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Long-term effects of optical defocus on eye growth and refractogenesis

Długotrwały efekt optycznego rozogniskowania na wzrost oka i refraktogenezę

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ABSTRACT

Introduction: The aim of this paper was to study the effect of binocular and alternating monocular myopic constant defocus prescribed in spectacle format on myopia onset and progression in children.

Material and methods: 129 children aged 5–12 years were divided into 4 groups: 48 children aged 5–8 years (1st group) with emmetropia and risk factors of myopia development were prescribed the continuous wearing of plus lenses to induce myopia of 1.0 D. 46 children aged 7–11 years (2nd group) with low myopia from –0.75 to –2.25 D were prescribed 2 pairs of spectacles for alternating continuous wearing. One eye was corrected for distance to obtain residual myopia in spectacles of about 0.50 D, and the fellow eye was corrected to obtain residual or induced myopia of about 1.50 D. The children changed spectacles every day. Control data were obtained from 15 children aged 6–9 years (1st control group) with pseudomyopia with no correction administered, and 20 low myopic children aged 7–12 years (2nd control group) wearing conventional spectacle correction. Autorefractometry before and after cycloplegia and ultrasound biometry were performed.

Results: A hyperopic shift caused by thinning of the crystalline lens and deepening of the anterior chamber in all patients of the 1st group was observed after 1 month and persisted over the follow-up period. Horizontal diameter (HD) increased more than 3 times as much as the axial length (AL). No cases of myopia onset were observed during the 9-year follow-up period. 36 (81.8%) children of the 2nd group had stable refraction over the 4 year follow up, an insignificant increase in the AL and a significant increase in the HD were revealed. A 3-year follow-up revealed an increase in cycloplegic refraction in both control groups; the AL increased significantly, while the HD showed an insignificant increase.

Conclusions: Permanent low myopic defocus of the image in binocular spectacle format inhibits eye growth and refraction shift to myopia in children with low hyperopia, emmetropia and low myopia. The method of alternating monolateral low myopic defocus arrests the myopia progression in 81.8% of children with low myopia for 4 years and 66% for 7 years.

Keywords: emmetropization, refractive development, myopia, optical defocus.

STRESZCZENIE

Wstęp: Celem pracy było zbadanie wpływu obuocznego i zmieniającego się jednoocznego stałego rozogniskowania krótkowzrocznego wypisywanych okularów na formowanie początku i postępu krótkowzroczności u dzieci.

Materiał i metody: 129 dzieci w wieku 5–12 lat podzielono na 4 grupy: 48 dzieciom w wieku 5–8 lat (grupa I) z emetropią i czynnikami ryzyka rozwoju krótkowzroczności wypisywano soczewki plusowe do ciągłego noszenia, aby wytworzyć krótkowzroczność 1,0 D. 46 dzieciom w wieku 7–11 lat (grupa II) z niską krótkowzrocznością od –0,75 do –2,25 D wypisywano 2 pary okularów do zmieniającego się stałego noszenia. Jedno oko korygowano do dali tak, aby otrzymać resztkową krótkowzroczność w okularach ok. 0,50 D, oko towarzyszące korygowano tak, aby otrzymać resztkową lub aby wytworzyć krótkowzroczność ok. 1,50 D. Dzieci zmieniały okulary każdego dnia. Dane kontrolne otrzymano od 15 dzieci w wieku 6–9 lat (I grupa kontrolna) z pseudokrótkowzrocznością i bez stosowania korekcji oraz od 20 dzieci z niską krótkowzrocznością w wieku 7–12 lat (II grupa kontrolna) noszących konwencjonalną korekcję okularową. Wykonywano autorefraktometrię przed i po cykloplegii oraz biometrię ultradźwiękową.

Wyniki: U wszystkich pacjentów z grupy I po miesiącu i dłuższym okresie obserwacji odnotowano przesunięcie w kierunku nadwzroczności spowodowane ścieńczeniem soczewki i pogłębieniem komory przedniej. Średnica horyzontalna (HD) powiększyła się 3 razy bardziej niż długość osiowa (AL). Nie zaobserwowano przypadku wystąpienia krótkowzroczności w ciągu 9-letniego okresu obserwacji. Stabilną refrakcję trwającą dłużej niż 4-letni okres obserwacji miało 36 (81,8%) dzieci z grupy II. Stwierdzono nieznamienny wzrost AL i znamienny wzrost HD. W ciągu 3-letniego okresu obserwacji w obu grupach kontrolnych AL wzrastał znamiennie, podczas gdy HD wykazywał wzrost niezamienny.

