CONTACT LENS COMPLICATIONS

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TABLE OF CONTENTS

As of 1984, there were approximately 16 million contact lens wearers in the United States. By 1986, there were over 20 million, and the number is still growing such that projections reach 45-90 million wearers by the year 2000. Along with the growing population of contact lens wearers comes a growing number of ocular complications secondary to contact lens wear. Therefore, differential diagnosis and management of contact-lens-related pathology is as important to successful contact lens practice as knowledge of proper fitting techniques. Although a well fitting lens is less likely to cause complications than a poorly fitting lens, there are, unfortunately, many other variables involved in the etiologies of contact-lens-induced problems. In fact, the only other factor in which the optometrist plays a role is in the responsibility of patient education regarding proper hygiene and habits of the contact lens care regimen. All the other variables depend solely upon the patient, his compliance with the care regimen, and the idiosyncracies of his own body and immune system. We all know how dependable many patients are when it comes to compliance, and we also know how varied indiviual reactions to solutions, wear schedule, and contact lens wear itself can be. So, contact lens complications are going to occur, and we must be able to properly diagnose and manage them. What follows, then, is a guide intended to aid the clinician in recognizing which complication has presented itself (along with the patient) and instigating the proper treatment.

I have divided the complications into two groups: major and minor complications. Major complications are considered threats to vision and include infectious corneal ulcers, and, rarely, corneal vascularization,

and superior limbic keratoconjunctivitis. Minor complications are non-vision threatening, although they may lead to contact lens intolerance. They include corneal staining, corneal edema, giant papillary conjunctivitis, SLACH syndrome, and pseudodendritic lesions, to name a few. Included at the end are systemic and ocular pathologies to consider before fitting with contact lenses or resuming lens wear.

BACTERIAL CORNEAL ULCERS

SYMPTOMS: The patient will come to your office in great pain, complaining of photophobia, tearing, and foreign body sensation. The eye will be extremely hyperemic, and there may be mucopurulent discharge in the eye at examination.

SIGNS: There will be an epithelial defect somewhere on the cornea, often with surrounding or underlying infiltration. The defect will be excavated and may have a necrotic base. An anterior chamber reaction will probably be present.

ETIOLOGY: The most common pathogens are Pseudomonas, Staph, and Strept species. There is often some history of improper disinfection methods, deposited lenses, overwear of the lenses, etc. The common pathogenic organisms cannot invade the cornea unless the epithelium has been broken, but there are many opportunities for that to occur with lens wear, especially if the lenses are not properly cared for or if the cornea is overstressed from extended wear. Exact percentages are difficult to determine, but ulcers seem to be more common with soft lenses than with hard lenses, and they are more likely to occur with extended wear than with daily wear.

TREATMENT: Intensive antibiotic therapy should be started immediately. Pseudomonas can penetrate the cornea within 48 hours. An aminoglycoside such as Tobramycin or fortified Gentamycin is recommended every hour, and Polysporin ointment should be used at night. With some ulc-

ers, you will want patients to instill drops throughout the night, especially if the ulcer is centrally located. You should also have a culture and sensitivity study done. The antibiotics may need to be changed, based on the sensitivity study. Taper the antibiotics when reepithelialization has occurred.

PROGNOSIS: If Bowman's layer has been damaged, scarring will occur. In some cases, scarring will occur even if you have done "all the right things." If the ulcer is centrally located, vision loss can occur. It would be prudent to refer this patient to a corneal specialist. Contact lens wear may be resumed when the eye has quieted completely, although you may want to consider switching an extended wear patient to daily wear.

OCULAR INFECTIONS

It is beyond the scope of this paper to give a detailed differential diagnosis of ocular infections, but the following are guidelines to follow in making your diagnosis and management decisions. With any infection, contact lens wear should be discontinued until the eye is quiet. One thing to remember is that any contact lens complication can become secondarily infected. This could alter the prognosis, and steps must be taken to prevent that from occurring.

BACTERIAL INFECTION: The most common cause of both chronic and acute bacterial infection is staph aureus. Patients will complain of a grittiness or foreign body sensation. The lids will often be matted shut in the morning with a mucopurulent discharge. The infection usually starts in one eye, then spreads to the other eye in a few days. Treat both eyes with antibiotic drops (Gentamycin) four times a day and antibiotic ointment (Polysporin) at night. Continue treatment for several days after the eye has whitened.

VIRAL INFECTION (NON-HERPETIC): A patient with a viral conjunctivitis will often have a recent history of an upper respiratory tract infection. Signs of viral inflammation include swollen lids, watery discharge, follicles, and preauricular lymphadenopathy. Adenoviral infections, such as epidemic keratoconjunctivitis, can take several weeks to resolve. Punctate keratitis and subepithelial infiltrates are also present in EKC infection. The only treatment for these viral

infections is supportive. Cold compresses, decongestants, and cycloplegics will help to relieve symptoms. A broad sprectrum antibiotic (Gentamycin) should be used four times a day while there is a keratitis in order to prevent secondary infection.

HERPES SIMPLEX: The Herpes Simplex Type I virus causes cold sores and fever blisters, as well as almost all cases of ocular Herpes. By age 15, over 90% of the people have been exposed to the virus, and it harbors in the sensory ganglion of the trigeminal nerve and autonomic ganglia. It remains latent until the body's immune system is weakened, and flare-ups usually occur in times of physical or emotional stress. The recurrent ocular infection is unilateral and usually causes a characteristic dendritic ulcer, although atypical corneal defects can occur. The lesion will stain with fluorescein and rose bengal, and the branches will usually have rounded end bulbs. The cornea will have reduced sensitivity in the affected eye. Steroids are absolutely contraindicated, and they should never be used with any epithelial defect until you are sure that you are not dealing with Herpes. The antivirals IDU and Trifluridine are the drugs of choice. The drops should be used every 1-2 hours during the day and every 2-4 hours during the night until the epithelium is intact, then every 2-4 hours during the day for 7 more days. Treatment should not exceed 21 days. When steroids are inadvertantly used, or if the patient delays seeking treatment, the inflammation can reach the stroma. This is a threat to vision, as stromal infiltration, vascularization, and disciform edema can occur. The patient should then be referred to a corneal specialist.

