

Abnormal Head Posture due to Ocular Problems: A Review

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Abstract: An abnormal head posture (AHP), or torticollis, is a common condition in children, with an estimated incidence of 1.3%. This condition is encountered commonly by primary care family pediatricians. AHP can be congenital or acquired. The cause of the AHP can be ocular, orthopedic and neurologic. The orthopedic causes of AHP include congenital muscular torticollis due to tightness of the sternocleidomastoid muscle, Klippel-Feil anomaly and brachial plexus injury.

Neurologic causes of AHP are mainly related to brain tumors, postinflammatory central nervous system conditions, psychomotor delay and focal dystonia. Other less common reasons for AHP are: Sandifer syndrome (hiatal hernia associated with gastro-esophageal reflux) and unilateral hearing loss.

Numerous ocular conditions can cause AHP or "ocular torticollis". Among them: superior oblique muscle palsy, lateral rectus muscle palsy, nystagmus, vertically incomitant horizontal strabismus (A or V patterns), Brown's syndrome, Duane's syndrome, refractive errors and DVD.

The AHP can take the form of head tilt, face turn, chin up, chin down or combined, depending on the specific etiology. However, there are many variations and the type of the head posture cannot reliably predict the underlying cause.

Since the etiology is not always obvious, these patients must be carefully evaluated, and sometimes a multidisciplinary approach is needed, including examinations by ophthalmologist, neurologist and orthopedist.

Ocular AHP is usually an attempt to improve visual acuity or binocularity. Some patients adopt the head posture to avoid diplopia caused by incomitant strabismus, those with nystagmus adopt a head position that brings the eyes to the null point (where the oscillations dampen or markedly diminish). Ocular AHP is usually a binocular phenomenon. Rarely, abnormal head position can be acquired following visual loss in one eye.

The majority of these ocular conditions require eye muscle surgery. Different ocular etiologies of AHP require different surgical strategy, for this reason careful etiological diagnosis is important.

The purpose of this article is to review the ocular conditions that cause AHP, their relative frequency, indication for surgery and the appropriate surgical treatment.

Keywords: Abnormal head position, abnormal head posture, torticollis, head tilt.

OCULAR CAUSES OF ABNORMAL HEAD POSTURE

There are many ocular causes for AHP that can be manifested as head tilt, face turn, chin up or chin down, depending on the specific etiology. The head posturing in the ocular cases usually serves to promote bifoveal fixation, allow binocular vision, or improve visual acuity. Less commonly it serves to compensate for refractive errors or visual field defects [1].

The abnormal head position can be simple (head tilt, face turn or chin up or down), or it can be combined from more than one element. In order to identify head tilt one can observe whether the ears and eyes are level. A face turn is diagnosed by checking whether the patient's nose is centered or whether both eyes can be seen equally. Seeing the patient's nostrils is indicating chin elevation, while chin depression is marked by inferior scleral show [1].

In a series of 73 patients with AHP published by Nucci and Kushner, an AHP in the form of a face turn was exclusively caused by ocular motility problems. Patients with superior oblique muscle palsy predominantly manifested a head tilt, while patients with nystagmus or Duane's syndrome manifested a face turn [2].

However, there are many variations and the type of the head posture cannot reliably predict the underlying cause.

Incomitance is the most common cause of ocular abnormal head posture accounting for 63-70% of ocular torticollis [3, 4]. The patient is assuming the head position in order to seek for alignment and fusion and to avoid diplopia.

Nystagmus is the second common cause for ocular torticollis accounted for 17-20% in different series [3, 4]. The patient is searching for the null-point where the oscillations dampen or markedly diminish.

