VOLUME IV

# Current concepts in OPHTHALMOLOGY

Editor

FREDERICK C. BLODI, M.D.

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**FREDERICK C. BLODI, M.D.**

Professor and Chairman of The Department of Ophthalmology,
University of Iowa College of Medicine,
Iowa City, Iowa

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# Preface

This volume is the second one emanating from The Department of Ophthalmology at Iowa. Here again we have tried to bring together some of the newer developments in ophthalmology that are not easily available to the general ophthalmologist. We have emphasized in this volume certain newer diagnostic methods and have incorporated some original chapters, together with some review chapters.

This volume is larger and has more contributions than the previous one. This is due to two factors. First of all, our full-time staff has been considerably enlarged during the last 2 or 3 years. A number of members have joined the faculty. Some of them have been recruited from Europe, and others have developed in our own training program. This has created a most active and productive staff. The second factor is that a larger number of our residents and fellows have been involved in research projects of general interest. They have greatly contributed to this new volume.

Again we wish to express our thanks for the continued support of the National Eye Institute and above all to the generous support of the Neurosensory Program Project. The Neurosensory Program Project is under the direction of Dr. M. W. Van Allen, Professor of Neurology, and is Grant No. NS03354-13 of the National Institute of Neurological Diseases and Stroke. Because of this close association with the Department of Neurology and the Division of Clinical Psychology, the large majority of these contributions are again slanted toward medical ophthalmology, and few of them are concerned with the surgical aspects of our specialty.

The acceptance of the previous volume has been most encouraging, and we hope that the ophthalmic community will find the present volume interesting and instructive.

**Frederick C. Blodi**

# Contributors

From The Department of Ophthalmology, University of Iowa College of Medicine, Iowa City, Iowa

**Lee Allen**
Research Associate

**Frederick C. Blodi, M.D.**
Professor and Chairman

**William M. Bourne, M.D.**
Instructor

**Thomas C. Burton, M.D.**
Associate Professor

**Jonathan Chua, M.D., Ph.D.**
Resident

**Gerhard W. Cibis, M.D.**
Resident

**William N. Clarke, M.D.**
Fellow in Pediatric Ophthalmology

**John B. Constantine, M.D.**
Resident

**James R. DeKock, B.S.**

**Bernd Ellerhorst, M.D.**
Special Fellow in Inflammatory Diseases of the Eye

**Richard T. Falter, M.D.**
Instructor

**Sohan Singh Hayreh, M.B., M.S., F.R.C.S., Ph.D.**
Professor

**Noah Hershkowitz, Ph.D.**
Associate Professor of Physics

**Chase P. Hunter, M.D.**
Instructor

**Thomas P. Keenan, M.D.**
Instructor

**Hansjoerg E. Kolder, M.D., Ph.D.**
Professor

**Jay H. Krachmer, M.D.**
Assistant Professor

**Frederick A. Mausolf, M.D.**
Instructor

**Edward S. Meek, M.D.**
Professor

**John H. Mensher, M.D.**
Assistant Professor

**John C. Nelson, M.D.**
Instructor

**Lance E. Olson, M.D., Ph.D.**
Resident

**Karl C. Ossoinig, M.D.**
Associate Professor

**Charles D. Phelps, M.D.**
Assistant Professor

**Raymond D. Ricklefs**
Research Associate

**William E. Scott, M.D.**
Assistant Professor

**H. Stanley Thompson, M.D.**
Associate Professor

**Russell M. Warren, M.D.**
Instructor

**Robert C. Watzke, M.D.**
Professor

**Thomas A. Weingeist, M.D., Ph.D.**
Resident

**Stephen H. Wolken, M.D.**
Resident

**Ira G. Wong, M.D.**
Heed Fellow in Experimental Pathology

# Contents

Chapter 1

# Aseptic corneal erosions and ulcerations

Jay H. Krachmer, M.D.

