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ACUTE POLIOMYELITIS

WITH SPECIAL REFERENCE TO EARLY SYMPTOMATOLOGY AND CONTACT HISTORIES

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In the middle of August, 1947, the Middlesex Hospital offered to the London County Council a unit of 30 beds for the early treatment of cases of acute poliomyelitis. Although the diagnosis of poliomyelitis could be confirmed in only 54 of the 104 cases which were admitted up to the middle of October, sufficient points of interest arose to warrant their description. The following classification was adopted: (1) non-paralytic, 24 cases; (2) paralytic, 26; (3) polio-encephalitis, 4; (4) abortive poliomyelitis, 6; (5) incorrect diagnosis, 44.

Early Symptomatology

Prodromata.—Prodromal symptoms suggesting a stage of systemic infection were present in 70% of the non-paralytic and paralytic cases. The symptoms were those that are common to certain other infections—namely, headache, a shivering feeling, malaise, a generalized muscular aching, and fever. To these were sometimes added either signs of an upper respiratory infection such as sore throat or coryza, or a gastro-intestinal upset such as nausea, vomiting, or diarrhoea. The duration of this prodromal stage varied from 14 days to a few hours. In 9 of the 10 patients who became severely paralysed, signs of invasion of the nervous system preceded the appearance of constitutional symptoms. In a few cases the history suggested the “dromedary” type of prodromata.

Non-paralytic Form

In deciding whether a case should be classified as abortive or non-paralytic we were guided by the presence of meningeal signs and by changes in the cerebrospinal fluid; if the latter was normal, despite the presence of severe headache, the case was considered as abortive, but if in addition to these findings nystagmus was present the case was labelled as non-paralytic.

Headache was the main symptom of the meningeal stage of the disease. At the onset it may be referred to the back of the head, but more usually it is frontal in distribution and throbbing in character; it was sometimes described by patients as the worst headache they had ever experienced. Pain referred to the neck or down the back was not uncommon. All degrees of neck stiffness were met with. In a mild form this showed itself by an inability on the part both of the patient and of the examiner to approximate the chin to the chest; during this manoeuvre the patient expresses discomfort in words or by facial expression. In the course of a routine examination of the

nervous system no abnormality may be detected beyond those already mentioned. In other cases certain minor signs may be found which clearly indicate invasion of the central nervous system, but since they are not accompanied by paresis such cases should be grouped as non-paralytic. The commonest of these signs is nystagmus; this may occur in lateral or vertical fixation, and may be either of the horizontal or the rotatory type. Its disappearance in the course of convalescence signifies its relationship to the infection. Of 24 non-paralytic cases 14 showed this sign. The following may be cited as a typical example of the non-paralytic form.

Case 52.—L. E., a girl aged 17, was well until Oct. 5, 1947, when she felt sick and retired to bed. Next day the temperature was 99° F. (37.2° C.), the headache was worse, and she vomited three times. On Oct. 7, when seen in casualty department, apart from mild backache she had no other symptoms. She was admitted for lumbar puncture. The cerebrospinal fluid contained 35 lymphocytes and 29 polymorphs per c.mm.; protein, 40 mg. per 100 ml.

Depression or absence of one or more deep reflexes was found in a few of the non-paralytic cases, either without any evidence of paresis or with a minimal degree of weakness which passed off in the course of a day or so.

Paralytic Form

As a general rule prodromata and meningeal signs preceded the onset of paralysis. Both our previous and recent experience of poliomyelitis confirmed the importance of severe low backache as a local sign in poliomyelitis, particularly when the lower dorsal and lumbar cord is attacked. In the majority of our cases sensory symptoms were prominent at the onset. They were confined to or more pronounced in that part of the trunk or limbs which subsequently became paralysed. Pain was a common early symptom, while tenderness of the skin and pins-and-needles or a numb sensation were occasionally present. The following case illustrates this point.

Case 14.—Mrs. K., aged 30, noticed soreness around the abdomen on Sept. 4, 1947; her clothes hurt her and any light touch on the skin was unpleasant. By the evening the soreness had spread to the front of both thighs. Next day the temperature was normal. She complained of severe headache and aching at the back of the neck and in her limbs and back. On the 6th the temperature was 99° F. (37.2° C.); the pain in the legs had disappeared, but her left leg was now completely useless and the right leg partially so. She was admitted to hospital on

Sept. 7; the temperature was 102° F. (38.9° C.). The following signs were noted: marked neck rigidity, horizontal nystagmus, and a severe degree of paralysis of both lower limbs with diminution of the deep reflexes. Cerebrospinal fluid showed: cells, 94 lymphocytes per c.mm.; protein, 100 mg. per 100 ml.

