JOURNAL OF INTERNATIONAL CRISIS AND RISK COMMUNICATION RESEARCH
ISSN: 2576-0017
2024, VOL 7, NO S9

A Comprehensive Review of Strabismus: Diagnosis, Types, and Management Strategies

Abdullah Shaker Algheshayan [1], Saleh Ali G Alribi [2], Ashwaq Abdullah Alghamdi [3], Osman Abdulaziz Alarifi [4], Ashjan Abdulaziz Khotani [5], Nourah Mohammed Ali [6], Maha Ali Alghamdi [7], Ebtehaj Saeed AlGhamdi [8], Fahdah Alrowaily [9], Zaied Jzaa Almutairi [10], Eman Rasheed Mubarak Alotaibi [11], Ansam Mohamed Bin Nooh [12].

1. *Optometrist, King Salman Hospital, Ministry of Health, Kingdom of Saudi Arabia. A.gheshayan@gmail.com*
2. *Senior optometry Specialist, King Salman Hospital, , Ministry of Health, Kingdom of Saudi Arabia. sa85gr@gmail.com*
3. *General physician, King Salman Hospital, Ministry of Health, Kingdom of Saudi Arabia. Ashwaqghamdiii@gmail.com*
4. *General physicians, King Salman Hospital, Ministry of Health, Kingdom of Saudi Arabia. A.39man@gmail.com*
5. *Ophthalmology Service Resident, King Salman Hospital, Ministry of Health, Kingdom of Saudi Arabia. Dr.ashjan.alkhotani@gmail.com*
6. *Optometrist, Aqiq General Hospital Albaha, Ministry of Health, Kingdom of Saudi Arabia. noorah919@hotmail.com*
7. *Optometrist, Aqiq General Hospital Albaha, Ministry of Health, Kingdom of Saudi Arabia. Dr.maha0@icloud.com*
8. *Optometrist, Ara PHCC Albaha, Ministry of Health, Kingdom of Saudi Arabia. Ebtehaja@moh.gov.sa*
9. *optometrist, Hospital Management, Hospital Administrator, King Saud Medical City, Ministry of Health, Kingdom Of Saudi Arabia. fofana99@gmail.com*
10. *Optometry Specialist, Ministry of Health, Kingdom Of Saudi Arabia. saifalmutairi66@gmail.com*
11. *Optometrist (Specialist Optometry), Atiqa Health Consultative Center, Ministry of Health, Kingdom of Saudi Arabia. Dr.eman221@yahoo.com*
12. *Optometrist, King Slman hospital, Ministry of Health, Kingdom of Saudi Arabia. abinnooh@moh.gov.sa*

Abstract

Strabismus, a condition characterized by misalignment of the visual axes, affects approximately 1% to 3% of children and can arise due to a variety of factors, including systemic conditions, genetic predispositions, or cranial nerve palsies. This review provides a comprehensive overview of the types of strabismus, with a particular focus on esotropia and exotropia. Key diagnostic techniques such as the Hirschberg method, Bruckner test, and cover testing are discussed, emphasizing their importance in initial evaluations. Treatment strategies range from conservative approaches, such as spectacles and vision therapy, to surgical interventions for persistent or severe cases. The review also highlights the impact of strabismus on binocularity, visual acuity, and quality of life in both pediatric and adult populations. Insights into cranial nerve palsies and their associated ocular motility disturbances are presented, underscoring the need for targeted diagnostic workups and multidisciplinary management. The discussion aims to equip clinicians with the knowledge necessary for effective diagnosis and treatment of strabismus across diverse clinical scenarios.

Keywords: Strabismus, Esotropia, Exotropia, Cranial nerve palsies

Introduction

Strabismus, also known as misalignment of the visual axes, is commonly first identified during a primary care visit. The misalignment can be intermittent or subtle, which can make evaluation challenging. Strabismus may also be referred to as squint. A fundamental understanding of the various types of strabismus facilitates better communication with both patients and their parents, as well as more appropriate referrals and treatment strategies. While the term “lazy eye” is frequently used in association with strabismus, it is also applied to other conditions such as amblyopia (reduced vision in one eye without any observable physical defects or pathology), ptosis, and strabismus itself. Strabismus affects approximately 1% to 3% of children, with a higher prevalence in those who were born prematurely, have systemic conditions such as cerebral palsy, genetic syndromes, or a family history of strabismus (McKean-Cowdin et al., 2013). Parents often mistakenly believe that one eye is weaker than the other, but most commonly, one eye is simply the dominant or fixating eye. If the dominant eye is covered briefly, forcing the deviated eye to fixate, the misalignment often appears to shift to the previously dominant eye. This observation demonstrates that the neuromuscular imbalance primarily affects the coordination between both eyes, rather than being specific to one eye. In cases of cranial nerve palsy or muscle dysfunction, the dominant eye typically continues to fixate despite involvement of the affected muscle (Harley et al., 2005). The most common form of strabismus involves horizontal misalignment, which is classified as either esotropia (crossed eyes) or exotropia (outward deviation of one eye). In vertical deviations, it is important to specify which eye is affected, as left hypertropia is clinically equivalent to right hypotropia. More complex forms of strabismus may present with varying misalignment depending on the direction of gaze. Deviations are also categorized by comitance; comitant deviations are consistent across all gazes, whereas incomitant deviations change with the direction of gaze. The basic diagnostic approach to strabismus involves identifying the type of strabismus, recognizing misalignment patterns, and determining the appropriate work-up and treatment plan. This review focuses on the common types of comitant esotropia and exotropia, as well as misalignments associated with cranial nerve palsies. Special syndromes and systemic diseases affecting extraocular muscles or restrictive processes are beyond the scope of this review.

