Review Article

The Overaction of the Inferior Oblique Muscle and Associated Vertical Strabismus: Prevalence, Etiology, and Diagnosis

Masoud Khorrami-Nejad[1,2], Huda Karim[2], Wisam Hatem[2], Mustafa Abdullah[2], Ali Gheibi[3], Mohammad Ghasemi Boromand[4]

[1] Translational Ophthalmology Research Center, Farabi Eye Hospital, Tehran University of Medical Sciences, Tehran, Iran.
[2] Optometry department, School of Rehabilitation, Tehran University of Medical Sciences, Tehran, Iran.
[3] Student Research Committee. Department of optometry, Faculty of Rehabilitation, Shahid Beheshti University of Medical Sciences. Tehran. Iran.
[4] Department of Optometry, School of Rehabilitation, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

Corresponding author: Ali Gheibi, PhD**.**
Student Research Committee. Department of optometry, Faculty of Rehabilitation, Shahid Beheshti University of Medical Sciences. Tehran. Iran
Tel: (+98) 21-77684579, Email: aligheibi79@yahoo.com

ORCID ID:
Masoud Khorrami-Nejad: 0000-0002-8270-9704
Huda Karim: 0009-0007-3652-8746
Wisam Hatem: 0009-0006-5826-8538
Mustafa Abdullah: 0000-0003-1012-9425
Ali Gheibi: 0000-0002-9588-7702
Mohammad Ghasemi Boromand: 0000-0003-2372-3861

Article Info:
Received: 3 May 2023
Accepted: 28 Nov 2023

Citation: Khorrami-Nejad M, Karim H, Hatem W, Abdullah M, Gheibi A, Ghasemi Boromand M. The Overaction of the Inferior Oblique Muscle and Associated Vertical Strabismus: Prevalence, Etiology, and Diagnosis. Journal of Modern Rehabilitation. 2024; 19(1):?-?

Running title: The overaction of the inferior oblique muscle

Abstract:
Vertical strabismus, often caused by the overaction of the inferior oblique (IO) muscle, is a common disorder of the oculomotor system. It occurs in a third of all patients with strabismus, and in 70% of these cases, it coexists with esotropia. From a clinical perspective, there is no universally accepted approach for quantifying the magnitude of inferior oblique overaction (IOOA). Existing classifications are subjective and may not be entirely appropriate. Therefore, given the need to understand the severity of this anomaly, standardizing the classification of this disorder is essential.

This will aid in formulating treatment strategies and predicting the outcomes of surgical intervention. The IO muscles' mechanism of action is highly complex, and it varies depending on the position of the globe during IO contraction. Beyond their role in horizontal and vertical movements, IO muscles also participate in torsional eye movements. Any disorder of the IO muscle can contribute to not only vertical strabismus but also cyclotropia, which may limit the effectiveness of strabismus treatment. To identify all the clinical signs and symptoms resulting from IOOA, practitioners should take into account the triple action characteristics of the IO muscle. Therefore, comprehensive diagnostic examinations are necessary for patients with IOOA.
Keywords: vertical strabismus, inferior oblique overaction, strabismus, overelevation.

Introduction

Strabismus is defined as a binocular vision disorder, where one eye deviates from the normal fixation point. The prevalence of strabismus ranges from 0.5 to 5%.(1) Strabismus is more than a cosmetic defect, it is associated with impaired binocular vision and stereopsis. It can negatively impact on the patient's self-esteem and interpersonal relationships.(2)

Deviation of the eyeball upward in the adduction position is a consequence of the overaction of the inferior oblique muscle.(3) It has been reported in 70% of patients with esotropia and in 30% of patients with strabismus.(4, 5) This anomaly can be either unilateral or bilateral. In addition, the inferior oblique overaction (IOOA) can be categorized into primary and secondary deviation.(6) rimary IOOA is the most common in children. The prevalence of this form of vertical strabismus is around 72% in cases of infantile esotropia and 30% in cases of acquired strabismus.(7) Among all variations of vertical strabismus, IOOA is observed in 16.7% of cases.(7)

Methods

In the current study, Google Scholar, PubMed and Science Direct search engines were used for searching the articles using the keywords: vertical strabismus, inferior oblique overaction, strabismus and overelevation. Papers published between 1965 and March 2023 that were relevant to the topic of this review were included. The selected articles and scientific evidence were collected, summarized, categorized, evaluated, and concluded upon.

