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Abnormal Head Posture and The Relationship Between Vision and Posture: The Role of the Optometrist

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Abstract

Purpose: Abnormal head posture (AHP) of ocular origin is a postural adaptation, also referred to as ocular torticollis, which occurs as a result of paresis or paralysis of the extraocular musculature. Binocular vision anomalies or visual disturbances can also establish AHP, which is intended to maintain single binocular vision. In the presence of a suspected or confirmed imbalance of the visual system, healthcare professionals from various disciplines, who are involved in postural assessment and rehabilitation, may request an assessment from the optometrist to determine whether or not the AHP is of ocular origin.

Methods: This review article examined the existing literature using electronic databases: PubMed, ResearchGate, ScienceDirect, and Google Scholar

Results: AHP can result from a variety of oculomotor disorders and be caused by eye-related or unrelated diseases. An estimated 1.1% of patients in ophthalmology clinics suffer from AHP, with 18% to 23% of cases having an ocular origin. The optometrist must first differentiate, through in-depth ocular examinations, between AHP of ocular or non-ocular origin, excluding other potential postural causes (orthopedic and neurological).

Conclusion: Understanding the distinction between ocular and non-ocular causes of AHP is essential for effective management. Collaboration among healthcare professionals, especially in posturology, is necessary to improve clinical outcomes. Establishing clear communication protocols will facilitate integrated management strategies. Promoting dialogue and continuous professional development is also important to meet the diverse needs of patients.

Keywords: Abnormal Head Posture; AHP of Ocular Origin; Ocular Torticollis; Postural Analysis; Abnormal Posture; Posturology; Strabismus; Anomalies of Binocular Vision

Abbreviations: AHP: Abnormal Head Posture; EOM: Extraocular Muscles; TPS: Tonic Postural System; CNS: Central Nervous System; RIO: Right Inferior Oblique; RSO: Right Superior Oblique; SO: Superior Oblique; IO: Inferior Oblique; IR: Inferior Rectus; SR: Superior Rectus; LR: Lateral Rectus; MR: Medial Rectus; APA: American Psychological Association

Introduction

The abnormal head posture (AHP) of ocular origin [1-4] or "ocular torticollis" [5], is a postural adaptation that is established with the aim of maintaining single binocular vision as a result of paresis or paralysis of the extraocular muscles (EOM) or in the presence of anomalies of binocular vision or visual disturbances. In the AHP, the position of the head presents an irregular inclination in relation to its normal position, that is, relative to the rest of the body along the vertical, horizontal, or antero-posterior axes. Many studies have demonstrated the relationship between vision and posture, and how they influence each other. Harmon [6], in his studies conducted in the 1950s, showed how vision and posture can influence one another, particularly in a school environment. He observed that astigmatists tended to tilt their heads laterally or occasionally rotate them, with the degree of inclination related to the axis of their astigmatism. Myopic and exophoric subjects typically tilted their heads backwards, accompanied by an upward rotation of the shoulder blades. Conversely, hyperopic and esophoric subjects usually tilted their heads forward, resulting in a downward rotation of the shoulder blades. Anisometropic subjects were more likely to rotate their heads in the horizontal plane. Furthermore, Harmon noted that the rachis was subjected to torsions and compressions on the intervertebral discs, which severely limited the freedom of movement. In his studies on proximal activities at school desks, Harmon theorized the existence of a concept known as the "Harmon distance" [6-9]. This distance is thought to reduce visual fatigue associated with tasks such as reading or writing at close range. Additionally, it

minimizes the energy expenditure required by the muscles to support the weight of the head and torso against gravity. Harmon further suggested that this distance corresponds to the placement of the head from the desk when the hand is closed in a fist and the elbow is resting on the work surface.

Clearly nowadays, we rarely use electronic devices at the distance theorized by Harmon. Therefore, if an incorrect posture and an AHP are maintained for a long period, anomalies of binocular vision can be determined with an alteration of the accommodative function and the appearance of phorias, which could alter the refractive state and the balance of the visual system [7,10]. Furthermore, if a subject is affected by an uncorrected or poorly compensated refractive error, evident or permanent postural imbalances and asthenopia may occur [7,10]. Finally, to further and more clearly highlight the role of visual function in posture [11,12], other studies have compared the oscillations of the body in an upright position, with eyes open and closed, with the posturographic examination in subjects with normal vision, visually impaired vision, and blindness, determining a detectable increase in oscillations and therefore a worsening in the precision of postural control of approximately 250% when the eyes were closed [11].

