Early diagnosis of acute anterior poliomyelitis

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-Senior Thesis-

The Early Diagnosis of Acute Anterior Poliomyelitis

Kenneth R. Drewelow
April, 1935.
-Outline-

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-Introduction-

In this short paper I will attempt to review briefly the facts of importance concerning the recognition of poliomyelitis in the preparalytic stages. A complete resume of the literature on the subject of poliomyelitis will not be accomplished as the number of articles on that subject is almost infinite. Especial emphasis will be placed upon symptomatology and diagnosis with a brief synopsis of the histo-pathology.

Treatment, prognosis and sequelae will knowingly be omitted.
-Definition-

Acute anterior poliomyelitis is a general, systemic infectious disease which tends to involve the central nervous system, attacking especially the gray matter of the spinal cord, but capable of producing widely scattered lesions throughout the tissues of the brain and cord.

Clinically the disease is characterized by two definite phases: first, a stage of general toxemia; the second, a manifestation of the results of damage to the ganglion cells of the anterior horns and ganglia of the posterior roots. The disease often occurs sporadically, but has strong epidemic propensities. (1c)

-History-

The first mention of poliomyelitis as it is understood today was by Michael Underwood in 1784, in the first edition of his treatise on the "Diseases of Children". (1) The only possible earlier reference was by Boerhaave in 1761 when he issued an extensive treatise, in two volumes, on diseases of the nerves. The section "De Paralysisi" is largely devoted to apoplexy and brain and nerve injuries, but there is one statement of possible significance. He states that he has heard of a peculiar type of paralysis in Asia that follows and seems to be produced by a cold (2).

The most important part of Underwood's description in 1784 is as follows:
"Debility of the Lower Extremities" - "This disorder either is not noticed by any medical writer within the compass of my reading, or is not so described to ascertain the disease here intended. It is not a common disorder anywhere, I believe, and seems to occur more rarely in London than in any other part of the kingdom. Nor am I enough acquainted with it to be fully satisfied, either in regard to the true cause, or seat of the disease, either from my own observation, or that of others, with whom I have corresponded, except in the instance of teething or of foul bowels; and I have not myself had an opportunity of examining the body of any child who has died of this complaint. I shall therefore only describe its symptoms, and mention the several means attempted for its cure in order to induce other practitioners to pay attention to it.

If it arises from teething or foul bowels, the usual remedies should be employed; and have always effected a cure. But the complaint as often seems to arise from debility, and usually attacks children previously reduced by fever, seldom those under one or more than four or five years of age. It is then a chronic complaint, not attended with an affection of the urinary bladder, nor with pain, fever or any manifest disease; so that the first thing observed is a debility of the lower extremities, which gradually become more infirm, and after a
few weeks are unable to support the body.

When only the lower extremities have been affected the above means in two instances out of five entirely removed the complaint; but when both have been paralytic, nothing has seemed to do any good but iron to the legs, for the support of the limbs, and enabling the patient to walk. At the end of four or five years, some have by this means got better in proportion as they have acquired general strength; but even some of these have been disposed to fall afterward into pulmonary consumption, where the debility has not been entirely removed (1).

In 1823, Shaw (2) published a paper on orthopedics in which he says: "But certain paralytic affections of the muscles are sometimes so instantaneous that we must consider them upon the change which has suddenly taken place in the brain or spinal marrow or in the nerve which supplies the affected parts." Shaw also cites several cases that he had seen in India.

In 1828, Abercrombie (2) reported a typical case, with autopsy, of acute respiratory paralysis in a healthy child of two years.

The next important article was Badham's (3) report in the London Medical Gazette, 1835. Badham's report was entitled "Paralysis in Childhood: Four Remarkable Cases of Suddenly Induced Paralysis in the Extremities, Occurring in Children Without any Apparent Cerebral or
Cerebro-Spinal Lesion." Ager believes this report to be very important for two reasons: First, it is the first recorded instance of a group of cases suggesting transmissibility, and may therefore be considered the first epidemic. Second, Heine cited this report as the direct stimulus to his systematic study of the disease.

In 1840, Heine of Stuttgart, (4) separated from a heterogeneous group of paralyses a clinical type characterized by cord lesions and referred to at that time as cases of infantile paralysis. In 1885, a quarter of a century later, Prevost and Vulpian studied the lesions in the anterior horn and attributed to them the symptoms of infantile paralysis. In 1881, Charcot, according to Amoss, was the first to accurately describe in his lectures the acute onset preceding the paralysis. Strumpell in 1884 presented several cases of acute brain disease and pointed out the epidemiological occurrence, and drew the analogy between them and poliomyelitis, the infectious nature of which he emphasized.

