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CLINICAL STUDY

Diplopia following cataract surgery: a review of 150 patients

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Abstract

Aim **To study the motility pattern, underlying mechanism, and management of patients who complained of double vision after cataract surgery.**

Methods **A retrospective case note analysis of 150 patients presenting with diplopia after cataract surgery to an orthoptic clinic over a 70-month period. Information was retrieved from orthoptic, ophthalmological, and operating room records.**

Results **A total of 3% of patients presenting to the orthoptic clinic had diplopia after cataract surgery. We grouped these according to the underlying mechanisms which were: (1) decompensating pre-existing strabismus (34%), (2) extraocular muscle restriction/paresis (25%), (3) refractive (8.5%), (4) concurrent onset of systemic disease (5%), (5) central fusion disruption (5%), and (6) monocular diplopia (2.5%). Twenty per cent of the patients could not be categorised with certainty. After infiltrational anaesthesia, extraocular muscle restriction/paresis was the commonest presentation, while decompensation of preexisting strabismus was commonest with topical anaesthesia.**

For the 150 patients seen, prisms were the commonest form of treatment prescribed (64%) either in isolation or in combination with other treatment, including surgery (19%). Convergence and divergence insufficiency/paresis patterns were also common.

A changing motility pattern was noted in some patients who had early documentation, with increasing comitance over time (spread of comitance). Partial resolution made it difficult to clearly identify the underlying mechanism in patients with late documentation.

Conclusion **Double vision is a troublesome complication of otherwise successful cataract surgery. The use of topical anaesthesia does not abolish this surgical risk.**

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Keywords: cataract; diplopia; strabismus; anaesthesia

Introduction

The recent rapid evolution of cataract surgical technique has made this one of the most commonly performed and successful surgical procedures. However, the substantial benefit of visual acuity improvement resulting from cataract extraction can be reduced by the introduction of post-operative diplopia. Most of the recent literature regarding the cause of this complication[1–20] has focused on anaesthetic myotoxicity, trauma during infiltrational anaesthesia, or the use of a rectus bridle suture.

In this study, we reviewed the motility characteristics, likely aetiology, and management of patients with double vision after cataract surgery, estimating the incidence of this problem in our area, with the aim of identifying potential factors, that could increase the risk of diplopia in patients undergoing cataract surgery. Finally, we wished to pay particular attention to causes of double vision in patients for whom infiltrational anaesthesia and/or superior rectus bridle sutures were not used at the time of cataract surgery.

Patients and methods

We reviewed the records of all the patients seen in the orthoptic clinic at British Columbia Children's Hospital from January 1995 to October 2000, with persistent double vision who had undergone cataract surgery. Patients were excluded if they had diplopia before surgery or their double vision had spontaneously resolved within 1 month of the surgery. The presence of

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strabismus itself was not an indication for exclusion, allowing the effect of cataract surgery on pre-existing asymptomatic strabismus patients to be discussed.

Data obtained from the orthoptic chart included the patient's age, sex, past ocular history, medical history, and interval from cataract surgery to initial orthoptic assessment. Details of each patient's complete orthoptic assessments were also extracted. This included a detailed motor and sensory evaluation. Motor evaluation consisted of prism cover test measurements in the diagnostic positions of gaze with documentation of ductions and versions. Serial Hess charts were also obtained in a large number of patients. A sensory evaluation was obtained on all patients using the synoptophore. The referring cataract surgeon's case notes were also reviewed, with particular reference to the past medical and ophthalmic history, the anaesthetic and surgical techniques used at the time of cataract surgery, as well as the initial nature of the post-operative double vision.

Results

In this 70-month period, a total of 5032 patients were seen in the orthoptic clinic of this institution. One hundred and fifty of these (3%) presented with double vision after cataract surgery. There were 89 women and 61 men, with a mean age of 75.4 years (range, 17–92 years). The patients were referred by a total of 39 different surgeons, operating at eight hospitals. One hundred and six patients had undergone bilateral (consecutive) surgery, and 44 had unilateral cataract surgery. The median interval from surgery to initial orthoptic assessment was 6 months (range, 0.15–384 months). The patients were grouped according to the mechanism underlying their diplopia (Table 1).

