Horner's syndrome because Müller's muscle contributes only to about 1.5 mm lid elevation.² More than sympathetic dysfunction alone is needed to cause the presented ptosis.

We recently reported a similar case in a 41 year old woman demonstrating involvement of both Müller's muscle and levator palpebrae superioris clinically and pharmacologically.³ Orbital imaging showed enlargement of the levator palpebrae/rectus superior complex, which also suggests a local pathology. We proposed a local, possibly inflammatory process of the lid surface anatomy as described by Rice and Gray.⁴ A similar explanation might account for the mild aching at the frontal region of the affected side in the patient of Sieb and Hartmann.

More recently a 62 year old man presented to our clinic with a 3 year history of recurrent right complete ptosis lasting 7 to 10 days, occurring once or twice a year with full recovery. The onset of the ptosis was associated with erythema and mild periorbital aching and swelling.

Unfortunately we have not yet been able to find an appropriate treatment. An initial trial with pyridostigmine using the rationale that 15%–20% of patients with myasthenia gravis have negative acetyl choline antibody⁵ was disappointing,³ as was treatment with non-steroidal anti-inflammatory drugs and oral prednisolone.^{1,3} Sieb and Hartmann tried the serotonin antagonist pizotifen and prednisolone also without significant improvement.

We do not know the reason for the recurrent complete ptosis in our two patients. Neither can we be sure that the siblings described by Sieb and Hartmann have the same disorder, particularly as in these patients the side of the ptosis alternated.¹ However, we suggest that the syndrome must be due to local disease causing loss of function of the levator palpebrae superioris muscle either alone or in addition to Müller's muscle.

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- 1 Sieb JP, Hartmann A. Relapsing alternating ptosis in two siblings. *J Neurol Neurosurg Psychiatry* 2000;69:282.
- 2 Small RG, et al. The effect of phenylephrine on Müller's muscle. A blepharogram study of eyelid motion. *Ophthalmology* 1995;102:599–606.
- 3 Petzold A, Plant GT. Recurrent ptosis in an adult due to isolated paresis of the levator palpebrae superioris and Müller's muscle of unknown aetiology. *Neuro-Ophthalmology* 2000;24:279–82.
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Sieb and Hartmann reply:

We greatly appreciate the interesting comments of Petzold and Plant who described two sporadic patients showing clinical similarities to our siblings.

Unfortunately, we cannot provide additional evidence for their suggestion that local disease of the Müller's muscle and the levator palpebrae superioris might cause relapsing, alternating ptosis. Magnetic resonance studies of the orbita as proposed by Petzold and Plant did not show any pathology in one of our patients during an episode of ptosis.

The literature states commonly that oculo-sympathetic paresis results only in slight

upper eyelid ptosis of 1 to 2 mm.¹ However, our clinical experience in patients with Horner's syndrome due to carotid artery dissection is different. We still think that an intermittent sympathetic dysfunction is the most likely explanation for the familial disorder observed by us. Hopefully, investigation of additional patients will shed light on the pathology of this unusual disorder.

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- 1 Patel AD. Autonomic nervous system and the eye. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology: the autonomic nervous system, part I*. Amsterdam: Elsevier, 1999:399–435.

Management of intracranial bleeding associated with anticoagulation: balancing the risk of further bleeding against thromboembolism from prosthetic heart valves

We read with interest the article by Crawley *et al.*¹ We respectfully take issue with the authors.

(1) In this article on the management of intracranial bleeding associated with anticoagulation, the authors reported on a patient who had an "intracerebral haematoma" and later developed two new "intracranial haematomas" after heparin therapy. We hope that the authors were referring to either lobar haemorrhage or basal ganglia haemorrhage. If the patient had a subdural haematoma, also classified under intracranial haemorrhage, then a surgical procedure as well as discontinuation of anticoagulation and reversal would have been the preferred treatment.

