

REVIEW ARTICLE

Infantile-Onset Esotropia

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ABSTRACT

Infantile onset esotropia is a non-accommodative esotropia starting within the first six months of life. Different approaches have been taken to manage this entity of esotropia. The pathogenesis of infantile onset esotropia is still obscure and several theories have been submitted in order to understand this type of esotropia.

Key words: infantile, esotropia, recession, Faden.

Introduction

Definition

Infantile onset esotropia is a non-accommodative esotropia starting within the first six months of life [1] It is prevalent in approximately 0.1% of general population [2].

Terminology & variation of infantile-onset Esotropia [1]

- Congenital esotropia (Costenbader)
- Essential esotropia
- Infantile onset esotropia syndrome
- Ciancia type esotropia
- Nystagmus blockage syndrome

Why is infantile-onset esotropia a special entity?

Conventional recession is associated with frequent under correction while large recession has better results but followed by consecutive intermittent exotropia.

It is a primary over action of medial recti.

Pathogenesis of Infantile Esotropia

It is obscure. The infant has normal but immature sensorial visual system. The infant has excess esotropogenic forces. A normal functioning vergence mechanism is capable of overcoming these forces and any defect of this vergence

mechanism may lead to esotropia. At birth both motor & sensory systems are immature. At 2 months both of them begin to mature to sustain normal binocular vision. Motor fusion can be normal in the absence of sensory fusion but not the reverse. Any defect in motor fusion fails to help both eye globes to sustain a normal binocular vision that leads one of them to block the eye in adduction position to receive the second image on the less mature nasal retina [3].

Characteristics of infantile-onset esotropia

It is characterized by large angle esotropia between 40-50 Δ and variable angles with or without convergence excess. The infant may have cross fixation and apparent limited abduction. The infant has low to moderate hyperopia. It is also characterized by adduction over-shooting [1].

Ocular alignment of the newborn

It is characterized by orthotropic to exotropic. It is rarely esotropic. Esotropia isn't present at birth except 6th nerve palsy or Duane syndrome. The ocular alignment is unstable until 2 months and any deviation after 2 months is abnormal [2].

Management of Infantile Onset Esotropia

Conventional treatment

- Large or augmented recession

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Non-conventional treatment

- Botulinum toxin injection of both medial recti
- Faden operation
- Recess resect of medial recti
- Y-splitting of both medial recti
- Pulley posterior fixation
- Slanting or differential recession

The idea of surgical treatment is how to decrease the contractility without affecting the tone of the muscle.



Figure 1: Pseudo paralytic 6th nerve palsy of infantile onset esotropia.

Large or augmented recession

Large recession 6-8 mm recession, which is behind the equator, decreases both contractility and muscle tone. Loss of the normal tone of both medial recti which is essential for maintaining the eye position against exotropogenic forces of the orbit and the passive forces and therefore leading to high incidence of consecutive intermittent exotropia (20%-26%).

But this procedure is easy and fast to do [4].

An effective pharmacological alternative to the surgical management of infantile onset esotropia.

Botulinum toxin

Bilateral simultaneous injection of botulinum toxin into the medial rectus muscle under direct visualization with an “open sky” technique 2.5-5.0 IU. The aim of botulinum toxin treatment is to temporarily weaken the inner muscles of both eyes causing the eyes to drift outwards. The child rediscovers their 3D vision and uses this to help keep their eyes in a straight position and no further treatment is needed. The child’s eyes continue to drift inwards, but not as far as they had done before botulinum toxin treatment. The child’s eyes return to their original in position.

In the second and third scenario further botulinum toxin treatment could be considered. Average 2-botulinum toxin treatments are required to successfully reduce the angle of the squint [5].

Faden or posterior scleral fixation

This procedure was founded by Cuppers and was named as “the so called thread operation” and was then modified by de ‘Decker to treat infantile onset esotropia. The

main principle is to weaken the contractility only in the direction of action but not against and does not affect the tone [6]. Dissection of the medial rectus up to 15 mm and three continuous non absorbable suture including both 1/4th of the upper and lower fibers of the muscle were taken as shown in Figure 2.

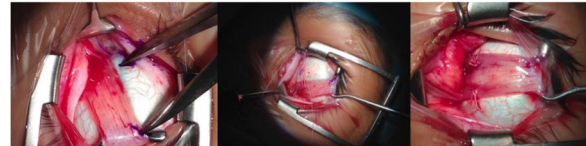


Figure 2: Posterior fixation sutures (Faden).

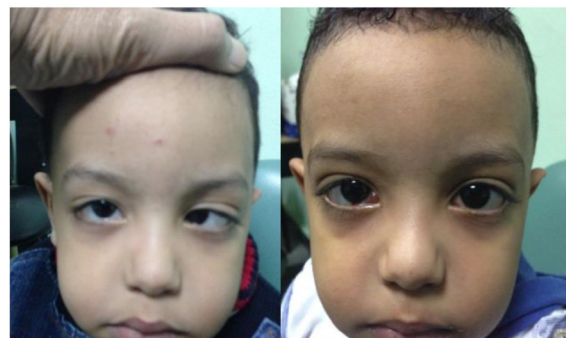


Figure 3: Pre-operative and postoperative of Posterior fixation sutures.