Muscles supplied by the same spinal segments may show a considerable variation in the extent to which they are paralysed. For example, in the upper limb the deltoid may be severely paralysed while the biceps and brachial radialis may show only a temporary and mild weakness. The extensors of the wrist may relatively escape compared with the triceps and extensors of the fingers. Similarly, the sternal portion of the sternomastoid may be more affected than the clavicular portion. Although these facts are not new, attention is drawn to them because of their diagnostic importance. Coarse fasciculation or muscular twitching, although not an uncommon early sign in children, is seldom seen in adults; it was observed in only two cases in the present series. Lastly, minor degrees of paresis are easily overlooked unless a systematic examination of the skeletal muscles is made.

The Myelitic Form.—This has received little mention in the classical descriptions of the disease. The clinical picture may be that of an acute dorsal myelitis, differing in no way from that due to other causes. A stage of flaccid paraplegia with extensor plantar responses, sensory loss on to the abdomen, and retention of urine is followed by a residual spastic paraplegia with minimal sensory loss. The combination of meningitis and acute myelitis with a lymphocytic pleocytosis in the cerebrospinal fluid should suggest the diagnosis, but only if other causes such as syphilis can be excluded. A partial myelitis characterized by temporary paraesthesiae and combined upper and lower motor neurone lesions is somewhat more common. The following is a typical example.

Case 5.—W. A., a youth aged 17, was driver's mate in a lorry which left Yorkshire bound for London on July 21, 1947. At 9 p.m. he felt a sudden dull ache in the lower part of his back; this pain passed off within an hour. At midnight he noticed a gradual onset of numbness in the left hand and the fingers, which felt dead. Within an hour the numbness had spread up to his elbow; at the same time he noticed a weakness in this limb. At 4 a.m. the following day the numbness was noticed in the toes, and within a quarter of an hour it had spread half-way up the left thigh. The leg became weak, and on getting out of the lorry he limped. At 8 a.m. there was a gradual onset of numbness in the right hand and arm, with weakness. At 10 a.m. the numbness had spread to the abdomen, lower chest, and groins. At 12 noon he was admitted to hospital. The temperature was 98.2° F. (36.8° C.). He stated that he had no headache and that he felt quite well. There was no neck rigidity. Both upper limbs were markedly weak, except abduction and flexion of the elbows. Paralysis of finger movements was complete in the left hand and partially so in the right. The triceps-jerk was absent on the left side. There was a generalized weakness of both lower limbs, left more than right. The left knee-jerk was depressed; the remaining lower-limb reflexes were exaggerated. The left plantar response was equivocal, the right flexor. There was diminution to cotton-wool and pin-prick in both hands; doubtful pin-prick and thermal loss on right side below D2. Cerebrospinal fluid: cells, 7 lymphocytes and 1 polymorph per c.mm.; protein, 30 mg. per 100 ml. On July 24 the temperature had remained normal since admission and there was good recovery in extension of left wrist, but triceps, extension of fingers, and small muscles of left hand as well as of right were still severely paralysed. Reflexes in left leg were now brisker than on right, left plantar response extensor, right flexor. On Aug. 16 he was much improved: mild residual weakness in left triceps and intrinsic muscles of both hands, left more than right, with early wasting. Left plantar response was still extensor. Sensation was now normal.

When the case was first admitted the absence of fever and meningeal signs and the prominence of sensory

symptoms suggested an acute myelitis rather than polio-myelitis. However, after 48 hours the distribution of the paresis in the arms and the greater loss of power in the quadriceps as compared with other muscle groups in the left leg established the diagnosis of poliomyelitis.

