

Article ▶ Myopia and Myopia Control: Options and Outlooks

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ABSTRACT

Though myopia has been studied throughout history, the topic has gained significantly more attention over the past few decades. An internet search displays over 5 million results and over 15,000 articles written on the topic. Yet there is great debate as to the clinical efficacy of current methods in myopia control and our duty as physicians to incorporate a means of intervention into our treatment protocol. In clinical care, many of the disease management treatments are not curative; they are simply supportive. Yet doctors willingly accept these therapies as the standard of care. Myopia control should be thought of in the same respect: though you cannot cure it or reverse it, and though the protocol is not equally successful for every patient, treatment is still worthwhile in many cases. In this article, we will review relative data to compare current theories of myopia control and their efficacy.

Keywords: atropine, myopia, myopia control, orthokeratology

Introduction

Myopia, present throughout history, has been studied for the past 150 years, and at least 15,000 articles have been written on the topic.¹ Yet our progress to find an effective solution has been slow. If something could be done to prevent a child from experiencing life as a high myope, surely the profession as a whole would take action. However, there has been great debate as to the clinical efficacy of current methods in myopia control and our duty as physicians to incorporate a means of intervention into our treatment protocol.

Normal Refractive Development

Before we consider abnormal refractive progression, we should examine the normal process by which a child's refractive status changes. Infants have an average cycloplegic refraction of +2.16 D at three months of age that decreases to +1.36 D at nine months.² In addition, 69% of newborns have ≥ 1.00 D of astigmatism;³ however, research shows that this astigmatism is likely to be reduced by roughly two-thirds between nine and twenty-one months of age.⁴ The emmetropization rate varies according to the magnitude of the prescription, with larger hyperopic and myopic refractive errors normalizing quicker. There is a significant and quick change toward emmetropia during an infant's first year. The process slows substantially but continues until around age two for hyperopes and age four to five for myopes and astigmats.⁵ The mean spherical equivalent refraction at age six, after the emmetropization process is usually complete, is between +0.70 D and +1.00 D.^{6,7} However, those children with moderate hyperopia may continue to experience a very small lessening effect up to age ten.⁸ Anisometropia is more prevalent in young children than adults; however, some may be transient due to different rates of emmetropization between the eyes.⁹ For children with more than 3.00 D anisometropia at one year of age, it is very likely to remain, and approximately 60% of those will develop amblyopia.¹⁰

It is suggested that prescribing even partial correction at a very young age may have a negative effect on the emmetropization process for the child, but more studies are needed.¹¹ However, the interference from a prescription is clearly evident in animal studies. Also, it is argued that if adults wearing monovision contact lenses can develop a consequential refractive error, the system of a young child is even more pliable and easily influenced.¹² In order to leave a strong stimulus for emmetropization, a good rule of thumb is to prescribe the amount of plus, minus, or cylinder that leaves young children just above the mean refractive error for their age. This will ensure they have a stronger-than-average stimulus for emmetropization.⁵ It is particularly important, though, to monitor partially-corrected hyperopic children closely for strabismus and to increase the prescription accordingly if needed. Since emmetropization is essentially complete by age six, it is common practice to prescribe full correction for all school-aged children.⁵ An astigmatic prescription may be titrated to allow for adaptation, and a small hyperopic prescription ignored.⁵

Theories of Myopia Control

As early as 1864, Donders hypothesized that myopia was correlated to the overuse of near vision. This belief continued to prevail, as studies demonstrated that participants with higher education exhibited a higher incidence of myopia.¹ Today, we have many new theories to consider.

