MECHANICAL STRABISMUS FOLLOWING OCULAR SURGERY

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LEARNING OBJECTIVES
1. Describe the mechanism and recognize the clinical course of strabismus due to anesthetic myotoxicity
2. Describe three causes of strabismus following implantation of ocular devices
3. List three ways to prevent the occurrence of postoperative strabismus
4. Understand appropriate indications for surgery to treat iatrogenic strabismus following ocular surgery

CME QUESTIONS
1. Which of the following best portrays the incidence of involvement of extraocular muscles in anesthetic myotoxicity in decreasing order?
   a. Superior rectus, inferior rectus, lateral rectus, inferior oblique
   b. Inferior rectus, inferior oblique, superior rectus, lateral rectus
   c. Inferior rectus, superior rectus, inferior oblique, lateral rectus
   d. Inferior oblique, inferior rectus, superior rectus, lateral rectus

2. A patient undergoes a scleral buckling procedure OD under peribulbar anesthesia with bupivacaine. The buckle is placed for 200 degrees superiorly. One day postoperatively he notices binocular vertical diplopia with the image from the operated eye lower than that of the fellow eye. This slowly improves over a few weeks but one month postoperatively he demonstrates a right hypotropia of 15 PD with an elevation deficit of the right eye. Which of the following is the most likely cause of the diplopia?
   a. Anesthetic myotoxicity to the right inferior rectus
   b. Anesthetic myotoxicity to the right superior rectus
   c. Restrictive strabismus from the scleral buckle
   d. Intraoperative stretch trauma to the right superior rectus

3. What is the most common cause of isolated unilateral inferior oblique paresis?
   a. Anesthetic myotoxicity
   b. Implantation of a glaucoma drainage implant
   c. Direct surgical trauma during lower lid blepharoplasty
   d. Fat adherence syndrome following orbital decompression

KEYWORDS
1. Diplopia
2. Mechanical Strabismus
3. Anesthetic Myotoxicity
4. Ocular Surgery
5. Bupivicaine

INTRODUCTION
Strabismus or diplopia after ocular surgery, particularly one that is otherwise uncomplicated, is a distressing situation for patients and physicians alike. Multiple mechanisms play a role in this phenomenon, including decreased visual acuity leading to sensory deviations, defects in the supranuclear fusion system resulting in manifest deviations, and changes in refractive error resulting in fixation switch or anisokonia/anisophoria. The focus of this presentation is postoperative strabismus secondary to iatrogenic mechanical causes.

MECHANICAL STRABISMUS AFTER CATARACT SURGERY
The most common cause of mechanical strabismus following cataract surgery is myotoxicity from local anesthesia, which occurs in 0.1% to 1% of cases using peribulbar or retrobulbar blocks; although much less common, sub-Tenon's delivery of local anesthesia can also produce a similar result. In addition to direct injury from the needle itself, myotoxicity from either lidocaine or bupivacaine (the latter more severe) also occurs. It is important to realize that direct injection of the agent must occur in order for myotoxicity to develop; mere exposure to drug does not produce any muscle damage.
Use of hyaluronidase in the injection cannula may help to spread the anesthesia more evenly and avoid higher concentrations of drug, thus diminishing the incidence of myotoxicity.  

The typical sequelae of anesthetic myotoxicity of the extraocular muscles are an initial paresis which is replaced by progressive segmental fibrosis and hypertrophy of the muscle over a period of 3-8 weeks; the segmental nature of the cicatricial response may produce a true overaction of the affected muscle. Thus, damage to the inferior rectus will immediately result in an ipsilateral hypertropia followed over several weeks by improvement of the hyperdeviation and an eventual hypotropia with the greatest deviation in downgaze. The temporal profile of this reversal of deviation is almost pathognomonic for anesthetic myotoxicity.  

By virtue of the location of the retrobulbar or peribulbar injection (generally given inferiorly), the most common muscle involved is the inferior rectus. However, Capo and associates have demonstrated via cadaver studies that the superior rectus may also be affected in such injections, bypassing the optic nerve and injecting the muscle very posteriorly in the orbit, although approximately 5 times less frequently than the inferior rectus.  Other authors have also demonstrated involvement of the horizontal rectus and oblique muscles.  

Over the years, the frequency of this post-cataract mechanical strabismus has progressively diminished, primarily due to use of topical anesthesia for cataract surgery. Discontinuation of a bridle suture around a muscle to stabilize the globe has also contributed as well. Anesthetic myotoxicity is also finding a new role in the treatment of strabismus. To increase muscle contractile strength and elastic stiffness, often in conjunction with injection of botulinum toxin to the direct antagonist; preliminary reports demonstrate that an average of 20 PD of strabismus may be treated with such an approach.  

