Ocular Abnormal Head Posture: A Literature Review

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Abstract

Purpose: To provide a comprehensive review on different characteristics of abnormal head postures (AHPs) due to different ocular causes, its measurement, and its effect on facial appearance.

Methods: In this review article, PubMed, Scopus, and Google Scholar search engines were searched for the scientific articles and books published between 1975 and September 2020 based on the keywords of this article. The selected articles were collected, summarized, classified, evaluated, and finally concluded.

Results: AHP can be caused by various ocular or nonocular diseases. The prevalence of ocular causes of AHP was reported to be 18%–25%. 1.1% of patients presenting to ophthalmology clinics has AHP. The first step in evaluating a patient with AHP is a correct differential diagnosis between nonocular and ocular sources by performing comprehensive eye examinations and ruling out other causes of orthopedic and neurological AHP. Ocular AHP occurs for a variety of reasons, the most important of which include nystagmus, superior oblique palsy, and Duane’s retraction syndrome. AHP may be an essential clinical sign for an underlying disease, which can only be appropriately treated by the accurate determination of the cause. Long-standing AHP may lead to facial asymmetry and secondary muscular and skeletal changes.

Conclusion: In conclusion, a proper differential diagnosis between nonocular and ocular causes, knowledge of the different forms of AHP and their measurement methods, accurate diagnosis of the cause, and proper and timely treatment of ocular AHP can prevent facial asymmetry and secondary muscular and skeletal changes in the patients.

Keywords: Abnormal head posture, Duane’s retraction syndrome, Facial asymmetry, Nystagmus, Superior oblique pals, Torticollis

INTRODUCTION

In visual sciences, an abnormal head position refers to any deviation of the head from its normal angle and direct position in which the head has an angle relative to the body in the vertical, horizontal, or anteroposterior axis. Abnormal head posture (AHP) is used as a clinical term and an important clinical sign in articles related to ophthalmology and visual sciences. The term torticollis is mostly used in other sciences, which is derived from two Latin words: Torque, meaning twisted and collum, meaning neck. Although torticollis is considered a nonspecific term indicating a head and neck tilt following contracture of the sternocleidomastoid muscle, the term “ocular torticollis” has been commonly used in ophthalmology and vision science since 1914.

AHP is not a diagnosis but a manifestation and a sign of an underlying disease, which may be present for no apparent reason in some patients. Ocular AHP may be congenital or acquired and can occur at any age depending on the cause of the disease. A primary care physician or an orthopedic specialist is not usually educated about the ocular causes of AHP, and when...
they examine a patient with torticollis, they are more likely to detect muscular causes. Muscular torticollis is caused by the shortening or fibrosis of the sternocleidomastoid muscle, resulting in a homolateral head tilt and turn. On the other hand, ophthalmologists may be more concerned about the ocular sources of torticollis. In addition to muscular and ocular sources, neurological, vestibular, and cosmetic causes can also be the sources of torticollis.

Although all examiners should consider all the possible causes of torticollis when evaluating these patients, it may have more than one origin and may be secondary to various causes in some of the patients. For example, one study found more than one cause in 16% of the patients with torticollis. Therefore, even if an ocular origin is confirmed for an AHP, other evaluations should be performed to rule out other neurologic and orthopedic conditions such as the shortening or fibrosis of the sternocleidomastoid muscle. In addition, ocular AHP may be due to two or more ocular causes at the same time in some patients. It is clear that a comprehensive assessment is necessary to evaluate all the possible causes of torticollis in these patients. This review was conducted to provide a comprehensive evaluation of the different manifestations of the head position in patients with ocular AHP, its measurement, and its effect on facial appearance.

Methods
In this review article, PubMed, Science Direct, Scopus, and Google Scholar search engines were searched for the scientific articles and books using the search terms: abnormal head posture, nystagmus, superior oblique palsy, Duane’s retraction syndrome, facial asymmetry, and torticollis. The books and articles that were published from 1975 to March 2020, included at least one of the keywords, and were relevant to the subject of this review study were included. More emphasis was placed on recent articles. The selected articles and valid scientific evidence were collected, summarized, classified, evaluated, and finally concluded by the first (M.R.A.) and correspondence (M.K.N.) authors. A written consent form and permission for publishing the photos was obtained from patients.