Why is myopia control receiving so much attention now? There is significance in the timing due to the recent trends that have been documented. Myopia is on the rise, with its prevalence increasing from 25% in the early 1970s to 41.6% in the mid-2000s in the United States alone.¹³ It is important to note that the methods and comparative data were identical in both studies and that the increase among African Americans was even more substantial, rising from 13% to 33.5%. A Taiwanese study reported that over 84%

of the 11,000 children enlisted were myopic, with 12% of the boys and 18% of the girls having a refractive error greater than -6.00 D.¹⁴ Observation of a higher myopic prevalence among industrialized communities, as well as the increase in myopia from young children to young adults, indicates an environmental factor influencing myopic progression. If this link can be determined and targeted as a way to retard myopic progression in children, millions of lives will be impacted. Not only should we consider the social, economic, and educational inconvenience of high myopia, but also the physiological ramifications.

Myopia can be pathological (onset before age six), school-age onset, or adult onset. School-age onset is most likely to progress but stabilize by the early twenties, while adult onset is generally related to one's ability or demand to accommodate. All three types involve axial length elongation. The current rate of progression in American children, ages eight to thirteen, is estimated at -0.55 D per year. The majority of the studies look at children between the ages of six and thirteen, in order to capture the time period in which myopia tends to progress the fastest.¹⁵ Most of the recent studies use axial length measurement as an indicator of progression along with refractive error. Though it is believed that the lens and cornea also play a role, their impact is still unclear.¹

The etiology of myopia is not completely understood. However, it is well known that there is a genetic component, as revealed in studies showing more myopia in monozygotic twins (single fertilized egg splitting into two cells) versus dizygotic twins (two different fertilized eggs).¹⁶ One study examining 1,224 twins showed that genetics are a factor even in the development of adult-onset myopia.¹⁷ There have been multiple links made between educational attainment and degree of myopia, but these same researchers concluded that even education level is somewhat linked to genetics.¹⁸ There are also studies examining familial influence and the increased incidence with two myopic parents versus one myopic parent.^{19,20}

Many studies support different theories of environmental influence on the development of myopia. Researchers have looked at similarities in the incidence of myopia within isolated communities as well as among ethnicities. Other studies evaluated the impact of the frequency of near and computer activities as well as outdoor activities before and after myopia onset.¹ There were conflicting results from different studies regarding the influence of near activity, but it was reported that myopic children did spend less time participating in outdoor activities both before and after the onset of myopia.²¹ Researchers Raviola and Wiesel were able to induce axial length elongation and myopia by partially suturing the lids of monkeys to create form deprivation.^{22,23} Chen demonstrated that myopia can be induced in chicks through form deprivation (environmental influence), but that some chicks were more susceptible to that influence due to their genetic make up.²⁴ It is common consensus that one's degree of myopia is due

to a combined influence of both environmental factors and genetics,¹ but the challenge is in being able to truly separate the two.

There is an existing concept that accommodation is somehow related to myopia and axial length elongation. This was the basis for attempts in the early 1900s to use vision training to reduce myopia.²⁵ While the vision therapy may have improved visual acuity, it did not change refraction.²⁶ Visual training studies were conducted as late as the 1980s but did not show any greater success.²⁷ In early animal studies, Young limited the viewing distance of monkeys to less than 20 inches for up to a year, and thereby induced -1.00 D to -2.00 D of myopia, possibly supporting the idea of excessive accommodation as a causative factor.^{28,29} Young postulated that extensive accommodation induced permanent changes in the lens structure, which created a secondary rise in intraocular pressure and a resultant deepening of the vitreous chamber.³⁰ Interestingly, in other early animal studies, up to -7.00 D of myopia was induced by keeping the monkey's head lower than his body for six hours per day for five to six months.¹ However, the connection between head posture and refractive state was never fully understood.

