talipes calcaneus on both sides. She made no attempt to walk, but could stand when supported with both legs adducted. Occasionally restless movements of all limbs were observed. The knee and ankle jerks were brisk, right greater than left, and plantar reflexes indefinite. Heart normal, pulse rate 76, and blood pressure 105/70.

Commentary

I am not aware of any previous record of a case of Sturge-Kalischer-Weber syndrome showing bilateral intracranial calcification. Cases of extensive cutaneous naevi on both sides of the body, usually more continuous on one side and patchy on the other, in association with Jacksonian epilepsy are occasionally encountered. One such case under my care is described in an addendum to Parkes Weber's paper of 1929; in this case no intracranial calcification was demonstrable on x-ray examination. In the case now reported the calcification is more extensive and more intense on the one side (left cerebral hemisphere) than on the other, and this corresponds to definite hemiplegic signs in the spasticity of the right arm and leg and the increased deep reflexes. Similarly, the epileptiform attacks usually begin on this (right) side, and even when more generalized the right side is the more affected. The blood found in the cerebrospinal fluid at the first lumbar puncture at the age of 7½ months was almost certainly due to contamination at the time of lumbar puncture (no doubt resulting from intense local vascularity) rather than to a recent sub-arachnoid haemorrhage from the intracranial angioma, as the red cells were quite fresh and there was neither haemolysis nor xanthochromia. Then again, the cerebrospinal fluid examined at the age of 3½ years was quite normal.

The calcification of the meningeal angioma evidently began between the ages of 7½ months and 1½ years, since no x-ray abnormality was seen at the earlier age, but a year later intracranial calcification was demonstrated on both sides, the more profound lesion of the left cerebral hemisphere showing the more extensive and denser calcification.

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Presiding at a conference of the Tuberculosis Association in Belfast, Dr. Frederick Heaf, Director of the London County Council tuberculosis services, said the organization in Ulster for dealing with the disease was the envy of the profession in England. Dr. Brice Clarke, Director of the Northern Ireland Tuberculosis Authority, informed the conference that there were 1,200 beds in tuberculosis hospitals in Ulster, as well as a number of beds in general hospitals for tuberculosis patients. Seven hundred additional beds were planned and 250 of them would be ready within the next twelve or eighteen months. A children's orthopaedic hospital was being built and an infants' and children's hospital for pulmonary cases was being developed. It was hoped to have five or six clinics in Belfast and thirteen in other parts of Ulster. In addition, a contract had been placed for 100 rest chalets. Since June, 1945, 93,000 persons had been examined by the mass radiography unit in Belfast and a second unit had been ordered.

POLIO-ENCEPHALITIS

BY

E. G. BREWIS, M.D., M.R.C.P., D.P.H.

AND

C. NEUBAUER, M.D.

(From the City Hospital for Infectious Diseases,
Newcastle-upon-Tyne)

The 1947 epidemic of poliomyelitis has again attracted attention to the question of polio-encephalitis, and there is no doubt that considerable confusion prevails concerning the definition of this condition. McAlpine (1947) stated that "during this epidemic the presence of nystagmus, photophobia, and neck rigidity in a patient who has been ill for a few days with headache and fever was considered sufficient evidence for the diagnosis of polio-encephalitis." Murray (1947) suggested that the brain-stem form of the disease should be regarded as an extension of the spinal type, and that the notification of polio-encephalitis as a separate condition should be discontinued.

Rothman (1931), in an excellent review of the situation at that time, stated that "the exact status of polio-encephalitis (using the term to mean cerebral and not bulbar involvement) and its relationship to the spinal form of infantile paralysis remains nearly as unsettled now as it was at the time of its description." This statement holds true to-day. It is hardly surprising that this should be so when it is remembered that the name was first given by Strümpell in 1885 to a form of cerebral paralysis in children which, although displaying many of the manifestations of poliomyelitis, was probably due to toxic action on the cerebral vessels causing thrombosis or haemorrhage without evidence of encephalitis (Ford and Schaffer, 1927).

