Corneal abrasion is the most common ocular complication in surgery.1–5 Injury is confined to the epithelial layer of the cornea and typically heals within 72 hours. However, this is of little consolation during the initial 24 hours of intense discomfort. Within hours of surgery, the patient experiences exquisite pain, tearing, blurry vision, and photophobia in the affected eye. These symptoms are alarming to both patient and surgeon, neither of whom expected eye pain to be a major postoperative issue.

Once an abrasion is diagnosed, treatment should minimize pain and prevent infection without negatively impacting wound healing. Traditionally, conservative pain control (e.g., oral analgesics, topical nonsteroidal antiinflammatory drugs/cycloplegics, patching) has been favored for fear that topical anesthetics slow the healing process, leading to secondary infection, ulceration, and scarring. A critical look at old and new evidence suggests that this approach is flawed, and topical anesthetics can provide significant pain relief without delaying reepithelialization.

Unfortunately, outdated treatment guidelines persist in the literature.6–11 The goal of this review is to update the prevention and treatment of perioperative corneal abrasion to improve surgical patient care.

ANATOMY AND PHYSIOLOGY

The cornea plays a vital role in normal vision through light refraction and light transmission.12 It forms the anterior one-sixth of the globe and has five layers (Fig. 1). The surface layer, the epithelium, is four to six cell layers thick. The cornea contains the most richly innervated tissue in the body, with nerve terminal density 300 to 400 times greater than skin or teeth.12,13 All corneal stimuli (mechanical, chemical, and thermal) are perceived as painful. When an abrasion occurs, nerve endings lack cover from the epithelial surface, increasing their sensitivity. Opening or closure of eyelids and chemicals in tears are among the various stimuli that cause constant discomfort. Intraepithelial nerve terminals detect cell injury and changes in cell shape and volume, and effect physiologic response with the release of neurotrophic peptides, signaling molecules, and inflammatory mediators.

Reflex tearing occurs when noxious stimuli cause activation of secretory cells of the lacrimal glands, conjunctiva, and corneal epithelium to produce aqueous and mucous tear components. In contrast, the lipid component of tears is presecreted into meibomian gland ducts and passively released on contraction of pretarsal orbicularis oculi fibers during blink. The three-layer precorneal tear film is important for maintaining the health and function of the corneal surface (Table 1). It minimizes friction and dryness on the corneal surface, both of which can cause epithelial slough (desquamation). In addition, it is important for corneal oxygenation and protects against infection. The tear film evaporates while...
the cornea is exposed and is renewed by blinking. Factors that increase tear film stability (increased viscosity, increased lipid component) decrease the rate of evaporation. Break-up time, or time to tear film rupture, is a quantitative measure of stability.

The cornea is avascular. The central cornea receives atmospheric oxygen dissolved in the precorneal tear film, and the peripheral cornea is supplied with oxygen from anterior ciliary vessels. The cornea is very sensitive to interruptions in oxygen delivery and becomes edematous when ischemia is present. Edema makes a dry corneal surface more prone to desquamation, resulting in abrasion (more below).

Eyelid closure shields the cornea from the external environment and renews the tear film. Tonic contractions of the orbicularis oculi maintain eyelid closure during normal sleep. If lid closure is incomplete (lagophthalmos), the corneal surface is prone to drying out. Bell phenomenon, if present, provides another mechanism of protection whereby, on eyelid closure, the globe rotates upward, moving the cornea under the upper lid.

Normal epithelium undergoes constant renewal, turning over every 7 to 10 days. The superficial squamous epithelial cells are continuously shed and replaced by cells that migrate upward from the mitotically active basal cell layer. The basal layer is replaced by means of progenitor cells that migrate centripetally from the limbus. Injury to the epithelium disrupts the normal balance between slough and proliferation. Cell proliferation and migration are increased to resurface the defect and restore structural integrity of the layer. All of these physiologic factors play a vital role in protecting and supporting the integrity of the corneal epithelium (Table 2).