Polio-encephalitis

Several factors account for the varied symptomatology that may occur in polio-encephalitis. First, although the virus of poliomyelitis is highly neurotropic for motor nuclear masses in the brain stem and spinal cord, it may attack other components of the nervous system. Secondly, although the brain stem is mainly affected, there is clinical and histological evidence that in polio-encephalitis higher levels of the brain may not be spared. Lastly, the relative incidence of polio-encephalitis and the signs arising from it vary considerably from epidemic to epidemic. Drowsiness may be present in the initial stages. In only one of our four cases of polio-encephalitis was it marked, although it appears to have been a prominent feature in a certain number of cases in the recent epidemic. Coma in our experience is rare. A degree of mental confusion with subsequent amnesia is not uncommon. Nystagmus is an important sign of invasion of the brain stem; it may occur alone or be associated with cranial-nerve or spinal paralyses. Pyramidal signs are not uncommon in polio-encephalitis.

There is a clinical picture which we have not previously encountered and which so far as our inquiry into the literature goes has not been reported in any previous outbreak of the disease. Some of the features of this unusual condition have been described by Strickland (1947), Marmion and Sandilands (1947), and one of us (McAlpine, 1947).

Case 28.—R. P., a captain in the U.S. Army aged 32, complained on Aug. 11, 1947, of mild backache, which persisted until the 21st, when he developed a headache, sore throat, and aching pains in the neck and legs. He vomited once and had mild diarrhoea. The temperature was 100° F. (37.8° C.). During the next few days he improved. Nystagmus was noticed for the first time on Aug. 25. On the 27th he was unable to raise his head because of muscle weakness and the nystagmus had increased. Next day he was admitted to hospital. The temperature was 98.8° F. (37.7° C.). He complained of backache, weakness, and blurring of vision. He was markedly euphoric, with emotional lability. His memory was good for both recent and past events; his orientation and co-operation were excellent. There were no disturbances of micturition and his sleep rhythm was normal. A slight degree of neck rigidity was noted. He showed violent jerky conjugate movements of the eyes both laterally and on elevation. There was a large rotatory element in the movements especially on lateral fixation. The slightest movements of the eyes, the act of blinking, and even an emotional stimulus would produce this phenomenon. After an object was fixed for a few seconds the ocular disturbance subsided. Occasionally, spontaneous movements would appear, though these may have been due to attempted voluntary fixation unnoticed by the observer. With the violent lateral movements a synchronous lateral tremor of the head and neck developed which was very obvious to the patient and which he could not control. The ocular movements were full and the pupils and other cranial nerves were normal except that he disliked lifting his head from the pillow. Jaw-jerk was unaffected. Power, tone, and co-ordination were normal in the limbs. In the arms the tendon reflexes were unaffected, but they were considerably exaggerated in the legs. The right plantar response was flexor, the left extensor. He had marked hyperaesthesia to stroking of the abdomen. There was no sensory loss. Cerebrospinal fluid contained 53 lymphocytes and 1 polymorph per c.mm.; protein, 80 mg. per 100 ml. The condition remained stationary for two days, and then the head movements disappeared. Both plantar responses became flexor by Sept. 2, but the eye movements were still very active, though much less so than formerly.

The euphoria was now less evident, and he continued to improve. He was transferred to an American Army hospital on Sept. 12, and still showed eye movements to the left and vertically.

Orzechowski (1927) described two forms of these spontaneous eye movements: (a) an ataxic dysmetria due to cerebellar disturbance, and (b) opsoclonia due to a lesion in the basal ganglia. Cerebellar signs, though not observed in our case, may sometimes be present. A common mechanism acting through the median longitudinal bundle may explain the synchronization of the head and eye movements, but the precise physiological basis of this unusual condition will be determined only at necropsy.

The incidence of polio-encephalitis in our series was very small, but during the recent epidemic we had the opportunity of seeing a number of cases in other hospitals in which varying degrees and combinations of cranial-nerve palsies have been present; these have not differed from those described previously in this and other countries. Details of these palsies as they occurred in cases in the London area will shortly be published by Sir Allen Daley.

Of the early symptoms of bulbar palsy, difficulty in swallowing and in talking are the commonest. In the following case stridor was the presenting symptom; other atypical features were the relative absence of fever and of changes in the cerebrospinal fluid.