Strabismus assessment is conducted using a range of techniques. Several basic screening methods are highly effective for initial evaluation. The Hirschberg method involves directing a beam of light toward the eyes and assessing the reflex in the pupils. In cases of alignment, the light reflex will be centered within the pupil. If the child is gazing directly at the light and one light reflex is central while the other is displaced, strabismus is suspected. Another useful method is the Bruckner test. In this technique, the room lights are dimmed, and the clinician stands a few feet away from the patient while using an ophthalmoscope to direct light at the child’s face. The red reflex should appear symmetrically in both eyes; asymmetry in the red reflex suggests potential strabismus or other ocular pathology. If the child is old enough to focus on a toy, a cover test may be performed. While holding the toy in front of the child, one eye is covered briefly, and the motion of the uncovered eye is observed. If the uncovered eye moves to fixate on the toy, strabismus is indicated. In the absence of strabismus, no refixation movement should be seen. The specific technique used depends on both the clinician's comfort level and the patient’s ability to cooperate. If strabismus is present for a significant portion of the time, the child may lose binocularity and become amblyopic. In adults, strabismus can lead to debilitating diplopia.

ESOTROPIA

Esotropia is a type of ocular misalignment in which the deviating eye turns medially, toward the nose. In the initial months of life, both the visual and oculomotor systems are immature and continue to develop. Parents may perceive their child’s eyes as crossed or drifting, but such misalignments typically last only for a brief period and realign spontaneously. Most children

achieve stable eye alignment by 3 months of age, although this can be delayed in premature infants or those with delayed visual maturation. If ocular misalignment persists beyond 3 months of age, a referral to a pediatric ophthalmologist should be considered. There are several forms of comitant esotropia, including pseudoesotropia, congenital esotropia, and accommodative esotropia, which are discussed below.

Pseudoesotropia

Pseudoesotropia accounts for a significant portion of consultations in pediatric ophthalmology. The term "pseudo" indicates a false form of esotropia, where the ocular alignment is normal, but the eyes appear to be crossed. This appearance is often due to wide nasal bridges and prominent epicanthal folds common in infants. These folds can obscure the medial sclera of both eyes, making them seem crossed. An ophthalmologist should be consulted to verify the true alignment. Treatment primarily involves explaining the cause of the apparent crossing and reassuring parents that true misalignment is not present. In some cases, children may initially present with pseudoesotropia, which later progresses to intermittent esotropia. Therefore, repeated examinations on separate visits may reveal true strabismus.

Congenital Esotropia

Congenital esotropia is a form of ocular misalignment that manifests within the first 6 months of life. Any child presenting with constant eye crossing should be referred to an ophthalmologist immediately. Small-angle, intermittent crossing may be observed during the first 3 months of life, as it can spontaneously resolve in some cases. In congenital esotropia, the angle of deviation is typically large, and patients may spontaneously switch the fixating eye. These patients generally do not exhibit significant refractive errors (Campos, 2008). Additional ocular findings can also be observed, such as latent nystagmus, which causes the eye to shake or jiggle when one eye is covered. These children may also have dysfunction of the oblique eye muscles, potentially leading to pattern esotropia. For instance, a V-pattern esotropia is linked to inferior oblique muscle dysfunction, resulting in a significantly larger angle of deviation when the eyes are in downgaze compared to upgaze. Dissociated vertical deviations, characterized by the unilateral upward drift of the non-fixating eye, are also commonly associated with congenital esotropia. The treatment for congenital esotropia is primarily surgical. Glasses are seldom helpful, and there is no evidence to support the use of eye exercises, often referred to as vision therapy.

