Diplopia after retinal detachment surgery

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SUMMARY Diplopia following retinal detachment usually responds to simple measures. Fifteen out of 311 cases developed diplopia lasting more than three months after conventional retinal detachment surgery. Binocular single vision was restored in 12 of the 15 cases (80%). The mean follow-up was four years. Diplopia was eliminated stepwise. If prisms were ineffective, our first surgical procedure was removal of the scleral buckle. If the retina was flat, we were prepared to remove the buckle early. When diplopia persisted after buckle removal, we proceeded to strabismus surgery. Our most consistent results followed strabismus surgery on the untreated eye. Prisms alone restored binocular single vision in six patients (40%), one of whom preferred to adopt a compensatory head posture. Removal of the scleral buckle restored binocular single vision in three patients (20%), with the help of a prism in one case and a compensatory head posture in another. Binocular single vision was restored after buckle removal and strabismus surgery in three further patients (20%), one requiring a prism in addition. Binocular single vision was not restored in three patients (20%).

Diplopia occurs in 3% to 30% of patients after conventional retinal detachment surgery. Possible causes include the breakdown of an existing fusion weakness, distortion of the globe by scleral buckles, damage to the rectus muscle insertions by traction sutures, and postoperative tissue swelling. Permanent adhesions and scarring often bind the rectus muscles down to the underlying sclera. Most cases of diplopia following retinal detachment surgery recover spontaneously. Recessus muscles swollen by cryotherapy return to normal after six weeks. Even when division of the rectus muscles was more widely practised, and diathermy was used instead of cryotherapy, heterotropia was usually transient. It has been postulated that phoria adaptation contributes to the resolution of the initial heterotropia after retinal detachment surgery. Phoria adaptation is observed when the normal fusional range is restored after any induced shift of oculomotor balance.

Many of those with transient diplopia learn to suppress the image from the operated eye, particularly if its acuity is reduced. It has been estimated that between one-quarter and one-third of all patients develop suppression in the treated eye after retinal detachment surgery. There are some who cannot suppress the second image; the ability to suppress lessens with age, and intractable diplopia may even occur after surgery on amblyopic eyes. Diplopia resolves spontaneously in most cases after retinal detachment surgery, but sometimes persists to become incapacitating. We have studied a group of 15 patients whose diplopia persisted longer than three months after retinal detachment surgery.

Material and methods

Four hundred and thirty-six consecutive cases requiring retinal detachment surgery at St Thomas’s Hospital from 1 January 1979 to 31 December 1982 were studied. Surgical techniques included cryotherapy and full-thickness scleral buckling with either Silastic sponges of standard sizes, or solid Silastic bands, gutters, and tyres. 3/0 silk sutures under the rectus muscles provided traction on the globe, but the muscle insertions were not divided. Closed pars plana vitrectomy was performed in 125 of the cases studied.

An orthoptic assessment was performed as soon as any patient complained of double vision after retinal detachment surgery. This orthoptic examination included the measurement of visual acuity and the range of ocular movements. Serial Hess charts were recorded, and the prism cover test was used to measure the angle of heterotropia (the modified
Krimsky test was substituted when the visual acuity was poor. Binocular functions were assessed with Bagolini's striated glasses, the Titmus stereotest, and the synoptophore. Further orthoptic and ophthalmic examinations were conducted at three and six monthly intervals.

First we tried to control diplopia with either adhesive Fresnel prisms or with a prismatic addition to the existing spectacles. We operated only if prisms failed to control the diplopia. Our initial surgical procedure was removal of the scleral buckle, and adjacent rectus muscle insertions were freed of scar tissue at the same time. We performed strabismus surgery as a separate procedure in two cases and combined it with buckle removal in one case.

