OCULAR CHANGES DUE TO CONTACT LENSES

BY *Joseph M. Dixon*, M.D.

THE STUDY PRESENTED HERE is concerned with the eye that wears a contact lens. It is not concerned with the optical principles or the fitting of contact lenses.

Each day several million people apply small plastic discs to their corneas and leave them in place from 12 to 16 hours. Adequate knowledge of the effects of these foreign bodies in contact with the ocular tissues has not been available. Scattered reports of the ocular changes due to contact lenses are for the most part concerned with scleral lenses. Fick,[1] Sattler,[2] Smelser,[3] and Kinsey[4] have reported clinical and experimental studies, all of which deal with scleral lenses, and which may be only partly applicable to the popular corneal lenses in general use today. Hirano,[5] Spaeth,[6] and Morley and McCulloch[7] studied the effects of corneal lenses on experimental rabbit eyes for short periods, and Black[8] made important clinical observations. Recent studies of the effects of corneal lenses have been made by Dixon and his co-workers.[12,19,34,39,40,45]

It is clear that basic research in the medical problems of contact lenses has lagged far behind the popular use of these lenses by the public. Attempts to further clarify some of the medical problems associated with corneal contact lens wear were started in November, 1960, at the University of Alabama Medical Center, Department of Ophthalmology. The purpose of this study has been to try to understand the changes caused by wearing contact lenses in order to avoid damage to the eyes.

An outline of these studies is as follows:

I. TISSUE CHANGES CAUSED BY CONTACT LENSES
   A. Alterations in the corneal epithelium
   B. Corneal vascularization

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C. Alterations in the corneal endothelium
D. Alterations in the conjunctiva
E. Alterations in the tear fluid
F. Histochemical studies
G. Spectacle blur
H. Detachment of the retina

II. ADAPTATION TO CORNEAL LENSES

III. MICROBIOLOGY OF CONTACT LENS WEAR

A. Bacterial infections and bacteriology of the eye with contact lenses
B. Bacterial flora in contact lens containers
C. Corneal herpes simplex infections complicating corneal lens wear
D. Fungus infection of the cornea complicating corneal lens wear

IV. ALTERATIONS IN THE INTRAOCULAR PRESSURE

V. GLARE CAUSED BY CORNEAL LENSES

VI. SUMMARY AND CONCLUSIONS

METHODS AND PROCEDURES

An attempt has been made to correlate the findings of basic laboratory and experimental studies with clinical observations.

The rabbit eye has proven to be suitable for tests because pathologic changes occur more rapidly in it than in the human. Methyl-methacrylate corneal lenses were fitted to one eye of each rabbit with the base curve made to coincide with the flatter meridian of keratometer readings. The opposite eye was reserved as a control. The lenses were tricurve, 11.1 mm. in diameter. Removal of the nictitating membrane was not necessary because it slides over the lens during blinking in the same manner that the lids do. One hundred and eighteen rabbits have been used in this study.

Ten dogs were fitted with similar lenses, 14 mm. in diameter on one eye of each dog. The morphologic changes in the dog were similar to those found in the rabbit except they developed more slowly.

Three human eyes wearing corneal lenses were obtained for pathologic examination by fitting the lenses to eyes with normal anterior segments 10 to 12 hours before enucleations were done because of intraocular malignant melanomas. The lenses were fitted with the base curves made to coincide with the flatter keratometer readings. Properly fitting lenses from trial sets were used.

Seven hundred and seventy-five patients seen in clinical practice wearing tricurve corneal lenses have been studied by routine cycloplegic refraction, tonometry, slit-lamp examination, keratometer readings, corneal diameter measurements, corneal sensory measurements, peripheral fields, and fundus examination. Special groups were studied with cultures of the lids and conjunctivas before and after wearing corneal lenses using blood agar plates. Conjunctival epithelial scrapings with Giemsa's stains were made on some and tear fluid samples taken on others as described in detail in the sections of this report devoted to those special subjects. Other observations made during follow-up visits by patients are reported which did not allow sharply defined statistical analysis. Statistical data included in published reports referred to are not all repeated.

The studies presented in the various sections that follow are intended to add to what is known with the understanding that the accumulation of new knowledge will continue with the passing of time.

I. TISSUE CHANGES CAUSED BY CONTACT LENSES

A. Alterations in the Corneal Epithelium

The intimate contact between corneal lenses and the soft corneal epithelium makes it probable that all eyes wearing these lenses have some form of epithelial alteration. Clinical and histologic observations on human eyes and experimental animals presented in this report confirm this view.

The corneal epithelium is soft and pliable. It is easily molded mechanically, and the cells are more loosely attached to Bowman's membrane than they are to each other. The absence of keratinization makes the surface easily indented. Foreign bodies readily become imbedded in this soft tissue, but defects are rapidly repaired. The tip of a cilium from the lid may touch the cornea so gently that the patient is not aware of it while sufficient cells are wiped away to make a staining area with fluorescein. Under normal conditions the swift blinking of the lids sweeps and evenly lubricates the corneal surface by the palpebral conjunctiva and thus maintains a highly polished optical zone. A thin plastic lens fitted to the corneal contour and separating the upper lid from the cornea is an imperfect substitute for the lid. Many alterations reported in this study have been observed in the epithelium of corneas wearing corneal lenses, but most are transient.

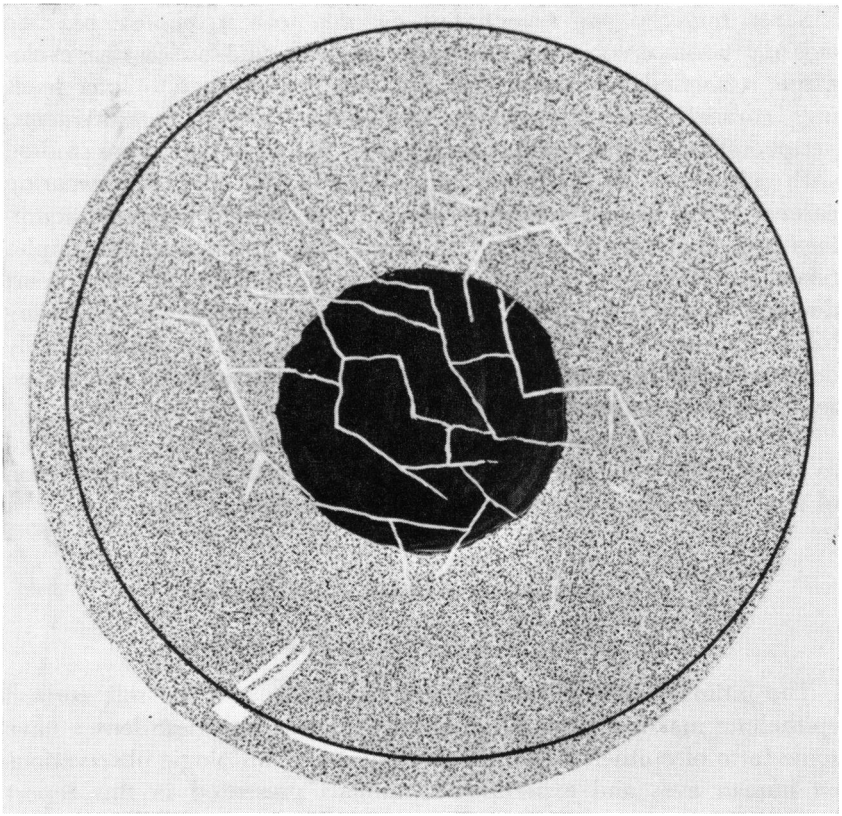

FIGURE 1. PATTERN OF BRANCHING WRINKLED FURROWS IN SURFACE OF CORNEAL EPITHELIUM UNDER CORNEAL CONTACT LENS SEEN WITH SLIT-LAMP AND COBALT FILTER

BRANCHING FURROWS IN THE EPITHELIAL SURFACE

Fine branching lines in the corneal epithelial surface are observed in one-quarter of contact lens patients provided they are examined with a slit-lamp using a cobalt filter and fluorescein (Figure 1). These lines appear to be minute wrinkled furrows in the epithelial surface that collect fluorescein. They cause no symptoms and rapidly disappear when the lenses are removed to allow the lids to repolish the corneal surface.

These lines appear to be identical with those produced by massaging the cornea with pressure through the closed lids. The pressure furrows were first described entoptically by Helmholtz[9] who accurately located them on the corneal surface. Friedman[10] and Finkelstein[11] also studied these lines entoptically but were unable to visualize or localize them

with the slit-lamp technique available at that time. Pressure furrows can be reproduced experimentally at will on the normal cornea and observed with the slit-lamp by using fluorescein and a cobalt filter.