Accommodative Esotropia

Accommodative esotropia typically develops between the ages of 1 and 3 years. This form of esotropia is associated with significant hyperopia (farsightedness). While mild hyperopia is common in young children, they are generally able to accommodate or adjust their focus without difficulty. As a result, these children do not usually experience problems with seeing both near and distant objects. However, the near reflex involves both convergence and accommodation. In children with accommodative esotropia and significant hyperopia, accommodation triggers convergence, but they are unable to execute a fusional divergence movement that would realign the visual axes, resulting in esotropia. Fortunately, correcting the hyperopia with spectacles relaxes the accommodation and resolves the associated esotropia. In some cases, excessive convergence for accommodation can cause further crossing when focusing on near objects. This condition is termed a high accommodative convergence to accommodation ratio. In these cases, bifocal lenses can further relax accommodation during near-target fixation, thus alleviating the residual esotropia. Once acceptable ocular alignment is achieved, children with accommodative esotropia may still require treatment for amblyopia (reduced vision in the non-preferred eye). As children grow, hyperopia typically diminishes, particularly in adolescence. If treatment is delayed, residual esotropia may persist despite spectacle correction, and in such cases, strabismus surgery can restore proper ocular

alignment.7 Early referral to an ophthalmologist for corrective glasses can prevent the need for surgery and amblyopia management.

The acute onset of comitant esotropia in older children may indicate a neurologic cause. Children over the age of 5 often report acute diplopia (double vision). In the absence of refractive error, such esotropia may be linked to intracranial pathology, including conditions such as Arnold-Chiari type I malformation, pontine gliomas, and astrocytomas.8 In these instances, urgent neuroimaging is necessary, along with immediate referral for further evaluation.

Exodeviations

Exodeviation refers to the outward deviation of one eye relative to the other. There are various forms of exodeviations, including pseudoexotropia, exophoria, intermittent exotropia, constant exotropia, and convergence insufficiency. In all instances, it is important to gather information regarding the frequency of misalignment and the child's preferred eye. A thorough medical history that includes craniofacial syndromes, neurological disorders, infections, and trauma is crucial, as these factors may predispose individuals to exodeviations. While specific genetic markers have not been identified, a family history of exotropia is often reported.

Pseudoexotropia

Pseudoexotropia occurs when the eyes are properly aligned, but they appear to diverge outward. This appearance may result from an increased interpupillary distance or a positive angle kappa, which is the angle formed between the visual and pupillary axes. If the angle exceeds 5°, the corneal light reflex is displaced nasally, creating the illusion of exotropia. A positive angle kappa can occur in isolation or be associated with intraocular abnormalities such as temporal macular dragging due to retinopathy of prematurity, high myopia, or macular scarring resulting from infections. Children with pseudoexotropia maintain a straight head position, and no refixation movement is observed upon cover testing. No treatment is required for the alignment, but if associated macular pathology is present, amblyopia should be addressed.

Exophoria

Exophoria is a type of exodeviation that is controlled by the sensory fusion mechanisms of binocular vision. Under normal conditions, the eyes are aligned; however, when binocular fusion is disrupted, an exodeviation may emerge. In the absence of symptoms, no treatment is necessary, but observation is recommended as decompensation can lead to progression into manifest exotropia.

Intermittent Exotropia

Intermittent exotropia is the most common form of exodeviation in childhood, with onset typically occurring between 6 months and 4 years of age. The deviation is intermittent, initially becoming apparent during periods of inattention, fatigue, or stress. Parents may notice squinting or the child closing one eye, particularly in bright light, to prevent diplopia associated with the exotropic eye. Bright light can disrupt binocular fusion, making the exodeviation more evident. In some cases, the eyes realign when the child focuses on near targets, deviating only when looking at distant objects. As the condition progresses, the deviation may occur during near viewing, and the misalignment periods may become more frequent, eventually progressing to constant exotropia. Children with intermittent exotropia typically have normal vision in both eyes, without amblyopia, and may alternate fixation.

Intermittent exotropia is further classified into three types: basic exotropia, where the deviation is equal at both near and far distances; divergence excess, where the deviation is more pronounced at distance than at near; and convergence insufficiency exotropia, where the deviation is greater at near than at distance.

Non-invasive treatments are preferred over surgical intervention when possible. The primary goals of treatment are the preservation of vision, binocular fusion, and proper alignment.

Treatment begins with the correction of any underlying issues, such as significant refractive errors, which should be corrected with glasses. Amblyopia, if present, should be treated using glasses or patching. Prisms may be used for small deviations causing diplopia, though they are typically reserved for adult-onset exotropia or patients who are not candidates for surgery. Vision therapy alone has not proven effective in treating exotropia. In cases where the exotropia is large, frequent, or constant, surgical intervention may be necessary. The timing of surgery is typically when the exotropia is present for at least 50% of the waking hours and measures greater than 14 to 16 prism diopters (PD). Surgery may also be considered earlier if the exotropia causes amblyopia or significantly disrupts binocular fusion. Surgical options include bilateral lateral rectus recessions or unilateral recession of the lateral rectus combined with resection of the medial rectus. While both procedures generally provide good initial alignment, long-term success can be more challenging to define. The most common complications of strabismus surgery include overcorrection, undercorrection, or the recurrence of strabismus after an initial period of realignment (Joyce et al., 2015).

