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Abstract
Contact lenses are a popular and frequently used mode of correcting refractive error and are occasionally used to treat diseased and degenerative processes. As both Rigid Gas Permeable (RGP) and Soft Contact Lenses (SCL) lie directly on the cornea buffered only by the tear layer, it is the cornea that shows the first signs of adverse effects of contact lens wear. Contact lenses can affect the cornea both mechanically, due to their physical presence on the eye, as well as indirectly through interference with the normal physiology of the cornea. As the mode of contact lens wear increases through new materials and designs, so too do the needs of the patient. The population trend is moving toward the older patient and this group of patients are less tolerant to mild corneal hypoxia and mechanical trauma than the younger population. At the same time, as their corneal topography and physiology is more fragile, there is an increased need for thicker contact lenses (e.g. bifocal lenses). With this situation, the early detection of contact lens complications is critical in order to preserve a healthy visual system. A good patient evaluation assessing the ocular health, combined with strong patient motivation is the first step in successful contact lens wear. Secondly, a thorough knowledge of materials, lens design, fits and care solutions also contribute to successful long term contact lens wear. Once a patient has been fitted with their lenses, the recognition of early contact lens complications is essential. The earlier the identification and correct treatment is undertaken, the better the prognosis for continued success in contact lens wear. Optometry students and new practitioners (who may have had little experience with contact lens complications), often find it difficult to correctly identify the problems and implement the correct treatment. This thesis was developed to provide a pictorial reference guide with an accompanying written identification and treatment guideline for eight of the most common contact lens complications. The early identification and implementation of appropriate treatment will usually stop the progression of the corneal/lid problem, preserving and/or re-establishing a healthy ocular system, while still maintaining good visual acuity. Successful treatment of contact lens associated ocular problems can lead to continued healthy contact lens wear by the patient.

Degree Type
Thesis

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ATLAS OF COMMON COMPLICATIONS ASSOCIATED WITH R.G.P. AND S.C.L. WEAR

by

Nancy A. Selinger

A thesis submitted to the faculty of the
College of Optometry
PACIFIC UNIVERSITY
FOREST GROVE, OREGON
for the degree of

Doctor of Optometry

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Advisor:

Cristina M. Schnider, O.D.
SIGNATURE PAGE

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CHRISTINA M. SCHNIDER O.D. (Advisor)

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To my children Meghan, Adam and Caitlyn, I would like to say thank you for your love and patience in allowing me the time to successfully complete this thesis.
Nancy Selinger was born and raised in Vancouver, B.C., Canada. After receiving her R.N. degree, she worked in the operating room for several years, primarily in Neurosurgery and E.E.N.T. During her nursing career Nancy lived in Regina, Saskatchewan, where she married John Selinger and had their three children Meghan, Adam and Caitlyn.

Upon returning to school she attended the University of Regina and Pacific University, where she received her B.Sc. degree in 1991 and is currently a fourth year student in the Doctor of Optometry program at Pacific. Upon graduation, Mrs. Selinger anticipates working in a full scope practice with an emphasis on contact lenses.
ABSTRACT

Contact lenses are a popular and frequently used mode of correcting refractive error and are occasionally used to treat diseased and degenerative processes. As both Rigid Gas Permeable (RGP) and Soft Contact Lenses (SCL) lie directly on the cornea buffered only by the tear layer, it is the cornea that shows the first signs of adverse effects of contact lens wear.

Contact lenses can affect the cornea both mechanically, due to their physical presence on the eye, as well as indirectly through interference with the normal physiology of the cornea.

As the mode of contact lens wear increases through new materials and designs, so too do the needs of the patient. The population trend is moving toward the older patient and this group of patients are less tolerant to mild corneal hypoxia and mechanical trauma than the younger population. At the same time, as their corneal topography and physiology is more fragile, there is an increased need for thicker contact lenses (e.g., bifocal lenses). With this situation, the early detection of contact lens complications is critical in order to preserve a healthy visual system.

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The early identification, and implementation of appropriate treatment will usually stop the progression of the corneal/lid problem, preserving and/or re-establishing a healthy ocular system, while still maintaining good visual acuity. Successful treatment of contact lens associated ocular problems can lead to continued healthy contact lens wear by the patient.
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HEALTHY EYE
CONDITION - "VASCULARIZATION"

VASCULARIZATION - the formation and extension of vascular capillaries within and into a normally avascular cornea.¹

NEOVASCULARIZATION - the formation and extension of new vascular capillaries within and into a previously vascularized cornea.¹

GHOST VESSELS - empty blood vessels where only the vessel wall remains. Seen as faint white lines.⁴ May persist indefinitely and will refill with blood at the slightest provocation.⁴

VASCULAR RESPONSE to CL wear occurs in three stages:³

- Stage 1 - the terminal capillaries that are normally empty now fill with blood
- Stage 2 - active stage where there is actual new vessel outgrowth from the limbal arcade
- Stage 3 - unless treated, the new vessels continue to grow into all layers of the cornea (may be straight or tortuous):
  a) possibly form new limbal arcades or
  b) possibly grow into cornea & approach or cross pupil zone jeopardizing V/A

Associated with:

1. tight fitting contact lens
2. EW SCL³⁹ > DW SCL³⁹ > RGP > 0 lens¹

ETIOLOGY

A. Hypoxia - decreased O₂ interferes with normal aerobic metabolism. Anaerobic metabolism takes place producing excessive lactic acid => cornea vascularization.¹⁴

B. Edema - due to chronic low grade hypoxia.  
  1. causes softening of the stroma thus allowing vessel growth into the stroma.¹³⁴³⁹

C. Mechanical Injury - chronic irritation of the C.L. to the cornea epithelium induces release of vaso-stimulating agent in the area leading to new vessel growth.¹³³⁹
VASCULARIZATION (continued)

ETIOLOGY (continued)

D. Refractive Error - increased C.L. thickness => reduced O₂ transmission.
   - reduced vascularization with patient RE from minus 4.00 => plano.

SYMPTOMS

A. Mild/Moderate - generally asymptomatic

B. Severe - burning Symptoms due to increased
   - itching corneal edema not
   - reduced V/A vascularization
   - possibly moderate redness

EVALUATION

A. Slit Lamp Evaluation
   - direct focal illumination for non-transparent cornea
   - direct retro-illumination for transparent cornea
   - direct illumination - narrow beam to determine depth
   - magnification 15-40X - white & red free illumination
   - marginal retro-illumination to observe ghost vessels
   - seen as faint white lines

B. Depth
   - Superficial (S) - vessels in the stroma are continuous with conjunctival circulation
   - Deep (D) - typically a large feeding vessel from the limbus (usually in mid stroma) develops into finer, tortuous branches that end in bulbs.

C. Limbus
   - The limbal grey zone contains the Girdle of Vogt with the inner margin of the limbus measured from here. (See figure 1.)
Fig. 1  
Vascularization  
Limbal Grey Zone

Fig. 2  
Vascularization  
Grade 1

Fig. 3  
Vascularization  
Ghost Vessels  
Grade 2
**VASCULARIZATION (continued)**

**GRADES AND TREATMENT**

A. **Grading**

A. All grading is done from the limit of the limbal grey zone under normal lighting conditions.

B. Applicable for DW and EW RGP and SCL

C. 5 level grading scale - may add (superficial) S or (deep) D as determined by SLE using parallelepiped illumination.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Distance from limit of visible limbal grey zone to stroma</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.0 mm vascular response</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>0.5 mm vascular response</td>
<td>1. Monitor limbal vessels at regular intervals.</td>
</tr>
</tbody>
</table>
| 2     | 1.0 mm vascular response                                 | 1. Increase Dk/L<sup>1,4</sup>  
2. New lens and/or material (SCL => RGP)<sup>1</sup>  
3. Reduce wear time (EW => DW)<sup>1,4</sup>  
4. Change solution (reduce allergic response) <sup>1,4</sup> |
| 3     | 1.5 mm vascular response - Serious<sup>1</sup>  
- Beyond 1.5mm indicates accelerated growth rate of vessels into stroma | 1. Discontinue lens<sup>1</sup> wear for extended time/permanently  
2. If CL wear resumed, change parameters as per Grade 2 |
| 4     | 2.0 mm vascular response - Vessels are approaching pupil zone at rapid rate and vision is threatened. | 1. Discontinue wear<sup>4</sup> permanently  
2. Medical referral laser/diatherapy /keratoplasty<sup>1</sup> |
VASCULARIZATION (continued)

