Review article

Restrictive problems related to strabismus surgery

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Abstract

Strabismus surgery may be responsible for some restrictions in ocular motility that may cause new problems after surgery. Most of the time these restrictions present as a complex motility problem after surgery that requires further treatment. There may be various reasons that cause motility restriction following strabismus surgery. Those are excessive shortening or inadvertent capture of extraocular muscles, transposition procedures and, the most challenging problem, postoperative scar tissue-adhesion formation. In this review the potential reasons for postoperative restrictive problems, preventive measures and finally the treatment options for such problems are overviewed.

1. Introduction

The aim of strabismus surgery is primarily to correct ocular misalignment and to keep both eyes aligned in nine positions of gaze with free ocular movements. However, in some patients that goal may not be achieved by surgical intervention and strabismus surgery itself may be the reason for ocular misalignment and restrictive ocular motility problems.

The tissue that causes a restriction may limit the rotation of the eye both in the opposite and in the same direction which is called as “leash” and “reverse leash” effect by Jampolsky [1]. Figure 1 demonstrates the schematic representation of “leash” and “reverse leash” effect.

The tissue that causes a restriction of ocular movements may be the extraocular muscle, conjunctiva, soft tissues around the extraocular muscles and orbital adhesions that possibly affect the extraocular muscle pulleys.

2. Diagnosis

When a postoperative limitation of ocular movement is observed, the surgeon first needs to know whether it is due to a restriction or due to weakness of an extraocular muscle. For differential diagnosis, forced duction test, forced generation test, intraocular pressure change on side gazes and saccadic velocity measurements can be used. Slit lamp examination may also give clues about excessive conjunctival scarring and the presence of orbital fat tissue under the conjunctiva at the early postoperative stage.

The forced duction test is a simple test that can be performed at the examination room with topical anesthesia in adults. Use of the forced duction test during surgery provides very important additional information if it is repeated at different stages of surgery. In order to find out which tissue is responsible for the restricted ocular movement, the forced duction test must be repeated before and after dissection of conjunctiva, Tenon’s capsule and extraocular muscles. That will allow to determine the tissue that causes the restriction. Sometimes the forced duction test may still be positive after disinsertion of the extraocular muscle, which indicates the presence of orbital fibrosis.

The forced generation test is useful to rule out a lost muscle problem after surgery. Intraocular pressure change on gaze positions also gives an idea about the presence of a restriction and any change exceeding 4 mmHg is suggestive of a restrictive motility problem. Saccadic velocity measurement gives reliable information but it is not as easy as the other methods to perform and requires some equipment which is not available in most clinics.
3. Etiology — preventive measures

The problem that causes restricted ocular movement may be at the extraocular muscles, conjunctiva, Tenon’s capsule, soft tissues surrounding the extraocular muscles and at the orbital structures.

Etiology of restricted ocular movements may be summarized in four groups:

1. restrictions related to excessive shortening of an extraocular muscle;
2. restrictions related to inadvertent capture of a neighboring muscle;
3. restrictions related to transposition surgery; and
4. restrictions related to postoperative scar tissue — adhesions.

3.1. Restrictions due to excessive shortening of an extraocular muscle

Excessive shortening of an extraocular muscle may develop either due to excessive resection or excessive tucking-plication of an extraocular muscle. Excessive shortening of an extraocular muscle causes a restriction towards the opposite side of the functional area of the shortened extraocular muscle. In order to avoid that complication, the amount of resection must be carefully determined. In surgical tables the conventional maximum amounts of resections show some variability. The surgeon needs to consider that the elasticity of an individual muscle may have some variability and in some certain conditions even the conventional amounts of resection may cause some restrictive motility problems in concomitant strabismus. In all resections, performing forced duction test must be a routine procedure. In dysinnervational problems such as Duane syndrome and congenital fibrosis of extraocular muscles, resections should be avoided as a golden rule. There may be only rare exceptions to perform resections in Duane syndrome which should be regarded with great caution [2].

3.2. Restrictions related to inadvertent capture of a neighboring muscle

If it is placed too anteriorly or the posterior fibers are spread too temporally. In order to reduce that risk, the posterior fibers are recommended to be sutured in a bunched up fashion and the placement of sutures should not be placed anterior to the inferior rectus insertion.

3.3. Restrictions related to transposition surgery

Transposition of rectus muscles may cause a restriction towards the eye movement in the opposite direction with the transposition. As an example, transposition of vertical recti laterally in an abducent palsy may cause limitation on adduction. In order to avoid that complication, the forced duction test should be performed and if any restriction is felt, transposition should be performed with a small recession of the transposed muscle [3]. The risk for a restriction increases with Foster augmentation sutures and a small restriction usually does not cause any problem in an abducent nerve palsy. However if it occurs in Duane syndrome where there is some limitation of adduction preoperatively, the restriction may cause significant problems. If there is a restriction in the preoperative forced duction test, the augmentation sutures may be placed more anteriorly, enough to allow a free forced duction test.