The branching furrows under corneal lenses appear to be identical with the mechanical alteration of the pliable epithelial surface produced by pressure and have not been a cause for concern. The average individual who vigorously rubs his eyes and notices transient blurring of his vision has produced this change many times.

EPITHELIAL DIMPLES

The finding of groups of dimples in the surface of the corneal epithelium with bubbles trapped in the dimples under corneal lenses has been reported by Dixon and Lawaczeck[12] in 65 of 353 patients.

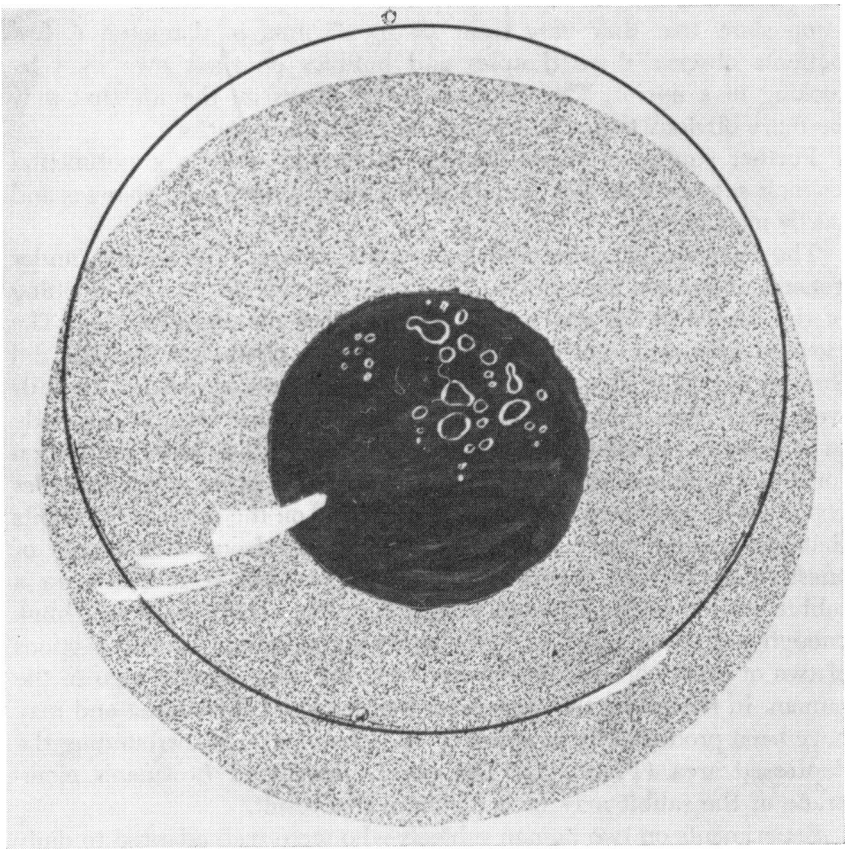

FIGURE 2. AIR BUBBLES TRAPPED IN EPITHELIUM DIMPLES UNDER CORNEAL CONTACT LENS

They aspirated these bubbles, and mass spectroscopy analysis demonstrated that they consist of air. The bubbles do not cause the dimples, and they are not a product of metabolism.

Clinically, these dimpled depressions in the corneal epithelium first appear as small scattered dots one or more hours after corneal lenses are inserted. They are separated by areas of normal epithelium. Fluorescein will collect in the depressions and give the impression of staining. There are no symptoms, and while under slit-lamp observation the dimples can be seen to increase in width and depth. The first dots are round, but with increasing size they may become oval- or horseshoe-shaped (Figure 2). As the lids blink, small bubbles of air at the lens margins are forced under the lenses where they collect in the dimples and remain fixed in that location in spite of lens excursions. Measurements of these dimples with a calibrated ocular on the slit-lamp show that they vary from .05 to .35 mm. in diameter. A few patients observe these dimples and bubbles on their own eyes by looking in a mirror. The dimples cast shadows on the iris that may be more obvious than the dimples on the corneal surface.

Further studies of these dimples on humans and in experimental animals suggest that they may be partly due to metabolic changes and partly mechanical.

The injection of saline into the corneal stroma of 4 rabbits under general anesthesia using a No. 27 hypodermic needle caused clouding of the cornea in the area of the fluid without dimple formation. The clouded area was mottled and not uniform. Epithelial dimples did form in the clear areas of the cornea in these same eyes while the lids were held open for observation. They would also form consistently in the corneas of rabbits under anesthesia if the lids were held open for 5 or 10 minutes and the tear film allowed to drain away. Dimples could not be produced by the same technique on the corneas of rabbits that had expired more than 15 minutes before from an overdose of anesthesia. On rare occasions dimples have been seen by us on a rabbit cornea under a contact lens, but the rabbit does not blink enough to trap air bubbles. Pathologic examination of stained sections of two of these corneas did not resemble the same picture seen in the human. In the rabbit the depressed area involved the stroma and may have been produced by swelling of the corneal stroma surrounding the depressed area (Figure 3). The poorly developed Bowman's membrane of the rabbit may be a factor in this result.

Experiments on two human subjects who were well adapted to daily wear of corneal lenses quickly produced groups of epithelial dimples and some deep furrows by simply removing the lenses and holding the

lids open until the precorneal tear film drained away (Figure 4). No anesthetics were used. Blinking of the lids later erased the dimples within 2 or 3 minutes. The same technique was used on two normal subjects who did not wear contact lenses. Also topical anesthesia was

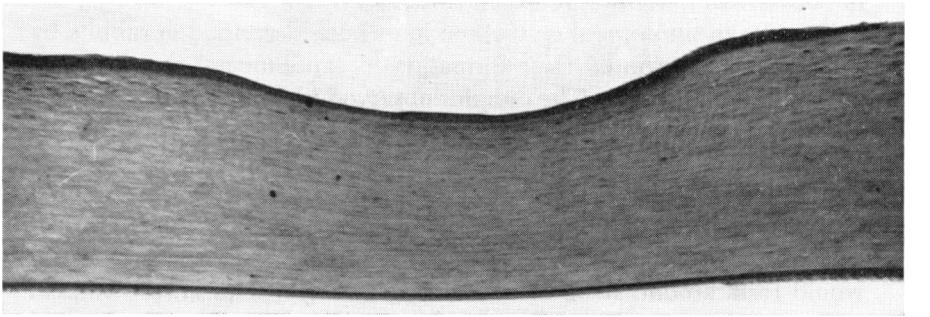

FIGURE 3. PHOTOMICROGRAPH OF DIMPLE IN RABBIT CORNEA UNDER CORNEAL CONTACT LENS
The epithelium is not broken (Defect in endothelium is an artifact).

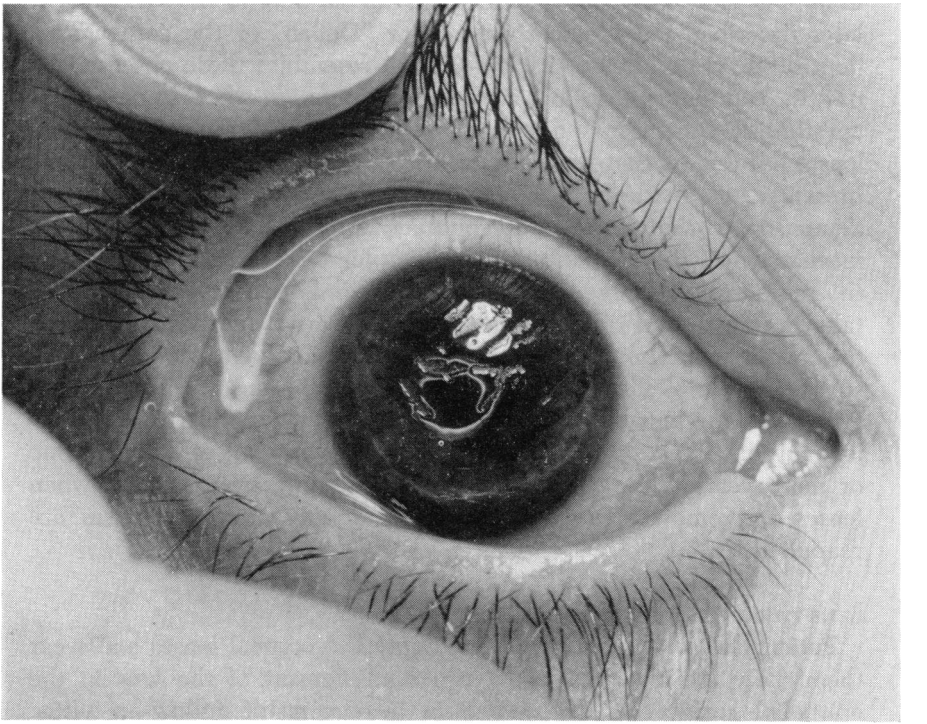

FIGURE 4. EPITHELIAL DEPRESSIONS IN HUMAN CORNEA PRODUCED BY REMOVING CORNEAL CONTACT LENS AND PREVENTING BLINKING WHILE TEAR FLUID DRAINS AWAY

used, and the lids held open until the precorneal tear film evaporated. Wide bands of depressed areas of the epithelium appeared, but typical round and crescent-shaped dimples did not develop. In these subjects the vision was reduced to 20/200. After blinking normally 30 to 60 minutes the vision returned to 20/20.

