Clinical and Experimental Ophthalmology 2005; **33**: 90–96

Perspective

Refractive surgery and strabismus

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Abstract

This review discusses the potential for strabismic complications after refractive surgery for hyperopia, myopia, anisomyopia, astigmatism and monovision, and how to avoid these complications. Guidelines are given for assessing patients with strabismus seeking refractive surgery. Screening tests are suggested that lead to stratification of refractive surgery patients into different risk groups each warranting a different intensity of evaluation.

Key words: anisomyopia, astigmatism, hyperopia, monovision, myopia, refractive surgery, strabismus.

Introduction

How to identify and modify factors that may lead to strabismic complications in refractive surgery patients and how to assess strabismic patients who seek refractive surgery requires a good understanding of the underlying physiology of strabismus. Data regarding the frequency of strabismic complications of refractive surgery is only available from refractive surgery centres in which all patients have a detailed strabismus exam before and after the surgery, and has been collected by only a few studies; for example, for monovision and for low myopia with no strabismogenic risk factors. This review will cover the main areas of refractive surgery and discuss screening and risk stratification for refractive surgery patients.

Hyperopia

Some refractive surgeons treat hyperopia as if it were the mirror image of myopia, which is not at all the case. Hyperopia is quite different for several reasons [apart from any difficulties of performing hyperopia surgery]:

1 Hyperopes are more likely to:
- have an esodeviation or
- be predisposed to esodeviation
- have amblyopia.[1]

2 The habitual spectacle correction may be worn to give good vision and also to control an esodeviation. Good vision may require less hyperopic correction than the correction required to control any esodeviation. Sufficient surgical correction of hyperopia to give good vision may thus leave a patient with inadequate control of an esodeviation.

3 As discussed in detail later, the laser target may not always be easily defined. Furthermore, it is a moving target, largely depending on age.

4 Early visual success depends on all of the following:
- corneal reshaping
- residual accommodative amplitude
- residual hyperopia (magnitude and symmetry)
- binocular status (especially phoria magnitude and motor fusional reserves).

Late visual success depends on all of these, and also:
- presence and magnitude of latent hyperopia; and
- decay of accommodative amplitude with time.

It is important to understand the different components of hyperopia (Fig. 1).

1 Absolute hyperopia: minimum plus that brings the patient to threshold distance acuity; that is, that part of the total hyperopia for which the patient cannot compensate by the usual accommodation.

2 Facultative hyperopia: hyperopia for which the patient can compensate by accommodating and that can also be relaxed; the difference between the maximum and minimum plus that keeps the patient at threshold distance acuity (the difference between absolute and manifest hyperopia).

3 Manifest hyperopia: maximum plus that allows threshold distance acuity.

4 Latent hyperopia: the part of the total hyperopia normally compensated for by ciliary body tone and which cannot be revealed other than by cycloplegia. It typically becomes increasingly apparent and symptomatic (i.e. becomes 'manifest') with time.

Total hyperopia probably remains constant throughout life. Facultative hyperopia is 'elastic' and allows for variations in hyperopic correction for a particular patient while still maintaining threshold distance visual acuity (dotted arrows

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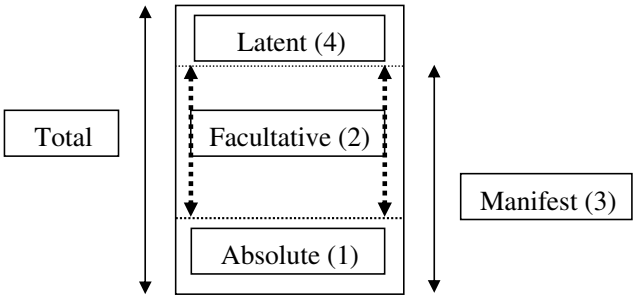

Figure 1. Components of hyperopia.

in Fig. 1). With time (years to decades), latent hyperopia gradually decreases to become manifest and more of the facultative hyperopia becomes absolute and requires correction as accommodative amplitude diminishes with age (Fig. 2).

