CANDY:
Controlling Astigmatism and Nearsightedness in Developing Youth

David Bartels, O.D., Peter E. Wilcox, O.D.

ABSTRACT:

Background

The progressive worsening of myopia and/or astigmatic refractive errors is a serious concern for both the child and their parents. The treatment procedure known as orthokeratology has been shown to slow the rate of progression in children. This study attempted to replicate these findings.

Methods

The "No-mold" retrospective refractive data from 20 children (40 eyes) who subsequently chose orthokeratology treatment were obtained from patient records. The duration of refractive data varied from as little as 2 to as much as 82 months.

The "Molded" phase included 28 children (56 eyes). Baseline data included refraction, Simulated K (Sim K) and topographic maps. They were fit with Wave custom designed orthokeratology mold lenses. Treatment duration was 7 to 57 months.

The "Unmolded" phase involved discontinuing orthokeratology treatment until flat Sim K returned to within 1D of pre treatment or 14 days.

Results

The mean change in spherical equivalent refractive error (SEQ) during the No-mold phase was -.37D per year. The mean change in SEQ during the Molded/Unmolded phase was -.03 D per year.

Conclusions

Orthokeratology treatment does slow the progression of myopia in children.

INTRODUCTION

The worsening of myopic and/or astigmatic refractive errors is a serious concern for both the affected child and their parents. Typical solutions include the use of spectacles and/or contact lenses. Neither of these choices addresses the concern surrounding progressive changes in refractive error but, in fairness to the clinician, there are few good options to present which work simply and predictably. There are many factors which are associated with these shifts in myopic refractive error such as: genetics (1, 2, 3), phoria (4, 5), near vision demands (6, 7), ethnicity (8, 9), sex (10) and younger age (11).

Options to slow the progression in refractive error are unpredictable and at times inconvenient. Atropinization (12, 13) is one of the most successful forms of intervention but the side effects greatly outweigh the outcomes. Bifocals and progressive addition lenses have some effect (3, 14,) but they are cumbersome for youth with active lifestyles. Daytime wearing of rigid gas permeable lenses has been shown to slightly reduce the progression but at rates which are of limited value to the patient (15, 16).

An effective, predictable and reproducible method to reduce the rate of change of myopia and astigmatism is Orthokeratology (also known as Corneal Molding (CM), Corneal Reshaping, Corneal Refractive Therapy, Advanced Orthokeratology and Custom Accelerated Orthokeratology to name a few). Orthokeratology employs the nightly wearing of rigid gas permeable lenses (corneal molds) which reshape the curvatures of the cornea and provides great unaided vision during waking hours.

Several studies have demonstrated the effectiveness of orthokeratology. The Loric Study (20) followed patients over 12 months and concluded that Orthokeratology has a positive effect on slowing the progression of nearsightedness and astigmatism. The Orthokeratology and Adolescent Myopia Control study (21), a 3 year retrospective study, found that those who participated in orthokeratology had an average increase in myopia of only -0.67D over three years versus the expected -1.50D of change over the same period of time. In 2007, Gerowitz, Eiden and Davis initiated a similar FDA approved, 5 year corneal molding – myopia control study of 300 youths ages 8 through 14. The outcome is pending.

Methods

This study was designed to replicate the findings that orthokeratology slows the normal progression of myopia/astigmatism.

The study enrolled 28 youth. Seven were male and 21 were female. Age ranged from 4 to 20 years. Age at commencement of orthokeratology treatment ranged between 9 and 16 years. Subjects' ethnicities were 27 Caucasians and 1 African-American. Subjects' pretreatment refractive error ranged from sphere of -1.00DS to -5.25DS (mean = -2.33D) and cylinder of plano to -1.00 (mean = 0.17DC). Mean SEQ was -2.25D.

No Mold Phase: Historical refractive data were obtained from a subset of 20 subjects of the study group (40 eyes). They were established patients with active medical charts but had not yet begun orthokeratology treatment.

The rate of progression of their myopia during this phase was calculated as the difference between the SEQ at their first visit to the office and the SEQ just prior to initiating orthokeratology treatment.

Mold Phase: This phase included all 28 youth (56 eyes). Refractive data prior to orthokeratology treatment were as follows: mean SEQ = -2.25D (range -1.00 to -5.50), mean sphere = -2.23D (range -1.00 to -5.25), mean cylinder = 0.17DC (range plano to -1.00).

Upon choosing orthokeratology, baseline data were collected which included a noncycloplegic refraction, topography (Scout-Eyequip), intraocular pressure and age of parents' onset of myopia/astigmatism.

