Corneal Response to Rigid and Hydrogel Lenses During Eye Closure

Melvin R. O'Neal,*† Kenneth A. Poise,* and Morton D. Sarver*

Corneal changes were monitored in 14 subjects following 3 hr of eye closure while wearing selected oxygen permeable rigid and hydrogel lenses. The mean increase in corneal thickness ranged from 82.5 to 29.5 nm for rigid lenses with oxygen transmissibilities (Dk/L) between $2 \times 10^{-9}$ and $57.0 \times 10^{-9}$ (cm/sec) (ml O$_2$/ml X mmHg), respectively, and ranged from 82.5 to 23.5 nm for hydrogel lenses with Dk/L between $2.5 \times 10^{-9}$ and $70.0 \times 10^{-9}$ (cm/sec) (ml O$_2$/ml X mmHg), respectively. No differences in the amount of swelling between rigid and hydrogel lenses of the same oxygen transmissibility were observed (t-test, $P > 0.20$). Combining the swelling data for both types of lenses shows that a minimum lens oxygen transmissibility of approximately $75 \times 10^{-9}$ (cm/sec) (ml O$_2$/ml X mmHg) is necessary during eye closure to prevent contact lens induced edema. The estimated oxygen tension under a lens with this Dk/L value is 40 mmHg. Recovery of the cornea to baseline thickness follows a nonlinear time course, with the rate of dehydration decreasing as the cornea thins. For initial swelling of 40-54 μm, 55-69 μm, and 70 μm and above, the time to reach baseline thickness was 1.5, 2.0, and 2.5 hr, respectively. Effects on vision, corneal curvature, distortion, and epithelial integrity were not clinically significant during this short period of eye closure. Invest Ophthalmol Vis Sci 25:837-842, 1984

When a contact lens is placed on the eye, the oxygen tension at the tear-lens interface is reduced.¹² If the oxygen tension falls below the amount necessary to maintain normal metabolism an increase in lactate content is believed to occur in the stroma, which results in corneal edema.³

During daytime wear, most oxygen permeable rigid and soft contact lenses allow sufficient oxygen to reach the cornea to maintain normal corneal metabolism.⁴ However, during eye closure (eg, sleep) the driving force of oxygen at the anterior lens surface is reduced from 155 mmHg in air to 55-57 mmHg.⁵ Under this condition, the currently available extended-wear lenses do not transmit enough oxygen and substantial amounts of corneal edema can result.⁶ The clinical significance of corneal edema has not been established completely; however, chronic hypoxia-induced alteration of corneal metabolism may cause some of the complications that have been reported to accompany extended wear of hydrogel lenses.⁸⁻¹¹

Providing sufficient oxygen to maintain normal metabolism during eye closure requires the contact lens to have a substantially greater oxygen transmissibility (Dk/L) than available with most hydrogel materials.¹² It is possible to make rigid lenses of silicon/acrylate polymers that have Dk/L values higher than that of present extended wear hydrogel lenses. Rigid lenses with high oxygen transmissibilities may meet the corneal oxygen requirement during sleep. Additional advantages of oxygen permeable rigid compared to hydrogel lenses would include correction of complex refractive errors, improved lens maintenance, flushing of trapped debris and metabolites upon awakening, and compatibility with most ophthalmic solutions.

If rigid lenses are to be prescribed for extended wear, it is important to first evaluate the corneal response under closed eye conditions. In making this assessment, several important questions can be investigated. For example: What is the effect of lens oxygen transmissibility on the degree of corneal edema that occurs during eye closure? What lens Dk/L is necessary to prevent hypoxic edema during sleep? Is the corneal swelling that occurs during closed eye lens wear primarily a result of hypoxia or does lens rigidity, movement, and pressure (ie, mechanical interference) have

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Table 1. Summary of selected ocular parameters for the 14 subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corneal thickness (μm)</td>
<td>509</td>
<td>37</td>
<td>465 to 570</td>
</tr>
<tr>
<td>Sphere ref. error (D)</td>
<td>-2.63</td>
<td>1.83</td>
<td>+0.25 to -6.25</td>
</tr>
<tr>
<td>Cylinder ref. error (D)</td>
<td>-0.68</td>
<td>0.66</td>
<td>0 to -2.75</td>
</tr>
<tr>
<td>Horiz. Keratometry (D)</td>
<td>43.40</td>
<td>1.85</td>
<td>39.87 to 46.62</td>
</tr>
<tr>
<td>Corneal toricity (D)</td>
<td>0.46</td>
<td>0.98</td>
<td>2.00 against to 2.00 with</td>
</tr>
</tbody>
</table>