Dimples in the corneal epithelium have been described in rabbits by Gaule[13] who attributed their formation to experimental sectioning of the Gasserian ganglion. The dimples observed in our experiments when the lids of rabbits were held open while under anesthesia appear to be the same.

Eckhard,[14] in commenting on "Gaul's Pits" in the rabbit, also noted that the pits could be produced easily without disturbing the trigeminal nerve. Eckhard noticed that depressed areas in the corneal epithelium would form around small bits of hair or dust particles on the corneal surface. Observations in our experiments confirm this. E. Fuchs[15] described epithelial dimples or pits near the limbus in patients who had conjunctival edema adjacent to the dimples. The dimples E. Fuchs described were large and measured 1.0 to 3.5 mm. in size. A. Fuchs[16] later described pathological dimples or "Dellen" of the cornea as a depressed area of 1.69 or 2.0 mm. which resembles those observed by us after exposing the normal cornea to evaporation when using topical anesthesia. Dickinson[17] mentioned epithelial craters under contact lenses but denied that the bubbles in the craters were trapped air. He presented no evidence for his statement. Dixon and Lawaczeck[12] demonstrated the nature of the bubbles in the dimples, but the exact mechanism of the formation of the dimples under contact lenses and the relationship between these and other dimples formed experimentally in human and rabbit corneas is an unsolved question. Neither is the relationship between mechanical and metabolic factors understood. The dimples are more common under tight lenses.[12] They do not form when the lens has a central perforation. They appear to be harmless and have been observed in patients who wore lenses daily for one or more years without symptoms. They disappear each night when lenses are removed and reappear the next day after the lenses are reapplied.

LENS IMPRINT ON THE CORNEA

Patients who have become well adapted to corneal lenses and wear them daily are observed to have a faint imprint of the lens in the epithelial surface of the cornea in 5 percent of follow-up visits. Typically, this can only be seen by using the mercury vapor lamp in

a darkened room after fluorescein has been instilled in the conjunctival sac. Usually these are cases in which the excursions of the lens during blinking of the lids are small, and the lens tends to settle in a relatively fixed position. This corneal imprint of the lens is seen when the observer displaces the lens. A crescent in the fluorescein pattern is then observed on the cornea at the position of the lower peripheral curves of the lens. This crescent is raised in the form of a ridge which presses the fluorescein away from the lens rather than a furrow which collects fluorescein.

The lens imprint on the cornea is more pronounced on rabbit eyes that are intentionally fitted with tight lenses. The rabbit blinks less frequently than the human, the lens remains in a more fixed position, and the rabbit cornea is larger in diameter, thinner, and more easily molded. A tight experimental lens may make an imprint so pronounced that when the lens is removed, the corneal impression resembles a lens that is still in place. Microscopic examination of one of these rabbit corneas that had been fixed with Zenker's solution and imbedded in celloidin shows that the epithelial cells under the lens position are distorted. Those cells immediately peripheral to this position of the lens are increased to 2 to 3 times normal number, and a ridge is created (Figure 5). This might explain the appearance of a faint ridge in the fluorescein pattern on the human cornea when the lens is displaced.

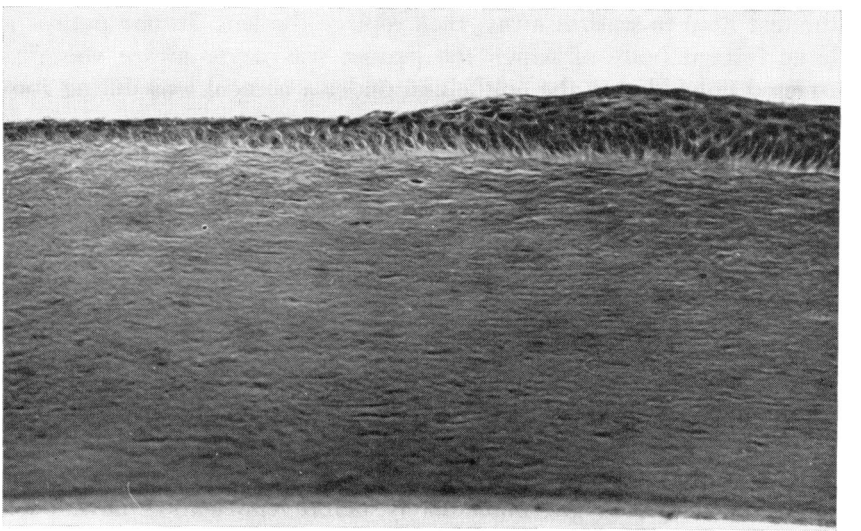

FIGURE 5. THICKENED AREA OF CORNEAL EPITHELIUM OF RABBIT AT MARGIN OF LENS IMPRINT ON CORNEA

In both the human and the rabbit this impression in the soft epithelium is transient and rapidly disappears after removal of the lens.

EPITHELIAL TRAUMA

The delicate corneal epithelium is vulnerable to trauma. Minor lesions caused by corneal lenses as reported by Dixon[18] are observed clinically in several forms.

Patients who are unskilled in handling the lenses frequently cause small abrasions of the corneal epithelium while removing and inserting the lenses. They may do this by striking the sharp edge of the lens or a fingernail against the cornea.

Foreign bodies blown into the eyes of individuals who are not wearing lenses are usually washed away by the tear fluid and by the blinking of the lids. These same foreign bodies that float into eyes wearing corneal lenses are often swept under the lenses where they are rolled between the lenses and epithelium. Routine slit-lamp examinations of patients during follow-up visits show that the faint scratches in the epithelial surface made by foreign bodies are so common that they are considered a normal part of wearing lenses. Patients who have decreased corneal sensitivity are not aware of the very small foreign bodies and faint corneal scratches. Most patients who wear lenses daily have experienced the sudden sharp pain of a large foreign body under a lens. If it does not float out quickly, they remove the lens and allow the tear fluid to wash it away, then replace the lens. In one patient a large foreign body of which the patient was never aware was discovered imbedded in the epithelium under a corneal lens during routine follow-up examination.

Abrasions produced by movements of the lens while blinking the lids are of two types. First are abrasions produced by the periphery of a lens that is too steep or poorly fitted. The epithelial cells are dislodged or abraded away by the edge of the lens faster than they can regenerate. This appears as a crescent stained with fluorescein on the cornea at the position of the lens edge. Second are those abrasions produced by the center of a lens that is too flat and causes excessive pressure and erosion of the epithelium over the center of the cornea. Both of these forms of abrasions have been consistently reproduced experimentally on 8 rabbit corneas by applying lenses too steep or too flat.

Pathologic studies of 3 normal human corneas that wore well-fitting lenses during the night immediately before enucleation have been made. Zenker fixation and imbedding in celloidin was done to avoid artifacts. The microscopic appearance of these corneas is as follows: as the margin of an abrasion is approached, the flat surface cells are

