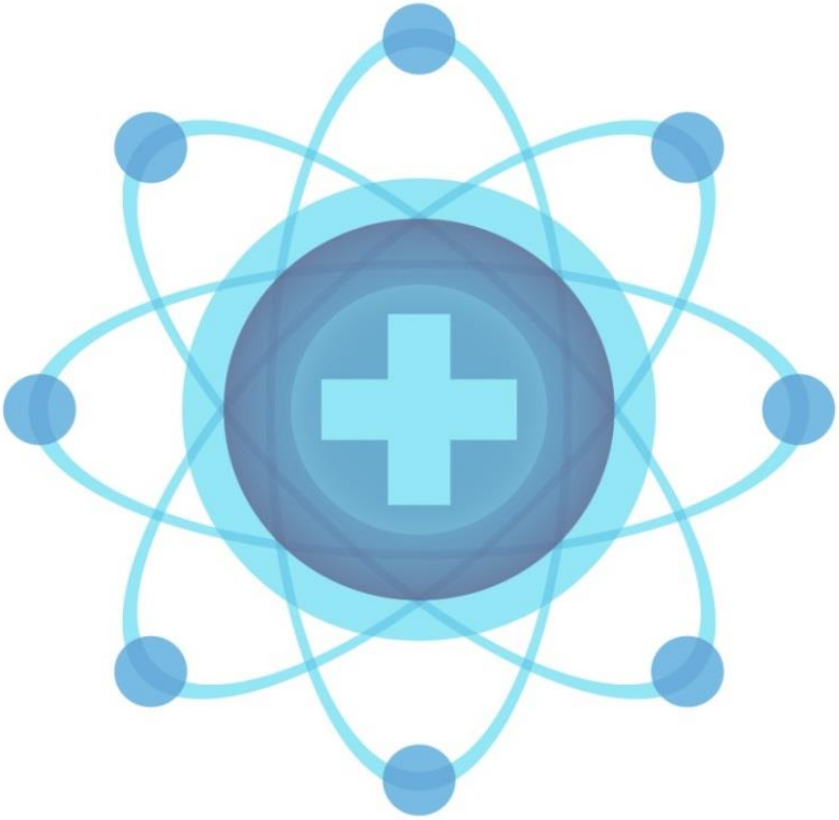

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SEQUENTIAL STRABISMUS
Literature review

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Abstract. Secondary strabismus is called an eye pathology that occurs during the treatment of strabismus and is characterized by a change in the deviation of the squinting eye to a diametrically opposite direction - convergent strabismus becomes divergent and vice versa. Prevention of secondary strabismus should be aimed at minimizing the hypereffects of surgical interventions for strabismus, leveling the causes of imbalance in the vergent system, and strengthening the binocular function that keeps the eyes in a symmetrical position.
Keywords: secondary sequential strabismus, hypereffect of surgical intervention, restrictive strabismus.

Introduction. Secondary strabismus in sequential or residual strabismus is a well-known complication of surgical treatment of strabismus. Secondary (synonymous with inverse) strabismus is an eye pathology that occurs during the treatment of strabismus and is characterized by a change in the deviation of the squinting eye to the diametrically opposite direction - convergent strabismus becomes divergent and vice versa. The new direction of deviation requires a change in the previous treatment tactics, increases the rehabilitation period, worsens the results, and negatively affects the psychological state of the patient and the doctor. There is no doubt that secondary strabismus is not one of the many types of strabismus but represents a severe complication of the treatment process [1,2,3]. Some ophthalmologists also call secondary strabismus a significant decrease in vision in one eye caused by congenital or early acquired pathology of the retina, optic nerve, and clouding of the refractive media that exclude joint binoculars function of both eyes. Many foreign ophthalmologists define such strabismus by the term "sensory strabismus" [3,4]. According to the literature, it was found that inverse divergent strabismus after surgical correction of convergent strabismus is observed in 10-25% of cases. It was reported that the incidence of insufficient correction after esotropia and exotropia surgery ranged from 20 to 40% and from 22 to 59%, respectively [8,11,15,16]. In contrast, the incidence of hypercorrection after esotropia and exotropia surgery ranged from 20 to 27% and from 2 to 20% respectively [9,14,18,21,24]. Secondary strabismus is heterogeneous both in terms of causes and timing of occurrence and in terms of development mechanisms.

