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Modern Interpretation of the Diagnosis, Treatment and Prevention of Divergent Strabismus with a Vertical Component

Abdullaeva Dilorom Rustamovna
Department of Ophthalmology, Samarkand State Medical University

Abstract: The complexity of the structural and functional organization of the visual and oculomotor systems determines the variety of types of strabismus (heterotropia), which are the result of damage to the organ of vision, differing in etiology and location. The success of strabismus treatment, especially surgical, largely depends on the correct determination of its type. This is facilitated by a rational classification of types of strabismus, which should reflect their etiology and facilitate the choice of treatment method. Many clinical classifications of strabismus have been proposed, which to a greater or lesser extent take into account the etiological factors of this disease and its main clinical symptoms. The rapid development of diagnostic methods for lesions of various parts of the binocular visual system at the end of the 20th century contributed to the clarification of the etiology of already known types of strabismus and the identification of its new clinical varieties, which led to the need to clarify and expand the classification of this disease. Currently, ophthalmologists in Europe and America use more sophisticated classifications that more fully cover the currently known types of strabismus and more accurately reflect their etiology and clinical features. A review of these classifications shows that the traditional division of strabismus remains clinically relevant to this day. With combined strabismus, there is no violation of eye motility, the angles of primary and secondary deviation are equal, the same in all directions of gaze, or their difference does not exceed 5 pr. This is called the primary or main form of strabismus. Currently, combined strabismus also includes such types of strabismus as syndromes A, V, X, in which the angle of deviation when turning the eyes up and down differs from the angle of deviation in the main position of the eye by more than 5 pr. diopters, as well as dissociated horizontal deviation of the eyes in the absence of DHDvi. Their difference is more than 5 pr, but eye mobility is normal.

Keywords: pathogenesis, etiology, origin, diagnosis, prevention and prognosis of strabismus.

Introduction: Classification of non-congruent strabismus The main symptom of non-congruent strabismus is a more or less pronounced limitation of eye motility, which most ophthalmologists previously considered a sign of paralysis or paresis of the oculomotor nerves. In non-congruent strabismus, the angles of primary and secondary deviation are unequal and differ in one or more directions of gaze. In addition to true neurogenic paralytic, paretic strabismus, which primarily requires examination and treatment by a neurologist, the limitation or absence of eye motility is characteristic of pseudoparalytic strabismus, anatomical anomalies of the extrinsic muscles of the eye, strabismus with limited mobility caused by anatomical anomalies of the extrinsic muscles of the eye, scleritis and some general diseases, their attachment sites and some diseases. In some patients with relatively rare hereditary multisystem diseases and disorders (Crouzon disease; Apert, Franceschetti, Waardenburg, Goldenhar, Mobius, Prader-Willi syndromes; chronic progressive myopathy: Graefe disease, Graefe disease "plus"), strabismus with limited or absent mobility may also be observed. In the classifications known in the CIS countries, these

forms of strabismus are classified into a group called "Special forms" or "Atypical forms" [1, 2, 4, 9, 13, 19, 23]. In our opinion, such a name does not reflect either the etiology of these types of strabismus or their common characteristic features (friendliness, limited mobility). It is not surprising that such forms of strabismus with impaired eye mobility are often not recognized and are diagnosed as paralysis or paresis of the oculomotor nerves. In such cases, instead of timely surgical treatment, which is necessary, useless complex neurological examinations and treatment by a neurologist are carried out. In recent years, the above-mentioned forms of strabismus have been designated by the term “restrictive strabismus” [30-34]. We believe that the division of non-paralytic types with limited mobility into a special group “Restrictive strabismus” or “Pseudoparalytic strabismus” firstly reflects their common clinical features, and secondly, indicates the need for differential diagnosis with paralytic or paretic strabismus in the first place.