Results

No patient complained of diplopia after vitrectomy (125 cases). Of 311 patients undergoing conventional retinal detachment surgery 15 (4.8%) developed postoperative diplopia lasting more than three months. Clinical details of these patients are summarised in Table 1, and the effect of scleral buckling in each case is shown in Table 2. The period of follow-up ranged from two to six years, mean four years.

Table 1  Clinical course of patients with diplopia after retinal detachment surgery

<table>
<thead>
<tr>
<th>Case no</th>
<th>Age</th>
<th>Number of retinal operations</th>
<th>Maximum heterotropia after retinal surgery (prism dioptres)</th>
<th>Time of buckle removal (months after retinal surgery)</th>
<th>Maximum heterotropia after buckle removal (prism dioptres)</th>
<th>Strabismus surgery</th>
<th>Present status</th>
<th>Corrected visual acuity of operated eye after retinal detachment surgery</th>
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<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>1 R/L 6</td>
<td></td>
<td>-</td>
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<td>No</td>
<td>BSV (prisms)</td>
<td>6/9</td>
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<tr>
<td>2</td>
<td>60</td>
<td>1 R/L 2</td>
<td></td>
<td>-</td>
<td>-</td>
<td>No</td>
<td>BSV (prisms)</td>
<td>6/9</td>
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<tr>
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<td>58</td>
<td>2 R/L 6</td>
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<td>-</td>
<td>-</td>
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<td>BSV (prisms)</td>
<td>6/60</td>
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<tr>
<td>4</td>
<td>57</td>
<td>1 R/L 8</td>
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<td>BSV (prisms)</td>
<td>6/12</td>
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<tr>
<td>5</td>
<td>61</td>
<td>1 R/L 9</td>
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<td>-</td>
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<td>No</td>
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<td>6/6</td>
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<tr>
<td>6</td>
<td>63</td>
<td>1 L/R 13</td>
<td></td>
<td>-</td>
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<td>BSV (with CHP)</td>
<td>6/36</td>
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<tr>
<td>7</td>
<td>52</td>
<td>1 L/R 14</td>
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<td>18</td>
<td>L/R 2</td>
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<td>6/9</td>
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<tr>
<td>8</td>
<td>68</td>
<td>1 R/L 16</td>
<td></td>
<td>15</td>
<td>R/L 4</td>
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<td>9</td>
<td>31</td>
<td>1 R/L 3</td>
<td></td>
<td>10</td>
<td>R/L 1</td>
<td>No</td>
<td>BSV (with CHP)</td>
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</tr>
<tr>
<td>10</td>
<td>54</td>
<td>2 L/R 9</td>
<td></td>
<td>-</td>
<td>-</td>
<td>No</td>
<td>Torsional diplopia suppression</td>
<td>6/36</td>
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<tr>
<td>11</td>
<td>21</td>
<td>1 L/R 2</td>
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<td>-</td>
<td>-</td>
<td>No</td>
<td>Weak fusion with intermittent diplopia suppression</td>
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<tr>
<td>12</td>
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<td>3 L/R &gt;20</td>
<td></td>
<td>5</td>
<td>L/R 14</td>
<td>No</td>
<td>BSV</td>
<td>6/60</td>
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<tr>
<td>13</td>
<td>34</td>
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<td>6/6</td>
</tr>
<tr>
<td>14</td>
<td>62</td>
<td>1 R/L 20</td>
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<td>14</td>
<td>R/L 3</td>
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<td>BSV (prisms)</td>
<td>6/9</td>
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<tr>
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<td>46</td>
<td>1 R/L 20</td>
<td></td>
<td>3</td>
<td>R/L 20</td>
<td>Yes</td>
<td>BSV</td>
<td>6/18</td>
</tr>
</tbody>
</table>

R = right eye; L = left eye; Exo = exotropia; Eso = esotropia; BSV = binocular single vision; CHP = compensatory head posture.
Diplopia after retinal detachment surgery

Table 2  Details of episcleral buckling and its effects in patients with diplopia after retinal detachment surgery