Lenses

Seven rigid (1 PMMA, 6 GPH) and five hydrogel lenses were used. All rigid lenses were 9.2 mm in diameter with a power of -3.00 D. The hydrogel lenses varied in diameter and power. The average thickness of each lens was computed from measurements made across the entire lens for the rigid lenses and across the central 11.0 mm for the hydrogel lenses. The oxygen transmissibility of each lens was measured at 35°C by the polarographic oxygen sensor method, using a curved surface (7.8 mm radius) electrode (Rehder Development Co.; Castro Valley, CA). Temperature control and lens handling were monitored strictly to increase the repeatability of the measurement.

The physical characteristics of these lenses are listed in Table 2.

Materials and Methods

Subjects

Fourteen subjects, (six women, eight men; mean age 27.5 ± 5.5 years, range 20 to 41 years) who were free of ocular disease and had no prior contact lens experience (unadapted) participated in the study. Informed consent was obtained from each subject. A summary of relevant ocular parameters is listed in Table 1.

Table 2. Physical characteristics of the test lenses

<table>
<thead>
<tr>
<th>Number</th>
<th>Proprietary name</th>
<th>Material</th>
<th>Oxygen permeability* (Dk) 35°C</th>
<th>Average thickness (mm)</th>
<th>Oxygen transmissibility† (Dk/L) 35°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PMMA</td>
<td>PMMA</td>
<td>0.2</td>
<td>0.12</td>
<td>0.2</td>
</tr>
<tr>
<td>2</td>
<td>B &amp; L Soflens</td>
<td>38.6$, HEMA</td>
<td>9.8</td>
<td>0.39</td>
<td>2.5</td>
</tr>
<tr>
<td>3</td>
<td>Boston Lens III</td>
<td>Sil/Acry</td>
<td>19.1</td>
<td>0.32</td>
<td>6.0</td>
</tr>
<tr>
<td>4</td>
<td>B &amp; L Soflens</td>
<td>38.6, HEMA</td>
<td>9.8</td>
<td>0.09</td>
<td>10.5</td>
</tr>
<tr>
<td>5</td>
<td>Boston Lens III</td>
<td>Sil/Acry</td>
<td>19.1</td>
<td>0.16</td>
<td>12.0</td>
</tr>
<tr>
<td>6</td>
<td>Boston Lens III</td>
<td>Sil/Acry</td>
<td>19.1</td>
<td>0.09</td>
<td>22.0</td>
</tr>
<tr>
<td>7</td>
<td>Boston Lens III</td>
<td>Sil/Acry</td>
<td>19.1</td>
<td>0.07</td>
<td>26.0</td>
</tr>
<tr>
<td>8</td>
<td>Hydrocurve II</td>
<td>55.0, HEMA</td>
<td>19.2</td>
<td>0.07</td>
<td>26.0</td>
</tr>
<tr>
<td>9</td>
<td>Boston Lens EW</td>
<td>Sil/Acry</td>
<td>26.3</td>
<td>0.07</td>
<td>38.0</td>
</tr>
<tr>
<td>10</td>
<td>Experimental</td>
<td>70.0, HEMA</td>
<td>38.0</td>
<td>0.08</td>
<td>50.0</td>
</tr>
<tr>
<td>11</td>
<td>Paraperm EW</td>
<td>Sil/Acry</td>
<td>57.0</td>
<td>0.10</td>
<td>57.0</td>
</tr>
<tr>
<td>12</td>
<td>Experimental</td>
<td>70.0, HEMA</td>
<td>38.0</td>
<td>0.06</td>
<td>70.0</td>
</tr>
</tbody>
</table>

* Dk units: (X10^-11) (cm^2/sec) (ml O2/ml X mmHg); † Dk/L units: (X10^-9) (cm/sec) (ml O2/ml X mmHg); $Percent water content.

lamp evaluation of corneal edema and epithelial staining was made using a Topcon SL-5D Biomicroscope (Paramus, NJ).

Baseline measurements made prior to lens insertion, included slit-lamp examination, corrected visual acuity, keratometry, and pachometry. Following lens insertion, both eyes were closed for 3 hr to obtain steady state corneal swelling.\textsuperscript{17} Also, no lens was worn during one session to determine the normal physiologic closed eye edema. Test lenses were worn on the right eye, with the left eye serving as a control. All subjects were not available to wear each lens; eight subjects wore lenses 3-8, while six subjects wore lenses 1, 2, and 9-12. At the end of 3 hr the lens was removed and central corneal thickness readings of both eyes were taken.

