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-fibrotic 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-fibrotic 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 appeared 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 stated 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

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