CHLAMYDIAL INFECTION: This is usually unilateral and begins with a follicular response. It can mimic viral or chronic bacterial conjunctivitis, and if there is a keratitis, it will usually be in the upper half of the cornea. It often lasts 2-3 months and does not respond well to topical antibiotic or supportive therapy. This is a sexually transmitted infection, and patients will often have a concurrent urinary-genital tract infection. This must be treated systemically as well as topically, because chlamydia causes pelvic inflammatory disease in women, which is one of the leading causes of infertility in the United States. Therapy should include tetracycline or erythromycin ointment applied twice a day, along with oral tetracycline, 250mg four times a day for two weeks. Pregnant and lactating women should take oral erythromycin. Recommend that sexual partners seek care.

CORNEAL VASCULARIZATION

SYMPTOMS: Corneal vascularization is in itself assymptomatic, however, there may be symptoms associated with the particular inflammation causing the blood vessel growth.

SIGNS: The corneal vessels can either be superficial (in the anterior stroma) or deep (in the middle to posterior stroma). Depending on the cause of the vascularization, there can be other associated signs, such as corneal infiltrates, corneal scarring, and conjunctival injection.

ETIOLOGY: Corneal vascularization is caused by inflammation, although not all inflammations will lead to vascularization. The inflammation can be caused by hypoxia, infection, or toxic reaction. The exact mechanism of vascularization is not known. If the vascularization is 360 degrees circumlimbal with the vessels encroaching only about 1mm, it is probably being caused by hypoxia secondary to contact lens wear. This is probably the most common vascularization seen with contact lens wear, and the vessels are superficial. If the vessels are more sectored with greater than 1mm extension into the cornea, other types of inflammation, such as infection, are more likely causes.

TREATMENT: Treat the underlying inflammation. In the case of contact-lens-induced hypoxia, it is recommended that you try a lens with higher oxygen transmissibility. Other tactics include fitting with a flatter lens or decreasing wearing time.

PROGNOSIS: If the vessels seem confined to 1mm encroachment, there is no threat to vision. At 1mm from the limbus, the structure of the cornea changes, with the collagen fibrils becoming more tightly packed, and the new vessels usually do not cross that point. Vascularization confined to 1mm from the limbus is much less likely to progress to the visual axis. Any vessel greater than 1mm from the limbus is a potential threat to vision. Whenever the vessels cross the visual axis, vision is decreased, and penetrating keratoplasty is necessary to restore vision.

NOTE: Corneal vascularization is also seen in other contact lens complications: superior limbic keratoconjunctivitis, corneal ulcers and infection, 3 and 9 o'clock staining and circinate pattern interstitial keratitis.

CONTACT-LENS INDUCED SUPERIOR LIMBIC KERATOCONJUNCTIVITIS

SYMPTOMS: The patient may present with complaints of chronic redness, burning or foreign body sensation, and mucus-like discharge. Occasionally, there may be a decrease in vision, and patients usually report a gradually increasing intolerance to lens wear.

SIGNS: Signs are usually limited to the superior cornea, limbus, and conjunctiva. There is bulbar conjunctival injection which is most intense at the superior limbal area and an epithelial hypertrophy of the superior limbus, giving it a gelatinous appearance. There is also a punctate keratitis with a fine, swirling, subepithelial opacification, often in the shape of a "V". Filaments may develop on the abnormal epithelium. An iritis may also develop. In very severe cases, a coarse epithelial keratits can develop with diffuse corneal scarring and vascularization leading to vision loss and possibly requiring penetrating keratoplasty for restoration of vision. Fortunately, this severe type of contact-lens-induced SLK is rare and usually occurs only when the patient refuses to discontinue lens wear.

ETIOLOGY: This condition has been reported with both daily wear and extended wear soft contact lenses. There are several different theories of the cause of this type of SLK. Because many of these patients used thimerosal-containing solutions, some researchers suggested that it was a type of thimerosal hypersensitivity reaction. However, cases have been reported in which patients used nonpreserved

solutions. Other theories propose relative hypoxia of the area under the upper lid, mechanical trauma caused by the edge of the lens, and deposits on the lens as possible causes. Most likely, it is really some combination of those theories.

TREATMENT: Discontinue lens wear. Cold compresses, artificial tears, oral analgesics, and cycloplegics provide symptomatic relief. Topical steroids may be used for 7-14 days in an attempt to relieve some of the inflammation. Antibiotics are not indicated unless a secondary infection develops. Stubborn cases may require debridement of the abnormal corneal epithelium (after which, you would pressure patch; see corneal abrasion, p 12) or silver nitrate cautery of the irregular conjunctival tissue. In the severe cases with central corneal scarring and vascularization, penetrating keratoplasty is necessary to restore vision.

PROGNOSIS: The symptoms of SLK usually take several weeks to resolve, and the signs may still be present for several months after removal of the lenses. Researchers disagree over the return to soft contact lens wear. Some say that it is possible to resume soft lens wear as long as the patient uses a care system without thimerosal. Others believe that SLK is caused by something more than a thimerosal hypersensitivity and that the risk of recurrence is too great. They recommend a switch to rigid gas permeable lenses.

CORNEAL ABRASION

SYMPTOMS: Patients will report pain, photophobia, and excessive tearing.

SIGNS: By definition, there will be an epithelial defect somewhere on the cornea, of any conceivable size or shape. There will also be conjunctival injection and, possibly, an associated iritis, dependent upon the severity of the abrasion. An overwear abrasion is usually centrally located and relatively symmetrical.