As animal studies expanded, myopia experiments were conducted using chickens, tree shrews, gray squirrels, mice, guinea pigs, cats, American kestrels (another bird), and even fish, supporting the hypothesis that there is a common mechanism of influence in play.¹ Chicks are often used because they can be manipulated as soon as they hatch and their visual system responds within hours.³¹ When plus or minus lenses are placed in front of the chick eye, the focal length of the eye is quickly regulated by directional modification of choroidal thickness (i.e. thinning or thickening) as an immediate response. This is followed by a hurried or slowed rate of eye growth to permanently affect the focal length and refractive state of the eye in response to the induced myopia or hyperopia.³² A recent study investigated the variation of choroidal thickness among high myopes using optical coherence tomography.³³ The researchers found that there is a direct correlation between choroidal thickness and axial length in high myopes. For each millimeter decrease in axial length they found an associated decrease in choroidal thickness of $25.9 \pm 2.1 \mu\text{m}$.

When high plus or minus lenses are used in animal studies, the axial length is shown to grow in a manner driven towards emmetropia with a speed relative to the degree of blur induced.^{34,35} Therefore, some hypothesize that if there is near blur due to accommodative lag, the eye may elongate in an attempt to correct the hyperopic blur.³⁶⁻³⁹ However, other researchers blame limited exposure to sunlight as the actual cause, as opposed to near activity.⁴⁰ When animal subjects had the nasal half of their visual field obscured by occlusion, blur, or light obstruction, it resulted in a corresponding axial elongation of the temporal retina and asymmetry in the shape of the eye.^{41,42} Varying the degrees of light obstruction altered the degree of myopia induced. This was done in the absence

of accommodation or even an intact optic nerve.⁴¹⁻⁴⁴ Also, axial length changes in response to induced blur occurred even after the macula had been ablated.⁴⁵ When peripheral blur is induced, yet central clarity is preserved, there is still resultant myopia.⁴⁶ Each of these variations indicate that the peripheral retina is more influential than the macula or optic nerve in axial elongation.¹⁵ A human example of this process occurs in infants with unilateral congenital cataracts presenting with concurrent axial length elongation.⁴⁷ It is known that myopic children exhibit peripheral relative hyperopia; however, there is conflicting data as to whether or not peripheral hyperopia can be used to predict the onset of myopia.¹

Ultimately it is believed that as the eye becomes myopic and elongates, it takes on an elliptical shape, inducing hyperopic defocus peripherally as the image now falls behind the retina in the periphery. This hyperopic blur is exaggerated further as the child accommodates at near.¹⁵ The hyperopic peripheral blur provokes a biochemical neuro-retinal signal for axial length elongation, thus prompting myopic progression.⁴⁸ Our attention is focused on disrupting or eliminating this signal for elongation. This is the motivation behind steering away from traditional lenses that only correct the central blur and using pharmacological agents such as atropine or pirenzepine to interrupt the signal.¹⁵ Keep in mind that even for the most agreed-upon theories, there is an alternate opinion. Logan suggests that Chinese people have more axial symmetry than Caucasians, therefore peripheral relative hyperopia may not be a universal causative agent among all ethnicities.⁴⁹

Spectacle Correction

As far back as the 1880s, practitioners were taught to prescribe the least amount of minus that allowed the patient to achieve clarity, while limiting accommodation.⁵⁰ From this, a common approach to lessening myopia progression was to under-correct the myopic patient by as much as 1.00 D. Though this was a longstanding and widely accepted approach, the reasoning was not well substantiated. Later research showed that spectacle correction had no effect on myopic progression, regardless of the child's wear time.⁵¹ Furthermore, in 2002 Chung⁵² demonstrated that under-correcting myopes by 0.75 D caused an increase in the rate of myopia progression and Goss⁵³ reported that overcorrecting by -0.75 D did not influence myopia progression in either direction.