The ophthalmologist is often confronted with the problem of corneal defects that fail to epithelize properly. Most lesions are not associated with active infectious agents. Treating these aseptic corneal erosions (epithelial defects) and ulcerations (epithelial and stromal defects) is most successful if (1) the lesion is properly diagnosed as an aseptic problem rather than a bacterial, fungal, or viral ulcer, (2) factors that cause corneal erosions and ulcerations and prevent healing are recognized, and (3) the ophthalmologist has a basic knowledge of anterior corneal anatomy and mechanisms of epithelization.

To meet these criteria, methods of treatment will be preceded by a discussion of etiologic factors, slit-lamp characteristics of these lesions, and basics of epithelial repair. *Punctate* erosions associated with corneal dry spots,[1] with staphylococcal blepharokeratoconjunctivitis, with viral diseases, and with many other disorders[2] will not be discussed in this chapter.

## CORNEAL EPITHELIAL ANATOMY AND REPAIR

The corneal epithelium consists of approximately five layers of cells.[3,4] Three basic types of cells are seen: the surface cells are broad and flat; the basal cells are columnar; the cells that are squeezed in between surface and basal cells are curved and are called "wing" cells. Beneath the basal cells, but anterior to Bowman's membrane, is a basement membrane. Small attachments called *hemidesmosomes,* which are seen between the basal cells and the basement membrane by electron microscopy, are thought to contribute to the adherence of the epithelium to the basement membrane.

Epithelial repair consists of epithelial cell sliding from adjacent epithelium, thickening by cell division, and adherence to underlying basement membrane.

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Figures in this chapter are of patients seen while I was doing a Heed Ophthalmic Foundation Fellowship with Dr. Peter R. Laibson, Cornea Service, Wills Eye Hospital and Research Institute, Philadelphia.

Khodadoust and co-workers[5] showed that, in the rabbit, the adherence of new epithelium depends on the presence of the underlying basement membrane. When the basement membrane is intact and just the overlying epithelium is removed, new epithelium becomes adherent in 1 week. However, when the basement membrane is removed, the reepithelized area is not firmly adherent until the new basement membrane and the basal cell–basement membrane attachments are present. This took several weeks in the rabbit.

## SLIT-LAMP CHARACTERISTICS

What clues may indicate that a corneal ulcer is not an active bacterial, fungal, or viral lesion?

Usually a superficial corneal defect with minimal surrounding or underlying infiltration (Fig. 1-1) is either an aseptic corneal erosion or an active herpes simplex ulcer. A dendritic or geographic pattern would implicate an active herpes virus. When a "clean" lesion has ragged edges from loose epithelium (Fig. 1-2) or is discretely round, oval, or square (Fig. 1-3), it is more likely to be an erosion. Active bacterial ulcers (Fig. 1-4) and those caused by a fungus have significantly more infiltration and edema surrounding them, extending to most or all of the cornea. In aseptic lesions, on the other hand, the remainder of the cornea is frequently uninvolved. Difficulty in differentiation arises in cases in which an erosion is accompanied by moderate or marked infiltration. In some cases of erosion, a hypopyon can be seen. If the infiltration around the defect