Tonic Postural System (TPS)

Our posture is mainly influenced by the gravitational force, which we must counteract at every moment. Just think of the vestibular system and how its receptors work in response to the action of the gravitational force. Therefore, when we find ourselves in conditions of reduced or absent gravity, such as in water or in space, our posture changes and we assume a different position than when we are subject to the terrestrial force [10]. To cope with this force and be able to maintain an upright position, stably and in balance, our body uses the TPS [10,13,14] made up of receptors (inner ear, eye, mouth, joints, muscles, foot, skin), which send information from the periphery to the hierarchically connected higher nervous centers, which, through the contraction of agonist and antagonist muscle groups, modify the posture in order to face any variation that tries to alter it, moment by moment. This is a system with multiple inputs and many functions. We can also consider the TPS as a self-regulated cybernetic system [10] where the receptors send an incoming afferent signal (input), which is processed by the central nervous system (CNS). In order to maintain the body in balance through complex postural reflexes, the CNS starts a response (output) by means of the complex myofascial system (muscular and connective), which involves the various myofascial chains or muscular chains.

Let us now analyze these three components.

I) The incoming afferent signal, or input, comes from exteroceptors and proprioceptors from the three main sensory systems: the Visual System (or exteroceptive), the Vestibular System (or exteroceptive), and the Somatosensory System (or proprioceptive). The Somatosensory System provides a set of information about our body (joint angles, muscle tension, load on the skeleton), with the aim of creating a geometric reference scheme, or a conscious internal representation of its conditions and orientation in space. It also helps in planning movements through motor control and in maintaining a stable upright position. Proprioception is therefore the conscious perception of the position of the body and the movement of one's limbs (kinesthesia) in space, independently of sight [15]. The receptors with proprioceptive function are located in the muscles and joints [15] and consist of three types of mechanoreceptors: muscle spindles, Golgi tendon receptors, and joint receptors.

II) The central nervous system is like a computer. It generates the activation of the muscle chains appropriate for maintaining the desired posture, sends commands through the musculo-osteo-articular system, and, through the real-time integration of the various systems, ensures that the human body is able to implement compensation strategies to maintain the upright position.

III) The responses or outputs of the efferent pathways can be both automatic and voluntary and serve the purpose of organizing the myofascial chains based on the needs of the movement to be performed. The myofascial chains are muscle groups connected by a system of fascia (connective tissue; [10]) and are also referred to as "muscle chains," which participate in a common action to achieve movement in a given direction through flexions, extensions, adductions, abductions, rotations, etc. Numerous authors have studied myofascial (or muscular) chains, and among the best known are those of Bousquet [16,17]:

a) Flexion chain or Anterior chain.

b) Extension chain or Posterior chain.

c) Anterior crossed chain or Closing chain.

d) Posterior crossed chain or Opening chain.

e) Posterior static chain (with an anti-gravity function).

A balanced posture is one that allows maximum effectiveness of the gesture in the absence of pain, in maximum energy economy, and where all forces acting on the body are balanced. In clinical practice, we may encounter patients who present an AHP, and we must differentiate through an optometric evaluation between one determined by ocular or non-ocular causes, excluding other potential postural sources (orthopedic and neurological). Let's now take a look at what a postural analysis is and what syndromes are associated with it that prompt posture healthcare professionals to send a subject for an optometric check-up.

Postural Assessment and Its Misalignment

In functional postural analysis, the first step is to observe the patient, evaluating the alignment of the body in an upright position in relation to an ideal posture. The ideal upright posture,

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in the absence of pathology, refers to an anatomical posture where the body is upright, the face is facing forward, and the upper limbs are in line with the hips. Therefore, by referencing the various planes and axes recognized at an anatomical and morpho structural level, we compare them on different observation planes, using fundamental body landmarks to draw a virtual horizontal line that intersects them [13,14]. Then we will look at the symmetry and alignment of this, because the lines must be horizontal and parallel to each other. Starting in a cranio-caudal direction, we will evaluate the alignment on the: anterior frontal plane, posterior frontal plane, sagittal or lateral plane, transverse or horizontal plane. In the postural assessment on the posterior frontal plane, through a plumb line that goes from the skull down to the intergluteal line describing a virtual line perpendicular to the floor and to be used as a reference, any misalignments to the right or left can be highlighted. This observational evaluation is also called the "Barré's Vertical Test" [18,19]. With the subject standing, still and relaxed, holding the arms along the body and looking forward (at eye level), it is possible to assess through the balance ratio between the various body landmarks, whether the virtual line or Barré's vertical joins the following landmarks on the posterior frontal plane:

- The Occiput or Vertex (head).
- C7.
- L3 or L4 for other posturology specialists.
- The Intergluteal Line.
- The "half of the intermalleolar distance" or space between the heels.

