felt better, however. The next morning, July 16, she had less headache and felt pretty well. She stayed in bed, and at one o'clock took a little nap. When she awoke at two, she tried to get up and found she could not move her right leg. Except for inability to move her legs, she has felt perfectly well. On the afternoon of July 17 she was moved to Saint Luke's Hospital. At this time her neck was slightly stiff and movements were painful. She has had no headache and has not felt especially sleepy. She has not vomited since the onset. Her appetite has been poor. Urination has been normal. She has had no pains in her legs since the onset.

Physical Examination.—The patient is a large, well developed girl. The skin is clear and not hot. There is rather marked brownish pigmentation all over the body, more especially over the lower part of the abdomen and upper thighs.

The face is symmetrical. *Eyes*.—The eyes move normally. Pupillary reactions are normal. *Ears*.—No discharge, no tenderness over the mastoids. *Mouth*.—The tongue protrudes straight and has a slightly gray coat. The teeth are in good condition. The tonsils are somewhat enlarged and ragged, but there is no exudate. Throat is otherwise normal. *Neck*.—The isthmus of the thyroid is palpable. There is no stiffness of the neck. *Superficial lymph nodes*.—The lymphatic glands are not enlarged. *Chest*.—Well formed, symmetrical, both sides move equally with respiration, costal angle is ninety degrees. *Heart*.—Normal. *Lungs*.—Normal on auscultation and percussion. *Abdomen*.—Level, soft, tympanitic, no masses or tenderness. The abdominal wall moves normally with respiration. Abdominal reflexes are present. *Liver*.—Flatness extends from the sixth space to the costal margin. Edge is not felt. *Spleen*.—Just palpable on deep inspiration. *Muscular system*.—Legs: reflexes; Kernig's sign, knee jerks, Achilles, Babinski, and Oppenheim reflexes are absent on both sides. Movements of the legs: right leg, complete flaccid paralysis of all of the leg muscles, including the iliopsoas. Left leg, movements are all possible but rather weak. Patient states that it is more difficult for her to move her leg than it was yesterday. Weakness is especially marked in the quadriceps extensor. Sensation is normal on both sides. There is no pain or tenderness over either leg. Arms: muscular movements are strong on both sides. *Temperature*.—99.8° F.

July 19. Blood count: leukocytes, 13,000; differential count of 200 cells: polymorphonuclears, 59 per cent.; small mononuclears, 17.5 per cent.; large mononuclears, 17 per cent.; transitionals, 4.5 per cent.; eosinophiles, 1.5 per cent.; mast cells, 0.5 per cent. Red cells are normal.

Spinal fluid: 15 c.c. of clear colorless fluid, not opalescent; no increase of pressure; cell count, 297 per c.mm.; smear, small mononuclears, 96 per cent.; large mononuclears, 4 per cent.; globulin, +; albumin, +; sugar, +.

July 22. 8:00 P. M. Leukocytes, 10,400. The pain is extreme. The patient no longer has pain in the back, but has great pain all through the thigh, front and back, extending down into the calf. This is especially marked on the right (paralyzed) side. The leg is tender, and there is a spot of especial tenderness over the anterior crural nerve, just below Poupart's ligament. Heat is comforting but does not relieve the pain. There is no pain in the head or stiffness of the neck. The ears and throat are negative. The heart is negative, lungs clear, abdomen soft, no tenderness.

July 23. The patient was given morphia twice and had a fair night. This morning the pain is starting again. The pain and tenderness in the back are less. Left leg: knee jerk is absent, Achilles reflex active. There is no marked pain, but there is tenderness over the sciatic nerve. Right leg: there is pain in the upper and lower leg like a "tired feeling." There is exquisite tenderness on pressure over the course of the sciatic nerve in the posterior thigh, popliteal space, and calf region. Tenderness is fairly accurately localized over the nerve. There is extreme tenderness over the anterior crural nerve just below Poupart's ligament, over the upper front of the thigh, and over the inner aspect of the thigh. Heart, lungs, and abdomen are negative.

July 28. Spinal fluid: 25 c.c. of perfectly clear fluid, not opalescent; pressure increased; cell count, 38 per c.mm.; smear, mononuclears, 99 per cent.; endothelials, 1 per cent.; globulin, ++ (heavy flocculent precipitate). Towards

evening the patient began to have considerable pain, after a very comfortable day. The pain was present over the left lower quadrant of the abdomen and especially over the left gluteal region and thigh. The upper border of quite a definite area of hyperesthesia is at the level of a line extending from the crest of the ileum to somewhat above the pubes. The lower line of the area of tenderness and hyperesthesia on the thigh is less definite. There is marked pain on pressure over the outer left thigh and over the inner thigh below Poupart's ligament. Palpation of the abdomen is painful over the left lower quadrant. The abdomen moves well with respiration. There is no spasm, no mass.

August 2. Spinal fluid: 15 c.c. of clear fluid; no pressure; no blood; cells, 26 per c.mm.; smear showed about 100 per cent. small lymphocytes; globulin, ++ (heavy flocculent precipitate).

August 3. Sensation for touch and temperature is normal.

August 4. Blood count: leukocytes, 18,100; differential count of 200 cells: polymorphonuclears, 67.5 per cent.; lymphocytes, 19.5 per cent.; large mononuclears, 3.5 per cent.; transitionals, 9 per cent.; eosinophiles, 1 per cent.

August 6. There is a general improvement but the patient still has considerable pain and needs codeine and often morphine nearly every day. The pain is all through the back and legs, especially the right, and tenderness is most marked over the nerve trunks. There is no more return of function in the right leg.

August 9. Spinal fluid: 25 c.c. of clear limpid fluid; no increase of pressure; no blood; cell count, 7 per c.mm.; all mononuclears; smear, mononuclears, ++; globulin, + ± (heavy white flocculent precipitate).

October 5. The general condition is excellent. Legs: reflexes on the right are absent. On the left the knee jerk is practically absent and Achilles reflex just obtained. Sensation to touch and pain is normal on both sides. The temperature is somewhat higher on the surface of the left leg than on the right. The right calf measures 28 cm., the left 31 cm. The right thigh measures 47 cm., the left 51 cm. Movements of the left leg are normal. Flexion of the right thigh on the pelvis is possible and patient also has some power of adduction of the thigh. Extension of the lower leg is not possible. She has very little power to flex the toes. She can stand on the right leg, if the knee is held stiff, and can walk a little in this way. The patient is transferred to the New York Orthopedic Hospital to have a brace fitted.

CASE 7.

James C., age, 18 months. Brooklyn, N. Y. Admitted, August 1, 1911. Discharged, August 13, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in a four family house in the country. The house is eight years old. It is clean and has good air. There are no animals in the house. The mother states, however, that there are lots of bedbugs, cockroaches, and chicken lice.

Past History.—Contains nothing of importance.

Present Illness.—The child was absolutely well until six days ago when, after a nap, he woke up suddenly, and his mother on trying to make him walk, found that he could not stand. He had been playing before that same morning. He vomited once two days later, the vomitus consisting of "curds." He has been very restless but not drowsy. The mother noticed that he would "shake his arms and legs" three or four times during the night. He has sweat profusely

and has cried out with pain on being touched. His appetite has been poor, and he has had a bad cough since onset. He has also had difficulty in urination and has been very constipated. Four days ago the left arm became very weak, but it improved on being rubbed. The right arm also was weak two days ago and appeared to be painful.

Physical Examination.—The patient is a rather thin, well developed baby, lips and skin pale. The skin is clear except for many small brownish papules the size of pinheads over the neck and shoulders (prickly heat?). The child lies quietly with his eyes closed, dozing, and objects to being touched. Anterior fontanelle not quite closed, frontal bosses prominent. Tache cérébrale is well marked but not of long duration. *Eyes.*—Pupils are equal, regular, and react normally. Ocular movements are normal, no strabismus. *Ears.*—No discharge, no tenderness over mastoids. *Nose.*—No discharge from nares, no excoriation. *Face.*—The face is symmetrical at rest, drawn more to the left than to the right on crying. *Mouth.*—The tongue has a moderate grey coat and protrudes straight. The teeth are in fair condition. Tonsils are not enlarged; pillars slightly reddened, no exudate. *Neck.*—The neck is somewhat stiff and resists flexion. *Superficial lymph nodes.*—Small discrete glands felt in neck, axillæ, and groins. *Chest.*—Well formed, symmetrical. Both sides move equally with respiration. Respiration is chiefly abdominal. Costal angle is ninety degrees. *Heart.*—Normal in size. Action is regular and rapid. Sounds are of good quality, no murmurs. *Lungs.*—Clear throughout on auscultation and percussion. *Abdomen.*—Level, soft, tympanitic, no masses or tenderness, symmetrical, moves normally with respiration. Abdominal reflexes are present. *Liver.*—Flatness extends from sixth rib to the costal margin. Edge is felt 3 cm. below the costal margin. *Spleen.*—Not felt. *Muscular system.*—Legs: all reflexes are absent on both sides. Kernig manipulation is painful on both sides. Movements: there is apparently a complete flaccid paralysis of both legs, including the iliopsoas. The only exception noted is power of flexion of the toes of the right foot and slight power of flexion on the left. Arms: movements are normal, but the right arm is weaker than the left. Neck and back muscles are weak. When the child sits up, the head drops forward, backward, or sideways. The spine is not held straight, but the buckling is not constantly towards one side. The back is not well supported from the hips. The child falls forward.

August 1. Blood count: leukocytes, 12,200; differential count of 200 cells: polymorphonuclears, 47 per cent.; small mononuclears, 40 per cent.; large mononuclears, 10 per cent.; transitionals, 2.5 per cent.; eosinophiles, 0.5 per cent.; mast cells, 0.

Spinal fluid: 15 c.c. taken; clear watery fluid, not opalescent; no increase of pressure; cell count, 7 per c.mm.; smear, small mononuclears, 98 per cent.; large mononuclears, 2 per cent. (no blood); globulin, ++ (heavy flocculent precipitate).

August 13. The mother came and wanted to take the baby home. No improvement has been noted in paralysis. He was discharged.

CASE 8.

Julia S., age, 2 years and 3 months. New York City. Admitted, September 29, 1911. Discharged, November 12, 1911. Result, improved. *Diagnosis.*—Acute poliomyelitis.

Family History.—Negative.

Past History.—The patient has always been a healthy child.

Present Illness.—On Tuesday, September 26, three days ago, the patient was perfectly well and happy. On Wednesday, September 27, she was feverish and lost her appetite. The mother said that she became very drowsy on that day. She did not vomit or cough. There were no convulsions but occasional twitchings of the legs and arms. The patient was constipated. Very little sweating occurred until after the doctor gave some powders to control the fever. Yesterday the patient complained of pain in the neck. She has gradually become more and more stuporous until today she seems to recognize no one.

Physical Examination.—The patient is a well nourished little girl, who lies apparently in coma vigil. For the most part the eye slits are held wide and the expressionless eyes are drawn usually to the right. She can be aroused by handling, and then she whines and cries in an irritated way. She evidently sees, but does not recognize. When a strong light is thrown in the eyes, she attempts to close the lids but does not succeed completely; a narrow slit remains. The eyeballs are rolled upwards synchronously, so that the pupils are covered by the upper lid. The head is held a little retracted and almost constantly on the right, although free voluntary motion is possible in all directions. Occasionally a slight twitch or tremor passes over one extremity or side. At times she lowers the upper lids and dozes off into deep sleep. The face is a little flushed and the lips tinged faintly with cyanosis. She breaths a little more rapidly than is normal. *Eyes.*—Pupils are equal and react to light. Sometimes a fine nystagmus is seen when the eyes follow an object well to the side. Occasionally slight incoördination of the eyeballs is seen. There is slight exophthalmos. *Ears.*—Normal. *Nose.*—No discharge. *Mouth.*—The tongue is heavily coated, and there is much thick greyish material posteriorly. The mucous membrane is reddened throughout, especially in the pharynx. The tonsils are very large and look almost inflamed. Teeth are in fair condition. *Superficial lymph nodes.*—The posterior cervical, axillary, and inguinal nodes are palpable. The posterior cervical are enlarged. *Neck.*—The neck is markedly stiff to flexion anteriorly, though the impression is gained that much of the resistance is voluntary and not reflex. The thyroid gland seems to be enlarged, especially the isthmus. *Back.*—Patient cannot be made to sit up because of resistance offered to flexing the spine. *Thorax.*—Well formed, moves properly in respiration. *Lungs.*—Clear. *Heart.*—Normal. *Abdomen.*—Soft, no tenderness, no masses. Spleen and liver are not felt. *Extremities.*—No weaknesses are found in either upper or lower. Knee jerks are obtained with difficulty on the right, absent on the left. Babinski reflex is absent. Kernig's sign: there is no resistance to complete extension of the leg on the flexed thigh. No complaint of pain on the right side, very slight objection at end of manipulation on the left. Achilles reflexes are slightly active on the right, absent on the left. *Temperature.*—102.8° F.

September 29. Spinal fluid: clear limpid fluid, pressure very slightly increased; cells, 113 per c.mm.; polymorphonuclears, 30 per cent.; mononuclears, 83 per cent.; globulin, very slightly hazy (normal); sugar, +.

Blood count: leukocytes, 14,200; differential count of 200 cells: polymorphonuclears, 58.5 per cent.; lymphocytes, 31.5 per cent.; large mononuclears, 7 per cent.; transitionals, 3 per cent.; basophiles, 0; eosinophiles, 0; stimulation form, 0.

September 30. General picture this morning is about the same. The child lies in a sort of stupor with eyes partly closed. She cries irritably on being aroused by prodding. There is no facial weakness, but the mask-like expression is striking. There is complete flaccid paralysis of the left deltoid and biceps, and perhaps also of the triceps. The left quadriceps responds very slightly to the patellar tap. This was not noted yesterday, probably on account of incorrect observation. The diaphragm is working. There is possibly a little weakness of the right shoulder muscles. The temperature remains at 102.4° F.

Tuberculin test (cutaneous) applied at 5:20 P. M.

October 1. Von Pirquet reaction negative this morning. The child seems certainly no worse. There is no advance in paralyses.

Spinal fluid: 17 c.c. of very slightly turbid fluid, no increase of pressure; cells, 239 per c.mm.; polymorphonuclears, 4 per cent.; mononuclears, 96 per cent.; globulin, very slightly +; sugar, +.

October 2. The patient continues to lie in a stuporous condition. There is definite paralysis of both deltoids this morning. The patient can move both legs, but the right less than the left. Knee jerk is absent on both sides this morning and the Achilles reflexes are also absent. The eyes show nystagmus easily and are still drawn chiefly to the right. The waxy or mask-like lack of facial expression continues.

October 3. The child is brighter this morning, but still lies sleepily with the head on the right, and the eyes drawn to the right. She can move them incoördinately toward the left, but there is a definite paresis of the right internal and left external recti. The temperature has dropped rapidly in the past 36 hours.

Spinal fluid: 25 c.c. of clear limpid fluid, pressure very slightly increased; cells, 112 per c.mm.; polymorphonuclears, 7 per cent.; mononuclears, 93 per cent., globulin, very slightly +; sugar, +.

October 4. The patient talks this morning and answers questions intelligently. She lies still, however, with head and eyes drawn to the right. Sometimes one gets the impression that she does not see. The temperature curve continued downward yesterday to 96.8° F., but this morning it has risen to 100° F. and now is normal again.

October 5. The child does not seem so well today. She lies more on her side with head retracted and drawn far to the right. The eyes also are still drawn to the right. She tries to avoid manipulation of the lower extremities. There is definite weakness, if not paralysis, of the quadriceps group of the right side. The leg cannot be extended on the thigh.

October 6. The child lies on her side with back arched, legs extended, and neck retracted. She moans constantly when approached. The neck is resistant. The eyes are usually turned to the right. The child cannot be made to respond at all. The arms are apparently weak. There is paralysis of the flexors of the right thigh. The child looks decidedly sick and her appearance suggests meningitis.

October 10. Spinal fluid: 20 c.c. of clear watery fluid, pressure slightly increased; cell count, 5 per c.mm.; mononuclears, +++; globulin, slightly +; sugar, +. Blood count: leukocytes, 11,200; differential count: polymorphonuclears, 52 per cent.; lymphocytes, 35.5 per cent.; large mononuclears, 7.5 per cent.; transitionals, 3 per cent.; mast cells, 0; eosinophiles, 1.5 per cent.; stimulation form, 0.

October 11. The patient is brighter and notices her surroundings, but is still irritable. There is no advance in paralyses. There is definite power now in the right quadriceps. This represents either a return or possibly an error in the finding of October 5.

October 12. Spinal fluid: 12 c.c. of clear limpid fluid; cells, 35 per c.mm.; mononuclears, +; globulin, very slightly +; sugar, +.

October 14. The child is much brighter. She is slightly propped up on pillows this morning and looks about with considerable interest.

October 20. Spinal fluid: clear colorless watery fluid, pressure not increased; cells, 12 per c.mm.; mononuclears, +; sugar, +; globulin, +.

October 23. The patient continues to improve, but she is still a little irritable, and there is pain when the spine is flexed. The left knee jerk is present, the right very indefinite. There is good power in the left leg, much less in the right.

CASE 9.

Dora O., age, 4 years. New York City. Admitted, September 30, 1911. Discharged, November 23, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative.

Past History.—The patient has always been well, though she has been a delicate child.

Present Illness.—On Wednesday, September 27, three days ago, the child was perfectly well and cheerful. She seemed well on Thursday also, until evening, when she vomited. Later in the night she was put on the toilet, left alone for a few minutes, and fell to the floor. She was put to bed yesterday morning. At that time she could not stand, though she could move her legs. This morning the father noticed that the left lower extremity was much worse than the right. She has had no convulsion and has not had any noticeable sweating.

Physical Examination.—The patient is a well nourished little girl who lies quietly and dozes when undisturbed. She objects to being handled and cries in an annoyed way on physical examination. *Eyes*.—Pupils are equal and react normally. Ocular motions are normal. *Ears*.—Normal. *Nose*.—The right inferior turbinate is red and swollen. There is no discharge. *Mouth*.—Lips are dry. The buccal mucous membrane is red. Pharynx is somewhat reddened. Tonsils are not very large. The tongue is heavily coated with a whitish fur through which the red points of the papillæ can be seen. *Superficial lymph nodes*.—The posterior cervical, axillary, and inguinal nodes are palpable. *Neck*.—The neck is stiff, attempts at flexion are resisted. The anterior muscles are not weak. *Back*.—The back is held rigid to prevent flexion. It hurts the patient to sit up. *Thorax*.—Well formed, respiratory movements are normal. *Lungs*.—Clear. *Heart*.—Somewhat rapid, otherwise normal. *Abdomen*.—Soft, not distended, no tenderness, no masses. *Liver*.—Not enlarged. *Spleen*.—Not enlarged. *Extremities*.—Upper: no weaknesses found. Lower: there is general weakness or possibly lack of willingness to move the right lower extremity. When prodded it is finally drawn up and moved in such a way that no definite paralyses can be made out. There is practically complete paralysis of the whole left lower extremity, except for power to flex the toes. There is also a little power left in the iliopsoas. There is definite foot drop. Knee jerks are slightly active on the right (belly of vastus internus twitches), absent on the left. Achilles reflex is

active on the right, and absent on the left. Babinski test gives plantar flexion on both sides. Kernig's sign: there is practically no resistance of a spasmodic nature on either side. Marked pain is caused on both sides by the manipulation, especially towards the end. *Temperature.*—102.4° F.

September 30. Spinal fluid: 25 c.c. of clear fluid, pressure not increased; cells, 133 per c.mm.; polymorphonuclears, 6 per cent.; mononuclears, 94 per cent.; globulin, 0; sugar, slightly +. Blood count: leukocytes, 20,000; differential count of 200 cells: polymorphonuclears, 49 per cent.; lymphocytes, 25 per cent.; large mononuclears, 26 per cent.; transitionals, 8 per cent.; basophiles, 0; eosinophiles, 1 per cent.; stimulation form, 0. *Temperature.*—101° F.

October 6. The temperature is normal. The child is very bright and lively, laughing and playing. Both legs are very tender to pressure and painful on movement. There is distinct tenderness below Poupert's ligament. The pain is so great that she can scarcely be induced to try to move the legs. The right leg does not seem to be paralyzed, but she will only move it a little. The left leg seems paralyzed except for the flexors of the toes.

October 19. The child is rather hysterical, laughs too easily and cries too easily. There is no improvement in paralysis of the lower extremities.

Spinal fluid: clear colorless watery fluid, no increased pressure; cells, 3 per c.mm.; mononuclears, +; globulin, slightly +; sugar, +.

October 24. The pain is less and the nervousness is less marked. There is no return of motion in the left leg. The quadriceps on the right is paralyzed.

October 30. The child still has pain on movement of the left leg and foot. There is no retrogression of the paralysis of the left leg. The psychic condition is about the same. She laughs hysterically and changes quickly from laughter to crying.

November 6. Spinal fluid: clear, colorless, and watery; pressure not increased; cells, 3 per c.mm.; mononuclears, +; globulin, normal; sugar, +.

November 7. The patient is in much better general condition. There is no improvement in paralysis of the left lower extremity. The right lower extremity has apparently regained complete power.

CASE 10.

Americo B., age, 2 years. West New Rochelle, N. Y. Admitted, October 6, 1911. Discharged, October 29, 1911. Result, improved. *Diagnosis.*—Acute poliomyelitis.

Family History.—Negative.

Past History.—The child has always been perfectly well.

Present Illness.—On Sunday, October 1, five days ago, the child seemed not quite so well as usual and vomited twice. The next day he was feverish and stayed in bed. Since that time he has been in bed. Four or five days ago when he had the fever, he also had slight convulsions. He has not cried much and has had no cough. Bowels and urination have been normal. He has been very sleepy. Has eaten well. Patient has not seemed very tender until today, when he has objected to being handled. Yesterday morning it was noticed that he could not move the left arm, which seemed tender. The other limbs have moved normally.

Physical Examination.—The patient is a well built, strong looking child,

lying on his back. He is quiet, does not cry, and is quite dull. He cannot be made to take an interest in anything. The skin is cool and rather dry. There is no rash. *Eyes*.—Pupils are small, equal, regular, and react to light. There is no strabismus. *Ears*.—No discharge or tenderness over mastoids. *Nose*.—No discharge or excoriation. *Mouth*.—The lips are dry, the tongue slightly coated. Teeth are dry and in good condition. Tonsils are slightly enlarged, not reddened. *Neck*.—The neck is stiff and resists flexion but is not retracted. *Superficial lymph nodes*.—Small glands are felt in the neck, axillæ, and groins. *Chest*.—Well formed, moves normally with respiration. Respiration is chiefly abdominal in type. There is no marked rosary. *Lungs*.—Clear on auscultation and percussion. *Heart*.—Not enlarged, action regular, sounds of good quality, no murmurs heard. *Abdomen*.—Slightly distended, soft, tympanitic, no masses or tenderness found. Abdomen moves normally with respiration. Abdominal reflexes are present. There is moderate tache cérébrale. *Liver*.—Flatness extends from the sixth space to the costal margin. Edge is not felt. *Spleen*.—Not felt. *Extremities*.—Upper: the right arm is normal. Left arm: movements of the fingers, flexion and extension of elbow are normal, but a little weak. There is apparently paralysis of the shoulder muscles, but the child is so drowsy that it is impossible to determine of which ones. Movement of the left arm is somewhat painful. Lower: movements are normal on both sides. Knee jerks are active on both sides. There is no Babinski reflex. Attempt to elicit the Kernig reflex on both sides causes pain, but no especial spasm. The child can support the head when sitting up. *Temperature*.—99° F.