Since secondary strabismus, whether recurrent or sequential, can occur long after good initial findings, the true incidence of secondary strabismus is often underestimated [17]. The causes contributing to the development of secondary strabismus are numerous. Secondary strabismus resulting from a change in the direction of deviation of the squinting eye is a complication in the treatment of strabismus and occurs mostly after surgical intervention. Secondary divergent strabismus may also develop during conservative treatment of convergent

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accommodative strabismus when its first symptoms are not detected in time. According to authors Goncharova S.A. et al., the pathogenesis of this complication is caused by the weakening of the interconnected functional system accommodation - convergence and prevalence of divergence in the prevailing conditions. The transition from esodeviation to exodeviation is also promoted by the absence or instability of binocular function and insufficiency of positive fusion reserves, which were noted in all children of this group. Weakening or cancellation of the plus spherical correction, strengthening of accommodation, convergence, positive fusional reserves, formation of binocular function prevent exodeviation and development of secondary divergent strabismus. The correctness of this opinion on the pathogenesis and measures to prevent secondary divergent strabismus in patients with congruent accommodative strabismus is confirmed by successful preventive treatment of children. The causes of the development of secondary strabismus are also: hypereffects of surgical intervention, incomplete orthopto-diploptic therapy in the postoperative period, untimely weakening or cancellation of plus spherical correction and age-related divergent changes in orbital configuration in children and adolescents.

The emergence of secondary strabismus in patients who have not received full orthopto-diploptic treatment for binocular vision is explained by the absence of such an important function that keeps the eyes in a symmetrical position, disrupting unstable muscle balance vergence system and the development of secondary strabismus. Strabismus may also be caused by untimely weakening and cancellation of plus spherical correction due to the failure of patients to visit an ophthalmologist for common control. In these cases, excessive plus correction, having weakened accommodation and convergence, increased divergence, violated physiological regulation mechanisms in the vergence system and led to the development of divergent strabismus. Studies have shown that secondary divergent strabismus is 11 times more common than secondary convergent [1]. Also, studies have identified the following causes of secondary (consequent) strabismus and factors favoring it: excessive recession of internal straights during surgery for convergent strabismus (distant place of attachment of internal straights after their recession - 7-10 mm from the original position); weakness or absence of convergence; the presence of associated dissociated vertebrae (2). Secondary strabismus occurring in the early postoperative period is thought to result from muscle slippage and is usually associated with marked hypofunction of the affected muscle. Late secondary strabismus related to varying degrees of muscle hypofunction, which occurs from months to years after the primary procedure, is thought to result from improper wound healing with stretching of scar tissue passing between normal muscle tissue and the sclera [10,22]. Secondary restrictive strabismus can occur after surgery to correct strabismus due to complications such as complete loss of the muscle during surgery or muscle detachment in the postoperative period. It also occurs with excessive recession or resection, scarring of the muscle, or fusion with orbital tissues. Secondary restrictive strabismus manifests as secondary deviation, with diplopia and impaired eye movement [5,6,10,20,22]. Stretched scar causes weakening of muscle activity with the development of secondary strabismus associated with poor

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inductance in the direction of action of the affected muscle. This phenomenon was first explained by Ludwig and Chow in 1999 using intraoperative observations and histological studies, and they called it the "stretched scar syndrome" [17]. In general, secondary strabismus caused by a stretched scar should be suspected in cases of late overcorrection or undercorrection associated with limited contraction of the previously operated muscle.

Complications of surgical treatment of vertical strabismus with hyperfunction of the inferior oblique muscle (IOM) may include recurrence of hyperfunction of the inferior oblique eye, requiring mandatory secondary surgical loosening. When performing transposition of the inferior oblique muscle, due to the lack of surgical dosing techniques, most often the authors note the development of hypercorrection of hypertropia of the operated eye, while recession or any other methods of loosening of the LCM give, on the contrary, insufficient corrective deviation effect [21].

Sequential exotropia is defined as exodeviation after hypercorrection of esodeviation. The literature has reported its prevalence as 4% to 29%. [7,19,25] This pathology occurs more frequently when combined with various concomitant factors, including amblyopia, high hyperopia, lack of binocular vision, DVD syndrome and large recession of the ERM, simultaneous strabismus surgery on three or four eye muscles, and error in measuring esotropic deviation [12,26]. Muscle loss or MR slippage may be considered probable causes if exotropia is observed immediately or in the short term after esotropia surgery, whereas prolonged sequential exotropia may be caused by gradual lengthening and weakening of the mitral muscle due to scar tissue formation at the site of the muscle tendon suture. Consequently, it will lead to insufficiency of the VPM limitation of adduction as well as weakness of convergence [13,23].

Conclusion. Thus, consecutive strabismus complicates the treatment of patients with strabismus, increases recovery time, and negatively affects the patient's psyche. Prevention of secondary strabismus should be aimed at minimizing the hypereffects of surgical interventions on strabismus, leveling the causes causing an imbalance in the vergence system, and at strengthening the binocular function holding the eyes in a symmetric position. The occurrence, treatment, and prevention of secondary strabismus require further study by ophthalmic surgeons.

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