In an ideal or correct physiological posture, a positional relationship is established between these points, which leads them to be all aligned with each other and arranged along the Barré's vertical. Thanks to this evaluation it is possible to understand whether the problem encountered is "causative" or "adaptive" of the postural imbalance [18,19]. If the reference points are not aligned along the Barré's vertical, but are arranged laterally to its right or left, then we speak of ascending, descending, mixed or disharmonic syndrome according to the following scheme [18]:

Ascending Syndrome: if C7 and Occiput are on the line, intergluteal line and L3 are on the right or left. The source of the postural imbalance must be sought below the center of gravity: probably on the podalic receptor; or for structural alterations of the lower limbs (e.g. foot, knee) or of the sacral triangle (e.g. sacroiliac, iliopsoas) [20].

Descending Syndrome: intergluteal line and L3 are on the line, C7 and Occiput are on the right or left. The source of the postural imbalance must be sought above the center of gravity: probably on the oculomotor or stomatongatic receptor; above the cervical district [20].

Mixed Syndrome: only one of the points is on the line, all the others are on the right or left. For example, L3 is on the thread, the intergluteal line is on the right and C7 and Occiput are on the left. The source of the postural imbalance can be due to either an alteration of the upper or lower part of the center of gravity; that is, either an alteration of the foot receptor or an alteration of the oculomotor or stomatongatic receptor.

Disharmonic Syndrome: none of the points are on the thread. They are all either on the right or left of the thread. The source depends on problems other than those described above and can be, for example: a trauma such as whiplash, toxic scars, psychosomatic traumas, neuro-vestibular disorders, neurological disorders, etc.

Therefore, the misalignment of one of the body segments caused by a disturbance, by an alteration in that segment, determines a cascade upstream or downstream, a repositioning of the other segments in order to maintain an erect posture, not only on the posterior frontal plane but also on the other planes (sagittal, anterior frontal and transverse) in a more or less evident way.

Generally, it is precisely the subjects affected by a Descending Syndrome, those who are sent to perform an optometric evaluation to differentiate between an AHP of ocular origin from one of a different origin. And during the optometric examination, one must differentiate the AHP as a consequence of an oculomotor alteration from one of a different origin, optical or of the neck muscles, in order to implement the correct therapy.

Usually, the family doctor or a specialist who deals with posture are not accomplished on the causes that determine an AHP of ocular origin. Likewise, due to his academic training, the optometrist focuses mainly on the ocular causes that can determine an AHP. However, it must be considered that, in addition to muscular and ocular sources, vestibular, neurological causes or alterations in other anatomical districts can also be responsible for torticollis.

Causes of Oculomotor Alterations

Whether latent or manifest, ocular deviations present common etiopathogenetic factors and the onset of one form or another depends on the cause and the fusional motor capacities of the EOM [2,3,21,22]. The task of the oculomotor system is to direct the gaze towards the target and maintain fixation and binocular vision on the target reached. In order to maintain a single binocular vision, it is necessary that:

- The images reach corresponding retinal points and have the same size.
- The brain is able to transform the visual impulse into a single visual perception.

Let us now try to describe generically what are the most

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common causes that can determine an oculomotor alteration [2,3,21,22].

Optical Causes: refractive errors can determine an alteration in the perception of images and/or the AC/A ratio. For example, when an uncorrected hyperopic patient exerts excessive accommodation and consequently excessive accommodative convergence to bring the image into focus and obtain single binocular vision, excess convergence may develop. If the latter is not adequately compensated by motor fusion skills, it may lead to the appearance of accommodative esotropia. It is therefore essential to compensate for the various refractive errors with adequate dioptric correction, through the prescription of glasses, in order to restore the normal AC/A ratio and maintain normal motor fusion skills both in convergence and divergence.

Sensory Causes: any functional or organic alteration, especially if it acts for a long time, of the ocular bulb and the visual pathways, which causes a distortion of the images or a significant difference in their size, produces an inability to fuse images, and can determine the onset of a strabismus in convergence or divergence. It is a defense mechanism that our organism adopts to avoid visual confusion or diplopia, which is established due to one of these alterations.

Anatomical Causes: alterations in the orbital plane, its size, or its direction can cause strabismus. Anomalies in the insertions of the EOM or their size. Or tendon anomalies can cause excessive adduction or abduction with consequent ocular deviation. Craniofacial dysostosis.

Paretic Causes: the causes of the various forms of oculomotor paresis-paralysis can be due to a multitude of etiological factors and whatever the etiology, there is a common semeiology between the various forms.