**Fig. 1-1.** Recurrent corneal erosion with minimal surrounding and underlying infiltration.

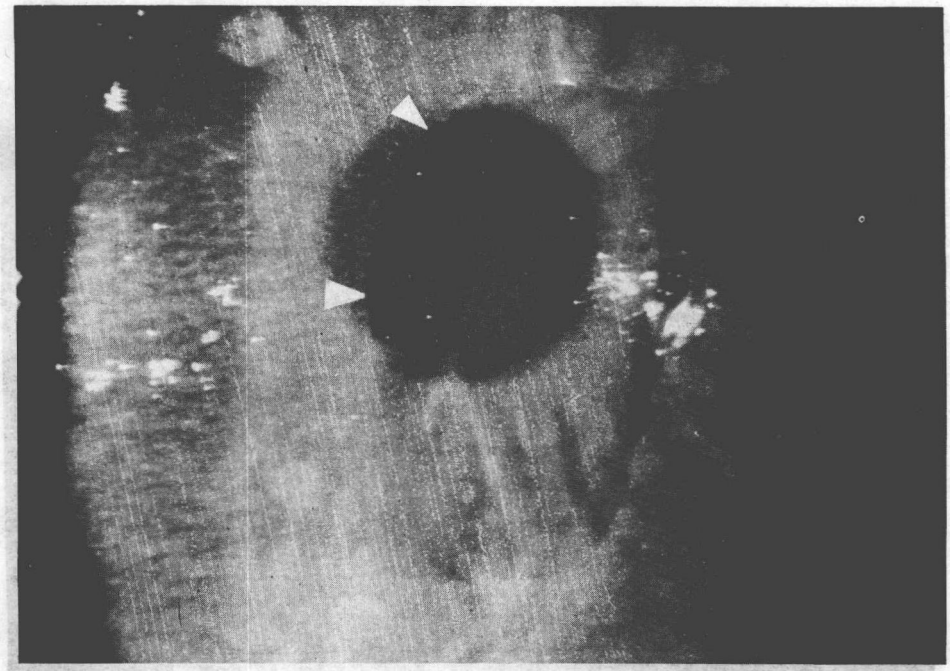

**Fig. 1-2.** Recurrent corneal erosion with a ragged edge and minimal infiltration.

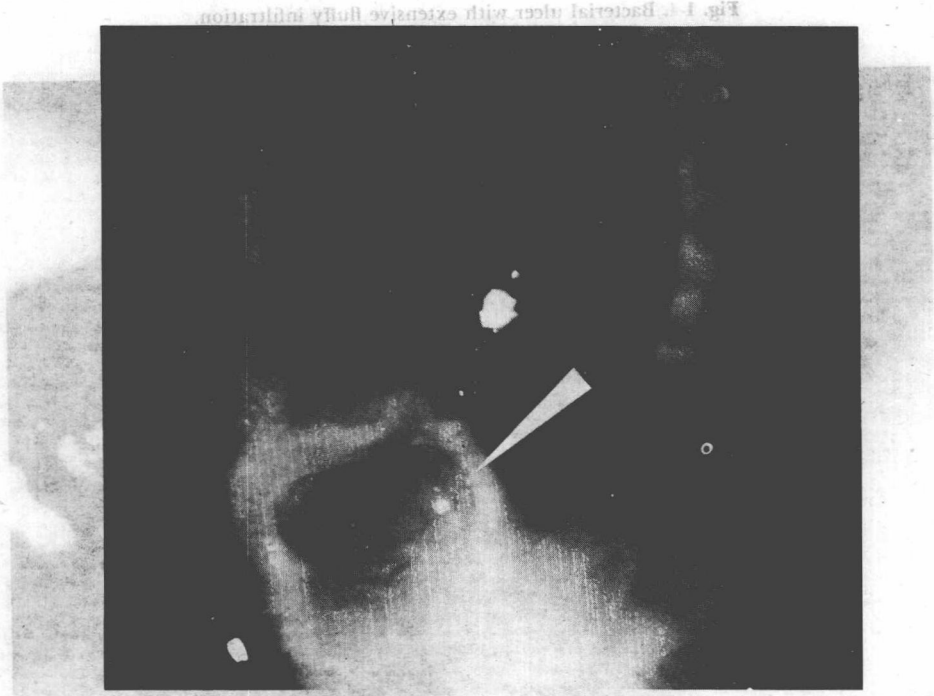

**Fig. 1-3.** Metaherpetic ulcer with minimal surrounding and underlying infiltration. Stromal scars from previous dendritic lesion are seen above.