MECHANICAL STRABISMUS FOLLOWING SURGICAL INSERTION OF OPHTHALMIC DEVICES  
Although in decreasing use in some areas, scleral buckles still remain in the therapeutic armamentarium of retinal detachment with retinal breaks. Transient postoperative strabismus and diplopia may occur in 50-60% of cases, but persistent symptoms remain in approximately 5-10% of cases. Mechanical etiologies are numerous, and include direct muscle damage from myotoxicity, surgical traction, necessity of muscle release and reattachment to insert the buckle, and motility restriction from the exoplant itself or adhesions between it and muscles or surrounding connective tissue. Involvement of the muscle pulley systems by insertion of any ophthalmic device is an increasingly recognized cause of postoperative strabismus as well, and, in part because of such changes, removal of a scleral buckle frequently has little effect on induced strabismus; although in some cases very early postoperative removal may be helpful, additional surgery is required to address the retinal detachment. Avoidance of strabismus may be enhanced by meticulous surgical technique, taking care to avoid encountering orbital fat, excessive muscle traction, and poorly placed buckles.  

Insertion of glaucoma drainage devices is another important cause of mechanical strabismus. Strabismus is possible regardless of the type or brand of glaucoma drainage device, with motility restriction from the device and/or underlying bleb and direct muscle damage being the most common causes. As with scleral buckles, adhesions can develop between the devices between muscles and surrounding tissue. Because many devices are implanted superotemporally, a typical strabismus pattern is ipsilateral exotropia and/or hypertropia. Although removal of the device seems to improve strabismus more than removal of a scleral buckle, results are unpredictable, and removal is often contraindicated due to the severity of the glaucoma. Treatment thus consists often of removal of scar tissue, prisms for smaller deviations, or surgery on yoke muscles of the fellow eye. In addition, use of the smallest plate possible is recommended during initial surgery to minimize risk of postoperative diplopia.  

Strabismus following episcleral plaque brachytherapy for uveal melanoma is also well documented in 1-2% of patients. The pattern of motility deficit varies with location of the plaque, and treatment involves a combination of prisms, surgery and chemodenervation.  

MECHANICAL STRABISMUS FOLLOWING OTHER OCULAR PROCEDURES  
Although seemingly a fairly straightforward procedure, strabismus following pterygium surgery is not uncommon. Significant scar tissue in the medial fornix may result in abduction deficits and an incomitant esotropia that is extremely difficult to treat. Alternatively, disinsertion of the medial rectus during intraoperative dissection produces a large exotropia with loss of adduction.  

Whether for functional or cosmetic indications, diplopia after blepharoplasty is well documented. Paretic strabismus is more common, thus, upper lid blepharoplasty may result in superior oblique paresis and lower lid surgery in paresis of the inferior oblique or inferior rectus. Although no large studies are reported, consensus is that the inferior oblique is likely the most commonly involved muscle, with blepharoplasty being the number one cause of isolated inferior oblique palsy. Less commonly, a Brown syndrome or inferior rectus fibrosis occur. Medial rectus restriction has also been reported. Most cases result from direct surgical trauma to the muscle, although injection of local anesthesia may contribute as well.
While pre-existent strabismus from thyroid orbitopathy, orbital masses, or inflammatory disease is often present in patients undergoing orbital decompression, the procedure itself may cause or worsen strabismus. The incidence of new-onset diplopia/strabismus seems to increase with the number of walls decompressed, ranging from 10% after single wall surgery to 20% with three. In addition, decompression surgery often alters vector forces of all muscles as well as globe position, further complicating the etiologic factors involved.

OTHER CONSIDERATIONS IN PREVENTION AND MANAGEMENT

As noted earlier, use of topical or general anesthesia for ocular surgery precludes post-operative strabismus from anesthetic myotoxicity. When peri- or retrobulbar anesthesia is needed, use of lidocaine over bupivacaine should be considered, as the latter is more toxic to the extraocular muscles. In all cases, careful surgical technique to minimize tissue disruption, muscle displacement, and postoperative scarring can aid in prevention, with avoidance of orbital fat and significant blepharoptosis paramount. An important principle in treatment is to avoid surgery until the deviation has stabilized. As noted earlier, the temporal profile of anesthetic myotoxicity evolves over weeks to months, and many cases of postoperative strabismus can improve or resolve spontaneously. For smaller deviations, prisms may be helpful, but for large or incomitant ones, monocular occlusion may be necessary. When surgical correction is indicated, judicious attempts to relieve restriction are indicated. Surgical treatment of anesthetic myotoxicity after otherwise successful cataract surgery is generally good, although removal of scleral buckles or other devices generally yields unimpressive results. Performing surgery on the uninvolved eye is often required to maximize alignment and improve incomitance. As in any case of vertical strabismus, careful assessment of the patient’s torsional status is required to ensure that the surgical approach to the vertical misalignment does not worsen the cyclodeviation. Surgical doses must be highly individualized, and standard formulas or tables are not applicable in these cases. Treatment of strabismus with bupivacaine and botulinum toxin may have an increasing role in these patients. However, the complicated nature and etiology of the motility disruption, as well as problems with sensory fusion in patients with loss of visual acuity or visual field, often contribute to residual diplopia.

CME ANSWERS

1. c. Inferior rectus, superior rectus, inferior oblique, lateral rectus
2. a. Anesthetic myotoxicity to the right inferior rectus
3. c. Direct surgical trauma during lower lid blepharoplasty

REFERENCES