A) They can be caused by an alteration or a primitive lesion [2]:

- in the muscle, in the muscle fiber or in the motor plate (and are defined as myopathies).
- In the neuromuscular junction (myasthenia).
- In the peripheral nerve, and here is the paralysis of the nerve.
- Of the nucleus or trunk of the oculomotor nerve (and in this case they are defined as neurogenic).
- Supranuclear (lesions of the conjugated motricity).

B) In the acquired forms, the causes may be traumatic, compressive, infectious, vascular, inflammatory, toxic (rare), metabolic, or from CNS alterations [2].

Assessment of Ocular Deviation

The assessment of the EOM is performed in the nine gaze positions to ascertain any Hypo or Hyper functioning of the extraocular musculature through the alignment of the visual axis with the muscular plane so as to test a single muscle per eye in each diagnostic position. Therefore, considering the action of the muscles according to the law [2,3,21,22]:

- Of Sherrington's reciprocal innervation, which states that "when a muscle contracts, its antagonist relaxes".
- Of the equal innervation of Hering, which states that "when an eye movement is made, the same innervation impulses are sent to both eyes", or rather that "the contraction (or relaxation) of a muscle in one eye corresponds to a similar innervation of the contralateral synergist".

When we go to evaluate the EOM in the presence of a paresis or paralysis, the evolution of the oculomotor paralysis will follow the following trend [2,5]:

1. The hypofunction of the involved muscle is followed by the hyperfunction of the homolateral antagonist muscle.
2. Then the hyperfunction of the contralateral synergist (of the involved muscle), with the hypofunction of the contralateral antagonist muscle.

To all of this, we must add that if we measure the ocular deviation, a difference will be observed between:

- Primary deviation, which occurs when fixating with the healthy eye.
- Secondary deviation, which occurs when fixating with the paretic eye, where the deviation is always greater than the primary deviation.

According to Hering's law, the secondary deviation will be greater than the primary deviation because a greater amount of nerve impulses will reach the eye affected by the lesion (to maintain fixation in the primary position). Consequently, receiving a greater impulse, the contralateral conjugate or synergistic muscle will contract more intensely, resulting in a greater ocular deviation. Therefore, in the various forms of oculomotor paresis-paralysis, depending on whether the fixation is in the healthy eye or the paretic eye, we can have an increase in the deviation in primary position, which will further complicate the innervational sequence between the ipsilateral and contralateral agonist muscles and their corresponding synergists.

After what has been described, it appears clear that knowing the sequence of muscle variations is important:

- To understand how, through the linked evolution of paralysis in EOM (frequent in congenital or infantile paralysis), one passes from a deviation with a variable deviation angle or incomitant form, to one with a constant deviation angle or concomitant form.
- To understand and analyze EOM, during objective and subjective evaluations.

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- To understand the AHP of ocular origin.
- To establish an effective and appropriate surgical therapy, when one decides to intervene.

In the presence of an ocular deviation, the tests generally used for measuring the angle are of two types [22]:

I) Objective tests, in which the patient's cooperation is reduced to the sole ability to fix and are:

- The Cover Test: which allows to differentiate between the presence of a phoria, from a tropia.
- The Prism Cover Test: with which using prisms the ocular deviation is quantified.
- The Synoptophore Measurements.
- The Hirschberg and the Krimsky Test: which are two tests based on the observation of the corneal light reflex (with respect to the pupillary center) and its realignment on the center of the pupil by means of the anteposition of prisms.

II) Subjective tests: in which the patient's response determines the results, which can be divided into tests:

A. Diplopic, based on the phenomenon of diplopia, such as:

- The red glass test, where the hypo functioning muscle, in which the lesion is located, is the one corresponding to the eye muscle that, in the diagnostic gaze positions, perceives the light furthest from its body.
- Maddox cross and Maddox wing, based on homonymous and crossed diplopia.

B. Haploscopic, based on retinal disparity, image separation and dissociation (obtained using red-green colored filters or polarized filters, as in the TNO test or the Randot Stereotest which evaluate stereopsis).

Among the haploscopic tests, the most important is the Hess-Lancaster Screen Test, for the study of the EOM, which graphically shows the hypofunctions and hyperfunctions of each muscle of the two eyes. The retinal correspondence must be normal, otherwise the graph and the resulting deviation do not correspond to the objective deviation. When examining the graph data, one looks for [2,5]:

1. the smallest diagram, given by the narrowing of the paretic muscle
2. On the same pattern, its enlargement in the direction of the homolateral antagonist.
3. In the larger diagram, however, it will be observed that the field of the synergist muscle contralateral to the paretic is enlarged
4. and that of the antagonist muscle contralateral is narrowed

During the inspection, when you are faced with a patient with an AHP, it is essential to first understand if it is an anomaly related to the skeletal system, muscular, or if it is due to an abnormal position related to an ocular problem. To do this, you can perform a simple test that consists of making the patient move the neck passively [3].

