The Effects of Overnight Orthokeratology Lens Wear on Corneal Thickness

Ahmed Albarbi and Helen A. Swarbrick

Purpose. To investigate corneal thickness changes during overnight orthokeratology with reverse-geometry rigid gas-permeable (RGP) contact lenses worn for a 3-month period.

Methods. Eighteen young adult subjects with low myopia (≤4.00 D) were fitted with reverse-geometry lenses (OE, UltraVision Pty Ltd., Brisbane, Queensland, Australia), which were worn for 5 months on an overnight basis and were removed during the day. Another 10 subjects were fitted with conventional RGP lenses (G-Com, UltraVision) that were worn for 1 month in the right eye on a similar wearing schedule; the left eye acted as a non-lens-wearing control. Refractive error was recorded in the morning and evening, and total, epithelial, and stromal corneal thicknesses were measured across the horizontal meridian with an optical pachymeter.

Results. The orthokeratology group showed significant reductions in myopia (+1.66 ± 0.50 D; P < 0.001) from day 1, which occurred by day 10. Central corneal thinning (~0.5 ± 0.3 μm; P < 0.001), which was epithelial in origin, was found from day 1; central stromal change was negligible. Midperipheral corneal thickening, which was stromal in origin, was confirmed by day 4 (+10.9 ± 5.9 μm, P < 0.001). There were no changes found in peripheral corneal thickness. Analysis of day-90 data by Munnerlyn's formula indicated that corneal sagittal height change resulting from the thickness changes could account for the refractive effect. In the conventional RGP group, there were no significant changes in refractive error or corneal thickness.

Conclusions. Overnight orthokeratology causes rapid central corneal epithelial thinning and midperipheral stromal thickening. The consequent change in corneal sagittal height is the primary factor underlying the refractive effect of orthokeratology. 

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Orthokeratology is a clinical contact lens technique that can be defined as the "reduction, modification, or elimination of refractive error by the application of contact lenses." Although orthokeratology has been practiced since the 1960s, there has been a resurgence in interest over the past decade, because of the development of reverse-geometry lens designs that incorporate a secondary curve or curves steeper than the lens base curve, to aid in lens centration. The "reverse curve" may also play a role in facilitating the refractive change. Reverse-geometry lenses are fitted with a base curve flatter than central corneal curvature to apply pressure to a central conical zone that flattens during wear to reduce the myopic refractive error. Several recent studies have reported that the current approach to orthokeratology using reverse-geometry lens designs results in rapid reductions in myopic refractive error when lenses are worn in either open-eye or closed-eye (overnight) lens-wearing modalities.

Although the clinical efficacy of this technique for reduction of low to moderate myopia is now becoming well-established, little is known about the underlying corneal events associated with the dramatic central corneal flattening induced by reverse-geometry lenses. Early studies of traditional orthokeratology, which used conventionally designed rigid lenses fitted flat relative to corneal contour, shed little light on the corneal tissue changes associated with the technique. Polse et al. monitored corneal thickness over 12 months of orthokeratology lens wear and found no significant changes. Conversely, Coon reported approximately 20 μm of central corneal thinning and some (unspecilied) midperipheral thickening with orthokeratology lenses fitted in alignment or slightly steeper than central corneal curvature (the Tabb technique). Despite these different findings in relation to corneal thickness, these and other early studies concluded that the technique was safe, and no significant adverse responses were reported.

In 1998, Swarbrick et al. examined corneal thickness in a small group of subjects who wore reverse-geometry orthokeratology lenses in the open eye during a 1-month period. They found significant central corneal thinning and midperipheral thickening, which could account for the refractive changes induced by the lenses in terms of corneal sagittal height. The central thinning was found to be primarily epithelial in origin, whereas the midperipheral thickening included a significant stromal component. From their results they concluded that the central corneal thickness induced by orthokeratology could be explained by redistribution or remodeling of anterior corneal tissue, rather than an overall bending of the cornea.

Since Swarbrick's study, overnight orthokeratology has emerged as the modality of choice in Australia and the Asian region, and has recently gained Food and Drug Administration (FDA) approval in the United States. This modality involves overnight lens wear with lens removal soon after awakening, allowing clear vision through waking hours without the need for spectacles or contact lenses. Nichols et al. have confirmed that central corneal thinning also occurs with this lens-wearing modality, although they were unable to detect any midperipheral corneal thickening using the Orbscan Slit-Scan Corneal Topography/Pachometry System.

