Matters arising

domyolysis,^{7,8} and in only one of the five cases were events adequately documented.⁹ In fact in one case thioridazine was started after an interval of one month without ill-effect.⁶ Another report described autonomic disturbances, of uncertain significance, occurring before and after an episode of NMS.¹⁰ In two other cases illness was separated by a 6 month interval. One of these, with a possible history only, was complicated by multiple seizures.¹¹ The other may have had NMS but the febrile episodes occurred with *Klebsiella pneumoniae* and *Staphylococcal septicemia*.¹² The source of two other referenced cases is unclear.⁶

Shalev and Munitz³ and Dr Adityanjee raise the important question of whether neuroleptic drugs can safely be used after an episode of NMS. Clearly if medication is restarted before recovery then relapse will occur. Abandoning full treatment of NMS is not likely to be helpful either. There is, however, no previously documented case acceptable to Dr Adityanjee's critical eye, where NMS has recurred after full recovery, allowing an interval of more than one month, for example. Indeed a tendency for non-recurrence is the generally accepted dogma,¹³ although under-recognition and under-reporting are likely. There is no reason why recurrences could not occur and if a patient requires further neuroleptic medication then caution suggests that neuroleptics should be started after at least three weeks recovery, preferably with gradual increments made under observation, of a less potent neuroleptic with intrinsic anticholinergic properties.¹⁴

In our case report¹⁵ fever, muscular rigidity, rhabdomyolysis and hyponatraemia occurred on two occasions seven months apart and without evidence of other contributory disease.¹⁴ Impaired consciousness should be a relatively late sign of NMS and "autonomic disturbances" likewise probably reflect severe illness alone. The interesting aspect of this case is the association of NMS with hyponatraemia and a stable drug regime, whereas in 53 of 56 cases NMS occurred after starting or increasing the dose of neuroleptics.^{14,16} Two other patients with hyponatraemia have now been described^{9,17} and in one of these cases NMS recurred, after 8 months on a stable drug regime, as in our case.¹⁷ The reason why dilutional hyponatraemia rather than an increase in neuroleptic medication may precipitate NMS is unclear. Conceivably an abrupt lowering of extracellular sodium concentration could lead to pre- or post-synaptic changes at the striatal dopaminergic synapse, with an effect anal-

ogous to the excessive stimulation of dopamine receptors postulated in NMS.¹⁴

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Distribution of multiple sclerosis in the United Kingdom

Sir: The excellent update of the UK distribution of multiple sclerosis by Swingle and Compston¹ emphasised the gradient in the association of HLA-DR2 and clinical multiple sclerosis prevalence which they showed declining progressively from SE England to NE Scotland. Their fig 11 showed the frequency of DR2 to be constant at 50% in multiple sclerosis cases throughout the UK whereas in controls it rose progressively from South to North.

My own general practice figures of 15 per 100,000 multiple sclerosis patients in an NHS list of 3,000 gives a remarkable prevalence of 500/10⁵ and has prompted a postal survey of other general practitioners in the New Forest area which showed 148 cases of multiple sclerosis in 140,765 people, giving a prevalence rate of 105/10⁵. However, from the four practices with Disease Registers the figure was 62 multiple sclerosis patients in 36,115 = 172/10⁵ (four other practices without such registers also reported figures giving prevalence rates of 143, 143, 145 and 150 per 10⁵ in a further population of 17,700). These figures are exceeded worldwide only by those in the Orkney and Shetland Isles, whose acid terrain of heather and wet peat is similar to the Forest.

HLA-status has not yet been defined in these cases but in unpublished figures obtained from the Wessex Region's Blood Transfusion Services, HLA-DR2 antigens are found in only 15% of 1,174 healthy volunteers for bone marrow transplantation in this region. This compares with the UK Transplantation Service's nationwide figure of 30% in those typed as cadaveric organ donors. The low Wessex figure for DR2 would suggest that this observed cluster of patients with multiple sclerosis is not genetically determined.

Using the DHSS Prolonged Study Leave scheme for general practitioners, I am now undertaking a survey of the multiple sclerosis patients (and controls) in the New Forest, designed to look at environmental factors, with specific interest in any significant differences pre-puberty, in the residential recreational habitats, and in animal contacts. If environmental histories are not significantly different in the two groups then the HLA-DR2 prevalence will be sought. As Swingle and Compston point out, if all are exposed to the same environment then only those with susceptible gene patterns will develop morbidity. Their graph would suggest that the 15% HLA-DR2 rate mentioned above in the healthy Wessex

community, will allow clear definition of any bias towards the 50% rate they demonstrated in UK multiple sclerosis patients. Conversely, a low level in the patients will reinforce the environmental argument for the observed New Forest cluster.