Table 1 Classification of aetiologies underlying presentation

Category	*No. of aetiologies*[a] *(patients = 150)*	*Percentage*[b]
Decompensating pre-existing strabismus	54	34
Extraocular muscle restriction/ paresis	41	25
Undetermined aetiology	32	20
Refractive	14	8.5
Concurrent onset of systemic disease	8	5
Central fusion disruption	8	5
Monocular	4	2.5
Total	161	100

[a]Since multiple aetiologies were occasionally implicated, nine patients were included in two categories and one in three categories. Hence the total number is out of 161.
[b]Percentage calculated from total number of underlying mechanisms accounting for diplopia. This includes patients having more than one underlying mechanism for their diplopia ($n = 161$).

Categories

Decompensation of pre-existing asymptomatic strabismus

This group of 54 patients comprised over one-third (34%) of all cases. Of these, 19 patients (12%) had convergence insufficiency (CI) and 10 patients (6%) had a decompensated superior oblique palsy. The remainder had previously documented strabismus or a history of childhood strabismus, which was asymptomatic preoperatively.

The 19 patients with CI were not documented to be symptomatic preoperatively.

The 10 patients with decompensated superior oblique palsy all had large vertical fusional amplitudes. Four of these patients had noticed intermittent vertical double vision before their cataract surgery but did not consider this problematic and had no difficulties in coping with this symptom. In five of the remaining six patients, the superior oblique palsy was contralateral to the cataract surgery.

Extraocular muscle restriction or paresis

Forty-one patients were in this category (25%). The details of the operated eye and the motility pattern seen in these patients are illustrated in Figure 1. Forty patients (98%) had a vertical strabismus at their initial orthoptic examination. Eighteen patients (44%) had a hypertropia of the operated eye, 22 (54%) had a hypotropia of the operated eye, and 1 patient (2%) had an abduction deficit of the operated eye. Eighteen patients (44%) had vertical strabismus, which increased in the direction of the deviation, that is, a hypertropia that increased in upgaze or a hypotropia that increased in downgaze. Four patients were examined within 2 weeks of their cataract surgery. Two of these initially had a hypertropia of the operated eye in the primary position, which changed into a hypotropia over a period of 2 months.

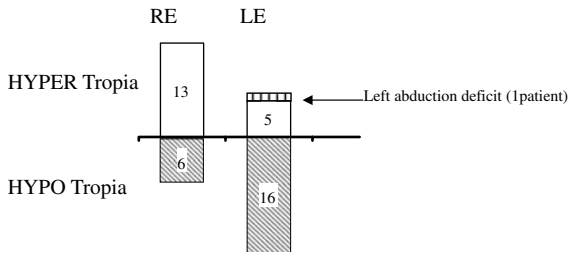

Figure 1 Extraocular muscle restriction/paresis group ($n = 41$). Strabismus at time of initial orthoptic evaluation following cataract operation. RE = cataract surgery on right eye, LE = cataract surgery on left eye.

Refractive

Two refractive causes of postoperative double vision were identified in this study: lens prismatic effect (seven patients) and aniseikonia induced because of spectacle correction of new anisometropia (seven patients) comprising a total of 8.5% of all aetiologies. A change to contact lens wear (two patients), the use of a piggyback intra-ocular lens (IOL) (two patients), a change in prescription (three patients), or followup with no intervention (four patients) eliminated the problem in 11 patients. Of the remaining three patients, two were lost to followup and one patient developed cystoid macular oedema in the operated eye.

Concurrent onset of systemic disease (including sixth nerve palsies)

Eight patients (5%) were included in this group. One had a new onset of thyroid orbitopathy and seven had a sixth nerve palsy. In the sixth nerve palsy patients, motor testing, including Hess chart, excluded a restrictive pattern. Two of the seven patients had undergone cataract surgery under topical anaesthesia. The remaining five had peribulbar anaesthesia, four of whom had cataract surgery in the ipsilateral eye and one had in the contralateral eye. The palsy resolved spontaneously in two cases. Three others were managed successfully with prisms and two underwent strabismus surgery.

Central fusion disruption (acquired loss of fusion)

This was thought to be the cause in eight patients (5%). The patients in this category had unilateral cataracts (one PHPV, seven traumatic) at a young age. All had undergone unilateral cataract surgery at least 17 years before presentation to us and had abandoned their refractive correction for many years (range, 8–32 years). They complained of diplopia after re-introduction of refractive correction in the form of a contact lens (seven patients) or a secondary IOL implant (one patient).

Monocular

Four patients (2.5%) presented with symptoms of monocular double vision. One had a subluxed IOL and became asymptomatic following re-positioning. One patient developed macular degeneration and in two patients the cause was undetermined.