Wnioski: Stałe niskie rozogniskowanie krótkowzroczne obrazu w okularach obuocznych hamuje wzrost oka i przesuwa refrakcję w kierunku krótkowzroczności u dzieci z niską nadwzrocznością, emetropią i niską krótkowzrocznością. Metoda zmieniania jednoocznego niskiego roogniskowania hamuje postęp krótkowzroczności w 81,8% u dzieci z niską krótkowzrocznością przez 4 lata i w 66% przez 7 lat.

Słowa kluczowe: emetropizacja, rozwój refrakcji, krótkowzroczność, optyczne rozoogniskowanie.

INTRODUCTION

The prevalence of myopia in children has increased substantially over recent years, and is approaching to 20–30% in non-Asian countries and to at least 80–90% in urban South East Asian countries [1, 2, 3]. Myopia is associated with pathological conditions such as glaucoma and cataract, and is an important risk factor for retinal detachment [4, 5, 6, 7]. The search for effective strategies to control the progression of myopia in children is of great importance. Previous attempts at controlling myopia have included optical means such as bifocal and multifocal (progressive addition) ophthalmic lenses and contact lenses, which have shown little effect in slowing myopia progression [8, 9]. Findings from the COMET Study showed that children wearing progressive addition lenses progressed on average 0.16 D less than children wearing single vision lenses [10]. Studies in which children were under-corrected have reported mixed results. A study of undercorrection of myopia found that myopia progressed significantly more rapidly in children who were undercorrected compared to those wearing a full correction, implying that myopic defocus in humans can increase the rate of myopia progression [11]. On the other hand, Phillips [12] showed that the use of monovision spectacle correction in a group of children slowed axial elongation and significantly reduced myopia progression in the near corrected eye. These results indicate the role of retinal defocus in myopia progression.

Several animal models were used to show that hyperopic defocus achieved by minus lens wear produces a compensatory increase in the axial eye length whereby the focal plane is shifted and myopia is induced.

In contrast, low-power plus lenses reduce the rate of axial elongation and develop hyperopia [13, 14, 15, 16, 17, 18, 19, 20].

Hung and Ciuffreda [21, 22] showed that both genetic and defocus-induced environmental factors play an important role in the development of refractive error and emmetropization, and suggested incremental retinal-defocus theory.

Our prior observations of children with low and moderate hyperopia who had worn special glasses to ensure optical penalization in strabismic or anisometropic amblyopia for several years showed that permanent low myopic defocus, i.e. penalization at distance with overcorrection lenses inhibited eye growth and natural refraction increase [23].

Phillips [24] reported that retinal image defocus influences eye growth and refractive development in schoolchildren in a manner consistent with the optical defocus in animal eyes, and thus supported the idea that childhood myopia progression could in principle be controlled by optical means.

The aim of this paper was to study the effect of binocular and alternating monocular myopic constant defocus prescribed in spectacle format on myopia onset and progression in children.

MATERIAL AND METHODS

129 children (258 eyes) aged 5–11 years were examined. The study protocol complied with the tenets of the Declaration of Helsinki of the World Medical Association regarding scientific research on human subjects. Informed consent was obtained from the subjects (parents) after explanation of the nature and possible consequences of the study. The study was approved by the Helmholtz Eye Research Institute human subjects Ethics committee.

The patients were divided into 4 groups.

The 1st group comprised 48 children (96 eyes) aged 5–8 years (6.5 ±0.35) with risk factors of myopia development: genetic predisposition to myopia (one or both myopic parents, among them 27; 32% – highly myopic), small reserve of age-specific hyperopia and pseudomyopia. The refraction before cycloplegia was -0.5 ±0.06 D, after cycloplegia +0.52 ±0.10 D. The patients were prescribed continuous wearing of plus lenses of 0.5–1.5 D (depending on the cycloplegic refraction), which induced myopia of 1.0 D. The children and their parents were ordered to wear the glasses during the whole day. The only exclusions were water-sports and some other activities during 1–2 hours 2 times a week, when they were allowed to take off the glasses.