Example 1

A patient with uncorrected vision 6/12 requires +1.00D to see 6/6 = his absolute hyperopia. He accepts an additional +1 (total +2) while maintaining 6/6 vision; +2.25 blurs. His manifest refraction is therefore +2. Facultative hyperopia is +1. Cyclopentolate refraction is +2.75 (total hyperopia). Latent hyperopia is +0.75.

Example 2

A 30-year-old with uncorrected vision 6/6 accepts +1.50 while maintaining 6/6; +1.75 blurs; absolute hyperopia is Plano. Manifest refraction and facultative hyperopia are both +1.50. Cyclopentolate refraction is +3.00D. Latent hyperopia is +1.50. Years later, the manifest refraction is +2.50, a dioptre of latent hyperopia having become manifest.

Short-term visual success of hyperopia surgery depends upon successfully treating the absolute hyperopia. Correction of just the absolute hyperopia may not maintain patient satisfaction in the medium to long term; as the accomodative amplitude (AA) declines and some of the facultative hyperopia becomes absolute and some of the latent hyperopia becomes manifest, the patient will seem to have recurrent hyperopia if only the absolute hyperopia is treated.

In Fig. 1, treatment of '1' in a 25-year-old will give good uncorrected distance vision and good near vision because the AA is still good. The same correction in a 40-year-old will probably give inadequate near vision. Treatment of {'1'+ '2'}, the manifest hyperopia, will give good distance vision at any age, and good near vision in a 40-year-old, but possibly inadequate near vision at 60 years. At any age, asymmetric treatment ('1' in one eye, {'1' +'2'} in the other) may cause one or more of visual discomfort, binocular blur relieved by closing one eye, and accommodative spasm with secondary esodeviation.

The strabismogenic potential of hyperopia surgery is in large part a manifestation of the presurgical binocular status,

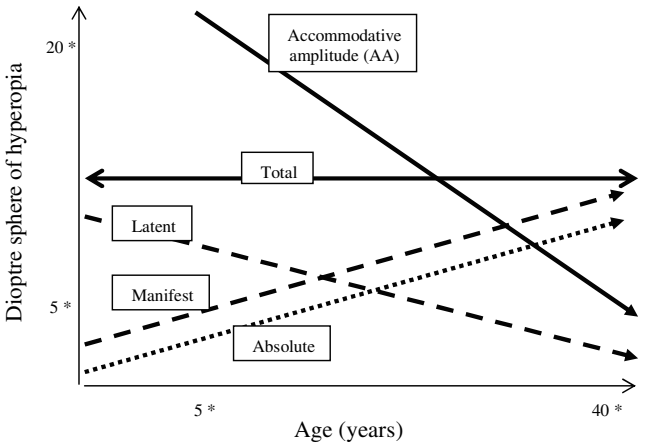

Figure 2. Change in the hyperopia components with age. Accommodative amplitude (AA) is initially +25.00D and slowly decreases with age. As AA decreases, absolute hyperopia and manifest hyperopia slowly increase. Latent hyperopia gradually decreases and becomes manifest. *Approximate numbers.

particularly predisposition to esodeviation with the absolute and manifest hyperopic correction in place. This potential can be appreciated by measuring the patient's range of motor fusion while wearing the absolute and the manifest hyperopia. It cannot be estimated – it has to be measured.

Motor fusion refers to the ability to align the eyes (or resist measures to misalign the eyes with prisms) while sensory fusion is maintained.[2] The range of motor fusion is measured with prisms of increasing strength placed before the eye(s) in a way that stresses both sensory and motor fusion, yet maintains binocular single vision. The fusion range is also referred to as fusional amplitude, fusional reserve, and positive or negative relative convergence or divergence!

