evidences of immunizing response as obtained by study of the agglutinating, baetericidal, and bacteriolytic power of the blood serum.

The vaccine should be prepared as follows: Pure cultures of the organisms are grown on agar for twenty-four hours, when the growth is washed off with sterile salt solution. The resulting suspension is thoroughly shaken to break up the clumps and a small quantity drawn into an ordinary red blood pipette, diluting 1 to 200, with salt solution for purpose of standardization. A small drop of this dilution is then placed upon the *blood-platelet* counter of Zeiss. The *blood-platelet* counter differs from the ordinary red blood cell counter in that with the former, the depth of the chamber is only $\frac{1}{8}$ mm. Twenty-five small fields are counted under the microscope, using dim direct illumination or indirect illumination by the dark-field condensor, and the sum divided by twenty-five to obtain the unit value. The unit value is then multiplied by the dilution, by the depth of the chamber, by 400, the number of small squares in a millimeter, and, lastly, by 1000 to convert to cubic centimeters. For example, if fifty baeterial cells are counted in twenty-five small squares, the unit value per square would be two, multiplied by 200, by 50, by 400, and by 1000, or 8,000,000,000, the number of baeteria per c.c. of suspension. The suspension is then heated to 54° to 55° C. for one hour in a sealed test-tube, in a water bath and then diluted with sterile salt solution, containing 0.35 per cent. phenol, so that each cubic centimeter of the finished vaccine contains approximately 1,000,000,000. Care must be exercised not to overheat the vaccine, since overheating impairs its immunizing properties. The thermal death point of most strains of typhoid bacilli varies between 53° and 56° C. Cultures should be taken from the vaccine to insure sterility before using.

CONCLUSIONS. 1. Not only should individuals who are typhoid carriers receive appropriate vaccine treatment, but all individuals including physicians and nurses, whose duties require the attendance and care of typhoid cases should receive immunizing treatment. By this procedure many individuals otherwise destined to become chronic carriers might secure protection which would prevent in themselves the possibility of cholecystitis or other late manifestations, as well as avoid the possibility of becoming a source of danger to others.

2. Such treatment has been found efficient and productive of little discomfort. Nurses, orderlies, and physicians should be regularly immunized at intervals of two or three years, as should all others exposed to endemic or epidemic outbreaks.

3. When typhoid bacilli have produced clinical or bacteriological evidences of cholecystitis, it is doubtful whether vaccine treatment will of itself be of avail, surgical drainage of the viscus then being indicated. On the other hand, appropriate vaccine treatment in

individuals showing evidences of intestinal or urinary tract perpetuation of the bacilli, without cholecystitis, during convalescence from the disease or subsequently, should be given the opportunity of vaccine treatment. Not all will be cured but the bacilli will disappear in some instances.

4. Chronic carriers who have been unsuccessfully treated by vaccines should be under the control of the local board of health and furnished employment if necessary which would not require the handling of food products.

EPIDEMIC POLIOMYELITIS: A CLINICAL STUDY OF THE ACUTE STAGE.¹

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It is an interesting fact, that although our knowledge of acute poliomyelitis dates back over seventy years, when the German surgeon, Jakob Heine² first described it under the name of "the spinal paralysis of children;" the true nature of the disease, its pathology, clinical manifestations, prognosis, infectiousness, contagiousness, have only lately been fully ascertained, and have, only in the last decade, become the subject of general knowledge. Furthermore, it is of interest to note that at the same time that Heine first made his observations in Germany, an American practitioner, Dr. Colmer, observed a small epidemic in Louisiana, and recorded it in the AMER. JOUR. MED. SCI. (1843, v, 248). Little or nothing was added to the knowledge of the subject for the next thirty years. No epidemics were reported until 1881, when 18 cases occurred in Sweden. Subsequently, epidemics were reported from Norway, Sweden, Germany, the United States, and Canada, in the eighties and nineties. The most extended epidemics occurred in the above-named countries in the last five or six years.

A noteworthy contribution was made by Strümpell³ in 1885, in his paper on Polienccephalitis Acuta in Children. Pathologically and clinically this disease was noted as identical with the well-known forms of spinal poliomyelitis. For the first comprehensive clinical studies we are indebted to the Danish physicians Medin⁴ and Wickman.⁵ Aside from his contributions to the pathology, Wickman was able to show conclusively, that the disease is contagious.