FIGURE 6. HUMAN CORNEAL ABRASION DUE TO CORNEAL LENS

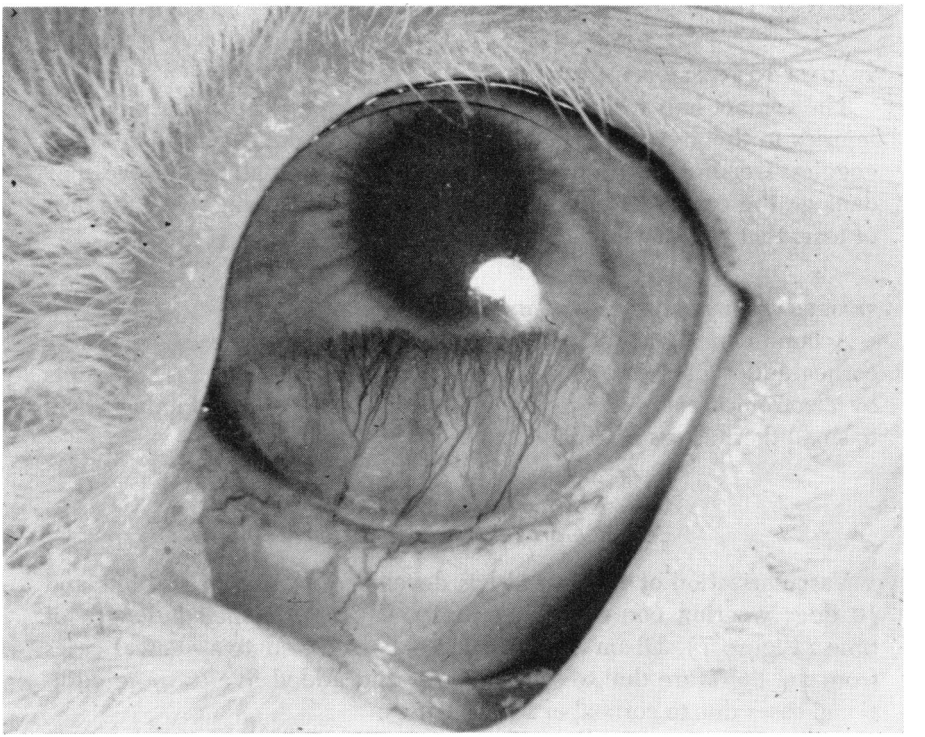

FIGURE 7. VASCULARIZATION OF RABBIT CORNEA DUE TO PROLONGED CORNEAL LENS WEAR

lost, some of the wing cells disappear, some of those remaining are macerated with rupture of the cell membranes and nuclei. Finally a row of remaining basal cells clinging to Bowman's membrane is distorted and macerated while in the center of the abrasion this membrane is completely denuded (Figure 6).

A form of central epithelial damage caused by prolonged lens wear is poorly understood and may not be a true mechanical abrasion. It is manifest clinically by severe pain and blepharospasm, usually bilateral, which appears several hours after lenses have been removed following a period of lens wear which is longer than usual or after sleeping with the lenses on the cornea. Frequently, it occurs in a patient who has recently acquired corneal lenses and because of overenthusiasm and poor adaptation exceeds the advised wearing time. Slit-lamp examination of these patients shows epithelial edema and an irregular central area staining with fluorescein and having a granular appearance. These areas may vary in diameter from .10 to 5.0 mm. Lid edema is present, and pain seems to be out of proportion to the severity of the lesions, but they heal within twenty-four hours after the lenses are removed. The result is a wiser patient.

TRAUMA TO THE LENS ON THE CORNEA

The contact lens resting on the cornea protects it from sharp cutting injuries in the area of the lens, but it gives less protection than safety goggles. Corrosive chemicals accidentally contaminating the eye may damage the cornea more severely by the lens interfering with the flow of tears that wash away these chemicals.

TEMPERATURE OF THE CORNEA UNDER A CORNEAL LENS

Accurate measurements of the temperature of the surface of the cornea under a contact lens have not been successful because attempts by electronics engineers to adequately insulate the conducting wires to small thermistors have failed.

B. Corneal Vascularization

Vascularization of the cornea has developed in all of 31 rabbits and 10 dogs wearing corneal lenses constantly for a sufficient length of time (Figure 7). Dixon and Lawaczeck[19] collected two clinical cases from the literature due to scleral lenses and added five or more additional cases due to corneal or scleral lenses.

CLINICAL OBSERVATIONS

Lauber[20] in 1929 and Strebel[21] in 1937 reported corneal vascularization due to wearing scleral lenses. Strebel stated that vascularization of the cornea in keratoconus developed after wearing the lenses in the presence of central erosions but that it was beneficial in flattening the corneal curvature.

Delgado,[22] Paton,[23] and Baird[24] have contributed clinical cases to this report. Delgado's case was a brush-like vascularization of the deep corneal stroma extending to within 2½ mm. of the corneal apex. The patient was a 50-year-old man with aphakia who used the lenses for 6 months, frequently sleeping with them on the corneas. After the lenses were discontinued, blood rapidly disappeared from the vessels. Corneal lenses were used in this case, the vascularization was bilateral, and there was no previous corneal disease. Paton[23] reported that he had observed several cases of vascularization of the cornea due to contact lenses and that it is not infrequent in conical cornea with scleral lenses. He also observed that the vascularization disappeared when the lenses were discontinued. Baird[24] reported corneal vascularization extending from the lower limbus in a man with keratoconus. Scleral lenses were started in 1947 and in 1950 changed to corneal lenses. There were several attacks of conjunctivitis, and vascularization was first noticed in January, 1962. The lenses had not been worn more than 10 to 12 hours a day.

Clinically we have frequently observed overfilling of the limbal vessels due to corneal lenses, and occasionally the vessels at the upper limbus proliferate a short distance into the clear cornea.

FINDINGS IN EXPERIMENTAL ANIMALS

One hundred percent of 31 rabbits and 10 dogs wearing methylmethacrylate corneal lenses fitted parallel to keratometer readings developed extensive corneal vascularization when the lenses were worn constantly 3 to 6 weeks. Lenses were tricurve, 11.1 mm. in diameter for rabbits and 14.0 mm. for dogs. One eye of each animal was fitted with a lens, and the opposite eye reserved for a control. Pathologic studies were made of both eyes of all rabbits. There was no vascularization or any other change in any of the control eyes of rabbits or dogs.

Vascularization started consistently from the lower limbus where the lens rested partly on the conjunctiva, but when a large corneal abrasion was present under the lens, new vessels later grew down from the upper limbus which was not covered by the lens. Corneas which had minor epithelial erosions or none at all vascularized only

under the contact lenses. Observations of the vessels with the biomicroscope showed that vascular systems grew in both superficial and deep layers of the stroma. Each brush-like vascular tree remained in the same level of the stroma whether it was deep or superficial. Any cornea would contain vascular systems in more than one level.

Vascularization appeared within an average of 2 to 3 weeks in rabbits without secondary infections and was usually complete in 4 weeks. Bacterial keratoconjunctivitis, which appeared in 6 eyes, accelerated the vascularization, and it was completed within 7 to 10 days. Secondary infections could be prevented by the use of antibiotic ointments once daily.

All eyes of 6 rabbits wearing lenses fewer than 16 hours per day for 8 months failed to vascularize.

Corneas which had completely vascularized in 13 rabbits showed rapid regression of circulation in the vessels within 5 to 7 days after the lenses were removed. These residual ghost vessels resembled clinically inactive interstitial keratitis when observed with the biomicroscope. Eight rabbit corneas that had completely vascularized by constant wearing of corneal lenses were allowed to recover by removing the lenses for a 40-day period. The same lenses were replaced on the same corneas, and the inactive ghost vessels rapidly refilled with circulating blood within 17 hours in all the corneas.

Eight rabbits were fitted with lenses containing 0.45-mm. perforations. The perforations varied in number from 1 to 8, and the lenses were worn constantly with no secondary infections. Corneal vascularization proceeded in the same way as with non-perforated lenses except that new vessels appeared one week later and were less numerous. Corneal vascularization was not influenced by the number of perforations in the lenses.

COMMENT

Cogan[25] studied the pathogenesis of corneal vascularization and proposed that corneal edema was an important factor, provided the edema was prolonged and involved an area near the vessels of the limbus. Our findings are consistent with this theory. In Cogan's experiments there was a latent period of 2 days before any changes could be observed in the limbal vessels. This seems to be an important factor, because in our studies none of the animals which wore lenses only a portion of each day developed corneal vascularization. This daily intermittent wear could also be the reason that the corneas of the several million people now wearing corneal lenses so rarely vascularize.

Smelser[3] reported corneal haze and halos in humans with fluid scleral lenses. He concluded that this resulted from interference with deturgescence of the cornea. Smelser and Ozanics[26] reported edema of the corneal stroma in guinea pigs wearing fluid scleral lenses. Kinsey[4] reported an increase in corneal thickness due to increased water content of the cornea in human subjects wearing fluid scleral lenses. In our studies the use of perforated lenses on rabbits in order to allow a greater exchange of gases and tear fluid did not prevent vascularization, but the mechanism of corneal edema due to prolonged epithelial abrasions and mechanical trauma was not excluded.

SUMMARY AND CONCLUSIONS

Corneal vascularization has appeared in human eyes after continuous corneal lens wear, and with intermittent wear, with prolonged abrasions in cases of conical cornea. In the small group of cases available at this time, corneal vascularization has been more frequent clinically in conical cornea.

Corneal vascularization will occur consistently in experimental animals with continuous corneal contact lens wear after 3 weeks or more. Vascularization in experimental animals is accelerated by prolonged and large epithelial abrasions or by secondary bacterial infections. Vascularization recedes rapidly in human corneas and experimental animals when corneal lenses are removed. Reactivation of vascularization is extremely rapid when corneal lenses are placed on previously vascularized and scarred corneas.