Fig. 4
Vascularization
Grade 3

Fig. 5
Vascularization
Grade 4

Fig. 6
Vascularization
Pannus with underlying infiltrates
Grade 4
**VASCULARIZATION** (continued)

**PREVENTION**

A. High Dk/L
B. Reduced mechanical irritation
C. Good movement
D. Good care system
E. Regular evaluation of lens.

**PROGNOSIS**

A. Grade 1 may undergo some regression of new blood vessels when contact lens wear is discontinued,¹
B. Grade 2 established corneal vessels do not fully regress and can persist indefinitely as ghost vessels.⁴
C. Cessation of lens wear will usually halt the progression of vessel growth into the cornea and resumption of contact lens wear must proceed with caution.
D. If there is subsequent injury or if inflammation occurs in the same area as old neovascularization, the ghost vessels will quickly refill with blood.
CONDITION - "CONTACT LENS- SUPERIOR LIMBIC KERATOCONJUNCTIVITIS"

CL- SLKC - is usually a bilateral chronic inflammation of the superior tarsal and bulbar conjunctiva, the superior limbus and superior peripheral cornea. Although CL-SLKC and Theodore's SLKC share many of the same diagnostic features, Theodore's SLKC is usually associated with hyperthyroid disease. While a history of thyroid disease predisposes patients of all ages, SLKC is usually found in older women. SLKC can occur independent of CL wear, seen in younger patients with history of thyroid disease. Pathological changes are:

- affected tissue is chemosed
- epithelial cells hypertrophy - epithelium contains lipid inclusion bodies filled with fluid vacuoles
- surface epithelium has fewer microvilli and is more keratinized
- inflammatory cells accumulate in underlying conjunctival stroma

Associated with:

1. abnormal corneal epithelium, altered tear film and abnormal lid posture
2. contact lens wear (SCL)

ETIOLOGY

A. **Specific etiology** - contact lens wear.

B. **Contributing conditions**

1. tissue hypoxia
2. mechanical irritation of the lens as it moves over the limbus
3. reaction to preservatives in lens care products e.g: Thimerosal
4. environmental antigens adhere to lens deposits and react with corneal tissue in an Ag- Ab reaction
CONTACT LENS - SUPERIOR LIMBIC KERATOCONJUNCTIVITIS
(continued)

SYMPTOMS

A. Photophobia7,12,16
B. Foreign Body sensation - increased lens awareness7,16
C. Burning7,16,42
D. Itching, Hazy vision 12,16,42
E. Pain7
F. Lens intolerance12,42
G. Dryness, Filaments 42

SIGNS

A. Superior tarsal conjunctiva and bulbar conjunctiva12
   - diffuse papillary hypertrophy reaction in tarsal conjunctiva11,16
   - punctate stain with rose bengal and fluorescein6,16,41
   - edematous

B. Superior bulbar conjunctiva surface16
   - thickened (due to edema)
   - increased keratinization
   - decreased surface microvillia
   - hypertrophied epithelial cells
   - hyperemia24
   - punctate staining24

C. Superior limbus - cornea and adjacent conjunctiva11
   - hyperemic
   - epithelial haziness
   - sub-epithelial infiltrates12,16,42
   - stains with rose bengal and fluorescein16
   - fibro-vascular micropannus consisting of vascularized connective tissue11,12,16,42
   - superior punctate staining42
   - superior corneal surface irregularities42

EVALUATION

A. Slit Lamp Evaluation
   - diffuse (low magnification) and direct focal (optic section or parallelepiped) illumination
   - moderate to high magnification
   - must raise upper lid to examine superior limbic region

B. Culture
   - negative6
SUPERIOR LIMBIC KERATOCONJUNCTIVITIS (SLK)

Fig. 7 SLK  
Grade 2

Fig. 8 SLK  
Grade 3

Fig. 9 SLK  
Grade 3+
**CONTACT LENS - SUPERIOR LIMBIC KERATOCONJUNCTIVITIS**  
(continued)

## GRADES AND TREATMENT

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal tarsal and bulbar conjunctiva</td>
<td>No treatment</td>
</tr>
</tbody>
</table>
| 1     | - Mild hyperemia - superior bulbar/tarsal conjunctiva  
- Mild burning/itching | 1. Reduce wear time (EW => DW)  
2. Ocular lubricants\(^{12,16}\)  
3. Monitor |
| 2     | - Increased conjunctiva inflammation\(^{41}\)  
- Moderate conjunctiva stain\(^{41}\)  
- Beginning of pannus formation on superior bulbar/tarsal conjunctiva | 1. Discontinue lens wear until all inflammation subsides\(^{12,16}\)  
2. Lubricants\(^{12,16}\)  
3. Change to non-preserved solutions \(^6,11,\) use heat disinfection\(^{11}\) |
| 3     | - Few corneal infiltrates\(^{41}\)  
- Corneal punctate stain \(^41\)  
- Conjunctiva hypertrophy  
- Pannus extends to and over the limbus | As for Grade 2 plus:  
1. New lens material - use greater deposit resistant material\(^{16}\)  
2. SCL => RGP: this may be the best option due to low incidence of CL-SLK with this type of lens\(^6,16\)  
3. Looser fit  
4. Redesign SCL edges  
5. Cromolyn Sodium 4% qid (mast cell stabilizer)\(^7\) |
| 4     | - Increased corneal stain => coalesce  
- Increased conjunctiva hypertrophy  
- Superior pannus formation extends into central cornea\(^{41}\)  
- Destruction of Bowman's membrane\(^{46}\) associated with formation of sub-epithelial opacities \(^{41}\) | 1. Discontinue lens wear until inflammatory process is completely resolved - may take weeks to months \(^{42}\)  
2. CL wear may be permanently discontinued  
3. Refit as per Grade 3 |
CONTACT LENS - SUPERIOR LIMBIC KERATOCONJUNCTIVITIS
(continued)

GRADES AND TREATMENT (continued)

Note: Mild CL- SKL is easy to miss unless the upper lid is elevated and a critical evaluation of the superior limbus is made.\textsuperscript{16}

PREVENTION:

A. Ensure good CL fit and design (e.g. rounded edges).
B. Monitor superior limbus during routine examinations.
C. Use of preservative free solutions.
D. Use of a good care regime which will reduce deposit build-up.
E. Use of high Dk/L lens material will reduce hypoxia.