Medin (1889), whose name was connected with Heine's in the designation of poliomyelitis (Heine-Medin disease), reported a large Swedish epidemic and accurately described the acute stage. Wickman, a student of Medin's studied the great epidemics of Sweden and his clinical, pathological and epidemiological contributions are classic.
The first epidemic (11 cases) was reported by Colmen (5) of Louisiana in 1841. Three other outbreaks were described before 1885, and since that time the disease has appeared in epidemic form in some part of the world every year. The first large epidemic in America occurred in Vermont in 1894 (132 cases) and was reported by Caverly (6). Caverly also called attention to a small number of cases (6) of apparently the same disease, but which did not reach the paralytic stage. Wickman later and independently described this type as abortive cases.

New York City was visited by a large outbreak (2,500 cases) in 1907 and again in 1916 (9,005 cases). In Wisconsin there were about 1,000 cases in 1908 and about 150 cases in Minnesota the same year. In the Nebraska outbreak of 1909, there were 999 cases reported. In 1914 in Vermont there were 350 cases among a population of 300,000. Every state in the Union has been visited by poliomyelitis within the past twenty years.
In perhaps no other disease than poliomyelitis has the parallel between the human illness and the experimentally produced affliction been more finely drawn. Not only the clinical features but the anatomical lesions are identical. Consequently in this paper the pathology in the experimentally produced disease in monkeys will be described.

In 1929, Fairbrother and Hurst (7) did some experimental work on the histo-pathology of poliomyelitis. A brief resume of their observations and conclusions will be given.

It is generally agreed that the condition in monkeys accurately reproduces the essential features of the disease in man. Fairbrother and Hurst inoculated the virus intracerebrally into the parieto-occipital region, and a histological examination was made at varying periods afterwards.

In monkeys killed one or two days after injection, there was a meningitis spreading from the site of the inoculation over the cerebral hemispheres. The meninges of the cerebellum and brain stem were rarely and those of the spinal cord never infected.

By the third and fourth days after injection there were early lesions consisting in cellular foci and early
perivascular infiltration in the thalamus of the same side of injection, in the cortex immediately adjacent to the site of inoculation, and in some cases in the midbrain and pons. The cord was normal. General meningeal infiltration had not spread to the brain stem.

On the fifth day, which was the day preceding or the day of appearance of paresis, in the cerebrum there were cellular foci and areas of perivascular infiltration often occurring in the absence of overlying meningeal infiltration. Marked lesions were present in the thalamus, mid-brain and the pons. The medulla and the cord were irregularly affected. At some levels, the appearance was normal while at other levels there were early lesions. Nerve cell degeneration was already seen in some of the anterior horn cells, even in the absence of local interstitial changes. Cellular foci were scattered at random over both the anterior and posterior horns.

By the eighth day after inoculation, the fully developed disease was present. While the lesions in the cortex and basal ganglia were not much altered from those seen the fifth day, those in the brain stem and in the spinal cord had increased greatly in severity and clinically had produced general paralysis and prostration. Nerve cell destruction was greatest at the lumbar and cervical enlargements of the cord. Pial infiltration
was insignificant in many cases. There was vascular congestion. Perivascular infiltration was not always observed even in the cases of most severe nerve cell destruction. The nerve cell destruction was always incredibly rapid. In the early stages, when tremor alone was present, only early lesions of the nerve cells were found. Twenty four hours later the majority of the affected anterior horn cells would be a necrotic mass of debris. After this twenty four hours there was a sharp distinction of the nerve cells into two groups: those which were obviously necrotic and those in which the changes seemed definitely recoverable. As degeneration of the nerve cells proceeds neuronophagia begins. Polymorphs, lymphocytes and glial cells quickly remove the dead cells.

In the medulla and pons nerve cell damage was never as severe as in the cord. In the mid-brain perivascular infiltration was often intense. Nerve cell damage was rarely severe and actual necrosis not seen. The basal ganglia, the cortex, the cerebellum, and intervertebral ganglia were all affected to some extent.