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Compston and Swingle reply:

Dr Markby's preliminary observations from the New Forest underline the need for case control studies of risk factors in multiple sclerosis from areas where the disease is inappropriately frequent or rare. Although we believe that genetic factors exert an important influence on the distribution of the disease, at least in the United Kingdom we are aware that there are too few areas where simultaneous prevalence and immunogenetic studies have been carried out, and there are exceptions to the rule that multiple sclerosis is common where DR2 is frequent in the at-risk population (for example, Hungarian Gypsies and some African tribes). Owing to misprinting of the horizontal axis in our recent paper, each point in figure 10 was inadvertently moved 10% to the left.

Subsequently¹ we have argued that under the multifactorial and polygenic model of susceptibility, the prevalence and specificity of genetic and environmental associations with multiple sclerosis will vary with changes in the absolute risk conferred by

individual susceptibility factors and their normal frequency. We agree that environmental exposure should be compared carefully in cases and controls in the New Forest where multiple sclerosis may be very common despite a relatively low frequency of DR2 in the normal population.

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C7 Radiculopathy: importance of scapular winging in clinical diagnosis

Sir: We were interested to read the article by Makin, Brown and Ebers.¹ They reported six cases of C7 radiculopathy in which weakness of the serratus anterior, manifested by winging of the scapula, was a feature. Recently, we have treated a further case.

A 35 year old electrical worker presented with two months history of neck pain, right brachialgia and deep infraclavicular and subscapular pain. The pain was worse on movements and straining. His right arm felt weak and numb. He gave a previous history of neck pain, which had no brachialgia or other element, and which responded to chiropractic attention.

On examination, there was a good range of cervical spine movements, weakness of the right triceps and an obviously winged scapula. The right triceps and supinator jerks were reduced. No sensory deficits or long tract signs were elicited. Plain radiographs showed a reversed cervical lordosis and narrowing of the C6/7 disc space. There was no instability on the flexion and extension views. Cervical myelography demonstrated a C6/7 disc prolapse compressing the right C7 roots. CSF protein content was 0.47 g/l.

The patient underwent anterior cervical discectomy and fusion. At operation a large sequestered disc fragment compressing the right C7 root was removed. Post-operatively, there was immediate symptomatic relief and return of power to triceps. Serratus anterior recovery was, however, slow.

The motor weakness in this case was confined to the triceps and serratus anterior contrasting with the cases reported where the pectoralis major, latissimus dorsi and extensor carpi ulnaris were frequently involved. Four of the cases reported had anterior cervical discectomy and fusion and all derived symptomatic relief and made a good neurological recovery.

It is perhaps surprising that weakness of serratus anterior does not occur more frequently in C7 radiculopathies. We suggest however, that if the serratus anterior function were to be tested in a more diligent manner according to the recommendations of Makin *et al.*, then what is apparently a rare clinical phenomenon may possibly be more commonplace. We would further comment that the thoracic distribution of pain seen in our case is not uncommon with C7 root compression but is frequently misinterpreted.

We are grateful to Dr Brian Phillips for referring this case for neurosurgical treatment.

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Book reviews

Subarachnoid Haemorrhage. By RP Sengupta and VL McAllister. (Pp 378; £114.00.) Berlin: Springer-Verlag, 1986.

Although it is 30 years since (Sir) John Walton published his famous monograph on subarachnoid haemorrhage, this is the first work in English that has appeared on the subject since then. During this time our

understanding of this complex neurosurgical catastrophe has expanded beyond recognition as has our ability to treat the lesions responsible. This is probably as opportune a time as ever for an up-to-date review of current knowledge on the subject, for following the explosion of both information and technology there are signs that the pace of advance may be beginning to slacken. Much of this present text is likely to be of permanent value.

Mr Sengupta and his radiological colleague, Dr VL McAllister, have produced what must surely become the definitive

guide to this field. The book is clearly written and beautifully produced. It contains a wealth of outstanding illustrations. Every aspect of subarachnoid haemorrhage and its management is covered in thorough, even recondite, detail, and the current literature is exhaustively reviewed. As is appropriate, the book commences with an historical review and ends with a chapter devoted to possible future developments.

Mr Sengupta is widely regarded as the premier aneurysm surgeon in Great Britain and the text draws generously on his own clinical experience and surgical results. The