The mechanism of vascularization could be prolonged corneal edema. The influence of restricted utilization of oxygen or exchange of gases is unknown.

It is concluded that in clinical practice, contact lenses in their present form should be worn intermittently with a minimum of mechanical trauma to the cornea and not worn at all in the presence of infections of the external eye.

C. Alterations in the Corneal Endothelium

No clear histological or clinical endothelial alterations have been observed in the endothelium of normal corneas due to corneal lens wear.

One patient with bilaterial aphakia and cornea gutatta type of degeneration of the endothelium of the center of the cornea developed bilaterial epithelial edema and clouding of the corneal stroma due to daily corneal lens wear. This condition receded after the lenses

were discontinued, and the endothelium appeared to be the same as before lens wear was started. One patient is insufficient for conclusions.

D. Alterations in the Conjunctiva

Minor transient alterations occur in the conjunctiva due to corneal lenses but are rarely a clinical problem. The rare complication of a lens becoming lost and buried under the conjunctival fornix has been described by Green.[27]

CONJUNCTIVAL HYPEREMIA

Hyperemia of the conjunctiva routinely accompanies the lacrimation and epiphora of patients beginning to wear corneal lenses. This has also been observed in microscopic sections of the eyes of all rabbits after a few hours of wearing experimental lenses. Occasionally patients will give up wearing lenses because the conjunctival hyperemia persists indefinitely and becomes a cosmetic problem. The lenses may be well fitting, the vision normal, and the corneal epithelium free of defects. The conjunctival hyperemia is transient in the usual case and disappears a few days or weeks after patients become adjusted to routine lens wear.

FOLLICULAR HYPERTROPHY

Hypertrophy of the conjunctival lymphoid follicles was a consistent microscopic finding in rabbits after wearing corneal lenses, and this was accentuated in those having secondary conjunctivitis. It is known that the follicles of the rabbit hypertrophy with mild provocation.

No pathologic material of the human conjunctiva is available after wearing corneal lenses. Only 2 cases have been observed clinically with follicular hypertrophy of the upper tarsal conjunctiva after wearing corneal lenses. These were non-symptomatic. The lenses were not discontinued, and after more than a year of daily lens wear, the follicular hypertrophy has seemed to regress slightly.

E. Alterations in the Tear Fluid

Samples of lacrimal fluid to be studied were taken from the lacrimal lake at the inner canthus. A white blood cell pipette was used to collect the fluid using the same technique used to take a sample of blood from a puncture of the tip of the finger. Tear fluid samples were taken from patients before applying corneal lenses and repeated after 6 or 8 hours of lens wear.

Tear fluid samples collected while increased lacrimation is present

do not give valid data on the composition of the tears under normal conditions. For this reason small quantities were removed from the lacrimal lake without disturbing the eyeball. The tests were limited to individuals who were accustomed to wearing corneal lenses and also accustomed to having tear samples removed.

LEUCOCYTE COUNT

Leucocyte counts were made on the tear fluid of 8 patients by putting the undiluted tear fluid on a blood cell counting chamber and counting the cells with the high dry objective of the microscope. Repeated leucocyte counts on these patients in the mornings before putting on their lenses averaged 1 to 3 cells per cubic millimeter. After wearing the lenses 6 or more hours the leucocyte count increased to 30 or 60. The origin of these cells in the tear fluid is presumed to be the conjunctival vessels. Microscopic sections of the eyes of rabbits wearing corneal lenses have many neutrophiles and some lymphocytes in the tissues surrounding the conjunctival vessels 6 or more hours after corneal lenses are applied.

SEBACEOUS CONTENT

Microscopic examination of the tear fluid on the blood counting chamber often showed an increase in the oil droplets in the tears after wearing corneal lenses. The oil droplets float up against the cover slip while the leucocytes lie at a lower level on the counting chamber.

VARIATIONS IN THE pH

Measurements of the pH of the tears using a Beckman pH indicator were not sufficiently valid for scientific conclusions. In the absence of lacrimation the small quantity of tear fluid obtained in the micropipette did not give sufficiently reliable readings. The tear fluid obtained during lacrimation is adequate in quantity but does not represent a reliable sample, Adler[28] does not consider the variations in pH to be significant.

F. Histochemical Studies

Histochemical studies of the corneal changes due to wearing scleral lenses have been made by Smelser and Ozanics[26] who reported that corneal edema is probably due to restriction of the access of atmospheric oxygen to the cornea. Smelser and Chen[29] reported an increase in lactic acid in the cornea after wearing scleral lenses.

Studies of the glycogen content of the corneal epithelium of rabbits after wearing corneal lenses have been reported by Hirano[5] and Spaeth.[6] Calmettes and his co-workers[30] made studies on 10 dogs. Hirano used about 30 rabbits while Spaeth used one rabbit. These writers report a decrease in the glycogen content of the corneal epithelium as interpreted from PAS stains on histologic sections. They infer that glycogen depletion is due to restriction of the access of atmospheric oxygen to the cornea by the corneal lens.

Preliminary histochemical studies in our laboratory on both eyes of 13 rabbits and 2 normal human corneas wearing corneal lenses have demonstrated that these findings must be interpretated with caution for the following reasons.

1. The staining reaction of the corneal epithelium varies from the center of the cornea to the periphery and sections of one eye compared with sections of a control eye must be cut from precisely identical areas of the cornea.

2. It is debatable whether one may infer that variations in glycogen content of the corneal epithelium under a contact lens are due to the availability of atmospheric oxygen. Several factors may influence the staining reaction such as profuse lacrimation, epithelial edema from irritation and trauma of the lens and increased blinking, as well as other metabolic factors.

3. Atmospheric oxygen must first go into solution in the tear fluid before it is available to the cornea. The role of oxygen in the mechanism of corneal edema in eyes wearing a corneal lens where the flow of tears under the lens may actually be accelerated by blinking and profuse lacrimation needs further study. The interpretation of histochemical reactions in the corneal tissues also needs further study. There may be a discrepancy between the histochemical and metabolic reaction of the rabbit and human corneas.

G. Spectacle Blur

Spectacle blur is the blurred vision most patients notice when they put on their spectacles immediately after wearing contact lenses. This is probably the result of several factors rather than a single specific cause. The condition occurs so commonly that it is considered a normal manifestation of contact lens wear. It is found in patients who are well adapted to corneal lenses which are well-fitting, nonsymptomatic, and correct the vision to normal. Spectacle blur may last for several hours to a day or two and leads patients to believe

that they see better with contact lenses than with glasses. This is actually true for a transient period. Some of the causes of spectacle blur are apparent and some are obscure.

EPITHELIAL CHANGES

Epithelial changes are a common cause of spectacle blur. Some of these changes may be seen with the slit-lamp as the branching furrows and dimples previously referred to. Examination of the cornea by the reflected mires of the keratometer shows distortion and loss of its regular refracting surface. Refraction of these patients during the period of spectacle blur does not correct the vision to 20/20.

On microscopic examination of stained sections of eyes of 6 rabbits wearing lenses on a 10- to 12-hour daily schedule for 8 months, the regular arrangement of the epithelial cells is altered. Many of the basal cells are no longer parallel or perpendicular to the thin Bowman's membrane. The wing cells are distorted, and the flat surface cells no longer present a flat surface. This was reported by Hirano[5] and confirmed by studies in our laboratory. It is necessary to exercise care in interpretation of these microscopic sections because the technique of fixation and sectioning often produces artifacts resembling abnormal changes.

ALTERATIONS IN REFRACTION

One patient seen clinically experiences transient but wide excursions in the refractive error after wearing corneal lenses, but there are no changes in keratometer readings.

This case is a 29-year-old white female with cycloplegic refraction of O.D. $-3.00\ -1.00 \times 30$ and O.S. $-3.25\ -1.25 \times 175$ which corrects her vision to 20/20 each eye. After wearing well-fitting lenses 14 to 16 hours during the day the myopia decreases until vision is 20/30 or 20/40 without glasses. Repeated cycloplegic refractions during this period of reduced myopia show a loss of two or more diopters of spherical power. This period that the patient refers to as "good vision" without the necessity of any correction lasts about eight hours, and refraction during this time does not correct the vision to better than 20/30. There is no significant alteration in pachyometer readings, and the corneas appear normal on slit-lamp examination. It may be speculated that this transient decrease in myopia is due to an alteration of the refractive index of the cornea possibly influenced by a change in the water content.

