SOME DIAGNOSTIC PROBLEMS SURROUNDING POLIOMYELITIS*

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In this paper reference is made to three problems in the recognition of poliomyelitis that are topical. Firstly, the diagnosis of non-paralytic poliomyelitis and its relationship to similar syndromes mainly of virus origin and often referred to as "aseptic meningitis"; secondly, the need to distinguish the paresis of so-called "benign myalgic encephalomyelitis" from paralytic poliomyelitis; thirdly, the existence of polioencephalitis unaccompanied by paralysis.

Recognition of Non-paralytic Poliomyelitis

The term "non-paralytic poliomyelitis" refers to the so-called major illness or nervous phase of poliomyelitis when this develops without the supervention of paralysis. In outbreaks of poliomyelitis one case of this type occurs for every two or three cases with paralysis. Looked at in another way, non-paralytic poliomyelitis is a mild meningitis of viral origin and is an example of a group of similar illnesses to which the term "aseptic meningitis" is often applied, especially when the aetiology is in doubt.

Viral meningitis cases have a cerebrospinal fluid of clear or near-clear appearance with a pleocytosis often between 10 and 500 or so cells per c.mm., but sometimes reaching 2,000 or over, with very variable proportions of polynuclear cells and lymphocytes, but over a series often leaning to lymphocytic preponderance. Perhaps half the cases show an increase of C.S.F. protein usually to less than 100 mg. per 100 ml., with normal sugar and chloride values.

Although sporadic cases of aseptic meningitis may be due to poliomyelitis infection they may also be due to the virus of mumps or of benign lymphocytic choriomeningitis, or may occasionally accompany glandular fever. If, on the other hand, they occur in large numbers, with an absence or near absence of paralytic cases, they are more likely to be due to one of the Coxsackie viruses or, as has been appreciated more recently, one of the so-called E.C.H.O. viruses, infection by which may sometimes be accompanied by a rubelliform rash (*British Medical Journal*, 1957). These two virus groups, together with the poliomyelitis viruses, are all very similar in their physical properties and form a larger group, to which the term "enteroviruses" is beginning to be applied (*British Medical Journal*, 1958). Whilst the virologist can assist by performing serological tests for mumps and lymphocytic choriomeningitis, and by isolating poliomyelitis virus from excreta, other relevant virological investigations are not yet available routinely.

Certain non-viral conditions need to be distinguished from aseptic meningitis of viral origin. In anicteric leptospiral meningitis, usually due to *Leptospira canicola* infection, there are similar findings in the C.S.F., but the correct diagnosis is suggested by the usual occurrence of conjunctival suffusion and some degree of nephritis (Ramsay, 1955); laboratory investigations, especially

for the development of antibodies, may confirm suspicion. It is especially important to distinguish aseptic meningitis from tuberculous meningitis with its steady clinical deterioration and increasing drowsiness, cranial palsies in advanced cases, wider C.S.F. changes, including the presence of acid-fast bacilli, and the frequent concurrence of other tuberculous manifestations. Aseptic meningitis may occasionally have to be distinguished from post-infection encephalitis, partially treated bacterial meningitis, aseptic meningeal reaction to an adjacent septic focus such as extradural abscess or venous sinus thrombosis following mastoid infection, and the rare acute syphilitic meningitis. In some districts it may also be necessary to bear in mind tick-borne virus infection of the Russian spring/summer encephalitis/louping-ill group, illness from which may be non-paralytic or paralytic (Likar and Dane, 1958).

The diagnosis of non-paralytic poliomyelitis being so often uncertain, many cases of aseptic meningitis due to viruses other than poliomyelitis have in the past undoubtedly been included in the poliomyelitis figures; only paralytic cases should therefore be counted in comparing rates (Medical Officers of Schools Association, 1948), and since 1950 this has been made possible by requiring notifications of poliomyelitis to state whether the disease is of the paralytic or non-paralytic form. It is also apparent that in situations where the aetiology of a sporadic case of aseptic meningitis is completely uncertain it is unfortunate if a speculative label of "non-paralytic poliomyelitis" becomes attached to it with its possibilities of needless public alarm.