It has been stated that increased tendon reflexes, absent abdominal reflexes, and extensor responses are evidence of cerebral involvement in poliomyelitis (Kiss and Fenyès, 1936). These signs, and the combination of spastic and flaccid paralyses, may be due to pressure on the pyramidal tracts from inflammation and oedema in the white matter of the cord.

Scheinker (1947), in a detailed study of six cases, found that no part of the central nervous system entirely escaped damage, and that the medulla, pons, mid-brain, and cerebellum were involved in every case, while the cerebral cortex was not affected at all. He also found that inflammatory changes were intense but neuronal destruction rare in the higher centres, in contradistinction to conditions in the anterior horns. Bodian (1947), reporting on 24 human cases, stated that to the pathologist all cases of poliomyelitis were encephalitic, that some areas of the brain were never affected but the main cerebral changes were from the brain-stem to the hypothalamus and thalamus. So far as the motor cortex was concerned, Bodian considered that only in rare cases were the lesions severe enough to cause clinical signs, and that spastic and psychic sequelae were not cortical in origin—nor were stupor, disorientation, and coma—but were produced by areas of focal softening in the basal centres.

The following case under our care appeared to show encephalitic manifestations and involvement of lower motor neurones.

Case Report

A girl aged 17½ had been well until Nov. 9, 1947, when she suddenly developed a cold and generalized headache. On Nov. 12 vomiting started, being repeated several times, and she was admitted to hospital as a case of suspected meningococcal meningitis.

Previous history revealed that she had had convulsions in infancy dating from birth, but these had gradually diminished in frequency and finally ceased at the age of 8. On admission the temperature was 100° F. (37.8° C.), pulse 104, respirations 20. The patient was extremely ill; she was unconscious, but resisted violently when disturbed. The pupils were very small but equal, and they reacted to light. Cranial nerves were normal. Neck stiffness and Kernig's sign were present. Power and tone in the limbs appeared normal and movement during the delirious phases was full. Tendon and abdominal reflexes were present and equal and the plantar reflexes normal. There was incontinence of urine. The cerebrospinal fluid was clear and under increased pressure, and 20 ml. was removed easily. She was treated with full doses of sulphathiazole and $\frac{1}{2}$ mega unit of penicillin daily (10,000 units intrathecaly) pending laboratory report on the fluid. She vomited once later but remained quiet unless disturbed.

Next day the temperature had risen to 100.6° F. (38.1° C.); she was still in coma, was difficult to rouse, and only mumbled unintelligently, and became violent on interference. By 1 p.m. she showed signs of returning to consciousness and reacted to stimuli but still resented interference. Forty minutes later she had a major convulsion which lasted only a short time, and then she became comatose again. At 3.10 p.m. she developed twitching of the right side of the face lasting a few seconds, became cyanosed and then pale, and was still unconscious. This twitching was repeated about one and a half hours later and also at 6.30 p.m., and the state of unconsciousness remained unchanged. By 9.30 p.m. she regained consciousness for a short time, was very restless and complained of frontal headache, but asked for a bedpan and passed urine normally.

The following morning (Nov. 14) the temperature was normal and remained so throughout the remainder of the illness. The cerebration had improved and she could answer questions, but she tired very easily and was not co-operative. The neck stiffness persisted, but Kernig's sign was negative. The left arm was now paralysed and the movements of the right forearm and hand impaired, while the tendon reflexes were absent in both arms and only the left ankle-jerk could be obtained. The abdominal and plantar reflexes were normal.

By this time the reports on the examination of the specimens of cerebrospinal fluid had been received. The specimen of Nov. 12 was sterile, contained 64 cells per c.mm. (87% lymphocytes), 700 mg. chlorides, 30 mg. sugar per 100 ml., 30 mg. protein per 100 ml., with no increase in globulin. The specimen of Nov. 13 was also sterile, 162 cells per c.mm. (70% lymphocytes), 740 mg. chlorides per 100 ml.; protein and sugar were unchanged.