October 7. Spinal fluid: 15 c.c. of clear watery fluid, pressure not increased; cell count, 12 per c.mm.; mononuclears, ++; globulin, slightly +; sugar, +.

Blood count: leukocytes, 9,800; differential count of 200 cells: polymorphonuclears, 64.5 per cent.; lymphocytes, 21.5 per cent.; large mononuclears, 7 per cent.; transitionals, 3.5 per cent.; mast cells, 1 per cent.; eosinophiles, 1 per cent.; stimulation forms, 1.5 per cent.

October 8. The child is still rather dull and pays no attention to anyone, but he is not at all comatose. The right arm moves normally. The left is paralyzed, apparently only in the shoulder. The legs are held stiff, flexed at the knees and thighs. They are not paralyzed, but all motion of the legs, and especially extension, is resisted as if painful. The right knee jerk is not obtained. The right Achilles reflex is barely obtainable. The left knee jerk is just present, left Achilles reflex is normal. There is no hyperesthesia to pressure.

October 14. The child remains in about the same condition, fairly bright, but rather irritable and whining. There is no change in the paralyses, the left upper arm only is affected. Movements and reflexes of the legs are present. There is pain on flexion of the neck, and there is resistance to flexion. There is also pain in the legs. When one attempts to handle the legs, the child flexes them sharply at the knee and resists strongly all attempts to straighten the leg, even attempts to straighten the leg when the hip is not flexed. There is no definite pain on pressure.

October 19. The patient is much less irritable. He can sit up alone and walk. He apparently has plenty of strength in the legs, but his sense of equilibrium is faulty. The paralysis of the left arm remains unchanged.

CASE II.

Moses F., age, 20 months. Brooklyn, N. Y. Admitted, September 18, 1911. Discharged, November 12, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives at Varrett Street, Brooklyn, in a house in which there are two families on a floor. There are seven other children on the same floor. Two months ago, a family with sick children had been dispossessed in this house. The nature of the sickness of those children is unknown. There are no pet animals in the family, but the usual parasites. The child had never been out of the city of Brooklyn.

Past History.—The child has always been strong and healthy, except that, according to the mother, he had pneumonia two weeks ago.

Present Illness.—Friday, September 8, ten days ago, he was perfectly well. On Saturday, September 9, the mother noticed a fever, which continued for the next eight days. The child vomited after taking medicine in the second week. Thursday, September 14, there were some choreic motions of the hands and head. On that evening, he could not move his right hand. The next morning he could not move the other hand. There was no weakness. The child has been irritable and crying and has not slept well. He complains of pain when handled. The bowels have been constipated but moved by enema.

Physical Examination.—The child lies comfortably on his back. He seems fairly bright and responds readily to stimulation. He is irritable on being handled. His legs are drawn up. At times the child has a loose cough. The head is well formed and the fontanelles are closed. *Eyes*.—There is no ptosis. The scleræ and conjunctivæ are clear. The pupils are not dilated, and both react to light, but the left pupil is slightly larger than the right. *Ears*.—There is no mastoid tenderness. *Nose*.—There is no discharge. *Mouth*.—The lips and mucous membranes are of good color. The tongue is red, moist, and has no coat. The pharynx is injected. The tonsils are visible but not large, and there is no exudate. *Neck*.—The anterior muscles seem weak. He holds his head erect, but it is unsteady, with a tendency to drop back. There is no retraction of the neck. There is marked overaction of the sternomastoids which apparently support the entire weight of the head. *Superficial lymph nodes*.—The glands in the right posterior triangle are palpable. The left axillary and both inguinal groups of glands are palpable and shotty. *Chest*.—It is well formed, symmetrical. The breathing is entirely costal in type. Expansion is fair, and slightly more on the left than on the right. The ribs move well. The accessory muscles of respiration are not brought into play. There is no apparent descent of the diaphragm. There is retraction of the abdomen on inspiration. *Lungs*.—The right side is resonant in front and back on percussion. Percussion note in upper front on left side is high pitched, in lower axilla and back it is somewhat flattened. The right side is clear on auscultation. No rales are heard over the area of impaired resonance on the left. The breath sounds over this area are somewhat more distinct than over the corresponding area on the right side. The quality, however, is normal. *Heart*.—Negative. *Pulse*.—Regular, quality good. *Abdomen*.—It is retracted on inspiration. There is no descent of the diaphragm. Abdomen is soft, no masses or tenderness. Superficial reflexes are weak, but present on both sides. The testicular reflex is active. *Extremities*.—Arms: complete

flaccid paralysis of whole right arm. Complete flaccid paralysis of left upper arm and shoulder muscles. Extension of the wrist and fingers is somewhat stronger than flexion. Brachial reflex absent on both sides. Legs: strong and can be moved in all directions. The knee jerks are active on both sides. Achilles reflexes are not obtained, Babinski reflexes are absent. Kernig's sign is absent. *Temperature*.—98.8° F.

September 18. Spinal fluid: 30 c.c. of clear limpid fluid; cells, 8 per c.mm.; globulin, very slightly +; sugar, +. Blood count: leukocytes, 20,100; differential count of 200 cells: polymorphonuclears, 45 per cent.; lymphocytes, 40.5 per cent.; large mononuclears, 0; transitionals, 9.5 per cent.; basophiles, 1 per cent.; eosinophiles, 3.5 per cent.; stimulation form, 0.5 per cent.

September 20. Percussion of lower left lung is unchanged. No rales are heard. The paralysis remains the same. The breathing is purely costal in type. The intercostals are strong.

September 25. Spinal fluid: 24 c.c. of slightly blood-tinged fluid; the specimen for the tests is clear; cells, 4 per c.mm.; globulin, very slightly +; sugar, +. Blood count: leukocytes, 25,600; differential count of 200 cells: polymorphonuclears, 59 per cent.; lymphocytes, 33.5 per cent.; large mononuclears, 0; transitionals, 7.5 per cent.; basophiles, 0; eosinophiles, 0; stimulation form, 0.

September 30. The diaphragm still does not act, but the patient seems perfectly comfortable with the intercostals acting alone.

October 2. Spinal fluid: 18 c.c. of clear fluid; cells, 3 per c.mm.; globulin, 0; sugar, +.

October 24. For the past week the patient has been walking alone. He carries his head far forward with his face a little down, so that he looks up under his eyebrow to see where he is going. The head is held also a little to the right. The trapezius seems to be doing all the work of holding the head up. This gives a peculiar sloping line from head to shoulder and makes the neck look very short and thick. There is no return of power in the arms.

October 30. The child is improving somewhat. He walks very well. He still holds his head tilted to the right. He tries to use his hands and can now pick up objects from the floor with his left hand, but there is little improvement in the arms.

November 12. The child is in excellent general condition. The heart, lungs, and abdomen are clear. The child walks very well and can even run a little. The pupils are equal, regular, and react normally. The neck, back, and abdominal muscles are strong. There is apparently some overdevelopment of the trapezius which supports the head. The pectorals are atrophied. There is a large fold of fat just inside the axillæ, over the pectorals, and a heavy collar of fat around the neck. The legs move normally. Both knee jerks are exaggerated. There is no true ankle clonus. No Babinski or Kernig's signs. The cremasteric and abdominal reflexes are present. The right arm and hand are completely paralyzed from shoulder to fingers. The left shoulder and apparently the extensors of the hand are paralyzed. The only movements possible are flexion and extension of the thumb. He has a remarkably strong grip with the left hand.

CASE 12.

John B., age, 3 years. New York City. Admitted, July 18, 1911. Discharged, August 20, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat:* the family lives in a sixteen family tenement. The home is clean and has plenty of air. As far as is known, there are no sick children in the house, and the child has come in contact with no one who is lame or paralyzed.

Past History.—He has always been well except for two attacks of measles, the last one a year ago.

Present Illness.—Two days ago, on July 16, the child was perfectly well. He usually takes a nap in the afternoon, but on this day he went to sleep in the morning. When he woke up at noon, he started to play, and one of the other children noticed that he could not lift his hand. However, in the afternoon he was well enough to play. He did not complain of pain, has not vomited, has not sweat, and has not been feverish. He has not been especially restless. Yesterday he was playing but could not use his arm. He has had no headache. Appetite has been poor. Bowels and urination normal. The arm has not become any worse. Face has been unaffected.

Physical Examination.—The patient is a well built little boy lying quietly half asleep. He takes little notice of his surroundings and is not easy to rouse. The skin and mucous membranes are of good color. *Eyes.*—The eyes move normally. The right pupil is larger than the left. The right pupil reacts to the light but the left apparently does not. *Ears.*—Normal. There is no discharge and no tenderness over the mastoids. *Mouth.*—The tongue has a grey coat and protrudes to the right. The teeth are in good condition. The tonsils are enlarged and ragged. There is no exudate, and they are not reddened. *Superficial lymph nodes.*—There are small lymphatic glands palpable in the neck, axillæ, and groins. *Chest.*—Well formed, symmetrical, and both sides move equally. *Heart.*—Normal. *Lungs.*—Normal on auscultation and percussion. *Abdomen.*—Full, soft, tympanitic throughout. It is symmetrical and moves normally with respiration. There are no masses or tenderness. Liver flatness extends from the sixth rib to the costal margin. Edge is felt 2 cm. below the costal margin. Spleen is not felt. Abdominal reflexes are not obtained. *Muscular system.*—Legs: knee jerks and Achilles reflexes are present on both sides. The other reflexes are negative. Movement of the legs: there is apparently no paralysis or weakness of either leg. Arms: the muscles of the right arm seem to be unaffected. There is a complete flaccid paralysis of the left arm and hand. The child does not move the arm or hand, and when lifted, it falls limply. Muscles of the back: there is no stiffness of the neck. Child sits up well and holds up its head. *Face.*—The right side of the face is slightly more flattened than the left. The folds of the skin are less marked on the right. The left corner of the mouth is drawn up slightly, and this becomes more marked when the child cries. The right eye slit is narrower than the left. There is apparently no weakness of the right upper lid as this can be raised normally. *Surface.*—There is a small area of herpetic eruption on the upper lip just to the left of the median line.

July 18. Blood count: leukocytes, 15,100; differential count of 200 cells: polymorphonuclears, 55 per cent.; small mononuclears, 26 per cent.; large mononuclears, 8.5 per cent.; transitionals, 1.5 per cent.; eosinophiles, 9 per cent. Red cells normal; blood platelets normal.

July 19. Spinal fluid: 20 c.c. of perfectly clear colorless fluid; pressure not increased; cell count, 37 per c.mm.; globulin, slightly opalescent, slightly +;

albumen, slightly +; sugar, +; smear, small mononuclears, 88 per cent.; large mononuclears, 10 per cent.; polymorphonuclears, 2 per cent.; a few red cells.

July 21. Left arm: it is difficult to make the child attempt movements, but this morning he can extend his fingers, flex the fingers slightly, and flex and extend the thumb. The child complains of pain in the legs, but reflexes and movements are normal.

July 23. The left pupil reacts normally.

July 25. Spinal fluid: 15 c.c. of perfectly clear watery fluid; not opalescent; no increase of pressure; cell count, 12 per c.mm.; smear, chiefly small mononuclears, too few to count; globulin, slightly +.

July 27. There is still very slight evidence of right-sided facial paralysis. The left eye slit is slightly narrower than the right. When seen this morning the child was sweating profusely about the head. The sweating was much more intense on the right side of the head, face, and forehead. Both pupils react normally to light. There is no more paralysis of dilation of the pupil. The tongue protrudes straight.

July 31. The knee jerk is active on both sides. There is hemicranial sweating. Sweating on the right side of the face is frequently observed, while the left side is practically without sweat. The left eye slit is narrower than the right. The right side of the face has a better pink color than the left. The pupils are equal, regular, and react normally.

August 20. His general condition is very good. The chest and abdomen are negative. The legs move normally. The reflexes are normal. The left eye slit is slightly narrower than the right (the lid is a little swollen). The pupils are equal, regular, and react normally. The eyes move normally. The right arm is normal. Left arm: there is complete paralysis of the shoulder muscles and of the flexors of the elbow. The child seems able to use the triceps to extend the elbow. Extensors of the hand are strong, flexors of the fingers and muscles of the thumb are weak but act fairly well. The child has had no hemicranial sweating or flushing of late. He is discharged.

CASE 13.

Victoria M., age, 4 years. Sandy Hook, N. J. Admitted, July 15, 1911. Discharged, September 9, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in a large house at Sandy Hook. There are three families in the house, but each has a separate entrance. The house is twelve years old and clean. There are no other children in the house. There is no one lame or paralyzed in the vicinity. The children have not been away from Sandy Hook since last October, and neither children nor parents have come in contact with anyone who is lame or paralyzed.

Past History.—The child was a normal baby. She is thought to have had scarlet fever four months ago, but the diagnosis is uncertain.

Present Illness.—On June 13 the child complained of pain in the stomach and vomited twice. She was very restless, slept poorly, and sweat a great deal, especially around the head. For the next two weeks she was in bed about half the time and was in poor health. She was slightly feverish and dull. She complained of no pain except in the stomach. On June 23 she complained of pain in her right foot and her mother noticed that when she got up in the morning she

limped and could not use her legs well. In the afternoon it was seen that she did not use her hands normally. The next morning she was taken to the Presbyterian Hospital. About a week before the onset of the paralysis, the mother noticed that while she was sleeping, her breathing was not like that of the other children. When breathing, her chest moved much more than normally, and her abdomen moved much less.

Physical Examination.—The patient is a fairly well built little girl, lying on her back. The skin is clear. *Eyes.*—The eyes move normally, and the pupils react normally. *Ears.*—Normal, no discharge. No tenderness over the mastoids. *Mouth.*—The tongue is clean. The tonsils are somewhat enlarged; the throat is clean. *Superficial lymph nodes.*—There are small glands in the neck, axillæ, and groins. *Chest.*—Respiration is very irregular and labored. After one or two shallow, quiet respirations, the child takes a deep forced inspiration which is associated with dilatation of the alæ nasi. There is marked retraction of the abdomen and flaring of the ribs. With each respiration, the sternomastoid muscles become very tense, and the front of the chest is raised to a marked degree. The left side of the chest is more prominent than the right. Respiration is for the most part of a very jerky character and is at times like hiccough. This is, however, much diminished when the child is lying quietly and has stopped sobbing. *Heart.*—Negative. *Lungs.*—Normal throughout on auscultation and percussion. *Abdomen.*—Level, soft, tympanitic, no masses or tenderness. At times, especially on expiration, there is marked bulging of the left upper quadrant. Abdominal reflexes are not obtained. *Liver.*—Flatness extends from the sixth intercostal space to the costal margin. Edge is not felt. *Spleen.*—Not felt. *Muscular System.*—Legs: knee jerks, Babinski and Oppenheim reflexes are absent on both sides. Achilles reflex is barely obtainable on both sides. Extension of the legs causes marked pain so that Kernig's sign cannot be tested. Movements: flexion of the thighs is possible. There is toe drop and apparently paralysis of the quadriceps and the anterior tibial muscles. Posterior tibial muscles on both sides are unaffected. Movements of the arms are normal. The head flexes without pain. The child can sit up and hold her head up but is apparently rather weak. She complains of pain in both thighs. *Temperature.*—100° F.

July 16. The child breathes quietly when let alone, but inspiration is always associated with retraction of the abdomen. On expiration there is bulging of the upper half of the abdomen. The muscular tone is good.

July 18. The nurse noticed yesterday that the patient was unable to hold a piece of bread well in her hands. Arms: movements of upper arms, and flexion and extension of forearm are normal. Dorsal extension of hand at wrist is impossible on both sides. Flexion of the fingers is fairly complete but weak on both sides.

July 19. The right side of the face shows some flattening, and the left corner of the mouth is drawn up. The left eye slit is narrower than the right. The tongue protrudes straight. The voice is very harsh, squeaky, and high pitched.

July 23. The chest and abdomen are clear. Paralysis of the diaphragm persists.

July 25. Spinal fluid: 18 c.c. of clear watery fluid, not opalescent; no increase of pressure; partly bloody; cell count, 163 per c.mm.; smear, small mononuclears,

87 per cent.; large mononuclears, 9 per cent.; polymorphonuclears, 4 per cent.; globulin, slightly +.

July 31. Spinal fluid: a few cubic centimeters of perfectly clear colorless fluid; no opalescence; no increase of pressure; cell count, 979 per c.mm.; many large phagocytic cells; smear, small mononuclears, 68 per cent.; large mononuclears, 16 per cent.; endothelial cells, 16 per cent.; no polymorphonuclears; many degenerated cells in the smear, apparently endothelial cells; globulin, slightly +. Temperature, 100° F.

August 2. Spinal fluid: 10 c.c. of perfectly clear watery fluid; no opalescence; pressure very low; cell count, 154 per c.mm.; a few phagocytic cells; smear, small mononuclears, 96 per cent.; large mononuclears, 4 per cent.; no blood; globulin, slightly +, opalescent; few small flocculi.

August 5. There is gradual increase of power in the quadriceps on both sides. Both knee jerks were elicited this morning, the left being more active.

August 8. Spinal fluid: 8 c.c. of clear watery fluid; pressure too low to measure; cell count, 31 per c.mm.; smear, large percentage of mononuclears; globulin, very slightly +, slightly turbid, within normal limits; sugar, +.

August 15. The diaphragm is now acting absolutely normally. The abdominal muscles act normally, and the reflexes are normal. Both knee jerks and both Achilles reflexes are obtained normally, there is no ankle clonus. The movements of both legs are normal, and fairly strong. She is walking and gets along well. The muscles of the neck are still weak (?), the head tends to fall backwards. The extensors of the hand and muscles of the thumb show little improvement.

CASE 14.

Gladys B., age, 6 months. New York City. Admitted, October 27, 1911. Discharged, November 11, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in a tenement. There are eighteen families in the building. The building is not old, and it is very airy and clean. There are no cats or dogs in the home; occasionally there are cockroaches. Max G. (a cousin of this child, and now in this hospital with poliomyelitis) lives on the ground floor of this building. There is very close contact between these two families, and Max plays continually with the B. children. On Tuesday, October 17, when Max was sick in bed, the baby was brought in and laid on a pillow on his bed. Shortly after that, Max was taken to the hospital. Since that time Gladys has not been in the G. home.

Past History.—Negative.

Present Illness.—On the morning of Saturday, October 21, six days ago, the baby was perfectly well when bathed. In the afternoon she vomited twice and had "high fever." The doctor thought it was angina and middle ear trouble. The next day she was feverish and very sick. There has been no vomiting since the first day of her illness. She has had no convulsions and has not sweat at all. Her bowels have been constipated. Urination has been normal. During her illness the child has taken no interest in anything, and has slept most of the time. She has wanted to lie quietly in bed, and apparently has objected to being handled. She has taken her nourishment well. Two days ago, October 25, the mother noticed that the child did not use her left hand. Even the day before,

the mother noticed that the child did not use her hands or her feet much. Yesterday she could move only the fingers of the right hand. The left arm and right leg were also paralyzed. The child looks brighter today and has less fever.

Physical Examination.—The patient is a well built and strong looking baby. Sensorium is clear. The neck resists flexion. There is no retraction. The skin is clear except for a little scaling on the upper lip. The anterior fontanelle is open, but not bulging. The face is symmetrical. *Eyes.*—Pupils are equal, regular, and react to light. Ocular movements are normal. There is no strabismus or nystagmus. *Ears.*—There is no discharge or tenderness. *Mouth.*—Tongue is clean. Tonsils are slightly enlarged and rather ragged. There is no redness. Pharynx is normal. *Superficial lymph nodes.*—Glands in the neck are palpable. *Chest.*—The chest is symmetrical and moves well with respiration, but with inspiration there is a very definite retraction and descent of the anterior chest wall. This is associated with a marked raising of the abdominal wall on inspiration. There is no special activity of the accessory muscles of respiration. Respiration is rapid, but not labored. Pressure over the abdomen causes great distress, but pressure over the chest does not affect respiration. *Heart.*—Normal in size. Regular. Sounds are of good quality. *Lungs.*—Clear on auscultation and percussion, except for a few scattered moist rales in both backs. *Abdomen.*—Full, soft, and tympanitic. There are no masses or tenderness. Abdominal reflexes are present. When the child sits up the abdominal muscles seem to lack tone. There is definite tache cérébrale. *Liver.*—Normal area of flatness. Edge not felt. *Spleen.*—Not felt. *Extremities.*—Right arm appears normal. Left arm: flexion and extension of fingers and wrists are normal; flexion and extension of the elbow are weak, and it is difficult to determine whether or not it is completely paralyzed. Lower extremities: right leg is paralyzed completely, except for the extensors of the toes and foot (anterior tibial and peroneal). In the left leg there is weakness of the quadriceps. Knee jerks, Achilles and Babinski reflexes are absent. Kernig's manipulation produces pain on the right, but not on the left side. *Temperature.*—99° F.

October 27. Spinal fluid: 15 c.c. of clear limpid fluid; pressure not increased; cells, 25 per c.mm.; mononuclears, ++; globulin, normal; sugar, +.

October 31. Paralysis of the intercostal muscles persists. There are no rales in the lungs. The general condition is excellent.

Spinal fluid: 15 c.c. of clear watery fluid; pressure slightly increased; cells, 5 per c.mm.; mononuclears, +; sugar, +; globulin, +.

November 6. Spinal fluid is clear, colorless, and watery; cells, 5 per c.mm.; mononuclears, +; globulin, very slightly +; sugar, +.

November 11. The child is in good condition. He has had a loose cough for some time and this persists slightly. The chest and abdomen are clear. His respiration is entirely abdominal. There is still some retraction of the thorax on inspiration, but it is not so marked as on admission. Paralysis of the intercostals persists. Both hands, the lower arms, and the elbows move normally. There is weakness of the shoulders, especially of the deltoids. The arms can only be raised incompletely, but still there is very good motion in every direction. The legs move normally. The knee jerks are both present. Achilles reflexes are not obtained. There is no Kernig's sign or Babinski reflex. The abdominal reflexes are normal. The residual paralysis consists of weakness of both shoulders and intercostals.

CASE 15.

Louis G., age, 3 years. New York City. Admitted, July 31, 1911. Discharged, September 3, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family live in a tenement with nineteen other families. The house is not very old, is fairly clean, and has good air. There are no bedbugs or other parasites found in the home, and no dogs or cats belong to the family. They know of no other sickness in the house or neighborhood and of no lame or paralyzed children or any possibility of contact.

Past History.—The child was always a healthy baby, nursed for eighteen months. He had a "convulsion" about every six weeks "when the teeth came out," but he has had none for the past six months. Six months ago he had measles, followed by whooping cough, the latter lasting five or six weeks.