Thus, Fairbrother and Hurst showed, as many others have done before and after, that poliomyelitis is an inflammatory disease of the whole nervous system with an especial involvement of the anterior horn cells.
They consider that the nerve cells are primarily affected by the virus and that their degeneration is not attributable to the accompanying interstitial inflammation. They also conclude that meningitis is not a necessary feature of the disease, and that while meningeal spread may take place, the usual route is by axis cylinder. They show that, although histologically the disease first affects the mid-brain and pons, clinically the first manifestation is the spinal involvement.
There are few infectious diseases so frequently incorrectly diagnosed. Gordon (8) found that of 446 patients referred to the Herman Kieffer Hospital, Detroit, as poliomyelitis only about one half of them were later confirmed after extensive clinical and laboratory study. Below is a table of correct diagnoses, based on thorough study at the Herman Kieffer Hospital.
Clinical Diagnosis of Acute Communicable Diseases
(Herman Kieffer Hospital 1928-1931)

<table>
<thead>
<tr>
<th>Disease</th>
<th>No. of Cases Referred</th>
<th>Diagnosis Confirmed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scarlet Fever</td>
<td>9605</td>
<td>9204</td>
</tr>
<tr>
<td>Whooping Cough</td>
<td>400</td>
<td>379</td>
</tr>
<tr>
<td>Erysipelas</td>
<td>972</td>
<td>910</td>
</tr>
<tr>
<td>Chicken Pox</td>
<td>346</td>
<td>316</td>
</tr>
<tr>
<td>Measles</td>
<td>900</td>
<td>816</td>
</tr>
<tr>
<td>Mumps</td>
<td>160</td>
<td>135</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>4980</td>
<td>3850</td>
</tr>
<tr>
<td>Cerebrospinal Meningitis</td>
<td>1591</td>
<td>1227</td>
</tr>
<tr>
<td>Small Pox</td>
<td>264</td>
<td>197</td>
</tr>
<tr>
<td>Encephalitis</td>
<td>52</td>
<td>35</td>
</tr>
<tr>
<td>Typhoid Fever</td>
<td>53</td>
<td>34</td>
</tr>
<tr>
<td>Poliomyelitis</td>
<td>446</td>
<td>226</td>
</tr>
</tbody>
</table>

From the above chart it can be readily observed that the diagnosis of poliomyelitis in the preparalytic stage is not as easily accomplished as it is in scarlet fever, whooping cough and the other acute communicable diseases. Kramer offers an explanation for this error in diagnosing poliomyelitis in the preparalytic stage have been due not so much to the absence of diagnostic signs and symptoms as to the lack of opportunity to observe that stage of the disease. It is unfortunate that in many instances the symptoms are so insignificant that parents are liable to overlook them. When a child is found to have lost the use of an extremity, the serious nature of the illness is realized and physician is called. By this time the diagnosis is obvious.
The low incidence of poliomyelitis gives little opportunity for physicians to become acquainted with this illness. This is true because even in severe outbreaks the incidence of poliomyelitis rarely exceeds two cases in 1,000 population.

Gordon (8) believes that the errors of early diagnosis of poliomyelitis are due to overemphasis of the disease, particularly during the period of seasonal prevalence and during epidemics. He believes that the average physician realizes the importance of an early diagnosis and in their eagerness to arrive at a diagnosis confuses poliomyelitis with other conditions that are as urgent. No less stress should be placed upon an early diagnosis of poliomyelitis but more critical judgement should be used in eliminating other conditions.

There are few diseases that give rise to such great alarm and hysteria in a community as does poliomyelitis. This is a factor which necessitates an early diagnosis and the institution of proper, effective treatment if a physician's reputation is to be preserved and honored in a community during an outbreak of the disease. In spite of the fact that poliomyelitis is not one of the more common epidemic diseases, it is not difficult to account for the general excitement during an outbreak. Undoubtedly, this is caused by the permanent crippling that so
frequently follows an attack of the disease. John Badham, one of the earlier observers of the disease said, "It is lamentable, indeed, to witness one of the most humiliating infirmities of age inflicted on infancy." This and the fact that there are as yet no adequate measures for control, undoubtably help to produce a feeling of helplessness and consequent hysteria.

There are a number of epidemicological features that are of value in making a diagnosis in the disease. These are the seasonal variation, geographical distribution and age incidence. Poliomyelitis is decidedly a disease of warm weather, occurring with great regularity in late summer and early fall. Winter epidemics have been reported but they have been small and rare. Poliomyelitis is a disease prevalent in temperate climates where seasonal changes in temperature are extreme. It is primarily a disease of early childhood, most cases occurring before the sixth or seventh year of life (13,16,27).