Undetermined aetiology

There was no obvious aetiological pattern in one-fifth of the cases (32 patients). However, two subgroups were noted: in group 1, 22 patients were found to have a small comitant vertical deviation measuring less than 12 prism dioptres. In group 2, 10 patients had a small comitant esotropia at distance with no near manifest deviation. These patients had no clinically detectable lateral rectus weakness. Saccadic velocities were not measured. There were no other neurological signs or symptoms. Both these groups responded well to treatment with appropriate prisms.

Analysis based on anaesthetic technique used for cataract surgery

The 86 patients for whom anaesthetic data were available were categorized according to the mechanism underlying their diplopia. Sixty-four patients received infiltrational anaesthesia (36 peribulbar, 16 retrobulbar, 12 not stated). Twenty-two of these had a superior rectus stay suture. Twenty-two patients developed diplopia after cataract surgery with topical anaesthesia only. Three of these were classified as having extraocular muscle restriction/paresis. Two of these three had superior rectus stay sutures; this information was not available for the third patient. The breakdown of these patients based on the underlying mechanism is shown in Figure 2. Table 2 categorizes the underlying mechanism in patients having stay sutures. Figure 3 shows the change in anaesthetic technique over the years studied (where data were available).

The use of hyaluronidase in the patients undergoing infiltrational anaesthesia was not documented in the surgical record.

Management

The treatment modalities used are shown in Table 3.

Discussion

To our knowledge, this is the largest reported series of patients who developed diplopia subsequent to undergoing cataract surgery. Diplopia was previously

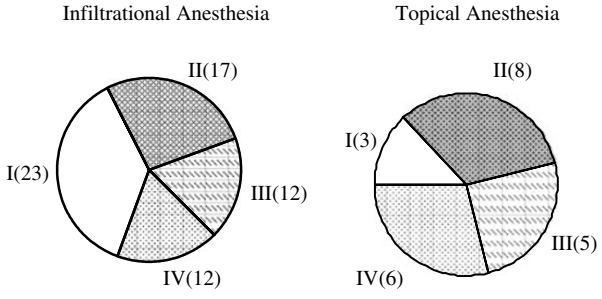

Figure 2 Mechanisms underlying diplopia in cases of infiltrational anaesthesia ($n=64$) versus topical anaesthesia ($n=22$). Group I – Extraocular muscle restriction/paresis. Group II – Decompensating preexisting strabismus. Group III – Undetermined. Group IV – Other.

Table 2 Classification of the mechanism underlying diplopia in patients having superior rectus stay suture[a]

Mechanism	*Anaesthetic technique*	
	Infiltrational[b] *(22 patients)*	*Topical*[c] *(2 patients)*
Extraocular muscle restriction/ paresis	9	2
Decompensating preexisting strabismus	6	0
Undetermined category	2	0
Other	7	0

[a]Includes only patients where anaesthetic data were available — infiltrational (64 patients) *vs* topical (22 patients).
[b]Suture not used in 31 patients, unknown for 11 patients. Two patients included in multiple categories making total number out of 24.
[c]Suture not used in 17 patients, unknown for 3 patients.

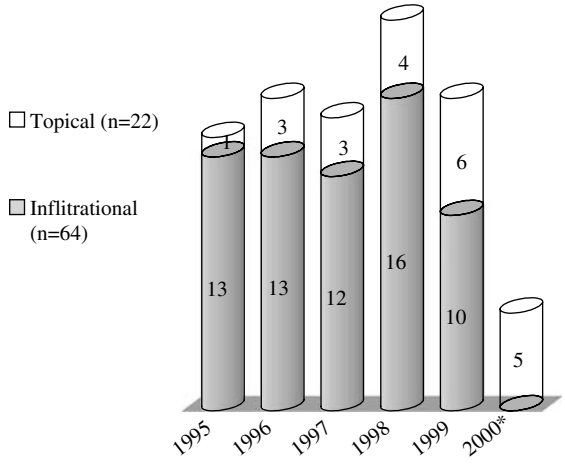

Figure 3 Cases of double vision classified by year of cataract operation. *Only includes patients up to October.

believed to account for 10% of the cases undergoing surgery in an adult strabismus practice.[3] We found that this problem accounted for 3% of the patients seen in the orthoptic clinic at this institution.