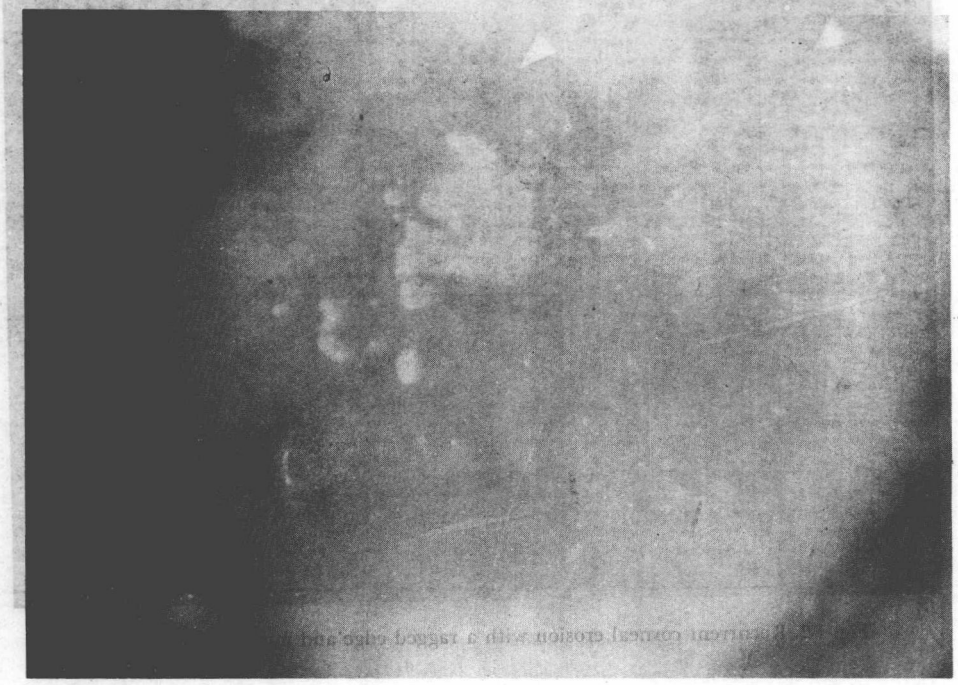

**Fig. 1-4.** Bacterial ulcer with extensive fluffy infiltration.

**Fig. 1-5.** Recurrent erosion with infiltration mainly limited to borders of lesion.

is grayish white and discrete (Fig. 1-5), rather than more yellow and radiating (Fig. 1-4), an erosion is a more likely possibility than a bacterial or fungal ulcer. When an aseptic case is complicated by secondary infection or when a long-standing erosion has fluffy and generalized infiltration and edema, differentiation by slitlamp appearance alone can be very difficult. Further clues to etiology may be gained from scrapings and culture and from knowledge of underlying pathologic conditions that are associated with aseptic corneal defects.

## ETIOLOGIC FACTORS

The variety of corneal insults characterized by aseptic lesions can provide the examiner with challenging diagnostic situations.

### Bullous keratopathy

Bullous keratopathy from any cause, such as Fuchs' dystrophy and glaucoma, is often complicated by the rupturing of a bulla with a resultant erosion. The diagnosis is usually not difficult because of the surrounding corneal edema and endothelial changes.

### Trauma

Corneal trauma can be followed days, weeks, months, or years later by variable episodes of erosions. The symptoms in recurrent erosions after trauma can range from barely noticeable to disabling. Recurrent erosions seem particu-