(2) In their review of the literature, the authors did not discuss the articles by Wijidicks *et al* and Babikian *et al* on the relative safety of discontinuation of oral warfarin after intracranial haemorrhage in patients with mechanical heart valves.^{2,3} These authors have found that temporary discontinuation of warfarin for 1 to 2 weeks was relatively safe.

(3) Crawley *et al* estimated a 0.016% daily risk of embolism or 0.67% over 6 weeks.¹ In our experience, although the risk is relatively low, it is in the order of 3% over 30 days.⁴

(4) Crawley *et al* correctly stated that having reversed anticoagulation in patients with prosthetic heart valves, it is uncertain when to restart it.¹ None of the patients in our experience had recurrence of intracranial haemorrhage on restarting warfarin (after a short period of discontinuation) during their stay in hospital.⁴ Thus we do not recommend a prolonged period of warfarin discontinuation in patients who are at high risk of embolisation. Additionally we recommend that these patients be screened with echocardiography in evaluating the risk-benefit ratio of warfarin discontinuation and the urgency of restarting anticoagulation.

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- 1 Crawley F, Bevan D, Wren D. Management of intracranial bleeding associated with anticoagulation: balancing the risk of further bleeding against thromboembolism from prosthetic heart valves. *J Neurol Neurosurg Psychiatry* 2000;69:396–8.
- 2 Wijidicks EFM, Schievink WI, Brown RD, *et al*. The dilemma of discontinuation of anticoagulation therapy for patients with intracranial haemorrhage and mechanical heart valves. *Neurosurgery* 1998;42:769–73.
- 3 Babikian VL, Kase CS, Pessin MS, *et al*. Resumption of anticoagulation after intracranial bleeding in patients with prosthetic heart valves. *Stroke* 1988;19:407–8.
- 4 Phan TG, Kott M, Wijidicks EFM. Safety of discontinuation and resumption of anticoagulation in patients with intracranial hemorrhage at high thromboembolic risk. *Arch Neurol* 2000;57:1710–13.

Wren replies:

We fully agree with Phan and Wijidicks that if a patient has a subdural haematoma while on treatment with warfarin surgical drainage might be required. The particular patient reported had recurrent lobar haemorrhage.¹ Photographs of the brain CT were supplied but not published.

We are aware of the articles by Wijidicks *et al*² and Babikian *et al*³ as well as the recently published article by Phan *et al*,⁴ which is an extension of the previous article published by Wijidicks *et al*.¹ The dilemma of reinstituting anticoagulation for patients with cardioembolic sources and intracranial haemorrhages is discussed by Hacke⁵ in the editorial accompanying the recent paper by Phan *et al*.⁴ Of particular interest is the apparent paradox between the reported embolic risk without anticoagulation with modern artificial heart valves in the range of 4 per 100 patient-years and observed risk in the order of 3% over 30 days in the retrospective studies of Phan *et al* going up to 20% in the study of Bertram *et al*.^{4,6}

We agree with Phan and Wijidicks and Wijidicks *et al* that patients at high risk of embolisation should have a limited period of warfarin discontinuation and that each patient needs to be assessed individually as suggested by Hacke.⁵ We also agree with Hacke's suggestion that a prospective registry would be very useful given the difficulties of setting up any form of randomised control trial. The potential prothrombotic effects of haemorrhage and reversal of anticoagulation are also subjects that merit investigation with, for example, thromboelastography.

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- 1 Crawley F, Bevan D, Wren D. Management of intracranial bleeding associated with anticoagulation: balancing the risk of further bleeding against thromboembolism from prosthetic heart valves. *J Neurol Neurosurg Psychiatry* 2000;69:396–8.
- 2 Wijidicks EF, Schievink WI, Borwn RD, *et al*. The dilemma of discontinuation of anticoagulation therapy for patients with intracranial haemorrhage and mechanical heart valves. *Neurosurgery*. 1998;42:769–73.
- 3 Babikian VL, Kase CS, Pessin MS, *et al*. Resumption of anticoagulation after intracranial bleeding in patients with prosthetic heart valves. *Stroke* 1988;19:407–8.
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- 5 Hacke W. The dilemma of reinstituting anticoagulation for patients with cardioembolic sources and intracranial haemorrhage: how wide is the strait between Skylla and Karybdis? *Arch Neurol* 2000;57:1682–4.