ETIOLOGY: Most often, there will be a history of trauma or a foreign body episode. Some patients cause abrasion with faulty insertion or removal techniques. Occasionally patients will self-abrade themselves by rubbing their eyes when they feel a foreign body sensation. Overwear abrasions are not as common anymore with the diminished use of PMMA lenses. The epithelial disruption in these cases is caused by the increase in corneal hypoxia and edema when lenses are worn more than a few hours over normal wear time.

TREATMENT: Instill a cycloplegic (Homatropine 5%) and an antibiotic ointment (Polysporin or Gentamycin) and then pressure patch the patient. The patch should stay on the eye 18-24 hours, and then the patient must be reexamined. If the new epithelium has been laid down, discontinue patching and prescribe antibiotic drops until the eye is quiet. If reepithelialization has not occurred, continue pressure patching until it does.

PROGNOSIS: In most cases, reepithelialization occurs overnight. In rare cases if Bowman's layer has been damaged, scarring will occur and could cause visual loss if on the visual axis. But in the vast majority of cases, the abrasion heals completely with no permanent damage. Contact lens wear can be resumed when the eye is quiet. The patient should be warned of the possibility of a recurrent erosion if the abrasion was caused by trauma or foreign body.

HYPOXIC CORNEAL EDEMA

SYMPTOMS: When the cornea is edematous, vision is blurred, especially after lens removal. In severe cases, patients will report halos around lights.

SIGNS: The cornea will lose its sharp, clear, appearance. The keratometer mires will be distorted. Striae occur when there is greater than 5% edema and appear as fine, white, nearly vertical lines in the posterior stroma. At 10% edema, endothelial folds appear as dark-looking grooves. Stromal transparency is lost when there is 20-25% edema.

ETIOLOGY: If the cornea becomes hypoxic, the epithelium begins to respire anaerobically, and lactic acid is produced, making the cornea hyperosmotic to the aqueous. This causes an osmotic force pulling water into the cornea, and the force becomes too strong for the endothelial pump to counter. "Baseline edema," the amount the cornea swells during sleep, is 4%.

TREATMENT: Edema should not be allowed to be greater than 5%, in other words, striae should not be visible, except with extended wear patients within two hours of awakening. Ways to decrease hypoxia are to use a lens with a higher Dk, decrease lens thickness, use a smaller lens, or fit with flatter base curves or peripheral curves. Other suggestions would be to switch from soft to rigid gas permeable lenses or to decrease wearing time.

PROGNOSIS: Long term chronic corneal edema can damage corneal integrity by allowing vascularization and leading to dysfunction of the endotheluim. Edema should not be tolerated in a contact lens patient.

CORNEAL STAINING

SYMPTOMS: There are many different causes and patterns of corneal staining with contact lenses. This is a quick overview of some of them.

STAINING WITH RIGID LENSES

3 and 9 o'clock staining: This is punctate staining on the inferotemporal and inferonasal margins of the cornea. It is caused by improper wiping of the upper lid over these areas due to a thick lens edge. Mild amounts can be tolerated, but more staining can lead to scarring and vascularization of the area. Dellen and pseudopterygia can also develop. Treatment includes re-edging and polishing the lens, changing lens parameters to thin the edge, using a smaller lens, and using lubricating drops.

Overwear abrasion: See Corneal abrasion, p. 12.

Dimple veiling: Air bubbles get under the lens because it has too much edge lift. There is no break in the epithelium.

STAINING WITH SOFT LENSES

Dehydration: This occurs with high water content lenses. Water evaporating from the lens pulls water into the lens from the tear layer. As the tears thin, dry areas develop beneath the lens and cause punctate staining.

STAINING WITH BOTH TYPES OF LENSES

Foreign body staining: This usually appears as straight tracks. It can be confused with a dendrite.

Poor lens fit: A flat lens will cause a central abrasion or punctate staining, and a steep lens will cause curved areas of staining in the periphery.

Deposited lenses: Large deposits can cause punctate staining.

Arcuate staining: With rigid lenses, this is caused by poorly blended junctions. With soft lenses, it is a sign that the cornea is not always covered by the lens in that area.

There are also distinctive patterns of corneal staining in some ocular disease states. See Fig. 1.