Constant Exotropia

Constant exotropia occurs when the eyes are persistently deviated outward. It is more common in older patients or those with decompensated intermittent exotropia, although a congenital form also exists. Constant exotropia typically has different classifications, including congenital exotropia, which occurs before 6 months to 1 year of age. This form of exotropia is rare in healthy children but more common in those with neurologic or craniofacial disorders. Congenital exotropia may begin as an intermittent deviation but can progress rapidly to a constant exotropia. As with congenital esotropia, the deviation angle in congenital exotropia is usually large, typically exceeding 35 PDs. Early surgical intervention is often recommended to attempt to regain some degree of binocularity. Close monitoring during the amblyogenic years is necessary to assess vision and any potential new misalignments, such as vertical deviations or esotropia.

Sensory exotropia generally refers to a unilateral exodeviation in patients with significantly reduced visual acuity in one eye. Potential causes include anisometropic amblyopia, corneal opacities, cataracts, optic nerve hypoplasia or atrophy, and retinal pathology. The eye with poor vision becomes strabismic, and this condition can be esotropic or exotropic, with exotropia occurring more commonly in older children and adults. Consecutive exotropia can develop following previous strabismus surgery for esotropia, and recurrent exotropia may follow surgery for exotropia. These forms of exotropia are observed in patients with poor binocularity, and surgical treatment is required to realign the eyes, though gradual misalignment may recur over time.

Convergence Insufficiency

Convergence insufficiency is characterized by an exophoria that is greater at near than at distance. Symptoms include asthenopia with near vision, blurred near vision, difficulty reading, a decreased near point of convergence, and reduced near fusional convergence amplitudes. Treatment options for convergence insufficiency include vision therapy exercises or the use of prism glasses. Vision therapy has proven to be effective in managing convergence insufficiency, while prismatic correction in reading glasses can provide symptomatic relief.

Ocular Cranial Nerve Palsies

The six extraocular muscles are essential for the coordinated motility of the eyes, and they are innervated by three cranial nerves. Consequently, disorders affecting any of these nerves result in specific motility disturbances. Cranial nerve palsies can cause partial or complete weakness of the corresponding muscles. Studies on cranial nerve palsies show that both men and women are equally affected, with approximately 38% of patients having concurrent systemic diseases (Pedro-Egbe et al., 2014). Among these, sixth nerve palsies are the most prevalent, accounting for 58% of cases (Rowe, 2011), followed by third nerve palsies at 26%, and fourth nerve palsies

at 16% of all cases. The patterns of motility disturbances caused by cranial nerve palsies, their underlying causes, diagnostic approach, and available treatment options are discussed.

Sixth Cranial Nerve

The sixth cranial nerve, also known as the abducens nerve, innervates the ipsilateral lateral rectus muscle, which is responsible for eye abduction. As a result, palsy of this nerve causes esotropia, which is more pronounced when gazing towards the affected side. Patients often compensate for this by turning their head away from the affected side to restore binocularity, or they may report horizontal diplopia during side gaze, depending on whether the palsy is partial or complete.

The abducens nerve originates from the pons and ascends to coordinate side gaze with the contralateral medial rectus fibers. Therefore, lesions within the abducens nucleus lead to gaze palsy. Nuclear palsies may result from pontine infarctions, gliomas, cerebellar tumors, or alcoholic encephalopathy. The abducens nerve exits the pons near cranial nerves V, VII, and VIII. Damage to the fasciculus may result in ipsilateral abduction weakness, which is often accompanied by ipsilateral facial weakness, analgesia, Horner's syndrome, and ipsilateral deafness. Vascular disease is the most frequent cause of fascicular palsies.

As the abducens nerve ascends along the clivus, it becomes vulnerable to compression from basilar tumors, such as acoustic neuromas, nasopharyngeal carcinomas, and meningiomas. Elevated intracranial pressure that leads to downward displacement of the brainstem can also stretch the abducens nerve. Once the nerve pierces the dura, it traverses the inferior petrosal sinus, making it susceptible to trauma from the temporal bone or infections of the mastoid. Congenital sixth nerve lesions are rare. The etiology of sixth nerve palsies falls into five broad categories: idiopathic (8% to 30%), tumors and other miscellaneous causes (10% to 30%), traumatic (3% to 30%), and vascular causes (0% to 36%). In adults over 50 years old, microvascular disease is the most common cause, and spontaneous resolution is frequent (Surachatkumtonekul et al., 2012). In elderly patients, giant cell arteritis must also be considered. In the absence of vasculopathic risk factors, neuroimaging, lumbar puncture, and blood tests (including CBC, sedimentation rate, Lyme disease, and syphilis testing) may be necessary (Table 1).