Following pachometry, slit-lamp examination was repeated to evaluate for corneal edema, striae, and staining. Each observation was graded on a 0–3 scale, corresponding to none, mild, moderate, and severe change, respectively. Spectacle acuity and keratometry measurements were then taken. All measurements were completed within 5 min of lens removal.

Recovery of the cornea to baseline thickness was monitored by measuring the central corneal thickness every 15 min for the first hour and each one-half hour for the next 3 hr.

Results

The mean change in central corneal thickness following 3 hr of eye closure while wearing the oxygen permeable rigid contact lenses is shown in Figure 1. There is an inverse relationship between the oxygen transmissibility of the lens and the degree of corneal swelling, with the difference between lenses being significant (\textit{F} test, \( P < 0.001 \)). The mean increase in corneal thickness ranged from 82.5 ± 6.1 \( \mu \)m to 29.5 ± 4.0 \( \mu \)m (16.5 to 5.9\%) for lenses with oxygen transmissivities (Dk/L) between 0.2 \( \times 10^{-9} \) and 57.0 \( \times 10^{-9} \) (cm/sec) (ml O\textsubscript{2}/ml \times mmHg), respectively. The curve was fitted by polynomial equation (sixth order, \( n = 48 \), \( r = 0.810 \)). The mean corneal swelling during the control sessions (no lens) was 21.0 ± 6.2 \( \mu \)m (4.2\%), range 14.6 to 31.5 \( \mu \)m, and is indicated by the dotted line.

Changes in central corneal thickness following lens removal (dehydration) are shown in Figure 2. The average time to return to baseline corneal thickness was related to the amount of induced edema. The mean recovery time was approximately 1.5, 2.0, and 2.5 hr from initial swelling levels of 40–54 \( \mu \)m, 55–69 \( \mu \)m, and 70 \( \mu \)m and above, respectively. Analysis of dehydration rates for different amounts of edema shows that the dehydration rate is the same for any given level of hydration regardless of the initial induced swelling. For example, in Figure 2, the rate of dehydration at 30 \( \mu \)m of edema is approximately 30 \( \mu \)m/hour and is the same for all three recovery curves.

The mean change in central corneal thickness following 3 hr of eye closure while wearing the hydrogel lenses is shown in Figure 3. The mean increase in corneal thickness ranged from 82.5 ± 6.8 \( \mu \)m to 23.5 ± 5.1 \( \mu \)m (16.5 to 4.7\%) for the lenses with Dk/L between 2.5 \( \times 10^{-9} \) and 70.0 \( \times 10^{-9} \) (cm/sec) (ml O\textsubscript{2}/
The mean change in central corneal thickness vs lens oxygen transmissibility (Dk/L) following 3 hr of eye closure while wearing hydrogel contact lenses. Error bars equal ±1 SD and line was fitted by polynomial equation. Dk/L units: (cm/sec) (ml O2/ml × mmHg).

There was a significant difference in the corneal swelling accompanying these lenses (F test, P < 0.001). The curve was fitted by polynomial equation (sixth order, n = 32, r = 0.958).

The swelling response data points for the hydrogel and rigid lenses were fitted by the method of least squares. Comparison of the regression lines shows no difference in the degree of swelling between rigid and hydrogel lenses of the same oxygen transmissibility (t-test, P > 0.20). The corneal swelling responses for the two types of lenses was, therefore, combined and is shown in Figure 4. The curve was fitted by polynomial equation (sixth order, n = 80, r = 0.914) and can be used to predict the effect of lens oxygen transmissibility on corneal hydration during eye closure. The derived polynomial indicates that a minimum lens oxygen transmissibility of approximately 75 × 10^{-9} (cm/sec) (ml O2/ml × mmHg) is necessary during closed eye contact lens wear to prevent more corneal swelling than the normal physiologic edema (4.2%) that occurs during eye closure.