Present Illness.—Two weeks ago yesterday, July 16, the child was well in the morning. At one o'clock in the afternoon the father found him very feverish, and sent for a doctor. The next day the child was worse, and still feverish. He had no convulsions, no vomiting, and no cough, but was very restless and sweat a great deal. His appetite was poor. After three days the child got better, but stayed in bed because his neck was weak, and he could not hold up his head. Eight days ago, July 23, the father says that he complained of pain in the left foot, and it was noticed that he could not move either foot. Since the first three days he has had no fever. For the past eight or nine days he has had a little cough, but no expectoration. The bowels have been constipated and appetite very poor. The arms have not been affected. The child has had no trouble with respiration, and was doing well until yesterday afternoon at three o'clock, when the parents noticed that he was having difficulty in breathing. Respiration was rapid and associated with much movement of the belly wall. Since then he has become much weaker, has not slept, and has eaten very little. He has had more cough and has cried all night. He has not seemed to have pain or tenderness since the onset, when he had pain in the neck and foot.

Physical Examination.—The patient is a well developed, very sick looking little boy. Sensorium seems clear. The lips are pale and have dry crusts on them; no herpes. The skin is hot and dry and rather grey. On the chin are two pinhead sized petechial spots, no rash, no bites. The head is well shaped and the face symmetrical. Child swallows milk well. *Eyes*.—Movements are normal, no strabismus. Pupils are equal, regular, about 4 mm. in diameter. They contract in bright light, but do not dilate well. The right eye slit is a trifle narrower than the left. On looking downward the sclera is seen above the left cornea. *Ears*.—No discharge or tenderness. *Nose*.—No discharge. *Mouth*.—The teeth are in good condition. Tongue protrudes straight, and has a thick grey coat. The tonsils are much enlarged, and rather ragged. No redness or exudate. *Neck*.—The neck resists flexion and there is slight retraction. *Superficial lymph nodes*.—The glands are not especially enlarged, but there are a few small ones in the neck and groins. *Chest*.—Symmetrical, well formed, costal angle is wide. Respiration is rapid. The neck muscles come into play actively, the sternomastoids stand out with each inspiration, and the *alæ nasi* dilate with respiration. Respiration is wholly of the abdominal type. The chest hardly moves at all with respiration, the only movement being a retraction of the lower ribs and of the lower intercostal spaces on inspiration. The abdominal

wall bulges forward on inspiration, and is retracted on expiration. Breathing is rapid and jerky, expiration is forcible. The expiratory sounds follow the retraction of the abdomen at a considerable interval. *Heart*.—Not enlarged, sounds obscured by rales, sounds of embryonic type. *Lungs*.—No dullness, no pure bronchial breathing, but both lungs are everywhere full of coarse moist rales. Respiration is harsh and loud. *Abdomen*.—Full, soft, tympanitic, no masses or tenderness. The left side is fuller than the right. Abdominal walls are very lax and bulge forward with inspiration. There is apparently a paralysis of the muscles of the abdominal wall. Abdominal reflexes are not obtained. *Liver*.—Flatness extends from the sixth rib to the costal margin, edge not felt. *Spleen*.—Not felt. *Muscular system*.—The arms move normally but there is apparently slight weakness of the right. Both legs lie limply on the bed and the child neither moves them nor resists passive motion. Movement of the flexors of the toes on the right is noted on plantar stimulation, but otherwise the legs are paralyzed. Knee jerks, Achilles, Babinski, Oppenheim, and Kernig reflexes are absent on both sides. The attempt to elicit the Kernig's sign is painful. It is impossible to determine about the back muscles, but they seem quite strong as the child is made to sit up. The child can hold his head up, but with difficulty. No pain or hyperesthesia is apparent except on attempting to flex the neck and in trying to elicit Kernig's sign. The child is very sick and it is impossible to determine the limits of paralysis accurately.

July 31. Blood count: leukocytes, 19,700; differential count of 200 cells; polymorphonuclears, 84 per cent.; small mononuclears, 6.5 per cent.; large mononuclears, 6.5 per cent.; transitionals, 3 per cent.; mast cells, 0; eosinophiles, 0.

August 4. Spinal fluid: 20 c.c. of perfectly clear watery fluid, not opalescent; pressure 100-110 mm.; cell count, 29 per c.mm.; differential wet count: 98 per cent. mononuclears; 2 per cent. polymorphonuclears; globulin, + (flocculent precipitate).

August 5. The chest is more free of rales. All the reflexes of the legs are still absent.

August 8. The chest is practically clear of rales. The abdominal and back muscles are strong. The neck muscles are apparently strong, but on sitting up the child has a tendency to let his head hang backwards.

August 19. The chest is clear. The child cries as soon as anyone tries to touch him or move his legs. He seems to have a good deal of tenderness still. The legs are the same as yesterday. Both knee jerks and Achilles reflexes are absent.

September 3. The child is very bright and active. He is able to sit up alone. The movements of the head and neck are good. The abdominal reflexes are active, and there is no sagging of the abdominal walls when the child sits erect. The spine does not deviate to either side. The knee jerks are absent on both sides; Achilles reflexes are absent; there is no ankle clonus, but strong plantar flexion of toes on the left on plantar stimulation, and wink-like response on right. Movements of the right leg are absent below the hip. The left thigh can be weakly flexed, extended, and rotated outward. There is no apparent power of abduction. Flexion and extension of the right lower leg are absent, the left lower leg can be rather strongly flexed and extension of the foot is good. The

toes on the left side can be both flexed and extended. The ribs move well on respiration, slightly more on the left than on the right side. The patient goes home today.

CASE 16.

Hyman W., age, 21 months. Brooklyn, N. Y. Admitted, September 22, 1911. Discharged, November 26, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis and acute bronchitis.

Family History.—Negative. *Habitat*: the family lives in a four family house on Willoughby Avenue, Brooklyn. They have four rooms on the first floor. The light in the bedrooms is bad, in the other rooms it is fair. There is no similar illness in the house. The child has played with other children of the street and house. There are flies and mosquitoes in the house, no roaches or waterbugs, but some bedbugs.

Past History.—The patient has always been a healthy child.

Present Illness.—On Sunday morning, October 15, five days ago, the child was perfectly well. In the afternoon he had two attacks of vomiting. The next morning, Monday, he had a temperature of 103–104° F., but was up and walked about during the day. On Tuesday, two days after the onset, paralysis of both legs was noticed and weakness of the right arm. On Wednesday, the child had some difficulty in swallowing. He has had no dyspnea or convulsions. Child has not been very irritable. For two days he has had pain when the head was raised, and on sitting up the head dropped backward. There has been no rigidity or retraction of the neck. He has had no discharge from the ears, no cough. Bowels moved last on Wednesday after enema and castor oil. There has been no retention of urine.

Physical Examination.—The child lies quietly in bed sleeping. On being awakened he responds slowly to stimulation and is drowsy and dull. He cries weakly on being handled. The skin is moist and warm. There is a slight degree of dyspnea. *Head*.—Well formed, slight frontal prominence. Fontanelles are closed. *Face*.—Symmetrical in repose, drawn slightly to the left on crying. *Eyes*.—Apertures are equal. Scleræ and conjunctivæ are clear. External ocular movements are good, no paralysis of orbicularis. Pupils are equal, not dilated, react slightly to light. *Ears*.—No tenderness or discharge. *Nose*.—No discharge, nares patent. *Mouth*.—Lips and mucous membranes are of fair color. Tongue has a heavy coat, muscles are intact. Pharynx is injected. Tonsils are not enlarged, and there is no exudate. *Neck*.—No retraction. There is slight resistance to flexion. On sitting up the head falls back and to the right. There is paralysis of the anterior neck muscles and sternocleidomastoids. *Superficial lymph nodes*.—The glands on both sides of the neck are palpable and shotty. *Chest*.—Well formed and symmetrical. There is slight flaring of the costal border. Breathing is diaphragmatic in type. Ribs do not move. There is retraction of the bony wall on inspiration. There is slight action of the accessory muscles of respiration. The diaphragm descends, and seems fairly strong. Respiration is slightly rapid, short, and jerky. *Lungs*.—Clear on auscultation and percussion. *Heart*.—Normal. *Abdomen*.—The abdomen moves well with respiration. Walls are relaxed. There is no bulging in the flanks, no masses or local tenderness. Superficial abdominal reflexes are absent. *Extremities*.—Upper: the right arm has very slight power of flexion of the fingers, otherwise it is in a

state of complete flaccid paralysis. The deltoid and posterior arm muscles are paralyzed on the left. Other movements are possible but weak. Lower: the right thigh can be extended with a strong tendency to outward rotation. The toes and foot can be extended. On the left side there is slight power of outward rotation with slight extension of the thigh. Otherwise flaccid paralysis is complete on both sides. There is pain on full flexion of the leg but no Kernig's sign. Knee jerks, Achilles reflexes, and Babinski test are all absent. *Temperature*.—99.6° F.

September 22. Spinal fluid: 20 c.c. of perfectly clear fluid, pressure slightly increased; cells, 5 per c.mm.; mononuclears, ++; globulin, 0; sugar, +. Blood count: leukocytes, 17,100; differential count of 200 cells: polymorphonuclears, 38 per cent.; lymphocytes, 49 per cent.; large mononuclears, 1.5 per cent.; transitionals, 11 per cent.; basophiles, 0; eosinophiles, 0; stimulation form, 0.5 per cent.

September 25. The alæ nasi are working this morning a little more than before. The child shakes his head from side to side frequently. Evidently this is the only way of attempting to escape from the discomfort of breathing and general sense of helplessness.

September 26. Spinal fluid: 30 c.c. of clear limpid fluid, pressure very slightly increased; cells, 15 per c.mm.; globulin, ±; sugar, +.

September 27. Spinal fluid: 10 c.c. of clear watery colorless fluid, pressure not increased; cells, 4 per c.mm.; all mononuclears; sugar, +; globulin, slightly +.

September 28. The patient continues to breathe only with the diaphragm. There is no return of power in the lower extremities or arms. The child sleeps little at night but dozes from time to time during the day.

October 3. Blood count: leukocytes, 17,200; differential count of 200 cells: polymorphonuclears, 49.5 per cent.; lymphocytes, 39 per cent.; large mononuclears, 6.5 per cent.; transitionals, 3.5 per cent.; basophiles, 0; eosinophiles, 1 per cent.; stimulation form, 0.5 per cent.

October 4. The patient has improved markedly in the past three or four days. He whines less and looks stronger and brighter. He breathes strongly with the diaphragm.

Spinal fluid: 14 c.c. of clear fluid, pressure not increased; cells, 2 per c.mm.; globulin, +; sugar, +.

October 18. During the past week the patient has seemed somewhat brighter. There is, however, no change in his paralysis. The neck is apparently unable to hold up the head. There is no return of costal respiration.

October 20. Spinal fluid: clear, colorless, and watery; cells, 2 per c.mm.; mononuclears, +; globulin, slightly +; sugar, +.

November 2. The child really makes no progress. He is a miserable, sick child. The neck muscles are still so weak that the head drops backward when he is sitting up. The back is very weak. The power of the right arm is completely gone except for flexion of the fingers. The left upper arm is paralyzed. Breathing is practically wholly diaphragmatic. The intercostals are paralyzed. The child still has pain on moving the legs. He does not look so bright or so well as he did two weeks ago. The chest is clear except for a few rales in the back.

November 3. Spinal fluid: clear, colorless; cells, 3 per c.mm.; mononuclears, +; sugar, +; globulin, very slightly +.

November 17. Yesterday morning there was a beginning bronchitis on both sides. In the evening both lungs, front and back, were full of coarse, moist rales. No rise in temperature was noted. Respiration was rapid and wholly abdominal. Complete paralysis of the intercostals persists. The chest has seemed to become smaller and more shrunken, an appearance probably due in part to atrophy of the pectorals and intercostals, associated with the soft protuberant belly. This morning the child looks better. His temperature is 100° F., and the rales are not quite so numerous.

November 19. The condition remains the same. Both sides of the chest are full of coarse moist rales. The heart action is good. There is no especial rise of temperature.

November 21. The temperature is still normal. The rales have cleared up remarkably, and the chest is almost clear. The child looks much better.

November 26. The patient continues to do well. This morning there are a few, low pitched, coarse rales in both sides of the chest near the bases. The breathing is still entirely diaphragmatic. The abdomen is greatly distended. The patient has gained in weight and looks very fat and well. The neck is not yet able to support the head, and the back is very weak. The child goes home today.

CASE 17.

Edward G., age, 3 years and 8 months. New York City. Admitted, August 12, 1911. Discharged, August 23, 1911. Result, well. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative.

Past History.—The patient has always been healthy.

Present Illness.—Last Sunday, August 6, six days ago, the patient came to his mother and said he did not feel well and wanted to lie down. He was sleepy and complained of headache. On Monday he walked about a little, but went to bed again and has been there since. He has had no appetite, has not been irritable, and has had no tenderness. On Tuesday he either could not or would not speak, and has not spoken since. He has eaten nothing since Tuesday. He had not vomited until yesterday, when he vomited a little after taking medicine. He did not lie quietly, but threw himself about in bed. At times he has seemed almost to have convulsions. Bowels have not been regular. He has passed urine normally, but has not voided since yesterday. Urination induced by application of hot compresses. No sore throat. The mother noticed no weakness of the arms or legs, but says there is some stiffness in the neck.

Physical Examination.—The patient is a well developed, rather thin, very sick looking child. He lies quietly on his back, eyes half closed, and pays no attention to his surroundings. He cannot be roused by questions and is very dull. The skin is dry, quite warm, and clear, except for a few old scabs on the legs and arms (bites). The face is symmetrical. *Eyes*.—The eye slits are equal. Eyes apparently move normally, but it is impossible to rouse him enough to make him follow an object with his eyes. Pupils are equal, and react normally. *Ears*.—No discharge or tenderness. *Nose*.—No discharge or excoriation. *Mouth*.—The lips are covered with dry crusts, no herpes. Teeth are dry and in fair condition. Tongue has a thick grey coat. The tonsils are only a little enlarged. Tonsils, pillars, and pharynx are reddened; no exudate. The pharynx is full of grey

mucopurulent material. *Neck*.—There is very slight resistance to flexion, no retraction. *Superficial lymph nodes*.—There are firm discrete enlarged glands in the neck, axillæ, and groins. *Chest*.—Somewhat chicken-breasted, no definite rachitic rosary or Harrison's groove. Chest is symmetrical and moves normally with respiration. *Heart*.—Negative. *Lungs*.—Clear on percussion and auscultation. *Abdomen*.—Level, soft, tympanitic, no masses or tenderness, symmetrical, moves normally with respiration. Abdominal reflexes are present. Bladder is distended. *Liver*.—Flatness extends from the sixth space to the costal margin. Edge is easily felt. *Spleen*.—Not felt. *Muscular system*.—Legs: reflexes, all normal. No paralysis or weakness of leg muscles. Arms: both move equally and strongly. Back and abdominal muscles are strong. Neck muscles: the child can sit up straight, but after a short time his head falls backwards. He has very little power to straighten his head up again, and usually lets it continue to hang backwards. *Temperature*.—99.8° F.

August 12. Blood count: leukocytes, 15,400; differential count of 200 cells: polymorphonuclears, 63 per cent.; lymphocytes, 17 per cent.; large mononuclears, 5 per cent.; transitionals, 8.5 per cent.; basophiles, 0; eosinophiles, 6 per cent.; stimulation form, 0.5 per cent.

Spinal fluid: 30 c.c. of perfectly clear watery fluid, not opalescent; pressure, 330 mm.; cell count, 31 per c.mm.; globulin, slightly cloudy, within normal limits; sugar, + after heating; sodium chloride, 0.70 per cent.; smear, small mononuclears, 96 per cent.; large mononuclears, 4 per cent.

August 14. The child is much brighter, is wide awake, and will sit up alone. He does not respond well to questions and still prefers to be let alone. The neck muscles are much stronger. Movement of the head is painful but he can hold his head up well. The right knee jerk is very slight and is obtained with difficulty.

August 15. Urination has been normal since admission.

August 18. Spinal fluid: 20 c.c. of clear colorless fluid; no blood; pressure slightly increased (crying); cell count, 7 per c.mm., all mononuclears; globulin, very slight opalescence, normal; sugar, +.

August 23. Spinal fluid: 15 c.c. of perfectly colorless watery fluid; cell count, 6 per c.mm., all mononuclears; globulin, very slightly +, turbid; no flocculi; sugar, +. Eyes, ears, and throat are negative. There is no stiffness of the neck. Neck, back, and abdominal muscles are strong. Movements of the legs and arms are normal. The child walks well. He blinks a good deal, and seems to have some photophobia. The patient is discharged well.

CASE 18.

Mary H., age, 8 years. New York City. Admitted, October 2, 1911. Discharged, October 22, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative.

Past History.—The patient has always been a healthy child.

Present Illness.—Last Thursday, September 28, four days ago, the patient vomited. She had been perfectly well on Wednesday night. The doctor gave her some medicine and she stayed in bed all that day and was drowsy. She did not sweat much. Slept fairly well Thursday night. On Friday she seemed much better and asked for food, but stayed in bed. That night she was restless. Satur-

day morning her father saw her suddenly become stiff, but she did not have a convulsion. This passed rapidly, and during the day she became more and more stuporous. The doctor said that it was either typhoid or meningitis. The child was restless during the night. Yesterday morning (Sunday), and throughout the day, she was in a very stuporous condition. Yesterday evening Dr. Huber was called and made a diagnosis of infantile paralysis. About 9:00 P. M. yesterday evening, the patient could swallow only very weakly. One eye could not be closed. She was restless during the night; no sweating. She has been constipated.

Physical Examination.—The patient is a well nourished little girl, tall and slender. She lies on her back with head to the right. The right eye is closed, the left open. She is in a stuporous condition from which she can be roused by handling or by sharp questions and orders. She will perform any simple act, such as putting out the tongue, raising the eyebrows, etc. She does not become irritated during the course of the examination, but accepts it all passively. She is very apathetic. There is incontinence of urine. *Face.*—The left half of the face is completely paralyzed. The eyebrows cannot be moved, the eye cannot be shut, and when she shows her teeth the right side pulls far over. *Eyes.*—Pupils are equal, react rather sluggishly. The left eye slit is wide and cannot be closed. The ocular motions are a little incoördinate, there being a definite weakness of the right internal rectus so that an internal strabismus of the right eye is present most of the time. *Ears.*—Normal. *Nose.*—Normal. *Mouth.*—The tongue is heavily coated. There is much thick, grayish, sticky mucous in the back of the mouth. The buccal mucous membrane is red. The pharynx is very much congested; tonsils not present. *Neck.*—The neck is stiff to flexion. Anterior muscles are weak. She cannot keep the head from falling backward. Swallowing is weak. *Superficial lymph nodes.*—The posterior cervical are somewhat enlarged. Axillary and inguinal nodes are just palpable. *Chest.*—Rather long and narrow, moves only very slightly in respiration, but if the patient takes a deep breath it moves normally. *Lungs.*—Clear. *Heart.*—Normal. *Abdomen.*—Soft, flat, no tenderness, no masses. Enlargement of liver and spleen is not evident. *Extremities.*—Upper: no weaknesses. Lower: no weaknesses. Knee jerks are very active; Achilles reflexes active. On testing for the Babinski sign, there is slight rapid initial flexion and then marked extension of the great toe. Oppenheim reflex is present. Kernig manipulation is resisted actively near the end and the child complains of pain. *Temperature.*—99.6° F.

October 2. Spinal fluid: 14 c.c. of clear fluid, pressure not increased; cells, 20 per c.mm.; mononuclears, ++; globulin, very slightly +; sugar, +. Blood count: leukocytes, 12,000; differential count of 200 cells: polymorphonuclears, 75 per cent.; lymphocytes, 13 per cent.; large mononuclears, 3.5 per cent.; transitionals, 8 per cent.; basophiles, 0; eosinophiles, 1 per cent.; stimulation form, 0.5 per cent.

This evening the patient was fed by gavage. She has difficulty in managing fluid in the mouth and pharynx and has much distress from thick saliva.

October 3. The patient is still very dull, but can be roused and responds slowly to questions. Some weakness of the right deltoid seems to be present this morning. The brachial reflex on the left is present, on the right absent.

October 4. The child is markedly brighter this morning. Both eyes are open

and she is no longer stuporous. She is still apathetic. There is slight internal strabismus of the right eye. There is no improvement of the left facial paralysis. The patient can speak coherently this morning, and says she has a little pain in her left foot. The knee jerks are present, but not especially active; they are about normal. The left Achilles reflex is much more active than the right. She has no loss of power in arms or legs, though she complains of pain in the left foot. The anterior neck muscles are weak, for the patient's head drops back when the trunk is lifted forward by the shoulders. In the sitting position she can hold her head up.

October 8. The child is much improved. She swallows easily and takes food well. Facial paralysis remains very marked. The right knee jerk is not obtained, the left is present. Both Achilles reflexes are present. Movements of both legs are normal.

CASE 19.

Fred S., age, 5 years. Orange, N. J. Admitted, July 14, 1911. Discharged, July 30, 1911. Result, improved. *Diagnosis.*—Acute poliomyelitis.

Family History.—One sister, 15 months old, admitted to this hospital at the same time as the boy, was paralyzed five days before him. Habitat: family lives in a separate house with a large yard. They have lived there for nine months. Last August, one year ago, there were two cases of paralysis, one an infant and one seventeen years old, occurring in different houses just across the street.

Past History.—Negative.

Present Illness.—The child vomited on July 19, the day the baby became sick. He was well for five days and then was taken sick with a high fever, 102° F., and complained of pain in his head. The next day the right side of the face was drawn up. There was no ptosis. The eye movements have been good. No trouble with swallowing. Can talk normally. Three days after the onset the left arm was limp for about twenty-four hours. Then the fever left and the condition has remained stationary. There has been no nasal discharge and no eruption. After the first twenty-four hours he seemed to have no pain. The bowels have been constipated during the illness. Appetite is good, no vomiting.

Physical Examination.—The patient is a bright active child who responds readily to questions. The head is well formed and held in normal position. *Eyes.*—External ocular movements are good, no ptosis. The left palpebral fissure is wider than the right. Pupils are equal and react to light and accommodation actively. The sclerae and conjunctivæ are clear. *Ears.*—No tenderness over the mastoids, no discharge. Hearing is acute. *Face.*—The right side is contracted and the left side lax and drooping. The mouth is drawn down partially to the right when in repose and moves well upward to the right on smiling. Both sides of the forehead can be wrinkled, but the wrinkles on the right go higher than on the left. On frowning, contraction is equal on both sides. He uses both eyes well. Masseters strong on both sides. *Mouth.*—The tongue can be protruded in the mid-line. Lips and mucous membrane are of good color. Teeth are good. The tongue has a slight white coat. Tonsils quite large on the right, easily visible on the left. They are not injected, their muscles move well. *Neck.*—Muscles are strong, no rigidity or tenderness. *Superficial lymph nodes.*—The right cervical, both axillary, and both inguinal glands are easily palpable, but not especially enlarged. *Thorax.*—Well formed; costal angle ninety degrees.

The chest moves easily with respiration. No rachitic rosary. Expansion good and equal. The thorax is everywhere resonant on percussion and clear on auscultation. *Heart*.—Negative. *Abdomen*.—Soft throughout; no tenderness or rigidity; moves with respiration. The muscles are strong. There are no masses. *Liver*.—Dullness extends from the fourth interspace to the costal margin in the right mammary line. Edge is not felt. *Spleen*.—The edge of the spleen is just felt on deep inspiration. *Extremities*.—No weakness of the arms. Grip a trifle stronger on the right than on the left. Brachial reflexes are active on both sides. Movements and strength of the legs are unimpaired. Patellar reflexes very active on both sides. Achilles reflex obtained on both sides. Plantar responses normal. *Sensorium*.—No hyperesthesia or muscular tenderness. *Temperature*.—99° F.