Discussion

The etiology of primary IOOA is still debated. However, Duane suggested that excessive innervation of vergences can lead to the development of strabismus.(8) According to his theory, over-elevation of the eye in adduction occurs due to an increased innervation of the adductive eye by the inferior oblique (IO) muscle compared to the contralateral superior rectus muscle. In contrast, Lisch and Simonsz hypothesized that ocular over-elevation in adduction could be observed in previously healthy eyes after prolonged monocular occlusion.(9) This may indicate that there is a natural tendency for the eye to lift on adduction, but under normal binocular conditions, these kinds of ocular movements are controlled by fusion.(10, 11) F.B. Chavasse considered it reasonable to compare the features of the superior oblique (SO) muscle and the IO muscle. He proposed that the releasing effect of the SO in adduction is less than the lifting effect of the IO, which is explained by their anatomical features.(12) In support of this theory, Berens noted that the IO as an elevator expends 42% of muscle energy, while the SO as a depressor spends only 37%.(13) In 1952, Scobee also supported the theory that IOOA is caused by muscle imbalance.(14) He believed that to take the globe away from the nose, an increased impulse is applied to the IO of the adducted eye, and an impulse with the same strength directs to its synergist,

i.e., the ipsilateral superior rectus muscle. The author also stated that the elevator action of the IO in adduction is greater than the depressor effect of the SO. As a result of the upcoming imbalance, over-elevation of the adducted eye develops. Lancaster also agreed with this point of view.(15) The overaction of the IO can be explained by the oblique location of the insertion of the medial rectus muscle or by excessive adhesion between the IO and the inferior or lateral rectus muscles. Guibor suggested that the excessive lifting effect of the IO muscle may be due to its synkinesis with the ipsilateral medial rectus muscle as a result of an impulse distribution in the central nervous system.(16) The secondary overaction of the IO muscle is more straightforward to explain. Its occurrence is caused by paresis or paralysis of the ipsilateral SO, or paresis or paralysis of the contralateral superior rectus muscle when the paretic eye is fixating. In the latter case, overelevation of the globe in adduction is caused by increased innervation transferring to the IO in accordance with Hering's law. However, in the first condition, overelevation in adduction is not caused by excessive innervation of the IO, but it is due to the lack of the tonus of the paralyzed SO antagonist, even a normal innervation impulse will be sufficient to overelevate the globe at the time of IO action.(17) Verhoeff believed that IOOA is always associated with paresis of the SO or hypoplasia of the nucleus n. trochlearis.(18) Thus, there are two main theories regarding the development of the secondary IOOA. White and Brown believed that weakness of the superior rectus muscle leads to the development of secondary IOOA 28 times more often than the lesion of the SO.(19) Bielschowsky and his coworkers, on the contrary, believed that SO paresis is observed more often in secondary IOOA.(20) Adler noted that part of the disagreement might be due to the fact that some authors consider only primary paresis,(21) while others argued that paresis associated with horizontal strabismus.(22) Adler also found that isolated primary vertical palsy studied by Davis was more often caused by SO paresis. However, the vast majority of vertical paralysis associated with horizontal strabismus studied by White and Brown were caused by weakness of the superior rectus muscle (19, 21).

Classification of vertical strabismus due to inferior oblique overaction:

The clinically quantitative classification of the IOOA is based on the assessment of the elevation effect of this muscle.(23, 24) According to the degree of IOOA, the most widely used classification ranges from 0 to +4, corresponding to the status of moving the globe at an angle of 45 degrees. Grade 0 corresponds to normal muscle function with no overelevation in the adductive state. The degree of +1 indicates the minimum vertical deviation of the globe in adduction, while +4 corresponds to the maximum elevation of the adducted eye. The degrees +2 and +3 are intermediate between these two extremes.(24) Based on this classification, Popova et al. proposed a grading system of the IOOA according to degrees A, B, C, D. In this case, the degree of muscle overaction was determined by the degree of deviation of the visual axis of the deviated eye from the horizontal line in the state of adduction of the globe. At the same time, degree D is diagnosed when in the depression of the globe; its visual axis forms a right angle (90 degrees) with the horizontal axis. Degree B corresponds to an angle of 45 degrees. For intermediate values of the vertical deviation of the eye in adduction, the degrees A or C are assigned.(25) However, such an assessment of the IOOA is subjective, especially in cases of intermediate degrees. Accordingly, most authors classified IO dysfunction in degrees according to the Hirschberg method or in prism diopters (PD). Alazme determined the degree of IOOA by the magnitude of the upward deviation of the eye during adduction in degrees, according to Hirschberg. With overaction of the first degree, the deviation of the eye is from 5 to 10°; the second degree corresponds to the interval from 15 to 20 °; the third degree from 25 to 30 °; and the fourth degree from 35 to 40°.(26) Given