Kramer (9) offers a simple classification of the disease. He divides into the paralytic form and the non-paralytic form. The non-paralytic form is subdivided into the abortive form and the true non-paralytic form. The type in which we are most concerned in this paper is the paralytic form as it is the one that necessitates an early diagnosis for full effect of therapy.
Below is a general description of the symptomatology as agreed upon by most writers. The onset of poliomyelitis is more or less sudden with general symptoms. A previously healthy child seems listless with a loss of appetite. During an epidemic, according to Draper (10), from 20 to 40% of all cases give a history of an upset from three to ten days previously. Usually the first indisposition passes quickly and the child remains well until the second phase or definite attack of acute poliomyelitis is evident. This is the so-called "dromedary" type described by Draper. In nine out of ten cases there are gastro-intestinal disturbances, either diarrhea or constipation. When diarrhea is present, it is usually of short duration followed by constipation. The patient usually vomits only a few times and the mother is prone to ascribe the upset to dietary indiscretion, as the vomiting is not projectile or different from the vomiting of a simple gastro-intestinal disturbance. A dull headache is the rule; however, Amoss (4) states that he has seen a number of cases in children old enough to describe their sensations in which the symptom was absent. Most of the cases are drowsy, but can be aroused. Some are restless and excited, and rarely there is delirium; fever is almost always present. The temperature
is usually from 100 degrees to 102.5 F though it may reach 105 degrees, endures for a variable period from three hours to four or five days, and returns rather rapidly to normal.

The febrile period may come and pass unobserved during the night and the only history obtainable will be that of a wakeful, fretful period. It is in this type of case, where the child has passed through an unrecognized febrile period, that he arises two or three mornings later with a group paralysis (so-called "morning paralysis"). Most observers, including Scott (12) have found this to be exceedingly rare.

At the onset the patient is flushed and presents a picture similar to that of the onset of other acute diseases of childhood. The sclera appear dulled and the face seems glazed all over. One writer describes the appearance of the patient as though he were being looked at through smoked glasses. The pulse rate is usually out of proportion to the fever.

Besides the headache the patient often complains of pain in the neck or between the shoulders and in the back. The pain is accentuated by the stretching of the neck when the patients' head rests on pillows. One of the outstanding diagnostic findings is a stiffness of the neck when the head is flexed on the chest and stiffness of the spine when anterior flexion of the trunk on
the thighs is attempted. The stiffness of the neck and spine is so striking that it can readily be detected on inspection. The patient assumes a protective attitude, lying flat on his back and with greatest comfort without a pillow. It is also noted when the patient attempts to sit up, a procedure that is usually accomplished by a series of shifts from side to side keeping the neck and back stiff with gradual elevation of the trunk on the supporting arms. The complete sitting position is usually not reached, the trunk remaining in a partially reclining position supported by the backwardly extended arms, the hands resting on the bed, the position of the trunk and head is so straight that the term "poker spine" has been applied to it. When forcible flexion of the head or trunk is attempted by the examiner, pain in the upper thoracic or lumbar region is elicited. Pain in the legs, hips or arms may appear suddenly in the night, sufficiently severe to awaken the patient. One of the more distressing symptoms is the exquisite hyperesthesia of the skin occurring in less than 50% of the cases. It usually lasts from five to ten days and is often prolonged by pressure from the bed clothing and massage.

General profuse or local areas of sweating on the head or back of neck, on an arm or leg, appear in some cases. Flushing of the skin or, more often, blanching of an arm or leg is often the precursor to paralysis in that
part. Morbilliform or scarlatiniform rashes with heavily coated tongue, congested throat and occasional minute red spots in the buccal mucosa, have been noted by some observers (13). In over 1200 cases, Amoss (4) has not seen a skin lesion in uncomplicated cases.

At some time during the course of the disease there is a disturbance in the reflexes. There does not seem to be any definite rule as to the characteristic reflex findings although it seems that in most cases presented in the literature there is a definite asymmetry and a greater variation in the lower extremities. The variations from normal are usually towards diminution and abolition rather than hyperactivity. When hyperactivity is present it is usually very early in the disease, later disappear and then finally the response being lost altogether. Concerning the reflexes, Gordon (8) says, "the cremasteric and superficial abdominal reflexes are among the earliest altered. Their absence is significant. The deep tendon reflexes usually gives little information from a single examination, but tend to be hyperactive early in the disease and later depressed or absent. Inequality of the reflexes on the two sides is important, and variation at successive examinations still more so. The Kernig, Babinski and Brudzinski signs are usually not demonstrable."