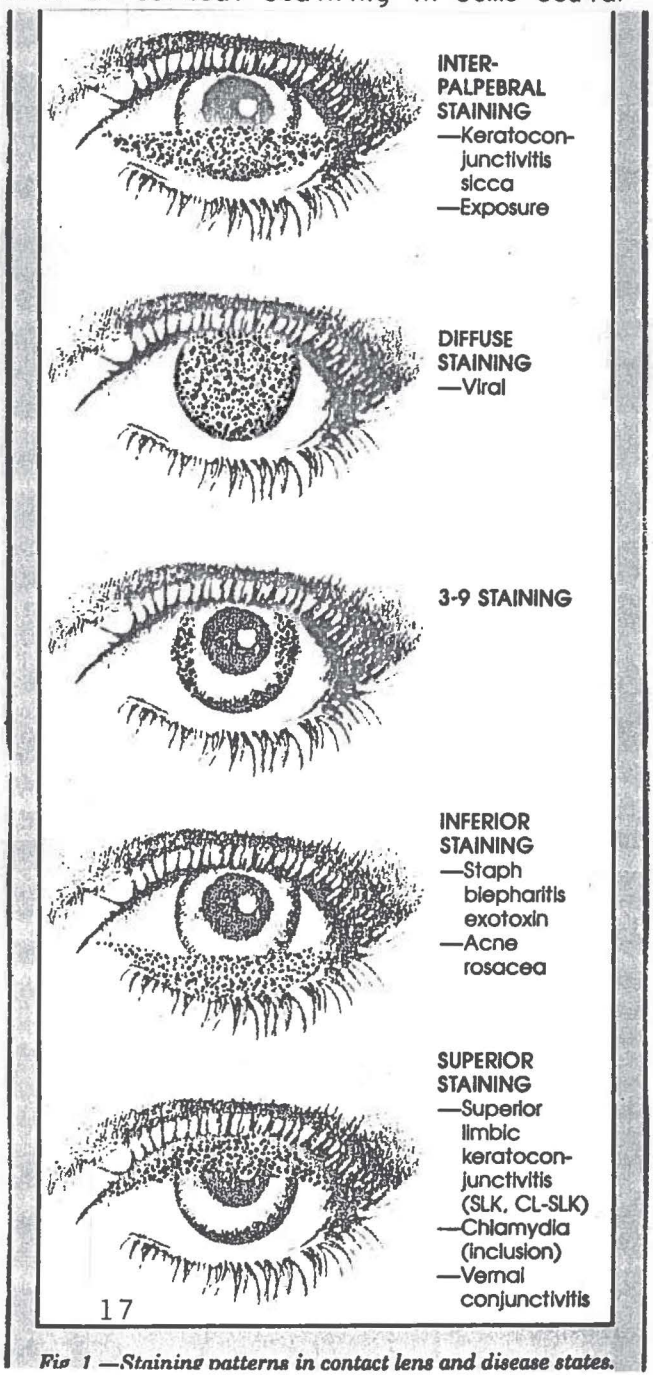

Fig. 1.—Staining patterns in contact lens and disease states.

CORNEAL INFILTRATES

SYMPTOMS: An infiltrate in itself is assymptomatic, but as it is a sign of an inflammation, the patient will experience the symptoms associated with that particular inflammation, such as hyperemia, discomfort, or photophobia.

SIGNS: Infiltrates are white or light grey dots found in the subepithelium or anterior stroma of the cornea. They can appear as a circumlimbal band, or in clusters anywhere on the cornea. There will be other ocular sugns that are dependent upon the particular inflammation that is causing the infiltrates.

ETIOLOGY: Infiltrates are white blood cells that migrate into the cornea from the limbal blood vessels. They can occur as an immune response to an infection or as a delayed hypersensitivity response to an antigen.

TREATMENT: Discontinue contact lens wear. Treat the underlying inflammation or infection, or remove the antigen causing the hypersensitivity response. Steroids can reduce the infiltrative response, but they prolong the healing time, and, after the therapy is discontinued, the infiltrates often return. Steroids are only recommended in cases where centrally located infiltrates are severely obstructing vision. Contact lens wear should not be resumed until the infiltrates have resolved.

PROGNOSIS: The prognosis is dependent upon the cause of the infiltrates. Most resolve with no loss of vision.

NOTE: Infiltrates are found in: infections, infectious ulcers, solution hypersensitivity, staphylococcal hypersensitivity, tight lens syndrome, and circinate pattern interstitial keratopathy.

MICROCYSTS

SYMPTOMS: If the number of microcysts is fewer than fifty per eye there are usually no symptoms. If there are more than fifty, the patient may experience blurred vision and/or ocular irritation.

SIGNS: Microcysts are tiny, translucent dots in the corneal epithelium. They are often irregular in shape and size and are best viewed with retroillumination and high magnification. They are usually seen in the central to midperipheral area of the cornea. Corneal staining can be seen when they migrate to the surface.

ETIOLOGY: Microcysts are most often seen in extended wear patients. Exactly what microcysts are and what causes them are unknown. They seem to occur in situations of relative hypoxia and poor tear exchange. It has been suggested that they may be metabolic debris from dead epithelial cells which is trapped in the basal cell layer of the epithelium and then migrates to the surface.

TREATMENT: If there are fewer than fifty microcysts per eye, monitor the patient and continue extended wear. If there are more than fifty, try a stricter cleaning regimen with decreased wearing time in an attempt to reduce their number below fifty per eye. If they do not resolve, try a higher oxygen permeable lens or switch to daily wear. If this doesn't work, try a rigid gas permeable lens.

NOTE: Microcysts are also seen in the Soft Lens Associated Corneal Hypoxia (SLACH) syndrome.

SLACH SYNDROME

(Soft Lens Associated Corneal Hypoxia)

SYMPTOMS: The patient initially experiences blurred vision, due to microcystic formation in the corneal basal epithelium. Twelve to twenty-four hours later, when the microcysts reach the corneal surface, the patient develops lens intolerance, tearing, and photophobia. Discomfort occurs, ranging from a mild foreign body sensation to severe ocular pain.

SIGNS: The condition is usually bilateral and relatively symmetric. The hallmark of the SLACH syndrome is an area of central microcystic edema surrounding central puctate epithelial defects. If the microcystic edema is severe enough, complete sloughing of the epithelium can occur, leaving a circular epithelial defect that could occupy as much as 30-40% of the corneal surface. Circumlimbal injection is also present. There is a risk of secondary infection and secondary iritis as well.

ETIOLOGY: SLACH syndrome has been noted with both daily wear and extended wear soft contact lenses. There is usually a history of little or no surfactant cleaning, although many of these patients report disinfection and enzyming as instructed. The cause, then, is thought to be hypoxia resulting from the decreased oxygen transmissability by lipid and protein buildup on the lenses.

TREATMENT: DO NOT PRESSURE PATCH THESE PATIENTS! This condition is caused by hypoxia, and patching will make it worse. In mild to moderate

cases, recovery seems fastest when the patients are given cycloplegics, cold compresses, oral analgesics, and antibiotics. When the symptoms are relieved, this treatment can be discontinued, although the microcysts may take weeks to months to resolve. In severe cases, the compromised epithelium will need debridement. Once you debride the cornea, then you must pressure patch the patient and treat as you would an abrasion. (See corneal abrasion, p. 12)

PROGNOSIS: The patient may be allowed to resume soft lens wear with a decreased wearing schedule and an appropriate sermon about the necessity of disinfecting, enzyming, and surfactant cleaning the lenses. The patient's lenses will, most likely, need replacement. If the patient had been in high water content lenses, you may wish to refit with a lower water content lens to try to reduce deposit formation. If SLACH syndrome recurs, the patient needs to switch to rigid gas permeable lenses or spectacle wear.