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**Table 1. Precorneal Tear Film Layers**

<table>
<thead>
<tr>
<th>Layer</th>
<th>Secreted By</th>
<th>Regulation</th>
<th>Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lipid (outer)</td>
<td>Meibomian glands</td>
<td>Blink</td>
<td>1. Prevents evaporation of aqueous layer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2. Increases surface tension</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3. Eyelid lubricant</td>
</tr>
<tr>
<td>Aqueous (middle)</td>
<td>Main and accessory</td>
<td>Neural control</td>
<td>1. Corneal epithelium oxygenation</td>
</tr>
<tr>
<td></td>
<td>lacrimal glands</td>
<td></td>
<td>2. Provides smooth optical surface</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3. Irrigates debris</td>
</tr>
<tr>
<td>Mucin (inner)</td>
<td>Goblet cells in conjunctiva</td>
<td>Neural control</td>
<td>4. Immune defense</td>
</tr>
<tr>
<td></td>
<td>Squamous epithelial cells</td>
<td></td>
<td>Makes epithelium hydrophilic</td>
</tr>
</tbody>
</table>

---
EPITHELIAL INJURY AND WOUND HEALING

An acute defect of the corneal epithelium (i.e., an abrasion) typically undergoes complete regeneration without scar formation. Initially, nearby epithelial cells slide and flatten to fill the defect. Reepithelialization follows by means of proliferation and maturation of basal cells near the zone of injury and migration of cells from the periphery. The new epithelium is fragile and dysfunctional compared with normal epithelium. Reattachment to the basement membrane stabilizes the layer, and further remodeling restores its integrity (Table 3).

Edema can have a significant role in precipitating an epithelial defect. Edema can be caused by trauma, ischemia, and increased intraocular pressure (i.e., from decreased venous outflow). Excess fluid in and between cells disrupts the normal architecture of the epithelial layer, causing fragility and dysfunction. If edema is sufficient to lift cells from the basement membrane, the overlying epithelium becomes very delicate, held together only by desmosomes, and readily separates from the cornea with minimal force (e.g., friction from forceful eyelid opening on dry corneal surface), producing an abrasion.

PERIOPERATIVE RISK FACTORS

The threshold for epithelial injury is lower in the unconscious surgical patient, because protective mechanisms are diminished by general anesthesia (Table 4). General anesthesia suppresses autonomic reflexes (i.e., corneal reflex, Bell phenomenon, reflex tearing), increases lagophthalmos (incomplete eyelid closure), and diminishes tear production and stability. The dry, exposed corneal surface is fragile and vulnerable to injury from (1) direct trauma and (2) desquamation, the two causes of perioperative abrasion.

Based on the largest studies to date (Table 5), the overall incidence of perioperative corneal abrasion is approximately 0.01 to 0.11 percent, however, the likelihood of injury can be higher in the presence of certain patient- and surgery-specific risk factors (Table 6).

Patient conditions that diminish the structural integrity of the corneal epithelium reduce the threshold for perioperative abrasion, namely, dry eye and recurrent corneal erosion. Dry eye is caused by inadequate tear film production and/or lagophthalmos. Advanced age is a risk factor for perioperative abrasion, likely attributable in part to physiologic tear film deficiency. Patients with prominent globe position (e.g., proptosis, exorbitism) are predisposed to lagophthalmos and therefore are at higher risk for perioperative abrasion. In recurrent corneal erosion, epithelial architecture is distorted and fragile. Recurrent corneal erosion can arise either as a result of prior trauma or corneal dystrophy. Therefore, history of ocular surface injury, isolated or recurrent, should be noted. Map-dot-fingerprint dystrophy affects 9 to 42 percent of the general population (more common in women and elderly) and results in weakened attachment of the epithelium to basement membrane. Patients with map-dot-fingerprint dystrophy, also known as basement membrane or Cogan-Guerry dystrophy, can sustain a corneal abrasion from minimal to no trauma.