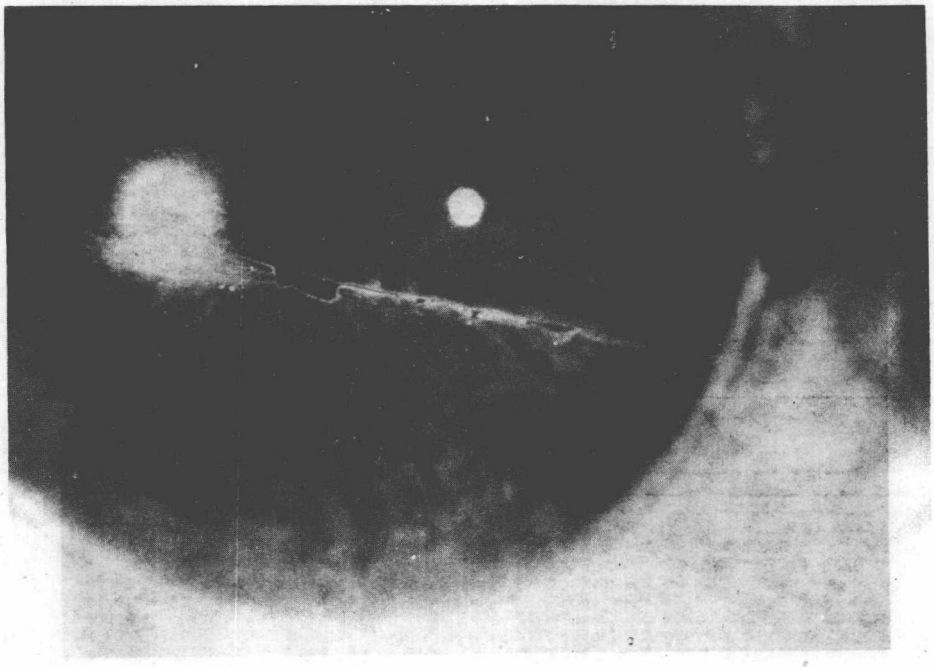

**Fig. 1-6.** Corneal abrasion from a piece of paper.

larly to follow shearing corneal abrasions such as those from paper (Fig. 1-6), fingernails, tree branches, metal edges, or cosmetic applictors. The history of trauma is undoubtedly helpful when it is accurate. Often, however, the patient forgets which eye was involved, when it was traumatized, and how it was injured.

During an episode of a recurrent erosion, the patient may present with a diffuse swelling of the upper lid (Fig. 1-7). Visual acuity depends on the location and extent of the erosion and is usually significantly reduced. There is an area of loose, edematous epithelium (Fig. 1-8), beneath which the anterior stroma shows a brawny edema (Fig. 1-9). The epithelium surrounding the erosion is loose (Fig. 1-10). After reepithelization many clear, semiopaque, and opaque epithelial spots are seen in the area of former erosion (Fig. 1-11). Most of these intraepithelial inclusions are not seen after a few weeks. However, they may remain long after the erosion. They become "markers" of previous erosion. Their eruption on the epithelial surface may be responsible for the more minor attacks of foreign-body sensation. These cysts are beautifully described both clinically and pathologically by Bron[6] and Tripathi.[7]

The fact that recurrent erosion typically occurs after the eyes have been closed have been attributed to hypotonic tears producing epithelial edema.[8] As the lids open, the loose epithelium is torn. Electron microscopy of a case of posttraumatic recurrent erosion showed loss of both basement membrane and associated hemidesmosomes. Goldman and associates[9] thought that recurrent breakdown of the epithelium in such cases might be due to poor adhesion of the epithelium to its basement membrane.