The corneal molding lenses were custom designed using the captured topography maps and Wave Software System. .

The dispensing visit consisted of educating the patient on the wear, insertion and removal processes and care of the lenses. A variety of care regimens were prescribed based on clinical judgment. These included Optimum and Boston cleaning and rewetting systems, Sauflon peroxide system, Allergan rewetting products and lens cleaning sponges. Following the training, unaided vision was measured. Then the patient was allowed to recline, closing their eyes for 30 minutes. Immediately upon opening their eyes, acuities were measured and the fit was assessed with white light and NaFl. The molds were removed and topographies and unaided acuities were measured.

Though all patients initially wore their molds every night, the lens wearing schedule eventually varied among the 28 subjects: Seven wore the lenses every night, 18 wore them every second night and 3 wore then every third night. This schedule was determined by the patient and their desired endpoint acuity.

Follow up visits included one-week, one-month, three months and six months as determined by the investigators. On rare occasion, a mold lens was changed to improve fit or post wear acuity. The treatment was considered successful if at the 3 month visit, the patient was "20/happy", topography was homogenous and no corneal pathology was observed. The duration of orthokeratology treatment varied per subject ranging from 7 to 57 months.

Unmold Phase: Given the clinical nature of this study and inconvenience to the patient, whenever possible, an effort was made to correlate discontinuing lens wear with any unscheduled mold lens replacement (lost or broken). Otherwise, patients complied with the request to unmold.

Lens wear was discontinued and unmolding was considered complete when a subject's flat Simulated-K returned to within 1.00D of their Sim K prior to molding or 14 days had elapsed since discontinuing lens wear. During the unmolding process, noncycloplegic refractions were performed at 3 to 7 day intervals until reversal was complete.

The rate of progression of myopia for the molded patients was calculated as the difference between the SEQ just prior to molding and the SEQ after unmolding was complete.

Results

Results for all phases are presented in Figure 1. Figure 1 superimposes both the No Mold and Molded/Unmolded phase data. The X axis represents the duration of the phases in months.

No Mold Phase:

This phase included the subset of 20 established patients (40 eyes). The mean change in SEQ showed a progression of -0.37D per year. This is represented in Figure 1 by the blue data diamonds and blue trend line.

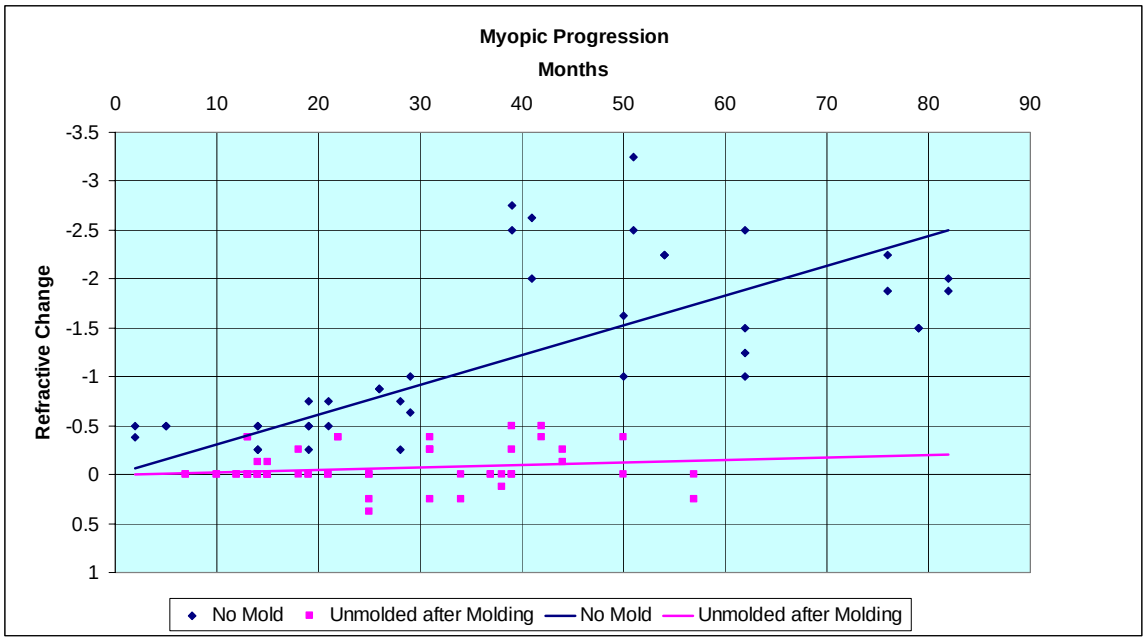
Unmold Phase:

This phase included all 28 subjects (56 eyes).