that in the state of adduction at an angle of 45°, the globe is able to rise within 30°, other correspondences equal to 0-7°, 8-15°, 16 -22°, 23-30°.(27) In 2006, Moon and Lee used not only degrees from +1 to +4 but also the corresponding values of hypertropia in PD (0–5, 6–10, 11–15, more than 15 PD) to assess the overaction value of the IO muscle, respectively.(28) According to the classification proposed by Kelkar et al. in 2015, grades 1+, 2+, 3+, and 4+ of IOOA approximately correspond to 1-15, 16-30, 31-45, and 46-60 PD hypertropia during globe adduction.(29) Some authors converted the degree of IOOA from 1+, 2+, 3+ and 4+ into 5, 10, 15 and 20 PD hypertropia when fixing into the distance, respectively.

To date, there is no single approach for defining IOOA by degrees, and the most common classifications are highly subjective and inappropriate. There is no unified classification of IOOA in the position of the adduction when we intend to determine the angle of deviation by the Hirshberg method. Due to the need to understand the severity of the disease for selecting the treatment strategies and predicting the results of surgical intervention, it is necessary to carry out standardization in the classification of the IOOA.

Examination methods of vertical strabismus caused by inferior oblique overaction:

The anatomy of the IO is somewhat atypical compared to other extraocular muscles. The IO originates at the lower inner edge of the orbit (in the area of the entrance of the lacrimal canal) and is attached to the posterolateral part of the globe near the lower border of the lateral rectus muscle, passing between the wall of the orbit and the inferior rectus muscle 16 mm from the limbus.

The IO is the shortest extraocular muscle, with a length of only 37 mm. The width of the attachment site varies widely, ranging from 5 to 14 mm, on average about 9 mm. The attachment site forms a concave line. The anterior point of attachment is 10 mm from the attachment of the external rectus muscle; its posterior point is 1 mm lower and 1 to 2 mm lateral to the macula. Unlike other extraocular muscles, which have both muscular and tendon components, the IO is almost entirely muscular, with an average tendon length of 1 to 2 mm.(30)

The mechanism of action of extraocular muscles is very complex and depends on the position of the globe at the time of their contraction. Clinically, the main (primary) action of the muscle is evaluated, while the primary position of the eye, and minor actions (secondary and tertiary), depend on the position of the globe. The primary action of IO is excycloduction. Moreover, elevation and abduction of the eye are minor actions for IO. Any impairment of the IO muscle leads to the development of cyclo-vertical strabismus, which is also the cause of the formation of torsion diplopia, and torticollis, which reduces the effectiveness of strabismus treatment. In general, IOOA leads to the development of the V-pattern strabismus.(31) To identify the entire symptom complex associated with IOOA, practitioners should consider the complexity of its triple action. From the clinical perspective, it is necessary to conduct a comprehensive diagnostic study. Simultaneously, to identify the IOOA, the displacement of the globe in nine gaze positions is assessed.(32)

An accurate differential diagnosis for the target muscle in cases of IOOA is critical. In particular, with the development of endocrine myopathy, frequent involvement of the inferior rectus muscles in the inflammatory process can also affect the IO muscle. This interaction may be related to the inferior anatomy of the orbit, as the inferior rectus and the IO are the only muscles in direct contact with each other. As a result, any inflammatory process will result in fibrosis of these two muscles and Lockwood's ligament. However, the treatment of endocrine ophthalmopathy is a complex process in which surgical treatment of strabismus is performed only in cases of failure of pathologically oriented therapy.(33)