July 16. Blood count: leukocytes, 13,400; differential count of 200 cells: polymorphonuclears, 58.5 per cent.; small mononuclears, 25.5 per cent.; large mononuclears, 14.5 per cent.; eosinophiles, 0.5 per cent.; transitionals, 1 per cent.; red cells, normal in size and shape; no nucleated forms seen. General condition is good. The arms and legs move perfectly well. The abdominal muscles are normal. There is paralysis of muscles of expression on the left side of the face (seventh nerve); the masseter is normal. The tongue protrudes slightly to the right. The folds of the face are obliterated on the left, the right side of the mouth is drawn up. The right eye is partially closed by drawing up of cheek. Pupillary and eye reactions are normal. Hearing is apparently good and equal on both sides.

July 29. The tongue protrudes to the right but it can be put out to the left. Face: the right side of the mouth is drawn upwards, especially when he laughs. The asymmetry is not marked when face is at rest. The forehead wrinkles on both sides but most on the right. Arms: movements of both are strong and normal. Legs move strongly and normally. Knee jerks and Achilles reflexes are active on both sides. Residual paralysis of the left side of the face (seventh nerve). Patient is discharged.

October 7. The child returned for observation. He is in good condition but the mother reports that he is very nervous and timid. He is afraid to go anywhere alone. His face is a little better, but still shows some asymmetry on smiling.

CASE 20.

Jerry E. S., age, 3 years and 10 months. Lakewood, N. J. Admitted, October 8, 1911. Discharged, November 14, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat*: the family lives in a single house, clean and airy. They have no animals. The family have been at Jefferson, N. H., for three months, and the children have all been well. Three families lived in the house there, but there was no sickness in the house. The house was clean, there were no parasites, and no pet animals except one dog. They know of no cases of infantile paralysis in Lakewood or Jefferson.

Past History.—Negative except for a birth palsy of the left arm.

Present Illness.—On October 4, four days ago, the child said that he did not feel well. "He laid around" and would not play or eat. The next day he felt worse and lay on the couch all day and seemed inclined to sleep. That afternoon

his speech was less plain. The next day, Friday, he was kept in bed all day; speech was about the same, he could talk, but not distinctly. He seemed rather dull and restless, but there was no evidence of pain until yesterday, when he kicked and cried and seemed to be in pain. Two days ago his mother noticed that he could not swallow, and his mouth seemed to fill with phlegm. He has not eaten or drunk anything for three days. He has tried to swallow milk and water but it comes back. He vomited at the onset and on the second day. Bowels have moved with enema, urination has been normal. Face has seemed symmetrical. The child has had no convulsions. He had a cough and raised some phlegm, but has difficulty in getting rid of it. He has not sweat at all and has had no chill, but has been feverish for the last three nights. There has been no evidence of paralysis of arms or legs. His speech has been worse than it is today.

Physical Examination.—The patient is a well nourished small boy. He lies on his side with head slightly retracted and knees drawn up. He objects to being turned on his back, and struggles actively to regain the lateral position. His face is flushed and he lies in a soporific condition from which he is easily aroused by any manipulation which tends to place him on his back. *Eyes.*—The left pupil is a trifle wider than the right; both react normally. Ocular motions are normal. There is no strabismus. *Ears.*—Normal. *Nose.*—Normal. *Mouth.*—Tongue is heavily coated, and the mouth is filled with thick grey mucous, which collects in the pharynx and which the patient cannot get rid of. He evidently cannot swallow or change the position of the fluids in his mouth. Pharynx is reddened, tonsils are somewhat enlarged. *Neck.*—The neck is a little stiff to flexion. Muscles are weak and head lolls back when the trunk is raised forward. *Superficial lymph nodes.*—The posterior cervical, axillary, and inguinal nodes are palpable but not enlarged. *Thorax.*—Well formed. Moves properly in respiration. *Lungs.*—Clear. *Heart.*—Normal. *Abdomen.*—Soft, not distended, no masses or tenderness. Liver and spleen not felt. *Extremities.*—Upper are normal and strong. Lower are strong and active. Knee jerks on both sides are, perhaps, slightly increased. *Temperature.*—101.4° F.

October 8. Spinal fluid: pressure not increased; cells, 18 per c.mm.; globulin, very slightly hazy; sugar, + (?). Blood count: leukocytes, 19,300; differential count of 200 cells: polymorphonuclears, 73 per cent.; lymphocytes, 18.6 per cent.; large mononuclears, 2.6 per cent.; transitionals, 4.6 per cent.; basophiles, 0; eosinophiles, 0; stimulation form, 0.6 per cent.

October 10. The child is somewhat brighter. There is no return of swallowing. He is receiving nasal feedings of milk and eggs, and salt solution by rectum. He usually regurgitates after the feedings and sometimes becomes quite blue. He has great difficulty in expelling the regurgitated food and the mucus from his mouth. When told to stick his tongue out he says, "I can't." When he tries he hardly gets it beyond his teeth; and it protrudes quite markedly to the right. No other paralysis is found.

October 12. Tongue paralysis is very definite, chiefly on the right side. Swallowing shows no improvement. The child is fed by nasal catheter. Speech has improved considerably since admission, but is still far from distinct. In general the child is somewhat brighter.

Spinal fluid: a few c.c. of clear watery fluid; no increase of pressure; cells, 90 per c.mm.; globulin, very slightly +; sugar, +.

October 14. The tongue protrudes definitely to the right. It is still impossible for him to swallow. Speech is not at all clear. The face is symmetrical. The pupils are equal. The neck resists flexion, which is painful. Movement of both legs is painful especially the right. He keeps the right leg flexed at the knee, and, when, any one handles it, resists any attempt to straighten the leg, this being very painful. Pressure over the calf and below the patient's leg is painful. The right knee jerk is not obtained (when the leg is held fairly flexed). Knee jerks and Achilles reflexes are active on the left. The movements are less painful.

October 20. The child is still being fed by stomach tube. He is not, however, losing much weight. For the last few days he has been getting more quiet, and takes very little interest in his surroundings, but he rouses when his mother comes. Yesterday his voice became much more weak and hoarse. He said his throat hurt him. A laryngoscopic examination was made but the vocal cords could not be seen. Today he breathes quietly, but has said very little and speaks in a very faint voice. His tongue deviates to the right. There are no other paralyses. The reflexes are normal.

Later in the day. Spinal fluid: clear, colorless; cells, 3 per c.mm.; mononuclears, +; sugar, +; globulin, slightly +.

October 21. Yesterday the patient caused surprise by swallowing a few cubic centimeters of water. Later in the evening he swallowed 20 c.c. of milk. This morning he took 150 c.c. of egg nog without difficulty.

October 30. The child swallows very well now, solid food better than liquids; in fact liquids still cause some trouble, but they have been swallowed much better in the last few days. His voice has been very weak and is still hoarse and weak but much better. He has complained greatly of pain, when being dressed or moved; but much of this seems to be fear, for today when his attention was diverted his legs were moved, and he moved them well himself and sat up straight without pillows and had no pain.

October 31. Spinal fluid: 5 c.c. of clear watery fluid; pressure not increased; cells, 3 per c.mm.; mononuclears, +; globulin, very slightly hazy (normal limits); sugar, +.

November 4. The patient walks better, but still with a swaying, shaky, almost ataxic gait. His legs are spread so that his feet are wide apart, and the right foot is usually held in outward rotation. The legs are moved and held rather stiffly. Both knee jerks are exaggerated, and both Achilles tendons give almost clonic response. There is a suggestion of ankle clonus but not a true clonus. The psychic condition is very interesting. The child is normally a rather quiet, silent, stubborn child. He is now distinctly nervously "on edge" and is ready at any moment to laugh or cry. Usually he ends by crying and without any particular reason. At times when some one is playing with him, he only answers by a series of nervous grunts, something between a giggle and a sob.

November 13. The child is in good general condition. He is still somewhat nervous and cries rather easily, but is quite different from what he was a fortnight ago. The child swallows all foods normally. His voice is loud but somewhat husky. The tongue still protrudes toward the right, and there is slight atrophy of the right side of the tongue. Both legs move actively and normally. Knee jerks and Achilles reflexes are exaggerated on the right, much exaggerated

on the left. Babinski and Kernig's signs are negative. There is a suggestion of ankle clonus on the left. The gait is much improved. He walks well and can run but still spreads his feet rather widely. His gait is perfectly steady, however. There is slight Romberg's sign. The child is discharged today.

CASE 21.

Helen N., age, 6 years. New York City. Admitted, September 17, 1911. Discharged, October 9, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. The family lives on West 57th Street. There are ten families in the house, one other on the floor on which the patient lives. The house is old and fairly clean and airy. There are many waterbugs and bedbugs, but very few flies and mosquitoes. There are, however, many fleas as the family has a pet dog and pups. There are no sick children in the neighborhood.

Past History.—The patient has always been a healthy child.

Present Illness.—Last Sunday, September 10, seven days ago, the patient was perfectly well and happy. On Monday evening she complained of nausea and vomited a little. From that time on she began to be dull, drowsy, and slept most of the time. She complained of headache and tired feeling. Wednesday evening she was a little irrational. A doctor was called, who said that the child had diphtheria and gave her antitoxin on Thursday morning. On Friday she did not recognize the family and had a vacant stare. There were no convulsions, but twitchings of the hands, feet, and eyelids. Another doctor saw her Friday and said she had meningitis. Temperature was then 105° F. On Saturday the patient was in a rather deep coma. Bowels have been constipated but moved yesterday with enema. The child has not spoken for the last two days.

Physical Examination.—The patient is a fairly nourished little girl who lies on her back, still, and apparently semicomatose. Occasionally the eyes roll under the half closed lids. She can be roused by handling. When the feet and hands are prodded, the legs or arms are drawn away sharply. She breathes regularly and by the normal mechanism, though perhaps a trifle less deeply than is normal. The face is rather flushed and has a slightly bluish tinge. *Eyes*.—The pupils are narrow, the right just a little smaller than the left. The eyes do not move quite coordinately. The conjunctivæ are injected, and there is a purulent secretion in the left eye. Eye grounds are apparently normal. The disc is sharply defined. The veins look a little full. *Nose*.—No excoriations or discharge. *Mouth*.—The teeth are in poor condition, some of the front ones broken. The buccal mucous membrane is reddened. The tongue is heavily coated. The pharynx is very red, the tonsils large. There is a thick mucopurulent mass in the pharynx with some yellowish white membrane-like shreds. *Superficial lymph nodes*.—Posterior cervical, axillary, and inguinal nodes are palpable. *Neck*.—Not rigid. *Thorax*.—Moves properly, though slightly in respiration. *Lungs*.—Clear. *Heart*.—Action is regular, not rapid, sounds not very loud. *Abdomen*.—Normal. *Extremities*.—Upper: apparently normal. Lower: power is apparently good. Both knee jerks are absent, both Achilles reflexes active. Babinski and Kernig's signs are absent on both sides. There is slight reaction to pain at the end of Kernig manipulation. *Surface*.—Clear. *Temperature*.—101° F.

September 17. Blood count: leukocytes, 23,200; differential count of 200

cells: polymorphonuclears, 76 per cent.; lymphocytes, 17 per cent.; large mononuclears, 3 per cent.; transitionals, 2.5 per cent.; eosinophiles, 1 per cent.; basophiles, 0; stimulation form, 0.5 per cent.

Spinal fluid: 25 c.c. of clear fluid; pressure 14 cm.; cells, 37 per c.mm.; mononuclears much increased; globulin, slight haziness, well within normal limits; sugar, +.

September 18. The child lies on her back in a drowsy condition. There are no rose spots and the spleen is not felt. There is no muscle stiffness of the neck, and no Kernig's sign.

P. M. There is slight but distinct weakness of the muscles closing the right eye.

September 19. This morning the patient is a great deal brighter. She looks at things about her a little more intelligently and she will slowly follow with her eyes an object that is moved in front of her face. She will answer simple questions. The ocular motions seem to be coördinate but slow. The pupils are equal and react sharply to light. The right palpebral fissure is a little wider than the left, and the patient is unable to close the eye. There is also very slight flattening of the right side of the face. No weakness of arms or legs is found. The knee jerks are still absent. The temperature which was 102.2° F. on the day of admission has been falling steadily for the past 36 hours and is now 99.2° F. The pulse rate has followed a parallel course. The von Pirquet reaction is negative. The neck is perhaps a trifle stiffer than yesterday. The patient complains of pain when her shoulders are lifted forward.

P. M. The patient is found wide eyed and crying. When questioned she says that she is better and wants to go home. Her mind is almost entirely clear, but she is a little slow in responding. The right knee jerk was obtained once or twice after repeated attempts, but not the left. No further paralysis is noted beyond that of the muscles supplied by the seventh nerve.

Spinal fluid: 22 c.c. of perfectly clear fluid; pressure slightly increased; cells, 44 per c.mm.; mononuclears much increased; globulin, very slightly +; sugar, +.

September 20. This morning the patient is still brighter. She lies quietly, however, preferably with knees drawn up, and does not like to be handled much. There is definite tenderness over the muscles on either side of the spine between the shoulders. There is no weakness in the arms or legs. The knee jerks are not obtained. Right facial paralysis is marked this morning. It is complete in the upper half with only weakness of the lower. The patient was taken from bed and put on the floor. She was a little unsteady in balance, but could stand and also walk.

September 21. Spinal fluid: 5 c.c. of clear limpid fluid; pressure not increased; cell count, 57 per c.mm.; globulin, very slight haziness; sugar, +.

September 25. This morning the child is bright and happy. During the last few days she has been inclined to be irritable. The paralysis of the right seventh nerve is receding a little. She cannot close the right eye. The knee jerks have returned on both sides. Achilles reflexes are as before. The child sits easily and also stands.

September 27. The patient is better. The knee jerks are both active. Paralysis of the upper half of the distribution of the seventh nerve is still marked. Spinal fluid: 25 c.c. of clear limpid fluid; pressure slightly increased; cells, 11 per c.mm.; globulin, very slight haziness; sugar, +.

October 9. No change in facial paralysis. The knee jerks are very markedly exaggerated, especially the right, which responds with almost a clonus. The Achilles jerks, too, are much exaggerated. There is no weakness in the extremities. The patient walks well and her general condition is good.

CASE 22.

Johanna W., age, 3½ months. New York City. Admitted, October 13, 1911. Died, October 15, 1911. *Diagnosis.*—Acute poliomyelitis.

Family History.—This baby slept in the same bed with her sister, Margaret, who was taken sick on October 4 and became paralyzed on October 5. Margaret used to let the baby suck the nipple of her nursing bottle.

Past History.—The child is a nursing baby and has always been very strong.

Present Illness.—On Wednesday, October 11, two days ago, the mother noticed that the baby looked sick and feverish. The next day she was taken to a doctor who said that the child had a fever of 102° F., but did not pronounce it infantile paralysis. She has not vomited until today when she vomited in the examining room, nor has she sweat much. She has had no convulsions and has not seemed to have much pain. The main symptom has been sleepiness. Bowels moved with medicine; movements green.

Physical Examination.—The patient is a well nourished infant who seems drowsy. When handled she tries to cry, but cannot make any noise. She looks like a very sick baby. *Eyes.*—Pupils are equal. There is possibly a slight convergent strabismus. *Nose.*—The alæ nasi move slightly in respiration. *Mouth.*—The tongue is slightly coated with a whitish fur. Pharynx is reddened, tonsils somewhat large. *Head.*—Anterior fontanelle bulges a very little (diminished after lumbar puncture). *Neck.*—Patient cannot hold her head up. The posterior muscles are not stiff to flexion. *Superficial lymph nodes.*—The posterior cervical and occipital nodes are palpable. *Thorax.*—Well formed. It does not move properly in respiration. The whole thoracic cavity is drawn down with each inspiration. There is apparently complete intercostal paralysis. *Lungs.*—Clear. *Heart.*—Apparently normal. *Abdomen.*—Full, not distended. The urinary bladder reaches nearly to the umbilicus. Patient does not void. Abdominal respiration is marked. Liver and spleen are not felt. *Extremities.*—October 13. There is complete flaccid paralysis of both lower extremities this afternoon. Knee jerks and Achilles reflexes are absent on both sides. There is no Kernig's sign present and Kernig manipulation does not cause pain. There is apparently flaccid paralysis also of the right deltoid and upper arm. The left has tone. October 14. The left arm also seems to be flaccid this morning. *Surface.*—A few fine petechiæ are scattered over the surface.

Spinal fluid: 30 c.c. of very slightly opalescent fluid, pressure not increased; cells, 423; globulin, slightly +; sugar, +.

Blood count: leukocytes, 25,300; differential count of 200 cells: polymorphonuclears, 59 per cent.; transitionals, 13.5 per cent.; lymphocytes, 23.5 per cent.; basophiles, 0.5 per cent.; large mononuclears, 2.5 per cent.; eosinophiles, 0; stimulation form, 1 per cent.

The child is drooling at the mouth. Her face and lips are cyanotic. The respirations are short and shallow and the lungs clear. There is practically no movement of the ribs on the left side, although there may be slight retraction

on inspiration; on the right side the ribs move slightly. Abdomen moves with respiration but the diaphragm seems weak. At intervals there is a deep forced inspiration. The accessory muscles of respiration are called into play.

Respiration is irregular this afternoon, and at times is of a Cheyne-Stokes character, two or three deep forcible inspirations and then a pause in which one hears a few very shallow and weak respirations; then again two or three deep breaths. The strong expirations are associated with a forcible jerky upward movement of the diaphragm. Pressure on the abdomen and blocking of the action of the diaphragm causes great distress and difficulty with respiration. The child is very limp; there is apparently paralysis of the legs, intercostals, arms, and neck. She is very pale and slightly cyanotic. The accessory muscles of respiration (sternomastoids) are used, and occasionally the child throws her head back and her lower jaw forward with inspiration.

October 15. The child gradually grew weaker during the night and, without change in symptoms, died at 6:30 A. M.

CASE 23.

Helen K., age, 3 years and 10 months. Bergenfield, N. J. Admitted, August 19, 1911. Died, August 21, 1911. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives alone in a house in the country. The next house is twenty-five feet away. The house is new and clean. They have one pet dog which is healthy. No parasites. No possible contact of child or parents with anyone lame. The family has lived in the country for five years.

Past History.—The child was normal at birth, had chicken pox as a baby and measles last year. No scarlet fever or diphtheria. She has always had a weak stomach and vomits occasionally.

Present Illness.—Last Wednesday, August 16, three days ago, the child went to Rockaway. The day before she was perfectly well. She ate very little breakfast that morning before starting for the seashore, vomited while on the cars, but on arrival ate a big dinner. In the afternoon she went wading in the surf and had a chocolate soda. Then she began to feel sick and complained of a headache. She ate no supper. At 8 P. M. she vomited, and vomited three times that night. She was restless at night and very feverish. No especial tenderness, but rather irritable. The next morning she vomited again. The doctor thought she had "wind on the stomach." At this time she walked but was "weak on her legs." There was no change all day. Yesterday she was about the same. Her legs were weak, she could walk, but did not want to stand. She sat up last evening and fed herself at supper. The mother says her arms and legs "quivered" yesterday. At three o'clock this morning the mother put her on the water closet and she "collapsed." The mother then noticed that she could not move her arms. She has had a fair appetite, has vomited frequently, and the bowels have been constipated. No convulsions, no especial sweating. She has been very dull and sleepy for several days. Her mother thinks she passed no urine yesterday. She complained of headache at first and pain in the back, but not in the arms and legs. Two days ago her voice became very weak. It is more normal now.

Physical Examination.—The patient is a well built, apparently strong child.

Sensorium is clear, but she is distinctly dull and sleepy. The cheeks are somewhat flushed, lips and conjunctivæ of good color. The face is symmetrical. *Eyes.*—Pupils are equal, regular and react normally. Ocular movements are normal. No strabismus. *Ears.*—No discharge or tenderness. *Nose.*—No discharge or excoriation. *Mouth.*—The tongue protrudes straight and has a thick gray coat. Lips are dry, no herpes. The teeth are in fair condition. Both tonsils are large but not reddened, and there is no exudate. *Neck.*—The neck resists flexion and attempt to flex is painful. *Chest.*—Well formed and symmetrical. Costal angle just under ninety degrees. Respiration is almost wholly abdominal; chest moves very slightly with respiration. When the hand is put over the abdomen and pressure made to obstruct the diaphragm, there is no increase in thoracic respiration, and she complains that she cannot breathe and respiration gets more rapid. There is no marked rosary or Harrison's groove. *Lungs.*—Inspiration is short, clear throughout on auscultation and percussion. *Heart.*—No enlargement. Left border inside nipple line. Action is regular and rapid. Sounds are of good quality. A soft systolic murmur is heard over the precordium. *Abdomen.*—Level, soft, symmetrical, tympanitic, no masses or tenderness. Abdominal reflexes not obtained. No tache cérébrale. *Liver.*—Flatness extends from the sixth rib to the costal margin, edge not felt. *Spleen.*—Not felt. *Muscular system.*—Legs: movements of both legs are possible, no paralysis can be made out. There is possibly some weakness, but the child is so sick that it is hard to be certain of this. The Achilles reflex is present on both sides. The other reflexes are negative. The left quadriceps acts well. The attempt to elicit Kernig's sign causes pain and muscular spasm which prevents complete extension. Arms: the shoulder muscles are completely paralyzed. The child can flex and extend the forearms at the elbows but does so very weakly. Flexion and extension of the fingers and extension of the hands dorsally are possible, but all movements are weak. The child can sit up, but the neck muscles are weak and the head has a tendency to drop backwards or sideways. The back muscles seem strong. She sits up well. Sensation: there is pain in the back of the neck on flexion and on attempting Kernig's sign. No especial hyperesthesia noted. The voice is somewhat rasping and harsh, but strong.

August 19. Blood count: leukocytes, 10,000; differential count of 200 cells: polymorphonuclears, 61 per cent.; large mononuclears, 6.5 per cent.; transitionals, 5 per cent.; lymphocytes, 26.5 per cent.; eosinophiles, 0.5 per cent.; stimulation form, 0.5 per cent. Spinal fluid: 20 c.c. of absolutely clear, colorless, watery fluid; pressure, 180 mm.; cell count, 120 per c.mm.; sugar, +; globulin, very slight turbidity, normal; smear, practically all the cells are mononuclears and nearly all are lymphocytes.

August 20. The temperature is still elevated. The weakness of the hands seems more marked. Legs: there is definite weakness of the quadriceps on both sides. Knee jerks are both absent. Other movements are pretty strong. The Achilles reflex is not obtained on the right, and is slight on the left. The respiration is 42 to the minute; it is wholly abdominal and is very jerky. On pressure over the abdomen and upwards towards the diaphragm, respiration becomes more rapid and labored, and her face becomes quite flushed. No movement of the chest surely referable to intercostals was discovered. The child complains of pain in the back, but of no other tenderness.