The diagnostic work-up for sixth nerve palsy depends on the patient’s age and general health. In children, an extensive evaluation is essential. In one study, tumor was found in 31% of children with isolated sixth nerve palsy, in the absence of papilledema or other cranial pathology (Dotan et al., 2013). Any associated signs, such as Horner's syndrome, involvement of other cranial nerves, nystagmus, papilledema, or contralateral weakness, require neuroimaging with MRI to assess the brainstem. Elevated intracranial pressure, which can lead to unilateral or bilateral sixth nerve palsies, accounts for up to 60% of cases of sixth nerve palsy. Sixth nerve palsies may also occur as part of postviral syndromes or in association with multiple sclerosis.

Microvascular causes, often secondary to hypertension or diabetes, tend to resolve spontaneously over a 6- to 12-month period, with spontaneous recovery occurring in approximately 66% to 73% of patients (Surachatkumtonekul et al., 2012). Traumatic causes resolve in 27% to 50% of cases. If no improvement is observed, further evaluation is warranted, especially if microvascular disease was initially suspected. Patching can alleviate the diplopia associated with sixth nerve palsies. For children under 8 years old, who are at risk for amblyopia, alternate eye patching is recommended. Prism therapy can help alleviate symptoms in the primary gaze, although diplopia may still occur during side gazes. Treatment is generally tailored to the underlying cause. In cases of idiopathic etiology or nonresolving microvascular disease after six months, surgical intervention may be required to weaken the ipsilateral medial rectus, strengthen the ipsilateral lateral rectus, or transpose the ipsilateral superior rectus or both the superior and inferior rectus to the lateral rectus, depending on the severity of the palsy

(Holmes et al., 2012; Mehendale, 2012). A study found that approximately 80% of patients achieved an acceptable range of single vision following surgery (Surachatkumtonekul et al., 2012).

Third Cranial Nerve

The third cranial nerve, also known as the oculomotor nerve, is responsible for controlling four out of the six extraocular muscles. Upon entering the orbit, it divides into superior and inferior branches. The superior division innervates the superior rectus and the levator palpebrae superioris, while the inferior division supplies the medial rectus, inferior rectus, and inferior oblique muscles. Additionally, the pupillary constrictor fibers travel alongside the inferior division. Consequently, complete or partial palsies of the oculomotor nerve can lead to various deviations. Complete palsies are more recognizable, presenting with total ptosis, pupillary dilation, exotropia, and depression of the globe. Diplopia may initially be obscured by the ptosis. Pupillary dilation often results in light sensitivity and impaired accommodation. Incomplete palsies are characterized by signs and symptoms specific to the affected muscles.

The oculomotor nucleus is a complex structure comprising multiple subnuclei within the brainstem. Fibers that innervate the levator palpebrae superioris are shared by a single midline subnucleus, whereas fibers supplying the superior rectus uniquely cross as they leave the nucleus. Therefore, lesions within the oculomotor nucleus can result in bilateral ptosis and contralateral limitations in elevation. Symptoms such as tremors or spastic paralysis indicate a nuclear or fascicular lesion, necessitating MRI for clarification.

In the subarachnoid space, the peripheral segment of the nerve is susceptible to injury from posterior communicating artery aneurysms. Acute onset of oculomotor palsy with pupillary involvement necessitates magnetic resonance angiogram (MRA) or arteriography to rule out such a life-threatening aneurysm. Other causes include uncal herniation and trauma, which can affect the nerve as it pierces the dura. Traumatic causes often affect pupillary fibers first due to their superficial dorsal-medial location within the nerve at this site.

Within the cavernous sinus, the oculomotor nerve runs along the lateral dorsal wall near the fourth cranial nerve. Conditions such as cavernous sinus fistulas, aneurysms, tumors, pituitary apoplexy, and infections or inflammations in the cavernous sinus may result in combined palsies involving other ocular cranial nerves. Within the orbit, trauma, local neoplastic processes, and infections can impair either the superior or inferior divisions of the oculomotor nerve. A review of head injuries indicated that approximately half of the cases had associated ocular morbidity, with 11.6% exhibiting complete or partial oculomotor nerve palsy (Sharma et al., 2014). Migraines may also cause partial oculomotor palsies, with recent studies attributing up to one-third of such cases to a demyelinating process or neuropathy (Gelfand et al., 2012).