Incomitant Strabismus

Superior Oblique Muscle Palsy

The fourth, or trochlear, cranial nerve palsy is the most frequent clinical entity responsible for abnormal head pos-

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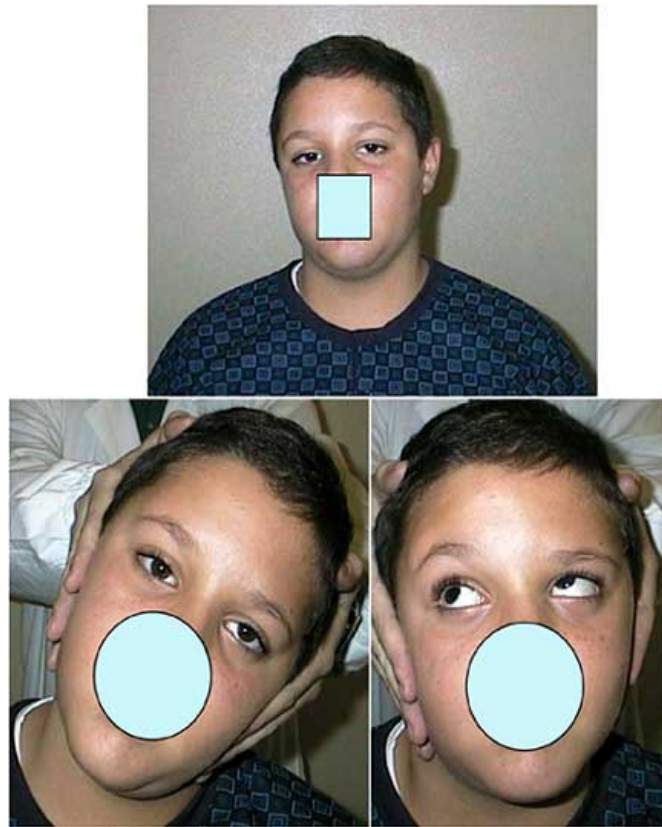


Fig. (1). Left eye superior oblique palsy.

tures in many series [2-4]. It is also the most common isolated palsy encountered by ophthalmologists. The palsy may be congenital or acquired. If acquired it is usually due to closed head trauma or, rarely, due to central nervous system vascular lesions or tumors, or diabetes. The palsy may be unilateral or bilateral. The examiner should have high index of suspicion for possible bilaterality. The patient may present with hypertropia of the involved eye if he is fixating with the unaffected eye or with hypotropia of the unaffected eye if he is fixating with the paretic eye. Abnormal head position is common, usually a contralateral head tilt (toward the side of the unaffected eye). Sometimes there is also face turn and chin elevation or depression (Fig. 1).

The diagnosis is supported by the three-step test that is positive when the vertical deviation is more marked with the head tilted to the side of the affected eye than with the head tilted to the unaffected side. In bilateral cases the test is positive when a right hypertropia increases on right tilt and a left hypertropia increases on left tilt. The double Maddox rod test is important in measuring the torsion.

Indications for surgery are abnormal head position, significant vertical deviation, or diplopia.

The performed procedure is often an inferior oblique weakening in the affected eye.

Lateral Rectus Muscle Palsy

Sixth cranial nerve palsy, which may be congenital or acquired, and causes incomitant esodeviation, is another common cause for ocular torticollis. There is usually a face turn toward the side of the affected eye to maintain fusion.

One third of these cases is associated with intracranial lesions and may have associated neurologic findings. Other cases may be related to post-infectious or immunologic processes that involve the sixth cranial nerve. These spontaneous lesions usually resolve over several months.

Surgery may be considered when spontaneous resolution does not take place after 6 months of follow-up and after exclusion of intracranial lesions. The goal of surgery is alignment in primary position with elimination of the face turn.

In a partial paralysis of the lateral rectus a large recession of the antagonist medial rectus muscle with resection of the lateral rectus is often successful. In cases of total paralysis muscle transposition procedure is needed.

Duane Syndrome

Duane syndrome is a developmental disorder in which the nucleus of the sixth cranial nerve is absent and an aberrant branch of the third cranial nerve is innervating the lateral rectus muscle in addition to the normal innervation of the medial rectus.

