be involved. This likelihood was mentioned by MacAlpine (1947) after the 1947 period of poliomyelitis had included similar cases; our present case, though affording no proof, does give this concept further support.

Summary

A fatal case of infection by the virus of poliomyelitis has been described.

Although the case clinically showed a mainly encephalitic and brain-stem involvement the cerebral changes were merely those of cortical congestion, and the pons showed only a mild inflammatory reaction. The spinal cord, on the other hand, showed gross and widespread destruction of anterior horn cells. No really satisfactory histological explanation was forthcoming from either cord or brain to account for the definite clinical evidence of upper motor neurone involvement. Congestion, oedema, or very slight early changes in these neurones might be adduced as causes of the signs. Inoculation of the nervous tissues of this case in monkeys resulted in symptoms and histological lesions closely akin to those in the human subject.

This seemingly encephalitic case was thus proved to be due to an infection by poliomyelitis virus, and its features are compared briefly with those shown by possible atypical cases of the same infection seen in the same district two years previously.

Our thanks are due to Miss M. H. Shaw for the photomicrographs of the sections.

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FATAL CASE OF ACUTE ENCEPHALOMYELITIS DUE TO POLIOMYELITIS VIRUS

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The number of instances in which poliomyelitis virus affects the fore-brain and brain-stem seems to vary in different epidemics. Reports dealing with the 1947 epidemic in this country indicated a higher proportion than usual of cerebral and bulbar lesions with or without spinal involvement (Kelleher, 1947; MacAlpine *et al.*, 1947). The picture has been confused by the occurrence, just before the poliomyelitis outbreak of 1947, of minor and localized outbreaks of virus encephalomyelitis recorded by Jennings (1947) and Laurent (1947) and regarded by them as a disease or group of diseases in which no relation to poliomyelitis was probable. On the other hand both Critchley (1948) and MacAlpine *et al.* (1947) believe that the virus responsible was probably identical with that of acute poliomyelitis.

Undoubtedly the lack of facilities for the isolation and identification of virus in this country has accentuated the confusion, and, whereas Toomey (1941) recognizes eight clinical types of poliomyelitis, doubts about the existence of types other than spinal and brain-stem are held by many authorities in this country—for example, Walshe (1947), who states: "It is probable, however, that only the spinal type and a few cases of brain-stem type really belong to this [poliomyelitis] infection." Baker and his colleagues (1947), in a comprehensive review in which the word "bulbar" has been used "with some degree of licence" to denote involvement of the entire brain-stem, including the medulla, pons, and mid-brain, present an acceptable classification of all forms of bulbar poliomyelitis. They include a "diffuse encephalitic" and a "focal encephalitic" group, and, in connexion with the latter, state that aphasic disorders have been reported rather infrequently. They quote one case of visual verbal agnosia from the literature.

The following record is a description of a case of acute polio-encephalomyelitis in which during life it was not possible to make a clear clinical distinction between poliomyelitis or other virus aetiology, but in which histological evidence and ultimately virus isolation established the aetiological agent.

Case Report

A man aged 25 with a record of excellent health was admitted to the Western Hospital on Oct. 12, 1947, with a history of illness which had begun six days earlier with a sense of fatigue and malaise, followed 48 hours later by headache and giddiness. On the following day (Oct. 9) he vomited once and, because of increasing malaise and the onset of giddiness, tremors, and blurring of vision, had to take to bed. On the fourth day after the onset feverishness and thirst were pronounced. He was restless and unable to sleep, and had difficulty in hearing. His condition rapidly worsened, until on Oct. 12 he was found by his relatives to be unable to move, to be generally rigid rather than weak or flaccid, and, though conscious, to be unable to speak.

On admission at 5 p.m. on the same day he had a temperature of 101° F. (38.3° C.) and a pulse rate of 82. He was conscious, and there was some cyanosis of the extremities. He was unable to speak. A fine tremor of the outstretched hands was noted. There was moderate neck rigidity and hamstring spasm. He moved his limbs very slowly. Movements were stiff, jerky, and apparently uncoordinated, and passive movements of the limbs further revealed the general rigidity and tonic spasm. The facial muscles were involved in the general hypertonicity, and commands were obeyed incompletely and with great difficulty and delay in execution. There was fine nystagmus in all directions but most pronounced to the right. Apart from a possible weakness of the left sternomastoid muscle there were no obvious cranial-nerve palsies. All tendon reflexes were present but depressed, and the abdominal reflexes were normal. There was a doubtful Babinski sign on the right; the left plantar reflex was flexor. The respiratory muscles were unaffected and the pharynx functioned. There was no retention of urine, but he was incontinent.

A few hours later the tendon reflexes in the right lower extremity were very sluggish, but apart from the dysfunction, which was apparently due to tonic spasm, there was still no apparent paresis or paralysis; the dysarthria might also be ascribed to tonic spasm, although possibly it may have been a true motor aphasia. Lumbar puncture showed a clear fluid containing 18 lymphocytes per c.mm. and 55 mg. of protein per 100 ml.; colloidal gold test, 0111210000; Wassermann reaction negative.