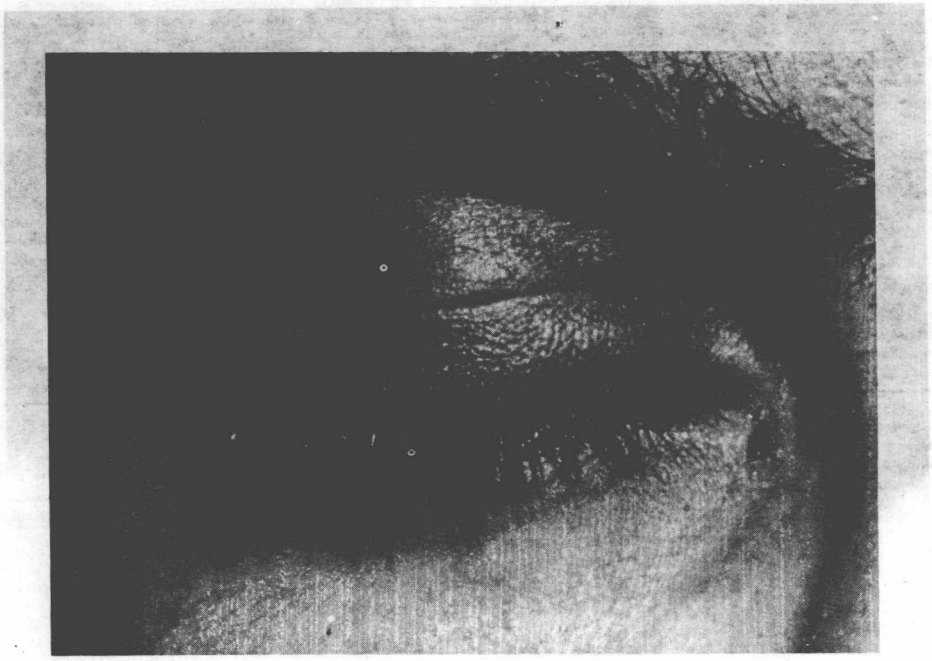

**Fig. 1-7.** Typical lid edema in a patient with a recurrent erosion.

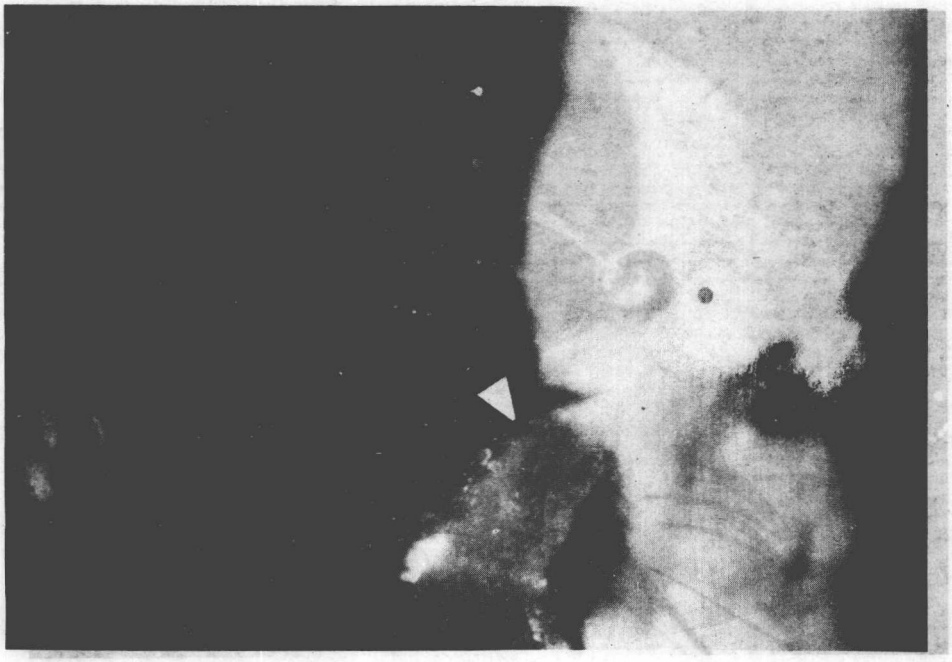

**Fig. 1-8.** Recurrent erosion with loose edematous epithelium.

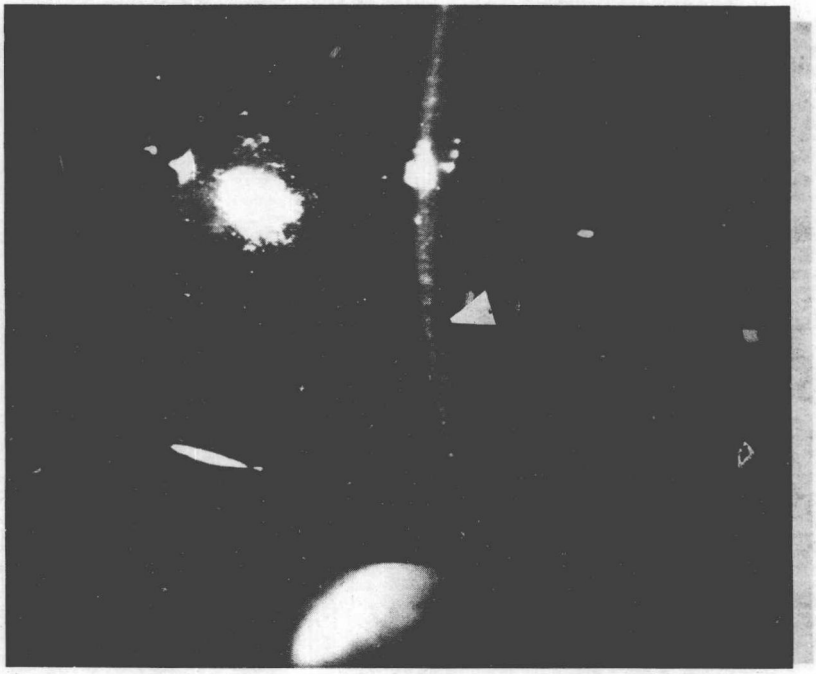

**Fig. 1-9.** Anterior stromal edema beneath a recurrent erosion.

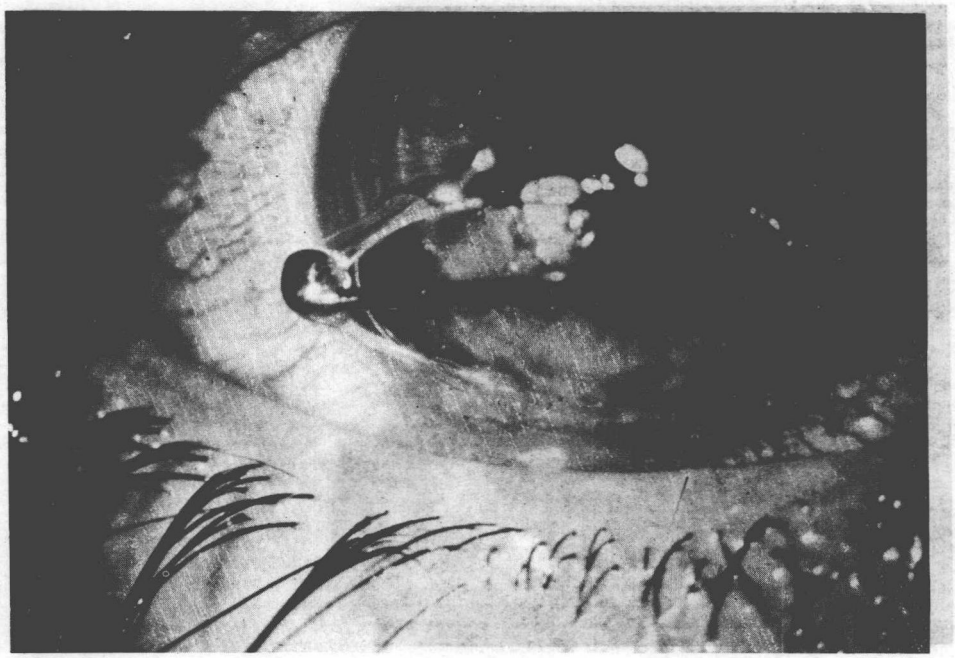

**Fig. 1-10.** Chalazion curette elevating loose epithelium surrounding a recurrent erosion.

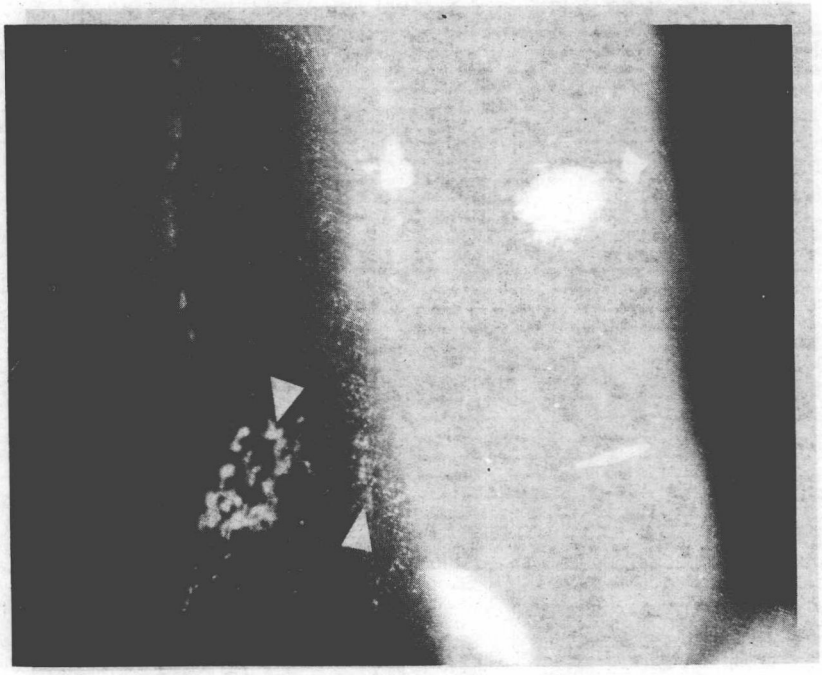

**Fig. 1-11.** Intraepithelial "markers" of a healing erosion.

## Corneal dystrophies

Recurrent corneal erosion is known to accompany several corneal dystrophies. Fuchs' dystrophy was mentioned earlier. Stromal dystrophies and anterior membrane (epithelium, basement