Research methods and materials: Accommodation and convergence states play an important role in the etiology, clinical manifestations and treatment of combined strabismus. Depending on the degree of expression of the accommodative component, it is customary to distinguish accommodative, partially accommodative and non-accommodative types of combined strabismus [1, 2, 4, 6-10, 14, 15]. In 1948, Kostenbeder F. divided accommodative convergent strabismus into two types: typical accommodative, in which the angle of deviation during distance fixation and near fixation is eliminated by correcting ametropia with glasses, and atypical accommodative strabismus, in which it is completely eliminated during correction at close range. In 1950, Kostenbeder F. replaced the term “atypical accommodative” with the term “hypoaccommodative”, since near esotropia is a consequence of tension during fixation near excessively weakened accommodation and an abnormally high AC / A ratio [1, 32, 37]. Pilman NI (1979), Serdyuchenko VI with co-authors (1992), Degtyareva NM, Serdyuchenko VI (1998), referring to Kostenbeder F., divide accommodative convergent strabismus into: 1) typical accommodative, 2) atypical accommodative, 3) hypoaccommodative, 4) partially accommodative. In addition, hypoaccommodation is divided into two subtypes: complete hypoaccommodation and partial hypoaccommodation. Comparison of the clinical signs of both subtypes of hypoaccommodative strabismus described by these authors with the clinical signs of atypical accommodative strabismus described by them does not allow us to identify significant differences between them in the nature of the deviation, refraction, or the effect of optical correction on the magnitude of the angles of near and far fixation. Their pathogenesis is based on a common etiological factor - accommodation impairment. Diagnosis of hypoaccommodative strabismus requires the determination of binocular and monocular visual acuity under visual tension and during near and far fixation without tension, which, as a rule, is not possible in all children under 4 years of age who develop this type of strabismus. Therefore, we preferred to use the classification of accommodative strabismus, currently used by ophthalmologists in Europe and America, which does not take into account visual acuity. In this classification, instead of the terms typical and atypical accommodative convergent strabismus, the terms “refractive” (instead of “typical”) and “refractive” (instead of “atypical”) are used, which more accurately reflect the etiology of this type of esotropia. Refractive accommodative esotropia is characterized by the presence of significant ametropia (4-10 diopters and more); the magnitude of the angles of deviation at distance and near fixation, which is completely eliminated by spectacle correction; the normal value of the AK/A ratio. In non-refractive accommodative esotropia, ametropia is absent or insignificant; the angle of deviation at near fixation is greater than the angle of deviation at distance; complete correction of ametropia does not eliminate the deviations that appear each time in the accommodative tension to achieve optimal visual acuity; the AK/A ratio is high. Kansky D. (2006) shows that non-refractive accommodative esotropia can be of two types: 1) a subtype caused by excessive convergence (large AC - accommodative convergence) with a normal state of accommodation (A) and 2) a subtype caused by accommodative weakness (hystraaccommodative high). The value of A is equal to the normal AC value, so the accommodative movement leads to excessive convergence [11]. In addition to these two forms of accommodative esotropia, two more forms are distinguished: decompensated accommodative

esotropia and mixed (combined) accommodative esotropia. Decompensated accommodative esotropia has a non-accommodative component that arises secondarily as a result of a long-standing accommodative deviation. The transition from accommodative to partially accommodative, and sometimes to non-accommodative, is observed in a number of patients with visual impairment or with non-systemic treatment, when wearing incomplete optical correction of ametropia. In such cases, it is indeed better to use the term "decompensated accommodative esotropia" to distinguish this form from the well-known primary partially accommodative strabismus, in which there is initially a non-accommodative component. Mixed accommodative esotropia is a combination of accommodative esotropia of the refractive and non-refractive type. It is characterized by the presence of age-related hyperopia; high AK/A; the magnitude of esotropia for near is greater than for distance. In such patients, the deviation is corrected with bifocal glasses (the upper part corrects the ametropia, the lower part is 2-3 diopters more). secondary as a result of prolonged accommodative deviation. The transition from accommodative to partially accommodative, and sometimes to non-accommodative, is observed in a number of patients with visual impairment or with non-systemic treatment, when wearing incomplete optical correction of ametropia. In such cases, it is indeed better to use the term "decompensated accommodative esotropia" to distinguish this form from the well-known primary partially accommodative strabismus, in which there is initially a non-accommodative component. Mixed accommodative esotropia is a combination of accommodative esotropia of the refractive and non-refractive type. It is characterized by the presence of age-related hyperopia; high AK/A; the magnitude of esotropia for near is greater than for distance. In such patients, the deviation is corrected with bifocal glasses (the upper part corrects the ametropia, the lower part is 2-3 diopters more). secondary as a result of prolonged accommodative deviation. The transition from accommodative to partially accommodative, and sometimes to non-accommodative, is observed in a number of patients with visual impairment or with non-systemic treatment, when wearing incomplete optical correction of ametropia. In such cases, it is indeed better to use the term "decompensated accommodative esotropia" to distinguish this form from the well-known primary partially accommodative strabismus, in which there is initially a non-accommodative component. Mixed accommodative esotropia is a combination of accommodative esotropia of the refractive and non-refractive type. It is characterized by the presence of age-related hyperopia; high AK/A; the magnitude of esotropia for near is greater than for distance. In such patients, the deviation is corrected with bifocal glasses (the upper part corrects the ametropia, the lower part is 2-3 diopters more).