Measurement of the magnitude of overaction of the inferior oblique muscle:
The Hirschberg method, based on the observation of the position of the light reflex of the ophthalmoscope on the patient's cornea, is widely used to assess the deviation of the eyes.(17) The method was proposed by Hirschberg in 1886. While holding a candle 12 inches from the patient, he observed the position of the reflex on the cornea of the deviant eye. He expressed the results of his observations in degrees. Hirschberg found that 1 mm decentration of the corneal reflex corresponds to 7° deviation relative to the visual axis of the examined eye.(34) At present, the angle of deviation is measured through the alternate cover test, with the occlusion of each eye separately while fixing an object located at a distance of 33 cm. Deviation and ocular motility are assessed in nine gaze positions.(35) Another less commonly objective method for determining the magnitude of IOOA is the method of achieving the absence of adjusting eye movements during the cover test and simultaneous deviation correction with the prism. In this case, the strabismus angle is corrected using loose prisms from the trial lens, Risley prisms, plastic Fresnel prisms, or prism bars. The study is carried out as long as eye movements are observed, and the angle of deviation is considered equal to the strength of the prism necessary to neutralize it. The magnitude of hypertropia, in this case, is expressed in PD. For the convenience of using various methods for studying the magnitude of the deviation between PD and Hirschberg degrees, the following relationship was found: 1° = 2 PD.(31, 35)

V-pattern syndrome:
IOOA is often associated with V-pattern syndrome, regardless of whether there is a deviation in the primary gaze position or IOOA is combined with eso- or exotropia.(36) To diagnose V-patterns syndrome, when examining a patient, it is necessary to identify the difference in the magnitude of the horizontal deviation when changing the gaze from the upward toward the downward position when looking at an object at a distance of 33 cm. In cases where the deviation angle after changing the fixation decreases by more than 15 PD, or 10° indicates the presence of V-pattern syndrome.(37)

Head tilt test:
The head tilt test (Bielschowsky test) was first described by the German ophthalmologist Alfred Bielschowsky in 1914. The test detects changes in vertical deviation when the head is tilted to the right or left shoulder in paralysis of the vertical rectus and oblique extraocular muscles.(38) The author gave a physiological justification for the method. When the head is tilted towards the shoulder, endolymph movement occurs in the otolithic apparatus (semicircular canals of the labyrinth), as a result of which an impulse to contract is given to the corresponding groups of extraocular muscles.(17) Given the condition of normal muscle response, this mechanism allows the individuals to maintain the correct position of the eyes without disturbing binocularity.
In cases of IOOA, the Bielschowsky test allows differentiating between primary and secondary overaction, in which this phenomenon is considered positive and consists of the appearance and/or increase in hypertropia when the head is tilted towards the affected eye.(39, 40)

Compensatory head position:
Forced tilting of the head in the form of ocular torticollis is characteristic of most patients with paretic, paralytic strabismus. The mechanism of its occurrence lies in the fact that when the gaze is shifted towards the action of the affected muscle, diplopia increases, and when tilting the head,

it becomes possible to avoid the field of action of the paretic muscle.(41) This avoids double vision and often preserves binocular vision.
In cases where fusion was not provided by torticollis, patients turn or tilt their heads to increase the distance between “double” images or use their nose as an occluder. It is essential to differentiate between true torticollis and ocular torticollis. True torticollis is caused by pathology of the cervical vertebrae or fibrosis of the sternocleidomastoid muscle.(42) However, treatments specific to torticollis cannot correct ocular torticollis.
Secondary IOOA caused by paresis or paralysis of the SO is characterized by a tilt of the head to the opposite shoulder with the chin-down.(43-45) It should be borne in mind that the degree of "correction" of the position of the eyes by tilting the head is much less than the degree of tilting the head. Long-term ocular torticollis contributes to the formation of facial asymmetry, which is an important clinical sign for differentiating congenital and acquired IOOA.(46-49)

Evaluation of cyclo-deviation:
In addition to horizontal and vertical movements, IO muscles provide excyclotorsional eye movements.(50) The occurrence of IOOA leads to the development of excyclodeviation. Patients with congenital or long-term IOOA have no complaints of torsional diplopia. This is due to adaptive compensatory mechanisms, which include ocular torticollis, compensation with cyclo-fusion reserves, and physiological suppression of one of the two monocular images. The value of physiological excycloduction does not exceed 12°, and its large values are excessive and confirm the presence of muscle overaction.(51)
The presence of cyclotropia, when its magnitude exceeds the cyclofusion reserves, can prevent the development of fusion and stereopsis and compromise the functional results of strabismus treatment.(52) Therefore, along with a standard ophthalmological examination, the evaluation of cyclotorsion movements is of fundamental importance in the diagnosis of IOOA.
Cyclotropia assessment can be qualitative or quantitative. There are several ways to determine the presence of cyclotorsion displacement of the globe qualitatively.(53-55)

1. During the eye movement examinations or performing a cover test: the presence of intorsion or extorsion is determined by observing the displacement of a large conjunctival vessel at the limbus.