There are few types of motility disturbances, common to all: retraction of the globe, narrowing of the palpebral fissure and upshoots and downshoots (leash phenomenon) on attempted adduction. Most cases are unilateral with predominance for involvement of the left eye. Most cases of Duane syndrome are sporadic, but about 10% show autosomal dominant inheritance. While most affected patients have Duane syndrome alone, many associated systemic defects have been observed, including Goldenhar syndrome (hemi-

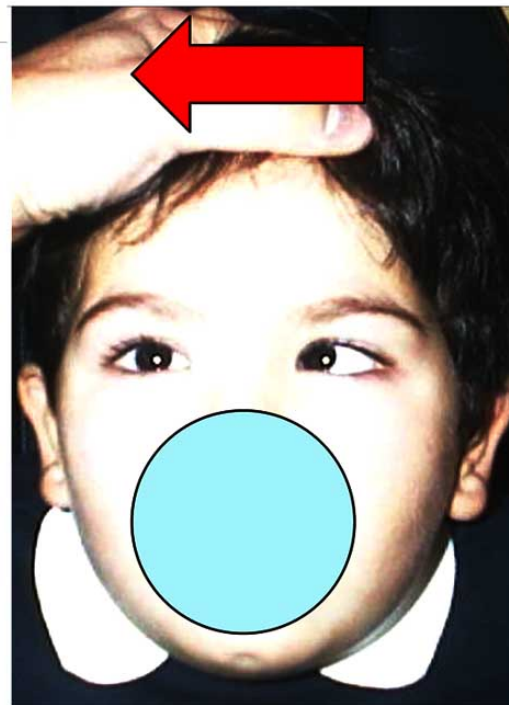


Fig. (2). Right eye Duane syndrome type 1.

facial microsomia, ocular dermoids, ear anomalies, preauricular skin tags, and upper eyelids colobomas). In Duane syndrome type 1 there is poor abduction, in type 2 poor adduction, and in type 3 both poor adduction and abduction. Esotropia and deficient abduction is the most common form (50-80%), while exotropia and deficient adduction is the next most common (20-33%). In primary position the eyes can be straight, esodeviating or exodeviating. If there is strabismus in primary position there is usually a head turn. The head turn is toward the affected eye for Duane syndrome type 1 (Fig. 2), and toward the opposite side in type 2.

Many Duane syndrome patients have some position of gaze in which the eyes are aligned and they thus develop binocular vision and amblyopia is less common.

Main indications for surgery are deviated eyes in primary position and abnormal head position. Surgery could be considered for those with marked globe retraction or large upshoot or downshoot in adduction.

For Duane type 1 recession of the ipsilateral medial rectus has been most often used to correct the esotropia in primary position and eliminate the head turn. Abduction will not improve. Resection of the lateral rectus should be avoided because of the likelihood that globe retraction will worsen. For Duane type 2 the recommended surgery is comparable: recession of the lateral rectus on the involved side for small deviations and of both lateral recti for large deviations, with avoidance of resection of the medial rectus. Duane type 3 patients, with both poor abduction and poor adduction, often have straight eyes in primary position, and since surgery will not improve eye movements, they are usually not operated. If the retractions are severe, it may be helpful to recess both the medial and lateral rectus muscles. For severe leash phenomenon some surgeons split the lateral

rectus muscle in a Y configuration and perform a posterior fixation of the muscle.

Brown Syndrome

Restriction of free passage of the superior oblique tendon through the trochlea is causing restriction of elevation in adduction.

Brown syndrome occurs in congenital, acquired, constant and intermittent forms. The acquired form is often related to trauma, local inflammation (as part of orbital pseudotumor) or systemic inflammatory (rheumatological) conditions. It is bilateral in 10% of cases. There is a deficiency of active and passive elevation in adduction, which is the field of vertical action of the superior oblique muscle. Out of this field the elevation improves in midline, with minimal or no elevation deficit in abduction. In adduction the palpebral fissure widens and a downshoot of the eye is often seen. Positive forced duction test demonstrating restricted elevation in adduction is essential for the diagnosis. This finding along with minimal or no superior oblique muscle overaction differentiate Brown syndrome from inferior oblique muscle palsy.

Brown syndrome is graded as mild, moderate or severe. The severe cases have both a downshoot in adduction and a hypotropia in primary gaze. These cases are often accompanied by abnormal head posturing. The most common head posture is chin up, and less common is face turn away from the affected eye (Fig. 3).