membrane, Bowman's layer) dystrophies also produce erosions. Although the epithelium in macular and granular dystrophies can erode, lattice dystrophy is probably the most common of the stromal dystrophies to produce such defects recurrently. The Reis-Bücklers anterior membrane dystrophy is characterized by recurrent erosions persisting until the third or fourth decade. Basement-membrane defects and loss of hemidesmosomes are thought to contribute to recurrent erosions in both early[10] and later[11] cases.

The map-dot-fingerprint corneal dystrophy can also produce recurrent erosions.[12] In 1950 Guerry[13] described what he referred to as *fingerprint-like lines* in the corneas of two patients. Dotlike changes were first described by Cogan and colleagues[14] in 1964 and have since been referred to as *Cogan's microcystic corneal dystrophy*. One year later Guerry[15] reported maplike corneal opacities. Further corneal findings, "blebs" and "nets," were added in 1971 by Bron and Brown.[16] A series of 35 patients with combinations of map, dot, and fingerprint changes was described by Trobe and Laibson,[12] who emphasized the frequency of the condition and the high incidence of recurrent erosions.

Maplike opacities can best be viewed with a wide slit-lamp beam at an

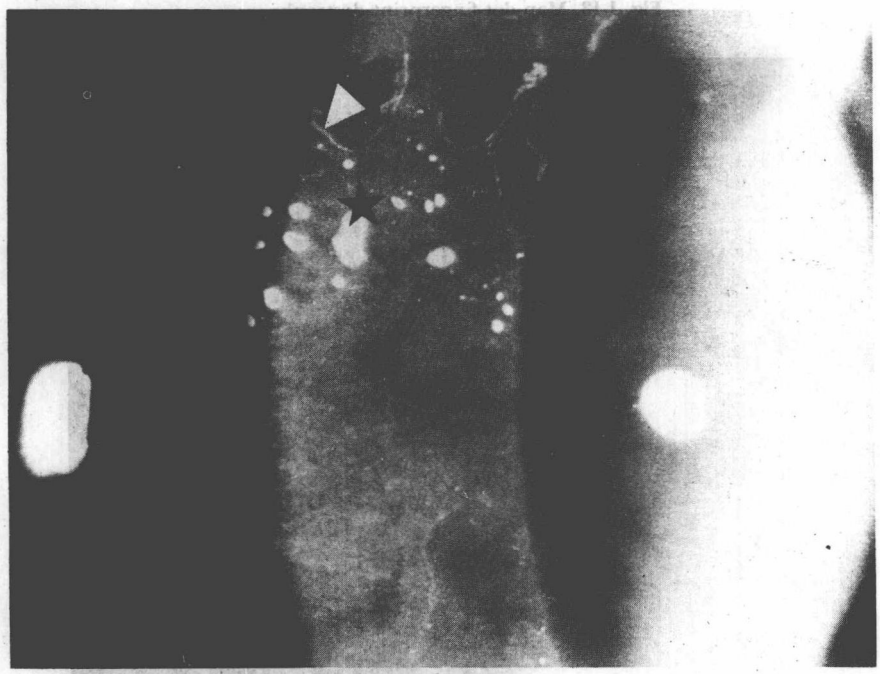

**Fig. 1-12.** Map-dot-fingerprint dystrophy with maplike (arrow) and dotlike (star) configurations. Note that the maps are actually the borders of intraepithelial sheets of tissue and that the dots are found only in the areas of the sheets.