Mechanism of Faden

$$T = F * r$$

T = torque r = lever arm F = force

It gives 80%-86% success, 11%-17% residual esotropia and 3% consecutive intermittent exotropia but it is difficult to approach with risk of scleral perforation. It is also technique dependable [6].

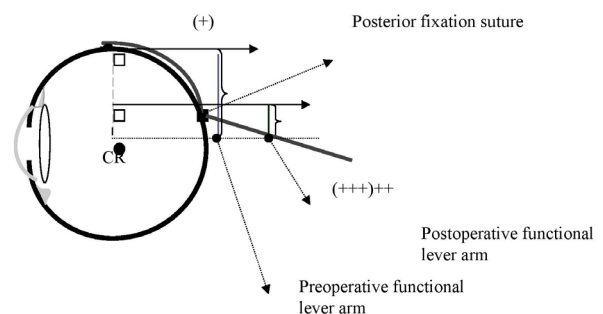


Figure 4: Mechanism of Posterior fixation sutures.

Recess resect of both medial recti

Both medial recti are resected 5.0 mm to compensate for lost tone and recessed 7.0 mm to decrease the torque as shown in Figure 5. This technique gets 57%-60% success, 18%-20% residual and 20%-23% consecutive exotropia [7].



Figure 5: Recess resect of medial rectus.



Figure 6: Preoperative and postoperative of recess-resect of medial rectus.

Y-splitting of both medial recti

Both medial recti are dissected 15.0 mm from insertion and divided into equal halves then a mark was taken 2.5 mm from the midpoint of insertion and from this point two marks 7.5 were taken so the two halves are separated by 15.0 mm. Both parts slide to side decreasing lever arm. So, triangle of equal limbs is formed 2.5 mm from insertion keeping normal muscle tone (Figure 7).



Figure 7: Y splitting of medial rectus. Preoperative and postoperative of Y splitting.

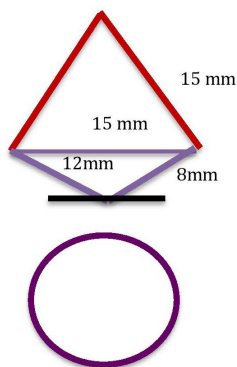


Figure 8: Mechanism of Y splitting.

Pulley posterior fixation

Dissection of the medial rectus is needed to the pulley sleeve, which is approximately 10 mm away, and non-absorbable suture is taken to the sleeve on each side of the muscle associated with medial rectus recession 3-5 mm (Figure 9). The overall success rate is 70%, 20%

are under corrected and 10% patients are overcorrected. Pulley fixation is a technically more difficult procedure that needs excessive MR dissection, which may lead to injury of the orbital fat, if not done by an experienced surgeon, but has a fast learning curve, the surgery is time-consuming. It avoids the additional surgical risks of scleral posterior fixation as Faden procedure [9].

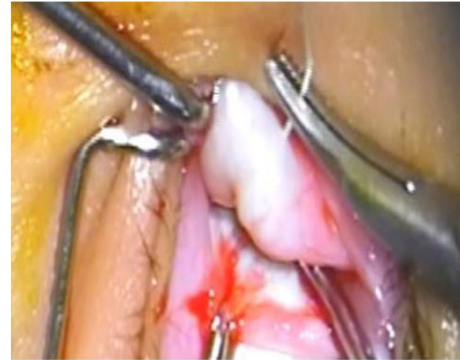


Figure 9: Pulley posterior fixation.



Figure 10: Preoperative and postoperative of pulley posterior fixation.

Differential/slanted recession of both medial recti

The upper half is recessed 4-5 mm while the lower half is recessed 7-8 mm. The two halves were separated after cutting the muscle into 2 parts to prevent re-unifying of both halves (Figure 11). It depends on selective action of both halves. The upper half decreases the tone while the lower half decreases the torque. This procedure can decrease both the tone and torque and decreases the incidence of intermittent exotropia. It is efficient in correction of infantile esotropia with or without convergence excess. It shows overall success 86.6%, 10% residual esotropia, while 3.4% shows consecutive exotropia [10].

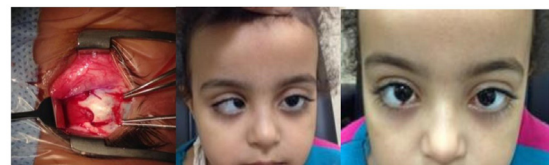


Figure 11: Slanting recession and its results.

CONCLUSION

The infantile-onset esotropia is a special entity and different approaches that decrease the muscle torque give satisfactory results.

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