Transposition of oblique muscles may also cause some restrictions. Anterior transposition of the inferior oblique muscle may cause a limitation of elevation—antielevation syndrome — if it is placed too anteriorly or the posterior fibers are spread too temporally. In order to reduce that risk, the posterior fibers are recommended to be sutured in a bunched up fashion and the placement of sutures should not be placed anterior to the inferior rectus insertion.

3.4. Restrictions related to postoperative scar tissue — adhesions

One of the major problems in strabismus surgery is the development of postoperative adhesions, particularly in patients who require multiple strabismus surgeries. The adhesions may develop in the conjunctiva, Tenon’s capsule, intermuscular membrane, orbital fat, sclera or extraocular muscle. Such adhesions may cause limitation of ocular motility despite an appropriate amount of extraocular muscle surgery.

Adherence syndrome is described by Parks [4] and he used this term specifically for the motility disturbance secondary to hemorrhage and prolapse of orbital fat tissue following inferior oblique surgery. Later on he suggested that the fat is not directly involved and the loss of elasticity of the septae in the extraconal space that
restrictive motility problems cannot be solved without the release of the tissues that cause the restriction. If it is the extraocular muscle itself that causes the restriction, it is relatively straightforward to release that restriction. If the problem is excessive resection, then the resected extraocular muscle needs to be recessed. In patients where excessive resection is responsible for motility restriction, botulinum toxin A (BTXA) may be very helpful, especially during the acute phase by allowing a reorganization of the length tension properties of the extraocular muscles. In our clinical practice our first-line treatment is to inject BTXA into the excessively resected muscle. If this fails, surgery may be considered. The following case represents an unusual example of the effect of resection in a case with cerebral palsy [25].

4.1. Case 1

A 7-year-old girl with Parinaud syndrome, right esotropia (20 prism dioptries) and cerebral palsy underwent bilateral superior transposition of horizontal recti, right medial rectus recession (3 mm) and right lateral rectus (5 mm) resection (Figure 2A). On the 1st postoperative day she had a large-angle exotropia (45 prism dioptries) with severe limitation of adduction (Figure 2B). She was reoperated with a preoperative diagnosis of a disinserted medial rectus muscle but the extraocular muscles were found in the appropriate position. A forced duction test was positive on adduction. The lateral rectus muscle was recessed for 5 mm in an attempt to neutralize the resection of the lateral rectus muscle. On the next day the exodeviation was slightly better but the severe limitation of adduction persisted (Figure 2C). Her eyes remained stable over the following 5 years. In this case, BTXA allowed a reorganization of the mechanical contractile forces and eliminated the abnormal contractility of the lateral rectus muscle [25].

If the problem is excessive tucking of superior oblique tendon, the treatment depends upon the severity. A mild to moderate amount of Brown syndrome resolves in time if tuck surgery is performed in a lax superior oblique tendon and does not require any correction. However if iatrogenic Brown develops in an acquired superior oblique palsy with normal tendon, it does not resolve in time and requires treatment if the patient is symptomatic.

If the restriction is due to the soft tissue scarring, such as orbital fat tissue prolapse, the problem is much more complicated because prolapse of orbital fat tissue causes a development of excessive adhesions and fibrosis among all of the tissues in the field. When adherence syndrome is developed, it is not possible to ‘cure’ it and all treatment attempts aim to make it ‘better’.

If the development of adherence syndrome is recognized during the acute phase, release of the antagonist muscle by BTXA injection may be helpful [26]. The mechanism of BTXA is similar with a traction suture where the globe is aimed to be kept in a certain position during the healing period. Injection of BTXA during the acute phase allows the eye to move towards the opposite side of the injected muscle, which keeps the eye in the primary position during the development of fibro fatty inflammatory reaction (Figure 3). Figure 4 demonstrates a sample case (Case 2) of the use of BTXA in a case with adherence syndrome and also a lost muscle.

4.2. Case 2

A patient was referred for lost lateral rectus muscle 7 days after her first surgery. She had a previous esodeviation, medial rectus...
Figure 2. Case 1. (A) Parinaud syndrome, right esotropia and cerebral palsy. (B) Consecutive exodeviation with severe limitation of adduction on the 1st postoperative day, following bilateral superior transposition of horizontal recti, right medial rectus recession (3 mm) and right lateral rectus (5 mm) resection. (C) Consecutive exodeviation and limitation of adduction persisted after second surgery. Preoperative forced duction was (+) on adduction, MR muscle was found in the correct location, LR was recessed for 5 mm. (D) No horizontal deviation 3 months later following botulinum toxin injection into the right LR muscle.

