Page et al reply:

We appreciate the opportunity to reply to the comments made by Dr Bernard Williams on our paper.¹ When one attempts to understand the mechanism of bleeding in minor trauma in patients with arachnoid cysts of the middle fossa (ACMF) the anatomy of the cysts has to be considered. They are for the most part intra-arachnoidal. Many workers have attempted to ascertain whether they are in communication with the general CSF space and some authors²⁻⁴ and ourselves have demonstrated communication with the basal cisterns at surgery. These represent the minority of cases. Gallasi⁵ and Ruscadella,⁶ working separately, are some of the most recent workers to publish equivocal results. There is also no specific evidence that non-communicating cysts are those that have undergone decompensation.

In addition to this, abnormal veins have been demonstrated at surgery, both stretched around the cysts and actually crossing the cysts⁷ (personal observation). Guidacella⁸ has shown persisting foetal venous sinuses in association with congenital ACMF, giving further evidence for the presence of abnormal venous channels ipsilateral to ACMF.

We, too, consider the final common pathway is rupture of the veins either bridging the subdural space and/or the ACMF themselves. To consider the first theory, the extra-cerebral paracortical CSF spaces are normally relatively small, excepting the basal cisterns. Thus turbulent flow is highly unlikely to develop in such a system in response to minor trauma. However, if a large communicating cystic space is present there is more potential for turbulent flow to develop both within the cyst itself and to be transmitted from the general CSF space via the communicating channels. If such an eventuality occurs, as we believe it does, abnormal vessels may then be stretched or compressed resulting in haemorrhage, either subdurally or intracystically.

With regard to the second theory, where subdural intracystic haemorrhage occurs in the presence of non-communicating ACMF, the response to minor trauma again results in abnormal redistribution of CSF within the calvarium. This hypothesis is not new and was initially proposed by Van der Meche and Braakman in 1983⁹ when they were discussing non-haemorrhagic complication in association with ACMF. Again we postulate that the lack of deformability of a non-communicating ACMF would lead to shearing forces on adjacent bridging veins.

We would like to point out that at no time

in this paper or in our other work have we stated that contralateral subdural haematoma occur frequently. To be precise, in our study, only two patients had evidence of contralateral haematoma and only three other cases have been specifically reported. We do, however, agree with Mr Williams that contralateral haematomas represent a difficult problem when one is hypothesising the mechanism of their formation in the absence of major trauma, in view of the presence of a presumably normal subdural and subarachnoid space on that side. It is interesting to note that none of the cases reported had unilateral contralateral subdural haematomas in association with ACMF. We believe that it is the lack of deformability induced by the presence of the ACMF that results in the potential for the development of bilateral haematomas due to rupture of bridging veins even if they are apparently of normal anatomical configuration.

AC PAGE

Department of Radiodiagnosis,
King's College Hospital,
Denmark Hill, London SE5 9RS, UK

RM PAXTON

Department of Radiology,
Freedom Fields Hospital

D MOHAN

Department of Neurosurgery,
Freedom Fields Hospital,
Plymouth, Devon PL4 8NL, UK

References

- 1 Page A, Paxton RM, Mohan D. A Reappraisal of the relationship between arachnoid cysts of the middle fossa and chronic subdural haematoma. *J Neurol Neurosurg Psychiatry* 1987;**50**:1001-7.
- 2 Robinson RG. Congenital cysts of the brain: arachnoid malformations. *Progr Neurosurgery* 1971;**20**:133-74.
- 3 Varma TRK, Sedzimir CB, Miles JB. Post traumatic complications of arachnoid cysts and temporal agenesis. *J Neurol Neurosurg Psychiatry* 1981;**44**:29-34.
- 4 Nagoulitch I, Petrovitch M. Les collections liquidiennes extracerebrales chez l'enfant. *Lyon Chir* 56: 726-39.
- 5 Galassi E, Piazza G, Gaist G, Frank F. Arachnoid cysts of the middle cranial fossa: a clinical and radiological study of 25 cases treated surgically. *Surg Neurol* 1980;**14**: 211-9.
- 6 Ruscadella J, Guardia E, Santos FM, Pos, Carvajal A. Dynamic study of arachnoid cyst with metrizamide. *Neuroradiology* 1980;**20**:185-9.
- 7 Dyke P, Gruskin P. Supratentorial arachnoid cysts in adults. *Arch Neurology* 1977;**34**:276-9.
- 8 Guidicella G, Hasson J, Choux M, Tonon C. Les kystes arachnoïdiens supratentoriaux. *J Neuroradiology* 1982;**9**:179-201.
- 9 Van der Meche GAA, Braakman R. Arachnoid cysts in the middle cranial fossa: care and treatment of progressive and non-progressive symptoms. *J Neurol Neurosurg Psychiatry* 1983;**46**:1102-7.

Sporadic adult onset distal myopathy

Sir: In the January issue of the Journal appeared an interesting letter of Orsico et al,¹ which described a case of late onset distal myopathy with histological evidence of proximal muscular involvement in the absence of clinical findings. The peripheral nervous system was reported to be normal. We would like to report the case of a young woman affected by late onset distal myopathy, with proximal muscular involvement and signs of peripheral neuropathy. A 32 year old woman complained of running and then of walking difficulties since the age of 19. At 21 she could climb stairs but could not walk on toes and bilateral stepping gait was present. At that time she was admitted to another hospital and the clinical evaluation revealed bilateral wasting of leg muscles, with marked distal weakness. Ankle reflexes were reduced. Upper limbs were completely normal. Serum creatine kinase levels were about three times normal values. An EMG study, performed only on the lower limbs, suggested a peripheral neuropathy and she was discharged with the diagnosis of "chronic peripheral neuropathy of undiagnosed origin, characterised by reduction of nerve conduction velocity". Her symptoms slowly worsened and recently she was admitted to our hospital. She was an obese woman, confined to a wheelchair, unable to walk, even though she could stand without great difficulty. Neurological examination showed severe wasting of lower limb muscles, with complete abolition of distal movements. Proximal movements were possible against moderate opposition. Upper limbs were less evidently affected, with mild global reduction of strength, without wasting. Reflexes were reduced, with bilateral absence of ankle jerks. Serum creatine kinase was about 3450 U/l (normal up to 180 U/l). An EMG examination revealed occasional insertional activity and very low