2. During ophthalmoscopy, observing the level of the optic nerve head (ONH) in relation to the macula: Anatomically, the macula lies 4 mm temporal to the ONH and 0.8 mm below the horizontal line. Thus, if the ONH is obviously above or below the macula, we can conclude that there is a cyclotorsion displacement (displacement of the ONH above or below the fovea indicates the presence of excyclodeviation or incyclodeviation, respectively).

3. When using double Maddox's rod test.

4. Photographing the fundus: it can determine the relationship between the optic disc and the fovea.
Quantification of the cyclodeviation is necessary to confirm the diagnosis and evaluate the effectiveness of surgical intervention for disorders of the IO muscle. The subjective and objective assessments determine not only the position but also the degree of displacement of the macula relative to the optic disc. A subjective quantitative assessment can be made on a scale of a trial lens using a Maddox rod. The rotation of the perceived image represents cyclo-deviation and can be compensated by rotation of the lens until the patient perceives the alignment of double images.

However, this technique is difficult to implement in pediatrics. The most reliable methods for determining cyclodeviation in children are those that do not require a patient response. These methods include the method of photographic recordings of images of markers in the fundus of both eyes during examination using a fundus camera, which allows for determining the position of the macula.(56)

Conclusion:
Any disorders of the IO muscle contribute to developing not only vertical strabismus but also cyclotropia, which may limit the efficacy of strabismus treatment. During the evaluation of patients with IOOA, it is necessary to conduct a complete diagnostic examination in addition to an assessment of torsional eye movements. In childhood, some diagnostic techniques are impossible since they require a conscious response from the patient. This limitation is the main reason for the necessity of performing a complete diagnostic evaluation of the IOOA. It is necessary to develop new diagnostic procedures to overcome clinical shortcomings. To identify all the clinical signs and symptoms that result from IOOA, practitioners should consider the characteristics of its triple action. Therefore, it is necessary to conduct comprehensive diagnostic examinations on patients with IOOA.

Funding:
There was no funding support.

Conflict of interest:
The authors declare no conflict of interest.

Acknowledgements:
Not applicable.

Authors contributions:
All authors equally contributed to preparing this article.

References:
1. Hashemi H, Pakzad R, Heydarian S, Yekta A, Aghamirsalim M, Shokrollahzadeh F, et al. Global and regional prevalence of strabismus: a comprehensive systematic review and meta-analysis. Strabismus. 2019;27(2):54-65.
2. Nelson BA, Gunton KB, Lasker JN, Nelson LB, Drohan LA. The psychosocial aspects of strabismus in teenagers and adults and the impact of surgical correction. Journal of American Association for Pediatric Ophthalmology and Strabismus. 2008;12(1):72-6. e1.
3. Choi DG, Chang BL. Electron microscopic study on overacting inferior oblique muscles. Korean Journal of Ophthalmology. 1992;6(2):69-75.
4. Caldeira J. Some clinical characteristics of V-pattern exotropia and surgical outcome after bilateral recession of the inferior oblique muscle: a retrospective study of 22 consecutive patients and a comparison with V-pattern esotropia. Binocular vision & strabismus quarterly. 2004;19(3):139-50.