Case 53.—R. A., a girl aged 17, was well until Nov. 5, when she developed a headache which had disappeared by the evening. Next day, she woke up with a cough, which worsened during the morning. About noon she found great difficulty in getting her breath. She was seen in the casualty department and was admitted to the E.N.T. department at once, where on laryngoscopic examination Mr. J. P. Monkhouse found a complete bilateral abductor paralysis. When seen at 3.30 p.m. by one of us her temperature was 99° F. (37.2° C.), pulse 106. There was a marked degree of dyspnoea with inspiratory stridor. The voice was feeble and husky and she had difficulty in swallowing. The palate was paretic. Both sternomastoids and trapezii were weak. The tongue could not be protruded beyond the line of the teeth. The limbs were normal. At 4.30 p.m. tracheotomy was performed. On Nov. 7 the temperature was normal. She complained of numbness of both hands, but no weakness could be detected. Nystagmus was now present. Palate and tongue movements had slightly improved; swallowing was less difficult. Further improvement was noted on the 8th. Cerebrospinal fluid showed: 1 polymorph per c.mm.; protein, 25 mg. per 100 ml. She continued to improve, and on Nov. 21 the tracheotomy wound was closed. Speech was normal apart from a mild nasal quality, and she no longer had difficulty in breathing and swallowing. There remained a mild residual paresis of the palate and of the abductors.

This case illustrates the value of tracheotomy as a life-saving measure in the bulbar form of the disease.

Cerebrospinal Fluid

The majority of our cases showed changes in the cerebrospinal fluid typical of poliomyelitis—namely, a moderate pleocytosis with a slight increase in the protein content. However, in eight out of the 30 cases of poliomyelitis and polio-encephalitis the cell count was less than 10 per c.mm. The average interval between the appearance of meningeal signs and lumbar puncture in these cases was four days. A normal cell count during the febrile stage of the disease may occasionally be met with. It occurred in approximately 5% of Service cases of poliomyelitis in the Middle East and India between 1941 and 1945. A high protein figure may be seen during the first two or three days of the illness, but is more frequent after the first week; it is usually accompanied by a pleocytosis, but occasionally may occur independently of it as in two of our cases. In three cases the protein exceeded 100 mg. per 100 ml. In one of these the fluid was examined on

the eighth day after the appearance of meningeal signs; the cell count was 12 lymphocytes per c.mm., and the protein was 180 mg. per 100 ml. This dissociation between cell count and protein increase has previously been recorded in poliomyelitis, although it is best known in connexion with acute toxic or febrile polyneuritis (Guillain-Barré syndrome).

Differential Diagnosis

The corrected diagnosis in 44 cases admitted as acute poliomyelitis was as follows: tuberculous meningitis, 3; pyogenic meningitis, 2; tonsillitis, 10; pneumonia, 6; typhoid fever, 2; gastro-enteritis, 2; various neurological conditions, 7; miscellaneous, 12.

Tuberculous Meningitis.—Acute poliomyelitis usually attacks individuals in good general health and the prodromal symptoms are of short duration, whereas in a typical case of tuberculous meningitis indefinite symptoms have been present for more than two weeks. A lymphocytic pleocytosis and an increase in the protein of the cerebrospinal fluid are characteristic of both conditions. The estimation of chlorides in the early stage of tuberculous meningitis may occasionally show a figure only slightly below normal—i.e., about 700 mg. per 100 ml. The characteristic fall in chlorides may be delayed and be obtained only if specimens are examined over a week or two (Smith and Daniel, 1947). Lastly, despite modern methods of staining, the tubercle bacillus may be overlooked unless a prolonged search is made. However, in practice this difficulty in diagnosis is only likely to arise initially, since the course of the illness and the continuation of fever for more than a week should suggest tuberculous meningitis rather than poliomyelitis.

Pyogenic Meningitis.—The clinical picture in the early stages of a pyogenic meningitis and of poliomyelitis may be indistinguishable except that as a rule in the former rigors may occur and pyrexia tends to be more pronounced. It is essential that lumbar puncture should be carried out at the earliest possible moment in every case presenting meningeal signs. The general similarity between these two conditions is exemplified by the following case admitted as polio-encephalitis.