August 21. Urine examination: yellow, turbid; reaction acid; Fehling's test for sugar negative; test for albumin with heat and acetic acid negative, with potassium ferrocyanide negative. No change in the child's condition yesterday. She was restless during the night but respiration remained the same until it suddenly changed at 7 o'clock this morning and the doctor was sent for. The child is lying on her back. The sensorium seems perfectly clear. The skin has a pale grayish color. The lips are dark red, rather cyanotic. The child is in a profuse sweat. The pupils are equal, regular, and react normally. Ocular movements are normal. The tongue protrudes straight. The temperature is normal. The child has difficulty in swallowing but takes some milk. She raises frothy mucus which she does not spit out, but holds between her lips and waits for it to be wiped off. Her respiration is short, jerky, and wholly abdominal. The upper part of the chest moves with respiration but is pulled down on inspiration. The abdominal respiration is normal. With inspiration the *alæ nasi* dilate, and there is a protrusion of the lower jaw. The sternomastoids and muscles of the neck stand out. Lungs: the right front is clear, respiration normal; left front, the respiration is very feeble; both backs are full of coarse moist rales. The heart is regular and normal. The abdomen is negative. The legs are as before, perhaps weaker. The arms are as before, movements of the hands weaker. The general condition is quite characteristic. She is perfectly clear mentally and speaks clearly but rather abruptly, telling exactly what she wants, "My arm hurts," "Turn me over," "Scratch my nostril." Apart from this, she desires to be let absolutely alone. "Don't touch my chest," "Leave me alone, doctor," she says. Her bowels have moved twice; after each movement the child is somewhat more cyanotic.

10 A. M. The child is very cyanotic, her respiration is irregular and jerky. She is moribund. The pulse is regular at 64; then it suddenly becomes more rapid, perhaps 100 (still regular) for a series of beats, then gradually drops down to about 64 again. Respiration becomes more feeble and suddenly ceases. The heart continues to beat forcibly and regularly for some minutes. Then it becomes more feeble and somewhat irregular, though in general the arrhythmia is of the type with a series of beats at regular rhythm, changing to a series of regular beats at another rate. The heart beats become more infrequent, and the heart stops beating five and a half minutes after respiration stopped.

CASE 24.

Alfred K., age, 15 months. New York City. Admitted, October 14, 1911. Died, October 17, 1911. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in bad surroundings. The house is an old wooden structure with a bakery in front and living rooms in the rear. The bedrooms are dark. There are flies, mosquitoes, and all varieties of vermin. There have been no acute infections in the house lately. There have been no cases of poliomyelitis in the immediate neighborhood, but there was one case two blocks away "some time ago."

Past History.—The patient has always been a healthy child with the exception of some slight gastro-intestinal trouble.

Present Illness.—The onset was rather sudden on Monday night, October 9, five days ago. The child was restless, could not sleep, cried a great deal, and seemed to have pain in the head. There was no spontaneous vomiting, but the child was

given ipecac and then vomited a little frothy mucus. On the next day he had a fever. Three days ago he had a slight convulsion, and on the following night had short general convulsions involving the whole body with the exception of the lower extremities. He has had muscular twitchings and a coarse tremor of the hands. There has been no nasal discharge. Sphincters have not been affected. Two days ago paralysis of both legs was noticed and at that time all reflexes were absent. Yesterday there was beginning weakness of the right arm. There has been no respiratory involvement. Temperature, pulse, and respiration have been as follows: October 10, temperature 102.6° F., pulse 140, respiration 60. October 11, temperature 102.6° F., respiration 48. October 12, temperature 101° F., pulse 130, respiration 36. October 13, temperature 100.2° F., pulse 120. October 14, temperature 98.6° F. Child has been very constipated, urination has been normal.

Physical Examination.—The child is a well developed, strong baby, lying quietly on his back. The eyes are open and the child is not asleep, but he seems languid and takes no interest in anything. The head is square and the cranium bulging. Anterior fontanelle is not closed. The face is symmetrical. *Eyes.*—Pupils are equal, regular, and react normally. Ocular movements are normal. Eyelids close normally. *Ears.*—No discharge or tenderness. *Nose.*—No discharge or excoriation. *Mouth.*—The tongue has a thick gray coat. There is a distinct groove in the median line. The lips are dry. Both tonsils are enlarged and boggy; no exudate and no especial redness. *Superficial lymph nodes.*—There are small glands in the neck, axillæ, and groins. *Neck.*—The child resists flexion, it evidently causes pain. *Chest.*—Well formed, costal angle ninety degrees, moves normally with respiration, no rosary or Harrison's groove. *Lungs.*—Clear throughout. *Heart.*—Normal in size, sounds clear. *Abdomen.*—Full, very soft, tympanitic, no masses or tenderness. Abdominal reflexes are barely obtained. There is no tache cérébrale. The abdominal muscles lack tone and seem very weak. There is no contraction when the child sits up. *Liver.*—Flatness extends from the sixth rib to the costal margin. Edge is just felt. *Spleen.*—Not felt. *Muscular system.*—Movements of the arms seem strong. Legs: both knee jerks, both Achilles reflexes, and both Kernig's signs are absent. There is no resistance to the Kernig manipulation on the right, and on the left only slight tightening of the external hamstring as the leg is extended (muscles all paralyzed), but extension is quite painful. There is no tenderness on pressure over the legs. Back: when the child is made to sit up the body falls forward over the legs, bending at the hips. There is paralysis of the gluteals, erector spinæ, and neck muscles. The head cannot be held up but drops loosely forward, backward, or to one side. Cremasteric reflexes are active. *Surface.*—The skin is rather dry but not hot. There is no rash. The lips are slightly pale.

October 17. Spinal fluid: 6 c.c. of clear, colorless, watery fluid, pressure low; cell count, 114 per c.mm.; mononuclears much increased; globulin, very slightly +; sugar, +.

The child does not look so well. He appears weaker and his respiration is rapid. The temperature has not risen. The child has some difficulty in swallowing, but can still take food. The tongue is apparently straight, but he will not stick it out. The chest moves well with respiration. The accessory muscles take some part in respiration. The abdomen moves paradoxically. There is distinct

retraction on inspiration, paralysis or weakness of the diaphragm associated with the weakness of the abdominal muscles noted before. On auscultation both sides of the chest are full of coarse moist rales, the fronts more so than the backs; there is no consolidation. The legs are paralyzed. The arms and face are unaffected. The child does not cry at all loudly (laryngeal involvement?).

11:30 A. M. The child has changed remarkably in the last hour. He is quite cyanotic and the respiration is short and labored. Breathing has become worse and the heart irregular (Luciani periods) until respiration practically stopped. Artificial respiration was then begun and camphorated ether injected. In a few minutes the heart was regular and rapid, and respiration fairly regular and deeper than before. His color was much better, and child could breathe alone. The intercostal muscles appear to be weakening now. With each respiration the lower ribs flare, but there is little or no movement of the upper ribs. The diaphragm is not descending. There is increasing activity of the accessory muscles of respiration in the neck. They are acting strongly, and with each inspiration there is a depression and protrusion of the lower jaw. The irregularity of the heart seems to bear some relation to cyanosis, increasing as the color gets worse. The child is unconscious. The skin has been gray and cyanotic since this attack began. It is quite edematous and marks of the stethoscope over the chest and also over the shins persist for a long time. During the period of apnea which occurred suddenly and in which the child appeared to be dying, the heart action assumed a curious irregularity, but one which was similar to that which we have seen in several other cases of respiratory failure. The heart would beat regularly at a rate of about 84 for a period of 10 seconds and then at a rate of 36 for 6 or 7 beats. This alternation of rapid and slow series of beats occurred over a considerable period of time, probably a minute and a half or two minutes. During the latter part of this time, artificial respiration was being carried on, and the heart action then became regular and rather rapid. When the patient began again to breathe spontaneously, the heart rate became rather slower and continued to be regular. After about 10 or 15 minutes the child again had respiratory failure. This time, however, the heart did not show the rhythmical changes in rate, but gradually became slower and weaker with slight sinus arrhythmia from time to time. The heart continued to beat, giving the normal sounds, at a fairly regular rhythm for three minutes after the breathing finally stopped, at least nothing could be heard after three minutes, except possibly the vaguest sound in the region of the second right interspace. Electrocardiographic records, however, show that an action current was formed for 24 minutes after breathing stopped and 21 minutes after the sounds had become inaudible.

CASE 25.

Jesse L., age, 5½ years. New York City. Admitted, July 20, 1911. Died, July 20, 1911. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat*: the family lives in a fifteen family house and knows of no sickness in the same house. The child and the parents do not remember ever having come in contact with anyone who is lame or paralyzed. The child has been in New York since last summer when he went for a few days to the country. There are no animals in the house. The building is old, but fairly clean and airy.

Past History.—The child has always been perfectly well.

Present Illness.—On Wednesday, July 12, eight days ago, the mother noticed that the child was feverish. She gave him citrate of magnesia, and the next morning he appeared to be well. On the following three days, Thursday, Friday, and Saturday, the child was up and played normally. Saturday night, however, five days ago, the child complained of pain in the back and was very restless and tired. He slept poorly and the next morning complained of severe pain in the head and back. He was given castor oil and vomited. Dr. Taylor saw the child in the evening. At this time he had pain and a stiff neck and his temperature was 102° F. Note by Dr. F. L. Taylor, who has had the case under his care since July 16: "The child has been ill for three days—apparently some digestive disturbance and coryza. Was given castor oil by mother. Last night complained of intense frontal and occipital headache; pain in the back of the neck and upper dorsal spine with retraction of head. Tendon reflexes were perhaps slightly exaggerated. There was twitching of the individual muscles of the extremities as the patient lay in bed. Temperature 102.2° F. (rectal); pulse 150. Has Kernig's sign and no Babinski reflex. Respiration 50 to 60 and shallow. Heart, lungs, and abdomen negative. Leukocyte count, 7,000; polynuclears, 81 per cent. Monday morning, July 17, condition unchanged. Lumbar puncture: 15 c.c. of crystal clear cerebrospinal fluid obtained under some pressure (*i. e.* came out in a spurt). Differential count shows polynuclears, 71 per cent.; lymphocytes, 21 + per cent.; endothelial cells, 2 + per cent.; incubation negative. Tuesday: reflexes obtained; tendency to somnolence; takes nourishment well. Temperature, pulse, and respiration about the same. Wednesday afternoon: respiration 36, temperature 101° F., pulse 120, quite somnolent, very weak. Urine: specific gravity 1.009, slightly acid, clear, heavy deposit of phosphates, no albumin, no sugar. Cell count: leukocytes, 3,000; polynuclears, 58 per cent.; small lymphocytes, 28 per cent.; mononuclears, 14 per cent. During the night the child had great difficulty in breathing, partial paralysis of the muscles of respiration, left arm, and left leg." The mother says that she first noticed weakness of the legs on July 18, two days ago. There has been no vomiting other than at the onset except yesterday when the child vomited once; no undue sweating. The mind has been clear.

Physical Examination.—The patient is a well built little boy, lying on his back. He is perfectly conscious and aware of all that goes on around him, but talks with difficulty in a very weak voice. Respiration is very labored and rapid, the *alæ nasi* dilating with inspiration, and with each inspiration there is a marked contraction of the sternomastoids on both sides. Associated with this is an extension of the neck and a protrusion of the jaw. The chest itself does not move at all with respiration. The abdomen moves in a normal manner, but the excursion of the abdominal wall is limited and weak. The skin is clear and white. Mucous membranes are pale. There is no discharge from the nose. The lips are covered with dry crusts. *Eyes.*—Movements of the eyeballs and pupillary reactions are normal. *Ears.*—Hearing is apparently normal. There is no discharge and no tenderness over the mastoids. *Mouth.*—The tongue is coated and protrudes straight. The teeth are in good condition. The tonsils are enlarged and ragged; there is no exudate. No herpes. *Superficial lymph nodes.*—There are small glands in the neck, axillæ, and groins. *Chest.*—Well formed, costal

angle about ninety degrees. The chest does not move with respiration. *Heart*.—Normal. There is a systolic murmur all over the precordium, most marked at the pulmonary area. *Lungs*.—Both fronts are normal on auscultation and percussion, but the breath sounds are very feeble. As far as can be ascertained, the backs are also normal, but turning the child over disturbs him greatly. *Abdomen*.—Level, soft, tympanitic, no masses or tenderness. Abdominal wall moves normally with respiration, but to a limited extent. The abdomen is symmetrical. *Liver*.—Flatness extends from the sixth rib to the costal margin. The edge is not felt. *Spleen*.—Not felt. *Muscular system*.—Legs: the Achilles reflex is present on the right side, the other reflexes are negative. Movements of the legs: it is difficult to ascertain how far the legs are paralyzed and how far lack of movement is due to weakness alone. His mother says that he can move the right leg, but no movements were seen. The left leg is very weak and flaccid, apparently completely paralyzed. Arms: the hands, wrists, and forearms can be moved on both sides. The muscles of the shoulders are apparently paralyzed, for the child seems unable to move his upper arms. Face: the face is not quite symmetrical. There is slight smoothing out of the right side of the face. The folds on the left side are somewhat more marked than on the right. This becomes more definite when the child talks or smiles.

July 20. 3:30 P. M. The pulse is irregular. At the apex a series of beats is heard at a rate of about 100; then the rate suddenly changes to 120 to 140, and a series of regular beats occurs at this rate. The rhythm of both rates is regular. Each rate may continue for three to four beats or perhaps twenty or more beats, then there is a quick change to either a slower or faster rate. The fast rate is not so much as twice that of the slow rate. No abortive beats and no compensatory pauses are heard. Between the most rapid rate and the slowest, other variations in rate seem to occur. The arrhythmia reminds one of rapidly recurring attacks of paroxysmal tachycardia.

Later. After admission the child's condition became gradually worse. He could at first speak a few words, though articulation was difficult. His lips moved and one could see that he was trying to talk, and after a few attempts he managed to utter a few words, such as "a drink of water"; the words were clear and distinct. After the first few hours he became unconscious. Movements of the limbs were few, presumably from weakness. Respiration was very labored and seemed to involve a voluntary muscular effort. The chest wall did not move. There was paralysis of the intercostal muscles. The muscles coming into play most actively were the sternomastoids and the platysma on both sides. The scaleni appeared not to act. On admission, the diaphragm was acting normally, but weakly. The diaphragm became weaker and weaker, *i. e.*, the movement of the abdominal wall became less and less, and towards the end it was scarcely noticeable. As death approached, respiration continued to be very rapid, 40 to 50, was even more short and gasping than before, and seemed to be carried on almost wholly by the sternomastoids and other neck muscles. The heart meanwhile continued to act strongly, showing constantly the type of arrhythmia noted above, an irregular hurrying and then slowing, simulating more a rapidly recurrent paroxysmal tachycardia, than an auricular fibrillation with pulse intervals of constantly varying lengths. The strong action of the heart up to the end was in marked contrast to the constantly failing respiration. The child took no

nourishment, only sips of water. Whether or not he could swallow could not be determined. He refused even on admission to take more than just enough fluid at one time to moisten his lips, which were very parched. Respiration grew weaker, the child became unconscious and at 7 P. M. died, of respiratory failure. The child was extremely pale on admission. Cyanosis was not especially marked until the very end, and even then it was no more marked than one usually sees it just at death. It was at no time a prominent sign.

CASE 26.

Roger P., age, 4 years. Hawthorne, N. Y. Admitted, September 20, 1911. Died, September 23, 1911. *Diagnosis.*—Acute poliomyelitis.

Family History.—The father and mother are living and well. There are two other boys in the family, aged two and nine years. Both have been perfectly well. All three children had measles last May. The two year old boy was slightly indisposed seven days ago. He was somewhat irritable and had little appetite but no vomiting or diarrhea. The family live in a small village in a house with grounds. The air and light are excellent. There are few mosquitoes, no roaches or other bugs, but large numbers of flies. The children have played about with other children in the neighborhood. T. R., who now has poliomyelitis in the Hospital, lives in the same village, but the patient had not been in the habit of playing with him. There are no other cases in the neighborhood.

Past History.—The patient has always been a strong healthy child.

Present Illness.—On Saturday, September 16, four days ago, the child suddenly complained of pain in the right leg. On trying to run about, the leg seemed somewhat weak. At that time he also had slight fever and very little appetite. He was kept in bed most of the time but on Monday afternoon, was up and able to walk about a little without assistance. He has not walked since, however. It has been noticed that his left arm has been very weak and also his back. The voice has seemed somewhat thin. He has not vomited and has had no convulsions. He has been rather drowsy, complained of headache and pain in the stomach, and has been constipated since Saturday. No retraction or rigidity of the neck has been noticed. On the evening of September 18, and yesterday morning, respiration was rapid and shallow, but it did not appear to be so last night. He has not urinated since this morning.

Physical Examination.—The child lies quietly on his back. He seems drowsy and does not respond readily to questions. The expression is rather dull. There is no evident distress. Color is good, skin hot and moist. *Head.*—The head is well formed; no frontal bosses. *Face.*—The right side of the face is slightly relaxed, and the mouth is drawn to the left on smiling. *Eyes.*—Pupils are equal, not dilated, react actively to light. There is no ptosis. External ocular movements are good. Sclerae and conjunctivae are clear. *Ears.*—No discharge or tenderness. *Mouth.*—Lips and mucous membranes are of good color. The tongue has a moderately moist white coat. Pharynx is not injected. Tonsils are not large; no exudate. *Neck.*—The head falls back on sitting up, so that there is evidently paralysis of all the anterior muscles. There is no retraction, the child resists bending forward because of pain. *Superficial lymph nodes.*—Not enlarged. *Chest.*—Well formed and symmetrical. Expiration is almost imperceptible. Breathing is purely diaphragmatic. The lower ribs do not move on

inspiration. The upper portion of the thorax is pulled up slightly on inspiration, although there is no marked action of the accessory muscles. The diaphragm descends well but seems a little weak. There is no pulling in of the ribs at the attachment. Respiration is shallow with an occasional, deep, sighing inspiration. Lungs are clear on percussion and auscultation. *Abdomen*.—The abdomen moves normally with respiration. The muscles seem soft and relaxed. There is no bulging in the flanks, no masses or tenderness. Superficial reflexes are absent on both sides. *Extremities*.—Upper: the grip of both hands is weak. There is no wrist drop. The fingers can be extended and flexed. Movements of the forearm are good. There is paralysis of the deltoid on both sides. Reflexes are absent. Lower: there is complete flaccid paralysis of the left leg. The toes of the right foot can be slightly flexed. Right leg: there is paralysis of the quadriceps, iliopsoas, and extensors of the foot, and weakness of the posterior muscles of the right lower leg. The toes can be fairly well flexed. The right gluteus and the hamstrings are active. All reflexes are absent on both sides. An attempt to elicit the Kernig's sign causes pain, but no muscle spasm.

September 20. Blood count: leukocytes, 12,400; differential count of 200 cells: polymorphonuclears, 53 per cent.; transitionals, 12 per cent.; lymphocytes, 24 per cent.; basophiles, 0; large mononuclears, 9.5 per cent.; eosinophiles, 0; stimulation form, 1.5 per cent. Spinal fluid: 22 c.c. of clear fluid; pressure slightly increased; cells, 55 per c.mm.; almost all mononuclears; globulin, very slightly +; sugar, +.

Later, near midnight, the patient could be seen just beginning to use the accessory muscles in the neck, and the *alæ nasi* were also working.

September 21. The child slept fairly well. This morning the costal muscles seem entirely paralyzed, and the accessory muscles in the neck are working much more obviously. The diaphragm is acting but not strongly. The lungs are clear. There is localized sweating of the upper lip. Later in the morning: the respiratory embarrassment is much more marked. The right side of the abdomen moves more than the left with inspiration, as though the left side of the diaphragm were not working. The child is drowsy but is easily aroused, and then is a little irritable. He can move the forearms and hands but not the shoulders. The pupils are equal and contracted.

In the afternoon the diaphragm seems to gain strength and the abdomen moves evenly and with a somewhat greater excursion. The rate of respiration is rather low, considering the amount of respiratory embarrassment. The breath sounds are not heard below the junction of the lower and middle third of the axilla. There are no rales. Towards evening the heart rate increased and a sinus arrhythmia was noted. There was some difficulty in swallowing noted, but this seemed at first due as much to increase of respiratory embarrassment as to actual failure of the muscles of swallowing. Later, however, quite definite failure of deglutition was noted. The child throws his head from side to side at times and cries weakly, seemingly trying to escape from some oppression. He cannot articulate clearly. The eyes are bright, but often he dozes, and the eyeballs roll up beneath the incompletely closed lids.

September 22. The patient slept fairly well. This morning he is breathing with about the same degree of embarrassment as yesterday evening, perhaps a trifle more easily. The rate of inspiration varied between 22 and 30 during the

night. The temperature is falling by a fairly steep curve. The patient's color is good. He seems to be getting enough air. This morning he is rather more drowsy than he has been. He cannot articulate properly, but he swallows much better than yesterday evening. Occasionally he moves his head vigorously from side to side. The arms are moved more extensively than yesterday. The abdomen moves about as much in respiration as yesterday, perhaps the right side a little more than the left.

P. M. The patient has gone on well through the day. He breathes a trifle more easily this evening. The abdomen certainly makes a slightly greater excursion on inspiration than it did this morning. The child takes his fluids much better this evening. This morning he had much difficulty in swallowing. He is more willing to try to speak and succeeded in articulating and phonating the words "good-night" weakly but distinctly.

September 23. This morning about 5:30 the patient began to weaken markedly. His color is not so good; there is a faint tinge of cyanosis on the lips and duskiess about the eyes. He is pale. The breathing is much shallower than last night. The heart action is fairly strong. From time to time there is definite sinus arrhythmia. The child's mind is quite clear and he can speak. He knows perfectly well what he wants and does not want. The left side of the diaphragm evidently does not descend at all, for that side of the abdominal wall remains motionless. From time to time faint, blotchy, localized flushings appear on the face and fade again. At 10:35 A. M. the first heart sound has become rather tapping and sharp in quality. The action is somewhat slower and also more constantly irregular, though the arrhythmia is very slight.

P. M. The patient has been breathing less and less deeply through the day. Cyanosis of the finger tips has been gradually developing and a slightly more dusky appearance of the face and lips. The left side of the diaphragm is apparently not acting at all. The abdomen is becoming distinctly more scaphoid, and the tissues are losing their elasticity. The child is in a rather deep coma, from which he can be roused, however, by persistent prodding. He swallows with difficulty. About two hours before death the child suddenly seemed to come out of his coma for a few moments and looked about him with perfectly clear eyes and apparently recognized his parents. This transient return to consciousness was of extremely short duration, and he rapidly lapsed again into coma. Following immediately upon this, the child's breathing became suddenly worse and in a few moments stopped entirely. He looked as though he had died. During the period of respiratory pause which lasted, with the exception of one or two shallow gasps, for nearly two minutes, the heart continued to beat, at first regularly as before, but after a few seconds with a noticeable irregularity. This soon took the form of alternating series of regular rapid beats and regular slow beats. The sounds conveyed the impression that tracings from the heart taken at the time would have shown a typical curve of Luciani periods. At the end of the long respiratory pause, the patient suddenly began to gasp more deeply and in a few breaths had resumed his previous respiratory rhythm. This continued for nearly two hours longer and then stopped suddenly as it had in the first instance. Electrocardiographic records were taken at short intervals throughout the last eight or nine hours of life.

CASE 27.

George G., age, 9½ years. Brooklyn, N. Y. Admitted, August 30, 1911. Died, September 1, 1911. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. Habitat: the family lives in a double house, which is quite new and is clean and airy. No parasites are found in the house. There is no history of any contact of parents or child with any lame or paralyzed person.

Past History.—The child has always been healthy.

