7 Zee DS, Robinson DA. A hypothetical explanation of saccadic oscillations. *Ann Neurol* 1979; **5**:405–14.
8 Dalmau J, Posner JB. Neurologic paraneoplastic antibodies. *Neurology* 1994;**44**:2241–6.
9 Dropcho EJ, Kline LB, Riser J. Antineuronal (anti-Ri) antibodies in a patient with opsoclonus-myoclonus. *Neurology* 1993;**43**: 207–11.
10 Fisher PG, Wechsler DS, Singer H. Anti-Hu antibody in a neuroblastoma associated paraneoplastic syndrome. *Paediatric Neurology* 1994;**10**:309–12.

Female predominance in spasmodic dysphonia

We were interested to see the sex prevalence results in the recent review of focal dystonia by Soland *et al*.[1] They found that of 956 patients with dystonia, 558 (58.4%) were female, for a ratio of 1.4:1. All but the cases of writer's cramp had more females than males. In their spasmodic dysphonia group the ratio was 2.6:1 female:male (n=36).

We have reviewed our database of the cases of spasmodic dysphonia seen at the Mayo Clinic, Scottsdale, between 1989 and 1996. There have been a total of 270 patients seen, 241 with adductor, and 29 with abductor spasmodic dysphonia. The overall ratio of females:males was 214:56 or 3.8:1. Thus females made up 79.3% of our population with spasmodic dysphonia. Broken down into subgroups, the female:male ratio was 4.1:1 for the adductors and 2.2:1 for the abductors. The mean age was 60 years for the men and 64.6 years for the women. Most (186) of these patients continue to be followed up for botulinum toxin injections.

Our results, in this much larger population of patients with spasmodic dysphonia, suggest a greater female predominance than most previous studies. The only other large magnitude study was by Blitzer and Brin[2] who reported 260 patients with idiopathic spasmodic dysphonia with a female:male ratio of 1.4:1. They later separated out their patients with abductor spasmodic dysphonia (n=32) and found a female:male ratio of 0.5:1. The other studies have been smaller, 21 and 41 patients, with a female:male ratio of 2.85:1[3] and 3.5:1.[4] Ludlow *et al*[5] had only 16 patients and all but one were females. Given the overall similarities (female predominance) among the published series, we do not think that the female predominance is due to ascertainment bias. Whether there are genetic, hormonal, or autoimmune factors causing this is unknown. Only one series of abductor spasmodic dysphonia showed a male predominance, although our series had a female predominance. Certainly further investigation is needed.

CHARLES H ADLER
BRIAN W EDWARDS
Department of Neurology

STEPHEN F BANSBERG
Department of Otolaryngology, Mayo Clinic, Scottsdale, AZ, USA

Correspondence to: Dr Charles H Adler, Parkinson's Disease and Movement Disorders Center, Department of Neurology, Mayo Clinic Scottsdale, 13400 E. Shea Boulevard, Scottsdale, AZ 85259, USA.

1 Soland VL, Bhatia KP, Marsden CD. Sex prevalence of focal dystonias. *J Neurol Neurosurg Psychiatry* 1996;**60**:204–5.
2 Blitzer A, Brin MF. Laryngeal dystonia: a series with botulinum toxin therapy. *Ann Otol Rhinol Laryngol* 1991;**100**:85–9.
3 Jankovic J, Ford J. Blepharospasm and orofacial-cervical dystonia: clinical and pharmacological findings in 100 patients. *Ann Neurol* 1983;**13**: 402–11.
4 Rosenfield DB. Spasmodic dysphonia. *Adv Neurol* 1988;**50**:537–45.
5 Ludlow CL, Nauton RF, Sedory SE, Schultz GM, Hallett M. Effects of botulinum toxin injections on speech in adductor spasmodic dysphonia. *Neurol* 1988;**38**:1220–5.

Recurrent Guillain-Barré syndrome and CNS demyelination

It is commonly assumed that multiple sclerosis exclusively affects the central nervous system but there is growing evidence that it is not so. There is evidence that dysfunction and tissue damage in Guillain-Barré syndrome, chronic idiopathic demyelinating polyneuropathy, and multiple sclerosis result from immune reactions within the peripheral or central nervous systems.[1] Although it is commonly assumed that multiple sclerosis exclusively affects the central nervous system, central nervous system demyelination has been suggested in chronic idiopathic demyelinating polyneuropathy.[2] There are few reports of acute inflammatory demyelinating neuropathy associated with multiple sclerosis.[3 4]

Here, we report a patient with a recurrent Guillain-Barré syndrome who developed a first episode of central nervous system demyelination several years later.

