interval being four days. In our case blindness set in immediately after the accident, and atrophy was distinct after a fortnight.

Concerning the prognosis of these cases there has been general agreement (Traquair) that if no signs of improvement in visual acuity occur during the first four weeks no further change can be expected. But in our patient there was a rapid and almost complete recovery as late as five months after the injury. This is difficult to explain; it is contrary to all experience that recovery should have started after so long a time. One might think of a hysterical fixation of an original organic blindness, but nothing was observed in the general behaviour of the patient to support this, and there were no psychogenic elements. There were, on the other hand, optic atrophy and a transitory scotoma—findings which do not permit a functional explanation.

It is likely that the primary site of the lesion was in or near the anterior chiasmal angle, affecting mainly the right optic nerve but also damaging the crossed fibres of the left. It is interesting to note that the temporal-field defect of the left eye gradually diminished and had entirely disappeared after the relapse. We will therefore have to assume that on this occasion the chiasma had been spared, the lesion being restricted to the right optic nerve.

Summary

A case is reported in which blindness and optic atrophy in the right eye, with a temporal-field defect in the left eye, followed a fall in an epileptic fit. X-ray examinations of the skull were negative. There was no fracture.

Not less than five months after the injury normal visual acuity with full visual fields in the left eye were re-established; during this period of recovery central vision was the last to return to normal. The temporal-field defect in the left eye was at the same time greatly reduced.

Shortly afterwards, following a trivial blow on the face, his sight and visual-field condition deteriorated, a gradually extending central scotoma appeared, and visual acuity fell rapidly until the right eye became blind again. There was no temporal-field defect in the left eye at this time. Atrophy of the right disk remained unchanged during the whole period. There was no further change one year later.

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Dr. Esmond R. Long, director of medical research and therapy, National Tuberculosis Association, has been appointed editor-in-chief of the *American Review of Tuberculosis*, official journal of the N.T.A.'s medical section, the American Trudeau Society, to succeed the late Dr. Max Pinner. A new position of managing editor has been created, and Dr. Walsh McDermott, associate professor of medicine, Cornell University Medical School, New York, has been appointed to the post. Dr. Long, who is also director of the Henry Phipps Institute, Philadelphia, has been director of the N.T.A.'s medical research since January, 1947. He is a past president of the N.T.A. and is a consultant on tuberculosis to the United States Army, the Veterans Administration, and the U.S. Public Health Service. A graduate of the College of Physicians and Surgeons, Columbia University, Dr. McDermott is associate attending physician of New York Hospital and was associate editor of the seventh and eighth editions of the Cecil-Loeb *Textbook of Medicine*. He is a member of the American Trudeau Society's Committee on Medical Research and Therapy and of the Antibiotics Study Section and the Tuberculosis Study Section, National Institute of Health, U.S. Public Health Service.

JUVENILE GENERAL PARALYSIS

A CASE WITH A SPONTANEOUS BUT SHORT-LIVED REMISSION

BY

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Although a considerable number of cases of juvenile general paralysis have been reported in the literature the disease is relatively rare and its incidence in cases of congenital syphilis is stated by Stewart (1933) to be about 1%. Schmidt-Kraepelin (1920) gives the figure as 1.7%, but White and Veeder (1922) and Smith (1933) are in agreement that the incidence is less than 1%. Most authorities agree that the sex distribution is approximately equal, although there is a slight tendency towards a preponderance of males; thus out of 145 cases reported by Mott (1899), Watson (1903), Schlicht (1915), Schmidt-Kraepelin (1920), Klauder and Solomon (1933), and Menninger (1930), 83 were males and 72 females. According to Menninger (1936) there are two types of onset, the first being insidious, when the child is apparently subnormal from birth, and the second in which an acute breakdown occurs after several years of seemingly normal development. Menninger in an analysis of 542 cases gives the age of onset as 3 to 5 years in 9.4%, 6 to 10 years in 24%, 11 to 15 years in 36.2%, 16 to 20 years in 26.8%, and 21 to 25 years in 3.6%. In the majority of cases, therefore, the illness begins between the ages of 10 and 15. Menninger states that in a series of 653 cases psychic trauma was regarded as the precipitating factor in 2.7% and that convulsions occurred in 17% before the diagnosis was established.

