therapeutic control. Because of these difficulties patients must be seen more often to maintain good therapeutic control: this is expensive in manpower and time and more than most hospitals can afford. Harries *et al*¹⁸ and McInnes¹⁹ confirmed that junior staff always play for safety when prolonged intervals are in use, and as a result underanticoagulation is the most common defect of control.

Mitchell²⁰ pointed out that so many statistical errors were made in the early trials of anticoagulant treatment, and the quality of thromboplastins was so poor and variable then, that the findings of most of these trials must be considered to be invalid.

The findings of the early Medical Research Council trials of anticoagulant treatment after myocardial infarction²¹⁻²³ were equivocal except for a reduction of the secondary effects of intramural thrombi. The value of anticoagulant treatment in this condition has therefore fallen into disrepute recently, and few now continue anticoagulant treatment for long after patients are discharged from hospital. The recent "Dutch trial,"¹¹ avoiding the errors of early trials and maintaining the therapeutic ratio range within close limits, showed that intensive and stable anticoagulant treatment substantially reduced the risk of recurrent myocardial infarction. This suggests that early trials failed due to inadequate control of treatment.

Over the next few years the number of patients receiving long-term anticoagulant treatment will increase owing to the reassessment of these new trials and the increased number of patients undergoing cardiac surgery.

Recommendation—Anticoagulant treatment is expensive and is not cost effective. It is invariably delegated to rotating junior staff, who may not have had previous experience, and so there is no consistent control. Medical assessment of anticoagulant treatment should be started. The method described here is simple and may be recorded in the patients' record cards after each visit. The "score" may then be evaluated annually. British comparative thromboplastin and the associated quality assessment scheme has given anticoagulant treatment a safe foundation. With the higher therapeutic BCR ranges now being advocated therapeutic quality control is required so that this expensive and potentially dangerous treatment may be used to the greatest effect.

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(Accepted 30 November 1981)

SHORT REPORTS

Lhermitte's sign in Behçet's disease

The neurological manifestations of Behçet's disease may resemble those of multiple sclerosis. The most common patterns in Britain are transient or persistent episodes of brain-stem dysfunction and focal cerebral or spinal cord disease.¹ The diagnosis of Behçet's disease is clinical and may be difficult, particularly when neurological disease precedes the more easily recognisable systemic features.

Lhermitte's sign consists of a sudden tingling or sensation of electric shock in the limbs on flexion of the neck and was originally thought to be pathognomonic of multiple sclerosis.² It is now recognised as occurring in various conditions affecting the cervical cord and cervicomedullary junction.^{3,4} We observed this sign to be a prominent feature in two patients with Behçet's disease.

Case reports

CASE 1

An 18-year-old Cypriot developed shock-like sensations "like a flash of blood or electricity" in his legs and arms on flexion of the neck. This persisted and was followed by left-sided hypoesthesia, leg weakness, and incoordination. He had suffered arthritis of the hands at the age of 15 and subsequently episodes of erythema nodosum.

On examination he had bilateral jerk nystagmus, left-sided cerebellar signs and sensory impairment with impaired joint position and vibration sense, and a spastic paraparesis. Lhermitte's sign was present. Ocular examination showed old anterior uveitis with active inflammation of the vitreous and periphlebitis. There were no other notable abnormalities. The clinical impression was of multiple sclerosis, but ocular inflammation and distinct inflammatory changes in the cerebrospinal fluid suggested an alternative such as sarcoidosis or Behçet's disease. A myelogram was normal.

Six months later he developed painful crops of oral ulcers and occasional painful penile ulcers, which subsequently recurred. His neurological condition gradually deteriorated despite corticosteroids and immunosuppressives. The clinical diagnosis was Behçet's disease, dominated by neurological symptoms. The original complaint of Lhermitte's sign persisted.

CASE 2

A 45-year-old woman developed neurological disease. Behçet's disease had been diagnosed nine years earlier on the basis of longstanding painful mouth and genital ulcers, polyarthralgia, and uveitis. She had been treated intermittently with steroids and immunosuppressives. She later complained of right-sided weakness and "brisk electric shocks" down her right side on flexion of the neck. These were momentary, shooting from the neck to the fingers and toes.

She was mildly cushingoid with active arthritis, orogenital ulceration, and anterior and posterior uveitis. Neurological examination showed reduced sensation on the right, including joint position and vibration sense. There was mild pyramidal weakness on the right but no evidence of incoordination or nystagmus. Lhermitte's sign remained present. Investigations showed an

erythrocyte sedimentation rate of 58 mm in first hour with acute-phase proteins. A computed tomogram and a myelogram were normal. Cerebrospinal fluid protein concentration was raised and showed a polyclonal globulin band with no cells.