Benign Myalgic Encephalomyelitis

Reference may now be made to "benign myalgic encephalomyelitis," which is the term suggested by the *Lancet* (1956) to denote a type of neurological disease which appears to have occurred in widely different parts of the world in recent years and which does not classify under any of the previously established entities. It is "benign" in the sense that there seems to be no appreciable mortality (although in some cases sequelae may persist) and "myalgic" in the sense that muscle pain is frequent rather than invariable. Other terms which have been used are "epidemic myalgic encephalomyelitis," "acute encephalomyelitis—cause unknown," "Icelandic disease," and in the Royal Free Hospital it was nicknamed "the pest."

In Coventry attention was aroused in the summer of 1953 when about a quarter of the staff (13 in number) coming into contact with poliomyelitis patients in the infectious disease unit in Coventry developed an illness in which all but one of those affected showed some degree of paresis often associated with a peculiar jerking quality of volitional movement (Macrae and Galpine, 1954). Slight pyrexia occurred in about half the cases, also headache, drowsiness, and discomfort, pain, and even rigidity in the neck and back. Tendon reflexes, though largely unaffected, were occasionally disturbed, with a tendency to early depression and later exaggeration. Muscular aches and paraesthesiae were complained of, and four of the affected staff had areas of hypoaesthesia or hyperaesthesia unrelated to segmental or peripheral nerve distribution. Disturbance of micturition occurred in four cases. In convalescence several complained of inability to concentrate mentally. The cerebrospinal fluids gave almost wholly normal

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findings. Wasting was either absent or, if present, not commensurate with the paralysis, from which there was a marked tendency to recovery earlier and more completely than would have been expected in poliomyelitis.

Extensive laboratory investigations failed to reveal any evidence of poliomyelitic infection, but electromyographic abnormalities with a discrete pattern of action potentials on attempted voluntary movement were found in three of the staff cases (J. Shulman, 1953, personal communication). Amongst a considerable number of patients admitted from Coventry and district as cases of paralytic poliomyelitis in the same season, an increasing proportion, most of them female, showed early recovery from their paralysis and a normal C.S.F. In many of the patients, therefore, the illness paralleled that in the nurses.

A similar outbreak affected some of the nursing staff of the Middlesex Hospital in 1952 (Acheson, 1954), but the largest outbreak recorded in this country occurred in the Royal Free Hospital in 1955, when nearly 300 members of the staff were affected. Malaise, lassitude, sore throat, enlarged cervical glands, and gastro-intestinal disturbances occurred at onset, and later symptoms included cerebral irritation, depression, cranial nerve disturbances (including vertigo), motor and sensory disturbances in the limbs, minor disturbance of reflexes, and sometimes intense muscle spasm (Dimsdale, 1957). As in Coventry, electromyographic abnormalities were found, and these were characterized on volitional movement by a reduction in motor unit potentials that individually were often polyphasic; in addition a peculiar grouping of potentials was seen, constituting a novel feature of obscure origin. The electromyographic pattern suggested involvement of the motor unit at cord level without, however, the electrodiagnostic signs of lower motor neurone degeneration common to such diseases as syringomyelia and anterior poliomyelitis (Richardson, 1956). Sporadic cases of a similar illness admitted in 1955 from the local population to the infectious diseases unit of the Royal Free Hospital formed the background to the staff illness (Ramsay and O'Sullivan, 1956), and further cases were admitted in 1956 and 1957 as reported by Ramsay (1957), who stressed the occurrence of sequelae, sometimes neurological, but characteristically including proneness to fatigue and emotional lability.

In Coventry seven further cases of the syndrome were admitted in 1956 (Galpine and Brady, 1957) and three more were seen in 1957 during a time of high poliomyelitis incidence locally. The most typical of the latter was the following case.