TRANSIENT ALTERATIONS IN CORNEAL CURVATURE

Amano[31] studied the keratometer readings of 72 people before wearing corneal lenses and repeated these readings 3 to 6 months later after the lenses had been worn daily at least 6 hours per day. He found the corneas had become slightly steeper by an average of .034 and .025 mm. for the two meridians in the right eyes and .043 and .015 mm. for the left eyes. This is an insignificant alteration for a young age group with normally increasing myopia.

Studies of changes in the corneal curvature by the author are inadequate for conclusions about this as a factor in spectacle blur. The distortion of the mires of a keratometer by the corneal irregularities when corneal lenses are removed disturbs the accuracy of the readings. Observations of the fluorescein patterns of corneal lenses on rabbits show molding of the cornea to the lens. Initially, a lens placed on a rabbit cornea may have a well-fitting fluorescein pattern. Twenty-four hours later the thin rabbit cornea has molded itself to the shape of the lens with close contact between the cornea and the entire base curve of the lens which is more pronounced with tight lenses.

H. Detachment of the Retina

Ueno[32] has reported 6 cases of retinal detachment developing while wearing contact lenses among 297 cases of retinal detachment seen from 1956 to 1960. All of the 6 cases had high myopia. Ueno did not think these findings indicated any significant relationship between contact lens wear and detachment of the retina.

One patient in the author's series who had aphakia developed a retinal tear with a small vitreous hemorrhage while wearing a corneal lens. The corneal lens was discontinued, and the retina never became detached.

II. ADAPTATION OF THE EYE TO CORNEAL LENSES

The adaptation of patients to contact lenses is a routine experience in clinical practice and consists of several factors. Obrig and Salvatori,[33] Black,[8] and Dixon and Lawaczeck[34] have reported the decreased sensitivity of the cornea to touch in patients who habitually wear corneal lenses.

PSYCHOLOGICAL ADAPTATION

Psychological adaptation to corneal lenses is an important factor and involves decreasing anxiety about the foreign body on the cornea

and decreasing anxiety about loss of the lenses. There is also increasing skill in removal and insertion of the lenses. Many psychological factors of adaptation are beyond the scope of this study of the eye that wears the lens.

IMMEDIATE SENSORY ADAPTATION

When corneal lenses are applied to patients who have never worn lenses, there is normally an immediate foreign body sensation with lacrimation and epiphora. This subsides within a few minutes. At the end of thirty or forty minutes most patients have relaxed and tolerate their new lenses well. This is consistent with the experiments of Adrian and Zotterman[35] who found that there is a rapid decline in the rhythm of a sensory nerve impulse due to a steady stimulus. They postulated that this might be due to a decrease in the excitability of the end organs or to a gradual increase in their refractory period or both.

DELAYED SENSORY ADAPTATION

Routine measurements of the corneal sensation of all patients during the initial examination have been made with the Cochet and Bonnet aesthesiometer. This device uses a nylon yarn of variable length, .0113 sq. mm. in section, and was calibrated by the author on an analytical balance. The weight in milligrams of the pressure required to bend the yarn slightly was measured for various lengths. The corneas were tested peripheral to the edge of the pupil. The upper lid was tested in the center of the tarsal conjunctiva and near the lid margin on its conjunctival surface. The average measurements are recorded in Table 1 for patients before wearing lenses and later after becoming well adapted to daily corneal lens wear.

TABLE I. AESTHESIOMETER MEASUREMENTS (IN MILLIGRAMS) OF THE CORNEA, CENTER OF UPPER LID, AND CONJUNCTIVAL SURFACE NEAR THE LID MARGIN BEFORE AND AFTER ADAPTATION TO CORNEAL LENSES

	Cornea	*Center of tarsus*	*Lid margins*
Before wearing lenses	1.0	50.0	13.0
Fully adapted to daily lens wear	13.0	150.0	50.0

It is clear that there is a consistent decrease in corneal and lid sensitivity to touch with increasing adaptation to corneal lens wear. Daily sensitivity tests were made on a well-adapted control patient by discontinuing the lens on one eye. There was a gradual return to normal sensation to touch in the cornea and conjunctiva of the upper

lid on this eye within five days, while the opposite eye wearing the lens remained at the average adapted level. After bilateral daily lens wear was resumed, the sensation in the recovered eye was reduced to equal that of the adapted eye within three days. Patients in this group who have not previously worn corneal lenses and who begin with one hour per day and gradually increase their daily lens wearing time do not reach average sensory adaptation for two to four weeks. Some patients who are well adapted do not have equal sensory adaptation in the two eyes. Two patients have been observed to be well-adapted to daily corneal lens wear of 12 to 14 hours without change of sensory measurements on the cornea or lids.

Sensory measurements have been made on 2 patients with well-adapted eyes early in the morning before lenses were applied and repeated daily for one week 8 hours later the same day with no change in measurements. The intraocular tension may be measured easily on these patients with the Schiotz tonometer without topical anesthesia.

Six patients who were well adapted to daily corneal lens wear were found to have the usual decrease in corneal sensitivity in the area of the position of the lens. Sensitivity of the cornea in the area of a small crescent below the lens position had not changed from the normal sensation for this area.

PHYSICAL ADAPTATION

The adaptation of the eyes to corneal lenses by physical or morphologic changes in its structure has been considered. Microscopic studies of sections of corneas from 6 rabbits wearing lenses daily for three months have not shown any consistent variation from the appearance of control eyes other than the epithelial changes described. These sections were stained with hemotoxylin and eosin. Sections stained for morphologic changes in the nerve fibers have not been of any value in our laboratory, and further study is indicated. The possibility of trauma to the delicate nerve endings in the area of the basal cells of the corneal and conjunctival epithelium must be considered.

III. MICROBIOLOGY OF CONTACT LENS WEAR

A. Bacterial Infections and Bacteriology of the Eye with Corneal Lenses

The most serious and feared complication of corneal lens wear is secondary bacterial infection. This complication develops quickly and

accounts for most of the eyes lost or those with permanently decreased vision.

Payrau and Perdriel[36] reported bilateral ulceration of the cornea with hypopyon following continuous wear of contact lenses. A preliminary report of a survey by the Contact Lens Committee of the National Medical Foundation for Eye Care[37] revealed 4 eyes lost in this country due to contact lenses in addition to 468 corneal ulcers with 157 cases of permanent scarring. Wild[38] reported a case of an aphakic eye with iris prolapse that developed purulent endophthalmitis due to wearing contact lenses (presumably corneal lenses). Useful vision was lost in that eye. Bilateral purulent keratoconjunctivitis with corneal lenses occurred in one patient with aphakia in the author's series. Clouding of both corneas reduced the vision to 20/200 in each eye, but the lenses were discontinued, and after hospitalization the corrected vision returned to normal three months later. It is interesting that the patient had obtained the lenses in another city two years previously and had used local anesthetic drops for comfort at least every hour daily since that time with no complications until the bilateral purulent infection suddenly appeared.

EXPERIMENTAL STUDIES

During the initial studies of pathologic changes in the eyes of 21 rabbits and 10 dogs wearing a corneal lens on one eye with the opposite eye reserved for a control, secondary purulent keratoconjunctivitis developed in 9 rabbit eyes wearing the lenses. No infection developed in the control eyes. Secondary infection was prevented in 7 other eyes by daily antibiotics, and 4 other animals were sacrificed for pathologic examination within 3 days of lens wear before infection developed. Two rabbits wore a lens constantly without the appearance of infection and without prophylatic antibiotics. Six rabbits wearing a lens on one eye fewer than 16 hours a day for 8 months without medication failed to develop secondary infections.

Cultures on blood agar made daily on rabbits were negative until an acute purulent infection suddenly appeared. At this time a growth of gram negative rods was the most common finding. None of these corneas perforated in spite of continued lens wear in the presence of the infection. After the corneas were well vascularized, the infection subsided spontaneously without treatment. Vascularization of the cornea in these animals was accelerated by the infection and developed much more rapidly than in the human. All 10 of the dogs developed a certain amount of purulent discharge. Cultures were not made on these.

In order to study other pathologic changes not complicated by infections, it was found that the daily local use of tetracycline ointment would prevent this complication. The ointment would cling to the cilia and collect under the lenses where it seemed to reduce irritation rather than increase it. Various antibiotic drops were ineffective in the prophylaxis of infections in these experimental animals when used only once daily.

BACTERIOLOGY OF THE HUMAN EYE WEARING CORNEAL LENSES

Winkler and Dixon[39] found that cultures from the lids and conjunctivas of more than 50 patients made before wearing corneal lenses and later repeated after lenses had been worn for a month or more were not appreciably altered. *Staphylococcus albus* and diphtheroids were recovered most frequently. The occasional finding of species of *Sarcina, Achromobacter, Flavobacterium, Pseudomonas,* and *Aerobacter* was no more frequent while wearing lenses than before.