The improvement in her condition was maintained; she was rational and co-operative on Nov. 15 and could sit up and move about easily, though she had no recollection of her illness. The power in the arms improved slowly and the tendon reflexes eventually returned, so that she was able to leave hospital on Dec. 8. There was some weakness of the forearms and hands, particularly on the left, due to impaired action of the intrinsic muscles of the thumb and fingers. Incoordination of the left arm and slight intention tremor were present.

She was next seen as an out-patient on Jan. 20, 1948, when she appeared normal mentally and had developed no character changes. She still had considerable disability in using the left hand, particularly for fine movements, although the range was full and the power improved. The tremor of the left hand was coarser than previously, was more pronounced, and could not be controlled voluntarily; past-pointing was still present and the tendon reflexes were more active than on the right. There was no muscular wasting.

Discussion

The inevitable criticism of such a case is that it may have been one of acute encephalitis lethargica. Toomey (1940), in discussing the diagnosis of encephalitis, states that, although theoretically a patient with encephalitis should have an upper motor neurone response, there may be such a degree of neural shock that he may be somnolent or

comatose and therefore not very reactive. In such cases, if there are combined upper and lower motor neurone lesions, the latter should be ignored and a diagnosis of encephalitis lethargica made, unless the lower motor lesion is segmental in type, when polio-encephalitis would be probable.

The case reported occurred towards the end of the polio-myelitis epidemic, when encephalitis lethargica was extremely rare. There was no cranial-nerve involvement and the rapid onset of delirium and coma was followed by a flaccid paralysis of the arms which cleared rapidly except for the left hand. An upper motor neurone lesion was suggested by the subsequent residual weakness, tremor, and incoordination, as well as by the initial hyperexcitability and coma, but without any subsequent character changes. The cerebrospinal fluid is of no help in distinguishing the two conditions.

It has been mentioned that the reason why all cases of poliomyelitis do not show symptoms resulting from the cerebral lesions is probably because they are rarely severe or destructive enough, but it is also true that considerable difficulty exists in assessing how far they may alter function. It seems reasonable to suggest that, although the lesions may be distinguished histologically, the symptoms resulting from them may, according to their position, resemble those of any other acute encephalitis. Differential diagnosis would therefore not be possible on clinical grounds alone, unless typical segmental lower motor neurone paralysis was also present.

Summary

It is suggested that a diagnosis of polio-encephalitis should be reserved for the comparatively rare cases of poliomyelitis with cortical or cerebral manifestations.

A case is described which appeared to show such evidence, with early flaccid paralysis of the arms and later spasticity and tremor of the left hand.

The difficulty of diagnosing such a condition from acute encephalitis lethargica is discussed, and the suggestion offered that on clinical grounds alone this may be impossible.

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The report of the British Council for the year 1947-8 has recently been published. It describes the work done to strengthen the bonds of common interest between this country and others in the arts and sciences and in the social and technical problems of the age. The Council has an active medical department which produces the *British Medical Bulletin* for distribution abroad. Two numbers were published during the year: the first, of 118 pages, contained a symposium on problems of personnel research, and the second, of 176 pages, a symposium on lactation. The medical library and information service was considerably expanded during the year, and many more inquiries were received from overseas, particularly about the social services and the new Health Act. A number of distinguished medical men were invited to visit this country as the Council's guests, and assistance was given to foreign delegates attending some of the international medical conferences which were held in this country. Programmes of work were arranged for over forty overseas medical graduates holding Council scholarships. In connexion with colonial students generally the Council's experience is that the early suspicions which these students are apt to harbour soon vanish and are replaced by gratitude and eagerness to co-operate once they know that they are treated as equals, or at least on the basis of their personal capacities and characteristics. Through the welfare service for overseas students of university status assistance was given to a total of 7,600 students.