A 26 year old woman was admitted to our hospital because of an acute right hemiparesis. In 1977, at the age of seven, the patient gradually developed a tetraparesis with areflexia. The neurological deficit reached a peak two weeks after the onset of symptoms. Her CSF showed 217 mg/dl protein without cells. The motor and sensory nerve conduction study included median, ulnar, peroneal, and sural nerves. The motor nerve conduction velocities (NCVs) and distal motor latencies were 46 ms and 5 ms respectively in the median nerve, 50 ms and 4 ms in the ulnar nerve, 39 ms and 7 ms in the peroneal nerve. Sensory nerve conduction and EMG studies did not disclose abnormalities. She slowly improved and was discharged three weeks later without symptoms. The patient was asymptomatic until 1991, when she was readmitted because of an ascending weakness and distal paraesthesiae, two weeks after a flu syndrome with fever. Neurological examination disclosed paresis in all four limbs, more severe distally in the upper limbs and proximally in the lower limbs. She was areflexic. Sensory examination showed a mild deficit to pin prick, and vibration and reduced propioception was found distally in her four limbs. Lassegue sign was positive in both legs. The CSF contained 148 mg/dl protein and two leucocytes/mm^3. Nerve conduction studies disclosed an absence of F responses and dispersion of motor nerve compound action potentials. Median, radial, ulnar, peroneal, and sural nerves were examined. Motor and sensory NCVs were normal except in the peroneal nerve (40 ms) and median nerve (48 ms). Distal motor latencies were 5 ms, 4 ms, and 8 ms in median, ulnar, and peroneal nerves respectively. The neurological deficit reached a peak three weeks after onset and she had recovered fully in two months and was thereafter asymptomatic. A control electrophysiological study was performed several months later and no abnormalities were found .

In 1996, she was readmitted because of an acute right hemiparesis. Neurological examination showed a right pyramidal weakness, with right Babinski's sign and without sensory impairment. The haematological and blood chemical values were normal. A CSF analysis showed 24 mg/dl protein with three leucocytes/mm^3 and oligoclonal bands were detected. Brain MRI showed multiple T2 weighted hyperintense white matter lesions in both cerebral hemispheres, brain stem, and cerebellum. One of the lesions located in the left corona radiata displayed enhancement after gadolinium administration. High dose intravenous steroids were started and there was a pronounced clinical improvement. When the patient was discharged 10 days later the neurological examination was normal. Motor NCVs were greater than 50 ms in the arms and 45 ms in the legs, and no prolonged distal motor latencies were found. Transcranial magnetic stimulation showed a delay in central motor conduction in the right arm. An EMG examination did not show abnormalities. A new MRI performed two weeks later showed a decrease in inflammatory activity.

Our patient had a recurrent Guillain-Barré syndrome with subsequent development of a first attack of a central nervous system inflammatory demyelinating disease. Recurrent Guillain-Barré syndrome consists of multiple episodes of typical acute Guillain-Barré syndrome with CSF findings and EMG abnormalities during each individual episode similar to those described in the acute monophasic disease.[5] Although our patient does not fulfil criteria of clinically definite multiple sclerosis, MRI and CSF findings make the diagnosis very probable. Cranial MRI lesions have been found in chronic inflammatory demyelinating polyneuropathy although without clinical symptoms suggestive of multiple sclerosis.[2] Acute inflammatory demyelinating neuropathy associated with multiple sclerosis has not been described very often.[3 4] As far as we know, this is the first case of recurrent Guillain-Barré syndrome associated with a central nervous system demyelinating disease. In our patient both peripheral and central nervous myelin involvement were clinically symptomatic.

It seems that dysfunction and tissue damage in Guillain-Barré syndrome and multiple sclerosis result from impairment of immune regulation within the peripheral or central nervous system. Peripheral and central nervous system myelins exhibit a similar macromolecular organisation and some protein molecules are present in myelin from both systems; thus it is likely that, under certain circumstances, immune processes may be directed selectively against either peripheral nerves or CNS white matter. In the animal model, experimental allergic encephalomyelitis, the induction of the disease is accompanied by different degrees of peripheral involvement. Because closely related epitopes may be expressed in peripheral and central myelin, it is not unexpected that in some people there may be cross reactivity between peripheral and central nerve white matter.[6] On the other hand, it has been suggested that activated T lymphocytes may function as effector cells that exert cytotoxic activity towards Schwann cells or myelin.[1]