lies. Accordingly, it is important to report further data on Parkinson’s disease in twins.

An advertisement was placed in the newsletter of the Parkinson’s Disease Society of the United Kingdom, requesting information on patients with Parkinson’s disease who had a twin. Twenty-two twin pairs were identified. The diagnosis of Parkinson’s disease in the index case of each twin pair was verified by communication with their general practitioner or neurologist. Criteria for accepting the diagnosis were the presence of typical clinical signs (tremor, rigidity and akinesia), the absence of features that might suggest an alternative diagnosis (severe dementia, disordered eye movements, postural hypotension or incontinence), and a definite response to levodopa treatment. Zygocity was assessed by asking the twins and their relatives whether they looked alike or dissimilar. The affected twin was asked whether their co-twin had any suggestion of a similar illness. If this was thought to be the case, the co-twin was approached by letter to describe the clinical features of their illness, and their physician was contacted for further information.

Of the 22 pairs of twins, one of whom was judged to have definite Parkinson’s disease, 11 pairs were thought to be identical and 11 pairs non-identical. Amongst the 11 identical twin-pairs (mean duration of illness 9–3 years), only one affected twin reported that her co-twin had Parkinson’s disease. The index case, a 76 year old lady, had had typical Parkinson’s disease with tremor for the previous 5 years. Her identical sister also had Parkinson’s disease, with tremor, for 3 years. Among the 11 non-identical twin-pairs (mean duration of illness 8–0 years), one affected twin reported that her co-twin had Parkinson’s disease. A 60 year old female who had Parkinson’s disease for the previous 18 years had a sister similarly affected for the previous 7 years. The remaining 10 identical twin pairs and 10 non-identical twin-pairs said that their co-twins were free of any symptoms or signs of Parkinson’s disease.

There has been no opportunity to examine these twin-pairs personally, so the results presented here must be tentative. However, amongst twins with Parkinson’s disease, the reported prevalence of this illness in the co-twin was no greater amongst identical twin-pairs than amongst non-identical twin-pairs. These data may be added to those of Ward et al. Amongst their 43 monozygotic twin pairs, only one co-twin had definite Parkinson’s disease; in a second twin-pair, the co-twin had possible Parkinson’s disease on the basis of a history of hesitancy in speech and the finding of variable cogwheel rigidity of one upper limb with a fine action tremor on examination. Thus, taken together, the total data suggest that amongst 54 identical twin-pairs, two (3.7%) or at most three (5.6%) were concordant for Parkinson’s disease. In the study of Ward et al, none of 19 dizygotic twin-pairs was definitely concordant for Parkinson’s disease; in one twin-pair, the co-twin had severe dementia which had pre-dated the onset of Parkinsonism by 5 years, and the index case also was demented. Adding the data from the present investigation, amongst 30 non-identical twin-pairs, only one (3.3%), or at most two (6.7%) were concordant for Parkinson’s disease.

It does not appear that the chances of a twin with definite Parkinson’s disease having a similarly affected co-twin is any different amongst identical twin-pairs compared with non-identical twin-pairs. Similar findings have emerged from a study of Parkinson’s disease twin-pairs in Finland (Martilla, personal communication). These data confirm and add to the conclusion of Ward et al2 ‘that the major factors in the aetiology of Parkinson’s disease are non-genetic’.

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Letters

Lymphomatous meningitis appearing as Garin-Bujadoux-Bannwarth meningopolyneuritis

Sir: We report a patient who developed lymphomatous meningitis without evidence of lymphoma outside the nervous system. The initial clinical and cerebrospinal fluid (CSF) findings and elevated antibody titres against the IgA-rabies-borrelia antigen led initially to the incorrect diagnosis of Garin-Bujadoux-Bannwarth meningopolyneuritis.

The 49 year old female patient was admitted in December 1984 with progressive lower back pain and radicular pain in the right SI segment. One month later she developed weakness in both legs and occasional incontinence of urine. The patient had had no serious medical complaints prior to this illness, and in particular no symptoms of Borrelia infection. Examination revealed a slight weakness of the legs (more pronounced on the left side); the left patellar reflex was hyperactive and the right ankle reflex was absent. This first examination gave otherwise unremarkable results, but after 10 days the patient developed a left facial paresis. EEG, CCT, myelography and chemical blood analyses gave normal results. CSF examination showed 70 WBC/mm3 (prevalently lymphocytes and few monocytes in Pappenheim staining), a blood brain barrier dysfunction with total protein of 116 mg/dl and intrathecal immunoreactivity with oligoclonal bands in isoelectric focusing. The titre of antibodies against IgA-rabies-borrelia antigen was elevated to 360 units in serum (normal less than 100 units), and was negative in CSF. The patient was treated with penicillin G (25 mega units/day for 10 days) and prednisone (initially 80 mg/day, dose reduced with time for 3 weeks) because of radicular pain. Sj depleted showed a marked improvement in her radicular pain and facial paresis after 2 weeks of therapy. At this time CSF contained 43 WBC/mm3 and a total protein of 61 mg/dl.

Two weeks after the end of therapy a relapse involving the recurrence of the left facial paresis and a hypesthesia in the right segments C8 and S1 occurred. Both ankle reflexes were absent. Now CSF showed 254 lymphocytes/mm3 and a total protein content of 136 mg/dl. Cytochemical and immunological analysis of CSF cells demonstrated a lymphoblastic lymphoma. Further investigations (radiographs of the chest, spine and skull, sonograms and CT of the abdomen, lymphangiograms of the lower extremities and bone marrow biopsy) gave no sign of lymphomatous infiltration outside the nervous system. However CCT now revealed a solid tumour in the posterior part of the corpus callosum with a diameter of 1.5 cm.