In the 1940s, efforts to reduce myopic progression involved the use of bifocals in children. In theory, incorporating the bifocal or multifocal reduces the lag of accommodation and thereby reduces the hyperopic blur and secondary signaling for myopic progression.¹⁵ The Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error (CLEERE) Study Group, which examined more than 1,100 children, determined that while myopic children do exhibit an increased lag of accommodation, it is not an accurate measure by which to predict the rate or degree of myopia.⁵⁴ Goss found that using bifocals did not cause a statistically significant difference

in progression for the group as a whole, but among esophoric children or those with a near cross cylinder of greater than +0.50D, the difference was quite significant (40% and 48% reduction, respectively).⁵⁵ Data from studies by Grosvenor and Fulk showed a similar reduction for esophoric children.⁵⁶⁻⁵⁹ However, Cheng's study, using high fitting executive bifocals with a +1.50 add and 3BI prism OU, demonstrated a 45% overall reduction in myopic progression among Chinese children.^{60,61}

When Leung and Brown looked at the efficacy of progressive addition lenses (PALs) versus single vision lenses, they found a statistically significant decrease in the degree of myopic progression for the group as whole; however, they were not masked to the treatment protocol.⁶² Another larger study, in which the researchers were masked, looked at children of similar ethnicity and saw no effect from progressive spectacles.⁶³ The Correction of Myopia Evaluation Trial (COMET) evaluated 469 children over the course of three years.⁶⁴ They compared myopic progression in children wearing PALs versus single vision but found only a clinically insignificant 0.20 D decrease. Two more recent studies evaluated the effect of progressive lenses on myopic children with near esophoria and large lags of accommodation to find a statistically significant but clinically insignificant effect.^{65,66} Overall, the results using bifocals and progressives are conflicting and not very supportive of a successful method for myopia control.

While bifocals and progressives reduce the accommodative demand for the child, they do not address the peripheral hyperopic blur. One study used three different designs that attempted to eliminate the peripheral blur, however they did not induce a statistically significant decrease in myopic progression.⁶⁷ A significant limiting factor to consider is that there is no control over where the child looks within the lens. This might lead one to assume that results may be enhanced through the use of contact lenses as opposed to spectacle lenses. However, studies showed no statistically significant difference in myopia between soft lens wearers and spectacle wearers.⁶⁸ In addition, any refractive difference between daily soft lens wearers and RGP wearers was attributed to the secondary corneal flattening, as the axial length for each group was not significantly different.⁶⁹

Orthokeratology

Orthokeratology (ortho-K) was first introduced in the 1960s. Morrison was the first to notice that myopia progression in children was slowed with the use of hard lenses.⁷⁰ Ortho-K lenses are unique in that while they flatten the central cornea, they actually steepen the mid periphery of the cornea. This would theoretically eliminate the hyperopic peripheral blur or even induce a myopic peripheral blur, both of which would disrupt the biochemical neuro-retinal signal causing myopic progression. In fact, multiple studies have found ortho-K to be effective in delivering somewhere between a 30% and

46% reduction in myopia (measured via refraction and axial length).¹⁵

Cho⁷¹ showed a 46% reduction in axial lengthening in the ortho-K group versus the spectacle group, while Kakita⁷² showed only a 36% difference in axial length between his ortho-K and spectacle groups. Walline demonstrated a 38% decrease in axial length between children corrected with ortho-K and those wearing soft contact lenses.⁷³ There were two relatively long-term studies done examining the efficacy of ortho-K. Kwok-Hei's seven-year study revealed an average rate of myopic progression for spectacle-corrected children to be -0.29 D/yr. versus -0.05 D/yr. in the ortho-K group.⁷⁴ His study did not include axial length measurements, but there was a wash out period for normalization of the corneal shape before the final refraction was obtained. Hiraoka looked at axial length changes in spectacle wearers and ortho-K patients over the course of five years.⁷⁵ He found the difference to be 30%, but it was only statistically significant over the first three years. This brings into question the long-term sustainability of ortho-K treatment for myopia control. At least one case report shows an increased rate of axial elongation following the discontinuation of ortho-K.⁷⁶ There is consistent positive support for this approach in the short term, but there is certainly a need for well-controlled long-term studies.

