CASE REPORTS

Bilateral failure of adduction following orbital decompression

F Kinsella, P Kyle, A Stansfield

Abstract

We report a case of bilateral complete failure of adduction following bilateral translid antral-ethmoidal orbital decompression. We believe the probable mechanism is neuropraxia (temporary dysfunction) of the third cranial nerves' supply to the medial recti, owing to these nerves' occupying an anatomically abnormal position. Partial recovery of adduction occurred over the ensuing six months.

In 1975 a 34-year-old woman in whom thyrotoxicosis and pretibial myxoedema had very recently been diagnosed was referred by the endocrinological service to our department for ophthalmological assessment. Examination at that time showed her best vision was 6/9 in both eyes (with a -2.0 sphere and $+5.50 \times 80^\circ$ in both eyes). Bilateral exophthalmos was present, the Hertel exophthalmometer measuring 23 mm on the right and 21 mm on the left side. Moderate lid lag and retraction were noted. A mild degree of lagophthalmos was present bilaterally, but corneal examination showed a normal appearance. Pupillary and fundal assessment gave normal results, as did measurement of intra-ocular tensions. The patient showed absence of convergence, but otherwise ocular movements were full.

Over the succeeding 11 years our patient's ocular status remained remarkably stable, her only symptoms being occasional grittiness. In 1987, however, her disease became much more active, with the appearance of marked congestion over the insertions of the horizontal recti and oedema of the caruncles. Corrected visual acuity fell to 6/18 bilaterally, and slight swelling of both optic discs became apparent. The degree of exophthalmos, however, remained static, and at

Figure 1: Both eyes divergent in the primary position on the first postoperative day.

no stage was there evidence of exposure keratitis. A CT scan of the orbits showed signs consistent with compressive neuropathy, and, because of her failing to respond to high-dosage systemic steroids, bilateral translid antral-ethmoidal decompressions were performed in August 1987.

The surgical procedure was uncomplicated, but it was noted at the time of surgery that the anterior ethmoidal arteries were located far more antero-inferiorly than normal.

On the day after surgery the patient's vision had improved to 6/12 in the right and to 6/9 in the left eye, but a marked divergent squint in the primary position was present (Fig 1). Examination of ocular movements revealed bilateral failure of adduction, the right eye being unable to reach the midline (Fig 2A) but reaching the midline on the left side (Fig 2B). The patient's other eye movements were normal. Forced duction testing demonstrated limitation of adduction in the right eye only on extreme adduction, whereas the left eye showed no limitation of adduction whatsoever. Force generation testing (whereby the patient performs ocular movements on command while the surgeon grasps the insertion of the appropriate

Figure 2A: Left lateral gaze immediately after operation, demonstrating inability of the right eye to reach the midline.

Figure 2B: Right lateral gaze immediately after operation, demonstrating partial adduction of the left eye.

Department of
Ophthalmology,
Southern General
Hospital, Govan Road,
Glasgow G41
F Kinsella
P Kyle
A Stansfield

Correspondence to:
Dr P Kyle, Southern General
Hospital, Govan Road,
Glasgow G41

Accepted for publication
28 September 1989

Figure 3A: Postoperative orbital CT scan showing evidence of antral-ethmoidal decompressions (arrows) as well as enlarged extraocular muscles consistent with Graves' ophthalmopathy.

Figure 3B: Postoperative orbital CT scan demonstrating that there has been no avulsion or disinsertion of the medial recti muscles (arrows).

rectus muscle) showed that active adduction from the abducted position was entirely absent in the left eye, and, though present in the right eye, its force was markedly diminished. A repeat orbital CT scan, apart from demonstrating signs of the bilateral floor and medial wall decompressions, showed that the recti muscles were essentially unchanged in appearance: in particular there was no evidence of avulsion or disinsertion of the medial recti (Figs 3A, 3B).

A trial of high-dosage systemic steroids failed to produce any improvement in ocular motility and was consequently discontinued after one week. Over the succeeding months, however, the patient's adduction began to improve spontaneously, so that six months after surgery some recovery of adduction had occurred bilaterally (Figs 4A, B). In addition the patient's convergence – which had not been present since first assessed in 1975 – had shown a certain amount of recovery (Fig 5).

At present (two years after surgery) our patient's ocular motility has shown considerable improvement. There has been significant recovery of adduction bilaterally, some convergence is present, and there is only 5° of left divergence in the primary position (Fig 6).