An increased dose of prednisolone and azathioprine improved both her ocular and neurological disease, the neurological signs and Lhermitte's sign disappearing after about three months.

Comment

Lhermitte's sign is probably due to movement stimulating demyelinated ascending tracts of the cervical cord,⁴ and these cases show its occurrence in neurological Behçet's disease, which, as far as we are aware, has not been previously reported.

Occasionally it is difficult to differentiate between multiple sclerosis and Behçet's disease.⁵ Disease of the optic nerve may occur in Behçet's disease, and remitting brain-stem syndromes and cerebellar ataxia are features of both. The presence of an internuclear ophthalmoplegia, painful tonic seizures, and Lhermitte's sign have been thought to be characteristic of multiple sclerosis.⁵ We think that the presence of Lhermitte's sign should not distract from the possibility of Behçet's disease in patients with neurological illness, particularly when associated with uveitis or other systemic abnormalities.

We thank Mrs P A Wright for secretarial help, and Dr D G James and Mr M D Sanders for permission to report on patients under their care.

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² Lhermitte J, Bollak Nicholas M. Les douleurs à type de décharge électrique consécutives à la flexion céphalique dans la sclérose en plaque. Un cas de forme sensitive de la sclérose multiple. *Rev Neurol* 1924;**31**:56-62.

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⁴ Jones A. Transient radiation myelopathy (with reference to Lhermitte's sign of electrical paraesthesia). *Br J Radiol* 1964;**37**:727-44.

⁵ Motomara S, Tabira T, Kuroiwa Y. A clinical comparative study of multiple sclerosis and neuro-Behçet's syndrome. *J Neurol Neurosurg Psychiatry* 1980;**43**:210-3.

(Accepted 18 November 1981)

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Reversible oesophageal dysphagia and long-term ingestion of chlormethiazole

Chlormethiazole is widely used in psychiatric practice, particularly in the treatment of alcoholism. We report a case of longstanding dysphagia due to an oesophageal motility disorder that responded to the withdrawal of chlormethiazole.

Case report

A 50-year-old woman with severe personality disorder and symptoms of anxiety and hysteria had been followed up in the psychiatric department of this hospital for 14 years. Numerous adverse social circumstances included relationships with three men and excessive consumption of alcohol. Her regular medication since 1972 had been diazepam and chlormethiazole (dose between 2 and 4 g daily).

One year after starting chlormethiazole she complained of dysphagia, with solid food sticking in the throat often associated with choking. Laryngeal examination showed no abnormality, but she failed to attend for barium swallow. It was decided that her symptoms were probably hysterical in origin. Over the next three years she lost about 13 kg in weight but subsequently maintained her weight. In 1979 she was referred to the general medical clinic with the same complaints. Barium swallow examination showed incoordination in swallowing, with delay and movement of the barium backwards and forwards in the mouth before passage into the oesophagus. Oesophageal manometry showed a gross motility disorder in the whole of the oesophagus, with numerous spontaneous, non-propagated contractions (fig

(a)). Lower oesophageal pH recording showed some evidence of gastro-oesophageal reflux during the manometry, but there was no reflux during overnight recording after removal of the manometry catheter. The endoscopic appearance of the oesophagus was normal. Chlormethiazole was withdrawn over four weeks; during part of this time nasogastric feeding was required. She initially noticed an exacerbation of her anxiety but within four weeks of stopping the drug began to feel generally better. Her dysphagia gradually improved and she began to gain weight. Oesophageal motility studies six weeks after the drug was stopped showed a return to almost normal motility (fig (b)). During the succeeding months she remained free from dysphagia and gained 10 kg in weight, although she retained her demanding personality.

Oesophageal manometry recordings (a) while patient was taking chlormethiazole and (b) six weeks after the drug was stopped.

Comment

Studies on the pharmacological properties of chlormethiazole have reported that it is an anticonvulsant with sedative and hypnotic properties but that it is without appreciable influence on the autonomic nervous system.¹ It has inhibitory effects on various central nervous centres: the cerebral cortex, respiratory centre, thermoregulatory centre, vomiting centre, and spinal cord.² It also has a non-specific action on reducing smooth-muscle tone but is not a neuromuscular blocking drug.²

The control of oesophageal motility is complex and may be affected by environmental factors including stress and drugs. This patient was taking only benzodiazepine tranquillisers in addition to chlormethiazole; only the chlormethiazole, however, was withdrawn. Her personal circumstances remained apparently unchanged during this time.