Results
The etiologies and frequency of abnormal head posture
The results of previous studies indicated a higher prevalence of nonocular sources of AHP compared to ocular causes. Ballock and Song found that 82% of the 288 patients with torticollis had congenital muscular causes, and only 18% of the causes were nonmuscular. In another multi-disciplinary study of 63 children with torticollis, an orthopedic, ophthalmologic, and neurologic cause was found in 56%, 25%, and 8% of the patients, respectively. Congenital muscular torticollis was the most common orthopedic cause, accounting for 49% of the patients (n = 31). The prevalence of congenital torticollis was 0.3%–2% in previous studies, and some studies found a prevalence of 1 in 250–300 infants, making it the third most common congenital orthopedic anomaly.

The most common complaints of patients with ocular AHP are ocular misalignment in 60%, abnormal eye movements in 3%, diplopia in 10%, and ptosis in 3% of the patients. It is difficult to determine the prevalence of ocular AHP in the general population accurately due to the lack of comprehensive studies. However, the prevalence of ocular AHP is 1.1% in patients presenting to ophthalmology clinic and 3.19% in pediatric ophthalmological practice.

As shown in Table 1, previous studies of the prevalence of ocular AHP found that superior oblique pals was the most common ocular cause. Some other studies reported nystagmus as the most common cause of AHP. According to five previous studies [Table 1], ocular AHP has more than ten main causes, including nystagmus, superior oblique palsy, A-V pattern deviation, Duane’s retraction syndrome, congenital esotropia, permitting foveal fixation (including ptosis, ocular muscle fibrosis, Moebius syndrome, and monocural lateral rectus palsy), Brown’s syndrome, inferior oblique palsy, and other rare causes (such as thyroid-associated orbitopathy, vertical and horizontal concomitant deviation, superior rectus palsy, double elevator palsy, torsional incomitance, ocular motor apraxia, spasmus nutans, astigmatism, etc.).

Abnormal head posture manifestations
The exact manifestation of AHP could be determined by direct observation from different axes. Three main manifestations of AHP could be observed: 1-pure head tilt, 2-simultaneous head tilt and face turn, and 3-pure face turn. In addition, in some patients, chin-up and chin-down could be observed alone or in combination with other main mentioned manifestations. The definition of head tilt is rotating the head around the anteroposterior axis of the skull, and head turn is the rotation of the anteroposterior axis of the skull from the normal position.

Abnormal head posture measurement
Previous studies used a goniometer (an instrument specifically designed for measuring angular differences) or computer analysis methods to measure different manifestations of AHP. For measuring any manifestation of AHP, it is necessary that the patients fixate on a target with fine detail, or it should be measured while recording best corrected visual acuity. AHP measurement methods in different manifestations are described below.

Head tilt measurement
To measure the head tilt, one arm of the goniometer should be placed perpendicular to the floor, and the other arm should be aligned with the axis of the face. The angle between the two arms represents the amount of head tilt. Another way to measure head tilt is to use computer software such as Corel Draw Graphics Suite. In this method, a picture is taken from the patient with a habitual AHP. The angle between the vertical line and the line that connects the center of the eyebrows to the center of the lip is calculated as the head tilt [Figure 1].
**Head turn measurement**

The amount of head turn is measured according to the degree of the rotation of the anteroposterior axis of the skull from the normal position. As shown in Figure 2, to measure the head turn, the patient is asked to fixate on a fine target at a distance. Next, the intersection of the two arms of the goniometer is placed on the center of the person’s skull. One arm is lined up with the visual axis, whereas the other is aligned along the anteroposterior axis of the skull. The angle between the two arms represents the amount of head turn in degrees. To measure head turn using the Corel Draw software (Corel Corporation, Ottawa, Canada) is shown in Figure 3.

**Chin-up and chin-down measurement**

To measure chin-up and chin-down, the goniometer is positioned with one arm perpendicular to the fixation target and the other aligned with the anteroposterior axis of the skull.