A woman of 29 had backache, general aches, and very marked lassitude for four days, followed by a feeling of instability and aching in her legs, worse after exertion. Her doctor found marked general weakness in her left leg, with normal reflexes and a temperature of 99° F. (37.2° C.). In hospital her left hamstrings were found to be painful on stretching and there was diffuse tenderness in the muscles of the left leg. This leg could with difficulty be lifted clear of the bed and then only after a characteristic pause for concentration. Voluntary movements of the left foot were very weak and restricted, and the left ankle-jerk appeared slightly reduced. Blunting of sensation to touch and pain and to vibration and position was found in the left ankle and foot. She complained of flickering sensations in the muscles of her weak leg. There was also slight weakness and aching on exertion in the left upper arm. The C.S.F. was normal. The patient was discharged home to have

out-patient physiotherapy after two and a half weeks in hospital, by which time sensation had very largely recovered and she walked fairly well, although dragging her left foot if she attempted to hurry. When seen seven months later her disability was minimal, but there was still slight weakness in the left foot and she complained of early tiring and aching in the left leg with a tendency for the left foot to turn in at such times.

Whilst the aetiology and pathology of this condition are, as yet, unknown, an underlying consistency exists in the versatile clinical picture and there seems good reason to suppose that it is a specific type of encephalomyelitis. Cases of this type, especially if occurring sporadically in the general population, may attract other and more familiar labels—for example, "paralytic poliomyelitis" and "hysteria."

Polioencephalitis

Finally, there is the problem of polioencephalitis. Although paralytic poliomyelitis has a reputation for not usually affecting cerebration, the occurrence of widespread paralysis in a patient, especially if including bulbar paralysis, is not infrequently accompanied by cerebral disturbance, either biochemical in origin and associated with respiratory inadequacy—a very important group—or truly encephalitic. In a description of his own attack of paralytic poliomyelitis made by a medical author (Personal Papers, 1953) the section on mental state includes the following:

"I was much troubled by flight of ideas; a succession of confused and unrelated thoughts sped through my mind so rapidly that I was unable to seize and hold any one of them. This experience was particularly frustrating and annoying at night, preventing any sustained thought or contemplation during many sleepless hours. Nevertheless it had its compensations, for the serious nature of my illness caused me relatively little apprehension during the early days. . . . During this period of confusion I had fantastic daydreams. My favourite was one in which I lay couched in my own medulla watching the outfall of nerve-cells from my nucleus ambiguus, an experience characterized by the complete absence of any affective element. For several days I was unable to appreciate the size and arrangement of my cubicle: at night, for instance, it expanded to an enormous room in a mythical house on the South Downs, or contracted to the box-like compartment where the stable-lad sleeps at my daughter's riding-school. In the confusion resulting from this uncertainty as to my whereabouts I was prone to get out of bed at night. . . . It was . . . about a month before my mental state was completely normal."

Whilst the foregoing may be presumed to refer to the encephalitic component of an attack of paralytic poliomyelitis classifiable and notifiable as such, the position is less clear if encephalitis occurs without paralysis but along with evidence—epidemiological or virological—that the poliomyelitis virus is at work. This kind of thing used to be notifiable separately as polioencephalitis before 1949, but in that year regulations were made abolishing this and including the condition in the term poliomyelitis. This is because it was felt that if polioencephalitis exists as a separate disease it is difficult to describe and to distinguish from other forms of encephalitis. There have, however, been a few reports of the isolation of poliomyelitis virus from cases diagnosable as encephalitis. For example, evidence of infection with poliomyelitis virus was present in seven out of nine cases of encephalitis-like illness seen during a poliomyelitis epidemic in Sheffield in 1955 (Snell *et al.*, 1957).

In Coventry in 1957, a year in which 141 paralytic cases were admitted to the local isolation unit, six cases of acute encephalitis-like illness were admitted at the height of the poliomyelitis incidence, with a good recovery in each instance. In two of them the faeces were examined for poliomyelitis virus, and type 1 virus was found in both. All six were pyrexial, with meningeal signs in all except one and C.S.F. changes consistent with poliomyelitis in all save one other. Two of these patients were girls aged 8 and 3 years who presented fully conscious but with ataxia—in one of them limited to the left arm and leg. Another girl of 3 was also conscious but exceedingly irritable, and showed violent generalized clonic spasms especially affecting her eyes, face, and head. Any attempt to sit her up caused her to scream and the movements to increase, but when she slept the spasms disappeared. Two boys aged 6 and 10 presented in stupor; one of them lay quietly on his side, but if disturbed resisted violently with extreme hyperpnoea and the development of carpopedal spasm in a matter of minutes—which passed off just as quickly when he was left alone. The sixth case was that of a business man of 35 who had episodes of confusion in which he performed ridiculous acts—for example, trying to post a bundle of his belongings in the ward kitchen. It is a reasonable clinical supposition that “non-paralytic” cases of the above type occurring in the midst of a poliomyelitis epidemic are in all probability examples of “polioencephalitis.”