To estimate the oxygen tension under each contact lens for the closed eye condition, calculations were made using a theoretic model proposed by Fatt and St. Helen13:

\[ \alpha P^{1/2} = (Dk/L)_{cl} \times (P_a - P), \]

where \( \alpha \) is 0.24 × 10^{-6} ml O2/cm² × sec × (mmHg)^{1/2}, \( P_a \) is the palpebral oxygen tension (55–57 mmHg), \( (Dk/L)_{cl} \) is the oxygen transmissibility of the contact lens, and \( P \) is the oxygen tension under the lens. This model assumes no tear exchange occurs under the lens when the eye is closed. The estimated oxygen tension under contact lenses for several Dk/L values is shown on the right ordinate of Figure 4. These calculations indicate that an estimated oxygen tension of 40 mmHg or greater is necessary for normal corneal metabolism.

The mean corneal and visual changes occurring after 3 hr of closed eye with and without wearing oxygen permeable rigid contact lenses are listed in Table 3. In general, corneal curvature changes and corneal dis-
Table 3. Mean visual and physiological response following three hours of closed eye with and without wearing PMMA and oxygen permeable rigid contact lenses

<table>
<thead>
<tr>
<th>Oxygen transmissibility* (Dk/L) 35°C</th>
<th>Corneal curvature (D)</th>
<th>Visual acuity (log MAR)</th>
<th>Corneal edema (grade)</th>
<th>Corneal striae (grade)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Horizontal</td>
<td>Vertical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.2</td>
<td>-0.30 ± 0.36</td>
<td>+0.42 ± 0.23</td>
<td>0.14 ± 0.05</td>
<td>2.50</td>
</tr>
<tr>
<td>6.0</td>
<td>+0.22 ± 0.38</td>
<td>+0.39 ± 0.45</td>
<td>0.07 ± 0.06</td>
<td>1.13</td>
</tr>
<tr>
<td>12.0</td>
<td>-0.19 ± 0.38</td>
<td>+0.19 ± 0.37</td>
<td>0.06 ± 0.07</td>
<td>0.63</td>
</tr>
<tr>
<td>22.0</td>
<td>-0.02 ± 0.20</td>
<td>+0.23 ± 0.42</td>
<td>0.01 ± 0.03</td>
<td>0.25</td>
</tr>
<tr>
<td>26.0</td>
<td>-0.05 ± 0.21</td>
<td>+0.17 ± 0.33</td>
<td>0.04 ± 0.05</td>
<td>0</td>
</tr>
<tr>
<td>38.0</td>
<td>-0.19 ± 0.17</td>
<td>0 ± 0.25</td>
<td>0 ± 0</td>
<td>0</td>
</tr>
<tr>
<td>57.0</td>
<td>+0.15 ± 0.20</td>
<td>+0.07 ± 0.27</td>
<td>0 ± 0</td>
<td>0</td>
</tr>
<tr>
<td>Control</td>
<td>-0.01 ± 0.14</td>
<td>+0.05 ± 0.11</td>
<td>0 ± 0</td>
<td>0</td>
</tr>
</tbody>
</table>

* (×10⁻⁹) (cm/sec) (ml O₂/ml × mmHg).

Discussion

The amount of corneal swelling following 3 hr of closed eye lens wear decreased as lens oxygen transmissibility (Dk/L) increased. Our data indicates a minimum lens Dk/L of approximately 75 × 10⁻⁹ (cm/sec) (ml O₂/ml × mmHg) is necessary to prevent the corneal swelling that results from contact lens wear during eye closure.

Corneal swelling induced by hypoxia tends to reach steady state in 3 hr¹⁷ and, therefore, a 3-hr test period provides a reasonable indicator of overnight edema accompanying extended wear. However, 3 hr of eye closure under laboratory conditions is an approximation of the normal overnight, sleep condition. The rapid eye movements (REM) and occasional eye openings that occur during normal sleep may allow some tear exchange (eg, increased oxygen) beneath the oxygen permeable rigid lens, which may reduce the amount of swelling. Also, these findings are based on a relatively small sample size, and testing with more subjects and longer closed eye duration are needed to verify these results. Our swelling responses, however, are in agreement with those reported for 6 hr of eye closure¹⁶ and following sleep during extended wear.⁶

We used an equation based on average corneal properties to estimate the oxygen tension at the tear-lens interface for closed eye wear of contact lenses having different oxygen transmissibilities. Our results indicate that an oxygen tension of 40 mmHg is necessary to prevent hypoxic edema. This oxygen level is in agreement with open eye measurements showing no edema for wear of a soft lens having a Dk/L of 22 × 10⁻⁹ (cm/sec) (ml O₂/ml × mmHg)¹⁹ and corresponding 5.0% equivalent oxygen percent (EOP) under the lens.²⁰

The minimum oxygen requirement, 40 mmHg, determined using the contact lens technique is higher than the thresholds determined by Polsky and Mandell (11-19 mmHg)²¹ and by Mandell and Farrell (23-37 mmHg)²² who used gas goggles to induce hypoxia. These differences may be due to increased metabolic activity accompanying contact lens wear, higher corneal temperature when a contact lens is worn with the eye closed, or differences between inducing corneal hypoxia with goggles as compared with contact lenses. During the goggle experiments, the flow of gas across the cornea may result in an increased effect of evaporation on corneal thickness and a limited swelling response.

