Progress.—August 1948 : Wasting was now apparent in the peripheral muscles of upper and lower limbs, but at the same time some return of power was noted. By the end of September nystagmus was present on direct fixation and was more vertical than horizontal. Arm reflexes were approximately normal ; tone was now somewhat increased in both legs. Right plantar response was equivocal and the left extensor.

Early in December his condition had deteriorated, incontinence was frequent, at times he was indifferent to it and was confused. Upper as well as lower limbs were spastic with increased reflexes and double extensor plantar responses.

8.12.48 : Patient became ill and drowsy, but retained consciousness. Speech and swallowing were impaired. There was conjugate deviation of head and eyes to right. Spasticity and paresis of all four limbs were intensified. Signs of a complete left hemiplegia with hemianæsthesia and hemianopia were present. A grasp reflex was obtained on the right side.

Further progress.—Power slowly returned in the limbs, more quickly in the upper than the lower ; the latter have remained severely paretic. A sensory level appeared at D8–9 segment. Cerebellar signs became more pronounced. Mental confusion was noted until March 1949 ; since then considerable improvement in his mental state has occurred. Except during the acute flare-up in December and on a few other isolated occasions, there has been no fever. The majority of the tests mentioned previously were repeated at intervals, and were negative.

C.S.F. : This was examined at monthly intervals until October 1948. It has shown the following features : (1) Xanthochromia. (2) Pleocytosis varying between 160 and 45 cells per c.mm., lymphocytes or polymorphs predominating. (3) Protein ranging from 1,200 to 3,500 mg. %. (4) A consistently negative W.R., and Lange colloidal gold curve. A specimen on April 20, 1949, contained 30 cells per c.mm. (28 lymphos., 2 polys.) and 400 mg. % of protein, and another on May 9, 1949, showed lymphos. 50, polys. 5, protein 350 mg. %.

Repeated attempts to isolate an organism from the C.S.F. have failed.

Discussion.—The diagnosis of a Guillain-Barre syndrome was at first considered in view of the four-limb flaccid paralysis and the xanthochromic C.S.F., although there were several atypical features, notably bilateral deafness and cerebellar signs. The prolonged course of the illness, the appearance of fresh encephalitic and myelitic signs a year after the onset, and the persistent pleocytosis in the spinal fluid suggested as an alternative diagnosis an obscure form of chronic encephalo-myelo-radiculitis. The man's occupation and the fact that he had nursed a cow with fever for three months prior to the onset of his illness suggested the possibility of a chronic form of neuro-brucellosis but laboratory tests carried out under the supervision of Professor F. Selbie of the Bland-Sutton Institute have failed to confirm this diagnosis. Judging by the clinical improvement and the recent diminution in the pleocytosis and the protein content of the C.S.F. it would appear as if the active process was dying out.

POSTCRIPT.—Since this case was shown, Professor Dorothy Russell has informed us of a fatal case of encephalo-myelo-radiculitis in a cowman which showed clinical features very similar to those shown by our patient.

Dr. H. G. Miller mentioned the difficulty sometimes caused by cases of porphyria in which the biochemical abnormality may for considerable periods be limited to the excretion of colourless porphyrinogens, and asked how completely this possibility had been excluded.

Dr. Russell Brain: Blindness due to optic neuritis and deafness may both occur in acute infective polyneuritis, and so also may cerebral symptoms. In spite of its chronic course it seems to me that the whole of the clinical picture in this case may be the result of infection with a virus allied to, if not identical with, that responsible for acute infective polyneuritis.

Mid-Brain Syndrome in a Professional Boxer.—DOUGLAS MCALPINE, M.D., F.R.C.P., and FRANCIS PAGE, M.D., M.R.C.P.

J. N., aged 24.

A boxer since the age of 14. Had never received severe punishment in the ring, despite three months' booth boxing, until January 1948 when he was twice knocked out. Amnesia on each occasion did not exceed a few seconds.

February 1948, after losing his temper, his whole body shook and he felt as though he wanted to "get going" with his fists. This quickly subsided but two days later he noticed slight tremor of the left hand, gradually increasing in severity and, during the next few weeks, spreading up the arm to involve the shoulder and head. Excessive salivation was also noticed during this period, and in March 1948 his speech became slow and indistinct and walking unsteady.