How to assess motor fusional amplitudes

To assess motor fusional amplitudes,[3] ask a 6/6 patient to look at a 6/12 letter with both eyes (e.g. a single F or H [letters allowing easy recognition of diplopia]). Run a horizontal prism bar Base In from 1Δ upwards, pausing with each new power, until the patient experiences diplopia that cannot be resolved within a few seconds. Do the same with a Base Out prism. Repeat for near using a 'paddle' with a miniaturized letter chart. Values greater than 10Δ for each measurement are safe.

Patients should be considered high risk if (i) the measurement is ≤5Δ, or (ii) diplopia is not experienced despite a tropia on cover test. There may be a suppression scotoma, which needs to be quantified.

It is probably safe to operate on these patients if the horizontal fusion range is greater than ±5Δ, unless the latent hyperopia exceeds +2. Latent hyperopia will become manifest with time and a previously safe, but small, motor fusion range may become insufficient to maintain orthotropia.

Hyperopic laser in situ keratomileusis (LASIK) may eliminate the hyperopia in patients with accommodative esotropia and allow alignment without correction. The small amount of international experience allows cautious optimism for this as a recommended treatment for accommodative esotropia in older patients (if amblyopia is not an issue).[4–7]

Correction of only the absolute hyperopia is safe if a patient has large fusional reserves to keep the residual hyperopia from inducing an esotropia. It is usually preferable to correct all the manifest hyperopia for an optimal and lasting visual result.

An important exception is the patient with an unrecognized exodeviation that is being unknowingly controlled by accommodative convergence. This patient is easily identified if looked for; there will be an exophoria (or exotropia) and poor (or no) Base Out fusional reserves with the absolute refraction in place. Such patients need skilled evaluation. Leaving this patient with some uncorrected hyperopia to allow for accommodative convergence to control the exodeviation may defeat the purpose of the refractive surgery. Some will have full hyperopic correction and require sequential strabismus surgery.

Case report 1

A 32-year-old patient[8] had a well-controlled accommodative esotropia when wearing a cycloplegic refractive correction of right +3.50/+2.50 × 115 and left +3.50/+3.50 × 60. After bilateral LASIK there was a cycloplegic refraction of right +1.75/+1.50 × 115 and left +1.25/+1.75 × 60. Uncorrected visual acuity in each eye was 6/9+. The patient did not wear any optical correction and soon developed a 15Δ esotropia with constant diplopia that required strabismus surgery, which was successful.

Comment

Despite the change of more than +1.50 (and the improved uncorrected vision), the patient's esodeviation decompensated to a constant tropia, which would indicate that a correction greater than the absolute hyperopia was required to keep the esotropia under control. If the fusional amplitudes had been measured, it is likely that the patient would have had poor Base In fusional amplitudes to begin with and would have required a higher hyperopic correction than the absolute hyperopia for a safe fusion range.

Case report 2

A 24-year-old patient[8] had accommodative esotropia with convergence excess (so-called high AC/A ratio). The strabismus was well controlled with progressive additional lenses (PAL). The refractive surgeon was unaware that the patient wore a near add. After LASIK there was a near esotropia with diplopia, but the final outcome is unknown.

Comment

Convergence excess usually decreases before adolescence, but had not done so in this patient. All patients undergoing refractive surgery should have their spectacles tested on an automated vertometer to detect PAL in the occasional pre-presbyope. You should also mark the pupillary positions on the lenses for every patient to check for prisms that have been inadvertently dispensed or of which the patient is unaware.

Myopia

Surgery for myopia corrects the least minus dioptre sphere required to reach distance threshold. Habitually overcorrected myopia is seen occasionally in the intermittent exotrope who has been overminused to encourage accommodative convergence; beware if the manifest myopia is –0.50 greater than the cycloplegic refraction. A habitually overcorrected myope may experience a breakdown of fusion and subsequent exodeviation once the manifest myopia is surgically corrected. If the manifest myopia is greater than the cycloplegic myopia, have the patient wear the cycloplegic correction for 20–30 min in the waiting room and then assess the deviation and motor fusion reserves.