The 2nd group comprised 46 children (92 eyes) aged 7–11 years (9.5 ±1.4) with low myopia from -0.75 to -2.25 D (before cycloplegia -1.46 ±0.13 D, after cycloplegia -1.29 ±0.10 D). Due to poor uncorrected visual acuity we adjusted alternating unilateral low myopic defocus. Two pairs of spectacles for continuous wearing were prescribed. One eye was corrected for distance to obtain residual myopia in spectacles of about 0.50 D and the fellow eye was corrected to obtain residual or induced myopia of about 1.50 D (lens from +0.75 to −0.75). If in the 1st case the refraction was -0.50 D and in the 2nd case it was -1.50 D, we used plane lens spectacles so that the natural defocus was preserved. The children wore spectacles alternatingly: 1 day the right eye was subjected to myopic defocus, the next day the same happened to the left eye [25].

The 1st control group comprised 15 children (30 eyes) aged 6–9 years (7.5 ±1.4) with pseudomyopia (refraction = −0.38 ±0.12 D before cycloplegia, +0.78 ±0.10 D after cycloplegia). The children were not administered any correction.

The 2nd control group comprised 20 children (40 eyes) aged 7–12 years (9.7 ±1.2) with low myopia (−0.97 ±0.15 D before cycloplegia, −0.83 ±0.11 D after cycloplegia). The children were administered conventional spectacle correction. Both eyes were corrected to obtain residual myopia 0.50–0.75 D, monocular distance visual acuity 0.7–0.8 (14/20–16/20) and binocular visual acuity 0.8–0.9 (16/20–18/20).

All subjects included in this study underwent a complete ophthalmic examination: visometry with and without correction (visual acuity was measured using a standard Snellen acuity chart at 5 metres (Snellen lines); automatic refractometry before and after cycloplegia; ultrasound biometry without cycloplegia with the measurement of axial length (AL) and horizontal diameter (HD) of the eyeball, anterior chamber depth (ACD) and lens thickness (LT); ophthalmoscopy of the central and peripheral retina; determination of the sensory dominance (binocular state; 4-dot Worth test); investigation of muscle balance (phoria) for far and near distance with a prism compensator and Maddox rod. To measure the

horizontal diameter of the eyeball we oriented an ultrasonic transducer perpendicularly to the optical axis at the equator of the eye with maximal deviation of the eye toward the nose. Horizontal diameter was measured three times, and HD value was stated as the average of the three measurements.

Mathematical treatment of the data included parametric and nonparametric methods. Statistical processing was done using Microsoft Excel. The main parameters for comparative analysis were the mean value (M) and the standard error of the mean (m). The significance level was determined with Student's t-test.

RESULTS

The 1st group was followed for 3–9 years (5.2 ±1.7), with control examinations after 1, 3, and 6 months, and then every 6 months. After 1 month of permanent low myopic defocus we observed a hyperopic shift in all patients. Ultrasound biometry revealed that this shift was caused by thinning of the crystalline lens and deepening of the anterior chamber. These changes persisted throughout the follow-up period. At the end of this period the refraction was +0.44 ±0.04 D before cycloplegia and +0.88 ±0.09 D after cycloplegia. Uncorrected visual acuity increased to reach 1.0. During the 9-year follow-up period no cases of myopia onset were observed.

Permanent wear of defocusing spectacles during the follow-up led to changes in echobiometric parameters of the eye: deepening of the anterior chamber by 0.29 ±0.05 mm ($p < 0.01$), flattening of the crystalline lens by 0.32 ±0.05 mm ($p < 0.01$), an insignificant average increase in the axial length by 0.34 ±0.04 mm ($p > 0.05$), and a significant increase in the horizontal diameter by 1.20 ±0.08 mm ($p < 0.01$); see Table 1.