All symptoms have been slowly progressive although the tremor was temporarily improved by moderate doses of belladonna.

Examination.—No evidence of intellectual deterioration. Speech moderately dysarthric. Slight loss of facial expression. Pupils and ocular movements full. No nystagmus. Rhythmic, coarse, static tremor of head, left shoulder, arm and hand with occasional tremor of right hand. Tremor intensified by emotion but disappears when limb is supported. On voluntary movement this coarse tremor is replaced by a less intense and more rapid intention type of tremor. Posture of hand not typical of Parkinsonism ; no cogwheel phenomenon. All reflexes very brisk, including jaw-jerk. Bilateral extensor plantar responses. No sensory loss. Gait—left arm fixed across body and fails to swing. Mild spastic gait, less apparent when running.

Investigations.—C.S.F. normal. Blood W.R. negative. X-ray of skull normal. E.E.G. normal, including a record taken during sleep after seconal medication.

Discussion.—Dr. Gordon Holmes in 1904 described a series of eight cases which showed a static form of coarse tremor replaced by an intention tremor on voluntary movement. Some of these cases showed evidence of third nerve paralysis and unilateral or bilateral pyramidal signs. The pathological lesion was either vascular or neoplastic in the mid-brain. Dr. Holmes attributed the tremor to involvement of the red nucleus or superior cerebellar peduncle. Kremer, Russell and Smyth in 1947 published a number of cases of a mid-brain syndrome following head injury. Several of these cases showed a mild Parkinsonian facies; in addition dysarthria, ataxia, a static coarse tremor and pyramidal signs were seen in all cases. Evidence of damage to the third nerve was inconstant.

In the present case the mild Parkinsonian features may be accounted for by damage to the substantia nigra, and the tremor to a lesion of the red nucleus or its connexions. The picture shown by this patient is not consistent with a previous attack of encephalitis, nor is there any evidence to suggest a neoplasm. On the other hand the likeliest explanation appears to be trauma resulting from contusion to the mid-brain caused by one or more knockouts.

REFERENCES

HOLMES, GORDON (1904) *Brain*, **27**, 327.

KREMER, M., RUSSELL, W. R., and SMYTH, G. E. (1947) *J. Neurol., Neurosurg. Psychiat.*, **10**, 49.

Dr. Russell Brain: I have recently seen a very similar syndrome in a professional boxer who exhibited a mixture of torticollis, Parkinsonism and cerebellar ataxia suggesting that the main damage had fallen upon the striatum and upper mid-brain.

Dr. Helen Dimsdale: It is interesting that this patient complained of excessive salivation, a symptom usually associated with encephalitic Parkinsonism.

Multiple Cranial and Peripheral Neuropathy.—REDVERS IRONSIDE, F.R.C.P.

Miss L. D., aged 62.

She thinks that her left upper limb has never been so well developed as the right. About eight years ago she noticed that the left half of her tongue was wasted and this has become progressively worse.

One year ago: Diplopia, left-sided ptosis, and a divergent squint. At that time she was found to have a partial third-nerve and a complete sixth-nerve palsy on the left side, with impairment of sensibility to cotton-wool and pin-prick over the first and second divisions of the fifth nerve. Corneal reflex diminished on the left side. Ptosis improved but sensibility on the face has become more affected and now involves all three divisions of the left fifth nerve. The motor division does not appear to be affected.

Six months ago: Suddenly developed left foot drop with analgesia to pin-prick and cotton-wool over the outer border of the foot and the outer aspect of the left calf.

C.S.F. normal. X-rays skull normal. Blood W.R. negative.

Patient has a fibroma over the right lower ribs in front and another on the flexor aspect of the left wrist.

Dr. Ironside drew attention to the length of the history, the presence of fibromata and a developmental anomaly, and commented on the frequency with which cases of multiple neuropathy were found to be due to tumours involving nerves.

Vestibular Syndrome with Facial Palsy.—REDVERS IRONSIDE, F.R.C.P.

Mrs. G. B., aged 62.

For the past eleven years has had attacks of vertigo, particularly after hot baths. For the last three years the attacks have become more frequent and have lasted as long as twenty-four hours. The present attack commenced four weeks ago with severe vertigo, pain behind the