Group (Kennard, Newland and Ridley), and the Hammersmith Hospital Group (Gross, Legg, Lockwood and Pallis), in the treatment of inflammatory polyneuropathy by plasma exchange, have prompted us to report our own experiences in this hospital in the management since February, 1981 of eight patients with acute polyneuropathy, all of whom were managed by plasmaphoresis commenced at varying times after the onset of symptoms.

In three, where treatment was initiated on the 3rd, 5th and 14th days of the illness, discernible improvement began after the first phoresis and was sustained with subsequent exchanges. In two, commencement of treatment was deferred because of additional medical problems for longer periods, yet improvement began after three or four exchanges and proceeded rapidly. One with a one week history responded after three treatments but then was slow to recover completely. In one, an elderly male with chronic obstructive airways disease, who required ventilation, no benefit was seen, and the patient died.

Of most interest was a 25-year-old woman, 3 months post partum, who presented with a six day history of aching pain in the calves and back, followed by paraesthesiae in the hands and feet, progressive limb weakness and speech and swallowing difficulties. Examination revealed moderate weakness in the limbs (worse proximally), bilateral facial weakness, areflexia in the lower limbs and peripheral sensory loss to pain, light touch, and temperature. The CSF contained a protein of 0.6 g/l and no cells. Nerve conduction studies showed asymmetric motor slowing and diminished sensory action potentials. Daily 31, plasma exchanges were commenced on presentation, but over the next week further clinical deterioration occurred with worsening of bulbar symptoms, increasing facial weakness and further limb disability. On day 8 after five exchanges, considerable improvement was observed. Further plasmaphoreses were therefore deferred, improvement continued thereafter, but 10 days later she had a relapse of left facial paresis and dysphagia and dysphonia. Further phoreses were begun, and within three days noticeable improvement in speech, swallowing and facial movements began and continued steadily while eight exchanges were made, only to relapse seven days after the last of these.

It is our very distinct impression that with the exception of one patient, the natural history of these inflammatory neuropathies has been greatly affected, at

times immediately after the first exchange and that relapse may occur after too premature termination, with subsequent improvement following its reinstitution, and that the period of disability is very significantly shortened by the procedure.

JE NORMAN

GH PURDIE

RH RISCHBIETH

Departments of Haematology and Neurology,

The Queen Elizabeth Hospital,

Woodville Road,

Woodville, South Australia, 5011

The treatment of acute polyneuropathy by plasma exchange

Sir: We were interested to read the experience of Kennard *et al* (*J Neurol Neurosurg Psychiatry* 1982;45:847-50) in treating cases of acute polyneuropathy by plasma exchange. They concluded that only marginal benefit was obtained and rightly point out that as the procedure is expensive and time-consuming, optimistic reports should be regarded with caution. There is however, a major difference between the form of treatment used by this group and our own practice.

Nine of their twelve cases received their exchanges between twenty-four hours and eighteen days after they had reached their lowest clinical rating. The reason for this delay in initiating treatment was not made clear.

In line with other published experience (Brettell *et al* 1978¹; Gross *et al* 1982²) we offer plasma exchange whilst the neurological disease is still progressing and before a plateau phase has been reached. In practice, this is at a stage when the need for assisted ventilation becomes imminent. A further point of difference between our experience and that of Kennard *et al* group is the routine use of at least two units of fresh frozen plasma for each exchange performed.

Clinical trials are presently taking place. Until a trial reports on the use of plasma exchange in treating polyneuropathy at a stage that the disease is still progressing, then we feel it is still justified to offer this treatment to cases in whom artificial ventilation may be necessary.

MICHAEL LP GROSS

The Royal Free Hospital,

Pond St, London NW3 2QG, UK

References

- 1 Brettell RP, Gross M, Legg NJ, Lockwood M, Pallis C. *Lancet* 1978;2:1100.
- 2 Gross MLP, Legg NJ, Lockwood M, Pallis C. *J Neurol. Neurosurg Psychiatry* 1982;45: 675-9.

Kennard *et al* reply:

We welcome the opportunity to reply to Dr Gross' letter since his criticism of our apparent delay in initiating treatment is based on a reading of the table and not of the Results section in our paper. He has, therefore, relied on the functional grading scale used, which in fact allows deterioration of the patient's condition to continue within one grade. All patients, except one (case 8) as we mentioned in the results, were still deteriorating on entry into the trial and plasma exchange was commenced as soon as the patients reached our hospital.

Although there are theoretical reasons why fresh frozen plasma (FFP) may be of value in immune complex disorders (for example systemic lupus erythematosus)¹ there is no evidence that FFP adds any benefit to the use of plasma protein fraction (PPF) alone as replacement fluid in possible antibody-related conditions such as the Guillain-Barré syndrome. Indeed there are published reports claiming early success using PPF alone² as in our series. In view of the increased side-effects, including allergic reactions, when FFP is used as a replacement fluid³ its use seems somewhat capricious when the value of the procedure in this condition appears in doubt.

References

- 1 Plasmaphoresis II: Review of clinical experience. Wysesbeck AJ, Smith JW, Krakauer RS. *Plasma Therapy* 1981;2:61-71.
- 2 Plasma exchange in Guillain-Barré syndrome. Durward WF, Burnett AK, Watkins R, Reid JM. *Br Med J*. 1981;283:794.
- 3 Citrate induced hypocalcaemia during cell separation. Watson DK, Penny AF, Marshall RW, Robinson EAE. *Br J Haemat* 1980;44:503-7.

C KENNARD

AC NEWLAND

A RIDLEY

This correspondence is now closed, pending the results of the controlled clinical trials presently underway. — Editor.