Present Illness.—The present illness began about a week ago. The child felt poorly, was given physic, and soon appeared perfectly well again. No definite symptoms were noted until Sunday, August 27, three days ago, when he complained of stiff neck. He vomited once the same day after a meal, but has not vomited since. He has had no convulsions, and the mother has noticed no profuse sweating. At the onset he had headache and indefinite pain in both thighs, lasting a few hours, but besides this has had no especial pain. Throughout the illness his mind has always been clear. He eats well, but his bowels are constipated. He has no trouble with voiding, no cough, or sore throat. He has been kept in bed, and his illness was considered to have been cerebrospinal meningitis. There was no history of paralysis until this morning (11:00 A. M.) when he had lost knee jerks, and the muscles of his legs were flaccid and motionless. Since then the left arm has become involved (noted at 4:00 P. M.). This morning his respiration was entirely abdominal and his thorax was motionless. Expiratory grunt was also noted. He has not been drowsy and not especially restless.

Physical Examination.—The patient is a rather slim, well built boy. He lies on his back with his eyes open and is evidently quite alert, noticing all that goes on. He responds to questions brightly but with a very weak voice. Respiration is rapid, rather jerky, and labored. The alæ nasi dilate with respiration and the muscles of the neck, especially the sternomastoids, take an active part in respiration. The chest itself is quite motionless. Respiration is wholly of the abdominal type, but the impression is given that the diaphragm is not acting very strongly. There is slight protrusion of the lower jaw with inspiration. The face is symmetrical. The skin is rather dark in color; no rash. *Eyes*.—The pupils are equal, regular, and react rapidly to light. Ocular movements are normal. Eye slits are equal. *Ears*.—No discharge or tenderness. *Nose*.—No discharge or excoriation. *Mouth*.—The tongue protrudes straight and has a thick gray coat. The tonsils are not enlarged. The pharynx is negative. The child can swallow milk well. *Neck*.—There is stiffness of the neck and resistance to flexion, but no retraction. *Superficial lymph nodes*.—The glands in the neck, axillæ, and groins are just palpable. *Chest*.—Well formed and symmetrical. *Lungs*.—Clear throughout on auscultation and percussion. *Heart*.—Not enlarged, action regular and rapid. Sounds are of good quality. A soft systolic murmur is heard all over the precordium, most marked in the pulmonary area. *Abdomen*.—Slightly distended, soft, tympanitic. No masses or tenderness. Abdominal reflexes and cremasteric reflexes are not obtained. *Muscular system*.—Legs: knee jerk and Achilles reflex are absent on both sides. Babinski reflex is negative on both sides and also Kernig's manipulation, which is slightly painful. *Movements*: no movements are obtained in either leg except flexion of the toes. *Sensation*: the

touch of a camel's hair brush is easily felt over both feet and lower legs. Arms: there is apparently a paralysis of both upper arms. The child can flex and extend the fingers weakly and can flex both elbows. Abdominal muscles: there seems to be some tone to the abdominal muscles. The neck and back muscles were not tested. The boy is so sick that an accurate localization of the paralysis is impossible.

August 30. Blood count: leukocytes, 16,700; differential count of 200 cells: polymorphonuclears, 75 per cent.; lymphocytes, 15 per cent.; large mononuclears, 0.5 per cent.; transitionals, 9.5 per cent.

August 31. The boy's condition is about the same. He is bright and alert and appears to be making a good fight. Each breath he takes comes with obvious effort. Except for breathing, however, he makes no effort to move. When he speaks it is in a low voice, and he only speaks in order to answer questions or to make known his requests. He says respiration is easier than last night. He asks to have his nightgown loose about his neck, to be turned over, etc. Physical examination shows nothing new. No rales in the lungs. The respiration is abdominal, and the movement of the abdomen seems weaker than last night. The upper part of the chest moves with respiration, but this movement seems to be due to the pull of the neck muscles. The arms seem a little weaker.

Afternoon. The excursion of the abdomen in respiration is very slight. Later in the evening there seemed to be a distinct improvement in the patient's condition. He answered questions more fully, moved his head more, and in general did not display the extreme economy of muscular effort seen earlier in the day. The breathing was distinctly less shallow. Abdominal excursion was fair. Urine examination: clear, amber; specific gravity 1.017; reaction acid; Fehling's sugar test negative; albumen test with heat and acetic acid negative; sediment, heavy, yellow, granular precipitate.

September 1. The patient dozed for short periods during the night. At 6 o'clock, breathing became worse. The patient was cyanosed, especially about the lips. The effort to expand the chest was great, and spent chiefly through the sternocleidomastoids, the muscles of the floor of the mouth, and the hyoids. The pull on the inner end of the clavicles was such that a partial anterosuperior dislocation of the sternoclavicular articulation resulted with each inspiration. Slight protrusion of the epigastric region indicated weak diaphragmatic action. This continued practically to the end. About one half to three quarters of an hour before death the heart action, which had been regular and moderately strong, began to show irregularity. At first this seemed to be an occasional sinus arrhythmia. A little later, however, alternating stretches of slower regular, and more rapid regular rates were observed. Sometimes the rapid period was entered by a gradually and evenly increasing rate. The slow rate was usually approached more abruptly by a few, irregular, slower beats. There was no change in the quality of the sounds. Notwithstanding the respiratory difficulty and the failing heart, the lungs remained clear of adventitious sounds until a few minutes before death, when some scattered rales appeared in the lower left axilla. The patient remained semi-conscious almost until the last, although a somewhat delirious muttering was present for two hours or more before death. There was a slight inequality of the pupils, the left being a little larger, during the last hour or more. The heart continued to beat very weakly and regularly for a minute or two after respiration ceased. The sounds could be heard over the base but not at the apex.

CASE 28.

Belle T., 8 months old. New York City. Admitted, August 21, 1911. Died, August 23, 1911. *Diagnosis*.—Acute poliomyelitis.

Family History.—The mother and father are living and well. There is one other child in the family, who subsequently also developed poliomyelitis and died.

Past History.—The child is one of twins. She has always been healthy except for erysipelas at the age of three months.

Present Illness.—On Friday, August 18, the mother noticed that the child was feverish. The doctor was called on Saturday morning. At this time, the child had a temperature of 103° F., pulse 120, and was very nervous. The throat was red and inflamed, and there was a slight patch on the right tonsil. In the evening the throat was clearer, but the temperature remained at 103° F., the breathing became more rapid, and a few rales were heard. The doctor said it looked like pneumonia. Later in the evening the temperature fell to 101° F., pulse 110. August 20, the child appeared brighter and not so nervous, but she was not so lively as usual. She slept well that night. Next morning, she was again seen by a physician who noticed twitching of the face and a peculiar movement of the toes of the left foot, almost rhythmical in character. About this time weakness was also noted in the left arm and leg. At noon the same day, August 21, Dr. Francis Huber made a diagnosis of poliomyelitis. During her illness there has been no vomiting and the bowels have been normal.

Physical Examination.—The patient is a well nourished infant who lies quietly and is rather somnolent. She frets a little if handled much, but lapses rapidly into the sleepy condition. The expression is a drowsy one, and not alert. *Eyes*.—There is a slight but definite internal strabismus. The pupils are equal and react normally to light and accommodation. *Ears*.—Normal. *Mouth*.—The buccal mucous membrane is clean. The pharynx is a trifle reddened. The tonsils are a little enlarged. *Head*.—The anterior fontanelle is open widely; no bulging or pulsation. *Neck*.—Not stiff. No power in the neck muscles is apparent; the head drops when the baby is sitting up. *Superficial lymph nodes*.—The cervical nodes are palpable. *Thorax*.—Well formed, moves very little in respiration. *Lungs*.—Apparently normal. *Heart*.—Apparently normal. *Abdomen*.—Rather prominent, moves properly and rather widely in respiration. It is soft, there are no masses, but palpation of the abdomen seems to produce discomfort. *Extremities*.—Upper right: no definite weakness is found. Left: there is definite loss of power in the shoulder muscles, probably in the biceps also. Flexors and extensors of the hand, and extensor of the forearm are weakened but not paralyzed. Lower right: there seems to be complete weakness of the thigh muscles. The flexors and extensors of the foot are still intact. Left: practically the same condition exists on the left though not quite so marked. The reflexes are absent on both sides.

August 21. This evening a few fine dry rales are heard along the inner border of the left scapula. Respirations are very rapid and short, but the diaphragm is working well. Blood count: leukocytes, 23,900; differential count of 200 cells: polymorphonuclears, 25 per cent.; large mononuclears, 2 per cent.; lymphocytes, 57 per cent.; transitionals, 25 per cent.; eosinophiles, 1 per cent.

August 22. Urine examination: pale yellow, clear; reaction acid; Fehling's test for sugar negative; test for albumen with potassium ferrocyanide negative; slight sediment.

August 23. Last evening the breathing, which had been extremely rapid since admission, was not so deep, and the *alæ nasi* were moving. They had not been active previously, even with the high respiratory rate. This morning the respiratory motions are much more short and jerky. The floor of the mouth and glottis are drawn down forcibly with each inspiration. There is considerable power in the dorsal flexors of the left foot. The right is much weaker. The knee jerks are both absent, likewise Achilles reflexes. The child is drowsy and apathetic. The heart action is regular and strong, 174 per minute. There are a few rather moist rales on the left lung. The abdomen seems to move more on the left side than on the right, and the viscera appear to move downward and toward the right with each contraction of the diaphragm, as though the left half of the muscle were acting more strongly than the right. It is almost impossible to determine whether the abdominal muscles are paralyzed. There seems to be a very faint reflex on the left, none on the right. During the morning the child's condition gradually becomes worse. From time to time beads of perspiration appear on the lips and forehead. The accessory muscles of respiration are brought more and more actively into play. There is a striking collapse of all the tissues. The rotundity of outline has gone almost visibly from the costal margins, the abdomen becoming more and more scaphoid and the tissues looking faded and inelastic. The motion of the left side of the abdomen is very marked, the right side is practically motionless. From time to time the tongue is protruded, and the lips are opened and shut as though to moisten the mouth. Frothy saliva covers the tongue and lips. There is cyanosis of the tongue, but the lips are marked rather by pallor than by blueness. The whole face is pale. Heart rate, 192 to the minute at 1:20 P. M., action strong and regular. The rales are still present over the left anterior axillary region. The breath sounds are somewhat better heard over the lower left chest than the lower right. The pupils are equal and remain strongly contracted. They react, however, to light. During the afternoon the respiration becomes constantly weaker. The left half of the diaphragm continues to act but the respirations are very shallow and jerky. The child dies suddenly at about 5 P. M.

CASE 29.

Frances K., age, 21 months. Perth Amboy, N. J. Admitted, September 11, 1911. Died, September 16, 1911. *Diagnosis*.—Acute poliomyelitis; bronchopneumonia.

Family History.—Negative.

Past History.—The child has always been healthy except for an attack of bronchitis last year.

Present Illness.—On Wednesday, September 6, the patient was perfectly well. On Thursday she had a slight fever, was restless, and evidently had pain when handled. The restlessness continued on Friday with loss of appetite and alternating constipation and diarrhea. On Saturday the doctor noticed paralysis of the right arm and slight weakness of the neck. There was an increase of paralysis of the neck on Sunday and the child vomited once. Today there is possibly some weakness of the left arm also. The child has had no convulsions. No urinary symptoms.

Physical Examination.—The patient is a well nourished little girl, who lies

quietly without moving and is very pale. She dozes easily, but can be aroused and made a little fretful by handling. She is breathing very fast. *Eyes*.—The pupils are equal and react to light. The left eye has a slight external strabismus. Ocular motions are coördinate. *Nose*.—No secretions, no excoriation. *Ears*.—Normal. *Mouth*.—The tongue is heavily coated, grayish yellow. The pharynx is reddened; the tonsils are much enlarged. *Neck*.—There is flaccid paralysis of the anterior and most of the posterior muscles. Attempt at flexion causes pain. *Superficial lymph nodes*.—The posterior cervical, axillary, and inguinal nodes are enlarged. *Thorax*.—Well formed; respiration almost entirely thoracic. *Lungs*.—Clear. *Heart*.—Rapid, regular. *Abdomen*.—Retracted in inspiration; soft. Liver and spleen not enlarged. *Extremities*.—Upper: there is flaccid paralysis of the entire right arm, and of the left shoulder and arm. There is some power in the forearm (flexors). Wrist drop is present. Lower: the reflexes are absent on both sides. Kernig manipulation is painful, but resistance to it is voluntary. The left quadriceps is weak. *Temperature*.—101° F.

September 11. Spinal fluid: 20 c.c. clear limpid fluid; pressure not increased; cells, 174 per c.mm.; globulin, 0; sugar, +; polymorphonuclears, 3 per cent.; mononuclears, 97 per cent. Blood count: leukocytes, 26,300; differential count of 200 cells: polymorphonuclears, 70 per cent.; lymphocytes, 16.5 per cent.; large mononuclears, 3 per cent.; transitionals, 9 per cent.; basophiles, 0.5 per cent.; eosinophiles, 0.5 per cent.; stimulation form, 1 per cent.

September 12. This morning the temperature is 103° F. The child is breathing rapidly and almost entirely with the thorax. The alæ nasi are working more than they were last night. There is no advance of paralysis. The child is very ill.

September 13. The temperature at 8:00 A. M. is 100.8° F. Breathing is rapid and slightly irregular, purely costal in type. The accessory muscles of respiration are not brought into play. The ribs move well, the costal border flaring well out. Condition of paralysis unchanged.

September 14. The patient is not so well this morning. There is a faint tinge of cyanosis on the lips and a duskiess over the forehead. The alæ nasi are working strongly. Accessory respiratory muscles in the neck are beginning to work. The thorax still expands well, but the impression is given that the musculature is becoming fatigued. The patient has rather a fixed distant expression in the eyes, and occasionally the lids droop and the eyeballs roll slowly upward, sideways, and back again. The temperature continues between 101° and 102° F. The pulse rose to 160 this morning. It is small and very weak. Throughout both sides of the chest there are numerous scattered moist and sibilant rales. Transient vasomotor phenomena are seen in the irregular flushings of the face.

Spinal fluid: 15 c.c. of clear fluid; pressure not increased; cells, 44 per c.mm.; largely mononuclears; globulin, 0; sugar, +.

September 15. This morning the patient seems perhaps a little brighter than yesterday. The costal breathing is as strong as ever. The cyanosis is distinctly less. The alæ nasi are working as before. There is slightly more marked impairment of resonance over the left upper chest anteriorly and in the left axilla than on the right. The adventitious sounds consist chiefly of loud squeaks and coarse sounds. Posteriorly the rales predominate and are of a distinctly moist character. There are also many scattered rales over the right lateral and

posterior aspects of the chest. There is a slight expiratory grunt. The temperature continues between 101° and 102° F. The pulse rate is high and is stronger this morning.

8:30 P. M. Numerous rales can be heard, and over the lower axilla there is a distinct bronchial quality to the breath sounds. The percussion note is less dull on the left than it was this morning. Weakness of the muscles of deglutition is very marked.

September 16. The patient is much worse this morning. She is pale with a bluish tinge on the lips and general duskiness. The breathing is shallower and very rapid. The patient is more apathetic. The sounds in the chest are chiefly squeaks and coarse rales. It seems now like a generalized bronchitis of the larger tubes. She coughs occasionally, a loose cough.

About an hour later, the patient suddenly became much worse. The respiration was shallow, heart rapid and weak, and death followed. The heart continued to beat for some minutes after respiration ceased. Spinal fluid removed post mortem: about 6 c.c. of clear limpid fluid; cell count, 118.8 per c.mm.; globulin, slightly +; sugar, +.

CASE 30.

John A., age, 13 months. New York City. Admitted, October 29, 1911. Died, November 4, 1911. *Diagnosis*.—Acute poliomyelitis, bronchopneumonia.

Family History.—Negative. *Habitat*: the family lives in a rather dirty tenement on East 17th Street. They have three rooms. There are about twenty children in the house. No sickness of any kind is known in the house or neighborhood. There are no lame children in the neighborhood.

Past History.—The child has never had any illness.

Present Illness.—Five days ago, Tuesday, October 24, the baby seemed somewhat ill. He was quiet and would not play. The mother gave him castoria and on Wednesday he seemed perfectly well again. On Thursday he again seemed ill and was very quiet. He was worse on Friday and stayed in bed all day. That night, that is, thirty-six hours ago, the mother noticed that he did not move his legs. Yesterday he was no better and last night it was noticed that he preferred to lie with his head well back. This morning, Sunday, a doctor was called in and diagnosed the case as poliomyelitis. There has been no cough, diarrhea, constipation, or sweating, no nervous excitement. The child has not been very cross and has cried very little. There has been no stupor.

Physical Examination.—The patient is a pale, but healthy looking child, who lies quietly on his back. He is not very drowsy but becomes fretful and whines if handled. *Eyes*.—Pupils are equal and react normally. *Nose*.—No discharge. *Ears*.—Normal. *Neck*.—The neck is stiff. The patient can hold his head up in the sitting position, but prefers to let it fall back. *Superficial lymph nodes*.—The posterior cervical, axillary, and inguinal nodes are palpable. Many are enlarged. *Thorax*.—Well formed; respiratory movements normal. *Heart*.—Rapid but normal. *Abdomen*.—Not distended, no tenderness. Several small but distinct nodules can be felt in the central part of the belly. They do not feel like fecal masses and strongly suggest enlarged lymph nodes. Liver and spleen are not felt. *Extremities*.—Upper: there is distinct weakness of the right deltoid. Lower: there is complete flaccid paralysis of both lower extremities. Knee jerks

and Achilles reflexes are absent on both sides. There is no Babinski reflex. Kernig manipulation is not resisted but causes pain. *Surface*.—There are numerous small scaling spots on the forearms, and over the legs are many pink dull spots about 5 to 10 mm. in diameter. They look like healed impetigo. There is also a smaller, more punctate eruption over the forehead and a few papules on the face. *Temperature*.—101.6° F.

October 29. Lumbar puncture today yielded a clear fluid apparently under somewhat increased pressure. Cell count, 90 per c.mm.; lymphocytes, 92 per cent.; polymorphonuclears, 6 per cent.; large mononuclears, 2 per cent.; globulin, 0; sugar, ++ (quick reduction).

October 30. The child is in good condition. He has considerable pain on moving the neck and legs. The left side of the mouth does not move quite so much as the right side does when the child cries. The left eye does not close so tightly as the right when the child cries. Lungs: there are generally scattered rales and a few piping sounds both anteriorly and more especially posteriorly. The child has a distinct grunting expiration at times. The temperature rose this morning to 102.2° F.

October 31. There are rales all through both sides of the lungs. In the right upper back the breath sounds are a little harsh. The upper right front is less resonant. Rales are everywhere coarse and bubbling.

November 1. Both sides of the chest are full of moist bubbling rales. The child looks badly. Respiration is almost wholly thoracic. There is inspiratory retraction of the abdominal wall, but when the thorax is compressed, the abdominal wall acts in a way that seems to indicate some weak diaphragmatic action.

November 2. There are not so many moist bubbling rales this morning, but numerous squeaks. There is a good deal of mucus in the throat, and the child frequently gives a loose bubbling cough. There is very definite and almost complete weakness of the diaphragm. The child looks pale and possibly very faintly dusky. The weakness of the right deltoid has advanced to complete paralysis of practically the whole extremity. Only weak action of the muscles of flexion of the fingers remains. Possibly the left deltoid is also weak. The temperature was 101° F. at noon.

November 3. The child has a high temperature this morning. Both sides of the chest are full of rales, coarse and bubbling. The diaphragm is hardly working at all. The child looks very badly. The temperature is 104.4° F.

P. M. The child has been out of doors all day. The physical condition is unchanged except that there are, if anything, fewer rales in the chest. The pulse is good.

November 4. Last evening the child looked rather badly, but the pulse was of good quality and not very rapid. When seen at 2:00 A. M. the condition was unchanged. At 6:00 A. M. just after the nurse had felt the pulse and found it unchanged, the child suddenly died.

CASE 31.

Max G., age, 4¾ years. New York City. Admitted, October 18, 1911. Discharged, November 11, 1911. Result, improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Negative. *Habitat*: the family lives in an eighteen family tenement. The house is new and is clean and airy. There are cockroaches, but

no other parasites in the house. The family have no cats or dogs. The father knows of no possibility of contact.

Past History.—The child has always been well and strong.

Present Illness.—On Sunday morning, October 15, three days ago, the child was perfectly well. That evening he had a headache and a little fever. The next morning he was feverish and vomited. Since that time he has been in bed. Monday evening the child had pain on moving his neck. He has not sweat much and has had no convulsions. Bowels and urination have been normal. On the first day of his illness the child was very drowsy, but he has been getting brighter. On Monday, two days ago, it was noticed that he was unable to move his eyes so as to look downward. He could close the eyes and move them normally in other directions. No other muscular weakness has developed. The child swallows normally but eats little. He has had fever each night.

Physical Examination.—The patient is a well built, rather thin boy, lying on his left side with his legs curled up. Both eyes are partly closed, leaving a slit about 0.5 cm. in width in which the sclera and lower edge of the cornea of the up-turned eyes are visible. The child mutters from time to time, but it is difficult to understand him. He responds weakly to his name and answers simple questions, but he is very drowsy and when left alone lies in a semistuporous condition. The skin is hot and dry. The face is symmetrical, is not flushed, and there is no rash. *Eyes.*—The eyelids close normally when the patient is told to close the eyes. When let alone his eyes are held closed. There is marked photophobia. The pupils are equal and react quickly to light and accommodation. Both eyes move upward, and to the right and left normally, but neither eyeball can be made to look downward. He cannot see anything below the level of the eye. There is no strabismus. He can count his fingers quickly and correctly. *Ears.*—No discharge, no tenderness over the mastoids. *Nose.*—No discharge or excoriation. *Mouth.*—The lips and teeth are dry. The mouth is held partly open. The tongue is slightly coated, protrudes straight and for a good distance. Both tonsils are enlarged and ragged, but not reddened. The pharynx is not red. *Superficial lymph nodes.*—There is moderate enlargement of the glands in the neck, axillæ, and groins. *Neck.*—The neck is not retracted but it is held stiffly, and resists all attempts at flexion. *Chest.*—Well formed, symmetrical, moves normally with respiration. There is no rosary. *Lungs.*—Clear throughout on auscultation and percussion. *Heart.*—Not enlarged, regular, sounds of good quality. The pulmonic second is louder than the aortic second. A soft systolic murmur is heard over the precordium. *Abdomen.*—Full, soft, tympanitic, no masses or tenderness. Abdominal reflexes are active. There is no tache cérébrale. The abdominal wall moves normally with respiration. Abdominal muscles are strong. Cremasteric reflexes are active. *Extremities.*—Movements are normal and strong on both sides. Knee jerks on both sides are active when obtained, but are hard to obtain. Achilles reflexes are absent on both sides. Kernig's sign: when the legs are extended a little more than a right angle, he complains of pain and tightens up his hamstrings. The Babinski test is unsatisfactory, first an extension and then a flexion. Oppenheim test: a slight but suggestive extension of the big toe on both sides was obtained. There is no tenderness on pressure over the legs. Both arms move strongly and normally. Arm reflexes are not obtained. *Temperature.*—104.2° F.