Longer procedures (>60 to 90 minutes) carry a significantly higher risk of corneal abrasion. Longer anesthesia times lengthen the period of diminished protective mechanisms. Furthermore, tear production and breakup time are reduced.
to an even greater extent with increasing duration of general anesthesia.

Patient head position is another important consideration. Procedures requiring lateral, prone, and Trendelenburg positions have a higher incidence of corneal abrasion. In lateral and prone positions, the head is turned to the side or face-down, with one or both eyes placed in a dependent position. Because of gravity, the dependent globe is prone to exposure. In addition, improper head positioning that places pressure on the globe can cause ischemia and edema secondary to compression of choroidal blood vessels. Lateral, prone, and Trendelenburg positioning can diminish venous outflow and increase intraocular pressure, both of which cause edema and increase the fragility of the corneal surface.

Other risk factors for perioperative corneal abrasion include head and neck surgery, intraoperative hypotension, and preoperative anemia. Like patient positioning, these factors can place the cornea in a vulnerable position and increase the propensity for ischemia and edema.

**PREVENTION**

Prevention is optimized when all participating providers (e.g., surgeons, anesthesia providers, and operating room and recovery room nursing staff) are educated and involved in eye care. Table 7 lists potential sources of physical, chemical, and thermal injury specific to each phase of care.

The cause of most abrasions is unknown (one study identified cause in only 20 percent of cases), and direct trauma is believed to account for a minority of identifiable cases. Segal et al. found oxygen use during transport and recovery to be a significant risk factor, supporting the idea that face masks are a preventable cause of abrasions. Eye shields are commonly used during periorbital surgery to protect the eye from direct trauma; however, these must be inspected for surface irregularities before each case and require careful placement/removal, as they have been known to cause corneal injury.

The most important risk factor for perioperative corneal abrasion is lagophthalmos. Although only 5 percent of individuals demonstrate lagophthalmos during normal sleep, the prevalence may be as high as 60 percent in those under general anesthesia. Incomplete lid closure exposes the epithelial surface, making it prone to desiccation. In one prospective study, corneal abrasions occurred in 26 of 59 patients (44 percent) whose eyelids were partly open during surgery. All patients should have eyelids secured in closed position immediately after induction. A strip of tape is generally sufficient; however, high-risk cases (e.g., those requiring Trendelenburg position) may benefit from use of transparent bioocclusive dressings, such as Tegaderm or OpSite, which can span the entire lid, providing strong uniform closure. Furthermore, by creating a tight peripheral seal at the skin around the eye, tear film evaporation is minimized and a barrier to physical and chemical trauma is provided.

Ocular lubricants support surface moisture. Studies comparing various types of lubricants fail to demonstrate differences in efficacy; however, preservative-free methylcellulose-based ointments are preferred. Paraffin-based (petroleum) ointments disrupt tear film stability (decrease breakup time), carry a higher risk of eye irritation (blurred vision, foreign body sensation, and photophobia), and are flammable. In addition, certain inhalational anesthetics (halothane and isoflurane) are highly soluble in paraffin-based lubricants, reaching concentrations high enough to cause corneal injury.
enough to cause ocular inflammation. In contrast, methylcellulose-based lubricants prolong tear breakup time and have a low complication rate. Ointments are preferred because they are retained in the eye longer than aqueous solutions (they have a longer half-life on the closed, nonblinking human eye). Methylcellulose-based ointments create firm adhesion between upper and lower lids, which may also support ocular surface moisture by preventing tear film evaporation. Many commonly used preservatives can irritate the ocular surface; therefore, preservative-free preparations are recommended. Gelatinous lubricants have also been described for perioperative use. There is no advantage to combining lid taping and lubrication over using lid taping alone. Moreover, lubricant can get onto the tape, making it difficult to stick to the skin. However, if lid taping is not possible or not desired (e.g., burn patients, need for perioperative eye monitoring), lubricant should be applied for corneal surface protection. Prevention guidelines are summarized in Table 8.