5. Ha S-G, Na G-H, Kim S-H. A valid indication and the effect of bilateral inferior oblique transposition on recurrent or consecutive horizontal deviation in infantile strabismus. Korean Journal of Ophthalmology. 2017;31(2):138-42.
6. Taylan Sekeroglu H, Dikmetas O, Sanac AS, Sener EC, Arslan U. Inferior oblique muscle weakening: is it possible to quantify its effects on horizontal deviations? Journal of ophthalmology. 2012;2012.
7. Wilson ME, Parks MM. Primary inferior oblique overaction in congenital esotropia, accommodative esotropia, and intermittent exotropia. Ophthalmology. 1989;96(7):950-7.
8. Duane A. Binocular movements. Archives of Ophthalmology. 1933;9(4):579-607.
9. Liesch A, Simonsz H. Up-and downshoot in adduction after monocular patching in normal volunteers. Strabismus. 1993;1(1):25-36.
10. Stager Jr D, Dao LM, Felius J. Uses of the inferior oblique muscle in strabismus surgery. Middle East African journal of ophthalmology. 2015;22(3):292.
11. Ludwig IH, Clark RA, Stager Sr DR. New strabismus surgical techniques. Journal of American Association for Pediatric Ophthalmology and Strabismus. 2013;17(1):79-88.
12. Worth CA, Chavasse FB. Worth's Squint, Or, The Binocular Reflexes and the Treatment of Strabismus: Baillière, Tindall and Cox; 1939.
13. Berens C. The Eye and Its Diseases by 82 International Authorities: WB Saunders; 1936.
14. Scobee RG. The oculorotary muscles: Mosby St. Louis; 1952.
15. Lancaster WB, editor Factors underestimated, features overemphasized, and comments on classification. Strabismus Ophthalmic Symposium; 1950: CV Mosby, St. Louis.
16. Guibor GP. Synkinetic overaction of the inferior oblique muscle. American Journal of Ophthalmology. 1949;32(2):221-9.
17. Von Noorden GK. Binocular vision and ocular motility. Theory and management of strabismus. 1990.
18. Verhoeff F. Occlusion hypertropia. Archives of Ophthalmology. 1941;25(5):780-95.
19. White JW, Brown HW. Occurrence of vertical anomalies associated with convergent and divergent anomalies: a clinical study. Archives of Ophthalmology. 1939;21(6):999-1009.
20. Edward J. Bielschowsky's Lectures on Motor Anomalies. American Journal of Ophthalmology. 1940;23(8):934-5.
21. DAVIS WT. Paresis of right superior oblique and of left superior rectus muscle: Differential diagnosis. Archives of Ophthalmology. 1944;32(5):372-80.
22. ADLER FH. Physiologic factors in differential diagnosis of paralysis of superior rectus and superior oblique muscles. Archives of Ophthalmology. 1946;36(6):661-73.
23. Avetisov E, Alazme A, Kashchenko T, Smol'ianinova I, Mats K, Lokhtina N. Surgical treatment of strabismus with superior oblique muscle insufficiency. Vestnik Oftalmologii. 1994;110(4):12-4.

24. Sanjari MS, Shahraki K, Nekoozadeh S, Tabatabaee S-M, Shahraki K, Aghdam KA. Surgical treatments in inferior oblique muscle overaction. Journal of ophthalmic & vision research. 2014;9(3):291.
25. Popova N, Sorokina V, Gorkin A, Yugay S. Method of dosed anterior transposition of the lower oblique muscle in vertical strabismus surgery. Fyodorov Journal of Ophthalmic Surgery. 2012;1:30-4.
26. Alazme A. ANATOMIC AND TOPOGRAPHIC FEATURES OF OBLIQUE MUSCLES AND SURGICAL INTERVENTIONS ON THESE MUSCLES. VESTNIK OFTALMOLOGII. 1991(2):70-6.
27. Min B-M, Park J-H, Kim S-Y, Lee S-B. Comparison of inferior oblique muscle weakening by anterior transposition or myectomy: a prospective study of 20 cases. British journal of ophthalmology. 1999;83(2):206-8.
28. Moon K, Lee S-Y. The effect of graded recession and anteriorization on unilateral superior oblique palsy. Korean Journal of Ophthalmology. 2006;20(3):188-91.
29. Kelkar J, Kanade A, Agashe S, Kelkar A, Khandekar R. Outcomes of asymmetric primary inferior oblique muscle overaction managed by bilateral myectomy and tucking of proximal muscle end: A cohort study. Middle East African Journal of Ophthalmology. 2015;22(4):457.
30. Porter JD, Baker RS, Ragusa RJ, Brueckner JK. Extraocular muscles: basic and clinical aspects of structure and function. Survey of ophthalmology. 1995;39(6):451-84.
31. Caldeira J. V-pattern esotropia: a review; and a study of the outcome after bilateral recession of the inferior oblique muscle: a retrospective study of 78 consecutive patients. Binocular vision & strabismus quarterly. 2003;18(1):35-48; discussion 9.
32. Rutstein RP, Daum KM. Anomalies of binocular vision: diagnosis & management: Mosby Incorporated; 1998.
33. Rødahl E, Seland J, Olofsson J, Aanderud S, Kråkenes J. Surgical treatment of endocrine ophthalmopathy. Tidsskrift for den Norske Laegeforening: Tidsskrift for Praktisk Medicin, ny Raekke. 1999;119(12):1737-42.
34. Brodie SE. Photographic calibration of the Hirschberg test. Investigative ophthalmology & visual science. 1987;28(4):736-42.
35. ESKRIDGE BJ, Wick B, Perrigin D. The Hirschberg test: a double-masked clinical evaluation. Optometry and Vision Science. 1988;65(9):745-50.
36. Nowakowska O, Broniarczyk-Loba A, Loba P. The reduction of AV patterns with oblique muscles overaction in unilateral and bilateral surgery. Klinika Oczna/Acta Ophthalmologica Polonica. 2008;110(4):361-3.
37. LANG J. The meaning of A and V pattern syndromes. International Ophthalmology Clinics. 1971;11(4):70-3.
38. Von Noorden GK, Murray E, Wong SY. Superior oblique paralysis: a review of 270 cases. Archives of Ophthalmology. 1986;104(12):1771-6.
39. Manchandia AM, Demer JL. Sensitivity of the three-step test in diagnosis of superior oblique palsy. Journal of American Association for Pediatric Ophthalmology and Strabismus. 2014;18(6):567-71.
40. Oh H, Roh Y. Head tilting in the patients with superior oblique palsy. J Korean Ophthalmol Soc. 1994;35(35):454-9.