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**Table 1: Causes of abnormal head posture in previous studies**

| Year     | Number of patients | Nystagmus (%) | Superior oblique palsy (%) | A-V pattern deviation (%) | Duane retraction syndrome (%) | Congenital esotropia (%) | Permitting foveal fixation* (%) | Brown syndrome, n (%) | Inferior oblique palsy (%) | Cosmetic (%) | Other causes (%) |
|----------|--------------------|---------------|-----------------------------|----------------------------|-------------------------------|--------------------------|---------------------------------|---------------------|--------------------------|--------------|-----------------|
| Kushner21 | 1979               | 188           | 38 (20.2)                   | 46 (24.5)                 | 4 (2.1)                       | 31 (16.5)                | 12 (6.3)                        | 10 (5.3)                        | 6 (3.2)                   | 7            | 4 (2.1)         | 30 (15.7)     |
| Mitchell22| 1999               | 630           | 120 (19)                    | 59 (9.4)                  | 116 (18.4)                    | 46 (7.3)                 | 69 (10.9)                       | 27 (4.3)                        | 25 (4)                    | 6 (1)         | 0              | 162 (25.7)    |
| Nucci et al.3 | 2005     | 25            | 4 (16)                      | 12 (48)                   | 0                             | 6 (24)                   | 0                               | 0                   | 3 (12)                   | 0            | 0              | 0             |
| Erkan Turan et al.18 | 2017 | 29            | 7 (24.1)                    | 4 (13.8)                  | 2 (6.9)                       | 6 (20.6)                 | 0                               | 3 (9.3)                        | 1 (3.4)                   | 0            | 0              | 6 (20.7)      |
| Erkan Turan et al.23 | 2017 | 163           | 16 (9.8)                    | 55 (33.7)                 | 10 (6.1)                      | 35 (21.5)                | 0                               | 18 (11)                        | 11 (6.7)                  | 0            | 0              | 18 (11)       |
| Total    | 1035               | 185 (17.9)    | 176 (17)                    | 132 (12.7)                | 124 (12)                      | 81 (7.8)                 | 58 (5.6)                        | 46 (4.4)                       | 13 (1.2)                  | 4 (0.4)       | 216 (20.9)     |

*Ptosis, ocular muscle fibrosis, Moebius syndrome, monocular lateral rectus palsy*
The angle between the two arms shows chin elevation and depression in degrees. 28

**Ocular versus nonocular causes of abnormal head posture**

In assessing a patient with AHP, it is essential to distinguish between nonocular congenital and ocular AHP in the first step. Nonocular causes of AHP are orthopedic, neurological, vestibular, and cosmetic conditions. 5,13-17 This diagnosis is based on five main cases:

1. Congenital nonocular AHP starts in the first 6 months, and it can even manifest itself in the 1st week of life, 21 whereas ocular AHP usually develops after 18 months of age. 32
2. It is impossible or difficult to return the head to the normal position in congenital nonocular AHP, 33,34 whereas the head often returns easily to its normal position passively or voluntarily in ocular AHP. 32
3. On neck examination, muscle palpation shows stiffness of the sternocleidomastoid muscle in congenital nonocular AHT while palpation is unremarkable in ocular AHP. 35,36
4. Vision is often normal and is not affected in patients with congenital nonocular AHP, whereas in patients with ocular AHP, the strabismus angle increases when the head is straightened with force or the head is positioned in the opposite direction to the habitual AHP, and moreover, diplopia may also occur in acquired cases. 37
5. Monocular patching does not change the head position in congenital nonocular AHP while patching the paretic eye corrects the head position in ocular AHP (in acquired and recent-onset cases). 38

If AHP is eliminated by monocular patching, it has an ocular source. 39 The patch test may not correct the head position in cases of significant vision loss in the nondominant eye or in patients with nystagmus or ptosis. If AHP does not resolve in the office using this test, the next step is to ask the patient to patch one eye at home for a few hours or more and patch the other day in some other. 21 If the result still does not change the head position, before a diagnosis of a nonocular source is made for AHP, secondary skeletal and muscular changes that may develop following a long-term ocular AHP should be examined. 32

As mentioned earlier, there are many ocular AHP causes; therefore, this study evaluated the characteristics of AHP in the three main causes of ocular AHP, including nystagmus, superior oblique palsy, and Duane’s retraction syndrome, which account for about half of the ocular AHP causes, and then discussed the characteristics of AHP in other ocular causes.