Incidence

In the 70-month period of patient recruitment, an estimated 87 500 cataracts (Hospital surgical data from Lower Mainland, British Columbia) were performed by the 39 ophthalmologists who referred patients for this study, suggesting an approximate incidence for this problem in the order of 0.17%. This is likely an underestimate of the true frequency of this complication since it is unlikely that all patients with post-operative diplopia would have been seen in this clinic. Some, with transient post-operative symptoms,[4] may not have been referred before their spontaneous improvement. Other diplopic patients may have been evaluated and treated elsewhere.

Table 3 Management of cases of double vision following cataract extraction

Management modality	*No. of patients*[a] *(n = 150)*	*Percentage*[b]
Prisms—Fresnel/ground in	96	64
Strabismus surgery	29	19
Observation	25	17
Refractive	8	5
Orthoptic exercises	8	5
Botulinum toxin injection	1	<1

[a]Some patients had a combination of treatment modalities and are therefore included in more than one category.
[b]Per cent based on total number of patients ($n = 150$), not the total number of treatment modalities used ($n = 167$).

Presentation

The median interval between the cataract surgery and orthoptic assessment was 6 months. This is probably because the referring ophthalmologist waited to determine if the diplopia was transient. However, the motility pattern in paralytic strabismus evolves with time owing to the spread of comitance and sometimes partial resolution. This can make it difficult to identify accurately the primary affected muscle. Two patients presented with an inferior rectus palsy immediately after cataract surgery, which evolved into an inferior rectus restriction over a period of 2 months. Capo and Guyton,[6] Brown *et al*,[4] and Koide *et al*[14] reported similar findings, which emphasise the need for early motor and sensory evaluation to understand the mechanism underlying this problem.

In this series, 10 patients' symptoms resolved spontaneously. Temporary diplopia could be due to the presence of a small pre-existing sensory strabismus, the prismatic effect of a new spectacle correction, which the patient eventually overcomes with fusion. Other potential mechanisms include prolonged anaesthetic effect, transient myotoxicity, or low-grade operative trauma to the orbital soft tissues.[5]

Diplopia occurred more often in patients who had undergone cataract surgery in both eyes (71%) as compared to unilateral surgery (29%). CI was much more common in patients who had undergone bilateral cataract surgery (see below). Also, it could be argued that patients undergoing surgery in both eyes had twice the risk of developing mechanical complications such as myotoxicity compared to unilateral patients.

Decompensation of preexisting asymptomatic strabismus

CI tends to manifest well before the age group considered here. Some patients are known to develop CI

when they first wear bifocals, owing to the resultant decrease in accommodative convergence. In our series, 19 patients developed findings of CI after becoming pseudophakic (14 bilateral). None of these patients had documented problems preoperatively, despite all being in bifocal correction for many years. The preoperative refractive errors of the patients in this subgroup were identified. Three had mild to moderate myopia (patient 1: R −2.25, L −2.25; patient 2: R−2.75, L−3.25); one of these had significant anisometropia preoperatively (patient 3: R−7.00, L−1.50). The remaining patients were found to have refractive errors ranging from plano to +2.75 DS with no one having anisometropia greater than 0.75 D.

The myopic patients may have been helped preoperatively by the base-in prismatic effect of their glasses when converging, masking an underlying CI. Eliminating myopic glasses after IOL implantation may have allowed their symptoms to manifest.

Eight of the 19 patients with CI were given orthoptic exercises. This treatment was successful in five of these eight patients. The three patients in whom this failed were subsequently managed with prisms.

Ten patients (6%) presented with vertical double vision resulting from a superior oblique weakness. Patients with congenital or long-standing superior oblique palsy develop large fusional amplitudes and their control may be disrupted by reduced vision in the cataractous eye or by cataract surgery.[7,21] It is possible that myotoxicity of the ipsilateral inferior rectus could be masquerading as a contralateral superior oblique palsy. However, the presence of large fusional amplitudes and lack of significant restriction of elevation in the operated eye are not consistent with inferior rectus restriction.

Patients with a past history of strabismus or amblyopia and appropriate sensory adaptations such as monofixation may present with diplopia as a result of change in their fixation pattern.[20] Twenty-five patients had a history of preexisting strabismus and developed new symptoms of diplopia following cataract surgery. Table 4 shows the preoperative documentation of strabismus in this group. In none of these cases did diplopia occur as a result of a switch in fixation. Two patients with long-standing exotropia and no fusion did however develop symptoms secondary to 'image jump' as a result of a new alternating fixation pattern after the vision improved in their operated eye.

