posterior segment neovascularization, and subsequent vitreous haemorrhage may occur.

This condition is frequently divided into two phases that often overlap. Early symptoms are systemic and nonspecific. These include fever, poor appetite, fatigue, weight loss, joint pains, and night sweats. During this time many patients have a raised ESR, this was found in 72% of one group of patients with active disease, and in 56% of those with inactive disease.² There are no specific markers for the disease.³ The gold standard test is vessel biopsy, but this is only possible if surgical intervention is needed. Therefore, disease activity is difficult to establish. Some patients that are classified as in remission may not be; one publication reported that histology specimens showed that 44% of those presumed to be in remission were not.³ Occlusive symptoms can occur later, secondary to narrowing of the arteries.

The condition can be confirmed by radiographic means such as arteriography, computerized tomography, and magnetic resonance imaging.

A number of options exist to control this disorder. Initially, prednisolone can be instigated; if this alone is unsatisfactory then additional drugs such as methotrexate, azathioprine, and cyclophosphamide can be used. With steroid treatment alone, one study observed that remission was achieved in 60% of patients but half of these subsequently relapsed. With additional cytotoxic drugs remission was accomplished in 40%. However, 23% of treated patients never attained remission.¹ Various mortality rates have been stated; a group of 120 patients studied had a rate of 82.9% over 15 years.⁴ Optic complications can improve with systemic therapy, carotid endarterectomy and pan retinal photocoagulation may be considered. Spontaneous regression of new vessels as in this case has not been previously reported.

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Sir,

Marcus Gunn syndrome

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The Marcus Gunn syndrome, described by Gunn in 1883, is one of the more common congenital oculofacial synkineses, and accounts for approximately 5% of all congenital ptosis.¹

Typically, the infant is noted soon after birth to have a ptotic lid, which elevates spontaneously when sucking or chewing. The lid elevates on contraction of the lateral (external) pterygoid muscle, which mediates opening, forward, and contralateral movement of the jaw. Less commonly, the lid elevates upon closure of the mouth, which is mediated by the medial (internal) pterygoid. A very rare variant, 'inverse' Marcus Gunn syndrome is characterized by the lid falling on opening the mouth, because of inhibition of the levator muscle in association with lateral pterygoid contraction. Although bilateral cases of classical Marcus Gunn are well documented, there are, to our knowledge, only two previous reports of a patient with the classical syndrome on one side and an inverse Marcus Gunn synkinesis on the other.^{1,2} This report describes the case of a 4 year-old boy with what could be described as 'see-saw' Marcus Gunn syndrome.

Case report

A 4-year-old boy, presented with a right congenital ptosis. His mother was concerned about the cosmetic appearance of the lid as well as anomalous movements of the lids when eating. The child was otherwise well, had no history of other ocular problems, and there was no family history of similarly affected individuals.

Examination showed his visual acuities to be 6/9 right 6/6 left. He had a 3–4 mm right ptosis, with typical Marcus Gunn syndrome on the right side, the lid elevating to the normal position with jaw opening. On the left side, the lid height was normal at rest, but became ptotic when the mouth was opened (Figure 1). There was no contraction of the orbicularis oculi. This resulted in

Figure 1 (left) Right congenital ptosis and left lid in normal position with the mouth closed. (right) Reversal of lid positions with the mouth open. 'See-saw' Marcus Gunn syndrome.

Figure 2 Postoperative appearance at 2 week.

the lids having a 'see-saw' motion with alternate opening and closing of the mouth. The voluntary levator function was right 3 mm left 10 mm. Ocular movements were full and there were no other abnormal findings on ocular nor general examination.

The child underwent bilateral extirpation of the levator muscle and frontalis slings with autogenous fascia lata as described by Crawford,³ with a satisfactory functional and cosmetic outcome (Figure 2).