- If passive mobility is normal, then the AHP is not caused by motor or skeletal abnormalities.
- If stiffness or limitations in mobility are found, then it could be indicative of muscular or skeletal problems (of the neck), which could be the basis of the patient's AHP condition.

Another simple test is the occlusion of one eye, which allows us to observe how in acquired oculomotor palsies the head tends to straighten involuntarily, while in congenital ones the abnormal position of the head persists. In cases of AHP of non-ocular origin, the head position maintains its abnormal position [2].

Types of Abnormal Head Positions

AHP of ocular origin is a postural adaptation acquired, following paresis or paralysis, with the aim of [1-4]:

- Trying to reduce or avoid diplopia.
- Allowing to maintain single binocular vision in at least one or more gaze positions.
- Trying to bring the eyes outside the field of action of the deficient muscle or muscles, since the diplopia disappears or is reduced in the opposite direction to the field of action of the deficient or paralysed muscle

Therefore, the patient mainly assumes 3 types of AHP, individually or in combination. Let's imagine that our subject's head is positioned at the center of a Cartesian reference system where the vertical axis corresponds to the Y axis, the horizontal axis to the X axis, and the antero-posterior axis to the Z axis. We will have:

Horizontal Rotation: It occurs when a muscle with horizontal action is affected. In this case, the head rotates around the vertical axis (Y axis) towards the patient's right or left shoulder. This condition is commonly called "Face turn". For example, if it is affected:

- The Lateral Rectus (LR), the head rotates towards the right shoulder.
- The Medial Rectus (MR), the head rotates towards the left shoulder.

Vertical Rotation: It occurs when a muscle with vertical action is affected. In this case, the head rotates around the horizontal axis (X axis). For example:

- Upwards, with the chin raised or "chin-up", if a levator muscle has been affected, such as the Superior Rectus (SR) or the

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Inferior Oblique (IO).

- Downward, with the chin lowered or "chin-down", if a depressor muscle has been affected, such as the Inferior Rectus (IR) or Superior Oblique (SO).

Inclination in the Frontal Plane: This occurs when a muscle with torsional action is affected. In this case, the head rotates around the antero-posterior axis (Z-axis). This condition is commonly called "Head tilt". For example, if it is affected:

- The Right Superior Oblique (RSO), the head is tilted towards the left shoulder, i.e., on the opposite side of the affected eye

- The Right Inferior Oblique (RIO), the head is tilted towards the right shoulder, i.e., on the same side as the affected eye.

In Table 1 we see a summary of the abnormal head postures that will be assumed in the primary gaze position in the presence of a muscular deficit [1-4]. We have seen how the AHP is established to compensate for a motor deficit, from paresis-paralysis of the EOM. However, it can also be established for the stabilization of a nystagmus and the head will then position itself so that the eyes reach the "null point" [3]. To reach a position or area in which the nystagmus is significantly reduced, the patient assumes an AHP in the "null point" or "neutral zone", generally positioning himself with his gaze on the opposite side to where the nystagmus "beats", in other words where the rapid phase is directed. For example, if a nystagmus beats to the right (rapid phase towards the right), that is, it increases its oscillations in the right gaze position and improves in the left gaze position, the subject will present an AHP with the head rotated towards his right shoulder and a preference of the gaze position to the left.

Table 1: Abnormal head posture assumed in primary position in the presence of muscular deficit.

Muscle Paralysed	Face Turn	Head Tilt	Chin
RLR	right	-	-
RMR	left	-	-
RSR	right	right	elevation
RIR	right	left	depression
RSO	left	left	depression
RIO	left	right	elevation
LLR	left	-	-
LMR	right	-	-
LSR	left	left	elevation
LIR	left	right	depression
LSO	right	right	depression
LIO	right	left	elevation

Methods

The aim of this research is to analyze abnormal head posture (AHP) of ocular origin as a postural adaptation acquired as a result of binocular vision anomalies. The study explores how these anomalies may be related to postural problems, examines the definition and assessment of posture, and delves into the concept of the postural tonic system. Additionally, it seeks to understand the motivation behind referrals from health professionals dealing with posture to optometrists for evaluating AHP and describes the clinical tests most commonly used to assess ocular deviations. This investigation adopted a systematic review methodology following the PRISMA guidelines. Relevant studies were identified through international journals, books, and reputable online sources, providing valuable insights into the topic. The search was conducted using electronic databases such as PubMed, ResearchGate, ScienceDirect, Google Books, and Google Scholar, including studies published up to November 2024. No restrictions were applied to the language of the articles to ensure comprehensive coverage. Given the specific nature of the topic and the limited availability of comprehensive studies on the prevalence and management of AHP of ocular origin in the general population, no strict exclusion criteria were applied.

