of the graduating student, a questionnaire was designed to analyse the neurological disorders seen in general practice and the difficulties experienced in their management. Replies were received from 47 practitioners. Of the 710 consultations for neurological problems, 70% comprised headache and facial pain (35%), vertigo and dizziness (16.5%), seizures (8%) and neuropsychiatric (7%) and cerebrovascular disorders (3.5%). Of all difficulties experienced by practitioners, 20% involved undertaking the neurological examination and interpreting physical signs and 57.5% the evaluation and treatment of headache and facial pain (20.5%), vertigo and dizziness (20.5%), seizures (5.5%) and neuropsychiatric (7%) and cerebrovascular disorders (4%). These results are in accord with Murray's² findings and support his conclusion that teaching programmes are failing to provide the knowledge and teach the basic skills for practitioners to evaluate and manage effectively patients with common neurological problems.

How can process and outcome be more appropriately and efficaciously matched? The design of undergraduate and postgraduate programmes must be based on defined educational objectives that reflect practical experiences and societal needs,³ with relevant teaching strategies directed to the acquisition of problem solving skills through education in pathophysiological processes, rather than emphasising pattern recognition conveyed by instruction. The preparation of students for practice as primary care physicians will also necessitate structuring undergraduate training to take greater account of the need for attaining competence in the diagnostic process. This additional emphasis could be accommodated through more rigorous selection of items of knowledge the student should know. Notable in this respect is the opinion⁶ that most basic and clinical undergraduate neurology could be covered through discussion of problems presented by patients with 13 selected disorders. Such adjustments would also contribute towards rationalising the undergraduate curriculum, now critically overloaded with information⁷ to the point of manifestly declining standards of patient management.⁸

The degree of success of programmes depends to a large extent on the calibre of the teachers.⁹ Contemporary career prospects in academic medicine are highly geared to research achievement¹⁰ which, as a result, dominates higher training leading to graduates manifesting a significant short-

fall in competencies deemed necessary for a credible educator.¹¹ As a consequence, the effectiveness of any innovations is likely to remain problematical until such time as expertise in educational methods and teaching skills receives formal recognition and comparable status in institutional terms.^{10 11}

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Methylprednisolone therapy in multiple sclerosis

Sir; It is evident that there is some interest in neurological circles regarding the place of intravenous therapy with methylprednisolone in multiple sclerosis patients who are in acute relapse. A recent letter in this Journal from the London Hospital (1982;**45**:179-80) was enthusiastic about the response in six cases, and this form of treatment was the subject of some discus-

sion at the April meeting of the Association of British Neurologists. In 1976, one of us (MS) began to use intravenous methylprednisolone in selected multiple sclerosis cases, and since that time this Department has acquired considerable experience with this therapy. We would like to record our earlier findings which have been gathered by a retrospective analysis of our case notes.

Data were available on 61 patients who had received a total of 98 courses of treatment. The response was judged to be good in 32, fair in 47 and absent in 19. Classification of response into these three categories was by clinical observation at the end of the course of treatment or at early follow-up, coupled with the subjective views of the patient. All patients had definite multiple sclerosis according to the McAlpine criteria, and all had suffered a recent deterioration in their symptoms. In most cases, methylprednisolone was given as a slow intravenous injection of one gram, and courses of treatment were for five or seven days, tailing off with a further week of oral prednisolone.

It would be naive to over emphasise the benefits of steroids in multiple sclerosis, but accepting the limited expectations of treatment, we would like to make the following points. The natural history of relapse in multiple sclerosis does tend towards resolution and this is particularly so in patients admitted to hospital who have rest and skilled physiotherapy. Nevertheless, we have been greatly impressed by the rapidity and extent of improvement that we have observed in our methylprednisolone treated patients, a significant number having responded dramatically within twenty-four hours of the first injection. Adverse effects have been minimal, a transient metallic taste follows most injections, a reversible facial reddening is frequently seen and mild ankle oedema has occasionally been noticeable. These reactions are much less evident than in conventionally steroid treated patients and we have not had any serious complications. The fact that several patients have had multiple courses of intravenous methylprednisolone, as many as five in three cases, testifies to the fact that such treatment appears safe, but also suggests that this therapy of acute relapse does not influence the next relapse or the continuing progression of the disease.

In addition to a study comparing this form of treatment with standard ACTH therapy, we are also exploring the use of shorter and smaller intravenous courses of

methylprednisolone, and considering in selected cases the administration of this drug at home, thus avoiding the expense and inconvenience of a hospital admission. Given the prevailing ignorance regarding the pathogenesis of multiple sclerosis in general, and the effects of steroids in particular, the mechanism of the observed beneficial effects of intravenous methylprednisolone remain speculative.