Comment

The Marcus Gunn syndrome is an interesting aetiological phenomenon. Electrophysiologic studies have documented cocontraction of the involved pterygoid and levator muscles in 'typical' cases,⁴ while one report documented inhibition of the levator muscle during ipsilateral lateral pterygoid contraction in a case of 'inverse' Marcus Gunn syndrome.⁵ Sano⁶ suggested, on

the basis of electrophysiologic studies, that Marcus Gunn syndrome and other oculo-facial synkineses represent release phenomena, as proposed by Pavesi *et al*.⁷ These phenomena are because of the reappearance of phylogenetically primitive synkinetic movements following injury to the involved cranial nerves *in utero*, with secondary degeneration of the cranial nerve nuclei. Cases may occur in isolation or in association with other anomalous ocular movements, such as double elevator palsy or Duane's syndrome,⁸ or with systemic anomalies such as cleft lip or palate. Bilateral cases have been reported in association with CHARGE syndrome.⁹ Such a heterogeneous group of patients suggests that the aetiological mechanism is complex and that more than one factor is likely to be involved in the development of such synkineses.

This case adds further to the complexity, with the patient demonstrating the combination of both 'typical' and 'inverse' Marcus Gunn syndromes, that is, a positive synkinesis on one side and an apparent inhibitory synkinesis on the other, although the electrophysiologic basis of the anomalous movements was not investigated.

Inverse Marcus Gunn syndrome also needs to be distinguished from Marin Amat syndrome, where lid closure because of orbicularis oculi contraction is associated with mouth opening.¹ Marin Amat syndrome generally follows seventh nerve palsy and is thought to be because of aberrant regeneration of the facial nerve. While some authors have used the terms 'inverse' Marcus Gunn syndrome and Marin Amat syndrome interchangeably, we believe the term inverse Marcus Gunn syndrome should be reserved for a congenital lesion, where lid closure is because of inhibition of the levator palpebrae superioris rather than orbicularis oculi contraction.

The treatment options in this case were more straightforward than in unilateral cases, as both lids

required surgery to abolish the synkinesis and correct the subsequent ptosis. In this case, bilateral levator extirpation combined with frontalis suspension using autogenous fascia lata was successful in achieving the desired result.

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Sir,

Pseudo-Terson's syndrome: bilateral simultaneous rhegmatogenous retinal detachment
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Vitreous haemorrhage secondary to subarachnoid haemorrhage was first described by Terson¹ in 1900. We report an unusual case of pseudo-Terson's syndrome

presenting to Stoke Mandeville Hospital's Eye Casualty with bilateral spontaneous vitreous haemorrhages and occipital headache.

Case report

A 57-year-old man presented to the eye casualty with an acute onset of bilateral blurred vision, photopsia, associated with sudden onset of occipital headache, tinnitus, and fever. Nausea was the predominant symptom with no vomiting, neck stiffness, or neurological complaints. He was hypertensive, on atenolol and amitriptyline.

He was fully alert and oriented. Examination revealed a visual acuity of 6/9 in the right eye and 6/60 in the left eye. Retinal examination was obscured because of vitreous haemorrhage in both eyes. A provisional diagnosis of bilateral vitreous haemorrhage secondary to subarachnoid haemorrhage (Terson's syndrome) was made. A full neurological assessment excluded signs of meningeal irritation or neurological deficit. CT scan of the brain showed high signal in the superior sagittal sinus; however, it was reported as normal. Absence of an increased intracranial pressure and red blood cells or xanthochromia on lumbar puncture excluded subarachnoid haemorrhage. B-scan of orbits revealed dense vitreous haemorrhage in both eyes. Systematic survey of each of the eye's four quadrants did not show evidence of small retinal tears or rhegmatogenous retinal detachment in the peripheral fundi. Full blood count, biochemical profile, clotting profile, and electrocardiogram were all normal. Blood pressure was 141/95. MRI of the brain was reported normal.

Vitreous haemorrhage cleared rapidly by day 3 in both eyes. Ocular examination showed a horseshoe retinal tear with minimal subretinal fluid in the right superotemporal quadrant and a suspicious area in the left superotemporal quadrant. Right retinal detachment repair was performed together with cryotherapy to the left eye.

After 1 week later, examination revealed a second horseshoe retinal tear with local subretinal fluid in the right inferotemporal retinal quadrant and three horseshoe retinal tears and bullous retinal detachment in the left superotemporal quadrant. Neither macula was affected by retinal detachment. Visual acuity recovered to 6/12 in the right eye and 6/6 in the left eye. Vitrectomy and fluid–gas exchange repair of the left retinal detachment and a second explant for the right eye were carried out.

Comment

Rhegmatogenous retinal detachment has a strong bilateral tendency. It results from chronic degenerative process, wherein both eyes are involved. The incidence of bilateral retinal detachment is 10% in normal population