Figure 3. The schematic representation of the effect of BTXA in adherence syndrome. The prolapse of orbital fat tissue induces an inflammatory reaction which pulls the globe and attaches to the point ‘A’. The injection of BTXA allows the eye to be kept in primary position (PP) and the fibro-fatty tissue attaches the globe at a more posterior point.
was recessed, lateral rectus was resected and then lost. At presentation she was found to have excessive inflammatory reaction and conjunctival edema. Orbital fat tissue was recognized under conjunctiva and the diagnosis was fat-adherence syndrome in combination with a lost lateral rectus muscle (Figure 4A). Because of the excessive inflammatory reaction we preferred to wait for 6 weeks. In order to keep the eye in the primary position during this period, BTXA was injected into the left medial rectus muscle. Orthophoria was achieved on the 1st postinjection week with limited abduction related to lost lateral rectus muscle and with limited adduction related to the BTXA effect (Figure 4B). Six weeks later she underwent operation. Forced duction test was severely positive both on adduction and abduction. Lateral rectus muscle was found within the orbital fat tissue and it could be reattached to the globe with some recession as it could not be possible to pull the muscle to the original insertion. Excessive scar tissue was also found at the medial rectus area and these tissues were excised. Two months after reattachment of lateral rectus muscle to the globe the eyes were well aligned but there is a limitation of both adduction and abduction related to fat-adherence syndrome (Figure 4C).

After the development of fibrosis BTXA cannot be effective and the problem can only be reduced with another surgery. In such

Figure 4. Case 2. (A) The patient was referred for lost lateral rectus muscle 7 days after her first surgery. Orbital fat tissue was recognized under the conjunctiva with excessive inflammatory reaction. Abduction was severely limited. (B) Botulinum toxin A is injected into the left medial rectus muscle and orthophoria was achieved on the 1st postinjection week with limited abduction related to lost lateral rectus muscle and with limited adduction related to the BTXA effect. (C) Two months after reattachment of lateral rectus muscle to the globe. Note that the eyes are well aligned but there is a limitation of both adduction and abduction related to fat-adherence syndrome.
cases the aim is to achieve orthophoria in primary position. Some limitation of ocular motility remains in all cases. A satisfactory improvement in primary position is reported in a series of 11 patients, with extensive 180° conjunctival peritomy and recession of conjunctiva in combination with inferior rectus recession [27]. The authors emphasized that the expectation of the patients must be realistic and it is not possible to obtain free ocular movements. It is important to remember that once the integrity of fascial system is lost and once the orbital soft tissue scarring occurs it is not possible to eliminate it in total with any type of treatment.

5. Conclusions

Restrictive problems related to strabismus surgery represent a challenging motility problem for the strabismus surgeon. The primary goal must be to prevent the development of such problems during surgery. Nontraumatic surgery is essential to prevent excessive scar tissue. Forced duction test that is carried out routinely is an effective preventive measure to reduce the risk of postoperative restrictive motility disorders and forced duction test must be repeated before, during and at the end of the surgery as a routine in all types of strabismus operations. BTXA may be very helpful to “rescue” a restriction if it is performed during “acute” phase of the problem.