The reversal requirement was achieved by 27 of 28 subjects within two weeks (within 1D of pretreatment Sims K). In fact 44 of 54 eyes were within .5D of Sim K and 49 of 54 eyes were within .75D of original Sim K. Only 1 subject failed and was excluded from the study.

During the mold to unmold phase, the mean SEQ progression rate was only -0.03D per year. This is represented in Figure 1 by the magenta data squares and magenta trend line. Some data points are superimposed due to duplicate results on some patients. Progression rates were calculated from the raw data, not the displayed graph.

Figure 1. Myopic Progression during No Mold and Unmolded after Molding Phases



Conclusions

The CANDY study demonstrated that Orthokeratology does both significantly reduce and even stop the rate of change of nearsightedness and astigmatism in developing youth (ages 9-18). This effect was independent of the age of initiation of orthokeratology and the premolding refractive error.

This reduction in the rate of change occurs for youth in all the familial risk groups (neither, one or both parents myopic by age 18).

The IOP had no effect on myopic progression.

Discussion

The CANDY study attempted to determine if orthokeratology halted refractive error change or simply masks the change due to the molding of the corneal surface. By unmolding the treatment and allowing the Sim K's to return to baseline levels, we believe some progress has been made to answer this question. The CANDY subjects had an average **premolding** progression in their SEQ of- 0.37D per year. This rate of progression in myopic children is coincident with published literature (11). These subjects' SEQ was essentially halted during the duration of their molding to an impressive mean rate of -.03D per year after being unmolded.

There is some concern that the 2 week duration of the reversal phase was insufficient despite the fact that the Sim Ks of 49 of the 54 subjects were within .75D of their baseline Sim K's. This issue should be the basis for further study. Data obtained from trials using longer reversal periods would provide more complete data.

Though Hyman, Marsh-Tootle et.al (COMET 2005) showed that there is a greater progression in myopic SEQ in their 6 to 7 year old age group and a slowing in the rate of progression by their oldest age grouping (10 to 11 years), there was progression in all the populations they followed. When the CANDY study reviewed the age of those molded, from 9 years to 18 years, essentially no correlation between age and progression of SEQ was found.

Since the CANDY study was initiated by evaluating patients who had previously established various wearing schedules of their corneal molds, other interesting conclusions surfaced. Some patients did not need to wear their molds every night to achieve visual success yet they had the same myopia/astigmatism stabilization. The patients who were best suited to sleep in their molds only every second to third night were those with lower refractive errors and steeper Sim K's. For this reason the authors submit that there is a greater incentive to mold the younger eye at lower refractive errors. Clinicians should present to the higher risk/lower SEQ patients and their family that:

1.) orthokeratology when done in the early stages of myopia is more cost effective,
2.) because their SEQ is low, it allows the child to wear the lenses every other night
3.) it is more acceptable for a younger patient to experience any partial unmolding during the alternate days when the lenses were not worn
4) it is better to stop axial elongation when the eye is still shorter, reducing the risk of retinal detachment.

The population of eligible patients in this study was significantly skewed toward a white population (27 White, 1 Black, 0 Hispanic and 0 Asians). As it has been shown that there is a greater incidence of and progression of SEQ in the Asian populations (19), a similar study within a primarily Asian population would be even more powerful.

The CANDY study was designed to investigate if orthokeratology slows the progression of myopia (with or without astigmatism). The study was not designed to investigate how specifically designed corneal molds affect the eye/vision. Additionally, the goal of each patient was to have good vision (with and without the molds), good fit and good comfort; hence, liberal boundaries

were granted on the complicated designs of the reverse geometry lenses. All designs were similar in they were tied to the global goals of success. An evaluation of the various designs does reveal more similarities than differences among the molds.

Despite the repeatable results of 'myopia control' by orthokeratology, some caution should be exercised in promoting this as a predictable way to halt the refractive error changes. Not all studies have found such consistent reduction in refractive error progression (Reim 2003). One of the limitations of this study is size of population. Larger, well controlled trials are needed to further validate the findings of this and other orthokeratology studies.

The investigators continue to monitor all those in the Candy study and are expanding their centers while broadening the data base. Additional variables which may be monitored are central corneal epithelial thicknesses and total central corneal thickness and axial length. They look forward to presenting their future findings.