On the following day (Oct. 13) there was little change, but some weakness of the right lower extremity, with greatly diminished reflexes, was noted. Consciousness appeared to be clouding and ultimately all response to stimuli failed. The tonic rigidity persisted and a fine tremor of the upper lids was

noticeable and persistent. A cinematograph record of the facies exists (Ministry of Health, 1948). A consultant neurologist made a provisional diagnosis of acute disseminated encephalomyelitis. By the next day (Oct. 14) there was marked deterioration, with a rising temperature and a tendency to accumulation of buccal secretions, but still no respiratory muscle involvement. Death, apparently due to bulbar failure, occurred rather suddenly the same evening.

Necropsy on Oct. 15 revealed a little focal haemorrhagic bronchopneumonia in the congested lungs, cloudy swelling and congestion of the liver and kidneys, and oedema and congestion of the spleen (all confirmed microscopically). The heart was normal, the air passages were clear, the pancreas and stomach were congested, and the urinary bladder was dilated. There was moderate oedema and congestion of the somewhat swollen brain and spinal cord.

Microscopical examination showed very severe perivascular "cuffing," mainly with lymphocytoid cells, and foci of acute inflammation and degeneration of parenchyma, in places almost purulent, in the basal ganglia, mid-brain, pons, and medulla—most severe in the mid-brain. The cerebral cortex in the left prefrontal, right Rolandic, and left occipital regions showed only oedema and congestion. The third and fourth cervical, fourth and eighth thoracic, and second lumbar segments of the spinal cord showed inflammation and degeneration of the grey matter, especially of the anterior horns, in which a considerable number of neurones were destroyed, and to a smaller extent of the white matter.

The findings were characteristic of acute polio-encephalomyelitis (A.B.B.).

The histological report was submitted to the consulting neurologist (Dr. T. Rowland Hill), who remarked: "A case of fulminating polio-encephalomyelitis in which, before death, it was not possible to demonstrate involvement of lower brain-stem motor nuclei. There were signs of severe mid-brain lesions in that there were tonic fits of decerebrate rigidity and marked nystagmus. There was also a degree of flaccid weakness of the right lower extremity with markedly diminished reflexes. Bulbar palsy developed before death."

Specimens of the brain and cord were stored at the Virus Reference Laboratory, Colindale, in glycerol-saline for ten months at -10°C . A 10% suspension of cord and medulla was then inoculated intracerebrally into one rhesus monkey under ether anaesthesia. The monkey developed typical signs of poliomyelitis ten days later and was killed with chloroform. Histological examination of the central nervous system showed lesions pathognomonic of poliomyelitis (F.O. MacC.).

Summary

A case is recorded of acute encephalomyelitis of clinically undetermined aetiology, in which histological findings and virus isolation established the virus of poliomyelitis as the cause.

Our thanks are due to Dr. W. H. Bradley, of the Ministry of Health, for helpful suggestions.

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Hospital matrons and nurses from Belgium, Canada, Cyprus, Denmark, Egypt, Finland, France, Greece, Italy, the Netherlands, Norway, and the U.S.A. have recently attended a two weeks' course on nursing arranged by the British Council in Edinburgh. Sir Andrew Davidson, Chief Medical Officer of the Department of Health for Scotland, gave the opening lecture.

STREPTOMYCIN IN DEVELOPMENT OF HYDROCEPHALUS IN TUBERCULOUS MENINGITIS*

BY

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[WITH PHOTOGRAVURE PLATE]

Since the introduction of streptomycin in the treatment of tuberculous meningitis several reports of hydrocephalus have appeared. Flory and his co-workers (1947) describe a case of relapse of tuberculous meningitis after cessation of streptomycin treatment in which an internal hydrocephalus was discovered at necropsy. Baggenstoss *et al.* (1947) record two fatal cases of tuberculous meningitis treated with intramuscular and subcutaneous streptomycin in which occurred heavy exudate at the base of the brain and moderate internal hydrocephalus. Wright and Rees (1948) report three cases of severe hydrocephalus associated with thick basal exudate after treatment with intramuscular and intrathecal streptomycin for at least eight weeks. Smith *et al.* (1948) describe two fatal cases with gross hydrocephalus after intramuscular, intrathecal, and intraventricular streptomycin; in two subjects who survived, ventriculography before treatment revealed some dilatation of the ventricles. In all of these cases exudate in the basal cisternae was found to be most intense.

An M.R.C. report (1948) includes 46 instances of hydrocephalus among 53 necropsies on cases of tuberculous meningitis treated with streptomycin by various routes. These studies led me, whilst interested in experimental hydrocephalus, to investigate the histological changes in the basal meninges in a small series of cases of tuberculous meningitis with the object of finding out if the administration of streptomycin had modified the lesion in any way that might lead to hydrocephalus.

Materials and Methods

The leptomeninges from the undersurface of the pons were available for histological examination from eight cases of tuberculous meningitis; six of these were hydrocephalic, two were not. Seven had been treated with intramuscular and intrathecal streptomycin, with or without intraventricular streptomycin, for periods varying from 12 to 231 days, while only one (hydrocephalic) received no antibiotic treatment. Fig. A shows the dilatation of the anterior horns of the lateral ventricles in the frontal section of the brain of a case of tuberculous meningitis treated with streptomycin.

FIG. A.—Hydrocephalus in an infant following tuberculous meningitis treated with streptomycin.

The leptomeninges, with a piece of brain tissue, were cut out from the undersurface of the pons and fixed in 10%

*Part of a thesis submitted to the University of London for the Doctorate of Philosophy.