References

1. Jampolsky A. Surgical leashes, reverse leashes in strabismus surgical manage- ment. In: Helveston EM, ed. Symposium on Strabismus, Transactions of the New Orleans Academy of Ophthalmology. St Louis, MO: CV Mosby Co; 1978:244–268.
2. Kraft SP. Lateral rectus resection strabismus surgery in unilateral Duane syn- drome with exotropia and limited abduction. Binocul Vis Strabismus Q. 2010;25: 149–157.
3. Rosenbaum AL. The efficacy of rectus muscle transposition surgery in esotropic Duane syndrome and VI nerve palsy. J AAPOS. 2004;8:409–415.
4. Parks MM. Causes of the adhesive syndrome. In: Helveston EM, ed. Symposium on Strabismus, Transactions of the New Orleans Academy of Ophthalmology. St Louis, MO: CV Mosby Co; 1978:269–270.
5. Parks MM. Discussion of Wright KW: the fat adherence syndrome and stra- bismus after retina surgery. Ophthalmology. 1986;93:415.
6. Mora JS, Sprunger DT, Helveston EM, Evan AP. Intraoperative sponge 5- fluorouracil to reduce postoperative scarring in strabismus surgery. J AAPOS. 1997;1:92–97.
7. Urban Jr RC, Kaufman LM. Mitomycin in treatment of hypertrophic conjunctival scars after strabismus surgery. J Pediatr Ophthalmol Strabismus. 1994;31:96–98.
8. Searl SS, Metz HS, Lindahl KJ. The use of sodium hyaluronate as a biologic sleeve in strabismus surgery. Ann Ophthalmol. 1987;19:259–268.
9. Elkas FJ, Gowda DC, Urcy DW. Synthetic polypeptide for strabismus surgery. J Pediatr Ophthalmol Strabismus. 1992;29:284–286.
10. Sondhi N, Ellis FD, Hamed LM, Helveston EM. Evaluation of an absorbable muscle sleeve to limit post operative adhesions in strabismus surgery. Ophthalmic Surg. 1987;18:441–443.
11. Ozkan SB, Kir E, Culhaci N, Dayanir V. The effect of Seprafilm on adhesions in strabismus surgery-an experimental study. J AAPOS. 2004;8:46–49.
12. Ryu WY, Jung HM, Roh MS, et al. The effect of a temperature-sensitive poloxamer-alginate-CaCl2 mixture after strabismus surgery in a rabbit model. J AAPOS. 2013;17:484–489.
13. Cruz OA, Matkovich L. Effects of intraoperative topical mitomycin-C on stra- bismus surgery in the rabbit: a preliminary study. Ophthalmic Surg. 1995;26: 237–240.
14. Minguzzi N, Monteiro de Carvalho KM, Akaishi PM, De Luca IM. Histologic ef- fect of mitomycin C on strabismus surgery in the rabbit. Invest Ophthalmol Vis Sci. 2000;41:3399–3401.
15. Egme A, Yildirim C, Tatlipinar S, Düzcan E, Yaylali V, Ozden S. Effects of intraoperative sponge mitomycin C and 5-fluorouracil on scar formation following strabismus surgery in rabbits. Strabismus. 2004 Sep;12(3):141–148.
16. Yamada M, Shinoda K, Hatakeyama A, Nishina S, Mashima Y. Fat adherence syndrome after retinal surgery treated with amniotic membrane transposition. Am J Ophthalmol. 2001;132:280–282.
17. Demirel S, Atilla H, Okcu Heper A, Erkam N. Effects of amniotic membrane on wound healing and adhesions in experimental strabismus surgery. Eur J Oph- thalmol. 2009;19:899–904.
18. Kasseem RR, Gawdat GI, Zedan RH. Severe fibrosis ofextraocular muscles after the use of lyophilized amniotic membrane in strabismus surgery. J AAPOS. 2010;14:548–549.
19. Strube YN, Conte F, Faria C, Yiu S, Wright KW. Amniotic membrane trans- plantation for restrictive strabismus. Ophthalmology. 2011;118:1175–1179.
20. Kasseem RR, Abdel-Hamid MA, Khodeir MM. Effect of lyophilized amniotic membrane on the development of adhesions and fibrosis after extraocular muscle surgery in rabbits. Curr Eye Res. 2011;36:1020–1027.
21. Chun BY, Kim HK, Shin JP. Dried human amniotic membrane does not alleviate inflammation and fibrosis in experimental strabismus surgery. J Ophthal- mol. 2013;2013:309126.
22. Kasseem RR, Khodeir MM, Salem M, et al. Effect of cryopreserved amniotic membrane on the development of adhesions and fibrosis after extraocular muscle surgery in rabbits. Acta Ophthalmol. 2013 Mar;51:e140–e148.
23. Tugcu B, Helvaciloglu F, Yuzbasoglu E, Gurec C, Yigit U. Amniotic membrane in the management of strabismus reoperations. Jpn J Ophthalmol. 2013;57: 239–244.
24. Kirsch D, Lowen MS, Fialho Cronemberger MF, Sato EH. Amniotic membrane for reducing the formation of adhesions in surgery: experimental study in rabbits. J Pediatr Ophthalmol Strabismus. 2014;51(6):341–347.
25. Ozkan SB. Role of botulinum toxin to rescue failed strabismus surgery. In: Ozkan SB, ed. Update on Strabismology. Ankara: Rotatip Publishers; 2010:37–41.
26. Ozkan SB, Kur E, Dayanir V, Dündar SO. Botulinum toxin A in the treatment of adherence syndrome. Ophthalmic Surg Lasers Imaging. 2003;34:391–395.
27. Burton B, Dawson E, Lee J. Adherence syndrome following inferior oblique surgery: management and outcome of 14 cases. Strabismus. 2004;12:169–174.