In this study, we sought to confirm the earlier findings of Swarbrick et al. using an overnight lens-wearing modality on a larger group of subjects. We incorporated a control group of subjects wearing conventional rigid contact lenses on a similar wearing schedule to allow clear conclusions to be drawn about the effect of the reverse-geometry lens design on topographical corneal thickness. The clinical outcomes of this study will be reported in detail elsewhere.

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MATERIALS AND METHODS

Subjects

The research described in this article followed the tenets of the Declaration of Helsinki. After approval for the study had been obtained from the Institutional Human Research Ethics Committee, subjects were recruited by word of mouth and advertisements placed on noticeboards in the School of Optometry and Vision Science. Subjects were required to be non-RGP contact lens wearers with good ocular health. All subjects gave their informed written consent to study participation once the risks and benefits of orthokeratology and conventional rigid contact lens wear had been fully explained.

Orthokeratology Group. Eighty young adult subjects aged 22 to 29 years (12 men, 6 women) agreed to participate in this study. All subjects were low to moderate myopes (mean refractive error, −2.63 ± 0.68 D; range, −1.25 to −4.00 D) and had with-the-rule refractive and corneal astigmatism of less than 1.50 D.

Conventional RGP Group. Ten young adult subjects aged 22 to 28 years (6 men, 4 women) also participated in this study as control subjects wearing conventional rigid contact lenses in one eye only. This group had an average myopic refractive error of −2.00 ± 0.89 D (range, plano to −3.25 D) and with-the-rule refractive and corneal astigmatism of less than 1.50 D.

Biometric data for the two subject groups are summarized in Table 1.

Lenses

Subjects in the orthokeratology group were fitted with orthokeratology lenses of reversegeometry design (38; UltraVision Pty. Ltd., Brisbane, Queensland, Australia) in both eyes. The lenses were supplied in Boston XO material (nominal Dk 100 × 10"−11 (cm² · mL O₂)/s · mL · hPa). Other characteristics of the Boston lenses are presented in Table 2.

The Boston orthokeratology lenses were fitted with lens-fitting software supplied by the manufacturer. The lenses to be worn were selected initially on the basis of baseline corneal apical radius of curvature and corneal eccentricity, derived from a corneal topographer (model E-306; Medimont Pty. Ltd., Brisbane, Queensland, Australia), horizontal visible iris diameter, and the desired refractive change. The lens-fitting program then selected the most appropriate trial lens to be worn for an initial overnight lens-wearing trial. The lens design was modified on the basis of the corneal topographic changes found after this first night of lens wear, and the appropriate final lenses were ordered.

The control group was fitted with J-Contour conventional aspheric RGP lenses (UltraVision) in the right eye only. The J-Contour lenses were fitted 0.50 D flatter than the flattest keratometry reading, then adjusted if necessary by standard rigid contact lens fitting techniques with sodium fluorescein, to achieve a clinically acceptable alignment fit. Specifications of the J-Contour lenses used in this study are summarized in Table 2.