Spinal fluid: clear, colorless fluid; cell count, 61.1 per c.mm.; differential count of 200 cells: polymorphonuclears, 22.9 per cent.; mononuclears, 78 per cent.; globulin, 0; sugar, slightly +.

October 19. The patient lies with a peculiar expression in his eyes like a fixed stare, but he says he feels much better than yesterday. He follows an object with his eyes until the object is moved downwards. Then he says, "I can't look down." There is slight external strabismus of the left eye. The pupils react normally. There is no paralysis of upper or lower extremities. The knee jerks are hard to obtain, but when they appear they are distinctly increased. Achilles jerks are both present. The Babinski reflex on the right is distinctly suggestive. The Oppenheim reflex is positive. There is marked ataxia of arms and legs. The patient contracts the hamstrings, and resists extension of the leg when Kernig manipulation is attempted. It is apparently quite painful.

Blood count: leukocytes, 13,000; differential count of 200 cells: polymorphonuclears, 51.5 per cent.; lymphocytes, 37 per cent.; large mononuclears, 7 per cent.; transitionals, 4.5 per cent.

October 20. Spinal fluid: clear watery; cells, 320 per c.mm.; sugar, +; globulin, very slightly +. Smear: a few red cells; polymorphonuclears, 66 per cent.; mononuclears, 34 per cent.; no organisms seen.

Note by Dr. Schirmer: "Position of the eyes when the boy looks in the distance is slightly divergent. Motion to the right and left side, and upward, is possible to physiological limits. In looking downward there is a marked restriction which is equal in both eyes. They can be moved downward to about 25° below horizontal. When the boy looks at a distance of one meter at my finger and I move the finger toward him, he is not able to converge at all. One of the eyes begins to deviate, and the movement develops into an associated one to one side."

October 21. His condition is about the same. The child can look down a little better. He is irritable and wants to be let alone. When he is asleep he has frequent jerky movements of the arms. Reflexes of the legs are the same as yesterday except that the knee jerks and Achilles reflexes are both more active.

October 23. The child is much better. There is slight strabismus (external on left?). The child can look down slightly better than before, especially with the right eye. Knee jerks are very active, Achilles reflexes active, Babinski negative, Oppenheim suggestive on both sides. Kernig's and neck signs are less painful.

Spinal fluid: 8 c.c. of clear limpid fluid; pressure not increased; cells, 6 per c.mm.; globulin, normal; sugar, +.

October 25. Eye grounds are negative.

October 26. Spinal fluid: 10 c.c. of clear, watery, colorless fluid; pressure not increased; cells, 25 per c.mm.; mononuclears, +; globulin, very slightly +; sugar, +.

October 31. Spinal fluid: 20 c.c. of clear, non-opalescent, watery fluid; cells, 11 per c.mm.; mononuclears, +; globulin, very slightly +.

November 4. The patient walked today. He showed a peculiar unsteadiness which at first looked like ataxia. On close examination, however, there is seen to be a distinct element of spasticity in the gait. The knee jerks are both exaggerated. The Achilles jerks are hard to obtain and certainly not exaggerated. The eye condition is unchanged. The patient cannot converge.

November 11. The general condition is excellent. The patient's gait is now quite steady. Eyes: the tendency is for him to roll his eyes up, but he can move them both down almost normally. There is a slight external squint of the left eye. He does not converge. He seems to focus with his right eye, and if this is closed, he has to rotate the left eye inward in order to see an object. Both knee jerks are exaggerated, both Achilles jerks are nearly absent, abdominal and cremasteric reflexes are very active. Kernig manipulation is still painful. Oppenheim and Babinski tests are negative.

CASE 32.

Patrick T., age, 23 months. New York City. Admitted, October 4, 1911. Discharged, October 15, 1911. Result, improved. *Diagnosis.*—Acute poliomyelitis.

Family History.—One brother, aged five years, had tonsillitis seven days ago and now has a cold in the head. He has had no muscular weakness or drowsiness. One sister, aged four years (Hannah T.), was admitted yesterday to Ward 1, with poliomyelitis. Another sister, aged three years, had a cold one week ago, lost her appetite and was drowsy for two days, but is now perfectly well. She has had no muscular weakness. A third sister, eight weeks old, is healthy. *Habitat:* the family lives on Lexington Avenue in a flat; they have five rooms, second floor front. The light and air are good. The mother and father, the sister with poliomyelitis, and the patient slept in the same room, the patient sleeping in a crib by himself and the sister in bed with the father and mother. The other three children sleep in a room together. There are flies, mosquitoes, bedbugs, and roaches in the house. The mother knows of no similar illness in the neighborhood.

Past History.—The patient has always been a strong healthy child.

Present Illness.—October 3, two days after the onset of the sister's illness, the mother noticed that the child was drowsy. This morning she noticed that he was "shaky" in his legs and could not rise from a sitting position. When placed on his feet he could walk fairly well, but dragged one leg. He has been able to use his arms well, and can feed himself. The mother has noticed no fever, but drowsiness has continued, and the child has been irritable. There has been no vomiting, no convulsions, and no retraction of the neck. Bowels and urination have been normal. The child had a cold and discharge from the nose one week ago, but it lasted only two days.

Physical Examination.—The patient is a well nourished, sturdy child. He is drowsy but can be easily awakened and then becomes irritable. On being let alone he quickly goes to sleep again. *Head.*—Well formed, forehead slightly prominent. *Face.*—Symmetrical in repose, not drawn to either side on crying. Both eyes can be closed. *Eyes.*—Conjunctivæ and scleræ are clear. Eye slits seem equal. There is a tendency to keep the eyes closed and when open to hold them away from the light. External ocular movements are good, no strabismus. Pupils are equal, moderately dilated, and react rather slowly to light. *Ears.*—No mastoid tenderness, no discharge. External ear, and meatus on the left side are reddened. Drums on both sides are obscured by wax. *Mouth.*—Lips and mucous membranes are of fair color. The tongue has a heavy yellowish white coat. Pharynx and fauces are red and injected. The tonsils are large and irregular; no exudate. *Neck.*—There is no retraction of the neck. The head is

easily held erect. There is no muscular weakness. The child resists anterior flexion slightly. *Superficial lymph nodes*.—The cervical, axillary, and inguinal glands are all easily palpable. *Chest*.—Well formed, expansion good, both sides move equally in respiration, diaphragm active. *Lungs*.—Clear on auscultation and percussion. *Heart*.—Action not especially rapid, sounds well heard, regular, no murmurs. The heart is not enlarged. *Abdomen*.—Soft, moves well with respiration, no masses or tenderness, no bulging. Abdominal muscles are strong. *Extremities*.—Upper: arm movements are normal, muscles show no weakness. Lower: the legs can be moved in all directions. The child stands alone and can walk well. The legs can be fully flexed on the abdomen without causing pain. The child, however, shows some irritability on being handled in this way. *Reflexes*.—Knee jerks, Achilles, and abdominal reflexes are active. Babinski and Kernig reflexes are absent. *Temperature*.—100.6° F.

October 4. Spinal fluid: 35 c.c. of clear limpid fluid; pressure definitely increased (child crying somewhat); cells, 94 per c.mm.; mononuclears, 93 per cent.; polymorphonuclears, 7 per cent.; globulin, 0; sugar, +.

October 5. Blood count: leukocytes, 15,000; differential count of 200 cells: polymorphonuclears, 52.5 per cent.; lymphocytes, 31.5 per cent.; large mononuclears, 13.5 per cent.; transitionals, 1.5 per cent.; basophiles, 0.5 per cent.; eosinophiles, 0.5 per cent.; stimulation form, 0.

October 8. There is no evidence of paralysis. Reflexes are present and active in the legs. The child can walk and stand well, but his gait is rather wobbly. He is irritable and cries a good deal, but it seems to be temper chiefly. His legs appeared weak, and he fell when first put on the floor, but when he was started walking towards his sister, he could walk very well.

October 15. The child walked very well this morning, several times up and down the hall. Both knee jerks and Achilles reflexes are active. The lungs are clear. The tonsils are both a little enlarged, but not especially red. Moving the legs (Kernig) seems to be painful still, though it is hard to distinguish the pain from temper. The child is discharged today.

CASE 33.

Helen B., age, 6 years. Bergenfield, N. J. Admitted, September 2, 1911. Discharged, September 17, 1911. Result, much improved. *Diagnosis*.—Acute poliomyelitis (abortive form).

Family History.—Negative.

Past History.—The patient has always been a healthy child and was never sick until July when she had whooping cough. She has always played with Helen K. (see case 29, page 174, who was taken sick on August 16 with acute poliomyelitis and died on August 21).

Present Illness.—The child was perfectly well yesterday morning, September 1. She came down to breakfast singing, ate her breakfast well, and played all the morning. She also ate a hearty lunch and played for a while in the afternoon. Later in the afternoon she complained of headache, which continued all the evening. She ate no supper, slept fairly well, was not very restless but seemed dull. This morning she ate no breakfast. She has not vomited, but there was no movement of the bowels yesterday. No weakness of any extremity has been noticed. She has had some pain in the back of the neck and forehead.

Physical Examination.—The patient is a well developed little girl who lies quietly in bed and looks acutely sick. The face is flushed, the expression of the eyes is heavy and the lids are half closed or closed most of the time; she is very apathetic. *Eyes.*—The pupils are equal and react normally. Sclerae are clear, ocular motions normal. *Nose.*—No discharge. *Face.*—There is no weakness of the face muscles. *Ears.*—Normal. *Mouth.*—Buccal mucous membranes are clear. The teeth are in fair condition. No Koplik spots. The tongue has a very light greyish coat. Pharynx and tonsils are slightly reddened. The latter are large, especially the right. *Superficial lymph nodes.*—The posterior cervical, submental, axillary, and inguinal nodes are palpable. *Neck.*—Not stiff. *Thorax.*—Well formed, respiratory motions are normal. *Lungs.*—Clear. *Heart.*—Rapid, sounds normal, action regular. *Abdomen.*—Not distended, soft, no tenderness, no masses. *Extremities.*—Upper: normal, no tenderness. Lower: there is good power in all the muscles, no tenderness. Kernig manipulation causes no pain. Knee jerks are very active on both sides; crossed adductor reflex is obtained on tapping the left patellar tendon. The Achilles reflex is also active, the Babinski reflex negative on both sides. *Back.*—No weakness, no pain or tenderness. *Surface.*—There are several fresh bruise marks over the legs. *Temperature.*—103.8° F.

September 2. Blood count: leukocytes, 8,800; differential count of 200 cells: polymorphonuclears, 77 per cent.; lymphocytes, 17.5 per cent.; large mononuclears, 0.5 per cent.; transitionals, 4 per cent.; stimulation form, 1 per cent.

Spinal fluid: 20 c.c. of almost clear limpid fluid. There is a faint suggestion of opalescence; cells, 5 per c.mm.; polymorphonuclears, 20 per cent.; mononuclears, 80 per cent.; globulin, 0; sugar, +.

September 3. The patient slept well. Her temperature is 99.1° F. This morning the temperature has fallen to 99° F. She is still rather listless but much less dull than yesterday. She has no headache. The knee jerks are still very active. The patient says she feels much better. She vomited after taking citrate of magnesia. In the afternoon the mother came and finding the child's temperature normal insisted on taking her home. She was advised to leave the patient but would not. The temperature was 98.7° F. The patient showed some unsteadiness on her feet but no definite weakness. About an hour later the mother returned in a taxicab with the child. On the way to the train she had complained bitterly of her head and vomited. The child begged to be put in bed. She looks greatly prostrated. There is a distinct tendency to keep the head back, and she assumes the position on her side so as to facilitate this. The neck is very slightly stiff, and an attempt to flex it causes pain. The Kernig manipulation causes pain about the knees. The knee jerks are still exaggerated.

Spinal fluid: about 2 c.c.; cell count, 62 per c.mm.; polymorphonuclears, 11.3 per cent.; mononuclears, 88.7 per cent.; globulin, slightly +.

Blood count: leukocytes, 11,200; differential count of 200 cells: polymorphonuclears, 79.5 per cent.; lymphocytes, 14.5 per cent.; large mononuclears, 0.5 per cent.; transitionals, 4.5 per cent.; basophiles, 0; eosinophiles, 0.5 per cent.; stimulation form, 0.5 per cent.

September 4. Temperature: 99.1° F. The patient seems a little better this morning. She is still apathetic and prefers to lie on her side. The neck resists flexion slightly, and the attempt to flex it causes pain in the occipital region.

Kernig manipulation causes slight pain in the knee. Knee jerks are not quite so exaggerated as before, but are still very active. No weakness is found in any muscles. The patient sits up well. The tongue has a heavy yellowish grey coating.

P. M. She has been drowsy and sleepy most of the day. When her father came the child paid hardly any attention to him and showed no signs of pleasure. She answered his questions only by nods and shakes of the head. As soon as he left the room she became drowsy again. At 3:00 P. M. the knee jerks are found to be so much less active than before that several blows of the hammer are required to elicit the jerk. They are fairly active when they do respond. No weakness is found. The neck is a little less stiff.

10:20 P. M. The patient is sleeping, but half waked during the process of eliciting knee jerks. They are less easily obtained than when last noted. When they respond it is with about normal activity. The stiffness of the neck late this afternoon (about 5:20 P. M.) had almost entirely gone.

September 5. The patient slept well this morning. She seems brighter, though still not very communicative. She has no headache. The neck is not stiff. Both knee jerks are active. She still prefers to lie in bed and does not feel energetic. The von Pirquet reaction made two days ago is positive, a raised urticarial-like area about 1 cm. in diameter, and red. The temperature dropped to normal last night. This morning it is 99° F. again.

P. M. The patient continues to be somnolent and takes very little interest in her surroundings. There is no change in her physical condition. It is possible that the right palpebral fissure is somewhat narrower than the left. Examination of the eye grounds reveals no abnormality.

September 6. The patient is a little brighter this morning but still prefers to lie in bed. The tongue has almost entirely cleared. The neck is a little stiff. "Signe de la nuque" is present. The left knee jerk is obtained on reinforcement only, right knee jerk normal.

Spinal fluid: about 8 c.c. of not quite clear, slightly opalescent fluid with faintly yellowish tinge; cells, 59 per c.mm., mostly mononuclears; globulin, slightly +; sugar, +. In the wet preparation for counting, a large phagocytic cell was seen containing five small mononuclears. Blood count: leukocytes, 5,600; differential count of 200 cells: polymorphonuclears, 68.5 per cent.; lymphocytes, 22 per cent.; large mononuclears, 1.5 per cent.; transitionals, 6 per cent.; basophiles, 0; eosinophiles, 1 per cent.; stimulation form, 1 per cent.

This evening the patient is somewhat brighter but continues to answer questions by shakes or nods and occasionally a monosyllabic reply. She is still drowsy. There is no change in her physical condition.

September 7. The patient seems better this morning. She sits in a chair easily, and also walks well, though perhaps with a little uncertainty. She continues to be most unresponsive. Her appetite is better, however, and she ate her supper last night, and breakfast this morning with a relish. There is still slight rigidity of the neck and "signe de la nuque." The knee jerks are the same as yesterday.

September 11. The patient is considerably better. She talks more and is losing her apathy. She still does not feel much like sitting up. The knee jerks are active. Spinal fluid: 20 c.c. of clear limpid fluid; pressure not increased; cell count, 4 per c.mm.; globulin, very slight haziness (normal); sugar, +.

September 13. Blood count: leukocytes, 11,800; differential count of 200 cells: polymorphonuclears, 78.5 per cent.; lymphocytes, 14.5 per cent.; large mononuclears, 4.5 per cent.; transitionals, 3.5 per cent.; eosinophiles, 0.5 per cent.; basophiles, 0; stimulation form, 0.5 per cent.

September 15. While the patient is better in every way, she still gives the impression of being a little apathetic. Her appetite is poor. Besides the unsteadiness that one would expect on first getting up, her manner of walking gave distinctly the impression of spasticity. This was quite in accord with the generally increased tone of the legs throughout her illness and with the exaggerated knee jerks. This morning the knee jerks are very much exaggerated.

September 17. The patient has been up in a chair for the past two days and was walking yesterday. She walks much more steadily. On physical examination nothing abnormal can be found but the still somewhat exaggerated knee jerks. The neck has no trace of stiffness and the "signe de la nuque" is gone. She feels well and is discharged.

CASE 34.

Clara T., age, 8 months. New York City. Admitted, August 22, 1911. Discharged, August 24, 1911. Result, not improved. *Diagnosis*.—Acute poliomyelitis.

Family History.—Mother and father are living and well. There is one other child in the family, a twin sister of the patient, who now has poliomyelitis in this hospital.

Past History.—The child has had no illness previous to the onset of the present trouble.

Present Illness.—The child was perfectly well until yesterday evening, August 21. At this time the mother noticed that she was rather irritable and objected to being handled. She vomited several times, and had a slight cough. Today she has again vomited several times and at four o'clock this afternoon her temperature was 102° F. When the child was examined in the receiving ward, handling of the right leg caused loud screaming.

Physical Examination.—The patient is a well nourished baby with good color, bright alert expression, and does not look sick. She lies quietly, and is sometimes playful. During examination she regurgitated suddenly and without effort. Occasionally she coughs. *Eyes*.—Pupils are equal and react normally. Ocular motions are normal, no squints. *Ears*.—Normal. *Nose*.—Normal. *Mouth*.—Buccal mucous membrane is clear. Tonsils are a little enlarged, pharynx slightly reddened. *Neck*.—Distinct rigidity. *Superficial lymph nodes*.—The posterior cervical and left axillary nodes are palpable. *Thorax*.—Well formed, respiratory motions normal. *Heart*.—Normal. *Lungs*.—Normal. *Abdomen*.—Well rounded and prominent, soft, moves properly with respiration. The spleen is easily felt. *Extremities*.—Upper: there is possibly a shade less strength in the right shoulder than in the left, but this is very indefinite. At times there is a distinct stiff shaking of the arms. Lower: knee jerks absent on the right, very faint reaction on the left; Achilles reflex absent on the right, present on the left; Babinski test negative on both sides; Kernig's sign: on the right side no resistance but some distress, on the left slight resistance. The left leg is moved about in a very active manner. The right, however, is not moved nearly so much, practically not at all spontaneously. When prodded, there is some move-

ment to withdraw the extremity, but this is chiefly in the lower leg muscles, and very little or not at all in the thigh. *Surface*.—There is a faint erythema over the upper part of the chest anteriorly. Over the inner end of the right eyebrow is a small superficial moist ulceration, sharply circumscribed and looking much like a collapsed water blister. *Temperature*.—102.6° F.

August 22. Spinal fluid: 30 c.c. of slightly greyish opalescent fluid; pressure at first much increased (child crying); later, slightly increased; cells, 990 per c.mm.; 95 per cent. polymorphonuclears. Smear: polymorphonuclears, 85.5 per cent.; transitionals, 4 per cent.; mononuclears, 10.5 per cent. (including probably both endothelial and lymphocytes, these cells being destroyed beyond recognition); globulin, +; sugar, + +. Blood count: leukocytes, 35,600; differential count of 200 cells: polymorphonuclears, 32 per cent.; lymphocytes, 55.5 per cent.; large mononuclears, 1 per cent.; transitionals, 9 per cent.; basophiles, 0; eosinophiles, 1 per cent.; stimulation form, 1.5 per cent.

August 23. Spinal fluid: about 7 c.c.. The first cubic centimeter was clear and slightly opalescent. This was used for cell count and smear. The smear contained a few red blood corpuscles, with great excess of mononuclears. Cell count, 627 per c.mm.; polymorphonuclears, 20 per cent.; mononuclears, 80 per cent.; no test for globulin, nor for sugar. On admission the child was irritable and cried a great deal. The knee jerks and Achilles jerks on each side were exaggerated. Both lower extremities were strong and active. This evening the left knee jerk is gone. The left Achilles jerk is very faintly present. The child cannot move the left thigh now, so that both lower extremities are affected. The flexors and extensors of the feet are still acting, but are weak. The child is rather more fretful this evening.

August 24. This morning there is no further advance of the paralysis, but both lower extremities are flaccid, save for some slight motion in the flexors and extensors of the feet and toes. The Achilles jerk on the left is gone. The arms are active and strong. The temperature has risen again to 102.8° F. The child looks bright, but is perhaps too alert and nervous. She is still inclined to be irritable if handled. If a sudden noise occurs, she blinks rapidly for several seconds and starts. Blood count: leukocytes, 23,200; differential count of 200 cells: polymorphonuclears, 49 per cent.; lymphocytes, 33.5 per cent.; large mononuclears, 5 per cent.; transitionals, 9.5 per cent.; basophiles, 0.5 per cent.; eosinophiles, 0.5 per cent.; stimulation form, 2 per cent.

This afternoon the child seems to be in about the same condition. It is possible that the patient is slightly more somnolent and does not show the nervous activity of the upper extremities that she did yesterday. Spinal fluid: a few cubic centimeters of slightly turbid, opalescent fluid were obtained; cell count, 1,221 per c.mm. In the wet count there are many very large phagocytic mononuclear cells with vacuoles; mononuclears, 92 per cent.; globulin, + (moderate flocculent precipitate); no test made for sugar. The father insisted on taking the child home this afternoon.

EXPLANATION OF PLATES.

PLATE 1.

FIG. 1. Interstitial meningitis in the anterior fissure of the cord, with beginning perivascular infiltration of the gray matter (human).

FIG. 2. Infiltration with small round cells in the perivascular lymph spaces (monkey).

PLATE 2.

FIG. 3. Diffuse areas of hemorrhage in the gray matter of the cord (human).

PLATE 3.

FIG. 4. Neurophages in nerve cells of the anterior horn (monkey).

FIG. 5. Extensive infiltration of the gray matter of the cord (human).

PLATE 4.

FIG. 6. The inflammatory process is seen extending throughout the whole anterior horn (monkey). Low power.

FIG. 7. Drawing of the lumbar cord of a monkey showing extensive interstitial meningitis and beginning perivascular infiltration.

PLATE 5.

FIG. 8. Perivascular and diffuse round cell infiltration in the medulla (monkey).

PLATE 6.

FIG. 9. Cervical ganglion, showing advanced inflammatory reaction.

PLATE 7.

FIG. 10. A spinal ganglion showing diffuse inflammatory process and beginning necrosis of the nerve cells (human).

PLATE 8.

FIG. 11. Neurophages in a spinal ganglion (monkey).

FIG. 12. Human lymph node. The pale centres of the nodules are formed by large endothelial cells. Low power.

PLATE 9.

FIG. 13. Human liver. Drawing showing necrosis of the liver cells and accumulation of lymphoid and polynuclear cells. High power.

FIG. 14. Electrocardiogram of the arrhythmia regularly seen in the fatal cases.

PLATE 10.

FIG. 15. Photograph of a case with profound stupor, showing irritated expression. The paralyzed left arm has been flung over by the shrugging of the shoulder. The retraction of the neck is also apparent.

FIG. 16. A case of intercostal paralysis, showing protrusion of the abdomen by contraction of the diaphragm and concomitant retraction of the thorax.

FIG. 17. Front view of patient shown in figure 2 with intercostal paralysis.

PLATE II.

FIG. 18. Paralysis of the lateral abdominal muscles. Bulging is produced by crying.

FIG. 19. Paralysis of the lateral abdominal muscles and right rectus. Bulging is caused by the attempt to sit up.

PLATE 12.

FIG. 20. The only evidence of paralysis is in the upper distribution of the seventh nerve. This was a profoundly stuporous case.