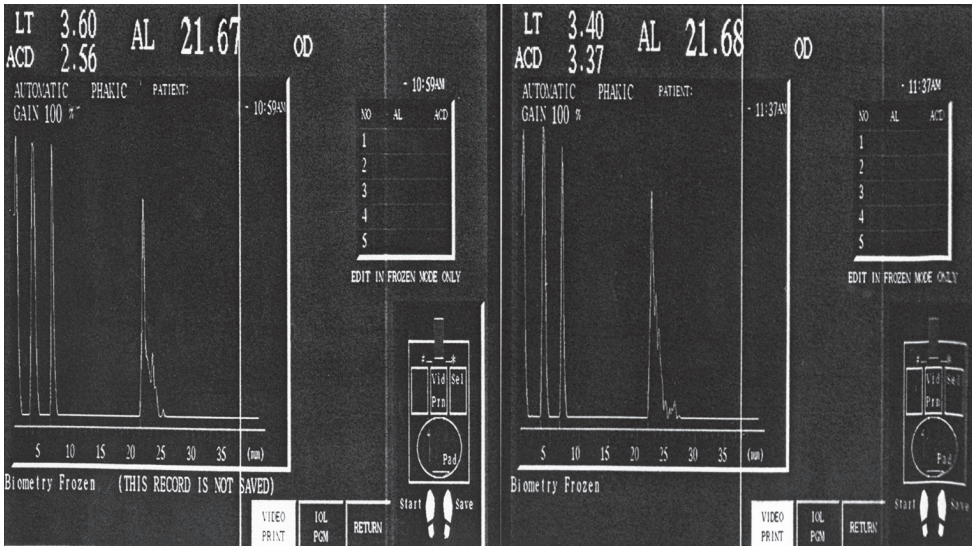
Obviously, the use of such emmetropizing factors, i.e. mechanisms of disaccommodation, reduced the refraction, eliminated pseudomyopia, and brought the focal point into coincidence with the retinal plane. Interestingly, the changes of the anterior chamber depth and the thickness of the crystalline lens initially induced by positive spherical lenses persisted over the follow-up period accompanied by inhibited axial growth. Horizontal diameter increased more than 3 times as much as the axial length (Fig. 1).

The 2nd group was followed for 3–6 years (4.1 ±0.9 years) with control examinations taken every 3 months. In 2 patients, alternating anisocorrection was discontinued after 6 months due to an excessive increase in exophoria and the development of variable exotropia. The initial exophoria in those patients exceeded the level of 10.0 prism D (12.0 prism D and 15.0 prism D, respectively). When alternating anisocorrection was discontinued the muscular balance was restored; yet the subjects were excluded from the study.

TABLE 1. Long-term changes of refraction, visual acuity and anatomic-optical parameters in the 1st group of patients

Follow-up period	Number of eyes	Refraction before cycloplegia D	Refraction after cycloplegia D	Uncorrected visual acuity	AL (mm)	HD (mm)	ACD (mm)	LT (mm)
Start	96	−0.50 ±0.06	+0.52 ±0.10	0.80 ±0.02	22.34 ±0.50	24.1 ±0.42	3.02 ±0.32	3.95 ±0.29
End	96	+0.44 ±0.04	+0.88 ±0.09	1.0 ±0.02	22.68 ±0.58	25.3 ±0.51*	3.31 ±0.34*	3.63 ±0.41*

AL – axial length; HD – horizontal diameter; ACD – anterior chamber depth; LT – lens thickness
* $p < 0.01$ as compared with the follow up start



AL – axial length

FIGURE 1. Changes of anterior chamber depth (ACD) and lens thickness (LT) in the 1st group of patients after one month of low myopic defocus

Of the remaining 44 children of the 2nd group who were still followed, 36 (81.8%) had stable refraction, which grew not more than 0.5 D over the whole follow up period. The mean value was -1.57 ±0.11 D. Uncorrected visual acuity remained the same (Tab. 2).

Visual acuity with optimal correction was 1.0 (20/20) in all children of the 2nd group, both at the start and the end of the study.