SOLUTION HYPERSENSITIVITY

SYMPTOMS: The most common symptoms are redness, itching, and burning which worsen shortly after lens insertion and are somewhat relieved with lens removal. Patients may also report a mild foreign body sensation and photosensitivity.

SIGNS: This condition is bilateral, with a diffuse conjunctival hyperemia and edema. More severe cases can have infiltrates, superficial punctate keratitis, and corneal edema. Occasionally, you may find inclusion cysts.

ETIOLOGY: This is a delayed hypersensitivity reaction to the ingredients in contact lens solutions. By far, the most common culprit is the preservative thimerosal, but others such as benzalkonium chloride, chlorohexidine, and sorbic acid have also caused reactions.

TREATMENT: Discontinue lens wear until the conjunctiva quiets. Cold compresses and decongestants will help relieve some of the symptoms. Then have the patient use thimerosal-free solutions. You may want to have the patient "purge" the lenses by soaking them several times in fresh saline, changing the solution every 2-3 hours.

PROGNOSIS: Once a patient has had a reaction to a certain solution, even one drop of the allergenic solution can trigger the hypersensivity response. Some patients will react to any preserved saline that you recommend and will need to use nonpreserved solutions.

GIANT PAPILLARY CONJUNCTIVITIS

SYMPTOMS: The first symptoms noticed are ropey mucous discharge present at awakening and itching upon lens removal. The symptoms gradually increase, with more mucous, more itching, and more discomfort, along with growing lens intolerance. In the more advanced stages, wearing time is decreased and the lenses tend to be pulled superotemporally.

SIGNS: The primary sign of GPC is a papillary reaction of the conjunctiva. In the early stages, the papillae are barely visible, but they grow in size and elevation until they are larger than 1mm. When the papillae are small, staining with fluorescein and viewing with the cobalt filter help make them more visible. As the amount of mucous discharge increases, the lenses will become more and more coated with deposits.

ETIOLOGY: The cause of GPC has been greatly debated since it was first recognized. Most authorities agree now that it is an immune reaction but are unsure ofthe exact triggering mechanism. A giant papillary reaction has been seen with hard lenses, ocular prostheses, and surgical sutures, but the problem is most prevalent with daily and extended wear soft lenses. Protein deposits on the lens, mechanical irritation of the upper lid, and individual hypersensitivity to lens polymers, deposits, or solutions have all been implicated as contributing factors. Some researchers recommend routine replacement of the lenses every six months to prevent GPC and other types of deposit-related complications.

TREATMENT: In severe cases where there is excessive mucous secretion and lens movement, contact lens wear will not be well tolerated and should be discontinued. Steroids may help relieve symptoms, but as this is a chronic condition, they are not recommended on a long term basis. Opticrom 4% is the recent drug of choice and is recommended three or four times per day until the symptoms are resolved and contact lenses can be tolerated. A maintenance dose of Opticrom once a day should then be prescribed for at least one month to prevent a recurrence. Soiled lenses should be replaced, and the patient should start a stricter cleaning regimen. Some contact lens companies are marketing lenses which are supposed to resist deposits--the CSI-T lens is one example.

In milder cases of GPC, lens wear may be continued with a rigorous cleaning schedule. Some researchers suggest replacement of the lenses. Sometimes this is enough to resolve the symptoms, but in more resistant cases, it may be necessary to use Opticrom. There is some disagreement as to whether Opticrom can be instilled while the lenses are in place.

PROGNOSIS: The papillae will never completely resolve, and there is a possibility of recurrence of the symptoms. Many patients are able to resume soft lens wear with adjustments in cleaning regimen and wearing time. A small number of patients with severe cases of GPC have not been able to tolerate any type of lens wear long after the other symptoms have resolved. Patients with recurrences of GPC should try rigid gas permeable lenses, as they tend to deposit less.

NOTE: Patients should be advised that Opticrom stings on instillation and that the bottle must be discarded 21 days after it is opened.

"TIGHT LENS" SYNDROME

SYMPTOMS: This is a problem seen with extended wear soft lenses. The patient will usually awaken with injection, photophobia, and discomfort after having no signs of a problem the night before.

SIGNS: The condition is usually unilateral, with marked limbal and conjunctival hyperemia. In more severe cases, there can be ciliary injection and stromal infiltrates. There may also be aqueous flare, endothelial bedewing, and epithelial microcysts and punctate staining. These patients will invariably have extended wear lenses with inadequate movement and debris trapped beneath the lenses.

ETIOLOGY: "Tight lens" syndrome is believed to be an immune reaction to debris trapped beneath the contact lenses. It can occur from days to months after starting extended wear. Almost every extended wear patient will have debris beneath the lenses upon awakening, but lenses with adequate movement will clear the debris from behind the lenses. It has been shown that the chances of developing this red eye response decrease as the amount of lens movement increases.

TREATMENT: Discontinue lens wear until the eye has quieted. Mild cases can resolve on their own in three or four days. If there is significant corneal involvement, it may be necessary to prescribe topical steroids. After the eye has quieted, you will need to either refit for greater lens movement or restrict the patient to daily wear. Also, a good practice which may prevent this immune response is to have patients

routinely move the lenses from the cornea to the sclera and back in the mornings to dislodge any debris which may have gathered beneath the lenses during the night.

PROGNOSIS: If a patient has recurrent episodes of the tight lens syndrome, he will probably need to switch to daily wear. Otherwise, after the eye quiets, extended wear can be resumed as long as there is adequate movement of the lenses.

MARGINAL CORNEAL INFILTRATES AND STERILE ULCERS
(STAPHYLOCOCCAL HYPERSENSITIVITY)

SYMPTOMS: If there is no ulceration, patients will feel mild discomfort and foreign body sensation with sectored injection at the limbus. If there is ulceration of the epithelium, the symptoms will be greatly intensified and will include pain, photophobia, tearing, and conjunctival hyperemia.