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Neuropathy after organophosphorus compounds poisoning

Sir: I read with interest the report by Senanayake: Tricresyl phosphate neuropathy in Sri Lanka: a clinical and neurophysiological study with a three year follow up.¹ However, in the table there is an error: terminal latency is not measured in m/s, but in ms. In fig 2, either the diagram or the legend should specify the terminal latency values measured in ms. As the author shows, the cases presented display a series of important differences from triorthocresyl phosphate (TOCP) neuropathy.

In our 12 patients who had accidentally ingested TOCP-polluted alcohol, the paralysis began slowly in the lower limbs (15–30 days) and later (5–10 days) spread to the upper limbs.^{2,3} In the acute phase the patients displayed flaccid paralysis mainly in the lower limbs. After 2–3 months there were signs of both peripheral and central nervous system lesions, i.e., pyramidal signs. The patients showed a good recovery from the peripheral nerve lesions, 1–2 years after TOCP ingestion, but not from the deficits due to the pyramidal lesions. The latter not only failed to improve, but even extended more distally, especially in the lower limbs. We presume that these signs of CNS lesions were present in the distal zones before examination (1–2 years after intoxication), but became evident only after improvement of the peripheral nerve lesions, which had masked them. On reexamination of two patients 13 years after TOCP ingestion, we found a tendinous retraction of toes I–V and fingers II–V in flexion, and of knees both in flexion and adduction. The gait was spastic. At the same time there was a marked weakness distally, especially in the feet.

Two to three months after intoxication all the patients showed concurrent peripheral and central nervous system signs of lesions, predominantly in the distal portions of the axons, especially the longer ones, which indicated that the process characterising neuropathy by TOCP is of the “dying-back” type, as described first by Cavanagh.⁴ However, the clinical picture in five patients was of a mixed, particularly motor and distal, polyneuropathy. Re-examination performed 13 years later in two of five patients with a predominantly neuropathic disorder showed mainly pyramidal signs. There was marked improvement of peripheral nerve lesions and poor improvement of central lesions in both cases which made the initial predominantly neuropathic form pass into the predominantly spinal form of neuropathy with a clinical and electrophysiological picture resembling amyotrophic lateral sclerosis. This finding is in agreement with the observation of Morgan and Penovich⁵ who showed that the TOCP lesion described as delayed neurotoxicity was not a neuritis but rather a spinal cord syndrome. Our electrophysiological findings suggest that a mixed process of axonal degeneration and secondary demyelination underlies this predominantly neuropathic form. We assume that in our patients the secondary demyelination occurred in fibres which had already undergone degeneration followed by regeneration, as shown by Dyck *et al*⁶ in uraemic neuropathy. In the remaining seven patients, pyramidal signs prevailed, objective sensibility was not disturbed, and motor conduction velocity was normal. These patients thus had a predominantly spinal form of neuropathy. The electrophysiological data suggest that this form of neuropathy was mainly an axonal degeneration.

We also studied two cases of poisoning with organophosphorus insecticides, Diphtherex and Divipan, in which there was footdrop, distal weakness especially in the lower limbs, abolishing of the Achilles reflex, and mild pyramidal signs. Electrophysiological findings suggest that this neuropathy also was a distal axonopathy.³

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The significance of the incidental finding of basal ganglia calcification on computed tomography

Sir: Harrington *et al*¹ state that “basal ganglia calcification identified radiographically has been associated with any one of 24 conditions”. However, their list omits a relatively recently described group of disorders in which mitochondrial abnormalities have been found, the signs and symptoms of which may be of some interest when compared with those described in their series of patients. The definition of such an underlying disorder may not be completely academic as there is some indication that treatment, particularly by dietary manipulation or steroid therapy, may be beneficial in some patients.^{2,3}

Many patients have now been described, presenting with a wide variety of neurological symptoms, in whom a characteristic feature is the presence of structurally and/or functionally abnormal mitochondria identified on muscle biopsy. Although myopathy is usually a prominent feature, hence the term “mitochondrial myopathy”, there is increasing evidence that the mitochondrial disorder is not confined to muscle and the term “systemic mitochondrial disorder” may be more appropriate. How frequently the mitochondrial abnormality is primary as opposed to a non-specific secondary change, is still open to debate⁴ and in only a few of over 100 cases reported of suspected mitochondrial abnormality, has a specific biochemical lesion been determined.² However it is now possible to study many aspects of *in vivo* biochemical function with the technique of ³¹P nuclear magnetic resonance (NMR)⁵ and this will almost certainly contribute to