Approximately the same amount of corneal swelling resulted when either oxygen permeable rigid or hydrogel lenses with similar oxygen transmissibility were worn during 3 hr of eye closure. This similarity of swelling responses between rigid and soft lenses of the same Dk/L suggests that the swelling is caused by corneal swelling that results when either oxygen permeable rigid or hydrogel lenses with similar oxygen transmissibility were worn during 3 hr of eye closure. This similarity of swelling responses between rigid and soft lenses of the same Dk/L suggests that the swelling is caused by corneal distortion were minimal, with only eight cases of grade 1, one case of grade 2, and one case of grade 3 distortion occurring. The corneal curvature changes were variable and do not appear to be related to the amount of swelling. Corneal staining occurred in 11 (seven cases of grade 1, four cases of grade 2) of the 44 GPH lens sessions and typically was associated with cases of de-centered lenses. Visual acuity was 20/20 in most cases and by the end of the recovery period acuity had returned to 20/20 for all subjects. These changes were similar to those found accompanying wear of the hydrogel lenses.

Corneal edema, as assessed with the slit lamp, was visible in only a few of the rigid lens sessions, appearing as a diffuse, nonlocalized haze. Corneal striae occurred either as single lines, or more commonly in groups of lines. Corneal striae was measured as grade 1 for 1-6 lines; grade 2 for 7-11 lines, and grade 3 for 12 or greater. For the rigid lens sessions, the grade of corneal striae correlated well with the amount of corneal edema (r = 0.83). Corneal swelling of <40 μm (8%), 40-50 μm (8-10%), 55-65 μm (11-13%), and 70 μm (14%) and above was usually accompanied by corneal staining occurred in 11 (seven cases of grade 1, one case of grade 2, and one case of grade 3 distortion occurring. The corneal curvature changes were variable and do not appear to be related to the amount of swelling. Corneal staining occurred in 11 (seven cases of grade 1, four cases of grade 2) of the 44 GPH lens sessions and typically was associated with cases of de-centered lenses. Visual acuity was 20/20 in most cases and by the end of the recovery period acuity had returned to 20/20 for all subjects. These changes were similar to those found accompanying wear of the hydrogel lenses.

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neal hypoxia rather than mechanical interference and that there is little or no tear exchange under either lens type during eye closure. The changes in vision, corneal curvature, distortion, and epithelial integrity were not clinically significant for wear of the rigid lenses during this short period of eye closure and were similar to that commonly reported for closed eye wear of hydrogel lenses. Corneal molding and epithelial staining may increase with long-term, closed, eye wear and needs additional study.

The ability to assess the corneal swelling accompanying closed eye wear of rigid lenses without the use of a pachometer is of clinical importance. The central corneal edema observed in daily wear of rigid lenses was not usually seen with closed eye wear and, therefore, is not a useful indicator of edema. However, corneal striae, which is observed in open eye wear of hydrogel lenses but not rigid lenses, did occur with rigid lenses during the closed eye condition. The degree of corneal striae correlated well with the amount of corneal swelling (r = 0.83), which may be helpful in monitoring corneal edema following sleep with rigid lenses.

We measured the recovery of the cornea from various levels of edema and found that the time to return to baseline thickness was related to the initial amount of swelling. Corneal dehydration follows a nonlinear time course, with the rate of dehydration decreasing as the cornea thins. Further, for any specific level of hydration, the rate of dehydration was the same regardless of the initial edema induced, suggesting that dehydration of the normal cornea is only affected by the current level of edema. During recovery, the corneal thickness always became thinner than baseline (overshoot) for a short period of time. These dehydration relationships and "overshoot" phenomenon have not been described previously and may be useful in investigating the function of the endothelial pump mechanism. Whether swelling recovery is similar for corneas with endothelial disease is not known and is the subject of further study.

Key words: cornea, swelling, closed eye, contact lenses, oxygen transmissibility, oxygen tension

References