Specialty Soft Lenses

Another method of correcting the central myopic refraction while addressing peripheral hyperopia is through new novel contact lens designs utilizing dual focus zones. After comparing 10 months of wear with this type of lens versus a control lens, Antstice and Phillips documented that myopia progression (measured by axial length and cycloplegic auto-refraction) was reduced by >30% in 70% of the children.⁷⁷ Holden evaluated another novel lens design and found a 56% reduction in myopic progression compared with spectacles.⁷⁸ He also noted an improvement in peripheral visual acuity and contrast sensitivity compared to traditional lenses, with no sacrifice of central visual acuity whatsoever.⁷⁹ However, similar to PALs and ortho-K, some studies point to reduced efficacy over time, noticed as early as six months. Kollbaum compared the visual performance and tolerance in subjects wearing MiSight (a dual focus soft lens by Cooper Vision available in Hong Kong) versus Proclear multifocal +2.00 D add (D).⁸⁰ There was visual compromise from both lenses compared to habitual correction, but there were no significant differences between the two, even at varied illumination and contrast. None of these experimental designs are currently available in the U.S.; however, one might assume that this would be a possible application for the Biofinity multifocal (D) lens. It is a silicone hydrogel lens with a central area of full myopic correction and an aspheric periphery of increased hyperopic correction; however, this lens has not been tested for this purpose.¹⁵ Acuvue Oasys for Presbyopia also has a distance-center design, with alternating peripheral rings.

Atropine

Atropine was used as early as the 19th century to slow myopic progression based on the principle that it reduced accommodation and would thereby reduce myopia progression. Studies have since disproved this correlation. Examples include using form deprivation to induce myopia in the gray squirrel, who has no accommodative ability, in chicks with severed optic nerves, and in fish, who do not have lenticular change in shape with accommodation.¹ Atropine did not remain as a strong avenue for correction due to its well-known side effects of near blur and photophobia. However, in the mid-1900s interest was renewed as more studies were released documenting its efficacy. Bedrossian showed that when 75 children, aged seven to thirteen, used 1.0% atropine in only one eye for one year, there was a mean decrease in myopia of 0.21 D versus an average myopic increase of -0.82 D in the fellow eye.^{81,82} When the treatment eyes were switched, the newly treated eyes showed a 0.17 D mean decrease in myopia after one year, while the untreated eyes showed an average myopic increase of -1.05 D. This was not the only study to show a greater progression in myopia after atropine discontinuation. Kennedy et al. looked at 214 children who were treated with 1.0% atropine daily for a median of 3.35 years.⁸³ The treatment slowed myopic progression significantly, however after discontinuation of atropine, the children previously treated progressed more quickly at -0.22 D/year versus the control group at -0.13 D/year. It is important to consider this but also to understand that the overall myopic change was still significantly less for the treatment group as compared to the control group.

Once the efficacy of atropine was confirmed, studies became much more elaborate, comparing multiple factors simultaneously. One such study examined the effects on children using 0.5% atropine with multifocal spectacles compared to a placebo drop with multifocals and a third group using a placebo drop with single vision specs.⁸⁴ The researchers concluded that the myopic reduction was attributable to atropine alone. Multiple studies compared the efficacy of atropine in different concentrations. The most notable may be the Atropine for the Treatment of Childhood Myopia study (ATOM) and ATOM2.^{85,86} They were both 2-year studies in which only one eye was treated with atropine, and the children wore progressive, photochromic lenses. Ultimately, between the two studies, the following atropine concentrations were compared: 1.0%, 0.5%, 0.1%, and 0.01%. Mean myopic progression was -0.60 D/year in the control group (untreated eyes). Mean progression in the 1.0% atropine treated group was -0.14 D/year, which represents a 77% reduction in myopia progression. The mean myopic progression among the other concentrations ranged from -0.15 D/year in the 0.5% group to -0.24 D/year in the 0.01% group. The researchers concluded that 0.01% has nearly no side effects and has comparable efficacy to the 0.5% concentration. Another study also reported a 77% reduction in myopic progression when atropine concentrations were varied according to the season.⁸⁷

Children used 0.1% in the summer, 0.25% in the spring and fall, and 0.5% in the winter. The concentration and usage was also reduced for very low myopes.