Ultrasound biometry revealed an insignificant increase in the axial length by 0.08 ±0.60 mm and a significant increase in the horizontal diameter by 0.75 mm ($p < 0.01$) – Table 2. At the start of the follow-up: AL = 23.92 ±0.62 mm; ACD = 3.60 ±0.29 mm; LT = 3.45 ±0.17 mm, HD = 23.95 ±0.11 mm. After 3–6 years of alternating anisocorrection: AL = 24.00 ±0.60 mm; ACD = 3.52 ±0.30 mm; LT = 3.43 ±0.16 mm; HD = 24.70 ±0.81 mm.

Over 2 years of alternating anisocorrection myopia progressed by 0.55–1.25 D (0.71 ±0.11) in 8 patients (18.2%) of the 2nd group (Tab. 3).

The analysis of echobyometric parameters revealed an increase in the axial length by 0.74 ±0.61 mm ($p < 0.01$), deepening of the anterior chamber by 0.42 ±0.35 mm ($p < 0.01$) with no changes in crystalline lens thickness, and an increase in horizontal diameter by 0.49 ±0.17 mm ($p < 0.01$) – Table 3. Due to myopia progression in those patients, we replaced the alternating defocus by other correction methods.

A 3-year follow-up in control groups revealed an increase in refraction both in children with pseudomyopia (1st control) and children with low myopia (2nd control group) – Table 4.

In the 1st control group myopia developed in 23 out of 30 eyes with pseudomyopia, which shifted the average value of non-cycloplegic refraction from –0.38 ±0.12 D to –1.07 ±0.10 D, cycloplegic refraction from +0.78 ±0.10 D to –0.60 ±0.07 D. The axial length and the horizontal diameter grew by 0.72 ±0.04 mm ($p < 0.01$) and 0.30 ±0.50 mm ($p > 0.05$), respectively.

In the 2nd control group myopia progressed by 2.05 ±0.10 D, the axial length increased by 0.68 ±0.52 mm ($p < 0.01$), and the horizontal diameter showed an insignificant increase of 0.20 ±0.50 mm ($p > 0.05$).

Twelve children (24 eyes) of the 1st group were followed up for 6–10 years (7.3 ±0.5). During the follow-up the children continued to use the correction prescribed. After 7.3 years the refraction was +0.23 ±0.03 before cycloplegia and +0.46 ±0.07 after cycloplegia, while the visual acuity with and without correction was 1.0 (20/20).

Ultrasound biometry revealed an increase in the axial length by 0.58 ±0.05 mm ($p < 0.05$) and the horizontal diameter by 1.31 ±0.09 mm ($p < 0.01$) during the follow-up (Tab. 5).

Thirty children (60 eyes) of the 2nd group were followed up for 5–9 years (7.1 ±0.6). At the end of the follow-up, the average refraction was –2.37 ±0.12 D before cycloplegia and –2.17 ±0.09 D after cycloplegia (Tab. 6). During the 7 years' follow-up the average increase of refraction was 0.73 D.

Of 30 remaining children of the 2nd group 20 had stable refraction (–1.79 ±0.08 D, change not more than 0.5 D) and in 10 patients myopia slowly progressed by 1.63 D.

Figure 2 shows echobiometric parameters in patients with stable and increased refraction. In patients with stable refraction, AL and HD increased by 0.18 mm ($p > 0.05$) and 0.83 mm

TABLE 2. Changes of refraction, visual acuity and anatomic-optical parameters in 36 patients with stable refraction

Follow-up period	Number of eyes	Refraction before cycloplegia D	Refraction after cycloplegia D	Uncorrected visual acuity	AL (mm)	HD (mm)	ACD (mm)	LT (mm)
Start	72	–1.60 ±0.05	–1.36 ±0.01	0.30 ±0.40	23.92 ±0.62	23.95 ±0.11	3.60 ±0.29	3.45 ±0.17
End	72	–1.78 ±0.03	–1.57 ±0.11	0.34 ±0.20	24.00 ±0.60	24.70 ±0.81*	3.52 ±0.30	3.43 ±0.16

AL – axial length; HD – horizontal diameter; ACD – anterior chamber depth; LT – lens thickness
* $p < 0.01$ as compared with the follow up start

TABLE 3. Changes of refraction, visual acuity and anatomic-optical parameters in 8 patients with myopia progression