- 6 Bertram M, Bonsanto M, Hacke W, *et al*. Managing the therapeutic dilemma: patients with spontaneous intracerebral haemorrhage and urgent need for anticoagulation. *J Neurol* 2000; 247:209-14.

Delirium episode as a sign of undetected dementia among community dwelling elderly subjects

Rahkonen *et al*¹ examine the complex issue of outcome after an episode of delirium and, in particular, whether an episode acts as an indicator of undetected dementia. Poor cognitive outcomes, including dementia, are well recognised after delirium but it is unclear whether delirium is merely a marker for dementia or if an episode contributes to the development of enduring cognitive impairment, possibly by a neurotoxic or kindling-type mechanism. Patients with mild cognitive impairment were not excluded at the outset but it is clear that there was a relation between mini mental state examination (MMSE) scores immediately after resolution of DSM IIIR delirium and risk of subsequent dementia. Given that persistent symptoms are common in delirium,² one possible explanation is that these cases reflect unresolved subclinical delirium or that incomplete treatment of delirium is a risk factor for subsequent cognitive decline.³ The data should allow estimation of the frequency of dementia in those patients with MMSE scores within the normal range after full resolution of DSM IIIR delirium. This information is highly relevant to planning of post-delirium management and although the MMSE does not provide a sensitive measure of the neuropsychological disturbances of delirium, may shed some light on whether these findings relate to persistent cognitive deficits of delirium that are measured on the MMSE.

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- 1 Rahkonen T, Luukainen-Markkula R, Paanila S, *et al*. Delirium episode as a sign of undetected dementia among community dwelling elderly subjects: a 2 year follow up study. *J Neurol Neurosurg Psychiatry* 2000;69:519-21.
- 2 Levkoff SE, Evans DA, Liptzin B, *et al*. Delirium: the occurrence and persistence of symptoms among elderly hospitalised patients. *Arch Intern Med* 1992;152:334-40.
- 3 Meagher DJ, Trzepacz PT. Delirium phenomenology illuminates pathophysiology, management and course. *J Geriatr Psychiatry Neurol* 1998;11:150-7.

Rahkonen and Sulkava reply:

We thank Meagher for his interest and for his comments on our paper. In the Kuopio delirium study, the disappearance of delirium (according to the DSM-III-R) was ascertained carefully. Instead of being discharged to their own homes (after the treatment of delirium and its underlying causes) the patients were transferred to a rehabilitation centre. During the 7 to 14 days stay in the rehabilitation ward the disappearance of delirium was confirmed. However, some symptoms also appearing in delirium, such as disturbances of the sleep-wake cycle or mild memory deficits, may have still been detected in these elderly patients, but they did not fulfil the diagnostic criteria for delirium any more.

In the Kuopio delirium study, 27% of the community dwelling elderly patients without serious predisposing factors for delirium were

diagnosed as having mild dementia immediately after the resolution of the delirium. The rate of subsequent dementia in the remaining non-demented patients was three out of 11 (27%) with the initial score on the mini mental status examination 24 or over after the delirium subsided (the incidence rate of dementia was 18.2/100 person-years). In the patients with an MMSE score less than 24 the rate of subsequent dementia after resolution of delirium was 11 out of 26 patients (42%) (the incidence rate of dementia was 25.4/100 person-years). However, in our article we published only the rate of the subsequent dementia in all the patients. The number of patients in the groups based on the MMSE scores was small and the usefulness of the scores in diagnosing dementia was limited in these elderly patients (mean age 82 years). In our study, no corrections were made to the scores of the MMSE test because of vision or hearing impairment or limited education.