The most frequent cause of pupil-sparing oculomotor palsy is ischemia. In patients over the age of 50 with atherosclerotic risk factors, isolated, pupil-sparing oculomotor palsy is typically due to microvascular disease, and neuroimaging may not be necessary (Murchison, 2011). Ischemic damage often resolves spontaneously within three to six months. In older patients, testing for giant cell arteritis is recommended, and a comprehensive workup should include a complete blood count (CBC), glucose tolerance testing, sedimentation rate, blood pressure measurement, syphilis testing, and antinuclear antibody testing. Close observation of the pupil is crucial because pupil involvement can develop within the first five days of presentation. Pupillary involvement warrants urgent neuroimaging with emphasis on identifying aneurysms (MRI/MRA) and immediate referral to neurosurgeons (see Table 1).

In children, congenital causes and trauma are the most prevalent etiologies of oculomotor palsies. However, a study from Nigeria involving young patients aged 20 to 29 years found systemic disorders in 38% of individuals presenting with cranial nerve palsy (Pedro-Egbe et al., 2014). Other non-localizing causes of oculomotor palsy include migraines, myasthenia,

post-vaccination inflammation, granulomatous diseases such as sarcoidosis, infectious meningitis, idiopathic intracranial hypertension (IIH), and multiple sclerosis (Muralidhar et al., 2013; Woo et al., 2014).

Spontaneous recovery of the oculomotor nerve can result in misdirection of nerve fibers to other muscles supplied by the same nerve, a phenomenon known as aberrant regeneration. Common misdirections include eyelid elevation during adduction or depression of the eye, adduction of the eye during attempted downgaze, and segmental pupillary constriction during adduction. Aberrant regeneration is commonly observed after recovery from trauma, aneurysms, or tumors but not following microvascular insults such as diabetic disease. The presence of aberrant regeneration should prompt a reevaluation to identify these underlying causes.

Treatment of oculomotor palsy focuses on addressing the underlying etiology when possible. If recovery from a microvascular cause is incomplete six months post-onset, strabismus surgery may be considered. Surgical management aims to alleviate symptoms in the primary gaze position while accounting for the complexity of the misalignment. If achieving a satisfactory range of single vision is not feasible, occlusion of one eye may be a reasonable alternative. Surgical options include globe fixation using various suture materials, lateral rectus disinsertion, superior oblique disinsertion, or muscle transpositions to re-center the globe. Preoperative assessment of residual muscle function within the oculomotor nerve's distribution is critical for guiding surgical decisions.

Fourth Cranial Nerve

The fourth cranial nerve, known as the trochlear nerve, innervates the superior oblique muscle. The primary function of the superior oblique is intorsion, which involves rotating the eye toward the nose. This cycloduction occurs when the head tilts to the side. To maintain visual field stability, the eyes perform a counter-rotation to the head movement. For instance, when the head tilts toward the right shoulder, the right eye intorts while the left eye extorts, or rotates toward the ear. The superior rectus muscle also assists with intorsion. In cases of superior oblique palsy, the elevation caused by the superior rectus cannot be counterbalanced by the superior oblique during intorsion, leading to a hyperdeviation when the head tilts toward the side of the affected eye. Additionally, the superior oblique serves as a depressor and an abductor of the eye. When the superior oblique is impaired, a hyperdeviation results. The superior oblique has the most significant effect on vertical positioning when the eye is adducted. Consequently, the severity of diplopia in superior oblique palsy worsens during gaze directed away from the eye with the weakened muscle.

The Parks three-step test is utilized to identify which vertical muscle is responsible for a hyperdeviation. The first step involves assessing the deviation in the primary position, where the patient gazes straight ahead with the head upright. A hyperdeviation in this position indicates weakness in the depressors of the affected eye or the elevators of the contralateral eye. The second step evaluates which horizontal gaze exacerbates the deviation. Vertical rectus muscles exert more influence when the eye is abducted, whereas the obliques have greater vertical impact when the eye is adducted. The third step examines whether the deviation worsens with intorsion or extorsion during head tilt. The results of these steps pinpoint the affected muscle. While the three-step test is valuable, it is not applicable in cases of restrictive conditions involving the muscles or multiple muscle palsies (Muthusamy et al., 2014).

Vertical and torsional diplopia are common in fourth cranial nerve palsies. Patients with congenital causes often experience intermittent symptoms or diplopia limited to specific gazes due to adaptive vergence mechanisms, whereas acquired cases usually present with sudden and constant diplopia. The ability to fuse significant vertical deviations strongly suggests a congenital origin. Many patients adopt a head tilt away from the side of the impaired muscle

to minimize and better control the vertical deviation. Evidence from old photographs showing a persistent head tilt can support the congenital nature of the palsy.