Results: The most common type of uncombined accommodative esotropia, the primary or main form, is based on a violation of the innervation of convergence and divergence: either increased convergence innervation or weakened or insufficient divergence innervation. In this main form of uncombined accommodative esotropia, there is no ametropia or it is mild, the AC/A ratio is normal and there is no accommodation disorder; the angle of deviation is the same for near and far or slightly larger for near (with excessive convergence) or slightly larger for far (sufficient convergence) and is not corrected with glasses. In addition to this main form, the following are distinguished: primary infantile esotropia, microtropia, blind spot syndrome and symptom, sensory esotropia, cyclic strabismus, acute-onset strabismus, secondary esotropia. In essential (idiopathic) infantile (congenital) esotropia, which occurs at birth or in the first six months of a child's life, ametropia is absent or insignificant, and eye mobility is preserved. Microtropia or microstrabismus is a specific form of strabismus, with a high degree of binocular interaction, but stereoscopic vision is always reduced or completely absent. It is characterized by the presence of an angle of deviation of less than 5 degrees. (Less than 10 pr. diopters) ametropia is combined with full spectacle correction, often with anisometropia, less often with amblyopia. Most often, microtropia is convergent (esomicrotropia) and rarely divergent (exomicrotropia). This condition was first described in 1951 by Jampolsky J. under the name "fixation imbalance". In 1966, Lang J. proposed to call this form of strabismus microtropia, microstrabismus or monofixation syndrome. Primary microtropia and secondary microtropia are distinguished [11, 12, 17, 4, 6, 9,

31, 8]. Primary microtropia (monofixation syndrome) is mainly sensitive in etiology, since it is the result of a high degree of adaptation of the visual system to abnormal binocular fixation, often observed in amblyopia with eccentric fixation and is characterized by retinal adaptation of the harmonic type of ACS. In primary microtropia, the angle of deviation is stable binocularly and monocularly; there are no accommodative movements of the eye during the closure test; the angle of deviation, the angle of anomaly and the degree of eccentricity of fixation are equal. In patients with primary microstrabismus, amblyopia can be treated, but the angle of strabismus cannot be corrected, and surgical treatment is usually futile. Secondary microtropia is a consequence of motor disorders, often occurs during the treatment of large angles of deviation (mainly surgical) and is characterized by the presence of surrogate binocular vision with non-harmonic ACS and the presence of accommodation of eye movements during the occlusion test in addition to microdeviation. A patient with secondary microtropia can be treated. Non-accommodative esotropia and esotropia accompanied by a small angle of deviation should be distinguished from blind spot syndrome (Swan syndrome) and blind spot symptom (mechanism) (Swan symptom) [38]. Blind spot syndrome was described by Swan in 1948 and is characterized by a stable angle of esotropia, the value of which corresponds to the angle of projection of the blind spot in the visual field (10-15 degrees or 20-30 diopters) and the presence of normal retinal correspondence, good visual acuity in both eyes. In the symptom (mechanism) of the blind spot, a similar magnitude of esodeviation is combined with ACS, amblyopia. In both cases, the blind spot of the deviated eye acts as a functional scotoma, which is used during binocular fixation to avoid diplopia. Unlike the blind spot syndrome, surgical correction of deviations in the blind spot symptom is not very effective. In recent decades, the concept of "sensory strabismus" or "sensory strabismus" has firmly established itself in foreign literature [11, 12, 4, 7, 2, 30, 38, 40]. This form of convergent or divergent combined non-accommodative strabismus occurs as a result of clouding of the refractive medium, as well as a decrease in visual acuity due to diseases of the retina and optic nerve. Functional treatment of this type of strabismus (restoration of binocular vision) depends on the possibility of improving visual acuity. Otherwise, only cosmetic surgery is possible to correct strabismus. The sensory form also includes convergent strabismus with high myopia, which is eliminated by spectacle correction due to a significant increase in visual acuity [1]. Some authors use the term “secondary strabismus” instead of the term “sensory strabismus” [10, 17]. However, most ophthalmologists consider secondary non-accommodative strabismus to be strabismus in the direction opposite to the primary one, which occurs with spectacles or surgical hypercorrection.

Discussion: Cyclic strabismus is a simultaneous eye twitching that occurs at regular intervals. During the period of absence of strabismus, the patient has normal binocular vision [1, 10, 11, 14, 16, 27, 38, 40]. The etiology of this type of strabismus is not clearly established, it is caused by disorders in the central nervous system (CNS), therefore, treatment is primarily neurological. Some authors define this condition by the term “intermittent or intermittent strabismus” instead of the term “cyclic strabismus”. From our point of view, in order not to confuse this type of strabismus with alternating strabismus, sometimes also called intermittent strabismus, it is better to use the term “cyclic strabismus”.

Acute strabismus (acute strabismus) is a deviation that occurs suddenly in school-age children and adults, accompanied by diplopia, but with all the signs of compliance [11, 14]. This strabismus is not caused by paresis of the oculomotor nerves, but by decompensation of heterophoria due to psychological trauma, stress, nervous tension, and fatigue.