PROGNOSIS:

A. Good if condition is diagnosed early and treatment is undertaken.
B. If grade 4 level is reached the prognosis is guarded as the contact lens wear may have to be permanently discontinued.
CONDITION - "EPITHELIAL MICROCYSTS"

EPITHELIAL MICROCYST - minute, scattered, irregular shaped dots that are postulated to be pockets of dead cellular matter\textsuperscript{4,9} 
- originate in the lower epithelial layers and migrate toward the surface along with normal epithelial cell growth\textsuperscript{13} 
- usually located para-central in the mid-periphery of the cornea\textsuperscript{9} 
- can occur in normal population non contact lens wearers - may occasionally see 1 or 2 microcysts\textsuperscript{10}

Associated with:

1. Chronic hypoxia - the development of microcysts is inversely related to the Dk/L\textsuperscript{7}
2. Chronic suppression of epithelial metabolism\textsuperscript{14}
3. Mechanical irritation
4. EW SCL (and to a much lesser degree, DW SCL and EW RGP)\textsuperscript{14}
5. Wearing EW SCL for 1-2 months

ETIOLOGY

A. **Etiology**: believed to be due to a disorder of epithelial cellular growth\textsuperscript{11,4}

SYMPTOMS

A. Generally asymptomatic\textsuperscript{4}
B. Decreased V/A with increased number of microcysts\textsuperscript{4}
C. Bilateral but asymmetrical
D. Cyclical - number may fluctuate during lens wear with microcysts disappearing periodically

SIGNS

A. **Slit lamp examination** 
   - irregular shaped grey-white translucent small dots (approximately 15-50 um)\textsuperscript{1,11} 
   - appear to be particles of debris in the tear film but remain fixed with blinking\textsuperscript{4} 
   - may observe negative stain if cysts cause small elevation at corneal surface but did not rupture\textsuperscript{8} 
   - may observe punctate corneal stain in dot like pattern if cysts have ruptured at the surface\textsuperscript{8,9} 
   - stained areas may coalesce to produce corneal erosion\textsuperscript{9}
EPITHELIAL MICROCYSTS (continued)

EVALUATION

Slit Lamp Evaluation
- moderate to high magnification (15-35X)
- marginal retro-illumination or direct retro-illumination

DIFFERENTIAL DIAGNOSIS:

A. Microcysts - shows reversed illumination where the dark part of cyst is on the side of the bright background and the light portion of the cyst is on the side of the dark background. Reversed illumination is possibly due to the high index of refraction material inside the microcyst relative to that in the surrounding tissue.

B. Vacuoles - transparent, fluid filled, round dots that look like tiny air bubbles, varying in size from 20 to 50 μm. Usually in discrete units but may be seen in clusters of 2. Do not stain with fluorescein as they are intraepithelial. Shows non-reversed illumination where the dark part of the vacuole is on the side with the dark pupil and the bright side of the vacuole is on the side with the brightly lit iris. Generally innocuous and require no action.
EPITHELIAL MICROCYSTS

**Fig. 10** Microcysts
No erupting
Grade 2

**Fig. 11** Microcysts and Vacuoles
★ Microcysts
▲ Vacuoles

**Fig. 12** Microcysts erupting
★ Black - Neg Stain
▲ Pool - Erupted
Grade 3
EPITHELIAL MICROCYSTS (continued)

**GRADES AND TREATMENT**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Acceptable</td>
<td>No treatment</td>
</tr>
<tr>
<td></td>
<td>- x &lt; 10</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Acceptable</td>
<td>1. No treatment</td>
</tr>
<tr>
<td></td>
<td>- 10 &lt; x &lt; 20</td>
<td>2. Monitor patient with follow-up visits</td>
</tr>
<tr>
<td>2</td>
<td>20 &lt; x &lt; 50</td>
<td>1. Discontinue lens wear until number of cysts reaches a more acceptable level and no epithelial stain is present4,9</td>
</tr>
<tr>
<td></td>
<td>- Break in cell layer</td>
<td>2. May reduce wear time (EW =&gt; DW)</td>
</tr>
<tr>
<td></td>
<td>- Stain with 2+ cysts</td>
<td>3. Follow-up/monitor</td>
</tr>
<tr>
<td></td>
<td>- May become infected</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>x &gt; 50</td>
<td>As for Grade 2 plus:</td>
</tr>
<tr>
<td></td>
<td>- Moderate to heavy number of cysts</td>
<td>1. Refit with high Dk/L DW RGP with reduced thickness or thin, high H2O DW SCL14</td>
</tr>
<tr>
<td></td>
<td>- Punctate stain is present</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>x &gt; 50</td>
<td>As for Grade 3</td>
</tr>
<tr>
<td></td>
<td>- High number of cysts</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Coalesced punctate staining suggesting corneal erosion9</td>
<td></td>
</tr>
</tbody>
</table>

**PROGNOSIS**

A. Prognosis is good.4
B. It takes 1 => 2 months of wearing EW lenses for microcysts to form.
C. It may take from 1 => 3 months for microcysts to resolve.
D. When initially discontinuing EW len wear, the number of microcysts typically increase for the first week and slowly decrease thereafter.4,9
CONDITION - "CORNEAL INFILTRATES"

CORNEAL INFILTRATES - aggregations of inflammatory cells such as monocytes and polymorphonuclear phils (PMN's)\textsuperscript{4,14,15}. They may be located near an area of necrotic tissue\textsuperscript{27}:
1. sub epithelial\textsuperscript{11}
2. deep epithelium\textsuperscript{11,14}
3. anywhere in the stroma between the collagen fibers\textsuperscript{14}

Associated with:
1. contact lens hypersensitivity (Thimerosal)\textsuperscript{11,15}
2. preserved solutions\textsuperscript{4,14,15}
3. infections\textsuperscript{4,15}
   a. bacterial exotoxins\textsuperscript{14}
   b. virus
   c. chlamydia\textsuperscript{14}
   d. staph immune complexes\textsuperscript{14}
   e. corneal hypoxia
   f. (adenovirus - patient aware of associated cold/flu like symptoms)
4. prolonged hypoxia\textsuperscript{4,15}
5. immune response\textsuperscript{4,15}
6. physical irritation\textsuperscript{4}
7. tight fitting lens\textsuperscript{4,11,14}
8. metabolic wastes trapped under the lens\textsuperscript{4}
9. risk - EWCL >> DWCL\textsuperscript{15}

ETIOLOGY

A. Inflammatory cells are believed to have entered the cornea via the tear film or limbal blood vessels\textsuperscript{11}

B. DIFFERENTIAL DIAGNOSIS:
1. Epithelial Microcysts - do not occur in discrete patches but appear more uniformly scattered\textsuperscript{14}
2. Vacuoles - do not reflect light and therefore are difficult to see with direct focal illumination\textsuperscript{11}
3. Scar - appears as more superficial or full thickness

SYMPTOMS

A. Condition may be symptomatic or asymptomatic
B. Usually asymptomatic with white quiet eye
C. If symptomatic, patient may present unilaterally with\textsuperscript{14}:
   1. red eye
   2. foreign body sensation
   3. lacrimation
   4. discharge
   5. photophobic
CORNEAL INFILTRATES (continued)

SIGNS

A. Slit lamp examination:
   - may observe single or multiple translucent area(s), round
grey-white, well demarcated bodies most commonly located in
the superior cornea under the upper lid (10 => 2 o'clock
location),11
   - 93% are found in the peripheral cornea15
   - may occur as band-shaped forms near the limbus or discrete
spots scattered throughout the cornea27

B. Size
   - Usually 0.5 => 2.0 mm diameter11

EVALUATION

A. Slit Lamp Evaluation
   - low => moderate magnification 10-20X (optic section)
   - marginal retro-illumination
   - indirect retro-illumination
   - with high magnification (40X), epithelial infiltrates will
appear as grey bodies within an infiltrative area14
   - direct focal illumination - infiltrates may appear as amorphous
shapes with a slightly dense center14
   - look for condition of focal or diffuse translucent grey-
white demarcated bodies commonly in 10 => 2 o'clock
area in peripheral superior cornea under upper lid

GRADES AND TREATMENT

A. Grading
   A. Graded as for color density nebula => macula=> leukoma
   B. Limbus is the key area to evaluate infiltrates14

The appearance of infiltrates indicates that a potentially serious tissue
reaction is imminent. Consider the possibility of an ulcer/infectious
process.
**CORNEAL INFILTRATES**