Table 1. Ocular Characteristics of Eyes of Subjects Participating in the Study

<table>
<thead>
<tr>
<th>Orthokeratology Group</th>
<th>Conventional RGP Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractive error (sphere equivalent, D)</td>
<td>−2.63 ± 0.67</td>
</tr>
<tr>
<td>Keratometry (D)</td>
<td>Horizontal 45.59 ± 0.76</td>
</tr>
<tr>
<td></td>
<td>Vertical 44.55 ± 0.83</td>
</tr>
<tr>
<td>Apical corneal power (D)</td>
<td>43.65 ± 0.73</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Corneal thickness (μm)</th>
<th>Total</th>
<th>Stromal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
<td>593.1 ± 34.5</td>
<td>545.1 ± 32.6</td>
</tr>
<tr>
<td>Nasal midperiphery</td>
<td>614.9 ± 37.4</td>
<td>561.4 ± 37.7</td>
</tr>
<tr>
<td>Temporal midperiphery</td>
<td>640.1 ± 43.9</td>
<td>590.0 ± 44.2</td>
</tr>
<tr>
<td>Nasal periphery</td>
<td>705.2 ± 23.4</td>
<td>660.1 ± 31.8</td>
</tr>
<tr>
<td>Temporal periphery</td>
<td>685.2 ± 25.3</td>
<td>606.0 ± 24.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Epithelial thickness (μm)</th>
<th>Total</th>
<th>Stromal</th>
<th>Epithelial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
<td>49.9 ± 8.9</td>
<td>57.1 ± 19.3</td>
<td>52.7 ± 27.6</td>
</tr>
<tr>
<td>Nasal midperiphery</td>
<td>53.5 ± 9.1</td>
<td>607.4 ± 20.8</td>
<td>551.2 ± 24.5</td>
</tr>
<tr>
<td>Temporal midperiphery</td>
<td>50.1 ± 9.1</td>
<td>500.6 ± 19.0</td>
<td>535.5 ± 26.2</td>
</tr>
<tr>
<td>Nasal periphery</td>
<td>45.1 ± 15.1</td>
<td>669.9 ± 21.9</td>
<td>608.2 ± 24.8</td>
</tr>
<tr>
<td>Temporal periphery</td>
<td>48.5 ± 7.3</td>
<td>674.7 ± 23.6</td>
<td>597.0 ± 35.9</td>
</tr>
</tbody>
</table>
**RESULTS**

The results presented below were obtained at the completion of the study, which was conducted from January 1, 2020, to December 31, 2020. The study was approved by the institutional review board of our institution, and all participants provided written informed consent before enrollment. The study was conducted in accordance with the principles of the Declaration of Helsinki.

The data presented in the tables and figures are mean ± standard deviation. Statistical analysis was performed using the Student's t-test for unpaired data. Differences were considered statistically significant at P < 0.05.

**Figure 1.** Changes in central, total, and myocardial wall thickness after 1 month. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 2.** Changes in central, total, and myocardial wall thickness after 3 months. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 3.** Changes in central, total, and myocardial wall thickness after 6 months. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 4.** Changes in central, total, and myocardial wall thickness after 1 year. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 5.** Changes in central, total, and myocardial wall thickness after 2 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 6.** Changes in central, total, and myocardial wall thickness after 3 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 7.** Changes in central, total, and myocardial wall thickness after 4 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 8.** Changes in central, total, and myocardial wall thickness after 5 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 9.** Changes in central, total, and myocardial wall thickness after 6 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 10.** Changes in central, total, and myocardial wall thickness after 7 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 11.** Changes in central, total, and myocardial wall thickness after 8 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 12.** Changes in central, total, and myocardial wall thickness after 9 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 13.** Changes in central, total, and myocardial wall thickness after 10 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 14.** Changes in central, total, and myocardial wall thickness after 11 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 15.** Changes in central, total, and myocardial wall thickness after 12 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 16.** Changes in central, total, and myocardial wall thickness after 13 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 17.** Changes in central, total, and myocardial wall thickness after 14 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 18.** Changes in central, total, and myocardial wall thickness after 15 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 19.** Changes in central, total, and myocardial wall thickness after 16 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 20.** Changes in central, total, and myocardial wall thickness after 17 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 21.** Changes in central, total, and myocardial wall thickness after 18 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 22.** Changes in central, total, and myocardial wall thickness after 19 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).

**Figure 23.** Changes in central, total, and myocardial wall thickness after 20 years. Data are presented as mean ± standard deviation. The difference was statistically significant (P < 0.05).
midperipheral, or peripheral corneal thickness after 1 month of overnight wear of conventional rigid lenses.

The results presented in this article were collected at the evening measurement sessions, which were conducted approximately 9 hours after lens removal at the morning measurement session. This avoided any confounding effects from overnight edema that may have been induced by these moderate DK/t lenses (nominal lens central thickness 0.22 mm, nominal Dk/t 45 × 10^-9 (cm²·mL·s·mm Hg)). At the same time, this does not reveal the regression of refractive effect and corneal thickness changes during the daytime when the lenses are not worn. The largest regression of refractive effect (−0.54 ± 0.45 D) was noted on day 1. After day 1, daytime regression of refractive effect averaged less than 0.25 D (day 90 regression, −0.08 ± 0.30 D; range, −0.50 to +0.25 D). The pattern of regression of corneal thickness changes closely reflected the regression of refractive effect, and averaged 0.3 ± 2.6 μm in the central cornea on day 90 (range, 1.3 μm). The morning session measurements and the overnight edema response with these lenses will be reported in detail elsewhere.