When discussing placing a child on a medical treatment for a sustained period of time, it is important to consider two things: potential adverse events and the longevity of treatment efficacy. Several of the atropine studies simultaneously tracked patient symptoms. As expected, there was a high rate of photophobia and near blur, and the incidence and severity of complaints were directly proportionate to the concentration administered. However, quite often, the complaint of photophobia subsided after several weeks of treatment and was very well tolerated. In several studies, children were prescribed photochromic progressive spectacles, which helped to alleviate both of these symptoms from the start. Despite the utilization of photochromic UV protection, one must consider the potential risk of added UV exposure to the lens, retina, and ocular surface secondary to a constantly dilated pupil.¹ Other reported side effects were ocular discomfort, ocular allergy, headaches, bad taste, dry mouth, and dry eyes in extremely small percentages.⁸³ Of note, those who used atropine concentrations of 0.25% or less had no complaints whatsoever.⁸⁸ Although serious and fatal side effects are possible (at 20 times the minimum dose), none were reported in any of the myopia or amblyopia studies involving atropine.¹⁵ In the ATOM study, amplitude of accommodation and near visual acuity were tested six months after the discontinuation of atropine, and there were no negative consequences reported.⁸⁵ Luu et al. evaluated retinal function via multifocal electroretinograms and reported that it was unaffected after cessation of atropine treatment.⁸⁹

Children in the ATOM study were evaluated for an additional year after the two-year atropine treatment course. During the first year following atropine cessation, their mean myopic progression was -1.14 D, compared to -0.38 D in the untreated eye.⁹⁰ The progression was significantly slower in the second half of the year, and axial length did not actually change after the atropine was discontinued. It was hypothesized that the presumed myopic progression may actually be much less than it appeared due to the patient's greater accommodative ability during the post-treatment refraction. During the study, refractions were measured while the patient was on atropine. However, cyclogel 1.0% was used for both the baseline refraction and any post-treatment refraction. Naturally, atropine has a much greater impact in relaxing accommodation compared to cyclogel. Because of this, it would appear that the initial beneficial effect of atropine was artificially high, as well as the apparent rebound effect after discontinuation of treatment.

Originally, it was believed that atropine worked by inhibiting accommodation, but that theory was disproved. McBrien was able to induce axial elongation via partial field deprivation with translucent lenses and accommodation via 10% carbachol.⁹¹ Unilateral intravitreal atropine injections

were shown to prevent axial elongation independent of accommodative influence. Understanding that atropine, a non-specific muscarinic antagonist, works via a non-accommodative mechanism has lead researchers to believe that the muscarine receptors in the retinal pigment epithelium are involved in axial elongation and refractive error.⁹² This has inspired a search for other muscarinic antagonists that do not affect pupil dilation or accommodation. Pirenzepine is not available in the U.S., but studies have shown it to have moderate efficacy in slowing myopic progression without significant dilation or near blur.⁹³ It does not, however, seem to be as effective as atropine. In a multi-center study of 174 children, there was a mean prescription change of -0.58 D after two years, compared to -0.99 D in the placebo group, however the drop out rate due to side effects was greater than in the studies using low concentrations of atropine. Furthermore, the axial length was not significantly different from the placebo group. Chen looked at racanisodamine 0.5%, another non-selective muscarinic antagonist, to find that it did not affect accommodation and triggered only a modest dilation, making it a good potential candidate for further study.⁹⁴