41. Rubin SE, Wagner RS. Ocular torticollis. Survey of ophthalmology. 1986;30(6):366-76.
42. Boricean I-D, Bărar A. Understanding ocular torticollis in children. Oftalmologia (Bucharest, Romania: 1990). 2011;55(1):10-26.
43. Adler FH. Adler S Physiology of the Eye: Clinical Application 5th Ed: Mosby; 1970.
44. Akbari MR, Khorrami-Nejad M, Kangari H, Baghban AA, Pazouki MR. Ocular abnormal head posture: A literature review. Journal of current ophthalmology. 2021;33(4):379.
45. Khorrami-Nejad M, Akbari MR, Kangari H, Akbarzadeh Baghban A, Masoomian B, Ranjbar-Pazooki M. Abnormal head posture in unilateral superior oblique palsy. Journal of binocular vision and ocular motility. 2021;71(1):16-23.
46. Dietrich D, Slack W. Scoliosis secondary to unilateral extraocular muscle paresis (ocular torticollis). Radiology. 1967;88(3):538-42.
47. Roussat B, Bourreau M, Hayot B, Besson D, Goddé-Jolly D. Benign isolated 6th nerve paralysis in children. Developmental aspects apropos of 9 new cases. Ophtalmologie: Organe de la Societe Francaise D'ophtalmologie. 1989;3(2):107-8.
48. Akbari MR, Khorrami-Nejad M, Amiri MA, Baghban AA, Khosravi B, Nourizad S. Facial asymmetry in unilateral duane retraction syndrome. Optometry and Vision Science. 2019;96(12):920-4.
49. Akbari MR, Khorrami-Nejad M, Kangari H, Baghban AA, Pazouki MR. Facial Asymmetry in Unilateral Congenital Superior Oblique Muscle Palsy. Optometry and Vision Science. 2021;98(11):1248-54.
50. PHILLIPS PH, HUNTER DG. EVALUATION OF OCULAR TORSION AND PRINCIPLES. Clinical strabismus management: Principles and surgical techniques. 1999:52.
51. Parks MM. Isolated cyclovertical muscle palsy. AMA archives of ophthalmology. 1958;60(6):1027-35.
52. Sharma P, Prasad K, Khokhar S. Cyclofusion in normal and superior oblique palsy subjects. SLACK Incorporated Thorofare, NJ; 1999. p. 264-70.
53. Bixenman WW, Von Noorden GK. Apparent foveal displacement in normal subjects and in cyclotropia. Ophthalmology. 1982;89(1):58-62.
54. Harada M. Surgical correction of cyclotropia. Jpn J Ophthalmol. 1964;8:88-96.
55. Troia RN, Nelson LB, Calhoun JH, Harley RD. Surgical correction of excyclotropia. American Orthoptic Journal. 1985;35(1):63-7.
56. Morton GV, Lucchese N, Kushner BJ. The role of funduscopy and fundus photography in strabismus diagnosis. Ophthalmology. 1983;90(10):1186-91.