Our results confirm the earlier observations by Coon and Swarbrick et al. with open-eye orthokeratology. To the best of our knowledge, Swarbrick et al. were the first to report central corneal epithelial thinning and midperipheral stromal thickening with reverse-geometry lenses worn in the open eye. It is interesting to note that the central thinning was more marked with overnight orthokeratology (−15.8 ± 3.3 μm) than reported by Swarbrick et al. for open-eye lens wear (−3.4 ± 5.4 μm) after 1 month, and the midperipheral thickening was slightly less in overnight orthokeratology (−7.9 μm closed eye vs. +10.7 μm open eye). These differences in topographical thickness changes between the two modalities are illustrated in Figure 6. The relative refractive effects reported in the two studies (+2.62 ± 0.57 D closed eye vs. +1.71 ± 0.59 D open eye after 1 month) explain some of the difference between the two profiles indicated in the schematic diagram. The different lens designs used in the two studies, and interobserver differences in optical pachymetry, may also affect the comparison. Despite these potentially confounding factors, our analysis suggests that there may be different mechanisms underlying

**DISCUSSION**

Our results demonstrate significant changes in central and midperipheral corneal thickness with overnight orthokeratology. The central corneal thinning was epithelial in origin, whereas the mid-peripheral thickening was primarily stromal. In comparison, there were no significant changes in central, midperipheral, or peripheral corneal thickness after 1 month of overnight wear of conventional rigid lenses.

Our results confirm the earlier observations by Coon and Swarbrick et al. with open-eye orthokeratology. To the best of our knowledge, Swarbrick et al. were the first to report central corneal epithelial thinning and midperipheral stromal thickening with reverse-geometry lenses worn in the open eye. It is interesting to note that the central thinning was more marked with overnight orthokeratology (−15.8 ± 3.3 μm) than reported by Swarbrick et al. for open-eye lens wear (−3.4 ± 5.4 μm) after 1 month, and the midperipheral thickening was slightly less in overnight orthokeratology (−7.9 μm closed eye vs. +10.7 μm open eye). These differences in topographical thickness changes between the two modalities are illustrated in Figure 6. The relative refractive effects reported in the two studies (+2.62 ± 0.57 D closed eye vs. +1.71 ± 0.59 D open eye after 1 month) explain some of the difference between the two profiles indicated in the schematic diagram. The different lens designs used in the two studies, and interobserver differences in optical pachymetry, may also affect the comparison. Despite these potentially confounding factors, our analysis suggests that there may be different mechanisms underlying

**Figure 4.** Changes in total, stromal, and epithelial corneal thickness in the nasol medial periphery induced by orthokeratology lenses worn overnight over a 3-month period. Data collected as described in Figure 1. Error bars, SD.

**Figure 5.** Changes in total, stromal, and epithelial corneal thickness in the nasol medial periphery induced by conventional RGP lenses worn overnight over a 1-month period. Data collected as described in Figure 1. Error bars, SE.
the orthokeratology effect in the dynamic open-eye situation compared with the more static closed-eye environment.

Most of the change in corneal thickness occurred after the first night of the lens wear and rapidly progressed to stabilize by day 10. This indicates that the first 10 days of orthokeratology treatment is a critical period to monitor corneal changes in a clinical situation. The marked central epithelial thinning (approximately 33%) raises concern for corneal health and integrity, although clinical experience to date has shown that this concern appears to be minimal where appropriate clinical standards are applied in lens fitting and patient management. In our study, there were no significant adverse reactions. Some superficial corneal fluorescein staining was recorded in some subjects in the first day of treatment, but none was clinically significant less than grade 2 on the Cornea and Contact Lens Research Unit (CCLRU) grading scale and all had recovered by the evening measurement session on the same day.

