

Report: OEPF Lecture • Myopia Management—How to Apply the Current Scientific Evidence to Our Clinical Practice

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The objectives of this presentation are that participants are able to: (1) apply to their clinical practice the current knowledge of myopia etiology, development, and progression; (2) educate families on myopia prevention and control options; and (3) practice evidence-based clinical care when managing myopia, including information on the possible benefits and risks of each treatment, determining the most appropriate length of treatment and understanding how to gauge success.

The first consideration is to define myopia, important for the management of myopia as well as the education of parents and children. Optically, myopia happens when distance images focus in front of the retina, rather than at the retina; we can use ophthalmic lenses to move the images back to the retina. It is important to understand that mismatch is typically due to an increased elongation (axial length) of the posterior segment of the eye.

Parents will likely want to know how frequent the myopia problem is. Currently, about a third of the world's population has myopia, heading for a majority of the population sometime around 2050. What is even more worrisome is that the prevalence of high myopia is also increasing. Prevalence does vary around the world, with the highest rates (80-99% of the "young" population) being found in south and southeast Asia.

When discussing myopia management options with parents, it is necessary to explain why it is important to control myopia. Myopia impairs distance vision when uncorrected (this is well-known), but parents may not know that myopia also impairs vision when corrected due to the stretch in the receptor grid of the retina caused by the excessive lengthening of the myopic eye. Higher amounts of myopia also limit the individual's options for refractive surgery later on. Overall, financial costs related to myopia in the U.S. exceed those of heart failure, lung cancer, or breast cancer. Even more importantly, myopia poses a significant risk for certain vision-threatening conditions such as myopic maculopathy or retinal detachment through retinal thinning. Myopic maculopathy, a relatively new condition, is already the second leading cause of blindness in Beijing, the third in Ireland and Israel, and the fourth in the UK—and these statistics are over 10 years old. Myopia is also associated with increased risk of primary open angle glaucoma and earlier onset of posterior subcapsular cataracts, as well as higher risk for complications after cataract extraction. The risk for these conditions increases with increasing amounts of myopia, but even low to moderate myopias pose higher risks than no myopia.

If we don't do anything to prevent it, a child with myopia will continue to progress until the late teens or early twenties, ending with higher myopia in adulthood if their myopia started earlier on. The good news is that we now have treatments available that can slow down, or even halt in some cases, the progression of myopia, so that individuals end with lower amounts. This is important because slowing myopic progression by just 1 diopter (e.g., ending with -5.00 instead of -4.00) may reduce the chance of myopic maculopathy

by 40%, retinal detachment by 30%, and primary open angle glaucoma by 20%.

Another question from parents that we may be asked is why their child got myopia. The myopia epidemic has shifted the world's viewpoint to acceptance now that the etiology of myopia is primarily environmental. Genetic disposition comes into play but may account for less than 10% of the variability. The environmental view reflects Skeffington's work, as well as early 1970s literature on native populations. Environmental changes associated with increasing myopia involve urbanization and schooling. The effect of schooling can manifest as myopia, and high myopia prevalence is higher in stringent religious schools versus general schools. Also, an increased total number of years of school is associated with a higher prevalence of myopia (and high myopia). Although we are not certain of the exact mechanism of myopia development, we know that the lengthening of the eye during childhood is a visually guided process. The normal process of emmetropization aims to match the length of the eye with the power of its optical components. When the lengthening of the eye continues beyond emmetropia, children develop myopia. Normal visual stimulation is required for the eye to know when to stop growing; for example, children need to be exposed to retinal images that include details (i.e., high spatial frequencies) for the eye to interpret the stop-growth signals correctly, but more specific information on the aspects of the retinal image that are important in normal emmetropization is needed to understand this process. Visual stimulation regulates retinal neurotransmitters and growth factors, which then modulate the scleral matrix to allow or cause axial elongation, but the specific pathway of these signals is also not well understood. To study some of these questions, the New England College of Optometry's Children's Vision Lab is conducting an NIH-sponsored study named PICNIC (Preventing myopia: Investigating Contributing factors to Nearsightedness In Children—neco.edu/PICNIC).