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St. Luke's Printing Works in Old Street, London, which was built as a hospital in 1784, was sold in January by the Bank of England for over £1m. Charles Dickens, who visited the hospital in 1852 (see *Journal*, 1951, 2, 1518), describes how it was founded as a new hospital for the insane by “half a dozen gentlemen” who were perturbed to see that the hospital at Bethlem was incapable of receiving and maintaining the great number of “melancholy objects of this sort who apply for relief.” The hospital's first home was a building called The Foundry (in what is now Tabernacle Street), where cannon had originally been cast and where later John Wesley preached. In 1784 George Dance, the architect, designed the new St. Luke's, the building in Old Street, which opened two years later. Since Dance's first important work had been the rebuilding of Newgate Prison it is hardly surprising that the new hospital had a massive and rather forbidding appearance. The Old Street building ceased to be a hospital in 1917, but during its long life it admitted 25,569 patients, of whom 11,077 were discharged cured. The hospital's name and purpose is perpetuated in the St. Luke's Woodside Branch of the Middlesex Hospital. In the nineteenth century the hospital's visitors included, besides Charles Dickens, Charles Lamb, who was one of its governors, and Mrs. Elizabeth Fry.

EPIDEMIOLOGY OF POLIOMYELITIS*

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It is now well established that there are three types of poliovirus—types 1, 2, and 3—and that type 1 is responsible for about 80% of all paralytic infections. Although these types are immunologically distinct there is evidence which indicates a degree of relationship between them, and this may be of importance clinically. It must be remembered that paralysis is not a common outcome of infection with a poliovirus, for, as is well known, infections with all types of poliovirus are a common experience; usually there is an alimentary infection which is clinically either inapparent or unrecognizable. It has been shown that infections at a clinical or subclinical level generally result in resistance to further attacks.

Transmission of the Virus

The virus is spread by person-to-person contact, and children are probably the most important disseminators of virus in the community. The reason for this is that virus multiplies better in the alimentary tract of children than of older persons, and the children therefore excrete much greater quantities of virus and are thus more infectious.

There is no evidence that animals or birds enter into the natural history of the disease. Recently there was a report from the West of Scotland which suggested that budgerigars might play a part in the transmission of the virus. Dr. D. S. Dane and I have so far been unable to infect budgerigars with any of 12 different strains of poliovirus.

After infection, virus is found in the throat secretions of a proportion of individuals for a few days and in the faeces for weeks or months. In addition to the fact that children excrete more virus than adults we have also some evidence suggesting that there is a family pattern of virus excretion. Thus the children of some families seem to excrete more virus than those of other families. There appear to be “hot” families in whom the virus multiplies well and “cold” families in whom virus multiplies poorly. This familial susceptibility may be of importance in the pathogenesis of the infection and also in the spread of virus in the community. The precursor of paralysis is viraemia, and it may be that, in those families in whom virus multiplies well because of its greater multiplication in the gut, there is more chance of the virus getting into the blood stream and thence to target areas in the C.N.S. where the damage is done. This does not mean, of course, that everyone who gets viraemia will become paralysed. The time relationship between the pathogenetic events and the clinical picture is shown in Fig. 1.

This represents the sequel of events in a paralysed child. Essentially the same picture occurs in the asymptomatic infection but with little or no invasion of the C.N.S.

If a child becomes infected it is a common experience for all members of the family to be also infected. This occurs whether or not any of the family is showing signs or symptoms of infection.

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