Divergent combined non-accommodative strabismus is divided into two types: permanent and periodic. Permanent concomitant non-accommodative exotropia is divided into infantile (congenital), which occurs in the first six months of a child's life and, except for nystagmus, has the same symptoms as infantile esotropia; sensory (due to organic changes in the refractive medium of the eye, retina and optic nerve); secondary (due to prolonged wearing of a convex sphere, surgical hyperesthesia); primary or fundamental, which is based on the absence of convergence (the angle of exodeviation for near and far is the same) or its significant weakening

(the angle of exodeviation for near is greater than for far). Intermittent concomitant non-accommodative exotropia has two phases or stages. In stage I, the deviation of one eye outward occurs during the occlusion test and periodically with the patient's attention turned off, with a distracting gaze into the distance. Stage II is characterized by exodeviation during distal fixation and occasionally during proximal fixation. Treatment of stage II is usually surgical.

Excessive innervation of divergence leads to excessive divergence, which is manifested by a constant deviation of one of the eyes to the distance during binocular fixation, while orthotropia is observed with normal binocular vision and normal convergence during near fixation.

In addition to the most common form of vertical strabismus, which can be characterized as the main form, accompanied by symptoms of diplopia, ptosis, and forced head rotation, there are also dissociated vertical deviation (abbreviated DVD) and divergent vertical divergent strabismus. DVD was first described in 1940 by Bielshovsky A. This condition is also called "occlusive hypertropia", "alternating hypertropia" and is manifested by alternating upward deviation of the right or left eye [11, 17, 7, 8-4]. In 1973, G. Noorden described divergent vertical concomitant strabismus, in which one eye deviates upward and the other deviates downward when fixed with this eye. Pseudoptosis is most often observed in the down-facing eye, which disappears when the eye is straightened. In addition to the well-known mixed form of horizontal and vertical strabismus, which is divided into horizontal (convergent or divergent) strabismus with a vertical component, if the horizontal deviation angle is greater than the hypotropia, and horizontal component if the vertical deviation angle is greater than the horizontal deviation angle, ES Avetisov in 1979 described a special form of mixed strabismus - moving vertical-horizontal deviation (ES Avetisov syndrome). In this form of mixed combined strabismus, when the first is fixed, there is a horizontal deviation of one eye, and the second, a vertical deviation of the paired eye.

As for the division of combined heterotropia into unilateral and variable, as well as classification according to the state of binocular vision, the state of monocular visual fixation, the presence and degree of amblyopia, we consider the opinion of N.I. Pilman (1967) and L.A. Dymshitsa (1970) to be quite fair, such a division does not reflect the etiology of the type of strabismus and is not logical, since the conditions of the factors listed above are not inherent in any type of strabismus.

Conclusion: Paralytic and paretic types of incommensurable strabismus occur with nuclear, fascicular, basilar (stem), orbital lesions of the oculomotor nerves. At the same time, the division of paralytic and paretic strabismus depending on the location of the oculomotor nerve damage requires specific methods of examining the brain. An ophthalmologist cannot independently, without the help of a neurologist, perform a topical diagnosis of most lesions of the oculomotor nerves, except for the orbital ones. At the same time, strabismus, diplopia, and eye motility are often the first manifestations of pathological processes in the central nervous system of various etiologies, and the patient first turns to an ophthalmologist. In such cases, the ophthalmologist should determine the initial diagnosis based on the assessment of eye symptoms and the general condition of the patient. In this regard, it is more useful for the ophthalmologist to classify paralytic, paretic strabismus depending on which nerve is affected, depending on the number of nerves affected (isolated paralysis of one of the oculomotor nerves or ophthalmoplegia), with damage to the oculomotor nerves and with damage to other cranial nerves (BlerMillard, Weber, Benedict syndromes, orbital apex syndrome, superior orbital fissure syndrome). Some authors distinguish paralytic strabismus between cortical (cortical oculomotor centers) and subcortical (pathways from cortical centers to the nuclei of the oculomotor nerves) lesions of the central nervous system [19, 23]. However, with such lesions, paralysis and paresis of the eye occur, voluntary lateral deviations of both eyes are impaired, paralysis or convergence and divergence paresis occur while the mobility of the monocular eye is preserved. Strabismus is absent due to the lack of uniform innervation of the corresponding eye muscles of both eyes. The presence of strabismus in cortical and subcortical lesions of the central nervous system, with the addition of

nuclear paralysis and paresis, indicates the involvement of the nuclei of the oculomotor nerves in the process.

Taking into account all of the above, we have developed a clinical classification of concomitant strabismus (Fig. 1), based on the etiology, pathogenesis and clinical features of the currently known types of strabismus, the most frequently observed in the practice of ophthalmologists. We understand that the classifications we propose are not ideal, but they fully reflect the diversity of currently known types of strabismus, their etiology and main clinical symptoms, which will help the ophthalmologist to more accurately determine the nature of the oculomotor disorder, correctly conduct a differential diagnosis and rationally choose the most effective treatment tactics.

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