Surgical treatment is indicated when a primary position hypotropia and/ or anomalous head posture is present, and spontaneous resolution seems unlikely. About two third of the cases are mild or moderate and these are most often left untreated. The common procedure is superior oblique tenotomy.

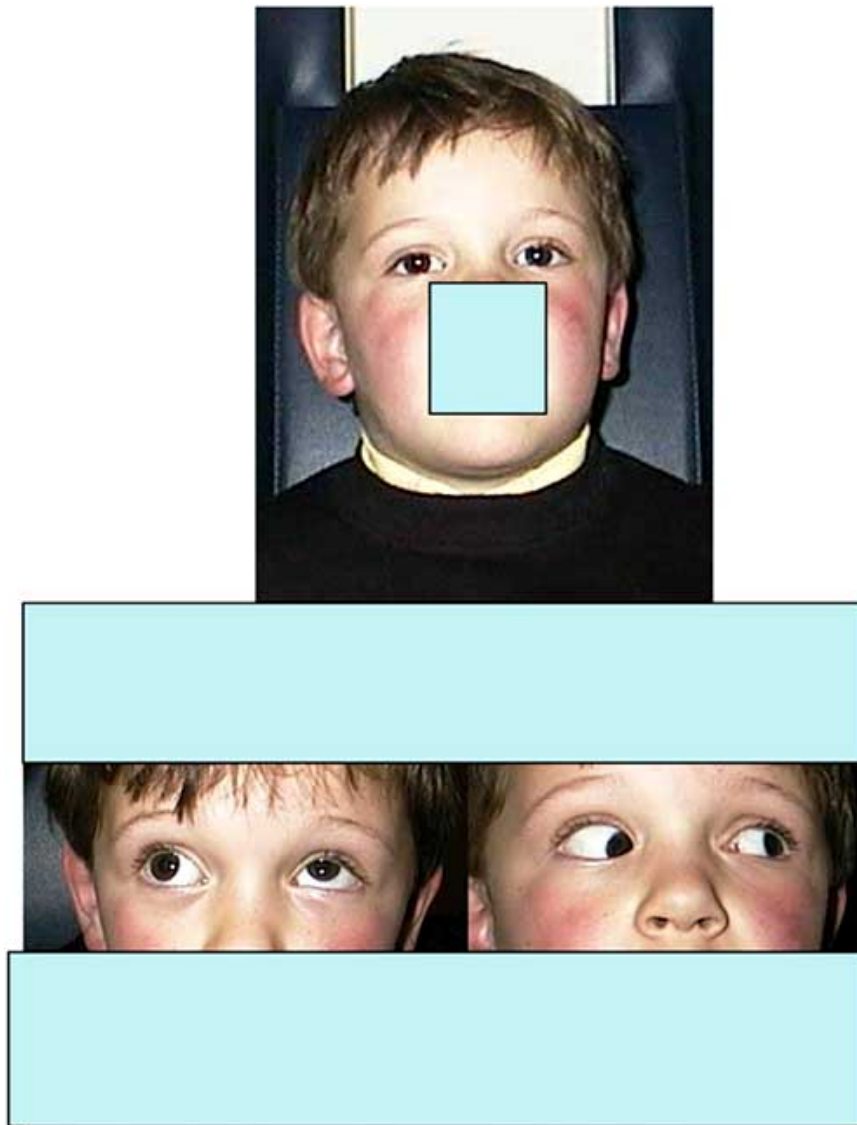


Fig. (3). Chin up in right eye Brown syndrome.

Dissociated Vertical Deviation (DVD)

DVD is a common innervational disorder that may be related to early disruption of binocular development.

The eye may spontaneously and slowly drift upward (sometimes with a simultaneous abduction and extorsion) when an eye is occluded or during visual inattention. When the vertically deviated eye moves down toward horizontal, the fixating fellow eye makes no downward movement. Hering's law of equal innervation does not apply to DVD. The condition is usually bilateral, although frequently asymmetrical. DHD, latent nystagmus, horizontal strabismus (especially congenital esotropia) are common associations.