Extraocular muscle restriction or paresis

Double vision after cataract surgery is most commonly attributed to extraocular muscle restriction or paresis from surgical trauma or anaesthetic myotoxicity.[1–15] Rainin and Carlson[8] were the first to suggest that myotoxicity from the local anaesthetic could result in temporary or permanent extraocular muscle paresis. Carlson *et al*[9] later demonstrated degeneration and regeneration in extraocular muscles of primates and humans as a consequence of retrobulbar administration of several local anaesthetics. Although inferior rectus/superior rectus muscle injury has been most commonly implicated, inferior oblique muscle injury[13] and transient paresis of the lateral rectus[4] have been reported.

Forty-one patients in our series (25%) had a motility pattern suggestive of restriction or palsy. Distinguishing with certainty between the two mechanisms was not possible as forced duction tests were not performed routinely. Two of four patients seen within 2 weeks of the cataract surgery were found to have an initial hypertropia in the primary position, which reversed to a hypotropia over the next 2 months. This changing pattern of hypertropia to hypotropia or vice versa has been attributed to early rectus palsy followed later by contracture of the palsied muscle from myotoxicity.[6] Much more commonly, (18 patients, 44%), patients presented with a hypertropia of the operated eye increasing in upgaze or a hypotropia of the operated eye increasing in downgaze. This pattern has also been

Table 4 Documented preexisting strabismus

Diagnosis[a]	*No. of patients (n = 25)*	*Subgroups*
Esotropia	12	Paresis, 3
		Mechanical thought to be secondary to high myopia, 2
		Childhood onset, 2
		Decompensating esophoria, 2
		Acquired unknown aetiology, 3
Exotropia	4	Childhood onset, 4
Hypertropia	9	Decompensating hyperphoria, 5
		Acquired unknown aetiology, 4

Patients having convergence insufficiency (19 patients) or decompensating superior oblique palsy (10 patients) were not included. All patients had documented strabismus before their cataract operation without symptomatic double vision. Two patients reported a new symptom of 'image jump' secondary to a new alternating fixation pattern from improved visual acuity of the operated eye.
[a]Includes intermittent deviation.

observed previously.[3,6,12] Scott *et al*[22] have recently shown that direct local anaesthetic injection into the muscle can lead to initial myotoxicity, which is followed by hypertrophy of the muscle and this is consistent with our findings. Also discussing this, Capo and Guyton[6] postulated that the involved muscle can overact as a result of posterior segmental myotoxicity, which is not extensive enough to produce classical restrictive strabismus with positive forced duction testing. Approximately equal numbers of patients were found with hypertropia (45%) and hypotropia (55%) of the operated eye. Interestingly, most patients undergoing right cataract surgery developed a right hypertropia, while those undergoing left cataract surgery developed a left hypotropia (Figure 1). We cannot account for this finding although the handedness of the person administering the local anaesthetic could be relevant.[23]

Refractive

Anisometropia producing a prismatic effect or aniseikonia can easily be overlooked as a cause of double vision after cataract surgery. All our patients who developed diplopia owing to anisometropia became asymptomatic after refractive manipulation to eliminate the effects of the anisometropia.

Concurrent onset of systemic disease (including sixth nerve palsies)

Thyroid eye disease, Myasthenia gravis, Parkinson's disease, and Abducens palsy are all recognised causes of diplopia after cataract surgery.[1,5,15,16] One patient developed symptoms due to a new onset of thyroid orbitopathy. Seven others became diplopic after they developed a sixth nerve palsy. Although this could be related to anaesthetic myotoxicity or surgical trauma, in four of these cases, it could not have been the mechanism for the two patients whose surgery was performed under topical anaesthesia, or the third whose palsy occurred in the contralateral eye.

Central fusion disruption (acquired loss of fusion)

Pratt-Johnson and Tillson[17] reported 24 patients who developed intractable diplopia after removal of a long standing cataract. All the patients in their series had prolonged interruption of fusion due to unilateral visual loss from the cataract and/or prolonged uncorrected aphakia. They suggested the term central fusion disruption when referring to this acquired intractable diplopia occurring in visually mature patients. Sharkey and Sellar[18] reported three cases of acquired central fusion disruption after surgery for senile cataract. In our series, eight patients had cataract surgery at an early age and did not wear aphakic correction for a prolonged period of time. One patient had undergone a secondary IOL. The remaining seven patients were found to have intractable diplopia after contact lens wear. In patients for whom secondary IOL implantation is being contemplated, it is important to obtain a complete sensory evaluation with a contact lens in place. Patients with long standing unilateral cataracts should be warned of the possibility of acquired loss of fusion, a phenomenon that may occur even after 2½ years of uncorrected aphakia or dense unilateral cataract.[17]

Botulinum toxin injection, used in one patient in this series, is occasionally helpful to determine the patient's fusional status or even correct the manifest deviation[19] by temporarily reducing the deviation. In some cases, reducing the deviation may allow fusion to occur and eliminate the diplopia.