Twitching of muscle groups often occurs during rest or sleep in the early period of the attack. These
twitchings and intention tremors are usually considered to forebode paralyses in those muscles.

Most observers mention the fact that occasionally a cerebral tache is present. Aycock and Luther (18) go so far as to say that it is almost always present and that frequently it becomes a purplish, irregular blotchy line a half inch or more in width.

-Classification-

There are a number of different classifications of the various types of poliomyelitis by approximately as many authors. Stephen (19) classifies the disease into the following six clinical types: (1) Spinal; (2) Ascending and descending, also termed "jump" or "stepping" owing to mode of spread of paralysis; (3) Bulbar and pontine (4) Encephalitic or cerebral; (5) Meningitic; (6) Abortive.

In the spinal form the lower lumbar segments of the cord are most commonly affected, the cells of the anterior tibial group seeming to be especially liable. When the lesions are in the thoracic segments the abdominal or intercostal muscles may be paralyzed. In this event respiration is carried on by the diaphragm and accessory muscles of respiration. The dorsal muscles of the back depend upon the anterior horn cells of this region and their paralysis leads to spinal curvature. Lesions in the cervical region results
in paralysis of the muscles of the upper limbs and neck.

In the ascending or "jump" type the lower extremities are usually first affected, then the abdominal muscles, then the muscles of the arm and neck and finally the diaphragm and intercostal muscles. Ten days or more may elapse between "jumps". Though the prognosis is grave if the respiratory muscles are affected, recovery may occur.

In the bulbar and pontine type any muscles of the head and neck supplied by the third to the twelfth cranial nerve may be paralyzed. If there is extensive involvement of the medulla, death from paralysis of the vital centers is likely. The nucleus of the facial nerve seems to be the most commonly affected nerve in this region (20).

In the cerebral or encephalitic form the general symptoms of invasion are present plus, often, convulsions. The frontal, motor or occipital region of the brain or the basal ganglia may be affected. The lesions may be unilateral or bilateral (21).

In the cerebellar type after the usual initial symptoms incoordinate movements of the arms, a drunken gait, nystagmus, and a jerky articulation may develop. It may be associated with other types of lesions mentioned (22).

In the meningitic type the cerebro-spinal fluid has the characteristic features of meningitis. All the symptoms of meningeal irritation develop (19).
There is considerable discussion in the literature concerning the abortive type. Those cases so labeled do not continue into paralysis. The initial symptoms of true poliomyelitis, that is fever, headache, pain in the extremities, general weakness, some gastrointestinal upset and occasionally a sore throat are present and are followed by recovery without paralysis. Whether this is a true type of poliomyelitis or just an upper respiratory infection with a gastrointestinal upset, occurring during an epidemic, is debatable.
-Hematology-

It is quite generally agreed by most observers that there exists abnormal white blood counts in poliomyelitis. Opinion seems to be divided upon the characteristic change in the white blood cell picture. La Fetra (14) has described a moderate leucocytosis as being characteristic of the acute stage. Gay and Lucas (15) summarize their blood findings as follows: "The acute stage of anterior poliomyelitis as it occurs in human beings and as it is produced experimentally in monkeys, is characterized by occurrence of a distinct leukopenia. The differential count shows a relative increase in number of eosinophils and lymphocytes." Peabody, Draper and Dochez (16) found in human cases a constant and marked leucocytosis. They also found a constant increase in polymorphonuclear cells of from ten to fifteen per cent and a diminution of lymphocytes of from fifteen to twenty percent.

Taylor (17) made blood counts on six series of monkeys before and at various intervals after inoculation with poliomyelitis virus, and used as a basis of comparison averages of one hundred and twenty one counts on forty normal monkeys. He found that all the counts made on monkeys during
the course of typical acute experimental poliomyelitis showed a variation from the normal and after injection of active poliomyelitis virus the lymphocytes were diminished but return to their former number and were actually increased by the sixth day of the incubation period. The polymorphonuclear count was high at this time. The normal average of lymphocytes of 11,1915 was increased to an average of 19,690 though the average percentage was slightly lowered. During the first three days after onset a marked diminution of lymphocytes took place. Instead of an average normal lymphocytic count of 11,815 the number was 3,302 and the average percentage 15.6. At the time the polymorphonuclear cells were materially increased in number, 18,231 as compared to the average normal of 9,116 and the percentage increased from 41 to 84 per cent of the total white cells. The total number of polymorphonuclear neutrophilic leukocytes returned to normal but there remained a relative increase averaging 77 percent. Finally, during recovery both types of cells returned to the average normal count and relation. No stimulation of the lymphocytes above normal appeared during recovery.