¹ Read before the Metropolitan Medical Society, Academy of Medicine, October 24, 1911.

² Die spinale Kinderlähmung, 1810 and 1860: a study of 150 cases.

³ Jahrb. für Kinderheilk., 1885, Band xxi, N. F.

⁴ X. Internationaler Congress, Berlin, 1890.

⁵ Beiträge zur Kenntnis der Heine-Medinischen Krankheit, Berlin, 1907.

Pathologically speaking, the disease has been characterized as a meningo-encephalo-polio-myelitis. Of late, it has been shown, however, that the lesions are not confined to the gray substance of the brain and cord, but may be found in the contiguous white substance, as well as in parts which have no direct communication with the gray matter.

Recently the study of poliomyelitis has entered into a new phase through its successful inoculation into monkeys.

In taking up this subject of poliomyelitis I wish to dwell mainly on its diagnostic features. If this be an infectious and contagious disease, as now seems certain, our duty as practitioners clearly lies not only with the patient, but also with the family and the community. Aside from the fact that the recognition of its inception is of great importance on account of the dubious prognosis as to life and limb, it is clearly our duty to diagnosticate it as early as possible, in order to prevent its transmission to others.

After a careful study of the manifold clinical manifestations lends up to a diagnosis of the case, the question of prognosis is the all important consideration. Having this in view, I would therefore, propose the following classification, which, empirical though it may seem, is comprehensive, and tends toward a practical purpose. Poliomyelitis can be divided into: (1) The abortive cases, mild or severe in their onset or course, which end in complete restitution in a short time; (2) cases with paralyzes, these may end in complete restitution or in partial or complete atrophy; (3) the rapidly fatal cases (*formes frustes*). This classification has some value, because it covers the whole ground and lays equal stress on the mild, transient forms, as well as on the paralytic and the fatal cases, putting the former on a par with the well-known paralytic forms.

By this classification I mean to lay stress on the fact that a large number of abortive cases pass unrecognized at the present time, while, judging from our experience of other infectious diseases, these same abortive cases spread the epidemic. Observations made during epidemics of poliomyelitis seem to show that the group of cases occurring not infrequently in infants and young children, hitherto styled meningitis serosa, as well as certain unclassified spastic or tetanic affections of childhood may well belong to the abortive type of poliomyelitis. This also holds good for certain unexplained fatal cases in children and adults. How large their number is, however, cannot at present, be estimated.

What means are at our disposal at the present time to recognize with any degree of certainty a case of acute poliomyelitis? What are the characteristic symptoms of the disease; what additional diagnostic aid does the laboratory offer toward making a positive diagnosis?

In studying the literature of the extended epidemics of Sweden, Norway, and Germany, it becomes at once apparent that the recognition of cases, even though they may be atypical in their clinical manifestations, seems comparatively easy. The existence of a widespread epidemic has been officially noted, the authorities are on the alert for every case, the practitioner is instructed to watch for and report all suspicious cases. An organized medical campaign is thus established in the country districts, the hamlets, villages, and small towns, and the progress of the epidemic can be followed from house to house.

In a city like New York, for example, conditions are totally different. It is true that we are forewarned that the epidemic generally appears in the summer months, and, furthermore, we know that the disease has become endemic with us. Thus, sporadic cases make their appearance at any time, and must be recognized without the aid of epidemiological data. If the cases are brought in from the country, it is sometimes possible to determine the existence of an epidemic in that outlying district. If the case is not an extraneous one, a clue as to its origin and transmission, in the city proper, can usually not be obtained, thus the etiological factor does not become apparent, and the possibility that one is dealing with a case of poliomyelitis is not even thought of or considered. Nothing would be of greater aid to the practitioners of large cities than the daily publication in the public press, by the Board of Health, State or National Health Boards of the daily number of cases of infectious and contagious diseases. In this way the family doctor would be apprised of the existence, the increase and decrease of the various epidemics, and exact information on these points would materially aid him in the diagnosis of suspicious cases.