Time Outdoors

The amount of time spent outdoors has gained significant attention as potentially having a protective effect on prevalent myopia, incident myopia, and myopic progression. Recently, Sherwin conducted a systematic review and meta-analysis of the work that has been done.⁹⁵ After aligning with inclusion criteria, the 1623 reports were narrowed down to 23 studies that could be examined for a comparative review. Ultimately, the data review included approximately 10,000 children and teens. Sherwin concluded that for every additional hour of outdoor time per week, there was a 2% reduced chance of myopia. Similarly, additional time outdoors slowed myopia progression and reduced axial length.⁹⁶ Guggenheim conducted a study that began with over 9000 children, and he also reported that increased time outdoors, especially between ages eight and nine, reduced the incidence of myopia.⁹⁷ The mechanism by which this occurs is not understood; however, there are multiple hypotheses. Possibly, the sunlight triggers a dopamine release in the retina that inhibits axial elongation,⁹⁸ the increased light intensity slows elongation by means of pupil constriction and secondary blur reduction,⁹⁹ or outdoor activities require less accommodation which lends a protective effect.¹⁰⁰

This is an encouraging correlation but is by no means straightforward. If there is a true link, it spurs the question of which came first, reduced time outdoors or myopia. Maybe myopes are of the personality type to enjoy more reading and less physical activity or time outside,¹⁰¹ or they are more likely to reside in urban communities where access to direct sunlight is restricted.¹⁰² If time outdoors does protect against myopic progression, it would be a legitimate assumption that myopic progression would exhibit some degree of seasonal variation. Some reports have shown such a seasonal swing.^{100,103} However,

when Fujiwara looked for seasonal patterns among Japanese children, he found that while there was a significant decrease in axial elongation during summer months, the amount of myopic progression on refraction was unaffected.¹⁰⁴ Of course, if additional studies show it to be effective, it is important to know whether its efficacy is consistent in regards to ethnicity, age, family history, frequency of near activity, and home region (especially since many of the studies were conducted in East Asia). Hopefully, future studies will also enlist more quantitative measurements of exposure such as conjunctival autofluorescence or vitamin D levels.^{105,106} While the actual key component is unknown, its efficacy is clearly related to the light and not the activity, because when children engage in indoor sports, there is no protective effect.^{96,99} Interestingly, bright UV-free light also slowed myopic progression in animals,¹⁰⁷ so there are many questions yet to be answered.

Myopic Consequence

Much of the drive to limit myopia in children is to prevent them from enduring the unwelcome consequences that it carries. The effects of myopia reach far beyond its inconvenience. Wearing a thick prescription or constantly sitting up front as a child can have many negative repercussions on their athleticism and self-esteem, which can certainly shape a child's personality. There is also a financial burden incurred, as some progressive myopes may have to change their prescription biannually.

In addition to the social, emotional, and financial effects, there are anatomical ramifications. Myopic patients are exposed to unique risks due to their elongated axial length and secondary structural challenges. While refractive surgery may eliminate a patient's myopic refraction, it does not eliminate the pathological threat associated with myopia.

The drive for refractive surgery may have decreased somewhat compared to its early years, but many people are still making the decision to have surgical correction. By now we should have a clear picture of any long-term comparisons between PRK and LASIK, however very few prospective studies have been conducted. LASIK, a mainstream mode of correction for myopia, is certainly a valuable solution, but it does not come without its own risk.

A recent study compared the stability of corneal aberrations and power at seven years post-LASIK versus post-PRK.¹⁰⁸ PRK patients achieved corneal stabilization by year 1, but the post-LASIK corneas continued to change throughout the entire 7-year study, increasing in corneal power, coma-like, and spherical aberrations. The researchers could not pinpoint any preoperative markers as to which patients might experience more or less stability. But they did determine that any PRK or LASIK-induced coma-like and spherical aberrations do appear to be permanent. Another

study reported on results at ten years postoperatively after LASIK.¹⁰⁹ Of the 97 eyes evaluated, 73% had a refraction of +/-1.00 D, and 92% were +/-2.00 D after ten years. In this study, there was no incidence of corneal ectasia.

