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.

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Sporadic adult onset distal myopathy

Sir: In the January issue of the Journal appeared an interesting letter of Orrioco *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

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amplitude interferential pattern in all muscles of lower limbs, especially in tibialis anterior. Many polyphasic potentials were recorded in all examined muscles. In the arms we also found signs of myopathic involvement, with predominant distal distribution, but they were of lesser degree than in the legs. Peripheral nerves evaluation disclosed no pathological changes in the arms, but showed a decrease in the motor conduction velocity in the peroneal nerve bilaterally (42 and 43 m/s, normal 49.5, SD 3.9). A neuromuscular biopsy was performed. Sural nerve biopsy revealed an increased number of clusters of small myelinated fibres, with a density of myelinated fibres at the lower limit of normal values (5850/mm²) and a shift to the left of the histogram of myelinated fibres, indicating an abnormal prevalence of small ones. There were no other abnormalities on electron microscopy. Biceps brachii muscular biopsy was consistent with a primary muscular disease. Endo- and perimysial connective tissues were markedly increased. Nuclear centralisation was present in some fibres, as well as fibre splitting and abnormal size variability. Inflammatory reactions were lacking. Histo-

chemistry (ATPase 9.4-4.6-4.3, NADH, PAS, Oil Red O, acid phosphatase) was normal. Some subsarcolemmal myeloid bodies but no vacuoles were seen on electron microscopy.² Our findings are in agreement with the data of Orrico *et al.*,¹ which suggest that distal myopathy could lead to generalised muscular involvement. This fact is more evident if patients are examined long after the onset of the disease, as in our case. The histological features of proximal muscular involvement are much more prominent than are suggested by the clinical evidence. We also found mild signs of peripheral neuropathy, and this finding was already described by Edstrom³ and by Stalberg and Ekstedt,⁴ but not confirmed by Markesbery *et al.*⁵ These morphological differences are probably due to the existence of different variants of the disease. In our case EMG evidence of peripheral nerves involvement were present since the earliest stage of the disease, so that they contributed to the initial misdiagnosis.

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Book reviews

Handbook of Parkinson's Disease. Edited by William C Koller. (Pp 520; \$79.75 (USA and Canada) \$95.50 (all others).) New York: Marcel Dekker, 1987.

It is a tribute to the progress achieved by modern research that the last decade has seen such a glut of symposia and proceedings devoted to Parkinson's disease and its treatment. But each of these publications is ephemeral and one suspects is of limited value to clinicians and neuroscientists. Perhaps daunted by the speed of progress and the fear of being quickly out of date writers have yielded few textbooks or handbooks—which by nature should be more comprehensive, yet more durable. Professor Koller of Loyola University, Maywood, Illinois, has braved this challenge, armed with contributions from neuroscientists, many well known, from the States and a few from Europe and Australia.

The result is a remarkably complete and detailed analysis of Parkinson's disease and of related topics, encompassed in 26 chapters and 475 pages. All essential aspects of the basic sciences and animal models are

covered; clinical sections embrace the mental state, autonomic disorders, dysarthria and classification. There are eight chapters on treatment describing all the drugs, pharmacology and a valuable section on stereotactic surgery which may be underused in the UK. The topical brain grafting is not neglected. Rehabilitation and psychosocial aspects are dealt with. And, there is a useful appendix listing in detail the clinical scales, disability and functional staging of the disease, lists of drugs used, their dose and cost; the addresses of national patient help organisations are appended.

I have only one grouse: the exhaustive references are in places chaotic—chapter 17 omits paper titles; chapters 5, 13, 15 and 26 list authors alphabetically whilst others are in numerical order.

Considering the enormity of the task confronted, Koller is to be congratulated in producing a most useful handbook, filled with detail and expertise. It is unfair to pick out individual contributions, but it would be even more unfair not to mention a superb and beautifully illustrated History of Parkinson's disease by Kenneth Tyler which sets such a high tone for the succeeding chapters. There are controversial statements and emphases which may provoke some

readers, but overall I would strongly recommend this book to all practising neurologists, trainees, and to investigators of Parkinson's disease.

JMS PEARCE

Physiology and Pathophysiology of the Cerebrospinal Fluid. By Hugh Davson, Keasley Welch, Malcolm B Segal. (Pp 1010; £120.00.) Edinburgh: Churchill Livingstone, 1986.

Davson's work and writings have inspired a wide spectrum of neuroscientists, basic and clinical, for almost half a century. *The Physiology of the Ocular and Cerebrospinal Fluid* was published in 1956 and subsequently expanded as *Physiology of the Cerebrospinal Fluid* in 1967. That book provided, for me, a voyage of genuine intellectual excitement both by its elucidation of the principles and by its idiosyncratic exposure of treasured observations long buried in the literature. My pleasure at being asked to review this third edition would have been all the greater had the invitation