**Fig. 13** Infiltrates
Grade 1

**Fig. 14** Infiltrates
Grade 2

**Fig. 15** Infiltrates
Grade 4
**CORNEAL INFILTRATES** (continued)

**GRADES AND TREATMENT** (continued)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Appearance</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No infiltrates</td>
<td>No treatment</td>
</tr>
</tbody>
</table>
| 1     | Mild inflammatory process - see slight haze | 1. Clean lens  
2. Non-preserved solutions  
3. Monitor |
| 2     | Moderate inflammatory process with increase in density of stromal haze and increased number of hazy areas | **As for Grade 2 plus:**  
1. Discontinue lens wear for 2-3 weeks, symptoms will cease in 1-2 days  
2. Non-preserved solution  
3. Refit with looser lens  
4. Clean lens |
| 3     | - Severe infiltrate process  
- Multiple or increasing density of infiltrates  
- No staining present | **As for Grade 2 plus:**-  
1. Discontinue lens wear until infiltrates completely resolved (usually resolve in 1 to 2 months) After a quiet period with no CL wear, no meds and cornea free of infiltrates, patient may resume CL wear  
2. Change material (SCL => RGP)  
3. Reduce wear time (EW => DW)  
4. Steroids  
5. Hypertonics - prn to increase visual acuity  
6. Increase Dk/L |
| 4     | EPITHELIAL DEFECT THAT STAINS - very serious | 1. Discontinue lens wear with immediate medical attention |
CORNEAL INFILTRATES (continued)

PROGNOSIS:

A. Good if diagnosed and treated immediately.
B. If condition progresses to infiltrative keratitis - severe visual consequences may occur.¹⁵
C. CL intolerance may continue long after infiltrates have resolved.
D. Patient may be left with residual scar.
CONJUNCTIVAL HYPEREMIA - benign response that is demonstrated as an overfilling of limbal vessels with RGP's and increased hyperemia of conjunctiva with SCL's\textsuperscript{31} - chronic hyperemia is common among contact lens wearers: SCL with preserved solution > SCL with non-preserved solution > RGP > non-CL wearers\textsuperscript{31} - SCL solutions are often preserved with Thimerosal which is an organic mercurial product with broad spectrum anti-microbial properties.\textsuperscript{42,43} It is widely used in non-ocular products such as vaccines, skin testing allergens and anti-venoms as well as ocular products such as CL solutions.

The mechanism of ocular Thimerosal sensitivity reaction is believed to be type IV (delayed cell-mediated) alone or possibly some effect from type III (delayed complex-mediated) allergic reaction\textsuperscript{45}. In both type III and IV allergic reaction, there must have been previous exposure to the allergen\textsuperscript{42} - may be active (inflammatory), passive (tight lens) or both\textsuperscript{3}

Associated with:

1. dry eyes/ocular dryness
2. eye irritation - possibly (but not exclusively) related to lens design, wear time, environmental factors (fumes, chemicals) or foreign bodies.

ETIOLOGY

A. Conjunctival hyperemia (limbus) can be a direct response to contact lens issues such as:\textsuperscript{31}
   1. surface deposits\textsuperscript{3,30} 
   2. hypoxia 
   3. poor fitting (e.g. tight edges)\textsuperscript{3} 
   4. solution preservatives (e.g. Thimerosal)\textsuperscript{3} 
   5. damaged lenses\textsuperscript{3,30} 
   6. contaminated lenses by toxic fumes, spray, liquids, etc.\textsuperscript{30} 
   7. residual "Foreign body" response\textsuperscript{3} 
   8. lens dehydration secondary to corneal/conjunctival dehydration of tear film

B. Response to issues not directly related to contact lens wear eg:\textsuperscript{3,31}
   1. reduced sleep 
   2. hay fever/allergies 
   3. eyestrain
CONJUNCTIVAL HYPEREMIA

Fig. 16 Hyperemia
Grade 1

Fig. 17 Hyperemia
Note the
Pinguecula
Grade 2

Fig. 18 Hyperemia
Grade 3
CONJUNCTIVAL HYPEREMIA (continued)

ETIOLOGY (continued)

4. wind, dust, smoke, smog, heat, etc.
5. alcohol
6. other environmental irritants to visual system
7. low ambient humidity
8. dry eyes

SYMPTOMS

A. Patient is generally asymptomatic
B. Foreign body sensation - stinging, burning, tearing

SIGNS

A. Conjunctiva Hyperemia
   - SCL is 360 degrees
   - RGP is usually at 3 and 9 o'clock

EVALUATION

A. Slit Lamp Evaluation
   - direct focal illumination
   - diffuse illumination with low magnification (10X)

B. Direct Observation

GRADES AND TREATMENT

<table>
<thead>
<tr>
<th>Grade</th>
<th>Appearance</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No hyperemia</td>
<td>No treatment (e.g. anaemia, albino)</td>
</tr>
<tr>
<td>1</td>
<td>Normal conjunctiva hyperemia</td>
<td>No treatment</td>
</tr>
</tbody>
</table>
| 2     | Mild hyperemia        | 1. Monitor
          |                                          | 2. Lubricants
          |                                          | 3. Assess lens for damage/ defects       |
| 3     | Moderate hyperemia    | As for Grade 2 plus:
          |                                          | 1. Reduce wear time
          |                                          | 2. Increase enzyme cleaning (Surfactant)
          |                                          | 3. Non-preserved solutions               |
CONJUNCTIVAL HYPEREMIA (continued)

Fig. 19  Hyperemia
Note indent on cornea due to bound lens
Grade 4

Fig. 20  Hyperemia
Grade 4+
CONJUNCTIVAL HYPEREMIA (continued)

GRADES AND TREATMENT (continued)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Appearance</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 (cont.)</td>
<td></td>
<td>4. Punctal occlusion if dry eye problem,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Artificial tears</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. Improve fit - blend RGP</td>
</tr>
<tr>
<td>4</td>
<td>Severe hyperemia</td>
<td>As for Grade 3 plus:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Discontinue lens wear until Grade 1 hyperemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Vasoconstrictor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Increase Dk/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. EW =&gt; DW</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Refit with new material, well blended RGP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. Flatter BC with SCL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7. If hyperemia does not resolve immediately with discontinuing lens wear,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>assess for underlying pathology.</td>
</tr>
</tbody>
</table>

PREVENTION:

A. Well fitting, well wetting lens.
B. Patients must be aware to use "full" blinks.
C. Supplemental lubricants.
D. Non-preserved solutions.
E. Moist environment (humidifiers).

PROGNOSIS

A. With well fitting lens and good lens care, hyperemia can be well maintained at a Grade 1 level.
B. Unless appropriate treatment is initiated, may progress to neovascularization (a more serious complication).
CONDITION - "EDEMA"

EDEMA - is a local or generalized condition where there is an excess accumulation of fluid in the tissue due to decreased O₂ to the cornea. This causes the cornea epithelium to undergo anaerobic respiration to conserve energy and results in a build up of the byproduct, lactic acid. The increase in lactic acid raises the stromal osmotic pressure and there is an influx of fluid entering the stroma in order to balance the osmotic conditions. This influx of fluid may result in folds and/or striae.

Associated with:
1. soft contact lens wear and to a much lesser extent, R.G.P. and PMMA.

ETIOLOGY

A. Hypoxia
B. Trauma after a perforating wound
C. Mechanical irritation
D. Inflammatory states
E. Diabetes
F. Idiopathic
G. Tear hypotonicity
H. Change in temperature and pH
I. Reduced barrier effect

Possible Mechanism:
- Swelling occurs due to reduced O₂ equally over the entire cornea with SCL.
- Edema appears primarily in the central cornea area (as CCC) with PMMA lenses. The sole source of the O₂ to the cornea with PMMA lenses is the tear pump. Once the O₂ exchange is restricted due to decreased tear pump activity, the cornea becomes cloudy in the central area.
- With RGP lenses, there is a more diffuse swelling as oxygen is able to pass through the lens itself unlike the PMMA lens. The cornea thickens in an anterior-posterior direction but due to the strong Bowman's membrane, the swelling is restricted primarily in the posterior direction. As the corneal limbus is fixed, the distance to be spanned by Descemet's and the posterior stroma is decreased which leads to a vertical buckling of the corneal endothelium and Descemet's membrane, and is seen as striae or folds.
EDEMA (continued)

SYMPTOMS

A. Patient is generally asymptomatic.21
B. Visual acuity is generally unaffected with lens on (RGP > SCL)21
C. Bilateral > unilateral21.24.26
D. Increase in myopia and astigmatism with lens removal.

