extrathecal shunting to be pooled in a few years time to try to assess whether flattening the syrinx is indeed worthwhile.

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Ruptured intracranial aneurysms

Sir: There are two points we would like to make with regard to the article "Ruptured intracranial aneurysms: has the incidence of early rebleeding been over-estimated?" *J Neurol Neurosurg Psychiatr* 1982; 45:774-9. The author attempts to comment on the occurrence of rebleeding and neurological deterioration following an intracranial aneurysmal rupture, having given the majority of his patients tranexamic acid, a drug used to prevent recurrent haemorrhage and a drug suspected of producing ischaemic neurological deterioration. We have recently demonstrated that the pattern of cerebral blood flow (CBF), measured daily over a three week period, differs considerably in patients on tranexamic acid compared to patients on no drug therapy. There is a substantially greater fall in CBF in the patients on tranexamic acid during the second week after haemorrhage.¹ The author, in his study, is clearly examining the effect of tranexamic acid on a group of patients following an aneurysmal rupture and not the natural history of the disease. Thus the comparing of this series with others where tranexamic acid was not used in a similar way is meaningless.

All neurosurgeons are aware of the difficulty of establishing the occurrence of a recurrent haemorrhage in patients already possessing an abnormal CSF and in the limited value of the CT scan in these circumstances. Small haemorrhages causing sudden and transient clinical deterioration, as suggested by the author are extremely

difficult to diagnose by these methods. Contrary to the author's statement it is high impossible at the time of surgery, especially as the author apparently does not operate for at least seven days from such a deterioration, to say whether the patient has had a small recurrent haemorrhage.

The paper, we feel, has asked the wrong question, considering the methodology, and not surprisingly produces a very questionable answer.

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Maurice-Williams replies:

Both points raised by Mr Neil-Dwyer and Mr Sharr are dealt with at length in the discussion section of my recent paper. I agree with them that it is possible that the administration of the anti-fibolytic agent tranexamic acid may have modified the natural history of the cases studied but I would point out that there is still considerable controversy as to whether or not anti-fibolytic agents have any effect on patients who have had a subarachnoid haemorrhage. As pointed out in the article, if the tranexamic acid has had any effect, it has been to replace one problem (rebleeding) with another problem (non-haemorrhagic deterioration), the classically described characteristic time-course of rebleeding having shifted to non-haemorrhagic deterioration while the time-course of rebleeding has become flattened and uniform. This would seem a very surprising effect. Furthermore the total number of episodes of rebleeding and non-haemorrhagic deterioration added together in the first three weeks of the study was very close to the number of "rebleeds" alone that the Co-operative Study would have predicted during this period. The confirmed rebleeds in my study had a very high mortality (90%), but the mortality of the cases of rebleeding and non-haemorrhagic deterioration added together (55%) approximated to the level of mortality of rebleeding reported by earlier papers. All these phenomena would

appear to be better explained by the hypothesis that earlier studies had confused episodes of non-haemorrhagic deterioration with rebleeding during the first few weeks after the subarachnoid haemorrhage. It is also possible that confusion between rebleeding and other causes of deterioration may account for the fact that some studies have reported anti-fibrinolytics to reduce the incidence of rebleeding while other studies have reported no such effect.

With regard to the second point raised by Mr Neil-Dwyer and Mr Sharr, it is indeed possible that some episodes of non-haemorrhagic deterioration were very minor rebleeds, too small in extent to be detected by repeat CT scan or repeat lumbar puncture. However this would not explain the totally different time-courses of rebleeding and non-haemorrhagic deterioration, nor the fact that patients who had an episode of non-haemorrhagic deterioration were not more liable to later frank rebleeding than those who had a more stable course. In addition, despite what Messrs Sharr and Neil-Dwyer say several patients came to surgery within 7-10 days of an episode of non-haemorrhagic deterioration and in no case was any evidence of a limited local rebleed found. The same was true of the eight patients who died after a period of non-haemorrhagic deterioration and who came to post-mortem examination.

I am interested by their statement that tranexamic acid appears to lower the cerebral blood-flow in patients during the second week after haemorrhage. This appears to be at variance with the findings of a recently published study from their unit on cerebral blood flow after subarachnoid haemorrhage in older patients which states that, in this group of patients at any rate, tranexamic acid did not selectively depress the cerebral blood flow.¹

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The treatment of acute polyneuropathy by plasma exchange

Sir: The different experiences reported to this Journal in September and August 1982, respectively, by the London Hospital

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 plasmapheresis 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 plasmaphereses 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.

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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.

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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.

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C KENNARD

AC NEWLAND

A RIDLEY

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