**Abnormal head posture in patients with superior oblique palsy**

Previous studies found that superior oblique palsy was the most common type of cyclovertical muscle palsy with a prevalence of about 40%. 40-43 Superior oblique palsy is diagnosed based on the presence of hypertropia in the primary position, which usually increases in ipsilateral head tilt and on contralateral gaze. 44 These patients habitually hold their heads away from the paretic muscle position. Although congenital patients are often asymptomatic, the most common symptoms in acquired superior oblique palsy include asthenopia, diplopia, and image tilting. 35,46 According to Figure 4, these patients tilt and turn their heads to the contralateral side of the palsy. Therefore, the affected eye moves away from the field of action of the paralyzed muscle and establishes a normal fusion by eliminating diplopia. 47

Although AHP is expected to manifest as a simultaneous head tilt and turn to the contralateral side of the palsy with chin-down in these patients, it may present as a pure head tilt or pure head turn alone in some cases. Erkan Turan et al. found that AHP manifested as head tilt in 87%, pure turn in 3.6%, combined head tilt and turn in 7.3%, and chin-down in only 1.8% of the superior oblique palsy patients. 23 Nucci et al. found AHP in 12 patients with superior oblique palsy, ten patients had head tilt, and only two had a combination of head tilt and turn. 48 Based on the results of previous studies, AHP is often seen as head tilt to the contralateral side of palsy without chin-down in these patients. 23,48

Many patients with superior oblique palsy who have the potential for bifoveal fusion may adjust the amount of their AHP to control the deviation easily by fusional vergences. 11 Therefore, the severity of head tilt in these patients manifests itself in various degrees [Figure 5].

In these patients, although AHP may be resolved by monocular patching of the eye with paralyzed muscle, AHP may not change with the dominant eye patching (even in acquired and recent-onset superior oblique palsy). The main reason for the presence of head tilt in superior oblique palsy patients is to decrease the vertical deviation. 11,49

Another point is that in some superior oblique palsy patients, the degree of vertical diplopia and the strabismus angle is so high that they cannot overcome them even with assuming a severe AHP. This diplopia causes confusion for the patient. As a result, AHP may be seen in these patients in contrast to what is expected in superior oblique palsy patients so that the patients eliminated confusion by further dissociation of the two images. This form of AHP in superior oblique palsy patients is called paradoxical AHP. 41,50-52 Von Noorden reported a prevalence rate of 3.4% for paradoxical head tilt in patients with superior oblique palsy. 43

Although the most common manifestation of AHP in unilateral cases was reported as head tilt to the contralateral side of the eye with muscle palsy, AHP in patients with bilateral superior oblique palsy manifested only in a chin-down position. 53 The presence of chin-down rather than a head tilt posture was reported as one of the most important clinical sign of bilateral superior oblique palsy. 54

**Abnormal head posture in patients with Duane’s retraction syndrome**

Duane’s retraction syndrome is a congenital syndrome mostly characterized by limited abduction or adduction, palpebral fissure narrowing, globe retraction on adduction, and upshot or
down shoot of the affected eye on adduction.\textsuperscript{53} This syndrome is found in 1\%–4\% of the strabismic patients. Some researchers suggest that Duane’s retraction syndrome should be divided into three subtypes, including esotropic, exotropic, and orthotropic, based on the deviation in the primary position.\textsuperscript{56,57}

Given the horizontal eye movement defects, AHP manifests as a pure head turn in these patients. This compensatory mechanism helps the patients to prevent diplopia and obtain a binocular single vision. Esotropic Duane’s retraction syndrome patients, due to abduction limitations, turn their heads to the same side of the affected eye [Figure 6] while the head turn is to the opposite side in exotropic Duane’s retraction syndrome patients that have adduction limitation [Figure 7].\textsuperscript{58} However, there may be no head turn in some patients with unilateral Duane’s retraction syndrome.\textsuperscript{57,59}

Abnormal head posture in patients with nystagmus

Nystagmus is one of the main ocular reasons for AHP development which can be manifested as head tilt, face turn, chin-down, and chin-up.\textsuperscript{5,18,21-23,25} The prevalence of AHP in infantile nystagmus was reported 73\% in Abadi and Bjerre study\textsuperscript{60} and 94\% in Spielmann’s study.\textsuperscript{61}

In some patients with nystagmus, the amplitude and frequency of nystagmus are reduced at an extreme gaze (known as the null point), resulting in optimal visual acuity.\textsuperscript{62} These patients change the head position form the normal primary position; therefore, when they look straight ahead, they place their eyes in the optimal position. The direction of the AHP is in the opposite direction to the null point that the amplitude and frequency of the nystagmus decrease.\textsuperscript{63} If the null point of these patients is close to the primary position or they have a similar visual benefit in different gazes, the patient will not have AHP; however, AHP may develop if the null point of these patients is far from the primary position.\textsuperscript{25}