Clinical experience and experimental studies indicate that contact lenses should not be worn in the presence of bacterial infections of the eye or adnexa.

B. Bacterial Flora in Contact Lens Containers

Patients store their corneal lenses in a wide variety of containers. During the night some patients place their lenses in "soaking kits," others leave them in tap water, some are placed in "screw cap" containers without solution, and some are placed in a variety of small containers with detergents or other commercial solutions advertised as antibacterial. A preliminary report on Pseudomonas contamination of contact lens containers has been made by Dixon, Lawaczeck, and Winkler.[40]

Chemical sterilization is difficult under the best of conditions even in the operating room. It is a delusion to expect the public to repeatedly place contaminated lenses in 2- or 3-cc. containers of any non-irritating solution at room temperature and expect it to maintain sterility or even prevent growth of pathogenic organisms.

PROCEDURE

In order to study the problem of reasonable sanitation of contact lens containers it was necessary to try several methods. Routine cultures were made on the lens containers of patients who returned

to the office for follow-up visits. After serious contamination was found consistently in 12 to 15 containers with one method of lens storage, a new method would be tried and cultures continued. Winkler and Dixon[39] found that cultures from lens containers, even those appearing to be dry revealed a variety of bacteria, primarily gram negative rods and staphylococci. While *Achromobacter* was encountered most frequently, *Pseudomonas* was recovered from over 10 percent of the 85 lens containers cultured.

Patients were advised to leave the lenses in the containers dry. Cultures of these containers thought to be dry were found to be contaminated with the same organisms. Further study indicated that patients habitually removed the lenses from their eyes and stored them moist in air tight containers with accumulated organic material which became a culture medium. The .6 percent protein content of the tears apparently becomes concentrated by evaporation and in the presence of moisture, bacteria flourish. One such patient in this study developed a pseudomonas corneal ulcer near the limbus which fortunately healed with a small scar.

Plastic mailing containers with a 5-mm. hole punched in each side for ventilation were then distributed to patients. These containers grew bacteria because the lens rested snugly against the wall of the containers and maintained moisture at the area of contact around the periphery of the perforation.

A new plastic container which holds the lens resting at its edge and has ventilating slots on each side has had the lowest contamination rate. This container may be boiled. Patients will occasionally seal a ventilated container in an envelope or carry it in a small change purse. Cultures from four of these containers had a higher rate of contamination and bacterial growth.

Several conclusions may be made from these studies of lens storage:

1. Absolute sterility of lenses and containers is probably not attainable in their routine use by the public. The fingers, lids, lenses, and containers suspend a few organisms that escape ordinary cleansing.

2. The prevention of conditions that favor bacterial growth during storage is necessary and is attainable.

3. At the time the lenses are removed from the eyes they should be cleaned of lid secretions, blotted dry, and placed in a well-ventilated container in a ventilated area. Containers should be cleaned every few days preferably by boiling.

4. Before the lenses are placed on the eyes, the hands should be washed, the lenses cleaned, and if the patient prefers, lubricated with

any sterile commercial preparation for that purpose. Ophthalmologists should be aware that a thin film of liquid on the lenses will have disappeared after a few blinks of the lids.

5. Those patients who wish to soak their lenses can use a fresh supply of ½ pint of tap water each night with proper cleaning of the lenses before and after use.

C. Corneal Herpes Simplex Infections

The experimental studies of Lawaczeck, Francis, and Dixon[41] on 36 rabbits indicate that this complication is to be expected occasionally in humans and that the clinical appearance and course of the infections is considerably altered. In rabbits it was found that the severity of the infection and resulting scar of the stroma increased in those eyes wearing corneal lenses when compared with control eyes. It is not certain whether the lens holds a greater concentration of the virus in contact with the cornea, or whether the mechanical trauma to the surface epithelium gives the virus better access to the cells. Metabolic factors could also be involved.

The appearance of corneas infected experimentally while wearing a corneal lens differs from the control corneas by the development of large geographic figures rather than dendritic figures with scattered stippling.

The experimental use of corneal lenses on old healed herpetic scars in rabbits did not reactivate the virus infection but did reactivate vascularization.

Because of the experimental increase in severity of herpes simplex corneal infections on eyes wearing corneal lenses and the reactivation of the vascularization in old healed herpetic scars, it is concluded that contact lenses are contraindicated in patients with a history of previous infections.

D. Fungus Infection of the Cornea Complicating Corneal Lens Wear

Moniliasis of the cornea was reported by Mendelblatt[42] who described it as having a bread crumb appearance. He pointed out that this fungus grows best on moist and macerated tissue.

Dr. T. A. Makley, Jr. has contributed sections of an enucleated eye received by him for pathologic examination from Dr. H. E. Possner which have *Candida albicans* in the corneal tissue (Figure 8). A 39-year-old woman who had worn contact lenses daily since 1947 developed several corneal lesions in July, 1961. Scleral lenses were

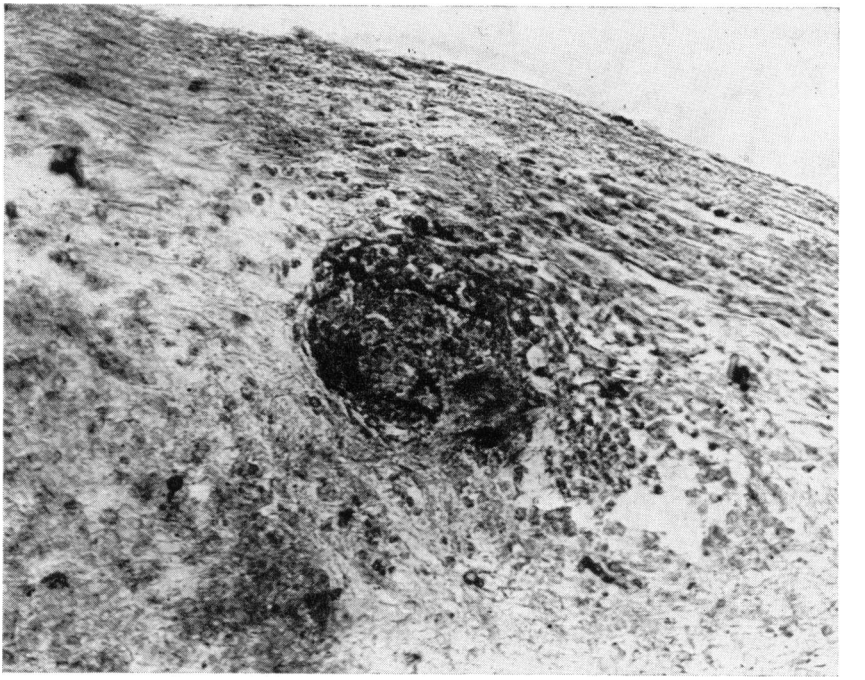

FIGURE 8. *Candida albicans* IN PERFORATING CORNEAL ULCER COMPLICATING HUMAN CONTACT LENS WEAR (GRIDLEY STAIN)

started in 1947 and changed to corneal lenses in December, 1950. The lesions were described as small, deep, whitish gray, near the center of the cornea. Some stained with fluorescein while others did not. The lesions healed in about four weeks after the lenses were removed and the eye treated with atropine and local steroids. Contact lens wear was started again, and in September, 1961, the same eye had a minor injury by a child's hand. October 24, 1961, she was admitted to the hospital for a more severe ulceration of the center of the cornea which perforated while a culture was being taken. Cultures were negative for bacteria and fungi. After an unsatisfactory clinical course including therapy with steroids, antibiotics, and copper sulfate, the eye was enucleated in December, 1961. At the time of enucleation cultures were positive for *Candida albicans.*

IV. ALTERATIONS IN THE INTRAOCULAR PRESSURE

The influence of corneal lenses on intraocular pressure dynamics is not clear, and there is a paucity of basic research in this area.