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CORRECTIONS

Bhakta B, Cozens JA, Chamberlain MA, *et al*. The impact of botulinum toxin type A on disability and carer burden due to arm spasticity after stroke: a randomised double blind placebo controlled trial. *J Neurol Neurosurg Psychiatry* 2000;69:217-21.

In table 2, p219 in the column relating to carer burden, BT-A, the values -0.77 (-1.8, 0) should read -0.67(-1.8, 0) and -1.0 (-2.0, 0) should read -0.5(-2.0, 0)

Bradshaw JL, Mattingley JB. Allodynia: a sensory analogue of motor mirror neurons in a hyperaesthetic patient reporting instantaneous discomfort to another's perceived sudden minor injury? *J Neurol Neurosurg Psychiatry* 2000;70:135-6. On page 136, para 3, line 8, the phrase "don't do that (meaning not to show him); he actually felt it" should have read "don't do that (meaning not to show him suddenly); he actually felt it".

Without the word *suddenly* the significance may be lost—It was because he "received the message suddenly" that he felt it (the experience of hyperaesthesia).

BOOK REVIEWS

Essentials of Clinical Epilepsy: 2nd Edition. By A GUBERMAN and J BRUNI. (Pp 207, £27.50). Oxford: Butterworth Heinemann, 1999. ISBN 0 7506 7109 2.

The authors of this text have sought to provide a handbook in which they present the core clinical knowledge on epilepsy while aiming to be comprehensive and up to date. Chapters cover the usual topics from epidemiology through to surgical treatment and this slim volume certainly packs in a huge amount of information. The reading lists are

very useful with statistical information and data from up to date studies. Inevitably a book whose text is made up almost entirely of bullet points proves very difficult to read, and this format does not allow enough space for critical appraisal of the reference studies which the reader will hopefully be prompted to look up. The authors practical approach to managing epilepsy is useful and I would have liked to have seen more of this. Anticonvulsant drugs are listed with standard accounts of pharmacokinetic and pharmacodynamic data. Most readers would have been interested in a discussion of the relative merits of the newer anticonvulsant drugs and the quality of evidence supporting their use in comparison with the older drugs.

This is an excellent little handbook well suited as an introductory reference text for the authors stated target audience of residents in training and general physicians.

S J WROE

Central Nervous System Diseases: Innovative Animal Models From Lab to Clinic. Edited by D I P EMERICH, R L DEAN III, and P R SANBERG. (Pp 512, US\$145.00). New Jersey: Humana Press, 2000. ISBN 0 896 03724 X.

This book takes as its main theme the neurodegenerative disorders and animal models of them for assessment and treatment. The book divides into five sections and concentrates on Alzheimer's, Parkinson's and Huntington's disease along with traumatic-ischaemic brain damage before concluding with some clinically relevant topics. The authors of each chapter are leaders in their field so that the accounts are generally well written, up to date, and authoritative—although some fields move so fast that some chapters in this book are already out of date. For example, the chapter on transgenic Huntington's disease mouse models by Gill Bates and coworkers is already missing some new interesting pieces of information on the possible pathogenesis of this condition. This having been said the large section, devoted to Huntington's disease in this book is welcome given just what an exciting field of research this is at the moment.

The overall structure of this book using sectional headings to group chapters is helpful, although the chapters themselves are a little uneven in their presentation. For example, the chapter on neural grafting by Roitberg *et al* consists of 27 pages of solid text with no figures whereas the chapter by Hantraye *et al* on primate models of Huntington's disease combines figures and text. The result is that the second is more accessible whereas the more wordy chapters are off putting even to those of us who are interested in the field. This also applies to the opening chapter on the cholinergic hypothesis of Alzheimer's disease, which can derail the reader before he has really got interested in the rest of the book.

This book presents an interesting collection of chapters, which provides a useful adjunct to those involved in research in this area of neuroscience, but it is not a book that will appeal to neurologists unless they are keen to update themselves on the emerging new therapies being developed in laboratories around the world. It is therefore a useful addition to the libraries of a few, rather than many which is a pity given its content and relevance to neurology in the next century.

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