SIGNS: This condition is unilateral. Most of these patients will have a blepharitis. In the infiltrative stage, there are one or more well-circumscribed, large infiltrates, separated from the limbus by a small area of clear cornea. If there are multiple infiltrates, they will usually be located circumlimbally in the marginal cornea (ring infiltrates.) The most common locations are where the lid margins cross the limbus, at 2, 4, 8, and 10 o'clock. If the area has ulcerated, there will be a significant break in the epithelium, and, possible, a mild iritis.

ETIOLOGY: The most common cause is an immune response to exotoxins produced by staphylococcal bacteria. In some cases, it is believed to be caused by an immune response to protein deposits on the lens. The ulcer is not infectious, but the possibility of secondary infection exists, as it does any time there is a break in the epithelium.

TREATMENT: Discontinue lens wear until the eye is quiet. If the underlying cause is staph blepharitis, prescribe lid scrubs and anti-

biotic ointment for the lids and prophyllactic antibiotic drops four times a day. If lens deposits are suspected, the antibiotic drops are still necessary. Antibiotic ointment for use in the eye at bedtime is also recommended. Since this is an immune reaction, some recommend careful use of a topical steroid in addition to the antibiotics in order to speed relief of the symptoms.

PROGNOSIS: The condition will usually resolve within 7-10 days. Contact lens wear can be resumed after that, with a decrease in wearing time and a stricter cleaning regimen if lens deposits are the problem. If the patient has staph blepharitis, lid scrubs will need to be added to the daily lens care routine.

PSEUDODENDRITES

SYMPTOMS: Tearing, photophobia, and changes in the quality of vision have been reported.

SIGNS: The dendriform corneal lesion has been reported in the midperipheral to paracentral cornea. It must be distinguished from the ulcer found with Herpes Simplex keratitis. The pseudodendrite is raised, rather than excavated, and may stain only lightly with fluorescein, and not at all with rose bengal. HSK is unilateral, while this is often bilateral. The lesion has been described as thinner, more annular and winding than HSK, and it does not have the rounded end bulbs of the branches seen in HSK. Associated signs of mild conjunctival injection, moderate papillary reaction, and mucous discharge have also been reported.

ETIOLOGY: Unknown. This condition is seen with soft lenses. The associated mucous discharge and papillary reaction have led researchers to suspect that pseudodendrites are some sort of a hypersensitivity reaction to certain solution preservatives such as thimerosal. But these lesions have also been reported in patients who were using nonpreserved solutions.

TREATMENT: Discontinue lens wear. Steroids are contraindicated due to the possibility of misdiagnosed Herpes Simplex keratitis.

PROGNOSIS: These lesions usually resolve within two weeks of lens removal. The patient may resume soft lens wear, but it is recommended that the patient not use solutions containing thimerosal. There have been no reports of any corneal scarring or other lasting effects.

NOTE: Another type of "pseudodendrite" may occur with hard or soft lenses. When a foreign body has been trapped under a lens, the "tracks" left may resemble a dendrite. There will most likely be a history of a foreign body sensation. Unlike HSK, there will be no reduction in corneal sensitivity and no staining of the lesion with rose bengal.

CORNEAL DELLEN

SYMPTOMS: The patient will usually have no complaints, although occasionally, one may report a mild irritation or a dull ache.

SIGNS: A dellen is usually found near areas of 3 and 9 o'clock staining or next to a pinguecula. If viewed shortly after lens removal, it will appear as a round area of corneal thinning at the limbus. Fluorescein will pool in the area, although there is really no break in the epithelium. About one hour after lens removal, the area will return to normal thickness, and, after six hours, becomes a greyish-white elevated area.

ETIOLOGY: Dellen are seen with rigid lenses and are caused by excessive drying of the corneal surface. This area of dessiccation is caused by a lens edge which is too thick to allow the lid to wipe over that section of the cornea. That area is not resurfaced with mucin, and the tear layer evaporates more rapidly there than on the rest of the cornea. This leads to 3 and 9 o'clock staining, and, if left untreated, may cause dellen formation. (See Staining, p. 16)

TREATMENT: Discontinue lens wear and prescribe lubricant drops to be used every two to four hours. Antibiotics are not really necessary, as there is no break in the epithelium. The dellen will usually resolve completely within a few days. The patient's lenses will need to be re-edged and polished, or may even need the parameters changed in order to get a thinner edge. If this problem recurs, you may need to refit with a soft lens to provide complete corneal coverage.

PROGNOSIS: Lens wear may be resumed after the corneal signs have resolved. Dellen usually leave no permanent damage if the corneal desiccation is relieved. But if the drying is not resolved, scarring could occur in that area.

CORNEAL MOLDING

(Unplanned corneal curvature changes secondary to PMMA wear)

SYMPTOMS: Spectacle blur.

SIGNS: Corneal edema and distorted or changing keratometer mires.

ETIOLOGY: This is caused by insufficient oxygen reaching the cornea, most commonly seen with the old PMMA lenses. When the cornea becomes hypoxic, it becomes edematous. With the contact lens in place, the cornea maintains its shape. But as soon as the lens is removed, the cornea is free to change its curvature as it swells and deswells. The combination of the corneal edema and the curvature changes cause the spectacle blur.

TREATMENT: The treatment of choice is to refit the patient with rigid gas permeable lenses, as the permeable material allows oxygen to reach the hypoxic cornea and the rigid lens holds the cornea's shape while the edema stabilizes. It is recommended to either use parameters similar to those of the patient's own PMMA lenses or to rely on the fluorescein pattern of diagnostic lenses. The patient should understand that the lenses may need to be changed again after the corneas stabilize. The patient should also be told that the new lenses may be less comfortable, with more lens awareness and more foreign body episodes, because corneal sensitivity will increase as the hypoxia decreases.

PROGNOSIS: Most often, there are no lasting effects. However, there have been reports of later development of irregular corneal astigmat-

ism, which has been called corneal warpage. In such cases, spectacle blur would be permanent, with rigid lenses being the only way to achieve clear vision.