We do not yet have all the answers as to how the visual environment affects emmetropization, but we know that schooling increases the risk of myopia development or progression, and conversely, outdoors time is protective against the development and likely the progression of myopia. Why is outdoors protective? The answer is likely related to light, but is it light intensity or spectral composition? Sunlight intensity is much higher than any indoor lights, but indoor lights are also much more limited in spectrum

than natural sunlight. Sunlight may also be important for its effect on the circadian rhythms, associated with eye growth rhythms, and may thereby alter postnatal growth regulation. The effect of vitamin D from sunlight exposure, increased depth of focus from pupil construction, and physical activity are not likely significant in this process. A factor independent of sunlight that may play a role is that the overall dioptric demand of outdoor environments is more distance-focused, so there is less dioptric variation across the visual scenes. Why is schooling and the associated near work a risk for myopia? Accommodative lag was suggested to be larger in myopes than in emmetropes, but the story seems more complicated; perhaps instability of accommodation is more important. The wider range of dioptric blur across the retina when we look up close may hinder the integration of signals across the retina; paracentral blur has been shown to be likely more important than central blur. Schooling, with the increased cognitive effort, also demands high levels of attention that favors central vision, decreasing peripheral and paracentral attention.

The next level of parental concern would be about treatment options. What options do we have for decreasing or preventing myopic progression in my child? Which option is best for my child? For each given treatment, do the potential benefits outweigh the risks? Should my child be treated if they only progress a little, or should they be treated only if progressing rapidly? Judging known risk factors, should my at-risk child be treated even prior to myopia being measurable? How will we determine success, and at what point should we stop the treatment?

First, prevention: a quick look at risk factors shows that parental myopia increases risk for myopia in children, as do decreased time outdoors and increased near work. Ethnicity also plays a role, with Asian children more likely to develop myopia than Caucasians. Refractive status measurements at young ages give us a good idea of where the child will end up; the risk of myopia is higher if at 6 years of age the child's hyperopia is less than +0.75 D. Given a higher risk profile, and understanding that outdoors time is protective against both development and progression of myopia, the first defense is to recommend that every child with or at risk for myopia spend more time outside: two or more hours per day. Near work considerations, such as moving viewing distances farther away and taking frequent breaks (every 20 to 30 minutes), are also helpful.

The recommended treatment should reflect the child's visual makeup, including accommodation and binocularity, as well as the family's availability. Tests include questionnaires about outdoors time and near work, as well as a thorough history of lens wear and family history, careful refractive status determination with retinoscopy and frequent cycloplegia, and measures of pupil diameter, binocular vision, and accommodation tests. Axial length is important, albeit not necessary, to monitor the treatment's effectiveness, and corneal topography is necessary if orthokeratology is chosen. Informed consent is recommended since most of the treatments are not FDA approved specifically for myopia control.

Available treatments are either optical (specially designed glasses or contact lenses) or pharmacological. With any of these, it is important to consider carefully what is the optimal lens correction for the child. We should aim to correct accurately, with minimal to no undercorrection, but also not over-minusing.

Bifocal and progressive glasses have been used for myopia control for decades, but their benefit is rather limited. They are more effective in children with larger lags of accommodation (more than 0.75 D) and esophoria at near. Newer designs of spectacle lenses use variations on peripheral defocus or peripheral light scatter. Preliminary studies with these lenses have shown promising results, although they are not yet available in the U.S. Spectacles may be the simplest and most economic treatment for many families.

Two general types of contact lenses are used for myopia control: orthokeratology and multi-zone soft contact lenses. Orthokeratology has been used for decades to correct myopia temporarily by reshaping the cornea. This treatment has been shown to slow down the progression of myopia, and the excessive elongation of the eye, in most children. The mechanism is not fully understood, but it may involve decreasing peripheral hyperopic defocus, although it also changes the aberrations' profiles and the accommodation and convergence dynamics. It even works in high myopia, when myopia can only be partially corrected. This is the treatment that shows the largest effect in myopia studies, although the studies have limitations: mainly the difficulty of having a placebo control group. If a child is using orthokeratology for myopia control, axial length should be evaluated to determine whether the treatment is being successful. A clinical consideration with orthokeratology includes the understanding that both the child and the family need to be willing and able to deal with contact lenses. The child's motivation is the number one factor for success! Orthokeratology

can be an appealing treatment to children because they no longer need to wear glasses. It is also appealing to parents because the child only uses the lenses at night, when at home. Correctly fitting and centering the lenses is important for success. Associated risks include infectious keratitis (similar to other types of contact lenses), endothelial changes, and microvascular complications. Clinical trials are ongoing on combinations of orthokeratology with low-dose atropine.