SIGNS

A. If edema > 15%:
   - displaces regular structural elements26.38
   - steamy cornea24.38
   - decreased visual acuity
   - infiltrates
   - bullous keratopathy38
   - light scattering (glare)11.26.38

EVALUATION: (measure central corneal region)

A. Pachometer (to determine corneal thickness)18
   - normal = 0.506 mm for the center cornea
   - corneal swelling of 5-6% = vertical striae are visible
   - corneal swelling 10-12% = folds are visible
B. Retinoscope - (12°) - see dark lines
C. Ophthalmoscope - (12-15 D) - observe dark lattice-like pattern
   - folds seen against red reflex18
D. Slit Lamp Evaluation
   - retro-illumination to see general disruption of the corneal structures
   - parallelepiped of 1-2 mm with diffuse-direct illumination at a 30° => 45° angle23 temporal to patient’s eye and microscope is 20° nasal18
   - sclerotic scatter with a 1 => 2mm beam over limbus while viewing the central cornea with or without magnification against a black pupil

DIFFERENTIAL DIAGNOSIS

A. Striae:
   - appear as shiny grey-white vertical linear streaks on the parallelepiped posterior curve.4
   - the number of striae is proportional to the degree of corneal swelling
   - striae are usually of uniform diameter lying parallel to each other and more commonly located in central cornea at the posterior stroma level or Descemet’s21.23
EDEMA (continued)

A. **Striae** (continued)
   - usually vertical but may be oblique or horizontal,\(^2^1\) may be branched,\(^2^1\) more sharply defined,\(^2^3\) shorter and more numerous

B. **Folds:**
   - appear as dark lines with specular reflection or white lines with direct illumination\(^1^8\)
   - appear to be a buckling of posterior cornea layer - **Danger Signal!**
   - occur as vertical, horizontal and oblique lines\(^1^8\)
   - horizontal folds appear larger than vertical\(^1^8\)
   - folds yield to digital palpitation\(^2^1\)

C. **Glassy Striae:**
   - vertical lines will not disappear momentarily with digital pressure; with corneal edema they disappear\(^1^8,2^1,2^4,2^6\)
   - only viewed with retro-illumination\(^1^8\)

D. **Nerve Fibers:**
   - more anterior and bifurcated \(^4,2^3\)
   - usually thinner, vertical pattern, shorter and more numerous
   - same whitish color as striae

E. **Central Corneal Clouding (CCC):**
   - greyish circular clouding in pupil area (see following scale):\(^5^0\)

<table>
<thead>
<tr>
<th>Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No CCC</td>
</tr>
<tr>
<td>1</td>
<td>Just detectable corneal haze without distinct borders</td>
</tr>
<tr>
<td>2</td>
<td>Distinct borders, but visible only against a dark pupil background.</td>
</tr>
<tr>
<td></td>
<td>Light density.</td>
</tr>
<tr>
<td>3</td>
<td>Borders very distinct, area of clouding visible against iris and in dimly lighted room</td>
</tr>
</tbody>
</table>

**Note:** Striae always accompany folds but folds will not always accompany striae\(^1^8\)
- Corneal swelling should not exceed 5-6%
Fig. 21  Edema
- Folds
  Grade 3

Fig. 22  Edema
  Folds and Striae
  High Mag. used
  Striae
  Folds
  Grade 4

Fig. 23  Edema
  CCC with RGP lens
  Grade 4
**EDEMA** (continued)

**GRADERS AND TREATMENT**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Edema</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No corneal swelling</td>
<td>No treatment</td>
</tr>
<tr>
<td>1</td>
<td>1% - &lt;5% corneal swelling</td>
<td>Monitor</td>
</tr>
</tbody>
</table>
| 2     | 5% - <10% swelling (Striae) | 1. Reduce wear time\(^{18}\)  
2. Fit with loosen lens\(^{18}\)  
3. Reduce center thickness (C.T.)  
4. Increase Dk/L (change material)\(^{16}\) |
| 3     | 10%-15% thickening (Folds) | As for Grade 2 plus:  
1. Temporarily discontinue CL wear  
2. Flatten base curve\(^{16,22}\)  
3. Smaller O.A.D. |
| 4     | Thickening greater than 15% | Discontinue contact lens wear permanently |

**PROGNOSIS:**

A. Good - following discontinued lens wear.  
B. Acute episode - resolves in 2-3 hours.\(^{4,16,21}\)  
C. Chronic episode - resolves in approximately 1 week.\(^{4,16}\)  
D. May result in stroma thinning.\(^{16}\)  
E. Condition is reversible with discontinuing of lens wear - vertical lines disappear in 2-4 day.\(^{21,24,26}\)
CONDITION - "CONTACT LENS PAPILLARY CONJUNCTIVITIS" (CLPC)

CLPC - is a reversible ocular inflammatory syndrome that is usually (but not exclusively) related to CL wear occurs with SCL > RGP
- papillae located on the upper tarsal conjunctiva
- Histologically - Papillae contain mast cells, eosinophil, basophil, plasma cells, PMN's and lymphocytes in the conjunctiva stroma
- CL induced papillae are not enlarged normal papillae; they are new abnormally large papillae associated with a pathological process
- although a common complication of contact lens wear CLPC is often overlooked/misdiagnosed due to:
  a. lack of awareness by patient of early symptoms of increased mucous/itchy eyes
  b. failure by attending Dr. to routinely evert the upper lid with each visit
- reduced CLPC with RGP as compared to SCL due to:
  a. minimal preservative uptake
  b. reduced adherence of deposits
  c. smaller O.A.D. => reduced contact with lid tissue

Associated with:

1. mechanical irritation of lens edge on lid
2. in conjunction with an immune response.
3. irritation may be a product of the fitting, design, inadequate lens cleaning and/or irregular or deposited lens surfaces

ETIOLOGY

A. Combination of mechanical, immunological and inflammatory mechanisms:

1. protein deposits on the CL become antigens that induce type IV allergy reaction
2. fitting and/or design
3. constant lens edge irritation of the lid combined with an immune response
4. genetic susceptibility to papillary response
5. inadequate lens cleaning
6. mechanical trauma to lid combined with the antigen response constitutes the afferent limb of immune response. The efferent limb involves the accumulation of mast cells, lymphocytes and basophil in the palpebral conjunctiva
CONTACT LENS PAPILLARY CONJUNCTIVITIS - (CLPC) (continued)

SYMPTOMS

A. Increased mucous (stringy & sheets),"mucus in eye on
awakening is first sign"
B. Itchiness (especially with lens removal)
C. Discomfort with lens insertion
D. Lens intolerance
E. Reduced visual acuity (blur)
F. Increased lens slippage
G. Injection
H. Foreign body sensation during CL wear

SIGNS

A. Enlarged papillae
B. Tarsal conjunctiva hyperemia

EVALUATION

Slit Lamp Evaluation
- evert upper lid and observe superior tarsal surface
- direct illumination with maximum illumination
- 3-5 mm wide beam
- low-medium magnification
- diffuse illumination to give best overall impression of lid
surface
- fluorescein stain with cobalt blue filter
- inferior conjunctiva is normal

DIFFERENTIAL DIAGNOSIS

1. Follicles
   - non vascular with no central blood vessel
   - usually in inferior tarsal conjunctiva
   - have pyramidal edges with respect to the tarsal plane
2. Papillae
   - vascular base with a central blood vessel
   - usually in upper tarsal conjunctiva
   - have perpendicular edges with respect to the tarsal plate
   - have white heads that look like scars
3. Allansmith et al. developed a mechanism to predict the
   development of papilla on the upper tarsal conjunctiva.
   The upper tarsal lid was divided in three zones:
   Zone 1 most superior;
   Zone 2 middle tarsal area;
   Zone 3 closest to the lid margin
Fig. 24  CLPC  
Grade 1  
Hyperemia  
Grade 2

Fig. 25  CLPC  
Grade 2  
Note large  
follies at fold  
Hyperemia  
Grade 2- Zone 2  
Hyperemia  
Grade 3 in lateral area