One of the authors has seen a patient in her 30s wearing –5.00D overcorrection to control an exodeviation and only recently develop asthenopic symptoms, and one in her 50s wearing a myopic correction –2.00D greater than manifest to control an exodeviation without discomfort.

Anisomyopia

Several different mechanisms may result in strabismus and diplopia in patients with anisomyopia, especially aniseikonia and reduced peripheral fusion (mildly strabismogenic).[9] Aniseikonia of only 3% can reduce central fusion, which can contribute to symptomatic strabismus.

If a vertical 10Δ prism is used to produce diplopia and the patient notices a difference in image size between the two images, aniseikonia is present and should then be quantified (e.g. with the Awaya New Aniseikonia Test book). None of the authors has experience with space eikonometers or synoptophores for these measurements.

It is important to get the previous optometry records in cases of significant anisomyopia. The current spectacle lenses may have been modified to minimize aniseikonia (e.g. by increasing front base curve, lens thickness and refractive index in front of the eye with the smaller image) and measurements may underestimate the aniseikonia.

Insofar as Knapp's Law is correct, axial ametropia corrected with a spectacle lens at the anterior focal plane of the eye results in a normal size retinal image. In high myopia, however, the retina is stretched with reduced retinal concentration of photoreceptors and this may abnegate Knapp's Law, with the resulting image minification demonstrable on an eikonometer. Contact lenses lessen the degree of image minification.[10,11]

Consider now the axial anisomyope who when corrected with optimal glasses has little/no aniseikonia and adequate motor fusion. If the anisometropia is now corrected by corneal surgery, corneal anisometropia will replace axial anisometropia. Any benefits of 'fine tuning' spectacle lenses to lessen aniseikonia are lost. This patient may now develop aniseikonia with disturbed motor and sensory fusion and risk of diplopia. It is not possible to reliably predict these effects for a particular patient vis-a-vis refractive surgery without doing a contact lens trial because there may be abnormal sensorial adaptations to pre-existing aniseikonia in addition to changes in photoreceptor density. A case could be made for giving contact lenses to all spectacle-corrected anisomyopes for at least a few hours to assess the subjective aniseikonic response and confirm a safe range of motor fusion.

Case report

A patient[8] had right –2.00D and left –8.00D with good fusion including 60″ stereopsis. After refractive surgery with resultant negligible refractive error bilaterally, he had intractable diplopia because of 7% aniseikonia.

Comment

Changing the anisometropic correction from the spectacle plane to the corneal plane created the problem, as predicted by Knapp's Law. Contact lens simulation of the post-surgical result may have predicted the possibility of aniseikonic diplopia in this patient (the positive predictive value of this simulation has not been studied). This type of diplopia is impossible to correct with strabismus surgery; 'size' (aniseikonic) lenses might be useful, but there is no local experience with them.

ASTIGMATISM

Refractive surgery for astigmatism can result in an undercorrection of power, a rotation of the axis or an entirely new astigmatism because of one of several mechanisms, including the entry of the wrong axis or power of the cylinder into the laser machine.

Some other mechanisms are monocular versus binocular fixation, postural change, subjective torsion and acquired astigmatism.

Monocular versus binocular fixation

Refractive surgery is done under monocular fixation. The eyes can cyclorotate when changing from binocular to monocular fixation, and topography done with monocular fixation can show an axis shift compared with topography done with binocular fixation.[12] The patients most likely to demonstrate this difference are those with cyclovertical muscle disturbances (e.g. hitherto compensated 4th nerve palsy), but this can also be seen in 'normals'.[12]

Postural change

Rotation has also been demonstrated while changing from sitting to the supine position, such as from the refraction or topography position to the surgical position.[12,13]

The combination of monocular fixation and position-induced torsion change can significantly alter the axis and amount of ablation, resulting in undercorrection of the astigmatism or induction of new astigmatism. The resultant blur and refractive asymmetry could stress fusion.