Since Medin's time, clinicians are wont to look for the predominance of certain irritative and paralytic symptoms of the nervous system, to aid them in recognizing the disease in its various forms. Aside from the abortive cases, which may present themselves under the most varied guise, the principal types of poliomyelitis are: The spinal, meningeal, cerebral, encephalitic, bulbar, and pontine, Landry's ascending and descending paralytic, the polyneuritic, and the ataxic types. It goes without saying, that these various types may overlap or combine. There is no room for doubt, that the systematic subdivision into the above-named groups, indicating as they do, the seat of the lesion, lend material aid in the diagnosis of poliomyelitis in its paralytic stage. In the acute stage, however, they only serve the purpose of indicating the existence of some infectious process in the cerebrospinal system, the nature of which must be especially determined. In other words, our method of diagnosis, at the present time, is a purely empirical one, and will remain so, until the diagnosis can be made from the secretions or excretions, from the blood or spinal fluid, bacterio-

logically, serologically, or by other exact biological methods. Our method of diagnosis, then, being purely empirical it is just as important to note certain other symptoms, which, though they may vary in different epidemics, must be carefully considered, as they are present at the very inception of the attack and may cover up or disguise the true nature of the underlying affection. A poliomyelitis may begin with or run its course under the form of an angina, tonsillitis, influenza, a gastro-enteritis, a rheumatic fever. In some epidemics most of the cases were accompanied by the various respiratory and intestinal symptoms of acute influenza. Indeed, a Swedish author² has attempted to show that poliomyelitis is only a form of influenza infection, although he has failed to demonstrate the constant presence of the bacillus of Pfeiffer in his cases. Angina and tonsillitis are often seen in the prodromal stage, or are the concomitant symptoms of the early acute stage. Again, the gastro-intestinal tract seems to be the principal seat of the infection in some epidemics, and the disease runs its whole course under the mask of a diarrhea accompanied with fever. The description of such a case is given below. When these cases occur in an abortive form, it is doubtful whether they can be recognized at all, especially in infants and young children, unless a careful search is made for symptoms of irritation of the brain and cord.

THE ABORTIVE CASES, THEIR DIAGNOSIS AND SIGNIFICANCE. Our knowledge of the abortive cases of poliomyelitis only date back to the observations of Wickman¹ made in the great Swedish epidemics of 1905 and 1906. He holds that they are frequent, probably exceeding in number the paralytic cases. As they have hitherto only been recognized when they occur in connection with typical cases, it follows that the number of abortive cases is much greater than is ordinarily supposed. Their epidemiological importance cannot be overestimated. Wickman cites a large series of family infections, in which abortive cases occurred in connection with paralytic cases of varying intensity, together with fatal cases. Some of the children after a short feverish attack were well in a few days or a week, and returned to school. Accordingly, the abortive cases are the principal carriers of the infection.

In my service at the German Hospital, in September, 1910, an abortive case of poliomyelitis of the gastro-enteritis type came under my notice, which was not recognized during its course, but was subsequently proved to belong to this group, through the kind interest of Dr. Simon Flexner. The case was published in the *Medical Record*, of July, 1911. As it is very instructive, I will mention it here, somewhat in detail:

¹ Th. Hronström, *Akute Kinderlähmung und Influenza*, Leipzig, 1910.

² Die akute Poliomyelitis, Berlin, 1911.

CASE I.—J. W., aged six years, entered the Hospital September 4, 1910, discharged cured, September 21, 1910. Diagnosis, acute dysentery, meningism. His father was living. His mother died of consumption. He had measles in February, 1910. For the several weeks before admission he had been restless at night. On September 3, at noon he was seized with a chill, then complained of headache, which gradually became very severe. He became drowsy, then unconscious, and had vomiting and diarrhea.

CHART I.—Case I. Abortive poliomyelitis.

Examination. The patient was somnolent but responded if aroused. There was rigidity of the neck and tenderness of spine. The eyes, ears, nose, were negative except for myosis and tardy reaction of the pupils to light. The abdomen was negative, the spleen was not enlarged. There was a slight Kernig and Babinski of both lower extremities, other reflexes were normal. The highest temperature was 103°.