Fig. 26  CLPC  
+ fluorescein  
stain  
Grade 2  
Same patient as in Fig. 25
CONTACT LENS PAPILLARY CONJUNCTIVITS - (CLPC) (continued)

DIFFERENTIAL DIAGNOSIS (continued)

SCL induced CLPC first appear in Zone 3 and progresses to Zone 1, whereas RGP induced CLPC generally appears in Zone 1 first and then progresses to Zone 3.5

4. **CLPC related to RGP**25,26
   a) less numerous papillae than with SCL
   b) tops appear crater-like

5. **CLPC related to SCL**25,26
   a) greater number than with RGP
   b) tops appear flatter or more round

**GRADES AND TREATMENT**
- Adapted from Allansmith et al.'s *Stages of GPC Development*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| 0     | - No papillae on tarsal plate  
       | - No tarsal conjunctiva hyperemia  
       | - Asymptomatic                   | 1. No treatment  
                                      | 2. Continue wearing lens         |
| 125   | - Mild conjunctiva hyperemia  
       | - No edema or increased swelling  
       | - Uniform microscopic papillae  
       | - Mild itch with lens removal25  
       | - Minimal increase of mucus25,37 | 1. Use non-preserved care system26  
                                      | 2. Increase care regime17,34      
                                      | 3. Decrease wear time (EW => DW)17,34  
                                      | 4. Weekly enzyme clean17  
                                      | (with papain)25,29,35             
                                      | 5. Monitor and evert lid with every visit34 |
| 225   | - Tarsal conjunctiva-thickened, edematous with moderate hyperemia  
       | - Papillae x<0.5mm  
       | - Increased lens  
       | - Awareness37  
       | - Moderate itch with CL | As for Grade 1 plus:  
                                      | 1. Discontinue lens wear until symptoms are resolved25,28,34,37  
                                      | 2. Hydrogen peroxide for disinfecting (daily) SCL20,29,32,36 |
**Fig. 27** CLPC
Grade 3
Hyperemia
Grade 2

**Fig. 28** CLPC
Note the GPC and the scarring
Grade 4
Hyperemia
Grade 2
CONTACT LENS PAPILLARY CONJUNCTIVITIS (CLPC) (continued)

GRADES AND TREATMENT (continued)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| 2 (cont.) | - Slight increase in number/elevation of papillae  
- In late stage, can observe papillae with fluorescein & cobalt filter with SLE | 3. No heat as the protein bakes on the lens  
4. No cold system as too irritating to eye20,35  
5. Alter lens fit, edge, design,20,27 material36  
6. Chromylon 4% qid34 with and without lens |
| 3<sup>25</sup> Mod/severe | - Tarsal conjunctiva is more edematous-  
- moderate => severe hyperedema  
- Papillae no longer uniform in size- may vary from 0.5 => < 1.0 mm  
- Pupillae increased in # and elevation<sup>25</sup>  
- Heavy mucus secretion/ observed lens coating  
- Papillae tops stain with fluorescein<sup>25</sup>  
- Increased lens movement<sup>25</sup> | As for Grade 2 plus:  
1. Enzyme clean to 3X/wk  
2. Replace lens q.6-12 months<sup>20,35</sup>  
3. Refit SCL => RGP<sup>17,28</sup>  
4. RGP - smaller OAD  
5. Good lenticular fit  
6. SCL - change to 13.8 mm  
   - CSI - small pore size  
   - smooth surface  
   - round edge  
7. Optichrom (Chromolyn)  
   - 4% indefinitely  
8. Monitor every 2 wks until CLPC is resolved  
9. Topical steroids eg.FML |
| 4<sup>25</sup> | - Severe tarsal conjunctiva hyperedema  
- Papillae are x >1.0mm  
- Tops of papillae flatten => deep clefts between papillae<sup>25</sup>  
- Total CL intolerance<sup>37</sup>  
- Coated lens => decentering<sup>25</sup>  
- Cornea may show:  
  a. cornea punctate stain  
  b. white arcuate infiltrate superior<sup>25</sup> | As for Grade 3 plus:  
1. Discontinue lens wear may be intolerant to future lens wear |
CONTACT LENS PAPILLARY CONJUNCTIVITIS - (CLPC) (continued)

PREVENTION

A. Educate patients of symptoms of CLPC. Itching, increased mucus or both are not normal to CL wear.20

PROGNOSIS

A. CLPC may persist for weeks or months but eventually will flatten out and leave a flat discoid scar.17
B. There is a good prognosis gained with the most simple changes such as:
   1. temporary discontinuance of CL.
   2. increased care regimes.
   3. use of non-preserved solutions for the control of CLPC during continued CL wear.34
   4. regular replacement of CL.
EPITHELIAL STAINING - is a common occurrence among contact lens wearers. Corneal staining can occur from a variety of sources and have been grouped into the following broad categories according to cause:

1. Mechanical/Trauma
2. Chemical Toxicity
3. Physiological
4. Desiccation

Staining occurs when fluorescein fills the gaps in damaged or displaced epithelium. Fluid is able to enter the damaged cells.

Staining indicates:

a. damaged epithelial cells
b. missing epithelial cells
c. breakdown of the epithelial barrier resulting in stromal edema

Corneal healing time:

- a. mild (superficial) => 12-24 hours
- c. moderate => 24-48 hours
- d. severe (deep) => 3-14 days

EVALUATION

A. Slit Lamp Evaluation
   - with fluorescein stain using Kodak Wratten #12 or #15,
   - Tiffen #2 yellow photographic filter and cobalt blue
   - Kodak #47 or #47A filter over white light source
   - diffuse direct illumination - low to medium magnification (10-20X)
   - parallelepiped - low to high magnification (10-45X)

B. Fluorescein with Burton Lamp

DIFFERENTIAL DIAGNOSIS OF EPITHELIAL STAINING

Observe:

1. extent, depth, location
2. recurrence with old lens/new lens
3. appearance of stain - symmetrical, circular, linear, punctate, circumcorneal or arcuate
4. time of onset - immediate or delayed
5. condition of lids/lashes (e.g. marginal staph bleph.)
6. lens material, design, condition (jelly bumps, defects, edges, etc.)
7. association with insertion and removal technique
8. lens fit and relationship to staining (movement, bearing areas)
9. lid position
1. **EPITHELIAL STAINING - MECHANICAL/TRAUMA:** Damage to the soft pliable corneal epithelial tissue can result in corneal abrasion ranging from superficial to deep and can occur from wearing both RGP's and SCL's. Superficial abrasions generally have little surrounding epithelial edema unlike deep abrasions. The superficial injury involves the external one to three layers of epithelial cells. Deep abrasion causes a full thickness of the epithelium to be lost over a small or large area.

Associated with:

1. contact lens wear
2. poor insertion and removal techniques

**ETIOLOGY**

A. **Specific Etiology**

1. lens defect or poor lens finish
2. SCL posterior surface defects due to jelly bumps embedded in the lens
3. foreign body - airborne material becomes trapped under the lens - seen as:
   a. thin spiral tracks (snail tracks)
   b. linear streaks ending in indented abrasion
   c. usually vertically linear in appearance (RGP > SCL)
4. poor insertion or removal of a contact lens e.g:
   fingernail gouges with RGP's - visible as an arcuate gouge in superior or inferior corneal epithelium.
   Finger print inferior para-central cornea (SCL's)
5. mascara brush abrasion - multiple fine linear superficial abrasions usually vertical
6. damaged lens, eg:
   a. nicks, tears, rough edges
   b. poorly polished surface
7. tight fitting lens. Tight adherence and distortion of the epithelial cells at lens bearing area (e.g. RGP)
   a. seen as circumcorneal dent that disappears with lens removal
   b. increased with aphakic, bandage EW lens or tight SCL; signs and symptoms may appear after 1-2 days wear and appear as circumcorneal punctate stain
8. trapped air bubbles - seen as discrete small round indents, but do not break through corneal epithelium if lens is removed immediately
EPITHELIAL STAINING / MECHANICAL TRAUMA