The inclusion criteria consisted of studies focusing on AHP of ocular origin, research examining binocular vision anomalies affecting posture, and studies that included assessments or treatments relevant to optometry or posturology. In contrast, exclusion criteria included studies unrelated to AHP or its ocular origins, editorials or reviews lacking sufficient methodological details, and articles without specific data on postural analysis or optometric interventions. The keywords and their combinations included: abnormal head posture, AHP of ocular origin, ocular torticollis, postural analysis, abnormal posture, posturology,

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strabismus, and anomalies of binocular vision. These terms were carefully selected to ensure a thorough exploration of the subject matter. In the search, a total of 482 articles were identified and, after removal of duplicates, 67 unique articles remained. Of these, 37 were evaluated in full-text and 25 were included in the literature review. In addition, 23 textbooks were reviewed, which provided additional reliable and meaningful sources for further study. The literature review adhered to the formatting and structural guidelines outlined by the American Psychological Association (APA).

Results

The relationship between oculomotor and postural defects has been extensively studied, with variations observed in the prevalence, causes, and associated anomalies of abnormal head posture (AHP). The Table 2 summarizes the key findings from multiple studies, comparing study designs, number of participants, results, and the primary causes of AHP. The studies indicate that AHP arises from both ocular and non-ocular causes, with some variation in the reported prevalence and causes across different clinical settings.

Table 2: Summary of Studies on Abnormal Head Posture (AHP): Prevalence, Causes, and Results.

Author(s)	Type of Study	Number of Participants	Results and Prevalence of AHP	Main Causes of AHP	Conclusions
Richards et al. [29]	Retrospective Study	4,373 cases	Prevalence of palsies: VI 43.8%, III 28%, IV 15%.	Vascular, trauma, or neoplastic causes of nerve palsies.	VI cranial nerve is the most affected, followed by Ill and IV nerves.
Cheng et al. [23]	Retrospective Study	624 children	Incidence of infantile torticollis: 1.3%, with a ratio of 3:2 (males to females).	Congenital muscular torticollis, ocular problems, and CNS disorders.	AHP has a low prevalence in the general population. Muscular causes are more common than ocular causes.
Ballock & Song [26]	Retrospective Observational Study	288 children	81.6% of cases caused by congenital muscular torticollis, 18.4% non-muscular causes.	Ocular disorders 23%, brachial plexus palsies 17%, CNS lesions 11%.	Ocular causes are significant but less prevalent than muscular causes. Prevalence of ocular causes: 18-23%.
Gallerani et al. [31]	Retrospective Observational Study	126 patients	Prevalence of palsies: VI 53%, III 36%, IV 11%.	VI cranial nerve most affected.	Emergency room findings show higher prevalence of VI nerve palsies compared to other settings.
Nucci et al. [27]	A prospective, consecutive case series	63 children	AHP caused by orthopedic 52.38%, ocular 39.68%, neurological 7.94%. Superior oblique paralysis caused 48% of ocular AHP.	Superior oblique paralysis.	Ocular causes represent a significant portion of AHP, particularly due to superior oblique muscle paralysis.
Akagi et al. [30]	Retrospective Observational Study	221 patients	Isolated cranial nerve palsies: VI 52.94%, Ill 28.51%, IV 18.55%.	Neurological causes: primarily cranial nerve palsies.	VI nerve palsy is most common in AHP-related neurological disorders.
Çolpak et al. [43]	Retrospective Observational Study	127 patients	Prevalence of palsies: VI 37.8%, Ill 35.4%, IV 26.8%.	Similar distribution as previous studies but slight differences in prevalence rates.	The distribution of cranial nerve palsies depends on the clinical setting.
Sabetti et al. [28]	Observational Study	47 children (7-10 years old)	Observed postural anomalies in children with Exo/Eso deviations: scoliosis 76.47%, knee valgus 47.05%, pelvic misalignment 29.41%.	Strabismus and compensatory AHP.	AHP in strabismus leads to postural imbalances. Postural abnormalities contribute to skeletal pathologies.
Akbari et al. [32]	Review Article	Case series	Nystagmus 17.9%, Superior Oblique palsy 17%, A-V pattern 12.7%, Duane syndrome 12%.	Multiple causes: superior oblique palsy, nystagmus, congenital esotropia, etc.	AHP is caused by various ocular and non-ocular conditions, with significant variation in prevalence rates.

Note: The table summarizes studies on the prevalence, causes, and effects of abnormal head posture (AHP) as reported by multiple authors.