Acquired lesions are typically caused by pathology along the course of the trochlear nerve and less commonly within the superior oblique muscle or tendon (Merino et al., 2014). Causes include trauma, neoplasms, ischemia, increased intracranial pressure, aneurysms, meningitis, and idiopathic factors. The trochlear nerve originates in the brainstem and decussates upon exiting. Conditions such as vascular disease, trauma, and demyelinating processes can damage the nerve within the brainstem, resulting in contralateral superior oblique palsy. These cases often involve additional symptoms from nearby structures, such as ipsilateral Horner syndrome caused by damage to descending sympathetic fibers.

The peripheral trochlear nerve loops around the brainstem, pierces the dura, and enters the superior orbital fissure via the cavernous sinus. Its extended course makes it particularly vulnerable to closed head trauma. Ischemic injury is the second most common cause of superior oblique palsy following trauma. A thorough history of hypertension, diabetes, and other vascular ischemic risk factors is crucial. Conditions such as hydrocephalus, idiopathic intracranial hypertension (IIH), and tumors compressing the nerve pathway can also lead to superior oblique palsy. Acquired cases warrant neuroimaging for diagnosis.

Ischemic superior oblique palsy often resolves spontaneously within six months. Treatment focuses on supportive measures until recovery. These measures include occlusion of one eye, prism correction within glasses to alleviate symptoms, or surgery if there is no resolution. Surgical intervention aims to balance the residual weakness and is guided by the Knapp guidelines, which recommend addressing the muscle corresponding to the maximum deviation. Target muscles include the ipsilateral superior oblique, inferior oblique, and superior rectus or the contralateral inferior rectus. Deviations exceeding 15 prism diopters (PDs) or 7 degrees of vertical misalignment typically necessitate surgery on two muscles (Nejad et al., 2013; Sekeroğlu et al., 2013). Torsional deviations are best managed with surgery targeting the superior oblique muscle (Li & Zhao, 2014). Surgical outcomes are more favorable when the preoperative deviation is less than 15 PDs, with fusion in the primary position achieved in most cases. When deviations are larger, success rates range from 60% to 65% (Durnian & Marsh, 2011).

Conclusion

Strabismus represents a significant clinical entity with implications for visual function and overall quality of life. Early recognition and intervention are crucial in minimizing long-term sequelae such as amblyopia and diplopia. Diagnostic strategies, including non-invasive tests and neuroimaging, are essential for accurate identification of the underlying cause. The management of strabismus is multifaceted, encompassing optical correction, vision therapy, and surgical interventions tailored to the specific type and severity of misalignment. Additionally, the intricate relationship between systemic conditions, cranial nerve palsies, and strabismus necessitates a holistic approach to patient care. Advances in surgical techniques and an improved understanding of the neuro-ophthalmological aspects of strabismus continue to enhance treatment outcomes. Clinicians must remain vigilant in identifying strabismus early and providing timely, evidence-based interventions to optimize patient outcomes.

References

Campos, E. C. (2008). Why do the eyes cross? A review and discussion of the nature and origin of essential infantile esotropia, microstrabismus, accommodative esotropia, and acute comitant esotropia. *Journal of American Association for Pediatric Ophthalmology and Strabismus*, *12*(4), 326–331. https://doi.org/10.1016/j.jaapos.2008.03.013

Dotan, G., Rosenfeld, E., Stolovitch, C., & Kesler, A. (2013). The role of neuroimaging in the evaluation process of children with isolated sixth nerve palsy. *Child's Nervous System*, *29*(1), 89–92. https://doi.org/10.1007/s00381-012-1929-z

Durnian, J. M., & Marsh, I. B. (2011). Superior oblique tuck: Its success as a single muscle treatment for selected cases of superior oblique palsy. *Strabismus*, *19*(4), 133–137. https://doi.org/10.3109/09273972.2011.620058

Gelfand, A. A., Gelfand, J. M., Prabakhar, P., & Goadsby, P. J. (2012). Ophthalmoplegic "Migraine" or Recurrent Ophthalmoplegic Cranial Neuropathy: New Cases and a Systematic Review. *Journal of Child Neurology*, *27*(6), 759–766. https://doi.org/10.1177/0883073811426502

Harley, R. D., Nelson, L. B., & Olitsky, S. E. (2005). *Harley's Pediatric Ophthalmology*. Lippincott Williams & Wilkins.