Multi-zone soft contact lenses, either standard center-distance multifocal soft contact lenses or special designs such as MiSight lenses, also show benefit in slowing the progression of myopia. The presumed mechanism is also that peripheral hyperopic blur may be the driving factor in myopia, and the paracentral add power changes that peripheral defocus to myopic. But more evidence is needed; the full mechanism of myopic progression is probably more complex and includes other higher-order aberrations, binocular vision, and accommodation. Clinical considerations, as with orthokeratology, include that both the child and family need to be ready for wearing and adequately caring for contact lenses. Studies have shown that children have fewer complications than adults when using contact lenses.

A number of pharmacological treatments have been tried to slow down the progression of myopia. Atropine, a non-selective muscarinic antagonist, has shown effective results, although with varied degrees of success among different studies. There is still controversy regarding which dose is optimal and whether we should individualize the dose depending on the age and progression of the child. Higher doses may have greater risk for rebound effects, and lower doses may be more beneficial long-term. Currently, low doses of 0.01%, 0.02%, and 0.05% are typically used. Additionally, it is unclear whether the effect is as positive in controlling the lengthening of the eye, which is the goal of myopia control, as it is in controlling the progression of myopia in diopters. The mechanism for atropine's effect in myopia control does not seem to be paralysis of accommodation. The inner retina has been suggested as the area of action, although this is yet to be determined, and some involvement of the accommodative system is plausible given the differences found between diopters and the axial length effect. Questions remain as to whether atropine use is appropriate for pre-myopes. The clinical considerations include possible short-term side effects of photophobia and near blur, although these are rare, and if they occur typically go away after

two weeks. Drug hypersensitivity or toxicity are not likely. Other pharmacological agents being tested are pirenzepine (M1-selective antagonist), brimonidine (alpha-adrenergic agonist), latanoprost (prostaglandin analog), and 7-methylxanthine (adenosine receptor antagonist), as well as bilberry extract and croetin (carotenoid).

If myopia control is undertaken, it is important to determine whether the treatment is effective. Is the treatment working? If no progression is noted, which is rare, in either diopters or in the length of the eye, then we are sure that the treatment is successful. In most cases, however, we need to compare the progression observed after the initiation of treatment with the progression expected for the individual child, considering age, sex, and race. Typically, myopia progresses at 0.75 D/year or faster between 6 and 9 years of age, but by 10-13 years old, that rate drops to about 0.50 D/year. Axial length typically increases 0.32 mm/year in the 6- to 9-year-old group in myopes and at roughly half that rate between 10 and 13 years of age (about 0.19 mm/year). Progression of axial length is expected even in emmetropes, at rates of approximately 0.15 mm/year in children 6 to 9 years of age and about 0.09 mm/year in children 10 to 13 years of age. Again, these numbers depend on the child race and sex, and more concrete centile tables are currently being constructed. Online calculators are available, but these are of limited use as they assume that the

expected progression and treatment effect is constant over time, which is incorrect. Note that the natural progression of myopia indicates that about half of all patients will stop progressing at around age 16 years, but that means that the other half will continue to progress until well into adulthood.

In summary, when it comes to myopia management, we have the obligation to recommend that all children with or at risk of myopia spend more time outdoors, reduce the amount of time spent doing near work, increase the viewing distance when doing these tasks, and have frequent breaks. Attempts to control myopia are necessary as it poses a high risk for vision-threatening conditions. Treatments such as low-percentage atropine, orthokeratology, and multi-zone soft contact lenses should be considered. Keep watching for studies in this area as new information is available weekly.

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