Subjective torsion

Subjective torsion can occur because of induced astigmatism and the inability to fuse these images results in diplopia (Kushner B & Guyton D, pers. comm.).

Case report 1

A 34-year-old patient[8] had a pre-operative cycloplegic refraction in the left eye of –5.00/+2.50 × 85. After radial keratotomy (RK) in the left eye, it was –2.00/+3.25 × 55. This 30° rotation in the axis of astigmatism resulted in an optically induced 7° excyclotropia as measured with Double Maddox Rods. Although he was orthotropic and had no objective fundus torsion, he remained symptomatic. He was able to fuse the torted image and had fusional amplitudes on the synoptophore when the torsional misalignment was simulated.

Comment

A patient may be unable to adapt to induced cylinder after an imperfect keratorefractive result. The absence of torsion on orthoptic evaluation and absence of fundus torsion indicated that the change in cylinder had induced a torsional diplopia that the patient could not fuse. Such symptoms may not be easily explained by the patient or appreciated by the refractive surgeon.

Case report 2

A 40-year-old patient[8] had refractive error right –4.00/+4.00 × 90, but –4.00/+4.00 × 180 was programmed into the laser computer. After surgery it was –8.00/+8.50 × 170 with binocular diplopia. Although orthophoric, the patient could not fuse the images because of 9% aniseikonia and image distortion.

Acquired astigmatism

A newly acquired astigmatism or new anisoastigmatism can cause accommodative spasm,[14] which can cause asthenopic symptoms in a patient whose astigmatism has been imperfectly or asymmetrically corrected or has been changed. This accommodative spasm can predispose to symptomatic esodeviation.

Planned monovision

With planned monovision, one leaves the patient Plano in one eye and –1.50 in the other eye, but problems may occur because (i) an amblyopic eye is now dominant for some tasks and (ii) a new anisometropia might lessen motor fusion (one can pre-test with contact lens simulation, although the positive predictive value of this simulation has never been evaluated).

Fawcett *et al*.[15] reported 118 refractive surgery patients of whom 48 had planned monovision. Of these 48, 11 had symptoms of abnormal binocular vision (being one or more of intermittent or persistent diplopia, visual confusion, and 'binocular … blur requiring occlusion to focus comfortably'). Of the 70 patients who did not have monovision, only two had abnormal binocular vision as defined earlier. Average anisometropia in the 13 patients with abnormal binocular vision was 1.90 compared with 0.50 in the 105 patients with normal binocular vision ($P < 0.05$).[1]

Fawcett *et al*. also showed that surgical monovision can produce an uncorrectable deficiency of high grade stereopsis, quite different to patients with contact lens monovision who typically spend some time every day without monovision.[15] Sherafat *et al*. have shown that patients with longstanding asymmetrical keratoconus experience a similar breakdown of binocular visual function.[16]

Kushner and Kowal report three mechanisms of diplopiogenesis in monovision patients.[8]

1. Intermittent strabismus or a phoria with poor reserves can decompensate into a constant tropia because of the degradation of high-grade foveal fusion (as demonstrated by Fawcett *et al*[15]).
2. Fixation switch. In some circumstances, the amblyopic eye (with the scotoma) becomes the fixing eye. The habitually fixing eye is now the deviating eye: there is no suppression or amblyopia scotoma in the now deviating eye, and diplopia may ensue.
3. Fixation switch diplopia can occur by another mechanism. In a well-compensated 4th nerve palsy, if the paretic eye is forced to preferentially fix, then secondary deviation will result in a larger tropia of the non-paretic eye, which could exceed the previously established fusional amplitudes and result in diplopia.

Temporary surgical monovision was seen routinely in the early days of refractive surgery when there was a planned 3 month delay between treatment of each eye. Of 50 patients reported by White, only one had fusional convergence decrease from 35Δ to 5Δ. All patients (including the one highlighted) were asymptomatic.[17] Surgical monovision of 3 months duration thus seems not to have the same morbidity as permanent surgical monovision.