DVD as a reason for manifest head tilt has recently been reported in few series [5, 6]. The assumed head tilts appear to decrease the magnitude and improve the motor control of the DVD (Fig. 4). The head tilt may be directed toward or away from the side of the hyperdeviated eye [5, 6]. Some patients with DVD will have a head tilt pattern with a hyperdeviation that increases when the head is tilted to the opposite side [7]. Others with DVD will have a head tilt pattern

with a hyperdeviation that increases when the head is tilted to the same side [8]. When fixating with one eye predominates, tonic fixational innervation to the contralateral superior rectus muscle can lead to a contracture of that muscle. Recession of the tight superior rectus muscle can reduce the head tilt. In DVD of the non-seeing eye, according to Nucci and Rosenbaum, the poorer seeing eye develops a dissociated extorsional movement that sends a consensual intorsional stimulus to the fixating eye. To correct this incyclotorsion the head is tilted toward the non-fixating eye [9].

Brodsky *et al.* described nine children with congenital esotropia who developed unexplained head tilts following horizontal realignment of the eyes in association with asymmetrical DVD. Five children maintained a head tilt toward the side of the fixing eye, which did not serve to control the DVD. Four children maintained a head tilt toward the side of the hyperdeviating eye, which served to control the DVD. A head tilt toward the side of the fixing eye corresponds with a postural manifestation of the underlying central vestibular imbalance that produces DVD, while a head tilt toward the



Fig. (4). Head tilt in asymmetric RE DVD.

side of the hyperdeviating eye serves to counteract the hyperdeviation and stabilize binocular vision [10].

Treatment for DVD is indicated if the vertical deviation occurs spontaneously, is frequent, and is cosmetically significant. Surgical treatment often improves the condition but rarely eliminates it.

A- and V- Patterns

Horizontal deviations that change in magnitude with up-gaze and downgaze are incomitant subtypes of horizontal strabismus. In horizontal strabismus with A or V pattern sometimes there is a chin elevation or depression. 15-25% of all strabismus cases have an associated A or V pattern. The head posturing avoids the increased deviation caused by the pattern. Chin up is characteristic of V-pattern exotropia or A-pattern esotropia. Chin down is sometimes present in V-pattern esotropia or A-pattern exotropia. Of those who do not assume head postures, many have deviations so large that they are not in the fusional range in any gaze field. Others may have no fusional ability [1].

In Kushner's series, only 9% of patients with A or V patterns exhibited a chin elevation or depression [4].

Clinically significant patterns are typically treated surgically. Surgery to eliminate horizontal deviation in primary position should be independently selected. Most patients with large A or V patterns will also have significant oblique muscle overaction. If so, a weakening procedure is done on the overacting oblique in addition to the recession-resection procedure. When there is no oblique dysfunction horizontal rectus muscle transpositions are indicated, meaning the horizontal rectus muscle is transposed vertically during the recession or resection.

Double Elevator Palsy

Double elevator palsy, or the more correct term monocular elevation deficiency, is any strabismus manifesting

deficit elevation in all positions of gaze. It can be due to paresis of one or both elevator muscles, but also due to inferior rectus muscle restriction. Chin elevation is often adopted in order to obtain fusion in down-gaze. Surgical indications include significant chin-up posture or hypotropia in primary position. The ipsilateral inferior rectus may be recessed if there is restricted elevation on forced duction test. If there is no restriction, the medial rectus and the lateral rectus muscles should be transposed toward the superior rectus muscle (Knapp procedure).

Inferior Oblique Palsy

Paresis of the inferior oblique muscle is demonstrated by deficiency of elevation from an adducted position. Patients may also show a mild hypotropia in primary position that increases with adduction, and sometimes there is an associated A pattern.

Patients usually assume a head posture with head tilt toward the side of the paretic eye and a face turn to the opposite side in order to keep the paretic eye in abduction [1].

Surgical indications include significant anomalous head posture, vertical deviation, and diplopia. Ipsilateral superior oblique weakening may be considered.