Neurological complications of non-Hodgkin’s lymphomas resulting from metastatic invasion of the nervous system usually
occur late in the course of the illness, when the systemic disease is widespread.1 Our case was thus unusual, in that lymphomatous meningitis seldom occurs in isolation as the first manifestation of a non-Hodgkin’s lymphoma, without other evidence of advanced disease. Not infrequently a lymphomatous meningitis is not discovered during routine cytological CSF analysis.2 Although the positive oligoclonal bands in the CSF3 and the patient’s general symptoms were consistent with a lymphomatous meningitis, the disease was initially diagnosed as a Garin-Bujadoux-Bannwarth meningopolyneuritis on account of (a) the elevated titre of antibodies against Ixodes-rici- nius-borrelia antigen and (b) the appearance of the CSF which seemed to indicate the presence of an inflammatory disease. The initial diagnosis was apparently supported by the patient’s improvement under therapy, although in retrospect this was probably due to the effect of cortisone on the tumour.4

Tick-associated *Borrelia* infections of the central nervous system such as Garin-Bujadoux-Bannwarth meningopolyneuritis and neurological complications of Lyme disease have, in recent years, increasingly been found to be the cause of lesions of the cranial and spinal nerves.5,6 Diagnosis is based on characteristic but unspecific changes in the CSF, including lymphocytic pleocytosis and intrathecal immunoreactivity, as well as the elevated titre of antibodies against Ixodes-rici- nius-borrelia antigen. This report demonstrates that an increased antibody titre does not necessarily indicate an acute *Borrelia* infection. In regions where the vector of the infection (*Ixodes ricinus*) is widespread it is necessary to assume a high incidence of subclinical infections. There are, however, no exact epidemiological data concerning this.

**Sporadic adult onset distal myopathy**

Sir: Distal myopathy is relatively common in Scandinavian countries, but it is much rarer elsewhere. Different types of distal myopathy have been reported. We describe a sporadic early adult onset case of distal myopathy with peculiar clinical and laboratory findings.

A young 19 year old man had been noting slowly increasing difficulty in running for about 2 years. General physical examination was normal. On neurological examination a bilateral stepping gait was found. Walking was difficult on the toes and it was impossible on the heels. There was bilateral wasting of leg muscles. Muscle strength was normal in the upper limbs. Routine laboratory findings and thyroid function were normal. Serum creatine kinase was 7720 U/l (normal 25–190); aldolase was 76 U/l (normal less than 7-6). Patient’s sister’s and brother’s CK and aldolase were normal. EMG examination was performed on tibialis anterior, extensor digitorum brevis and first interosseous dorsalis of the hand. Insertional activity was found in the examined muscles, especially in tibialis anterior, characterised by high frequency discharges. With voluntary effort a low amplitude interference pattern was seen. Many brief small amplitude polyphasic potentials were recorded. Peroneal nerve motor conduction velocity was 51 m/s. Sensory evoked potential latency from median nerve was normal.

A neuromuscular biopsy specimen (with quantitative and qualitative studies) of the superficial peroneal nerve showed no abnormality. Peroneus brevis muscle histology showed some degenerating muscle fibres with nuclear centralisation. As an expression of regenerative phenomena, some basophilic fibres were present. There was a striking increase of end- and perimisial connective tissue. There was no inflammatory reaction. Muscle fibre splitting was evident. Histochemistry (ATPase 9–4• 40–3• 4–35• NADH–GPD–PAS–ORO) showed type I predominance without type grouping. There were no abnormalities of structure or glycogen, or lipid storage. No vacuoles were present (fig a). Electron microscopy (EM) did not show any abnormality. The deltoid muscle was also biopsied. There was some variability of fibre diameter with sparse necrotic fibres and nuclear centralisations (fig b). No histochemical abnormality was found. No vacuoles were seen. EM did not show any abnormality, including motor end plates.

Kratz and Brooke1 in their review of distal myopathy distinguish between “Swedish” and “non-Swedish” types. To the first group are ascribed the cases reported by Welander,2 by Burrows and Duemler3 and by Edström.4 This type of distal myopathy is characterised by: (1) dominant autosomal inheritance, (2) late adult life onset, (3) involvement of hands and forearm extensors, (4) CK levels slightly elevated, (5) EMG and histopathological findings of a myopathy. In contrast, the “non-Swedish” type of distal myopathy is more heterogeneous. Familial and sporadic cases have been reported5–11 and among them it seems possible to identify some patients with peculiar features quite similar to those of our patient. In fact, the patients reported by Marksbery et al.,2 Miller et al.,10 Orrico et al.11 and our patient can be characterised by the following essential features: (1) early adult onset of distal hypotrophy and weakness, (2) CK levels markedly increased, (3) EMG findings suggesting a myopathy, (4) pathological findings suggesting a primary myopathic disease.

More recently two familial cases with similar findings have been reported by Scopetta et al.12 suggesting the possibility of an autosomal inheritance of the disease. Moreover, our findings in the biopsy performed on the proximal muscle suggest that the involvement of muscles in this disease can be more diffuse even in the absence of clinical evidence.

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