The case ran the course of an acute dysentery; the stools contained mucus and blood. The urine was negative. Gradually the nervous symptoms became more marked; opisthotonos, delirium, and extreme restlessness developed. A clear fluid escaped under moderate pressure from a lumbar puncture. The number of stools diminish, the temperature and pulse became normal in the course of a week. The symptoms of meningism subsided. The reflexes were never absent at any time.

Although meningism occurs as a symptom of various infectious diseases, especially in children, our original interpretation

of the case did not seem wholly satisfactory, especially after hearing of several cases of poliomyelitis of the gastro-enteritic type which had occurred in the course of the summer. On looking up the patient at his home, a careful examination of his reflexes showed no anomalies. There were no paralyses of any kind. Two ounces of blood were taken from the arms of the patient and subjected to the neutralization test.

This test is made by mixing for many hours, the active virus derived from apes with the serum of an immunized human being, and injecting it intracerebrally in apes. Controls receive in the same manner, virus mixed with the serum from an individual who never had anterior poliomyelitis. The controls contract the disease, while the test cases escape, presumably because of the antihodies present in the immunizing serum. The test in our case showed a positive result. As there was nothing in the patient's previous history pointing to an attack of poliomyelitis, we had doubtless been dealing with a case of the abortive type of this disease.

CHART 2.—Case II. Abortive poliomyelitis (meningism).

CASE II.—Since then another case came under my notice and partial observation which allows of the same interpretation. A family with a number of children, travelled through Norway, Sweden, Germany, and France. While in Norway, one of the children had a mild attack of tonsillitis. He subsequently complained of being very tired. Two weeks after leaving Norway, the child complained of headache, which gradually became so

intense that he could not bear his head to be touched, and had to lie down. On the second day, the temperature gradually rose to 103°, pulse, 108. There were no gastro-intestinal symptoms nor cough. The patient gave the impression of being very ill, was extremely irritable, and when he awoke from a restless sleep, complained of severe headache. The pupils were small, but equal, and reacted to light slowly. The patellar reflexes could hardly be elicited, and no Babinski or Kernig phenomena were present. The cremaster and abdominal reflexes were present and equal. There was stiffness of the neck and inability to put the chin on the chest. A tentative diagnosis of cerebrospinal meningitis was made and a doubtful prognosis was given. In the course of the fourth and fifth day, however, the meningitic symptoms gradually disappeared, and the child made an uneventful recovery. Six weeks later, the left patellar reflex was present, the right one absent. There seemed to be a slight paretic condition in the distribution of the right facial nerve. Examinations in the seventh, eighth, and ninth weeks after the attack showed that the patellar reflex of the right leg was gradually becoming normal. This was undoubtedly a case of abortive poliomyelitis.

CASE III.—A child, aged three and one-half years, was brought to the German Hospital, August 30, 1911, with a history of fever and diarrhea for a number of days. The mother said the child was unable to stand or walk alone.

Examination. With the exception of a slight rise of temperature, and a number of diarrheic movements, the child, as it lay in bed, did not present anything worthy of note. On placing the child on the floor, however, it could not rise, the feet seemed flabby, the big toes bent under the feet. In a sitting or standing posture, when held by the arm, the trunk reclined to the left side. The pupils and face were normal. The right patellar reflex was diminished, the left normal. Abdominal and cremaster reflexes were present and equal. The electrical reaction of the affected muscles of trunk and extremities were normal. In two weeks after treatment by urotropin, baths, and electricity, the child was able to walk. At the present time there is a distinct limp of the right leg and the patellar reflex on the right side is diminished.

Cases like Case III are not uncommon I presume. On account of the shortness of the febrile stage, they first come under observation when paralysis has set in, and pass on to partial or complete restitution or complete paralysis, according to the severity of the lesions in the anterior horns and surrounding nerve tissue. Frequently, the orthopedist is the one who first sees these cases, as occurred in the following case.

CASE IV.—A perfectly healthy but very neurotic girl, aged eight years, complained of headache and dizziness, but insisted on going to school. During the night the child's temperature

rose to 103°; she complained of headache, pain, and heaviness in the lower extremities. On the following morning the mother was thrown into a frenzy, because the child could not sit up or move her legs and had slight difficulty in passing urine. Both patellar reflexes were absent. The temperature was normal on the fourth day, and remained so. Examination showed that the peroneal group of muscles of both limbs were affected, also the posterior tibial muscles, more on the right than on the left side. This case was treated by Dr. J. Teschner with exercises and reëducation. The child never made use of a crutch, nor was a splint applied. She now walks with the characteristic swing and drag of infantile paralysis.