Third Nerve Palsy

There are limitations in adduction, elevation and depression. The eye is usually markedly exotropic and hypotropic in primary position. If ptosis is complete there will be no need for head posturing. Most fusing patients will assume a face turn opposite to the affected eye, putting the eye in the field of action of the normal lateral rectus muscle.

Surgical options may include a large recess/resect procedure for the exotropia combined with vertical transposition of the horizontal muscles, or combined with superior oblique weakening.

Nystagmus Dampening

A null point, or neutral zone, is a position of gaze, where the intensity of the oscillations of the nystagmus is diminished and the visual acuity improves. If the null point is not in primary position, anomalous head postures may be assumed to dampen the nystagmus and provide the best visual acuity. The head posture usually consists of face turn that brings the eyes to the null point position. If the null point is to the left, there will be a head turn to the right and vice versa.

Congenital motor nystagmus (CN) is dampened by convergence and therefore often associated with an esotropia. These children will fixate with the inturned eye and turn their heads to look across their noses.

Latent/manifest latent nystagmus (LN, MLN) is a congenital nystagmus that occurs under conditions of monocular fixation. It may become manifest when both eyes are open but only one eye is being used for vision (the other eye is suppressed or amblyopic). Latent nystagmus is often present in children with congenital esotropia and is sometimes associated with DVD. It is often associated with a face turn toward the fixating eye which is in adduction.

Patients with Ciancia syndrome, a specific form of nystagmus and esotropia, have a very large angle constant esotropia and often show a pattern of cross fixation. They may also alternate head postures with cross fixation.

The combination of a nystagmus with an esotropia has been termed- nystagmus blockage syndrome, in which the angle of esotropia and the amplitude of nystagmus have an inverse relationship.

Hertle *et al.* studied 37 children with nystagmus and strabismus who also had anomalous head postures. They distinguished between “gaze null” and “adducting null”. A “gaze null” is the null point where there is the best visual acuity due to smallest amplitude nystagmus. It is characteristic of congenital nystagmus, and it composed 62% of Hertle’s patients. The “adducting null” was the null point in adduction characteristic of manifest latent nystagmus in 32% of the patients [11].

In Kushner’s series of 38 patients with head posture and nystagmus, 20 had idiopathic congenital nystagmus with a null point other than in the primary position, 6 had congenital nystagmus with one blind eye while the seeing fixating eye had a jerk nystagmus with a null point in adduction, and 10 of 38 patients had congenital nystagmus associated with ROP [4].

Spasmus nutans is an acquired condition associated with a triad of findings including nystagmus, head nodding and torticollis. Age of onset is usually 3-15 month. In most cases it is a benign disorder that disappears by age 3-4 years.

Surgery for nystagmus is indicated to correct a head turn or improve visual function. By shifting the null point to primary position, surgery can obtain the best vision without the need for a head turn. In Kestenbaum or Kestenbaum-Anderson procedure the eyes are surgically rotated in the direction of the head turn, that is, away from the null point by a recess-resect procedure on both eyes.

An alternative is to recess all the horizontal rectus muscles posterior to the equator.

OTHER OCULAR REASONS FOR AHP

Beside incomitance and nystagmus there are other less common ocular reasons for abnormal head posture.

Some restrictive strabismus can cause pain or discomfort during the effort of fixation. In order to fixate with less duress against the restriction, a compensatory abnormal head position may occur whether or not fusion results. Examples include postoperative surgical restrictions, dysthyroid ophthalmopathy and blow-out fractures with entrapment [1].

Cases of head posturing in order to compensate for refractive errors were described rarely, but the mechanism is poorly understood. Havertape reported 5 patients with chin down posture. All had symmetric high hyperopia of more than +5.00 diopters, and none had strabismus. The chin down posture was present without the spectacle correction in place, and was eliminated by wearing the refractive correction [12].

Another reason for abnormal head posture is compensation for visual field deficit. Paysee and Coates described 10 children with early onset homonymous hemianopsia that assumed an ipsilateral head turn [13]. This adaptive posturing expands the useful peripheral field.