Follow-up period	Number of eyes	Refraction before cycloplegia D	Refraction after cycloplegia D	Uncorrected visual acuity	AL (mm)	HD (mm)	ACD (mm)	LT (mm)
Start	16	–1.32 ±0.06	–1.22 ±0.03	0.34 ±0.12	23.63 ±0.75	24.32 ±0.23	3.09 ±0.43	3.48 ±0.21
End	16	–2.14 ±0.05*	–1.93 ±0.20*	0.19 ±0.20	24.37 ±0.63*	24.81 ±0.12*	3.51 ±0.32*	3.43 ±0.12

AL – axial length; HD – horizontal diameter; ACD – anterior chamber depth; LT – lens thickness
* $p < 0.01$ as compared with the follow up start

TABLE 4. Changes of anatomic-optical parameters in patients of control groups

Group No	Number of eyes	Follow up	Refraction before cycloplegia D	Refraction after cycloplegia D	AL (mm)	HD (mm)
1st control	30	start	–0.38 ±0.12	+0.78 ±0.10	22.93 ±0.60	23.9 ±0.55
		end	–1.07 ±0.10*	–0.60 ±0.07*	23.65 ±0.50*	24.2 ±0.59
2nd control	40	start	–0.97 ±0.15	–0.83 ±0.11	23.69 ±0.70	24.1 ±0.51
		end	–3.05 ±0.17*	–2.88 ±0.15*	24.37 ±0.60*	24.3 ±0.69

AL – axial length; HD – horizontal diameter
* $p < 0.01$ as compared with the follow up start

TABLE 5. Changes of refraction, visual acuity and anatomic-optical parameters in the 1st group of patients over the 6–10 year follow-up

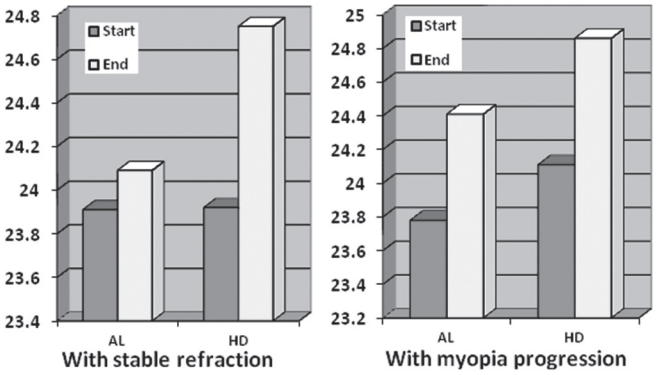
Follow-up period	Number of eyes	Refraction before cycloplegia D	Refraction after cycloplegia D	Uncorrected visual acuity	AL (mm)	HD (mm)	ACD (mm)	LT (mm)
Start	24	−0.34 ±0.11	+0.23 ±0.03	0.83 ±0.07	22.31 ±0.51	24.32 ±0.12	3.05 ±0.22	3.86 ±0.18
End	24	+0.21 ±0.05	+0.46 ±0.07	1.0 ±0.03	22.89 ±0.41**	25.63 ±0.37*	3.30 ±0.14	3.53 ±0.32

AL – axial length; HD – horizontal diameter; ACD – anterior chamber depth; LT – lens thickness
* $p < 0.01$ as compared with the follow up start; ** $p < 0.05$ as compared with the follow up start

TABLE 6. Changes of refraction and anatomic-optical parameters in the 2nd group of patients over the 5–9 years' follow-up

Follow-up period	Number of eyes	Refraction before cycloplegia D	Refraction after cycloplegia D	AL (mm)	HD (mm)
Start	60	−1.56 ±0.04	−1.41 ±0.11	23.86 ±0.11	23.98 ±0.09
End	60	−2.37 ±0.12	−2.17 ±0.09	24.20 ±0.12	24.79 ±0.24
Dynamics of parameters		0.81*	0.76*	0.34	0.81*

AL – axial length; HD – horizontal diameter
* $p < 0.01$ as compared with the follow up start



AL – axial length; HD – horizontal diameter

FIGURE 2. Changes of echobiometric parameters in the 2nd group of patients over the long-term follow-up period

($p < 0.01$), respectively. In patients with increased refraction, ultrasound biometry revealed a significant growth of AL by 0.63 mm ($p < 0.01$) and HD by 0.75 mm ($p < 0.01$).