Mrs. R. J., aged 36, was well until Aug. 19, when she had severe abdominal pain. She had a miscarriage of twin foetuses 13 weeks old on Aug. 10, but she felt quite well the next day and got out of bed on the 12th. For the next two days she remained in bed with severe backache. On the 15th she was unable to walk upstairs owing to weakness and giddiness, and on the following day her legs were weak and she had occipital headache. On the 17th the temperature was 103° F. (39.4° C.). She had several rigors and vomited on taking solids or fluids. On the 18th she had a rapid onset of deafness. Next day she felt better but complained of marked weakness in the back and legs. On Aug. 22 she was admitted to hospital. The temperature was subnormal and she appeared collapsed, dehydrated, and stuporous. Neck rigidity was marked. Among the positive findings were nystagmus, an exaggerated jaw-jerk, and deafness, which subsequently proved to be of the nerve type. Both plantar responses were extensor. Other findings were auricular fibrillation and mitral stenosis. Blood pressure was 110/80 and there were signs of consolidation of both bases. A diagnosis of pneumococcal meningo-encephalitis was made. Cerebrospinal fluid was purulent: 14,000 cells per c.mm. (96% polymorphs). Culture grew pneumococci. Pneumococci were also grown from the blood and sputum. This patient eventually recovered after systemic and intrathecal penicillin, intravenous sulphamezathine, and digitalization. Recovery of hearing was an impressive feature in the early stages of treatment.

Acute Benign Lymphocytic Meningitis.—There can be no doubt that in this country a form of benign or aseptic meningitis exists which is caused by one or more strains of a virus differing from that originally described by

Armstrong and Lillie (MacCallum, Findlay, and Scott, 1939). Further, it must be realized that this form of virus meningitis may appear in epidemic form. Nevertheless, we are not convinced that up to the present time this condition has been at all common in this country, although it should be added that many cases were seen in Service personnel in the Middle East during the late war. The symptomatology is so similar to that seen in the prodromal and meningitic stages of poliomyelitis that no separate description is called for. Involvement of the central nervous system is rare in benign lymphocytic meningitis. The degree of pleocytosis in the cerebrospinal fluid is, as a general rule, greater than in poliomyelitis, counts of over 500 cells being not uncommon. As in poliomyelitis, polymorphs may predominate initially. Of the other conditions complicated by a lymphocytic meningitis only mumps and glandular fever (mononucleosis) may be mentioned. In both conditions signs of an encephalitis may rarely accompany those of meningitis. In glandular fever the enlargement of glands, the course of the illness, and the result of a Paul-Bunnell test should suggest the diagnosis.

Epidemic and Other Forms of Encephalitis.—Epidemic or lethargic encephalitis has not occurred in epidemic form in this country for over 20 years. Sporadic cases are seldom recognized in the acute stage, the diagnosis being made in retrospect in a case of Parkinsonism. It may be difficult to classify a case of encephalitis showing drowsiness and ocular paresis unless other cranial-nerve or spinal palsies are present. As to the other forms of encephalitis which may occur in this country, precise knowledge is lacking not only of their incidence but also of the viruses responsible for them, but it would appear that such cases are comparatively uncommon. Now that the virus of acute poliomyelitis is well established in Great Britain it should be assumed that it is the usual cause of brain-stem encephalitis accompanied by signs of meningitis unless there is good proof to the contrary. The majority of the cases of meningo-encephalitis which occurred in the London area in 1946, and which were attributed by Jennings (1947) and more recently by Laurent (1947) to an unknown virus, appear to have been similar in most respects to those seen in the recent epidemic of poliomyelitis. In Jennings's series 22 patients showed signs of meningitis or of a meningo-encephalitis. Cranial-nerve palsies and/or pyramidal signs were relatively common. With one exception the cell count in the cerebrospinal fluid was increased. In 13 "influenzal" or "non-meningitic" cases headache and slight stiffness of the neck were the chief features of a brief febrile illness. The cerebrospinal fluid in these cases was normal. These appear to have been abortive cases of poliomyelitis.

Contact Histories

Since the work of Wickman (1907) it has become increasingly clear that abortive cases of poliomyelitis and healthy carriers of the virus are of more importance in the spread of the disease than the paralytic case. Reference has already been made to the symptomatology of the abortive case, which differs little from the prodromata of the disease itself except that catarrhal or intestinal symptoms may constitute the whole illness. The significance of minor illnesses preceding or accompanying an outbreak of poliomyelitis can be firmly established only by proving or disproving the existence of the virus in the pharynx or faeces of such patients by means of a passage to monkeys or cotton-rats. Unfortunately, facilities in this country for this type of work are at present extremely limited.

In an attempt to determine a possible source of infection patients and relatives were carefully questioned concerning

the occurrence of minor illnesses in the family, at school, and among friends or fellow workers. In 19 of 54 paralytic and non-paralytic cases a positive history was obtained. In five cases symptoms were those of an upper respiratory or intestinal infection. In the remaining 14 cases constitutional symptoms were followed by headache severe enough to suggest a mild meningitis. Eight of these were admitted to the Middlesex or some other hospital as suspect cases of poliomyelitis; in three of them there was a pleocytosis in the cerebrospinal fluid.