Some other nystagmus patients change the head posture in a way that helps them to converge the eye because the convergence will damp the nystagmus.\textsuperscript{64} Another cause of AHP is periodic alternating nystagmus which consists of horizontal nystagmus that cyclically reverses its direction.\textsuperscript{65} The prevalence of AHP in periodic alternating nystagmus is found in up to 95\% of patients.\textsuperscript{25} These patients may also
demonstrate periodic alternating head position to minimize the nystagmus; therefore, they should be observed for an extended time to rule out alternating head position from the constant head position. Patients with supranuclear disorders may also have nystagmus. Some of these patients also need to adopt an AHP to either fixate or achieve binocularity.

In some other cases, the severity of nystagmus is not so high to be considered the source of AHP. In such cases, binocular visual acuity should be measured once when the patient’s head is straight and once in the AHP position. Visual acuity improvement in AHP is a strong indication that AHP is caused by the presence of a null point in nystagmus. However, this is not always correct since a particular gaze may cause visual benefit in these patients, which does not necessarily mean improved visual acuity. It may take the patients a longer time to maintain an image in the fovea; this prolonged foveation time may lead to improved visual efficiency, such as more comfortable and higher reading speed, which cannot be detected by routine ophthalmic tests. Therefore, although nystagmus damping and increased visual acuity may be reported in the abnormal head position in most patients, lack of visual acuity improvement in the abnormal head position compared to the straight head position cannot rule out nystagmus as a cause for AHP. Another important point to note is that AHP is not coincident with the null point in 30% of the patients with nystagmus.

Attention should be paid that nystagmus patients do not usually have a constant AHP. AHP often tends to increase in intensity when the patient is focusing on fine details such as looking at the blackboard or watching television, and at other times, these cases do not have significant AHP. The most severity of AHP is often observed when the patient is making a maximum effort to fixate on distant target, but in near vision, the severity of AHP is considerably less than distant vision. This distance-near disparity in AHP is seen in 70% of the patients.

Other causes of ocular abnormal head posture

In addition to the above sources, other sources of ocular AHP, although less prevalent, include A-V pattern deviation, congenital esotropia, ptosis, ocular muscle fibrosis, Moebius syndrome, monocular lateral rectus palsy, Brown’s syndrome, inferior oblique palsy, superior rectus palsy, double elevator palsy, torsional incomitance, ocular motor apraxia, spasmus nutans, and astigmatism. AHP may manifest in various forms such as head tilt, head turn, chin-down and chin-up, or a combination of these forms. Most of the patients who have one of the above-mentioned etiologies of AHP habitually position their heads to avoid the field of action of the defective muscle, eliminate diplopia, and achieve a binocular single vision. Although the head position does not precisely match what is expected in all cases, the manifestations and the position of the head in these ocular causes are shown in Table 2. Chin-up position is a common posture in patients with bilateral ptosis including blepharophimosis syndrome.

Previous studies reported that 29%–73% of patients with lateral rectus palsy, 50% of patients with third nerve paresis, and 58% of patients with Brown syndrome had an AHP. In Brown syndrome, the most common type of AHP was chin-up; however, in some cases, contralateral head turn combined with chin-up was observed. In most of the patients with lateral rectus palsy, due to abduction limitations, the face turns toward the side of the eye with muscle palsy. This compensatory mechanism helps the patients to prevent diplopia and obtain a binocular single vision. The most common manifestation of AHP in patients with inferior oblique palsy was reported as a head tilt toward the side of the eye with muscle palsy and head turn away from the side of the eye with muscle palsy. As shown in Table 2, it is important to know that in some causes of AHP such as unilateral superior rectus palsy and inferior rectus palsy, both ipsilateral and contralateral head tilt might be observed. These findings are in contrast with the result of Bielschowsky three-step test for the diagnosis of these conditions; therefore, this test is not valuable in the diagnosis of unilateral superior rectus palsy and inferior rectus palsy.

The frequency of AHP in patients with dissociated vertical deviation, who had fixation preference, has been observed in 35%. The exact manifestation of dissociated vertical deviation is a controversial subject. Santiago and Rosenbaum found 12 of 14 patients with dissociated vertical deviation had contralateral head tilt. Nevertheless, in most patients in another study, increasing the size of the dissociated vertical deviation was observed on forced contralateral head tilt, and the severity of dissociated vertical deviation tends to decrease in ipsilateral head tilt.