EPITHELIAL SPLITTING OF THE SUPERIOR CORNEA

SYMPTOMS: There are usually no symptoms; this is often found on routine examination. At the most, the patient may report a mild irritation.

SIGNS: There is an arc-shaped breakdown of the epithelium in the superior cornea, 1-3mm from the limbus. This area stains brilliantly with fluorescein, and there may be infiltrates in the area. One report describes "an arc-like opacity" and several opacities in an arcuate line which precede epithelial breakdown.

ETIOLOGY: Unknown. This is seen with soft lenses, and the splitting occurs in the area where the junction zone of the lens would ride, pointing to a mechanical cause. It has also been suggested that hypoxia may play a role.

TREATMENT: Discontinue wear of those particular lenses. Use artificial tears during the day and ointment at night for lubrication. You may want to consider a topical antibiotic to ward off secondary infection.

PROGNOSIS: Epithelial splitting usually resolves within three days and rarely recurs when you refit with a soft lens of a different design or with a rigid gas permeable lens.

CIRCINATE PATTERN INTERSTITIAL KERATOPATHY

SYMPTOMS: There have been very few of these cases reported in the literature. Most often, there was no presenting complaint, although sometimes slightly blurred vision or questions about "this white spot on my eye" led a patient to seek treatment.

SIGNS: The condition is bilateral with deep corneal stromal vascularization and lipid deposition in a circular pattern either parallel to the limbus or around a deep stromal blood vessel. There is a relatively clear area between the lipid exudation and the limbus. There has been no report of associated hyperemia or anterior chamber reaction.

ETIOLOGY: Unknown. The condition resembles both the interstitial keratits seen in ocular manifestations of syphillis and the lipid deposition seen in some patients with high serum triglyceride levels. Because of the clear interval between the limbus and the lipid deposits, some sort of antigen-antibody immunological reaction has been suggested.

TREATMENT: Discontine lens wear. Topical steroids do not help.

PROGNOSIS: To my knowledge, this condition does not progress after removal of contact lenses, but neither does it regress. The blood vessels and the lipid deposits are probably in the cornea to stay.

COMPLICATIONS OF RIGID GAS PERMEABLE EXTENDED WEAR LENSES

At this time, we are on the verge of a large transisiton in contact lens wear. With the recent approval of certain rigid gas permeable lenses for extended wear comes the need for changes in the way we will manage many of our rigid lens patients. Also on the horizon may be a new set of contact lens complications, along with many of the conditions common today. The following are complications suggested by the preliminary findings from the clinical trials that led to FDA approval of the extended wearing of these lenses.

In general, many of the complications are similar to those seen with soft extended wear lenses. However, microbial corneal infiltrates and ulcers seem to be less prevalent, and giant papillary conjunctivitis and superior limbic keratoconjunctivitis do not seem to occur as often.

DEPOSITS: Coating of the lenses is common. Enzyming of the lenses is necessary along with surfactant cleaning to extend the life of the lenses and to help ward off the complications which might by caused by deposit buildup.

LENS ADHESION: Suction between the lens and the cornea is relatively common, especially upon awakening. Several possible causes have been suggested. Dryness of the eye seems to be a factor, and lubricating drops may resolve the problem. Buildup of mucous, protein, and other debris is also believe to play a role. Finally, if the cornea is

toric, flexure may contribute to the problem. In these cases, a flatter base curve or an increase in center thickness may help.

DESICCATION (3 and 9 O'CLOCK STAINING): As with all rigid lenses, peripheral corneal staining is common. (See Staining, p. 16)

GRADUAL CORNEAL FLATTENING: The pressure of the lids during overnight wear is believed to be the cause of a gradual corneal flattening of 0.50D to 0.75D after six months of wear by some patients. This effect is much more prevalent in the morning and is rarely observed during afternoon office visits.

EPITHELIAL GLAZING: This seems to be very rare. It is similar in appearance to central corneal clouding seen in some PMMA wearers but with borders that are less defined. It is believed to be due to a change in the basement membrane of the epithelium, giving it a more translucent appearance.

OCULAR AND SYSTEMIC CONSIDERATIONS

Some ocular or systemic conditions can lead to or exacerbate certain contact lens complications. Some must be taken care of before beginning contact lens wear, some require careful observation, and some contraindicate contact lens wear altogether.

STAPHYLOCOCCAL BLEPHARITIS: This common problem can cause foreign body episodes, chalazia, conjunctivitis, inferior punctate staining of the cornea, and recurrent styes. It can also lead to marginal corneal infiltrates and sterile ulcers. These problems occur in response to the organism itself and to the exotoxins it produces. Chronic blepharitis can lower chances for success with contact lenses. If you decide to try fitting these patients, you should prescribe lid scrubs and antibiotic ointment for the lid margins for two or three weeks prior to dispensing. After dispensing, the patient will need to do lid scrubs at least 3-4 times per week for as long as contact lenses are worn.

ACNE ROSACEA: This is also a staphylococcal problem which affects the sebaceous glands of the forehead, cheeks, nose, and lids. It can be controlled somewhat using long term systemic antibiotics, but it contraindicates contact lens wear altogether, as these patients are already prone to keratitis and vascularization without contact lenses.

SEBORRHEIC BLEPHARITIS: This type of blepharitis is characterized by large, greasy-looking scales at the lid margin and is almost always

associated with a dandruff of the eyebrows and/or scalp. Lens wear may be attempted only after aggressive treatment ofthe skin problem. You can treat the scalp and eyebrows with a dandruff shampoo or selenuim sulfide solution, but these must not be used near the eyes. A sulfacetamide-containing ointment used on the lids will help break up and remove the scales. Vasocidin is a good choice, as the steroid will relieve some of the lid inflammation. As this is a chronic problem, lid scrubs with baby shampoo should become part of the patient's normal hygiene.