<table>
<thead>
<tr>
<th>Type of buckle</th>
<th>Case No.</th>
<th>Diameter of sponge(s) (mm)</th>
<th>Muscle(s) in contact with scleral buckle</th>
<th>Maximally restricted ocular movement</th>
<th>Number of patients with diplopia</th>
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</thead>
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<tr>
<td>Silastic sponge explant: radial</td>
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<td>Adduction</td>
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<td>5/7-5</td>
<td>LR</td>
<td>Adduction</td>
<td>2</td>
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<td>8</td>
<td>4</td>
<td>SR/LR</td>
<td>Depression</td>
<td>2</td>
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<tr>
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<td>5</td>
<td>SR/LR</td>
<td>Depression</td>
<td>2</td>
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<tr>
<td></td>
<td>14</td>
<td>5</td>
<td>SR</td>
<td>Depression</td>
<td>2</td>
</tr>
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<td>SR</td>
<td>Depression</td>
<td>2</td>
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<td></td>
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<td>IR</td>
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<td>7-5</td>
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<td>Depression</td>
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<td>Abduction</td>
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<td>Abduction</td>
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<td>5</td>
<td>5/5</td>
<td>–</td>
<td>Abduction</td>
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<td>5/7-5</td>
<td>–</td>
<td>Abduction</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>7-5/7-5</td>
<td>–</td>
<td>Depression</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>7-5/7-5</td>
<td>–</td>
<td>Depression</td>
<td>1</td>
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<tr>
<td>Total cases</td>
<td>9</td>
<td>–</td>
<td>–</td>
<td>Depression</td>
<td>15</td>
</tr>
</tbody>
</table>

LR = lateral rectus muscle. SR = superior rectus muscle. IR = inferior rectus muscle.

Prisms were of no benefit in four cases (27%). Case 10 had torsional diplopia which was relieved by occlusion. Case 11 had very weak fusion with intermittent suppression, case 12 suppressed completely, and case 7, with a large vertical tropia, was able to fuse after removal of the scleral buckle.

**Response to corrective surgery**

**Removal of the scleral buckle.** The scleral buckle was removed in seven cases (47%) between three and 18 months after retinal detachment surgery. Changes in the position of the eyes are recorded as changes in the prism cover test readings in Table 1. Diplopia was eliminated completely in only one patient (case 7), who had a large hypotropia. There was a reduction of the heterotropia in three patients: one became orthophoric with prisms (case 8), one controlled the diplopia with a compensatory head posture (case 9), and the other suppressed (case 12). No change was seen in case 15, but the diplopia was actually worse after buckle removal in the two remaining patients (case 13 and 14); their clinical course is described below.

**Strabismus surgery.** Three patients (cases 13, 14, and 15) required definitive surgical correction of their strabismus. Case 13 had a medial rectus recession and lateral rectus resection on the buckled eye, but even with prisms horizontal diplopia was not controlled. Medial rectus recession and lateral rectus resection in the fellow eye eventually resolved the esotropia without the need for prisms. In case 14 the scleral buckle was removed at the same time as resection of the superior rectus muscle. Vertical diplopia persisted because of overcorrection, and further surgery was necessary. The superior rectus muscle was recessed and residual diplopia was eliminated with prisms. Case 15 had an exotropia, with hypotropia in the buckled eye. After removal of the scleral buckle strabismus surgery was performed only on the fellow eye: the lateral rectus was recessed and the medial rectus was resected, with downward transposition of the muscle insertions. Although there was some reduction of the hypotropia, diplopia was not controlled, and further strabismus surgery was needed. Recession of the inferior oblique was combined with further surgery to the horizontal recti, an adjustable suturing technique being used. Diplopia was eliminated in all directions of gaze except extreme laevodepression.

**Discussion**

Several factors influencing the occurrence of diplopia after retinal detachment surgery have been proposed. They include operative trauma, repeated retinal surgery, and the size, location, and type of scleral buckle used.