CASE V.—This was a healthy, well-developed boy, aged sixteen years, seen in consultation last summer. He had returned from the Adirondacks, with the history of remittent temperatures for three days. The patient complained of heaviness in the lower extremities and pain in the back. An incubation history could not be obtained. Patellar, cremaster, abdominal reflexes were present, equal and slightly exaggerated. There was a distinct Kernig's sign in both lower limbs. The pupillary reaction was normal to light and accommodation. There was slight stiffness of the neck. The spleen was not palpable. There was no bradycardia, the temperature was about 103°. Leukopenia and relative lymphocytosis were present. The Widal reaction was negative. The possibility of typhoid fever or cerebrospinal meningitis was considered. A spinal puncture, however, gave a clear fluid, containing 90 per cent. of lymphocytes. Subsequently, partial flaccid paralysis of the extremities followed. This was a typical case of the spinal form of poliomyelitis.

CASE VI.—S. F., a girl, aged about five years, whose mother was tuberculous, was suddenly seized with tonic and clonic convulsions. The patient moved both arms and legs convulsively or held them in tonic flexion; at times the arms and legs were relaxed; then again the legs were held in hyperextension. This was accompanied by twitching of the face, especially on the right side and rolling of the eyes. Severe opisthotonus occurred, which persisted for a number of days. All the reflexes were exaggerated. The pupils were unequal, Kernig's sign was present in both lower extremities, and there was a tache-cerebrale. Her temperature was steadily high, between 104° and 105°. After these irritative symptoms, the patient gradually lapsed into a comatose condition in which she remained for two or three days. During this period, stiffness of the neck and back persisted so that it was impossible to roll the child on her side, as she seemed to resist enforced motion. Spinal puncture was repeatedly made, a clear fluid showing small and large mononuclears escaped under pressure. There was occasional rolling of the bands inward, general tremor, and nystagmus.

The facials seemed at times unequal. The nystagmus gradually ceased, but there remained internal strabismus on the left side. The diagnosis of tubercular meningitis was made, but the tubercle bacillus could not be found in the spinal fluid. Subsequently the child had great difficulty in standing, and the patellar reflexes were much diminished and unequal. Gradually recovery set in. The patient tottered when walking and fell frequently when not guided by the band. The strabismus persisted for a number of weeks, but gradually disappeared. This was a case of poliomyelitis of the cerebral type.

This case was one of three children belonging to a family that lived in close quarters. During this severe illness of one child the two other children had short accesses of fever, to which not much attention was paid. Both children seemed much prostrated after a few days' illness. It does not seem improbable to me, at the present time, that they too had the short febrile abortive type of poliomyelitis. This may also have been the case with the brother of Case II.

CHART 3.—CASE VII. Bulbar and pontine poliomyelitis.

CASE VII.—O. E., male, aged twenty-nine years, entered the German Hospital September 8, 1909. Died September 12. The duration of the whole illness was eleven days. A positive diagnosis was not made, though the existence of an acute poliomyelitis was deemed probable. There was a history of tuberculosis in the family. On September 1 the patient had a slight chill; dull frontal headache and stiffness of neck. He was unable to move the arms at the shoulder-joint; had difficulty in dressing and could hardly

feed himself. He was, however, able to move the elbow and wrist-joints, but muscular action of the arms was weak. No pains.

Examination. The patient was bathed in profuse perspiration, lay helplessly in bed; he was unable to sit up, as he could not support himself on his arms or hands. The eye muscles were normal and the pupils reacted normally to light and accommodation. There was stiffness of the neck, inability to move the head from side to side, or flex it on the chest, and moderate rigidity of the spine. No hyperesthesias. There was complete loss of motion at both shoulder-joints, yet extension of the forearms was accomplished promptly. The hands and fingers were readily moved. Considerable muscle weakness existed. There were no paresthesias or hyperesthesias, no special points of tenderness along the nerve trunks. The knee-jerks were not obtainable; Kernig, Babinski, and Romberg signs were absent, and there was no loss of function or weakness of the leg muscles. No disturbance in sensation or electrical reaction of either upper or lower extremities occurred. Lumbar puncture was dry. The patient acted peculiarly, screamed during sleep, talked irrationally, wanted to get out of bed. On September 11 he was slightly delirious, and had some difficulty with speech and swallowing. The patient died suddenly during the night. The autopsy showed hemorrhages in the upper part of cord and pons. This was a case of the bulbar and pontine poliomyelitis of Oppenheim and Erh.