Opinion seems to be divided as to the characteristic blood picture of the early stages of poliomyelitis. Many observers in their reports on clinical studies of their private cases fail to mention the possibility of the blood as
aiding in the establishment of a definite diagnosis of poliomyelitis. This consistent omission must mean either lack of knowledge as to proper interpretation or granted unimportance of the blood picture in the acute stage.

-Spinal Fluid-

The spinal puncture is of utmost importance in the early diagnosis of poliomyelitis. Gordon (8) says that the diagnosis in the preparalytic stages depends upon a healthy clinical suspicion plus a lumbar puncture. Every observer agrees that the key to ultimate diagnosis is the examination of the spinal fluid (26, 28, 31).

In poliomyelitis the spinal fluid is delivered from the lumbar puncture needle under increased pressure. The exact amount of pressure does not seem to be necessarily known but it is usually between 150 and 200 millimeters of water. The gross appearance seems to be of distinct value. Normal spinal fluid is clear. The fluid of a poliomyelitis victim is relatively clear but when viewed in transmitted light it gives a slight haziness or ground glass appearance. A definitely cloudy fluid is found in patients with purulent meningitis. Also a fibrin web does not form in the fluid from a poliomyelitis case as it does in conditions of chronic meningeal involvement, such as in tuberculous meningitis or chronic meningococccic meningitis.
Determination of the number of cells per cubic millimeter in the spinal fluid is one of the most valuable laboratory tests. The usual and more simpler way of doing the count is to dilute the fluid in a white blood cell pipet with acetic acid containing a small amount of methylene blue (The methylene blue facilitates the counting). The normal spinal fluid count is 0 to 10 cells per cubic millimeter. In poliomyelitis the variation is from 50 to 200 cells. Amoss (4) reports a case where the spinal fluid cell count was 1200 per cubic millimeter and Draper reports a case with a spinal fluid cell count of 2400 per cubic millimeter. The cells should be counted promptly and must be free of red blood cells as they simulate lymphocytes.

The kind of cell in the spinal fluid is also of diagnostic significance. Observers agree that in early involvement, neutrophilic polymorphonuclear cells predominate. Within 24 hours there is usually a shift with the lymphocytes predominating almost to the exclusion of the neutrophils. (29)

On the second day a slight excess of globulin usually appear and gradually increases each day for several days. Excess of globulin usually persists after the cells have disappeared (4).

All the observers spend considerable time and space in telling of the importance and value of the spinal fluid
in making a diagnosis of poliomyelitis; yet none of them have failed to say that they have seen many cases in the acute stage that had a relatively normal spinal fluid and progressed into typical paralytic cases. No observer ventured a percentage of the cases with a relatively normal spinal fluid.

The following table is Gordon's (8) conception of spinal fluid findings in poliomyelitis and other conditions:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Appearance</th>
<th>No. of Cells</th>
<th>Predominant Cells</th>
<th>Mg. Sugar</th>
<th>Bac</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polio</td>
<td>Clear</td>
<td>0-10</td>
<td>Lymphocytes</td>
<td>60-80</td>
<td>None</td>
</tr>
<tr>
<td>Encephalitis</td>
<td>Clear or Slightly Hazy</td>
<td>15-500</td>
<td>Very Early Neutrophilic; Later, lymphocytes</td>
<td>50-80</td>
<td>&quot;</td>
</tr>
<tr>
<td>TB Meningitis</td>
<td>Clear</td>
<td>15-500</td>
<td>Lymphocytes</td>
<td>10-60</td>
<td>Present</td>
</tr>
<tr>
<td>Purulent Meningitis</td>
<td>Purulent</td>
<td>500-20,000</td>
<td>Neutrophils</td>
<td>10-60</td>
<td>&quot;</td>
</tr>
<tr>
<td>Syphilis, Cerebrospinal</td>
<td>Clear</td>
<td>10-500</td>
<td>Lymphocytes</td>
<td>10-60</td>
<td>None</td>
</tr>
</tbody>
</table>
-Examination-

In epidemics, a child with a gastro-intestinal upset, headache and fever, less alert and bright than with ordinary fevers, somewhat cranky and unapproachable, should be examined more carefully with the possibility of poliomyelitis in mind.