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- **Prevalence of AHP:** In the general population, the prevalence of AHP is relatively low, with Cheng et al. [23] reporting an incidence rate of 1.3%. However, the prevalence is notably higher in clinical settings, especially ophthalmology clinics, with 3.19% of cases in pediatric ophthalmology settings [24,25]. This supports the notion that AHP is more common in clinical environments dealing with visual disorders.

- **Muscular vs Ocular Causes:** The majority of cases of AHP are caused by congenital muscular torticollis, as reported by Ballock & Song [26]. However, ocular causes contribute significantly, particularly superior oblique paralysis, as shown by Nucci et al. [27]. The prevalence of ocular causes of AHP is estimated to range from 18% to 23%, according to various studies, such as those by Ballock & Song [26] and Nucci et al. [27].

- **Postural Anomalies:** AHP is often associated with postural abnormalities, particularly in children with strabismus. Sabetti et al. [28] found that children with Exo/Eso deviations exhibited a range of postural anomalies such as scoliosis, knee valgus, and thoracic hyperkyphosis, which may contribute to skeletal pathologies. These findings underscore the importance of a multidisciplinary approach in managing AHP.

- **Cranial Nerve Involvement:** Cranial nerve palsies, particularly of the VI nerve, are a significant contributor to AHP. Richards et al. [29], Akagi et al. [30], and Gallerani et al. [31] all confirmed that VI nerve palsies are the most common, followed by III and IV nerve palsies. This pattern was observed across different clinical settings, although emergency room data [31] showed a higher prevalence of VI nerve palsy, likely due to the nature of cases seen in emergency departments.

- **Variation in Prevalence Across Settings:** A key finding is the variation in the prevalence of AHP across clinical and research settings. For example, Akbari et al. [32] found significant variation in the causes of AHP, including a range of ocular and non-ocular conditions such as nystagmus (17.9%) and Duane syndrome (12%). These differences emphasize the need for accurate diagnosis and specialized care in the management of AHP.

In conclusion, the data from these studies highlight that AHP is a multifactorial condition, with both muscular and ocular causes being prevalent. Ocular causes, particularly superior oblique paralysis, contribute significantly to AHP, while cranial nerve palsies, particularly of the VI nerve, are often involved in neurological cases. The postural abnormalities associated with AHP in children with strabismus further emphasize the need for comprehensive evaluation, including both visual and musculoskeletal assessments.

Discussion

The AHP of ocular origin is a postural adaptation acquired primarily as a result of paresis or paralysis of the EOM, with the purpose of maintaining single binocular vision [1-4]. We have described how a subject in the presence of paresis-paralysis of the EOM, in order to avoid blurred vision and diplopia, assumes in primary position mainly three types of AHP [4] or a combination of them, given by the action of the extraocular muscles in the various diagnostic positions of gaze. Healthcare practitioners involved in the postural assessment of the patient can ascertain during their examination the presence of head misalignments in various planes of observation (frontal, sagittal, posterior, and transverse), which may lead them, as in the case of "descending syndrome," to request an optometric evaluation. The optometrist will then need to determine whether the origin of the AHP is due to visual or oculomotor disorders, with or without the presence of strabismus.

When there is a refractive defect that is not compensated or poorly compensated by glasses, it can be observed, or it is reported to us by the posturologist. that patients tend to protrude their head forward or tilt it backwards while observing a target. This protrusion of the head is done with the intention of utilizing accommodative convergence, which in turn stimulates accommodation, in order to improve focus and therefore visual acuity. As highlighted by Taub and Harris [33], patients often adopt different postures in response to visual stress and an imbalance between vergence and accommodation. For far and up to intermediate distances, if, for example, a hyperope [34] or a myope [35] moves their head towards the target (assuming a chin-down position), it means that the patient is trying to increase the accommodative demand already induced by the convergence of the medial recti, through the use of the superior recti, which, in addition to their primary action of elevation, also have adduction as a tertiary action [22].

Similarly, in near vision and up to more distal and intermediate distances, by moving the head backward (assuming a chin-up position), they attempt to utilize the action of the inferior recti, which also have adduction as a tertiary function [22]. Furthermore, it is not unusual to observe that, during these actions, they also increase the illumination on the plane of the target they are observing, to induce greater miosis and reduce the circle of confusion of the retinal image. Therefore, through the process of synkinesis between convergence, accommodation and miosis [3], they seek to extract any possible accommodative residue. Thus, the addition of the muscle action of the superior or inferior recti indicates that they are seeking an accommodative surplus and that they have already reached the limit of accommodation that can be induced through convergence, by virtue of the AC/A ratio [22,36].