CASE VIII.—Female child, aged two and one-half years. Bottle-fed, well nourished. In September, 1908, she had an attack of enteritis with fever. The child seemed very restless and nervous; complained of headache, and was suddenly seized with general convulsions which lasted for two days and nights. Chloroform was constantly administered. There was convulsive twitching of the face and tongue, with inability to swallow. The temperatures was up to 105°. The pupils were contracted. Bilateral Kernig's sign was present. The abdominal and patellar reflexes were not exaggerated, and there was no foot clonus. Trousseau's sign was present. Spinal puncture gave a perfectly clear fluid. At no time was there stiffness of the neck. The irritative symptoms persisted about a week, then the upper and lower extremities gradually relaxed and the pupils became dilated. The patellar reflexes disappeared. It was observed that the child did not take notice of objects placed in front of it, or held before its eyes; an examination of eye grounds by Dr. Koller showed hyperemia of the retinal vessels, but no optic neuritis. The child had a flaccid paralysis of the lower extremities, and was unable to stand. The hands were held in flexion. No reactions of degeneration were present. As there was an extended epidemic of cerebrospinal meningitis at the time, the question as to whether we were dealing with this infection was also favorably considered. In view of the

fact, that opisthotonus was never present, that the spinal fluid was clear, and that the attack was ushered in by a severe enteritis which ran parallel with the nervous symptoms, I am inclined to uphold my diagnosis of encephalitis, in which I was supported by several competent observers. I would group this case with the encephalitic type of poliomyelitis, in the light of our present knowledge of this disease.

Observers of the meningeal and cerebral forms of poliomyelitis like Wickman⁸ and others go into a very careful differential diagnosis between this type of poliomyelitis and cerebrospinal meningitis. In exhaustive treatises⁹ of epidemic cerebrospinal meningitis, however, the differential diagnosis of the two diseases is not even considered. From this I infer that the cerebral type of poliomyelitis has hitherto often passed unrecognized.

CHART 4.—Cerebral type of poliomyelitis.

CASE IX.—The following case is of interest in this connection. A native German, male, was brought to the hospital in a comatose condition on September, 1911. There was no history available excepting the fact, that he had come from the Catskills, where a few cases of infantile paralysis had occurred. Soon the patient was seized with tonic and clonic convulsions of the right side of the face, arm, and leg. Between the attacks the arms were held in fixed flexion. The right pupil was larger than the left. All

⁸ Loc. cit.

⁹ Goepfert Epidemic Cerebrospinal Meningitis (Genickstarre) Ergebnisse der Inneren Medizin und Kinderheilkunde, 1909, Band iv, p. 163.

the reflexes were exaggerated and were stronger on the right than on the left side. Foot clonus, Oppenheim and Babinski reflexes, were present on both sides. Abdominal and cremaster reflexes were intact. On stroking the plantar side of the right foot with the hammer a very strong contraction of the vastus of the right thigh was produced, which on repeated manipulation became tetanic. There was slight stiffness of the neck and complete aphasia. His temperature was up to 103°. Leukocytosis, 12,000.

The convulsions ceased, then started in again. They were controlled by the administration of bromides with the stomach tube. There was a strong probability of this being a case of poli-encephalitis, poliomyelitis of the encephalitic type, first described by Strümpell, with the identical symptoms, especially in consideration of the etiological factor. Careful subsequent examinations, however, showed a very distinct scar on the penis, the contours of the pupils were unequal (anechoria syphilitica), the Wassermann reaction was strongly positive. The spinal fluid showed an excess of lymphocytes, and the Nagouchi reaction was positive. After recovering from the coma, the patient gave a complete history of syphilitic infection. The patient is still at the Hospital, convalescent, and improving daily.