Fig. 29 Vertical Abrasion

Fig 30 Linear abrasion due to foreign body

Fig 31 D.W. Bound lens (lens is in place)
EPITHELIAL STAINING MECHANICAL/TRAUMA (continued)

SYMPTOMS

A. Asymptomatic - Possibly due to the corneal threshold injury being lower than that of corneal sensitivity

B. Symptoms
   1. Foreign body sensation
   2. Blepharospasm
   3. Lacrimation
   4. Mild discomfort $\Rightarrow$ severe pain
   5. Reduced vision

SIGNS

A. Signs:
   1. Injection
   2. Corneal edema
   3. Staining with rose bengal or fluorescein.

GRADES AND TREATMENT

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Asymptomatic/ no staining</td>
<td>No treatment</td>
</tr>
</tbody>
</table>
| 1     | None to mild discomfort  | 1. Evert upper lid: remove foreign body if present via rinse, manual and/or surgical removal  
   2. Discontinue lens wear until staining has resolved  
   3. Check lens for defect/ tear  
   4. Blend/modify lens if rough edges are present  
   5. Reorder new lens if defect is present
| 2     | Moderate discomfort + staining | As for Grade 1 plus:  
   1. Refit with:  
      a. flatter B.C.(looser fit)$^{11}$  
      b. smaller OAD$^{11}$ (e.g. RGP)  
      c. different lens material$^{41}$  
      d. re-teach,correct insertion/removal techniques

30
**Fig. 32** E.W. bound lens. No lens in place - imprint.

**Fig. 33** Rose bengal staining due to low riding RGP.

**Fig. 34** Dimple veil staining due to aerosol sterile saline solution.
<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 (cont.)</td>
<td></td>
<td>2. Advise regarding applying mascara and possibility of corneal abrasion</td>
</tr>
<tr>
<td>3</td>
<td>Moderate to severe discomfort; + staining</td>
<td>As for Grade 2 plus:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. Discontinue lens wear until eye is quiet and no stain observed for several days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Antibiotics (gtts/ung) broad spectrum e.g:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. tobramycin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>b. Gentamycin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Lubricants</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Observe for signs/symptoms of recurrent corneal erosion (RCE):</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. blurred vision 2-3 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>b. increased pain upon wakening in a.m.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Oral analgesic prn</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. RTC 24 hours</td>
</tr>
<tr>
<td>4</td>
<td>Severe discomfort; + staining</td>
<td>As for Grade 3 plus:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. RTC, 24 hrs, 3 days, 1 week post injury/deep abrasion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Patching contraindicated unless adult patient with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a. increased lacrimation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>b. increase blepharospasm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>c. large defect/abrasion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Usually no steroids</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. May cycloplege if large abrasion =&gt; decrease risk of possible uveal response</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Discontinue lens wear permanently</td>
</tr>
</tbody>
</table>
EPITHELIAL STAINING / MECHANICAL TRAUMA (continued)

**Fig. 35** Back surface debris => corneal staining Grade 3

**Fig. 36** Scratched and Protein deposited lens.
EPITHELIAL STAINING - MECHANICAL /TRAUMA (continued)

PREVENTION:

A. Educate patient re: symptoms.
B. Immediate lens removal for the previously described signs and symptoms; seek professional care as necessary.\textsuperscript{41}
C. Good insertion and removal techniques (short nails).
D. Good cleaning regimen to prevent build up of protein or formation of jelly bumps (SCL).\textsuperscript{41}
E. Careful application of mascara.
F. Proper lens handling (prevent damage to the lens).\textsuperscript{41}
G. RTC - with continued pain.
    - with blurred vision 2-3 days.\textsuperscript{47}
H. Evaluate cornea prior to CL fit.\textsuperscript{41}

PROGNOSIS:

A. Good if staining resolves quickly.
B. Moderate to poor if abrasion is deep and staining continues.
C. Risk of recurrent corneal erosion.\textsuperscript{47}
D. Healing power greater with younger patients than older.\textsuperscript{47}
2. EPITHELIAL STAINING - TOXICITY: Toxicity is a form of chemical trauma that causes a diffuse pattern keratopathy.\textsuperscript{11,41} The main chemical involved in most CL related chemical toxicity is benzalkonium chloride (BAK)

Associated with

1. use of contact lens solutions
2. use of antibiotics
3. poor technique in cleaning regime

ETIOLOGY

A. Contributing Factors
   1. improperly neutralized hydrogen peroxide (H\textsubscript{2}O\textsubscript{2})
   2. primarily (BAK) found in CL solutions and eye lubricants\textsuperscript{41} but other cationic preservatives such as chlorhexidine gluconate (CHG) and alkyltriethanol ammonium chloride (ATAC)
   3. SCL > RGP
   4. topical antibiotics\textsuperscript{46} - e.g. sulfa, neomycin
   5. SCL stored in cationic preserved solution:
      a) becomes impregnated with the solution and acts as a reservoir so when the lens is placed on the eye, it results in prolonged chemical contact\textsuperscript{43}
      b) binds to protein deposits on the lens as well as to the lens material itself
   6. failure to rinse or neutralize CL prior to insertion\textsuperscript{11}
   7. increased protein on lens => increased binding of chemicals to CL\textsuperscript{11}

SYMPTOMS

A. Sting/burning with lens insertion\textsuperscript{11}
B. Bilateral Conjunctival hyperemia\textsuperscript{11,43}
C. Foreign body sensation, lacrimation\textsuperscript{43}

SIGNS

A. Superficial punctate keratitis;\textsuperscript{11}
   1. central cornea - mild keratitis
   2. whole cornea - moderate keratitis
   3. confluent - severe keratitis
B. Onset - few hours => weeks => several months\textsuperscript{11}
C. Pseudodendritic epithelial lesions (usually bilateral)\textsuperscript{41}

33
EPITHELIAL STAINING / TOXICITY

Fig. 37 BAK toxicity
Grade 3

Fig. 38 BAK toxicity
Grade 4

Fig. 39 toxicity
SPK
RGP in place
Grade 4 (area)
EPITHELIAL STAINING - TOXICITY (continued)

GRADES AND TREATMENT

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal conjunctiva with no corneal staining</td>
<td>No treatment</td>
</tr>
<tr>
<td>1</td>
<td>- Mild hyperemia (bilateral), - Stinging with lens insertion</td>
<td>1. Monitor 2. Rinse lens well prior to lens insertion</td>
</tr>
<tr>
<td>2</td>
<td>- Sting/burn with lens insertion - Superficial punctate keratitis in central cornea area - Mild chemosis</td>
<td>1. Identify and remove toxin 2. Rinse lens well prior to lens insertion 3. Change to preservative free solutions</td>
</tr>
<tr>
<td>3</td>
<td>- Follicles - Foreign body sensation /lacrimation - Possible pseudo dendrites - Superficial keratitis</td>
<td>As for Grade 2 plus: 1. Discontinue lens wear until stain resolves 2. New uncontaminated lens (SCL)</td>
</tr>
<tr>
<td>4</td>
<td>Confluent superficial keratitis</td>
<td>As for Grade 3 plus: 1. Topical antibiotics</td>
</tr>
</tbody>
</table>

PREVENTION:

A. Educate patient re: rinsing SCL well before insertion.  
B. Educate to early signs and symptoms of toxicity.  
C. Prescribe preservative free care system.