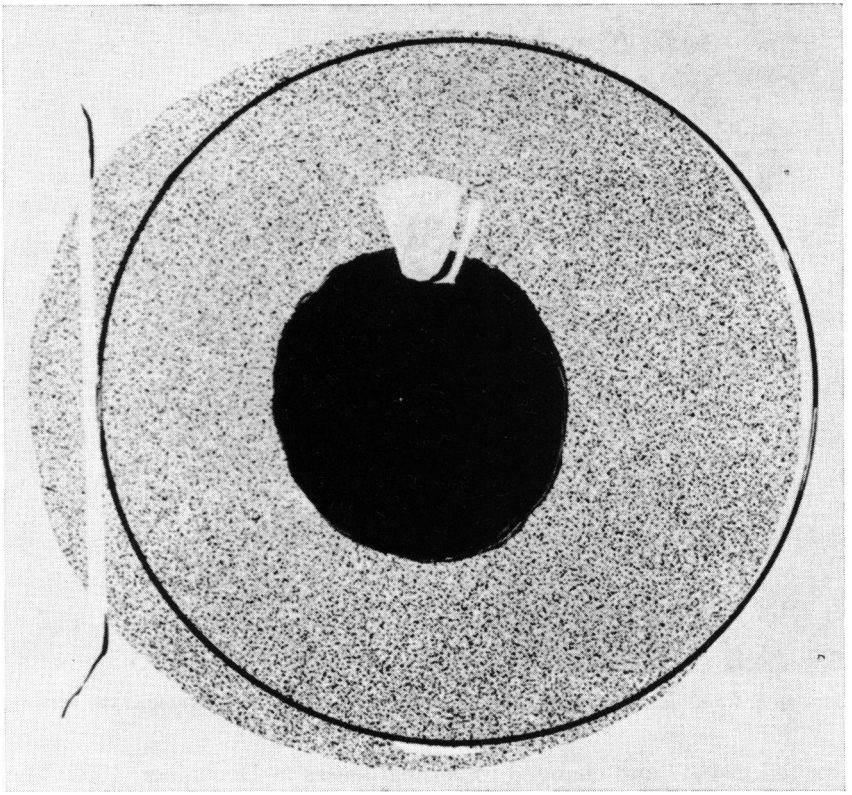

FIGURE 9. VERTICAL BEAM OF LIGHT FROM SLIT-LAMP ON CORNEA NOT IN CONTACT WITH CORNEAL LENS MARGIN IS NOT BENT TOWARD PUPIL AND DOES NOT CAUSE GLARE

Ascher[43] reported the reversal of flow in the aqueous veins caused by compression from the rim of scleral lenses. Huggert[44] reported a transient increase in intraocular pressure in 25 of 33 eyes caused by scleral lenses. It is debatable whether these findings would be applicable to corneal lenses. It is possible that the increased massaging effect of corneal lenses with blinking of the lids would facilitate the outflow of aqueous; however, any conclusions without further studies would be premature.

A large aqueous vein has been observed frequently on one patient while wearing a corneal lens. The corneal lens does not affect the circulation in the aqueous vein which is on the nasal side of the cornea except when the patient rotates the eye in the temporal direc-

tion. The lens is then pushed to the nasal side of the cornea by the lids, and as the corneal lens margin gently rests against the limbal vessels, the flow in the aqueous vein stops and does not flow in any direction until the eye resumes the primary position. This same rotation of the eye without the lens on the cornea does not affect the aqueous vein. An aqueous vein on another patient is not influenced at all by a corneal lens.

V. GLARE CAUSED BY CORNEAL LENSES

Discomfort from glare while wearing corneal lenses is a common complaint of patients. This has been studied by Dixon and Lawaczeck[45] and is included in this report because irritation of the eye by the lens has been implicated as the cause. The glare is present immediately

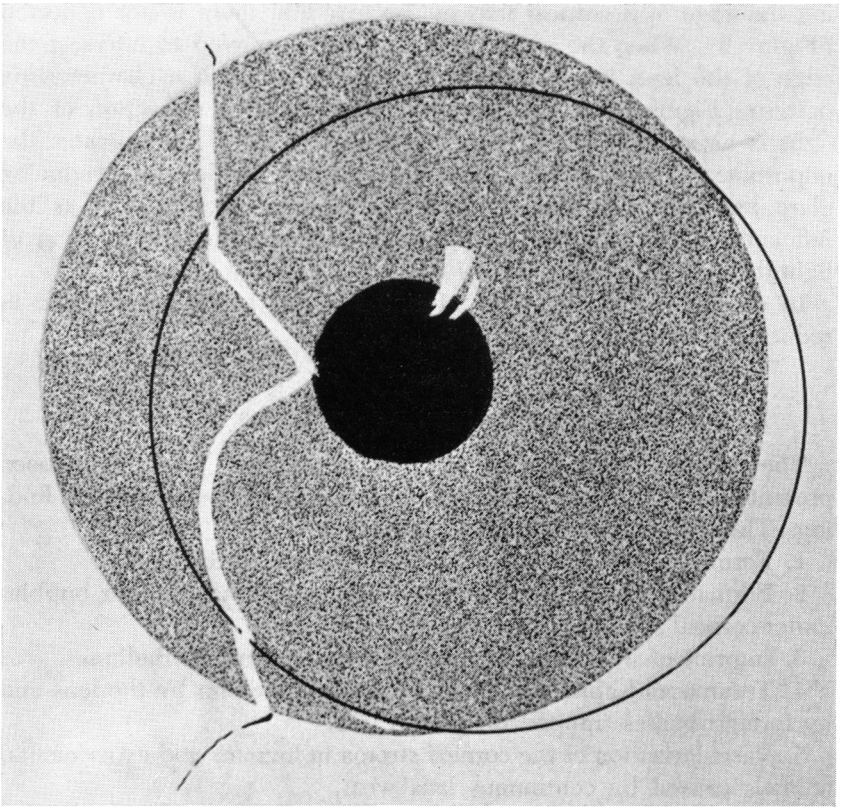

FIGURE 10. VERTICAL BEAM OF LIGHT FROM SLIT-LAMP CROSSING CORNEAL LENS MARGIN IS BENT TOWARD PUPIL AND CAUSES GLARE

after lenses are applied and immediately relieved when the lenses are removed. It was not altered by the use of topical anesthesia on a control cornea while wearing the lenses. These tests suggest that local irritation or a reflex from the cornea are not important factors. Rabbits wearing corneal lenses experimentally are observed to habitually sit in their cages with the eye wearing the lens directed away from a window or other light source.

One theory advanced by some is that the corneal plastic lenses transmit more light than spectacle lenses. This fails to explain why glare is absent in persons without corneal lenses or spectacles but does appear immediately when corneal lenses are applied.

Dixon and Lawaczeck[45] demonstrated a source of glare by the use of a narrow perpendicular slit-lamp beam on the cornea. The narrow beam of light interesecting the periphery of the cornea but not touching the edge of a corneal lens or the tear film there is not deflected (Figure 9). When the same slit-lamp beam is moved to intersect the edge of the lens, it is deflected toward the pupil in a characteristic pattern (Figure 10). The pattern of deflection or refraction of the light is also made by the lens suspended in air. This indicates the importance of the base in prismatic edge of the lens in producing glare but does not exclude other contributing factors such as the influence of the tear fluid at the lens margin or the transmission of light through the plastic.

In clinical practice the use of tinted lenses has been effective in reducing glare.

VI. SUMMARY AND CONCLUSIONS

The changes in the eye due to wearing contact lenses have been presented with a correlation between clinical and experimental findings. The following subjects were discussed:

1. Formation of wrinkled furrows in corneal epithelium.
2. Formation of dimples in corneal epithelium that trap air bubbles under corneal lenses.
3. Imprint of a corneal lens made in the corneal epithelium.
4. Trauma and abrasions to the corneal epithelium by the lens and by foreign bodies trapped under the lens.
5. Vascularization of the corneal stroma in humans and experimental animals caused by continuous lens wear.
6. Transient conjunctival hyperemia due to corneal lenses.
7. Increase in the leucocyte count of the tear fluid.

8. Debatable alterations of the glycogen content of the corneal epithelium.

9. Spectacle blur caused by tissue changes in the cornea and possible alterations in the corneal refractive index.

10. Descrease in corneal and lid sensitivity.

11. Bacterial flora of the lids and conjunctiva unchanged by wearing corneal lenses.

12. Danger of secondary bacterial infections and ulceration of the cornea.

13. High rate of wet or moist contact lens containers acting as culture media for pathogenic bacteria.

14. Aggravation of corneal herpes simplex infections.

15. Case of corneal infection with *Candida albicans.*

16. Inadequate knowledge of influence of corneal lenses on intraocular tension.

17. Mechanism of glare due to light bent toward pupil by base in prismatic edge of lens.

18. Relatively small number of cases of permanent eye damage compared with large number of people wearing contact lenses successfully.

The large number of people wearing corneal lenses has demonstrated that usually they can be worn safely with care. Many of the complications that have occurred have been preventable with proper understanding and care by ophthalmologists and their patients.

The multitude of physiologic and pathologic changes in the eye caused by wearing contact lenses demonstrate clearly that this is a medical problem. The excellent results with the use of corneal lenses in patients with aphakia and young people with myopia have made the care of these patients a permanent part of the practice of ophthalmology.

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