Such a child is difficult to examine. The friendly and tractable child becomes suspicious and distrustful and is obviously annoyed by the physician. A few minutes devoted to making friends with the patient is very helpful, as much depends upon cooperation. Before any manipulation, the chest and abdomen should be observed carefully to note any intercostal or diaphragmatic paralysis. To test the intercostals, the diaphragm is immobilized by pressure with both hands on the abdomen and likewise the intercostals are immobilized by pressure on the chest in testing the diaphragm. This examination should be made gently and quickly. It is inadvisable to subject the patient to a prolonged and exhausting neurological examination at this stage. After the usual physical examination, leaving the pharynx to be examined last, the reflexes are tested with a soft rubber hammer. In addition to the abdominal and plantar response, valuable information is often obtained by stroking the plantar surface and simultaneously palpating the inner hamstring on the same side. In this test the knee should be slightly flexed. The normal
response is a tightening of the hamstring muscle, but in poliomyelitis there is often no response. (31, 32, 30).

If there is no involvement of the respiratory center and no other contra-indications, the patient is allowed to sit on the edge of the bed. In early poliomyelitis the patient assumes a characteristic attitude; The back is held straight and both hands rest on the bed slightly behind the buttocks with the arms straight and stiff in an attempt to take the strain from the painful back. When the patient is asked to bend over and place the head between the knees, the back is held straight and rigid and the patient bends only from the hips. This is one of the most constant findings in early poliomyelitis. Additional evidence of the tender spine is obtained in the manipulation of the Kernig's signs, the response to which is not a true reflex. Finally the attempt is made to bring the chin to the chest by anterior flexion of the neck. The manipulation will cause pain. This again is not true rigidity but a voluntary mechanism for protection. (32)
-Differential Diagnosis-

There are three conditions which in the early stages are likely to be confused with acute poliomyelitis. There are enumerable other diseases that may cause confusion but here only the differential between epidemic meningitis, tuberculous meningitis, epidemic encephalitis and early poliomyelitis will be briefly discussed.

Epidemic meningitis referable to high portions of the cord may in occasional instances begin with group paralysis. In the first few hours, the number of cells in the spinal fluid may be well within the upper limits of the number found in acute poliomyelitis and many of them found cells in the proportion not exceptionally found in poliomyelitis. The globulin tests usually, however, show a much heavier precipitate than occurs at this stage in poliomyelitis. In epidemic meningitis there is a true reflex rigidity of the neck and back instead of the voluntary stiffening in poliomyelitis. The body surfaces should be searched for red splotches which suggest epidemic meningitis (23).

Tuberculous meningitis often begins with a gastro-intestinal upset similar to that observed in poliomyelitis, and the latter particularly in young children may give the typical picture of tuberculous meningitis with strabismus. Chemical examination of the spinal fluid usually gives the clue as the cell content in both diseases may be identi-
cal, but in tuberculous meningitis the excess globulin appears early in the course of the disease. In tuberculous meningitis close questioning usually brings out the history of a period of indisposition, failing appetite, and lassitude before the actual onset, whereas in poliomyelitis, this history is lacking.

Web and fibrin formation, which were formerly considered pathogenomic of tuberculous meningitis, occur sometimes in poliomyelitic spinal fluids. In fact, fluids from the first taps in these two diseases may be practically indistinguishable. It is obvious that the differential diagnosis cannot be made by the laboratory except in the small percentage of cases of tuberculous meningitis in which the tubercle bacilli are found in the first fluid withdrawn (24).

Epidemic encephalitis without localizing signs and the encephalitic form of acute poliomyelitis may simulate one another so closely as to present a difficult problem in differential diagnosis, especially in the early stages. The diffuseness of the lesions, an exaggeration of the stupor and a clouded psyche point toward epidemics of that disease. The spinal fluid findings assist in about half of the cases. In epidemic encephalitis, the fluid is abnormal in about fifty percent of the cases and the cell count in these is usually not as high as in the encephalitic form of poliomyelitis. The course of the encephalitic poliomyelitis is relatively short, but mortality is low, and in recovered cases there are not sequelæ which are confused with distressing after-
malt of epidemic encephalitis (23).
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