About the authors:

Dr. Peter E. Wilcox is in solo private practice in Hayes, Virginia. He received his OD from UAB and attended a Residency at PCO.

Dr. David Bartels is in group practice in Buffalo, NY. He received his OD at ICO.

The authors would like to extend their gratitude to Caroline Guerrero Cauchi, O.D., F.O.A.A. and Richard Anderson, O.D., F.O.A.A. for their editorial assistance in preparing this article.

On a personal note, being one of the investigators and the father of two of the subjects, I am pleasantly surprised at the outcome of the study. My daughter and son both mold every 72 hours. They have been threatened with myopic progression if they don't mold more often, only to fall on deaf ears. I don't know the correct number of nights needed to hold the progression back but both my children are doing fine. Corneal Molding has become the norm in my family and it is very gratifying to observe no change in my children's vision over many years. David Bartels OD.

.

References

1. Kurtz D, Hyman L, Gwiazda JE, Manny R, Dong LM, Wang Y, Scheiman M and the COMET Group. Invest Ophthalmol and Vis Sci. 2007; 48:562-570.
2. Hammond CJ, Snieder H, Gilbert CE, Spector TD. Genes and environment in refractive error: the twin eye study. Invest Ophthalmol Vis Sci 2001 May; 42(6):1232-6. 2001 May; 42(6):1232-6.
3. Paluru PC, Nallasamy S, Devoto M, et al. Identification of a novel locus on 2q for autosomal dominant high-grade myopia. Invest Ophthalmol Vis Sci 2005 Jul;46(7);2003-7.
4. Goss DA, Grosvenor T. Rates of childhood myopia progression with bifocals as a function of near point phoria: consistency of three studies. Optom Vis Sci. 1990. 67: 637-40.
5. Fulk GW, Cyert LA, Parker DE. A randomized trial of the effect of single-vision vs. bifocal lenses on myopia progression in children with esophoria. Optom Vis Sci 2000, 77: 395-401.
6. Saw SM, Zhang MZ, Hong RZ, Fu ZF, Pang MH, Tan DT. Near-work activity, night-lights in the Singapore-China study. Arch Ophthalmol. 2002; 120:620-627.
7. Mutti DO, Mitchell GL, Moeschberger ML, Jones LA, Zadnik K. Parental myopia, near work, school achievement, and children's refractive error. Invest Ophthalmol Vis Sci. 2002; 43:3633-3640.
8. Young F, Leary G, Baldwin WR, et al. The transmission of refractive errors within Eskimo families. American Journal of Optometry 1969; 47:244-9.
9. Kleinstein RN, Jones LA, Hullett S, et al. Refractive error and ethnicity in children. Arch Ophthalmol. 2003; 121:1141-1147.
10. Braun CI, Freidlin V, Sperduto RD, Milton RC, Strahlman ER. The progression of myopia in school age children: data from the Columbia Medical Plan. Ophthalmic Epidemiol. 1996; 3:13-21.
11. Hyman L, Gwiazda J, Hussein M, Norton TT, WangY, Marsh-Tootle W, Everett MA. Relationship of age, sex, and ethnicity with myopia progression and axial elongation in the Correction of Myopia Evaluation Trial. Arch Ophthalmol. 2005; 123:977-987.
12. Gimbel HV. The control of myopia with atropine. Can J Ophthalmol 1973 Oct;8(4):527-32.
13. Lee JJ, Fang PC, Yang IH, et al. Prevention of myopia progression with 0.05% atropine solution. J Ocul Pharmacol Ther 2006 Feb; 22(1):41-6.
14. Leung JT, Brown B, Progression of myopia in Hong Kong Chinese schoolchildren is slowed by wearing progressive lenses. Optom Vis Sci. 1999, 76:346-54.
15. Walline JJ, Jones LA, Mutti DO, Zadnik K. A Randomized trial of the effects of rigid contact lenses on myopia progression. Arch Ophthalmol 2004 Dec; 122(12):1760-6.
16. Katz J, Schein OD, Levy B, et al. A randomized trial of rigid gas permeable contact lenses to reduce progression of children's myopia. Am J Ophthalmol 2003 Jul; 136(1):82-90.
17. Smith EL. Mechanisms of myopia.
18. Jensen H. Myopia progression in young school children and intraocular pressure. Documenta Ophthalmologica 1992Sept;82(3) 249-255.
19. Park DJ, Congdon NG. Evidence for an "epidemic" of myopia. Ann Acad Med Singapore 2004 Jan; 33(1):21-6.