The following is an example of minor illnesses, suggestive of abortive attacks, in a family of which one member suffered from a non-paralytic form of the disease.

Case 50.—R. J. W., aged 11½. Between July 22 and 29, 1947, he suffered from sore throat, mild headache, and fever. At the beginning of August he was apparently well, though pettish, but on the 16th he was again unwell and feverish for a day. He complained of aching in the shoulders on the 23rd, and on the following day of stiffness in the neck. On the afternoon of the 24th his temperature was 101° F. (38.3° C.). Next day the shoulder-ache was no longer present, but he had mild headache, and was admitted to hospital; temperature normal. The only abnormal sign was a mild degree of neck stiffness. The cerebrospinal fluid showed 28 lymphocytes per c.mm.; protein, 30 mg. per 100 ml. The patient had one brother and two sisters, aged 10, 7, and 4 respectively. Between Aug. 7 and 12 both sisters were in bed with a temperature of 102° F. (38.9° C.). The elder girl had headache; the younger had no headache, and fever lasted only one day. Between Aug. 17 and 20 the elder girl had a recurrence of headache and complained of stiffness of the neck; a temperature of 100° F. (37.8° C.) was recorded on one of these days. The father had fever and headache between Aug. 13 and 16. The charwoman's child, aged 8, a contact of the children, was unwell with fever between the 14th and 21st. The mother stated that several other children in the same road, as well as their parents, had "stomach-aches and slight fever" about this time.

The following family history is of interest as it would seem possible that the mother and son were both infected by the daughter.

Case 2.—Miss A. L., aged 24, and her mother were on holiday at the seaside from Aug. 18 to 25. Her brother (Case 1) and her father remained at home in London. On the 18th Miss A. L. suffered from diarrhoea and backache, which lasted for 36 hours. On her return to London (Aug. 25) she complained of aching pain in the lower part of the back and both loins; she was drowsy and slightly jaundiced. The temperature was normal. These symptoms lessened over the next few days. On Sept. 3 a "rheumatic" type of pain in the back spread to the right shoulder and down to the hips, followed next day by headache. On the 8th she noticed stiffness of the left leg on getting out of bed, and on the following day there was weakness of both legs. On admission to hospital on the 11th she showed signs of mild paralysis in the lower limbs. On Aug. 25 her mother "felt rotten" and had fever for one day only.

Case 1.—M. L., aged 21, brother of Case 2. On Aug. 30, five days after his mother's and sister's return from holiday, he complained of pain across the chest, backache, and abdominal discomfort. Temperature was 100° F. (37.8° C.). A mild degree of jaundice was present. By Sept. 4 he felt better, but the next day he noticed stiffness of the neck, slight sore throat, followed in a few hours by severe headache and profuse sweating. On the 6th he felt a dull ache in the back and both legs. Headache had disappeared by the 9th, but his neck was still stiff and for the first time he noticed weakness in both arms and legs. On admission on Sept. 11 a mild degree of paralysis in the left arm and both legs was noted.

Examples of two nurses contracting the disease from patients they had nursed have previously been given (McAlpine, 1945). One of our cases is also interesting in this connexion.

Case 5.—This patient (W. A.) was in all probability the source of infection in a nurse. The diagnosis of poliomyelitis was not

made until 48 hours after his admission; during this period he was nursed without precautions. Nurse B. C. nursed him from the day of his admission (July 21) to July 25. Three days later she had a dental extraction but was well until the 29th, when she complained of pain behind the sternum and of a slight aching pain in the left wrist. On July 31 the headache was marked: temperature 100° F. (37.8° C.); cerebrospinal fluid: 18 lymphocytes and 1 polymorph per c.mm.; protein, 30 mg. per 100 ml. Slight weakness of extensors of wrist and fingers of left hand was noted on Aug. 1.