Case Report

A boy, aged 11 years 3 months, was admitted to this hospital in November, 1946, as he was not responding to out-patient treatment at a child-guidance clinic in another area.

Family History.—The father, a Naval petty officer aged 38, was stated to be fit and well. He has had little to do with the boy's upbringing owing to his service overseas. When interviewed in December, 1946, he admitted that he had had primary syphilis in 1929 but had received a full course of treatment over two years. His Wassermann reaction was: blood +, C.S.F. negative. He was having treatment at a V.D. clinic. The mother, aged 36, is fit and well. She is a kindly woman, but ineffective in handling the family and lacking in understanding. Her Wassermann reaction was: blood +, C.S.F. negative. She has had no treatment. **Siblings:** There are three sisters, aged 16, 7, and 4. All are bright and alert, with negative serology.

Personal History

The boy weighed 8 lb. 12 oz. (3.98 kg.) at birth, walked at 12 months, and began talking at 15 months. Clean habits were established early, and he appears to have been a perfectly normal child until a few days before his fifth birthday, when his home was destroyed by enemy action. He was uninjured, but was said to have screamed persistently for several hours after the incident. He was apparently terrified during the next three days, and there were many outbursts of screaming during this time. On the fourth day he had an epileptiform attack during which he exhibited loss of consciousness and left-sided rigidity followed by generalized clonic movements. In the succeeding nine months he had frank epileptic major attacks with incontinence and tongue-biting at the rate of about one a week. These persisted despite anticonvulsant drugs prescribed by his doctor. During this time he progressively became more dull and was excessively timid and anxious. There was, in fact, a complete personality change, and whereas he had formerly been a bright, cheerful, alert, self-possessed child, he now became very quiet and reserved, and was made fun

of by other children. Six months after the original epileptic manifestation he was seen by a children's psychiatrist, who stated that he was suffering from an anxiety state but that no special treatment was required. Shortly before this it had been noticed that he was becoming very clumsy and had very great difficulty in picking up objects. It was also noted that he had to be assisted in dressing, as he was unable to do up his buttons or tie his shoe-laces. His speech, which had previously been quite clear, became slurred, and his parents and teachers had difficulty in understanding what he was trying to say.

In July, 1942, the family moved to Scotland, and shortly after this a remarkable change was noted in the child. In the course of a few months he completely lost all signs of muscular incoordination, his speech became clear, he regained confidence, and he began to stand up for himself with other boys. He rapidly made progress in education, and during the next two years became recognized as one of the best scholars in his year. Evidence of this exists in the fact that he was awarded several books as prizes in arithmetic, writing, spelling, and drawing. In September, 1944, the family moved to another area and very shortly John's condition began to deteriorate once more. His standard at school fell rapidly, but his mother attributed this to the change of school. By February, 1945, however, it became obvious to his parents that his symptoms were exactly similar to those noted at the age of 5. A school report in May, 1945, noted him as "dull and backward" but "very nervous." He was accordingly referred to a child-guidance clinic and was treated as a "maladjusted child." His I.Q. was reported as 100. It is of interest to note that his mother had observed that his drawings were becoming completely indecipherable, being entirely obscured by a wealth of unnecessary detail. As three months' treatment did not make any impression on the child, but on the contrary had failed to prevent deterioration, he was sent to this hospital for in-patient treatment.

On examination he was a plump, awkward-looking child who was very restless and apprehensive. It was noted that the incisors were of the Hutchinsonian type. Gross motor incoordination was present, and this rendered him ludicrously clumsy. Slurring of articulation was a prominent feature, and although the pupillary responses were fairly normal there was irregularity of the margins. Tongue tremor was present and all reflexes were exaggerated; plantar responses were flexor. He was dull, slow in comprehension, and his I.Q. was found to be 69. The diagnosis of juvenile paresis was confirmed by a triple positive Wassermann reaction in both blood and cerebrospinal fluid and a paretic gold curve in the Lange test.