DISCUSSION

Recent studies have shown that genetic predisposition to myopia and decrease of age-specific hyperopia reserve are risk factors of myopia development; and pseudomyopia, or excess of accommodation, is not only a risk factor but also a stage in the clinical manifestation of myopia [26]. The myopic type optical defocus of the image in patients of the 1st group triggered the emmetropization mechanisms, which included flattening of the crystalline lens and anterior chamber deepening, i.e. the mechanisms of desaccomodation, which provided pseudomyopia elimination and brought the focal point into coincidence with the retina. Eventually, these changes of anterior chamber depth and crystalline lens thickness initially induced by positive spherical lenses remained the same during the follow-up and were accompanied by the inhibition of the axial elongation. It is possible that the role which the accommodative-crystalline mechanism plays in the regulation of eye growth and refractive development is focused on the direction of the accommodative reaction: it may be positive or negative. In the 1st case the dynamic refraction will be increased, and in the 2nd case it will be reduced. It is also possible that the most important factor is not a particular type of ciliary muscle contraction but the periods of defocus which are inevitable and recur in the course of the day due to ciliary muscle fatigue and relaxation. In this case the defocus may either be hyperopic (if the accommodative apparatus was strained, and contributed to the increase of refraction), or myopic, as in the case of permanent wearing of low-positive lenses, which caused the accommodation to contribute to the decrease of refraction. The exact mechanism of optical image defocus impact on the refractive development of the human eye needs a further versatile study. However, the results obtained from the 1st group of patients enable us to assert even at this stage that the permanent wearing of low positive lenses in binocular format eliminates pseudomyopia and prevents its conversion to real myopia due to the inhibition of axial eye growth; at the same time active equatorial growth is observed, which contributes to crystalline lens flattening and anterior chamber deepening. At the initial stages of acquired myopia onset in children, as Mutti et al. showed, the emmetropizing factors such as crystalline lens flattening and anterior chamber deepening compensate for a time the axial elongation and the clinical manifestation of myopia [27]. Obviously, myopia occurs when the potentials of this emmetropizing mechanism are exhausted. This fact is confirmed if we compare the biometric parameters of the 1st (premyopia, pseudomyopia) and the 2nd (low myopia) groups. Children of the 2nd group showed a significantly lower crystalline lens thickness than those of the 1st group (3.48 ±0.25 mm against 3.95 ±0.29 mm; $p < 0.01$) and a significantly deeper anterior chamber (3.58 ±0.34 mm against 3.02 ±0.32 mm; $p < 0.01$). At the same time, further changes of these parameters are practically impossible (see Table 2 above).

Thus, low myopic defocus induced by positive spherical lenses and natural defocus induced by the onset of myopia have the same effect on the accommodative-crystalline apparatus, which responds by decreasing the dynamic refraction of the eye. It is

known, however, that natural low myopic defocus, in contrast to artificially induced defocus, does not impede further myopia development; otherwise the whole of the myopic refraction would stop in its development at this stage. Possibly, positive spherical aberrations induced by a plus-lens have a strong effect in the 1st group. This may be due to the particular stage at which the induced defocus was applied: before the myopia was clinically manifested or after, when all reserve mechanisms are exhausted. However, the obtained results show that dosed low myopic defocus inhibits myopia progression even at this stage. The singularity of the proposed method of alternating monolateral defocus is induced anisometropia, which definitely affects the binocular functions and muscular balance. The state and dynamics of these parameters are very important for determining the possible contraindications and side-effects of the proposed method of correction. They can also help to reveal the mechanisms of myopia progression and enhance the potential of its prevention. These parameters will be the object of future work.

CONCLUSIONS

1. Permanent low myopic defocus of the image in binocular spectacle format inhibits eye growth and refraction shift to myopia in children with low hyperopia, emmetropia and low myopia.
2. The proposed method of alternating monolateral low myopic defocus arrests myopia progression in 81.8% of children with low myopia for 4 years, 66% for 7 years.

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