Nothing is known of the factors that render a person susceptible to an attack of poliomyelitis. The concept that immunity to the disease is conferred in childhood by a subinfective dose of the virus is not entirely borne out by recent facts (Van Riper, 1947). Sabin (1947) has pointed out that there is evidence to suggest that there may be no relationship between an attack of the disease or the passing of an epidemic through a community and the appearance of a neutralizing antibody as tested for against a single standard strain of virus, and occasionally even against some more recently isolated strains. Burnet and Jackson (1939) have stated that poliomyelitis antibody is not a result of exposure to or infection by the virus of epidemic poliomyelitis. During the past quarter-century there has been a definite rise in the age incidence of the disease in all poliomyelitis countries. Furthermore, the disease and the mortality rate have both been more severe in adolescence and in adults than in children. Such information as is available about the recent epidemic in this country would seem to confirm this tendency. One of our cases—a doctor's wife—apparently passed the infection to her brother-in-law, aged 30, but failed to infect her own two children, with whom she was in much closer contact, despite the fact that one, aged 3, had had a dental extraction during the time she showed prodromal symptoms and the other child, aged 5, had had a tonsillectomy a few days before.

Two of our cases gave a previous history of close contact with poliomyelitis in the early febrile and paralytic stages of the disease, yet both cases remained well at that time but contracted the disease in the recent epidemic.

Case 23.—Mrs. C., aged 21. During September, 1938, when 12 years of age, she was on holiday and sharing a bed with her sister. The sister developed headache, high temperature, and general malaise, followed by severe paralysis of her right leg. A diagnosis of poliomyelitis was made. Mrs. C. continued to share her sister's bed until the latter was removed to hospital. She herself remained perfectly well, but contracted a mild attack of poliomyelitis on Sept. 2, 1947.

Case 28.—Captain R. P., aged 32. In 1945 in Washington, U.S.A., he shared a room with another man, aged 28, who developed headache, aching all over, and stiff neck, followed by paralysis of the small muscles of his right hand. He was removed to hospital as a case of acute poliomyelitis. For two days he was fed and cared for by Captain R. P., who remained well at that time but contracted polio-encephalitis on Aug. 21, 1947.

Thus it would seem that if a child or an adult is exposed to the virus and does not develop paralysis, immunity either may not occur or may be short-lived. Judging by recent reports, second attacks of the disease are not so rare as were formerly supposed. In Alves and Pugh's (1947) case eight and a half years elapsed between the attacks. The three examples recently reported by Lipscomb (1947) concerned officers in India. In one of these the second attack occurred within six months of the first. A similar short interval was recorded by Cunningham (1947). Variations in the strains and virulence of the virus may in part explain second attacks of poliomyelitis, although it is possible that unknown factors underlying susceptibility may be of equal importance.

Summary

Symptomatology in 54 cases of acute poliomyelitis is discussed—24 of the cases belonged to the non-paralytic form. There were no deaths.

Several case histories are given, including an example of a rare ocular complication (opsoclonia), as well as an unusual case of bulbar paralysis successfully treated by tracheotomy.

Attention is drawn to certain atypical features in the cerebrospinal fluid.

In nearly one-third of the proved cases contact histories suggested the probability of an abortive attack of the disease in other members of the family or in friends.

In this country the virus of acute poliomyelitis should now be regarded as the usual cause of a benign type of lymphocytic meningitis and of a brain-stem encephalitis accompanied by meningitis.

A brief reference is made to the problem of immunity.

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POLIOMYELITIS

THE PRE-PARALYTIC STAGE, AND THE EFFECT OF PHYSICAL ACTIVITY ON THE SEVERITY OF PARALYSIS

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[PRELIMINARY REPORT]

The current epidemic of poliomyelitis provides an opportunity of studying the clinical features of this disease which is fortunately seldom available in this country. Though a great variety of symptoms have been reported in the present epidemic (Kelleher, 1947) many students of other epidemics have insisted that the pre-paralytic symptoms and signs in poliomyelitis are specific, and that it is possible to diagnose the disease before paralysis develops (Kauders, 1938; Gordon, 1932; Aycok and Luther, 1928; Adamson and Dubo, 1942).

A history of great physical exertion preceding the onset of severe paralysis has often evoked comment in cases of poliomyelitis (de Rudder and Peterson, 1938; Tucker, 1941), but no attempt (so far as I am aware) has been made to investigate this aspect of the problem statistically.

It is generally agreed that a study of the pre-paralytic symptoms gives no clue to whether or not paralysis will develop. Further, the changes in the cerebrospinal fluid in the pre-paralytic stage are equally abnormal whether or not paralysis subsequently develops (Collier, 1927; Adamson and Dubo, 1942).

It is not surprising, therefore, that in recent years attention has been directed to the physiological state of the