Since admission his condition has continued to deteriorate despite treatment, and he now falls about, has numerous epileptiform attacks, is incontinent, and requires constant nursing care and supervision.

Discussion

The above history has been carefully checked by reference to practitioners who previously treated the child, to teachers, and to others. It will be noted that the onset was just before the age of 5. So far as can be ascertained there are only 14 cases recorded in the literature in which the age at onset was the fifth year. Spontaneous remission is indeed very rare, and Stewart (1933) states that "the disease is never interrupted by a remission." On the other hand, Alzheimer (1895), Thompson and Dawson (1895), and others have recorded instances of spontaneous remissions, but the total number of cases does not exceed 20. Most authorities agree that the prognosis in such cases is very poor, and Lurie *et al.* (1943) report on 8 cases of juvenile paresis seen at the Cincinnati Child Guidance Home. Despite early diagnosis and treatment all these children showed progressive mental and intellectual deterioration.

The fact that this boy was examined by two child psychiatrists and treated by one for three months should make plain the need for a careful physical examination of all children seen at child-guidance clinics. In this connexion it is of interest to note that out of 125 in-patient children

reported by Barker and Milligan (1947) 13 were found to be suffering from organic disease of the nervous system and only three of these cases had been diagnosed as such before admission to hospital.

Summary

A case of juvenile general paralysis is described, with an onset at the age of 5 and an apparently complete spontaneous remission of two years' duration.

I wish to thank Dr. Thomas Beaton, Physician-Superintendent, St. James Hospital, Portsmouth, for permission to publish this case record.

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Medical Memoranda

Gastric Herpes Zoster

The following report of a case of haematemesis due to gastric herpes zoster is worthy of record.

CASE HISTORY

A diabetic woman aged 66 was admitted in a precomatose condition with a blood sugar of 450 mg. per 100 ml. She had been under regular observation in the diabetic clinic, and had no history of gastric symptoms. One year previously she had fallen and bruised her left lower ribs, and since that time she had noticed intermittent vague pain in the same region. A week before admission this had become constant and more severe, radiating round to the left abdomen. Four days later, feeling ill, she remained in bed, and at noon had a sharp haematemesis. At 3 p.m. she noticed a rash on the abdomen to the left of the umbilicus. On account of her malaise she discontinued both food and insulin; her diabetic precoma was the direct result.

On examination, apart from the signs of dehydration, she showed slight central epigastric tenderness. A typical eruption of herpes zoster was present on the left chest and abdomen, apparently involving thoracic segments 9 and 10. The rest of the examination was normal. Her diabetic condition was successfully treated, but unfortunately it was severe enough to preclude gastroscopy. A barium meal x-ray film eight days later was normal, and a fractional test meal showed complete achlorhydria. Haemoglobin was 90%, and rose subsequently with treatment. A radiograph of the spine and ribs was normal. The cerebrospinal fluid on admission contained 70 mg. of protein per 100 ml. and 23 lymphocytes per c.mm. Nine days later the protein was 50 mg. and there were 10 lymphocytes.

COMMENT

The sympathetic supply of the stomach by way of the coeliac plexus almost certainly includes branches from the ninth and tenth thoracic nerves. The appearance of the somatic eruption within three hours of the haematemesis is very suggestive of a true gastric herpes zoster as the origin of the latter. The relation of the previous injury to the herpes is of interest. Although there was no evidence of bony injury, it is possible that the repeated painful stimuli from an organized haematoma in muscle may have been sufficient to render the affected segments liable to attack by the virus. The cerebrospinal fluid changes are typical of those found in herpes zoster.

I wish to thank Prof. T. H. Oliver for permission to describe this case.

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