HORDEOLUM: This is an acute staph infection of a Meibomian gland (internal hordeolum) or a gland of Zeiss or Moll (external hordeolum). If the lens is not irritating the inflamed area, contact lens wear may be continued cautiously unless there is discomfort or exudation. Treat a hordeolum with frequent hot compresses. Add an antibiotic if there is any exudate.

CHALAZION: This is a more chronic, granulomatous inflammation of a Meibomian gland. If it is small and not inflamed, continue lens wear. If it is too large, it may interfere with lens movement, and you should refer the patient for excision. If it becomes inflamed, stop lens wear temporarily and prescribe hot compresses and antibiotics.

MOLLUSCUM CONTAGIOSUM: This is a viral infection which produces nodules on the lid margins and may have an associated keratitis and follicular conjunctivitis. Contact lens wear will probably not be successful until the nodules are surgically removed from the lid margins.

PINGUECULA: There is no problem in fitting hard contact lenses on a patient with a pinguecula. But if the pinguecula is near the limbus, it could complicate the fitting of a soft lens because the lens must drape over the raised area. You must make sure that there is adequate movement of the lens and that there is no compression of the pinguecula. It may be necessary to use flatter, smaller, or thinner lenses in order to accomplish this. Pinguecula can also have associated dellen. (See Dellen, p. 33)

PTERYGIUM: Contact lenses may trigger further growth of a pterygium, therefore, they are contraindicated. Even after surgery to remove a pterygium, contact lenses are not recommended due to the high percentage of recurrences.

DIABETES: Rigid lenses and extended wear lenses are contraindicated in diabetics, even those who are well controlled. If contact lenses are necessary, hydrogel or silicone elastomer lenses are recommended on a daily wear basis with careful monitoring of the patient's progress. Diabetics heal slowly and have a poor attachment of the corneal epithelial basement membrane, both of which may complicate contact lens wear. If the patient's diabetes is poorly controlled or of long duration, even soft contact lenses are contraindicated because these patients may have reduced corneal sensitivity which could lead to neurotrophic corneal ulcers. If the corneal anterior basement membrane problem does lead to recurrent erosions, bandage soft lenses may be used in some cases to relieve the patient's symptoms.

HYPERTHYROIDISM: Only five percent of patients with hyperthyroidism develop malignant infiltrative ophthalmopathy; most have only slight exophthalmos and/or lid retraction. These patients may wear soft contact lenses, as these lenses will provide some corneal protection. But the patient should be aware that lens deposits may become a problem requiring more frequent lens replacement, and lubricating drops may be necessary several times a day. Contact lenses should not be prescribed until the patient's hyperthyroidism is under control, because an uncontrolled hyperthyroid patient tends toward impulsuve and erratic behavior, which may lead to problems with lens care and handling.

CHRONIC GLAUCOMA: Rigid gas permeable lenses usually cause no problems in glaucoma patients. However, soft lenses easily absorb any drugs which are placed in the eye, with high water content lenses absorbing more than lower water content lenses. Sometimes this is used to advantage with continuous-delivery systems such as Ocusert, in which pilocarpine is placed in a soft contact lens, then slowly released to the cornea as the patient is wearing the lens. In this way, a constant amount of the drug is delivered to the cornea, and a lower percentage of the drug can be used. But if absorption of the drug is not taken into account, and neither the glaucoma therapy nor the wearing schedule is altered accordingly, the risk of systemic side effects of the drug is increased, due to the increased contact time of the drug. Another problem with soft lenses is that they are discolored by epinephrine. This is easily remedied by using Propine, which is a prodrug of epinephrine. Epinephrine is produced only when the drug reaches the anterior chamber, thus reducing the chance of discoloration problems.

VERNAL CONJUNCTIVITIS: This chronic allergic condition mostly affects males throughout their adolescent years. Its symptoms are usually most apparent in the spring and summer months, and patients will often ask to be fit during the times when the eyes are quiet. This is not recommended. Vernal conjunctivitis is usually benign and self-limiting, but complications of keratitis, vascularization, and ulceration can arise. The disease usually resolves by early adulthood, after which lens wear may be attempted, depending on the resultant health of the cornea and conjunctiva.

HERPES SIMPLEX KERATITIS: Patients who have a history of HSK should probably not wear contact lenses. The keratitis may be recurrent, and contact lenses may be a triggering mechanism.

THEODORE'S SUPERIOR LIMBIC KERATOCONJUNCTIVITIS: During the course of Theodore's SLK, bandage soft contact lenses may be used to relieve the patient's symptoms from the filamentary keratitis which may develop. After the SLK has resolved, however, contact lenses are not recommended.

EPITHELIAL BASEMENT MEMBRANE DYSTROPHY: This is also known as Cogan's microcystic or map-dot-fingerprint dystrophy. These patients are prone to recurrent erosions. Because of this, hard lenses should be avoided, as they rub aginst the cornea. Soft lenses may be worn if the patient does not develop significant edema.

FUCH'S ENDOTHELIAL DYSTROPHY: Any eye with poor endothelial function is more prone to corneal edema, and these patients must be closely monitored if they are to wear contact lenses. Extended wear lenses are contraindicated.

PREGNANCY/ORAL CONTRACEPTIVES: Some women who are pregnant, taking oral contraceptives, or undergoing menopause have been reported to develop decreased contact lens tolerance. Symptoms include dryness, burning, and reduced wearing time. High water content soft lenses are not recommended, as they are prone to dehydration. Rigid gas permeable or standard thickness low water content soft lenses are better at reducing symptoms. Lubricating drops will probably be necessary. Advise patients of the possibility of increased lens deposits and the need for more frequent lens replacement.

HYPERTENSION: Antihypertensive and diuretic medication can also cause dry eye symptoms. Manage these patients as you would those with symptoms from oral contraceptives. (See above.)

LONG TERM ORAL STEROID THERAPY: Contact lens wear is contraindicated in these patients. Steroids cause suppression of the immune system, and patients on steroid therapy are less able to resist infections and other complications which may arise with contact lens wear.

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