From the sketch of the histories given above, it becomes apparent that the diagnosis of this disease in the acute stage is, to say the least difficult, and under certain circumstances impossible. Some experienced observers state, that the four cardinal symptoms presented are, subjective pain, hyperesthesia, profuse perspiration, and leukopenia. Each one or all of these symptoms may be absent or present. Leukopenia, which is mentioned as a constant symptom is by no means always present. Moderate leukocytosis may be found due to the accompanying catarrhal affection of the respiratory tract or intestinal canal. As the early stages of measles and the great majority of the cases of typhoid fever also show leukopenia, the symptoms is only of value, if it occurs at all, in connection with the whole clinical picture. If the above-mentioned symptoms fail or are inconstant, how then is it possible to obtain the *first clue* to the existence of this disease and to differentiate it from other febrile conditions? I strongly suspect that this is impossible in certain elementary or transitory attacks of a mild character. In most cases, however, we will be rewarded by a most painstaking search for symptoms pointing to an irritation of the cerebrospinal system. It is unnecessary here, to go into a detailed analysis of the well-known signs of the presence and absence, exaggeration and diminution, and variability of the reflexes of the eyes and the muscular system which belongs to a complete neurological examination. The variations exhibited in the reflexes is due in great part to the location and to the irritative or destructive nature of the inflammatory process in the neurones. Recent

pathological study has furthermore shown, that it is not only the meninges, the anterior horns of the cord, the cortex of the brain, and the central nuclei which may be affected, but also the posterior horns, in fact, any part of the brain and cord may be implicated. The variability of the nervous symptoms is in itself if not characteristic, very suggestive. The taking of a complete nerve status at each visit will soon uncover the mutability of the reflexes, the motor and sensory disturbances. The first point to be looked to is, therefore, to discover, if possible, any sign of meningeal or cerebrospinal irritation. John Lovett Morse¹⁰ has recently corroborated the observations of Brudzinski, who first pointed out two distinct phenomena which are not present in healthy children, or in diseases other than those of the brain and cord. These are: (1) The contralateral reflexes of the legs, and (2) the neck sign. The contralateral reflex consists in the concomitant reflex of the leg on one side, when passive flexion of the leg on the other side is made. There are some variations of this sign. Brudzinski's neck sign is present in meningitis when passive flexion of the neck forward causes flexion of the legs at the hips and knees, and a marked flexion of the legs on the pelvis. Various writers have attached great importance to these signs in the diagnosis of meningitis. Further study must show, whether these reflexes are present in the meningeal forms of poliomyelitis. In a former paper¹¹ I dwelt upon the importance of spinal puncture in the diagnosis and treatment of various diseases in young children. At that time I hardly suspected the significance of the procedure in the diagnosis of the subject now under discussion; although it will take a far more extended experience than we now possess, and perhaps the devising of new methods of investigation to differentiate specifically the spinal fluid found in the various infections. In a careful analysis of over 500 specimens Lucas¹² tabulates the findings—macro- and microscopic of the spinal fluids of the various infections of the brain and cord in young children.

The cases under consideration may be divided into two large groups: (1) Cases with a predominance of polynuclears; (2) cases with a predominance of mononuclears or lymphocytes. The first group includes cerebrospinal meningitis as well as influenzal, pneumococcus, and streptococcus meningitis. In addition to this the characteristic organism is found in the spinal fluid in each instance. The second group, with predominance of lymphocytes, includes meningism, encephalitis, syphilis of the central nervous system, tubercular meningitis, and poliomyelitis. There is no reference to spinal puncture in tetany in the literature.

Let us attempt to differentiate the diseases belonging to the

¹⁰ *Archiv. Pediatrics*, 1910, xxvii, No. 8, 561.

¹¹ *AMER. JOUR. MED. SCI.*, 1910, cxxxix, 542.

¹² *Amer. Jour. Diseases of Children*, March, 1911, p. 230.

group with lymphocytosis in the spinal fluid. In syphilis of the central nervous system, which is comparatively rare in children, the spinal fluid may give the Wassermann reaction. It also gives the positive Noguchi butyric-acid test. In tuberculous meningitis the Noguchi reaction is negative; the tubercle bacillus is present in nearly 100 per cent. of the cases in the spinal fluid, as was shown by Hemenway. As far as encephalitis is concerned, a number of these cases may be of a parasyphilitic nature and give the Wassermann reaction, or they belong to the cerebral or encephalitic forms of poliomyelitis. It is not unlikely that some cases cannot at present be classified.