Regarding astigmatism, this refractive condition, if not compensated or poorly compensated by optical correction, can negatively affect both visual quality and head posture. In patients with inadequate correction, it is common to observe abnormal head tilts or rotations, a phenomenon that can be explained through the principle of vectorial summation of the axes, which is proportional to the degree and axis of the astigmatism. For example, in a patient with hypermetropic astigmatism, with an axis at 90° but corrected with a lens having an axis at 120°,

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the individual will tend to slightly tilt the head towards the left shoulder. This tilt serves to align the new axis resulting from the vectorial combination of the refractive axes, allowing the visual system to perceive an intermediate orientation between 90° and 120°, determined by the sum of the associated vectors. Therefore, the head tilt acts as a compensatory visual strategy aimed at reducing the perceived refractive error and optimizing visual quality.

Studies like that of Fesharaki et al. [37] have shown that the head tilt is proportional to the induced axial error. For example, a 25° tilt corresponds to an average error in the astigmatic axis of 18.6° (±4.2°), while smaller tilts, such as 5°, produce more contained errors, equal to 3.2° (±1.5°). However, the natural compensation through ocular counter-torsion [22], a movement that involves the rotation of the eyeballs in the opposite direction to the head tilt, is often insufficient to fully correct the astigmatic error, pushing the patient to adopt a tilted head posture as a necessary compensatory strategy to optimize vision.

Patients with anisometropia also tend to rotate their heads horizontally towards the side of the better-seeing eye to reduce visual confusion caused by disparate retinal images. This movement improves perception, likely by bringing the object closer to the dominant eye [32,38]. Studies have shown that head rotation or tilt can induce compensatory ocular torsions and vertical movements that help stabilize vision [32,38]. However, in significant cases, when the image size difference exceeds 5% or the anisometropia surpasses 3 diopters, fusion becomes impossible, leading to suppression or diplopia [2,22]. This phenomenon is particularly evident in hypermetropic anisometropia, where the better-seeing eye dominates while the other is excluded from the binocular vision process [2]. In children, refractive differences greater than 2 diopters can result in anisometropic amblyopia, while in adults, contact lenses are more effective than spectacles in reducing aniseikonia [39].

Subjects who use standard progressive lenses may often adopt incorrect postures, especially when using the computer or other activities at intermediate distances. This occurs because the variation in power along the progression corridor is primarily designed for use in open spaces, where observation distances differ significantly from those typical of indoor work environments [40,41]. The structure of progressive lenses allows, through eye and head movement, access to different correction zones along the progression channel until reaching the addition for nearby. This process of self-adjustment allows the subject to obtain a correction more appropriate for the desired viewing distance. In such contexts, the chin-up posture, characterized by raising the chin, becomes a compensatory strategy to improve visual acuity at intermediate distances or to correct suboptimal vision [40,42]. This behavior is particularly evident in under corrected hyperopes, who lift their chins to utilize areas of the lens with greater power, and in overcorrected myopes, who adopt the same posture to access zones of the lens better suited to their visual needs [40]. To prevent problems related to these incorrect postures, such as muscle tension, cervical pain, and postural disorders, it is essential to conduct a thorough professional evaluation and customize progressive lenses to the specific needs of the patient. Alternative visual solutions, such as indoor lenses designed for closed environments, can reduce the need for postural compensation and ensure optimal visual comfort, even during prolonged work sessions [40,44-48].

Conclusion

The abnormal head posture (AHP) of ocular origin requires a multidisciplinary approach to recognize and manage the condition. Although the interactions between vision and posture are known, the training of optometrists on the concept of posture, as interpreted by posturologists, requires further study. Similarly, posture professionals should broaden their understanding of the complexity of the visual system. In the training of optometrists, the study of posture is often relegated to those causes that can determine an abnormal head posture as a function of an alteration of the visual system. Furthermore, posture takes on different meanings among the various health professionals who deal with the functional and objective evaluation of movement and posture. In fact, in the assessment of the patient's state with respect to an "ideal posture", the visual system is taken into consideration only when there is a postural imbalance above the center of gravity, the cause of which is often sought in the involvement of the oculomotor or stomatognathic system, and in particular in a district above the cervical one.

Therefore, dialogue between the various professional figures occurs mainly when an assessment of the visual apparatus is required. However, communication on why such an assessment is necessary can vary greatly depending on the academic training and specialization of each professional, which can lead to confusion. Establishing clear reference protocols and creating a common clinical language between professionals can facilitate integrated patient management. It is essential that all health professionals reflect on how their practices can evolve to address the challenges related to the AHP, aiming to offer adequate and cooperative treatment. Only through collaborative work and continuing education will it be possible to ensure a comprehensive and attentive approach to the needs of patients.

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