Monocular

Reported causes of monocular diplopia after cataract surgery include foveal dystopia,[1] macular degeneration,[7] IOL subluxation,[7] and high degrees of induced astigmatism. Four patients (2.5%) had monocular diplopia in this series. One was due to a subluxated IOL, which was surgically repositioned. One patient had macular degeneration; no aetiology could be identified in the remaining two patients, one of whom became asymptomatic over the next 2 years. Cortical monocular diplopia has been reported in the literature.[24]

Undetermined aetiology

We subdivided the 32 patients whose aetiology was undetermined into two groups: group 1 (22 patients) consisted of patients with a small comitant vertical deviation. These could have been caused initially by a traumatic palsy or anaesthetic myotoxicity with gradual partial resolution and spread of comitance, resulting in the small residual comitant deviations observed. Superior rectus bridle sutures were known to be used in two of these patients, not used in eight patients, and not known in the remaining 12 patients.

Patients in group 2 (10 patients) had a small esotropia (<20 PD) in the distance with only a small phoria (6 PD or less) at near, and full abduction of each eye. This small distance esotropia pattern is reminiscent of divergence paralysis. However, none of these patients had any associated neurological symptoms and formal neurological opinions were not sought. Our patients became asymptomatic with small base-out prisms in the distance segment of their glasses. This entity has not been

widely reported to be a cause of diplopia after cataract surgery.[15,25]

Anaesthetic technique

Twenty-two patients presented having undergone cataract surgery with topical anaesthesia. Whereas anaesthetic myoxicity and surgical trauma are the main cause for the motility disturbance after infiltrational anaesthesia, decompensation of a preexisting strabismus is the most common aetiology after cataract extraction with topical anaesthesia. Previous reports[26,27] have documented a potential protective effect with hyaluronidase reducing the incidence of diplopia following infiltrative anaesthesia.

With the increasing use of topical anaesthesia (Figure 3), the mechanism of diplopia after cataract surgery is likely to change from a mainly mechanical to a mainly sensory problem.

However, restrictive and paretic problems still occurred in the patients operated with topical anaesthesia particularly where a superior rectus bridle suture had been used (Table 2).

Management

The management of patients with double vision after cataract surgery depends to a large extent on the underlying aetiology. In some patients, the diplopia was temporary and observation is advisable in the early post-operative period. Early evaluation of the ocular motility pattern may help to identify the mechanism for the double vision, since spread of comitance may mask the true aetiology after several months have elapsed.

Prisms were the most commonly used treatment modality in this series (64%). These are usually prescribed as Fresnels, particularly where the deviation is small and comitant, and changed to a 'ground in' lenticular form once the deviation has been stable for 3 months or more. These are rarely suitable for markedly incomitant deviations, where strabismus surgery with adjustable sutures would be preferable once the deviation is stable. Twenty-nine patients (19%) underwent strabismus surgery in our series. Convergence exercises (5%) and optical manipulation (5%) were other useful treatment modalities. The use of Botulinum toxin, discussed above, should also be considered.

Conclusion

Diplopia after cataract extraction is an uncommon, but often distressing, sequel to otherwise successful cataract surgery. It accounted for 3% of the orthoptic workload in a tertiary care centre. In this series, we have discussed the likely causative mechanism in each case. Although myotoxicity remains a common underlying mechanism, decompensation of a preexisting phoria and CI are also important causes of double vision. This series highlights two unexplained causes of post-cataract extraction diplopia, a divergence insufficiency pattern and a small comitant vertical deviation. Both these groups of patients are helped by prism correction.

Although myotoxicity and surgical trauma have dominated the recent literature on diplopia after cataract surgery, this series shows that patients are still at risk of developing double vision after cataract surgery when topical anaesthesia and no bridle suture is used. As topical anaesthesia becomes the standard, a shift in the pattern of underlying aetiology can be anticipated, with an increasing preponderance of sensory rather than mechanical problems.

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