The subject of meningism¹³ presents serious difficulties and needs further study. The cases belonging to this group must be considered in connection with the underlying infection; the cytology and bacterial findings will vary according to the early or late stage of the disease.

As to poliomyelitis, Flexner and Lewis,¹⁴ Gay and Lucas,¹⁵ and others, have studied the spinal fluid in monkeys and in young children. The result of these studies can best be illustrated by the precise diagnosis of an obscure case from the microscopic examination of the spinal fluid. This was the case of Dr. Frissell: The patient was a young adult in whom all the reflexes were exaggerated, the Kernig sign and Babinski reflex negative, and the neck a trifle stiff. The Widal, and blood culture for the *Bacillus typhosus* were negative. Leukocytes, 12,000. Physical examination was negative. On the fourth day of the disease, the first lumbar puncture was made. Flexner reported that the fluid was turbid, the sediment consisted of a small number of large lymphocytes, a considerable number of small lymphocytes, and very few polynuclears, and that the clear supernatant fluid gave the Noguchi reaction. The condition of the fluid resembled that seen in inoculated monkeys, just before the onset of the paralysis. On the fifth day of the disease, the reflexes nearly disappeared and there were indications of paralysis of the lower limbs and abdomen. A second spinal puncture showed that the fluid was clear and still contained an excess of lymphocytes. The Noguchi reaction was not so strong. This second fluid resembled that of monkeys after paralysis had set in. The diagnosis and prognosis of this case were made by the most advanced clinical and laboratory investigations.

Nothing is more commendable in clinical medicine than to lay special emphasis upon the thorough observation of the case in hand in all its various clinical aspects. All our methods of observation, both at the bedside and in the laboratory, should be brought to bear on the determination of the given case, just as in criminal

¹³ Johns Hopkins Hosp. Rep., 1904, xii, 370, and Richard Stein, *Ibid.*

¹⁴ Archives of Pediatrics, 1910, xxvii, No. 2, p. 93.

¹⁵ Archiv. of Int. Med., 1910, cxxix, 542.

law the jury sifts the evidence of the witnesses and takes note of the impression made by the prisoner. The finger-print method of identification seems at present, however, destined to become the scientific test which will outweigh the sum of all the circumstantial evidence; so in our own field the special diagnosis of disease by laboratory methods is advancing in leaps and bounds. Bacteriological, serological, biochemical tests have opened a great vista in the recognition of disease by scientific methods. In acute poliomyelitis we are dealing with a disease, sudden in onset, short in its course, with symptoms obscure and elusive. Although the attempt to diagnose it by the serological method has so far been futile, let us hope that its recognition by a simple scientific laboratory test—not to speak of its prevention, cure, and extermination—will be effected in the not too distant future.

A CLINICAL CONTRIBUTION TO OUR KNOWLEDGE OF POLIOMYELITIS WITH CORTICAL INVOLVEMENT.¹

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It can hardly be supposed that the more intensive study of epidemics of poliomyelitis of recent years accounts for the bizarre clinical manifestations of this disorder which are now of frequent report. The situation would seem of more rational explanation on the ground that the isolated atypical forms of the disease were overlooked or disregarded in the past. The bulbar and pontine types are now common and well defined, but the cerebral and especially cortical involvements are still subjects of debate despite the classic and well-known contentions of Strümpell and his school on the subject.

The existence of a true encephalitic type of poliomyelitis is difficult to differentiate clinically as well as pathologically. In the first place, a spastic palsy is not definite proof of cortical or encephalitic involvement, and secondly, when the autopsy has shown cortical or meningeal alteration the findings have been susceptible of explanation upon other causative grounds. It is not sufficient to state that an encephalitic type must show a cortical lesion and present evidences of notable meningeal irritation, resulting in hemiplegia, which later becomes spastic, but the character, onset, and clinical course of such cases must be a matter

¹ Read before the New York Neurological Society, October 3, 1911.