Matters arising

Transient uniocular visual loss in association with intraorbital tumours

Sir: We read with interest the article of Dr Bradbury *et al* on transient uniocular visual loss on deviation of the eye in association with intraorbital tumours.¹ We recently observed a similar case which also suggested that temporary occlusion of the ophthalmic artery might be the underlying defect, however without the presence of an intraorbital mass.

A 59 year old man, suffering from nasopharyngeal carcinoma, was treated in March 1985, with a total dose of radiotherapy of 7000 cGy to the nasopharynx and anterior base of the skull with shielding of the anterior part of both orbits. In August 1986 he developed gradual loss of vision of the left eye with papilloedema and haemorrhagic exudates and aching of the affected eye. The CT scan showed an enlarged optic nerve and fluorescence angiography indicated anterior ischaemia of the optic nerve. Despite treatment with prednisone he completely lost his vision of that eye, with optic atrophy and his pain gradually subsided. A diagnosis of anterior ischaemic optic neuropathy as a late effect of radiation was made.

In July 1987 he developed an aching right eye with partial loss of vision (acuity 3/6). On two occasions he had noticed that on left lateral gaze he lost after a few seconds all sight from his right eye during about 10 seconds, making him blind. Thereafter, the vision in his right eye gradually recurred in 5-10 seconds. Funduscopy showed a similar picture as his left eye, nine months earlier, with a swollen right optic disc and haemorrhagic exudates. There was no proptosis and the CT scan showed a thickened right optic nerve. Fluorescence angiography indicated anterior ischaemic optic neuropathy, the electroretinogram was normal and VEPs from the right eye were delayed. With prednisone treatment his vision deteriorated no further and he has had no more episodes of visual loss. Radiation injury again was thought to be the cause of the ischaemic optic neuropathy.

We think we observed an identical phenomenon of temporary (uniocular) visual loss on lateral gaze, as Dr Bradbury *et al*. The findings on fluorescence angiography, VEP and ERG are similar and give further support for their explanation of a (transient) ischaemia of the ophthalmic artery which might be further compromised by move-

ment or stretching of the vessel during lateral gaze.

Apparently, not only the presence of an intraorbital mass, but other causes of an ischaemic optic neuropathy may induce temporary visual loss on lateral gaze.

CH.J VECHE

WMH EIJKENBOOM

*Dr Daniel Den Hoed Cancer Center,
Rotterdam Radiotherapeutic Institute*

GS BAARMSA

BCP POLAK

*Eye Hospital, Rotterdam,
The Netherlands*

Reference

- 1 Bradbury PG, Levy IS, McDonald WI. Transient uniocular visual loss on deviation of the eye in association with intraorbital tumours. *J Neurol Neurosurg Psychiatry* 1987;50:615-9.

Arachnoid cysts of the middle fossa and subdural haematoma

Sir: The article from Page, Paxton and Mohan which appeared in the August issue of your journal discusses a fascinating problem and that is the association between subdural haematomas and intracranial CSF containing fluid collections.¹ I prefer to call these "pouches" rather than cysts since I believe most of them have a single opening which serves as an entrance and an exit and I also believe that this opening commonly has a valve-like mechanism.² I think the explanations offered by the authors from Plymouth are probably not correct. The explanation given that "flow changes within the CSF could be magnified by the arachnoid cyst leading to rupture of bridging veins or vessels in the cyst wall" seems to be highly specious if not completely meaningless. What sort of flow changes do the authors mean? If they mean quantity of flow it is difficult to see how an arachnoid cyst can do anything but diminish the quantity of fluid transmitted in response to pressure anywhere within itself or within its vicinity. If they mean pressure changes then it is again difficult to imagine how such a fluid collection can produce "magnification". It is certainly true that pressures may build up within the skull in response to elasticity of the skull itself and, particularly when considering shock waves travelling at sonic

speeds, damage produced by concussion may be focussed by the shape of the skull and its surroundings. This is possibly partly responsible for the mechanism of contrecoup injuries. To imagine that the cyst is somehow focussing pressure changes seems most improbable; in any case why should pressure changes produce disruption of the veins? The disruption of the veins is produced by stretching and then mechanical disruption of the walls or alternatively by gross distention of the vein so that it bursts open as an aneurysm bursts from pressure inside. I should think that stretching and avulsion of the veins from either the inside of the skull or from the surface of the brain is much the more probable in traumatic subdural haematoma. The second explanation that the authors offer is that the arachnoid cyst is less compliant than normal brain is almost certainly true, but what is "intra calvarial cushioning"? If this is what some authors call intracranial "compliance" and I prefer to call "capacitance" then what has that to do with subdural venous haemorrhage?

The probable association is that the arachnoid pouches commonly put some veins in their vicinity on the stretch and this will limit their ability to lengthen further when subjected to accelerative or rotatory stresses. They may therefore rupture more easily than veins crossing the subdural space which are not so distorted. I am not entirely happy with this explanation and in particular it does not account for the cases of contralateral haematomas. My experience of these lesions has been almost exclusively that the haematoma is in the immediate vicinity of the pouch and I would be interested to see more evidence on the claim from Plymouth that they occur frequently contralateral to these pouches.

BERNARD WILLIAMS

*Consultant Neurosurgeon,
Midland Centre for Neurosurgery
and Neurology,
Smethwick, Warley,
West Midlands B67 7JX, 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 Williams B. Surgery of arachnoid pouches. *Kinderchir* 1986;Supplement 1:1981.