PLATE 13.

FIG. 21. Residual facial palsy in a case with profound stupor.

FIG. 22. The same as figure 21.

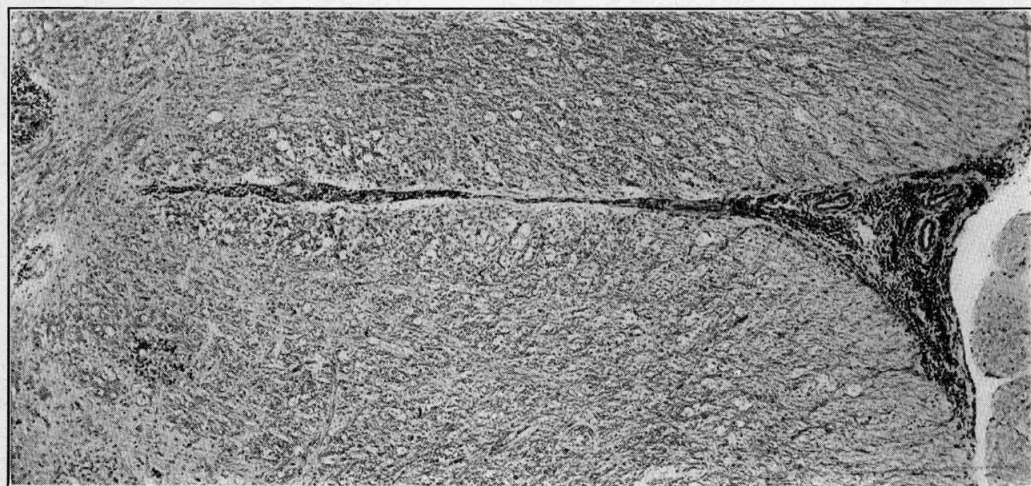


FIG. 1.

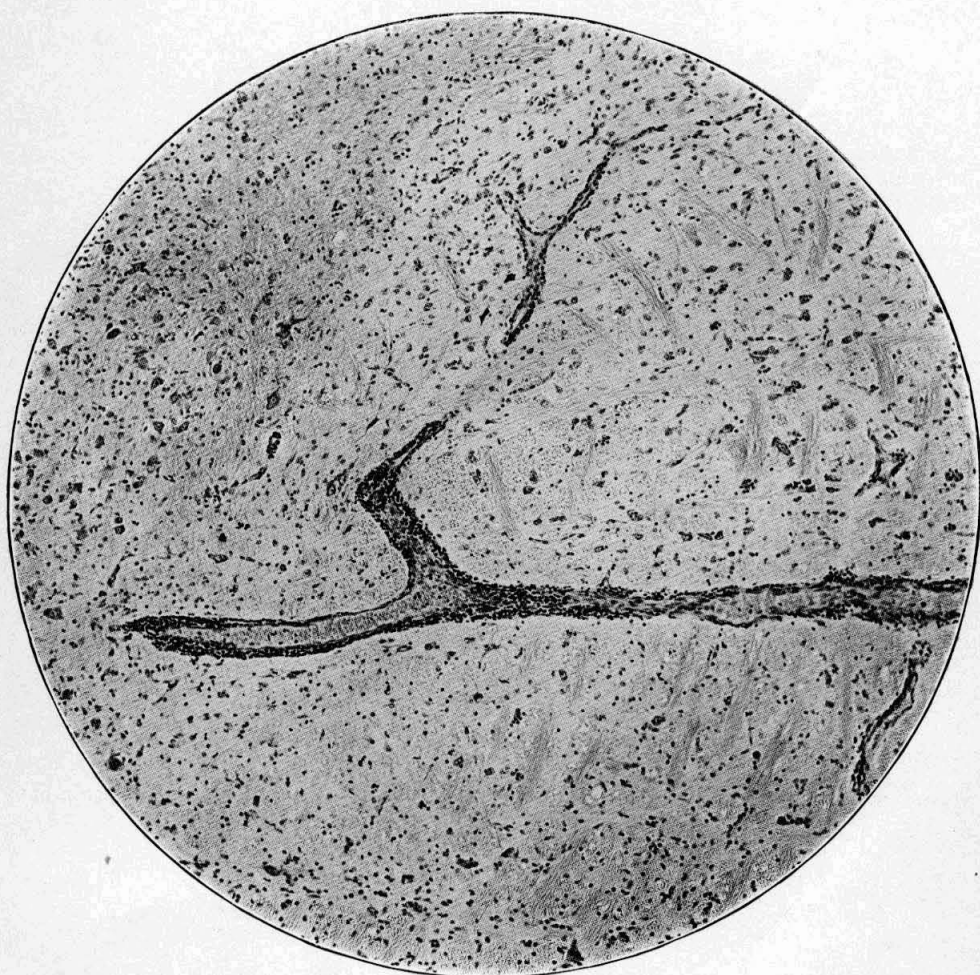


FIG. 2.

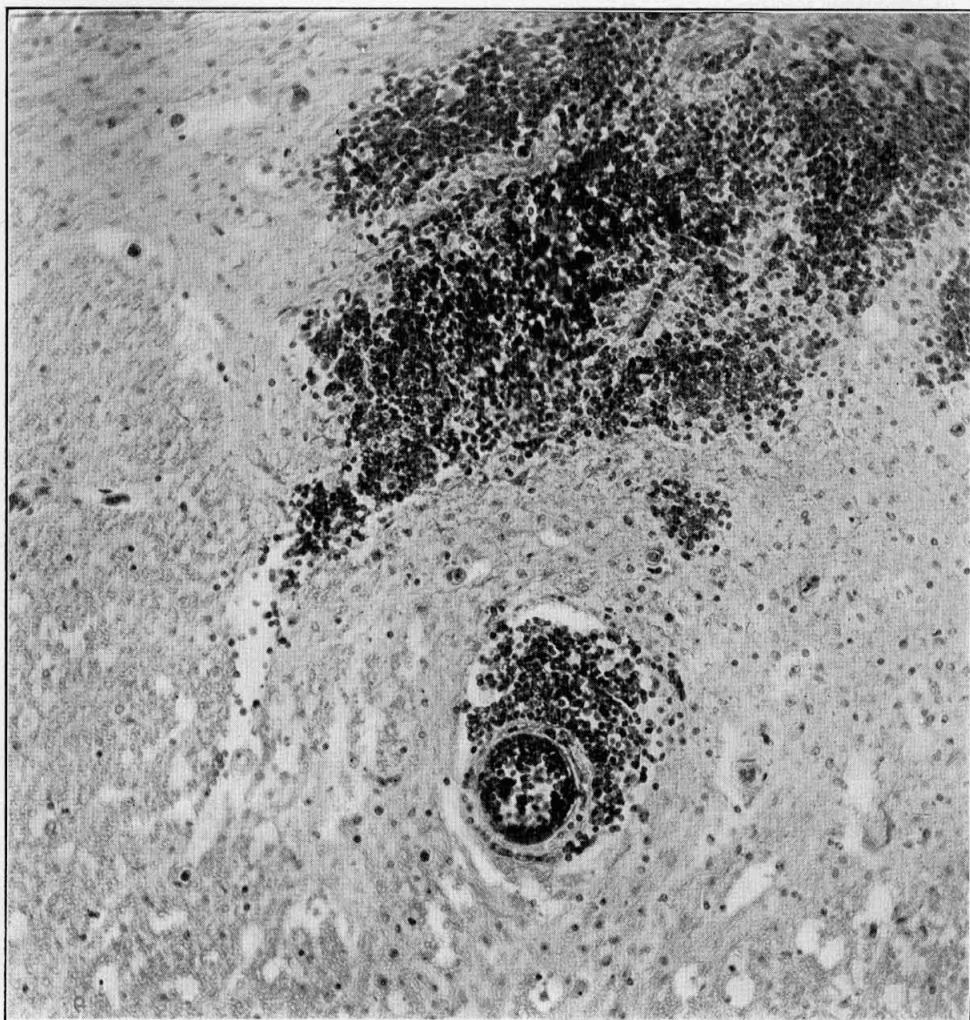


FIG. 3.



FIG. 6.

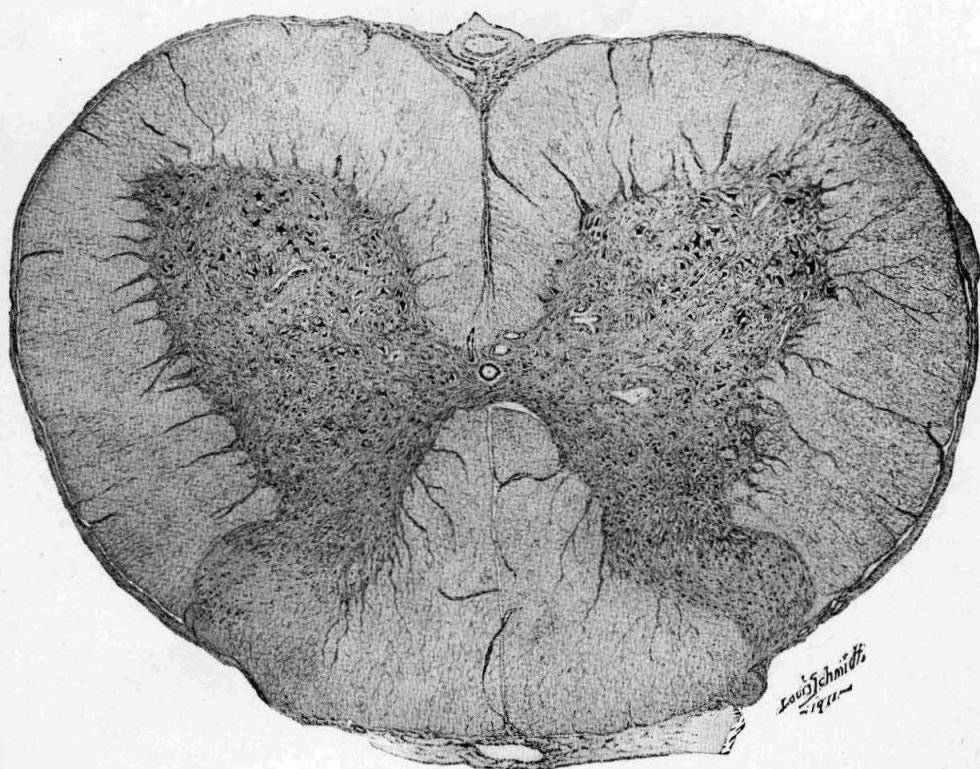


FIG. 7.



FIG. 8.



FIG. 9.

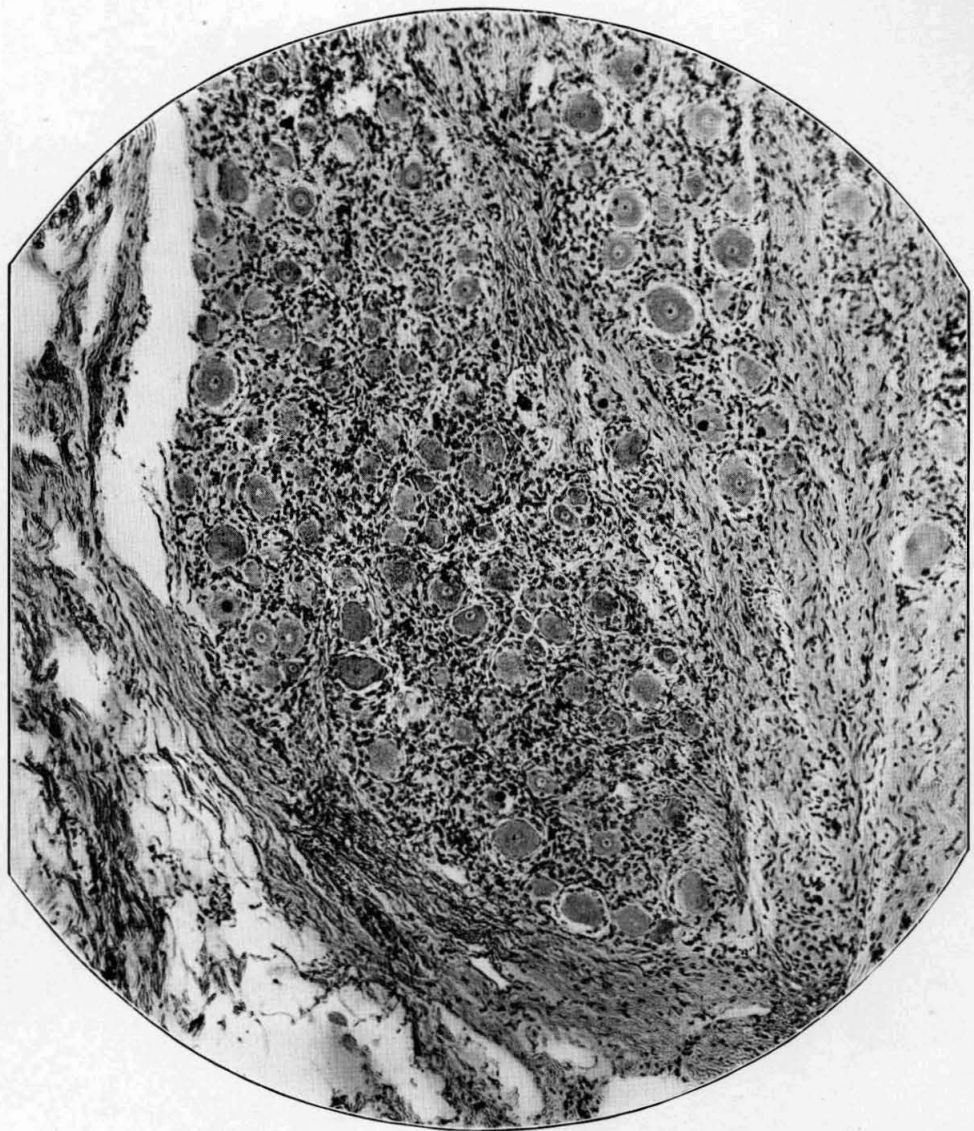


FIG. 10.

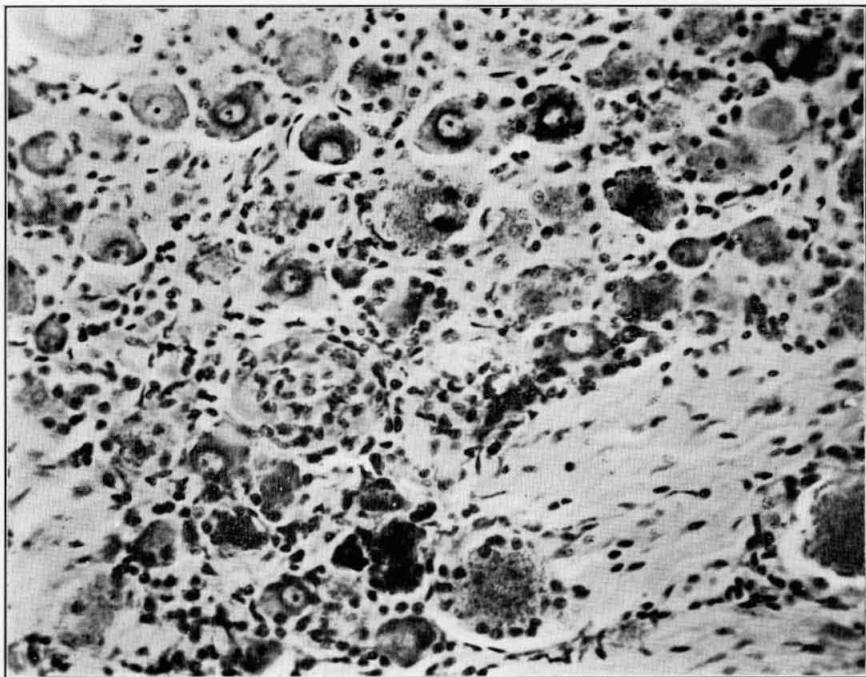


FIG. 11.

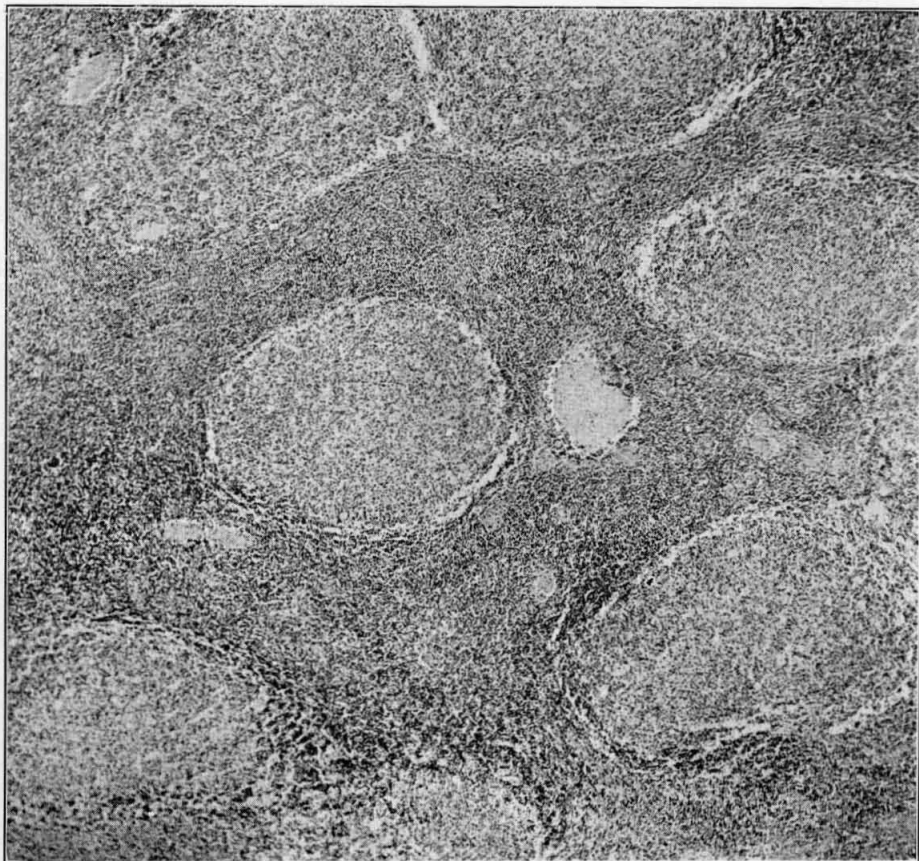


FIG. 12.

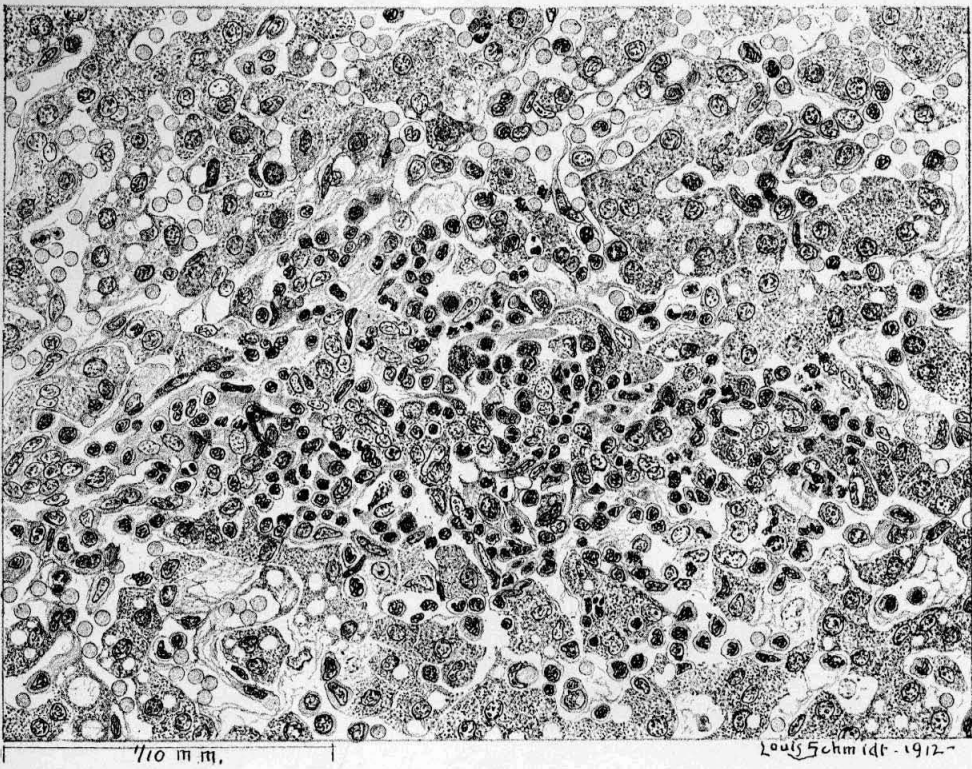


FIG. 13.

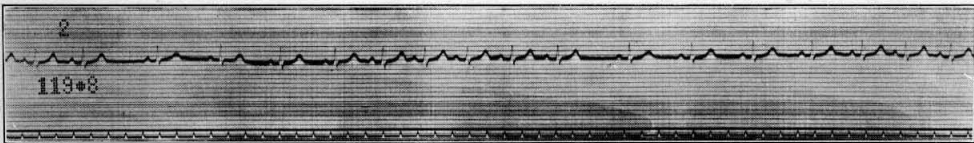


FIG. 14.

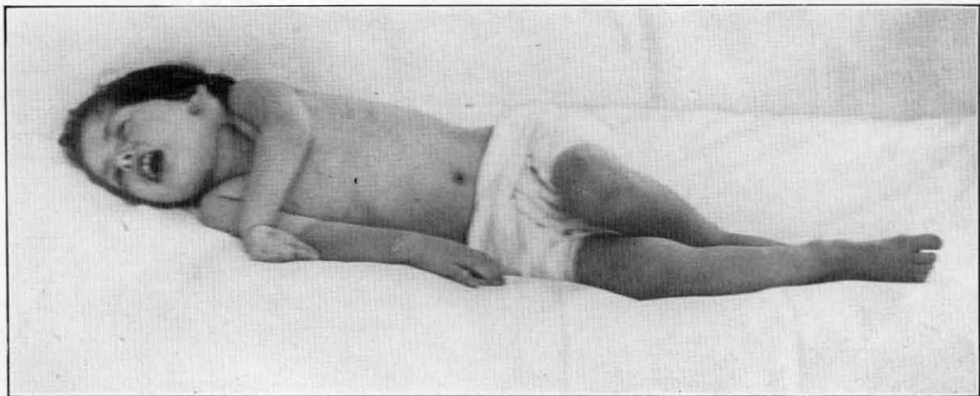


FIG. 15.

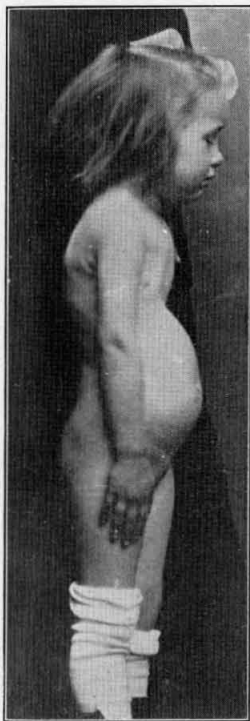


FIG. 16.



FIG. 17.



FIG. 19.



FIG. 18.



FIG. 20.



FIG. 21.



FIG. 22.