Case report

A 52-year-old patient[3] had right –6.00 =6/6 and left –6.00/–1.00 × 95 = 6/8. It was planned to correct the right eye for distance and the left eye for near. If the left is mildly amblyopic and the right the habitually fixing eye, then the 3Δ microesotropia, which has not been appreciated, is not important. However, if the left is forced to be the dominant (fixing) eye for near vision, the right does not have any sensory adaptation to a 3Δ microesotropia and diplopia ensues whenever the patient reads (fixable with prisms).

Angle kappa and decentration

An angle kappa means that the line-of-sight of the patient does not coincide with the centre of the pupil. It is not uncommon and can result in both flap and treatment zone decentration, creating a significant horizontal prismatic effect. The prismatic effects of such decentration are typically well within the range of normal horizontal motor fusion, but a patient with poor fusion range is at risk of developing strabismus and diplopia in this situation.

Vertical fusional ranges are normally poor ($\leq \pm 3\Delta$). A small vertical decentration is more likely to cause diplopia.

Case report

A patient[18] developed postoperative binocular diplopia after unilateral LASIK for –23.00D. He had difficulty maintaining fixation during the procedure and the treatment zone was decentred upwards, inducing a 16Δ vertical prism. Overlap of two different-sized images was achieved with either a hard contact lens or a prism. Follow-up of several years showed stability of symptoms and continuing need for the prism.

Comment

Such large corrections are no longer attempted with corneal refractive surgery. The vertical motor fusional range is too small to compensate for the large induced prismatic deviation. Following evaluation of this patient, L.K. examined approximately 5000 consecutive patient in a strabismus practice for vertical angle kappa and it was found in only one patient.

Refractive surgery in patients with strabismus

One needs to answer two related, but different, questions for each patient.

Question 1. What is the risk of deterioration of the strabismus?

1. Spontaneously (no refractive surgery).
2. Following successful refractive surgery.
3. Following imperfect refractive surgery.

If the strabismus deteriorates it is likely that it will be blamed on the refractive surgery. Spontaneous deterioration is more likely if there is a version or duction deficit already

present or a cyclovertical disturbance (e.g. oblique 'overaction') or alphabet pattern is already present.

An imperfect refractive result may worsen a strabismus. An eso tendency will be produced by a surprise hyperopic result (especially in the dominant eye), surprise astigmatism, and surprise anisometropia, all of which may produce unexpected accommodation or accommodative spasm.

Question 2. What is the risk of diplopia developing?

1. Spontaneously (no refractive surgery).
2. Following successful refractive surgery.
3. Following imperfect refractive surgery.

The strabismus patient requesting refractive surgery is not the usual strabismus patient. The amblyopic eye will be 6/12 or better, not the usual case seen in adult strabismus clinics. Acuity is a guide to the depth and size of suppression scotoma, and we thus anticipate an over-representation of shallow and small scotomas in this population. It then becomes important to assess the 'depth' and 'size' of the scotoma: a patient with a small shallow scotoma is at higher risk of diplopia than a patient with a large deep scotoma.

To assess the depth of a suppression scotoma, use a Bagolini (Sbisa) filter bar,[19] which measures how much retinal rivalry is required to successfully overcome a suppression scotoma. A value of '1' or '2' is indicative of a shallow scotoma; some of the patients will recall episodes of spontaneous, transient non-troublesome diplopia. A value of ≥'5' indicates a deeper and probably 'safe' scotoma.

One way to measure the size of the suppression scotoma is the polarized 4-dot test developed by Arthur and Lai,[20] which uses circularly polarized stimuli of the same colour viewed through polarized translucent glasses. There is no retinal rivalry (images are the same colour) and there is minimal dissociation. The size of the suppression scotoma can be mapped from a fraction of 1° to 5°. A large scotoma probably predicts a tolerance for a change of strabismic angle.