Holmes, J. M., Hatt, S. R., & Leske, D. A. (2012). Intraoperative monitoring of torsion to prevent vertical deviations during augmented vertical rectus transposition surgery. *Journal of American Association for Pediatric Ophthalmology and Strabismus*, *16*(2), 136–140. https://doi.org/10.1016/j.jaapos.2011.11.010

Joyce, K. E., Beyer, F., Thomson, R. G., & Clarke, M. P. (2015). A systematic review of the effectiveness of treatments in altering the natural history of intermittent exotropia. *The British Journal of Ophthalmology*, *99*(4), 440–450. https://doi.org/10.1136/bjophthalmol-2013-304627

Li, Y., & Zhao, K. (2014). Superior oblique tucking for treatment of superior oblique palsy. *Journal of Pediatric Ophthalmology and Strabismus*, *51*(4), 249–254. https://doi.org/10.3928/01913913-20140527-01

McKean-Cowdin, R., Cotter, S. A., Tarczy-Hornoch, K., Wen, G., Kim, J., Borchert, M., & Varma, R. (2013). Prevalence of Amblyopia or Strabismus in Asian and Non-Hispanic White Preschool Children: Multi-Ethnic Pediatric Eye Disease Study. *Ophthalmology*, *120*(10), 2117–2124. https://doi.org/10.1016/j.ophtha.2013.03.001

Mehendale, R. A. (2012). Superior Rectus Transposition and Medial Rectus Recession for Duane Syndrome and Sixth Nerve Palsy. *Archives of Ophthalmology*, *130*(2), 195. https://doi.org/10.1001/archophthalmol.2011.384

Merino, P. S., Rojas, P. L., Gómez De Liaño, P. S., Fukumitsu, H. M., & Yáñez, J. M. (2014). Bilateral Superior Oblique Palsy: Etiology and Therapeutic Options. *European Journal of Ophthalmology*, *24*(2), 147–152. https://doi.org/10.5301/ejo.5000362

Muralidhar, R., Vijayalakshmi, P., Mahesh, S., Srinivasan, K. G., & Karthik, S. N. (2013). Ophthalmic Migraine With Pupil-Sparing Third Nerve Palsy. *Journal of Pediatric Ophthalmology & Strabismus*, *50*(5), 320–320. https://doi.org/10.3928/01913913-20130222-01

Murchison, A. P. (2011). Neuroimaging and Acute Ocular Motor Mononeuropathies: A Prospective Study. *Archives of Ophthalmology*, *129*(3), 301. https://doi.org/10.1001/archophthalmol.2011.25

Muthusamy, B., Irsch, K., Peggy Chang, H.-Y., & Guyton, D. L. (2014). The sensitivity of the bielschowsky head-tilt test in diagnosing acquired bilateral superior oblique paresis. *American Journal of Ophthalmology*, *157*(4), 901-907.e2. https://doi.org/10.1016/j.ajo.2014.01.003

Nejad, M., Thacker, N., Velez, F. G., Rosenbaum, A. L., & Pineles, S. L. (2013). Surgical Results of Patients With Unilateral Superior Oblique Palsy Presenting With Large Hypertropias. *Journal of Pediatric Ophthalmology & Strabismus*, *50*(1), 44–52. https://doi.org/10.3928/01913913-20121113-01

Pedro-Egbe, C. N., Fiebai, B., & Awoyesuku, E. A. (2014). A 3-Year Review of Cranial Nerve Palsies from the University of Port Harcourt Teaching Hospital Eye Clinic, Nigeria. *Middle East African Journal of Ophthalmology*, *21*(2), 170. https://doi.org/10.4103/0974-9233.129770

Rowe, F. (2011). Prevalence of ocular motor cranial nerve palsy and associations following stroke. *Eye*, *25*(7), 881–887. https://doi.org/10.1038/eye.2011.78

Sekeroğlu, H. T., Sanac, A. S., Arslan, U., & Sener, E. C. (2013). Superior oblique surgery: When and how? *Clinical Ophthalmology (Auckland, N.Z.)*, *7*, 1571–1574. https://doi.org/10.2147/OPTH.S46382

Sharma, B., Gupta, R., Anand, R., & Ingle, R. (2014). Ocular manifestations of head injury and incidence of post-traumatic ocular motor nerve involvement in cases of head injury: A clinical review. *International Ophthalmology*, *34*(4), 893–900. https://doi.org/10.1007/s10792-014-9898-8

Surachatkumtonekul, T., Soontrapa, P., Kampanartsanyakorn, S., & Dulayajinda, D. (2012). Causes and treatment outcomes of third, fourth and sixth cranial nerve palsy. *Journal of the Medical Association of Thailand = Chotmaihet Thangphaet*, *95 Suppl 4*, S96-101.

Woo, E. J., Winiecki, S. K., & Ou, A. C. (2014). Motor palsies of cranial nerves (excluding VII) after vaccination. *Human Vaccines & Immunotherapeutics*. https://doi.org/10.4161/hv.27032