Discussion

Muscle imbalance following orbital decompression is well recognised,¹⁻⁵ but apart from the Naffziger⁶ anterior cranial fossa approach, which may be complicated by damage to the superior orbital fissure structures⁷ (resulting in third, fourth, or sixth cranial nerve pareses), the mechanism is almost invariably a mechanical

disturbance of the extraocular muscles. The risk of muscle imbalance depends on the particular procedure performed, being a frequent complication of three wall (36% in the series of Harting F, *et al*)⁸ and transantral antral-ethmoidal (33-41%),^{2,3,5} but rarely following translid antral-ethmoidal decompression (5-6%).⁴ The much higher incidence of motility disturbance following transantral rather than translid antral-ethmoidal decompressions is thought by some to be related to a more posterior decompression being achieved with the former approach, causing loss of support for the muscle cone at the orbital apex,⁴ though lack of direct visualisation with the former approach may contribute somewhat to this higher incidence. The occurrence of an A-pattern of motility disturbance in eight out of a series of 14 patients following anterior ethmoidal orbital decompression has recently been reported,⁷ the postulated cause being selective disruption of orbital fascial septa by the relieving periosteal incisions.

We believe that our patient's bilateral failure of adduction was due to neuropraxia (temporary dysfunction) of the third cranial nerves' supply to the medial recti for the following reasons: force generation testing demonstrated an inability to adduct the eyes on command, forced duction testing failed to show any significant mechanical restriction, while inadvertent avulsion of the medial recti was excluded by CT scanning. Additional evidence of neuropraxia is that some recovery of adduction occurred over a six-month period. So far as we are aware this is the first documented case of bilateral isolated adduction failure following orbital decompression.

As the medial recti are normally innervated from the global aspect, trauma to these nerves

Figure 4A: Right lateral gaze six months postoperatively demonstrating some recovery of left adduction.

Figure 4B: Left lateral gaze six months postoperatively demonstrating some recovery of right adduction.

Figure 5: Some recovery of convergence six months postoperatively.

Figure 6: Recent photograph showing the eyes in the primary position. The corneal reflexes are asymmetrical, and the cover test reveals that 5° of left divergence is present.

Figure 7: Diagram of the medial rectus branch of the inferior division of the third cranial nerve entering the orbit through the superior orbital fissure. The usual route into the orbit through the annulus of Zinn, as well as the aberrant route outside the annulus of Zinn postulated in our patient, are illustrated.

during an uncomplicated translid antral-ethmoidal decompression can be explained only by postulating an aberrant anatomical route for these nerves. The medial rectus muscle is usually innervated from the global aspect because its supplying nerve enters the orbit through that part of the superior orbital fissure surrounding the annulus of Zinn. We postulate that in our patient the nerves to the medial recti entered the superior orbital fissures outside the annulus of Zinn (as does the trochlear nerve), thus gaining access to the orbital aspect of the medial recti muscles (Fig 7), where it was injured during surgery. This theory is supported by the symmetry of the lesions produced on both sides and the peroperative observation that the anterior ethmoidal arteries occupied an abnormal position in the orbits.

A surprising outcome was partial recovery postoperatively of the patient's convergence

(which had been absent for many years). This was presumably related to the increased mechanical advantage afforded the medial recti after retrodisplacement of the globes.

We thank Ms E McMeekin and Mr C Orr, medical illustration department, Southern General Hospital, Govan Road, Glasgow G41, for their great assistance in producing the illustrations for this article.

- 1 Trokel ST L, Cooper WC. Orbital decompression effect on motility and globe position. *Ophthalmology* 1979; **86**: 2064-70.
- 2 DeSanto LW. The total rehabilitation of Graves' ophthalmopathy. *Laryngoscope* 1980; **90**: 1652-78.
- 3 Shorr N, Neuhaus RW, Bayliss HI. Ocular motility problems after orbital decompression for dysthyroid ophthalmopathy. *Ophthalmology* 1982; **89**: 323-8.
- 4 McCord CD, Jr. Current trends in orbital decompression. *Ophthalmology* 1985; **92**: 21-33.
- 5 Harting F, Koornneff L, Peeters HJF, Gillissen JPA. Fourteen years of orbital decompression in Graves' disease. *Orbit* 1986; **5**: 123-9.
- 6 Naffziger HC. Progressive exophthalmos following thyroidectomy: its pathology and treatment. *Ann Surg* 1931; **94**: 582-6.
- 7 Fells P. Orbital decompression for severe dysthyroid eye disease. *Br J Ophthalmol* 1987; **71**: 107-11.