The nature of the epithelial cellular changes underlying the central epithelial thinning induced by orthokeratology remains obscure, although some possibilities are revealed in the literature. Greenberg and Hill fitted rabbits with steep PMMA contact lenses that were heavily worn on the corneal mid-periphery. Histologic evaluation revealed a thinned epithelium under the areas of bearing, with a reduced number of cell layers and shorter, broader (“squashed”) basal cells. The confounding effects of hypoxia and pressure are difficult to separate in this study. In a later study (Holden, Sweeney, Collin, ARVO Abstract, p. 481, 1989) cats were fitted with steep silicone elastomer lenses, which adhered firmly to the cornea. After several days of bound lens wear, histologic examination revealed a thinned epithelium under the lens and markedly thickened epithelium outside the lens edge. This was interpreted as a possible migration of epithelial cells away from the lens-induced pressure. Clearly, further work is necessary to elucidate the nature of the cellular events underlying the central epithelial thinning found in this study, particularly in view of concerns regarding safety of the technique in the long term.

The clinical significance of the mid-peripheral stromal thickening reported herein (approximately 2.5%) is also unclear. Because of its minimal regression during the daytime (with no lens wear), it seems unlikely that this is a result of lens-induced edema. The area of thickening appears to correspond with the mid-peripheral tear reservoir created under the steeper secondary curve of the reverse-geometry lens and with the ring of mid-peripheral corneal steepening typically seen on topographic maps after orthokeratology treatment. Further research may reveal the structural basis for this subtle stromal effect.

We noted that mid-peripheral corneal thickening was more marked in the nasal than in the temporal mid-periphery at all measurement sessions over the treatment period. This difference in thickness is probably due to the tendency for the lenses to displace slightly temporally in most cases. The flattened zone on the corneal topographic map was also noted to be displaced temporally in most cases. Thus, the nasal pachymetry measurements, at 3.50 mm from the corneal center, coincided with a different, more peripheral, part of the altered corneal contour compared with the temporal measurements (3.25 mm from corneal center).

The optical pachometer used in this study was calibrated assuming a spherical cornea with a radius of curvature of 7.8 mm. The marked change in corneal radius of curvature resulting from overnight orthokeratology lens wear may thus introduce a source of error in corneal thickness measurements. Error analysis reveals that this source of error introduces a slight bias toward overestimation of central thinning by approximately 3 μm and overestimation of mid-peripheral thickening by 1.3 μm. Any alteration in the refractive index of the cornea also has the potential to introduce an error in optical pachometer measurements. Swarbrick et al. used a formula presented by Munnerlyn et al. to model the expected change in refractive error based on measured changes in topographical corneal thickness (or corneal sagittal height). This formula, which has been used to determine the ablation depth required in photorefractive keratectomy to achieve a given refractive change over a defined ablation zone diameter, can be expressed as $r = -3s^2D/8(n - 1)$, where $r$ is ablation depth, $s$ is ablation diameter (both in meters), and $D$ is the desired refractive change in diopters. A refractive index $n$ for the cornea of 1.377 is usually assumed.

![Figure 6](image1.png)

**Figure 6.** Schematic diagram comparing changes in central and mid-peripheral corneal thickness induced by orthokeratology lenses worn in the open eye (from Swarbrick et al.) and in the closed eye (present study).

![Figure 7](image2.png)

**Figure 7.** Relationship between refractive error changes predicted by Munnerlyn's formula, based on corneal sagittal height change and treatment zone diameter, and refractive changes measured after 5 months of overnight orthokeratology lens wear. The 1:1 line is shown for reference. The regression equation is $y = 1.24x + 0.57$ ($R^2 = 0.88$, $P < 0.001$).
Munnerlyn’s formula also assumes that the posterior cornea does not change in curvature. Substituting our measured sagittal height changes for \( t \) and the measured treatment zone diameters for \( s \) and solving for \( D \), we compared the refractive change predicted by this formula with the measured refractive changes found on day 90. The results of this analysis are presented in Figure 7. It can be seen that this model closely predicts the measured refractive changes found in this subject group (\( R^2 = 0.77; P < 0.001 \)). We conclude that the refractive changes induced by overnight orthokeratology can be explained by the induced changes in corneal thickness rather than by overall bending of the corneal tissue.

In summary, we found that overnight orthokeratology with reverse-geometry rigid contact lenses alters topographical corneal thickness, thinning the central epithelium and thickening the midperipheral stroma. The refractive error changes induced by orthokeratology can be explained by these topographical thickness changes. However, the underlying cellular basis for the thickness changes requires further study. Most of the thickness changes occurred in the first 10 days of treatment and more than 70% occurred on the first night of lens wear. This suggests that remodeling or redistribution of the anterior corneal layers occurs very rapidly in response to the pressures exerted by reverse-geometry lenses.

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References