PROGNOSIS:

A. Good - fast recovery following removal of causative agents.  
B. If lens worn during recovery period - slower recovery.  
C. Grade 1-2 will resolve in few days.
EPITHELIAL STAINING / PHYSIOLOGICAL

**Fig. 40** Exposure 3 and 9 o'clock staining
Grade 2 Inferior
Grade 3 Nasal
Grade 3 Overall

**Fig. 41** Exposure rose bengal staining at 3 o'clock
3. **EPITHELIAL STAINING - PHYSIOLOGICAL:** The anterior surface of the corneal epithelium becomes disrupted due to chronic metabolic stress\(^4,11,40\)

- Two examples of physiological trauma leading to epithelial stain are:
  1. **Epithelial Microcysts** (see section titled "Epithelial Microcysts")
  2. **Soft lens anterior chronic hypoxia (SLACH)** syndrome - hallmark of this syndrome is microcystic corneal edema\(^48\)
    - Associated with:
      a. Daily wear soft contact lens
      b. Corneal hypoxia\(^11,48\)

**SYMPTOMS**

A. Earliest sign - blurred vision\(^48\)
B. Mild foreign body sensation with lens on
C. Tearing, moderate to severe pain upon lens removal and photophobia
D. Bilateral/asymmetrical

**SIGNS**

A. Circular epithelial defect with surrounding microcystic edema\(^48\)
B. Fluorescein staining seen as irregular shaped defects\(^4\)

**GRADING** - none

**TREATMENT**

A. Discontinue lens wear until there is no longer any staining\(^41\) and epithelium shows uniform wetting.
B. Dilate\(^48\) (to decrease risk of iritis occurring).
C. Prophylactic antibiotics.
D. Analgesics - oral.
E. Pressure patch is contraindicated\(^48\) - due to decreased oxygen to the cornea when it is already hypoxic.
F. Refit with new lens with increased Dk/L.\(^40\)
G. Refit with RGP.\(^40\)

**PROGNOSIS:**

A. Fast recovery with immediate and appropriate treatment.
B. Early diagnosis will:\(^48\)
   1. Decrease length of time patient experiences discomfort.
   2. Decrease risk of corneal infection.
   3. Recurrence of SLACH is common even when refit with high water content lenses.\(^48\)
4. EPITHELIAL STAINING - DESICCATION: Desiccation is the drying out of the corneal epithelium cells leading to the disruption and or death of these cells. The focal depletion of the tear layer causes corneal epithelial cell desiccation as these cells are no longer protected by the tears. This allows fluorescein to enter the damaged epithelial cells and penetrate deeper into the stromal cells allowing observation of punctate staining.

Associated with:

1. RGP wear
2. dehydration with SCL
3. dry eye
4. exposure

SYMPTOMS

A. Asymptomatic
B. Mild discomfort, burning, stinging
C. Moderate conjunctival injection - associated with 3 and 9 o’clock staining

SIGNS

A. If condition persists:
   1. pseudopterygia
   2. limbal inflammation
B. Mild superficial => coalesced stain with fluorescein in specific patterns as indicated below.

RGP:

A. Large, base-in triangles diffuse stain due to the incomplete wetting of the cornea adjacent to the lens edge, nasal and temporal area
B. Characteristic in form and known as "3 and 9 o’clock" stain
C. Stain may vary from mild diffuse punctate spots to coalesced lesions
D. Occurs with PMMA and other RGP lenses including silicone
E. May progress to hyperplasia, infiltration, vascularization (vascularized limbal keratitis (VLK))

SCL:

A. Commonly associated with thin, high water contact lens as the lens dries out, it pulls the fluid out of the cornea to osmotically maintain a balance
B. Seen almost 100% of the time with ultra thin, high water contact lens with moderate minus Rx
EPITHELIAL STAINING - DESSICATION (continued)

SIGNS (continued)

SCL (continued)

C. Seen with other designs of contact lens although less frequently\(^\text{11}\)
   1. thin, high water content, parallel sided contact lens
   2. ultra thin, low water contact lens
   3. thick, high water contact lens
D. Characteristically, the epithelial erosions have a course punctate appearance and may be referred to as "flake-like", or "crumb-like"\(^\text{11}\). They are usually localized and appear to be mild and superficial no with fluorescein staining, but there are cases where they have penetrated the full thickness of the epithelium and stroma and staining is seen\(^\text{11}\)
E. Staining may appear anywhere in central or para-central areas of the cornea\(^\text{11}\)

DRY EYE

A. Commonly seen in patients with a poor tear layer or decreased TBUT
B. May be seen in patients with collagen disease such as rheumatoid arthritis, arthritis, Sjorgen syndrome
C. Seen as generalized superficial punctate staining, usually inferior\(^\text{11}\)

EXPOSURE

A. Commonly due to incomplete blink with both SCL and RGP
B. The lower part of the lens dries out the most frequently even when there is adequate blinking
C. Seen as coarse punctate staining in an arcuate or band staining across the inferior cornea (lower 1/3 of the cornea)

GRADES AND TREATMENT

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No staining</td>
<td>No treatment</td>
</tr>
<tr>
<td>1</td>
<td>Decreased TBUT x &lt; 10 seconds</td>
<td>1. Monitor</td>
</tr>
<tr>
<td></td>
<td>- History of collagen disease</td>
<td>2. Lubricant (Dry Eyes)</td>
</tr>
</tbody>
</table>
EPITHELIAL STAINING / DESSICATION

Fig. 42  SPK Dehydration
Grade 2
(central)

Fig. 43  Conjunctival staining
Grade 4
<table>
<thead>
<tr>
<th>Grade</th>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| 2 (cont.) | Mild punctate stain superficial epithelium | 1. Discontinue lens wear until there is no staining$^{41,4}$=> resume wear  
2. Artificial tears  
3. Modify edge of RGP$^{11,41}$  
4. Lenticulate high minus$^4$  
5. Blend to increase tear exchange  
6. Educate patient regarding full blink  
7. Lubricants$^{41}$ |
| 3 | Moderate, full thickness punctate stain  
- Mild discomfort  
- Moderate conjunctival infection  
- Grade 1-2 limbal infection | As for Grade 2 plus:  
1. Refit with smaller OAD  
2. RGP lens for 3 and 9 o'clock stain$^{11}$  
3. Discontinue ultra thin, CL and refit with thicker, high Dk/L lens  
4. Refit with low water content lens (LWC) thinner edge (RGP) |
| 4 | Severe punctate stain => coalesced  
=> +/- ulcer$^4$  
- Moderate discomfort  
+/- pseudopterygium  
- Grade 2-3+ limbal infection with RGP | As for Grade 3 plus  
1. Discontinue CL wear until staining is resolved.$^{41}$ Then resume wear and refit with:  
   a. thinner edges (RGP)  
   b. smaller OAD (RGP)  
   c. thicker, high Dk/L (SCL)  
   d. LWC (SCL)  
2. Artificial tears/ Lubricants  
3. Educate pt. re: full blink$^4$  
4. If ulcer refer out  
5. Do culture and sensitivity  
6. Prophylactic antibiotics monitor daily until ulcer resolves  
7. Permanently discontinue. CL wear |
EPITHELIAL STAINING DESSICATION (continued)

PREVENTION:

A. Routinely stain patients who wear contact lens at each visit/a.m. and p.m. visits regardless of the type of lens they are wearing.

B. Prior to fitting CL - assess:
   1. tear layer\[41 \]
   2. TBUT\[41 \]
   3. blink rate\[41 \]
   4. lid action\[41 \]
   \-----to ensure that all are within normal ranges

C. Also Assess:
   1. history of collagen diseases (arthritis)
   2. history of dry eye, allergies, current medications
   3. previous successful/unsuccesful contact lens wear

A CAREFUL ASSESSMENT OF PATIENTS OCULAR AND SYSTEMIC HEALTH MAY ALLOW YOU TO RULE OUT UNSUCCESSFUL CL WEARERS PRIOR TO PRESCRIPTION AND SUBSEQUENT DIFFICULTIES.

PROGNOSIS:

A. With RGP:
   1. Good with refit with thinner edges, moderate edge lift and smaller OAD.

B. With SCL:
   1. Good with thicker or low water content (LWC) lenses.

C. With Dry Eye:
   1. Fair.
   2. Slow recovery.
   3. May reoccur with high water contact (HWC), low water content (LWC) or no lenses.

D. With Exposure:
   1. Fair.
   2. Slow recovery.
   3. Teach patient to make frequent full blinks lubricants/artificial tears.
BIBLIOGRAPHY


BIBLIOGRAPHY (continued)


BIBLIOGRAPHY (continued)


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