If strabismus is present with refractive error, refractive surgery should normally be performed first because it may have an effect on the strabismus angle. Some time should pass to allow for regression of the refractive effect before the strabismus is assessed for realignment surgery.[21] If strabismus surgery is performed first, the so-called fornix approach is better because it enables normal microkeratome suction.

Surgical correction of marked ametropia in an amblyopic eye can correct the associated strabismus by improving peripheral fusion.[22]

Recommended screenng tests

Minimum tests

1. History. Routinely ask about prior strabismus, episodes of diplopia, any known prism in spectacles, bifocals in a prepresbyope or eye exercises in the past.
2. Check current glasses for prism and PAL. Mark the optical centres of the lenses while the patient is wearing them. Use an automated vertometer that will detect prism without any special effort on your part.
3. Cover–Uncover and Alternate Cover Test for distance and near while the patient is wearing the habitual and then the targeted optical correction.
4. Refraction. (a) Manifest: For myopes, least minus required for threshold acuity. For hyperopes we need to know the least plus for threshold (= absolute) and the most plus for threshold (= manifest). For near, use a threshold card that can test to N3+ (M 0.3 or 0.4). Near threshold testing will give an explanation to the patient with N5 vision with near blur whose complaints have not been appreciated because s/he has always had suprathreshold testing. (b) Cycloplegic: The difference between cycloplegic and manifest hyperopia is latent hyperopia. Any value >+1.00D is worth noting, and >+2.00D may be a relative contraindication to hyperopia surgery. In myopia, any value >0.50 difference between cycloplegic and manifest myopia is significant.

Additional tests

1. Fusional amplitudes: To be performed in all hyperopes and if there is a history or finding of diplopia, strabismus, prism in spectacles or a moderate-sized phoria.
2. If the patient habitually wears prisms, conduct a trial with neutralizing prism.
3. Astigmatic axis viewing monocularly and binocularly: If substantially different, measure again on the operation table.
4. Trial of monovision contact lenses if monovision is the desired outcome and the patient has substantial phoria, prisms in glasses, or poor motor fusion.

Risk stratification

No risk group

These are patients who have all of the following.

1. Myopia with ≤4 anisometropia.
2. No history of strabismus or diplopia.
3. No prism in the glasses.
4. No or minimal phoria on an alternate cover test.
5. Current spectacles, manifest refraction and cycloplegic refraction are all within 0.50.
6. Accommodative esotropia with good fusional reserves (≥± 10Δ) while wearing the absolute hyperopic correction.
7. Previous strabismus surgery and have good fusional reserves while wearing their correction.

Moderate risk group

Patients not satisfying the above criteria are to be considered at least at moderate risk. One should test for motor fusion in these and the following patients.

1 Patients undergoing surgery to correct a substantial astigmatic error and who have a considerable difference between the axis of astigmatism under binocular and monocular fixing conditions are at risk of inadequate or inappropriate correction of their astigmatism. Patients who also have subtle strabismus with cyclovertical disturbance are at particular risk.
2 Accommodative esotropes with poor fusional reserve (<5Δ) are at risk of recurrent strabismus.
3 Presence of ≥2.00D latent hyperopia may result in late decompensation to esotropia.
4 Patients wearing spectacles with prism. A trial of spectacles with the prism neutralized ('stuck on' or Fresnel prism) is required to predict the risk of postoperative diplopia.

High-risk group

1 Monovision patients who develop diplopia while on a monovision trial with contact lenses are at risk of developing postoperative diplopia.
2 An accommodative esotrope requiring substantially more plus correction than the absolute hyperopia to control the deviation.
3 Patients with >4.00D of anisometropia with good fusion are high risk candidates for post op diplopia (possibility of aniseikonia).
4 Patients with manifest strabismus.

A patient who falls into the high-risk group can usually still undergo refractive surgery if all the risks are evaluated and the patient accepts the possibility of undergoing strabismus surgery if required later. If refractive surgery has to be done on a patient with strabismus, it is preferable to do the refractive surgery first.

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