Another essential point to keep in mind is that in many patients with incomitant strabismus, AHP may only reduce the strabismus angle to the extent that peripheral fusion can be established (usually ten prisms or less). This is especially common in patients with “A” or “V” pattern strabismus and is associated with subjective benefits that patients understand from peripheral fusion. Therefore, incomitant strabismus may cause AHP only by reducing the angle and not by eliminating manifest strabismus. Some people also assume an AHP only for cosmetic reasons, including (1) to hide the conjunctival scar on one side of an eye that is esthetically acceptable to the patient, (2) to make incomitant strabismus less noticeable (this AHP does not have any effects on the central or peripheral fusion), and (3) to look attractive. Visual field defect and uncorrected refractive errors with a prevalence of <1% were also reported as fewer common causes of AHP. In patients with visual field defect, the head turns toward the defected side with a gaze preference contralateral to the visual field defect.

Abnormal head posture with more than one ocular cause

It should be noted that AHP might have more than one ocular source in some cases with the most common being coexisting nystagmus with null point and incomitant strabismus. In such cases, before any treatment, the effect of each factor on the
AHP development should be examined. Obviously, the surgical treatment protocols are quite different in nystagmus with AHP and incomitant strabismus. The best method to determine the exact cause or contribution of each factor in the development of AHP in this situation is to use a prism. For example, in a patient with V-pattern esotropia who has nystagmus with a null point in the up gaze, if base-down prisms placed in front of both eyes can eliminate AHP, the source of AHP is nystagmus, and surgical intervention using the vertical Kestenbaum-Anderson method can change the null point and correct AHP. However, if base-out prisms eliminate the AHP, the source is V-pattern esotropia, and esotropia surgery eliminates AHP. Therefore, when there is more than one ocular cause for AHP, prisms can be used to determine the exact cause of AHP (at the office using trial prisms or at home using Fresnel prisms).\(^1\)

**Facial asymmetry due to long-standing abnormal head posture**

One of the main reasons for early treatment of ocular AHP is to prevent secondary muscular and skeletal changes and facial asymmetry.\(^8\) AHP may affect normal facial development, and previous studies have shown that most of the patients with congenital AHP have facial asymmetry.\(^2,9,30,83\)

Long-standing AHP may affect the symmetricity of the neck,\(^2\) face, cheeks, nostrils, nasal tip, and columella.\(^29\) The effect of AHP on the facial appearance depends on the type and severity of AHP.\(^2,9,30,81-83\) In patients with a pure head tilt such as superior oblique palsy patients, columella is diverted to the same side of the head tilt, and nostril compression is more obvious on the opposite side [Figure 9].\(^29,83,84\) The main reason for these changes is the effect of gravity on the size of nostrils, causes widening of one of the nostrils on the same side of the head tilt and compression of the other nostril. However, gravity applies the same force to both sides of the face in patients with pure head turn.\(^30\)

A previous study by the authors showed that facial asymmetry was not necessarily an abnormality, and different types of facial asymmetry could be seen in orthotropic participants.\(^30\) However, the frequency of different types of facial asymmetry in patients with AHP is significantly higher compared to orthotropic participants.\(^29,30,82,83\) All quantitative and qualitative facial asymmetry parameters have a high frequency in patients with ocular AHP compared to orthotropic participants.\(^29,30,82,83\) Early strabismus surgery for correcting the AHP may avoid facial asymmetry, but ensuring that the infant sleeps with alternating head positions may be more important.\(^8\)

**DISCUSSION**

The first step in evaluating a patient with AHP is a correct differential diagnosis between nonocular and ocular sources by performing comprehensive eye examinations and ruling out other causes of AHP such as orthopedic and neurological conditions. AHP may be an essential clinical sign for an underlying disease, which can only be appropriately treated by the accurate determination of the cause, and long-standing
AHP may lead to facial asymmetry and secondary muscular and skeletal changes.\textsuperscript{38}

In conclusion, AHP can be caused by various ocular or nonocular diseases. Ocular AHP is an essential clinical sign for an underlying disease, with the most important causes being nystagmus, superior oblique palsy, and Duane’s retraction syndrome. The accurate diagnosis of the cause of ocular AHP and timely treatment can prevent the development of facial asymmetry and secondary muscular and skeletal changes in these patients.

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There are no conflicts of interest.

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