Vision fluctuation is a common complication following refractive surgery. A recent study enrolled 34 patients who had LASIK performed on one eye and PRK on the other. Patients complained of more fluctuating vision in the PRK eye during the first month after surgery compared to the LASIK eye.¹¹⁰ Otherwise, all self-reported symptoms were equivalent for both procedures at each visit and had returned to baseline by the 1 year post op visit.

Future Studies

Studies investigating myopia and myopia control are being conducted at a faster pace and with greater intensity than ever before. Yet, our understanding is still very limited, and our current solutions offer only modest results. Future testing will include new pharmaceutical agents as well as uniquely designed contact lenses, but researchers will continue to explore the impact of basic lens power. McBrien et al. discovered that when tree shrews wore a plus lens for one hour per day, it negated the progressive effect of wearing a high minus lens the rest of the day. Subsequently, the mission is now to determine the appropriate plus power needed and the suitable wear time and age at which to apply the treatment. Once this is established, they believe it may be an extremely effective tool in preventing and controlling the progression of myopia in children.¹¹¹

Clinical Application

Too often, some practitioners feel that they are not obligated to intervene in myopia progression because there is no one clear answer that works for everyone. But it is the optometrist's responsibility to inform young myopic patients and their families about available options and their potential to slow progression. When we spend time thinking about the effect of myopia on the individual and on society, we may realize the need to take a more active role with these patients. There are multiple options, and while their efficacy varies, one option is not necessarily the right solution for every patient.

Consider different options for approaching different patients, according to their needs and lifestyle.¹⁵

- Consider atropine (at a low concentration or prescribed with photochromic progressives) for a patient with:
 - ~ Greater risk
 - ~ Rapid onset
 - ~ Rapid progression
 - ~ Strong family history
 - ~ More sedentary lifestyle
 - ~ Expect ~58-90% reduction

- Consider ortho-K for a patient who is:
 - ~ Less progressive
 - ~ More active
 - ~ Athletic
 - Expect ~30-46% reduction
- Consider Biofinity D or Acuvue Oasys (off label use):
 - o If the parents have objections to drugs or overnight CL wear
 - Expect ~40-56% reduction
- Consider progressive spectacles with or without BI prism for:
 - ~ An esophoric child
 - ~ The least invasive option
 - Expect ~20-45% reduction
- Consider increasing the child's time outdoors:
 - ~ As an add-on to all other protective treatments
 - ~ For every myopic child
 - Expect ~2% reduction per additional hour outdoors

Any treatment undergoes an experimental phase and growth phase before being adopted as the standard of care. Unfortunately, this process can take decades. In clinical care, many of the disease management treatments are not curative; they are simply supportive. Yet, doctors still initiate these therapies, and they are widely accepted as the standard of care. Think of myopia control in the same respect. Though you cannot cure it or reverse it, and the protocol is not equally effective for every patient, the treatment is still worthwhile; it is still beneficial to do something rather than nothing.

Much independent research is being done, but it seems as though corporate industry may have abandoned the topic. Perhaps if the myopic child is already buying glasses or contact lenses, it does not promise great potential for additional corporate revenue. But even without industry leading the way, there is a need for the professional leadership of optometry to set forth guidelines and to help establish standards. Optometry as a profession is slow to change and slow to adopt new protocols. Currently it is up to the individual practitioner to be astute enough to acknowledge the need, compassionate enough to take the time, and bold enough to incorporate protective strategies when there are no guarantees.

Conclusion

Eye care physicians are being called upon to look at patients with a more holistic view. You are challenged to go beyond the eye and get involved in the systemic health and nutrition of the patient. It is well known how intricately these systems overlap, and the goal is not simply to address the patient's chief complaint, but to do all we can to influence their overall health. Some doctors approach this challenge more aggressively than others. Similarly, some doctors will embrace the opportunity to

impede the myopic progression of their young patients, while others will ignore the research. For those who embrace this science, it will allow them to do more than just spin the dials and prescribe. It will allow them to differentiate themselves as true optometric physicians and easily create a niche for patient referrals and practice growth.

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