A chin up posture has been reported in children with loss of central fixation from various etiologies resulting in bilateral central scotoma. Presumably this posture is used in attempt to “over-look” the central scotoma by fixating with the superior retina [14]. Nucci and Rosenbaum described a child with blind eye that assumed a face turn toward their non-seeing side in order to center the remaining field of vision [9].

Many children with congenital ptosis, particularly bilateral, adopt a chin elevation to make better use of the ptotic eyes. This has been reported to be a good sign and to be associated with a low risk of amblyopia [15].

NON-OCULAR CAUSES OF ABNORMAL HEAD POSTURES

The non-ocular causes of abnormal head position are generally orthopedic and neurologic.

The most common orthopedic cause is congenital muscular torticollis, which may be caused by contracture or fibrosis of the sternocleidomastoid muscle causing usually a head tilt to the affected side, turn to the opposite side, and sometimes a chin elevation. Palpation often reveals a firm and contracted muscle, and it is often difficult to straighten the head [1]. If the neck muscles are extremely tight a mass can be felt in the sternocleidomastoid region, which has been referred to as pseudotumor of infancy [16, 17]. This condition is usually benign and self limiting and rarely requires neck muscle surgery.

Other orthopedic causes of AHP include the Klippel-Feil anomaly (congenital fusion of any 2 of the 7 cervical vertebrae, autosomal dominant or recessive inheritance) and brachial plexus injury [18]. Any damage to cervical vertebrae may also cause a variety of abnormal head postures.

Neurologic causes of AHP are mainly related to brain tumors, postinflammatory central nervous system conditions, psychomotor delay and focal dystonia [2].

Other less common reasons for AHP are: Sandifer syndrome (hiatal hernia associated with gastro- esophageal reflux) [19] and unilateral hearing loss, in which there is a face turn toward the side of hearing impairment [1].

IN SUMMARY

An abnormal head posture is a common condition encountered frequently by primary care family pediatricians. Few articles have attempted to evaluate the prevalence of different causes of AHP.

Nucci and Kushner evaluated 63 children with AHP. The cause was orthopedic in 35, ocular in 25, and neurologic in 5. In 8 patients no specific cause could be found. The most common orthopedic cause was congenital muscular torticollis, which accounted for 31 patients, and was also the single most common cause of an AHP in the whole series. The most common ocular cause was superior oblique muscle palsy, which accounted for 12 patients [2].

In a study by Ballock and Song, 235 out of 288 patients with AHP had congenital muscular torticollis. In the 53 patients with a nonmuscular AHP, the AHP was attributed to ocular disorders only in 12 patients [18]. Their figures, however, may be misleading as to the actual prevalence of different causes of torticollis in children, because it was done in a tertiary care orthopedic clinic. Children who obviously had ocular or neurologic problem would not have been referred to the orthopedic clinic.

Khawam studied 158 patients with ocular torticollis. 70% was caused by incomitance and 17% by nystagmus. Of the incomitance group- 39% had superior oblique palsy, 24% had Duane syndrome, 14% had lateral rectus muscle palsy, 8% had double elevator palsy, 5% had Brown syndrome and only 4 patients were considered to have AHP due to refractive errors [3].

AHP serves to improve visual acuity and/ or binocularity, and it is almost always seen in patients with functional vision in both eyes. Rarely, AHP has been reported following visual loss in one eye. Nucci and Rosenbaum reported 5 patients with acquired visual loss in one eye and associated head position. 3 patients had head tilt toward the non-seeing eye, possibly related to excyclotopia of the fixating eye. 2 presented face turn toward the fixating eye due to adduction blocking nystagmus, and 1 patient assumed a face turn to center the visual field. Different mechanisms for AHP in monocular viewing patients require careful etiological diagnosis prior to developing a surgical strategy [9].

Although the majority of children with AHP have congenital muscular torticollis, one should put in mind that the presence of tight neck muscle does not preclude the possibility of ocular cause of the AHP. In the series published by Nucci and Kushner 2 patients had muscle contracture that suggested an orthopedic cause, however, the tight neck muscles were secondary to a head tilt caused by superior oblique muscle palsy [2].

When the cause of the AHP is not obvious, a multi-disciplinary approach should be taken.

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