The importance of minimising operative trauma by careful surgical technique has been stressed repeatedly. A rectus muscle was divided at the time of retinal surgery in only one of our patients (case 12). The inferior rectus muscle had been detached from its insertion twice before, and its tendon was never identified on exploration of the residual scar tissue. In all other cases the rectus muscle insertions were located, and their integrity was guaranteed by passing traction sutures beneath them. It has been suggested that repeated retinal
detachment surgery is a significant cause of post-operative diplopia, but we did not confirm this finding: 11 of our patients (73%) developed diplopia after their first retinal operation (Table 1).

Prospective studies have indicated that the size of a buckling element, and its location under a rectus muscle, may influence the onset of postoperative diplopia. Wide-diameter buckles (7.5 mm or wider combinations) were used in eight (53%) of our cases (Table 2). We found that circumferential sponges were associated with diplopia in six cases (40%) and radial or oblique sponges in three cases (20%), which is in contrast to the conclusion of a recent study. Only one of our 15 cases was treated with an encircling band alone (case 4), a procedure which is less often associated with diplopia, though cases have been reported with varying frequency. This factor probably accounts for the absence of diplopia in the 125 vitrectomy cases in our series. Nine of our 15 patients with diplopia (60%) had a scleral buckle directly related to one or more rectus muscles (Table 2). Restricted ocular rotation in the direction away from the rectus muscle with the underlying scleral buckle occurred in six of these cases. In the other three patients (cases 7, 11, 14) elevation was limited, and the scleral buckle lay beneath the superior rectus muscle, causing a mechanical restriction. A vertically acting muscle (superior or inferior rectus) was involved in eight of these nine cases. This finding is strikingly similar to a report of cases in which the rectus muscle adjacent to the scleral buckles were all detached at the time of retinal surgery.

Eleven patients (73%) have confirmed the benefit of prisms in the course of their treatment. Five patients (33%) had their symptoms controlled by the use of prisms alone. It is worth emphasising that seven patients (47%) still require prisms to maintain binocular single vision (Table 1). Torsional diplopia cannot be controlled by prisms, although surgical correction is possible. However, in case 10, we resorted to occlusion and did not attempt strabismus surgery.

It has been stated that no surgical correction of postoperative diplopia should be attempted until six months have elapsed after retinal detachment surgery. However, our experience of buckle removal suggests that its timing is not critical. We removed the scleral buckle in seven cases (47%), when the use of prisms was impracticable because the angle of deviation was too great. Removal of a circumferential sponge three months after its insertion had no effect at all on one patient (case 15); in another (case 7) the removal of a wide radial sponge under the superior rectus muscle after 18 months allowed elevation of the eye, eliminating the large vertical muscle imbalance. We see no practical advantage in waiting to remove the scleral buckle once the retina is safely reattached, since early removal should minimise episceral fibrosis. Although we cannot predict the effect of buckle removal in a particular case, we consider that this is the logical first step in corrective surgery. We were able to control diplopia by removing the scleral buckle in four of the seven cases whose diplopia could not be joined with prisms.

Diplopia persisted after buckle removal in three cases. Definitive strabismus surgery was then performed. After forced duction testing we operated first on the eye which had already been treated. We could thus divide muscle adhesions and correct the underaction of affected rectus muscles. In our small series we did not find it necessary to overcorrect the strabismus, employ marginal myotomy, or recess the conjunctiva to enhance the effect of rectus muscle recession, techniques which have all been advocated in the management of diplopia after retinal detachment surgery. In both patients with horizontal diplopia who required strabismus surgery (cases 13 and 15) we eliminated diplopia by operating on the eye which had not been buckled. This confirms that strabismus surgery for this condition may be performed on either eye. In one patient (case 15) we used an adjustable suturing technique with excellent results to achieve an early and accurate restoration of fusion.

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References

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