### DIAGNOSIS

Evaluation of postoperative eye pain begins with a focused history of current symptoms. Pain from corneal abrasion has an abrupt onset, typically arising within 2 hours of procedure completion as anesthesia wears off. The patient experiences intense pain and discomfort. Patients often describe a foreign body sensation and excessive tearing, blurry vision, and/or photophobia. A history of similar symptoms may reveal an underlying ocular surface abnormality (e.g., chronic dry eye, recurrent erosion syndrome) that may predispose the patient to this complication.

The eye examination begins by everting the eyelids to rule out the presence of a foreign body. If present, removal can be attempted with irrigation or topical anesthetic and a cotton swab.

### TREATMENT

Effective treatment requires (1) pain control, (2) infection prevention, and (3) daily symptom monitoring. Pain is greatest in the initial 24 hours after injury, when nerve fibers are exposed at the base of the wound. Previous treatment guidelines made effective analgesia challenging; however, updated recommendations, specifically regarding eye patching and topical anesthetic use (Table 10), have improved pain control during this initial period of heightened sensitivity.

### Table 8. Prevention Summary

| All participating providers educated on eye protection |
| Begins immediately after induction |
| Protect from direct physical, chemical, thermal injury* |
| Apply preservative-free lubricant† |
| Tape lids until ready to awaken from anesthesia |
| Continue to protect through recovery in PACU |

PACU, postanesthesia care unit.

*Evidence suggests that lubricant is only required if not taping lids.

†Evidence suggests that lubricant is only required if not taping lids. Consider combining lubricant with lid taping in high-risk cases (see Table 6).

### Table 9. Findings That Warrant Ophthalmologic Consultation

| History suggestive of underlying ocular surface abnormality |
| Foreign body embedded in ocular surface |
| Rust rung after removal of iron-containing foreign body |
| Visual acuity worse than 20/40 |
| Persistent vision abnormality |
| New or worsening visual symptoms after 24 hr |
| Irregular, dilated, or fixed pupil |
| Extruding ocular contents |
| Corneal infiltrate or ulceration |
| Blood (hyphema) or pus (hypopyon) in anterior chamber |
| Injury extending through Bowman membrane into stroma |
| Dendritic/branching fluorescein pattern (herpetic keratitis) |
| Worsening pain or vision after 24 hr |
| Still painful after 48 hr |
| Failure to heal completely after 72 hr |
Topical anesthetics were previously contraindicated during treatment of corneal abrasion because of concern for deleterious effects on healing and potential for patient misuse, both of which can result in serious complications. Concern for delayed reepithelialization derives from early animal studies; however, more recent laboratory and clinical investigations suggest that the effect of topical anesthetics on the corneal epithelium is insignificant.

Verma and colleagues conducted two randomized clinical trials testing the safety and efficacy of topical anesthetics in controlling pain after surgically creating an epithelial defect during photorefractive keratectomy. In the first study, patients instilled drops of either 1% tetracaine hydrochloride or placebo (physiologic saline) every 30 minutes while awake for 24 hours after surgery. The tetracaine group demonstrated delayed healing rates for 24 hours; however, thereafter, rates were similar between tetracaine and placebo groups. Wound healing was uncomplicated and complete within 72 hours in all patients. The tetracaine group experienced a lower maximum pain level during the immediate postoperative period, and fewer patients regarded the postoperative period as “painful” versus “nonpainful” (39 percent versus 85 percent of the placebo group). In the second study, they compared topical anesthetics: 1% tetracaine and 0.75% bupivacaine. Again, wound healing was uncomplicated in all patients. Surprisingly, despite the increased duration of action of bupivacaine, tetracaine was much more effective in reducing postoperative pain.

More recent randomized clinical trials have investigated the use of topical anesthetics in corneal abrasions presenting to the emergency room. Ting et al. randomized 47 patients to receive either topical 0.4% amethocaine or placebo (saline), with follow-up at 36 to 48 hours. The treatment group had better pain control, and neither group had complications at the 2-week follow-up. The authors conclude that topical amethocaine is effective but not definitely safe, as they felt the study lacked sufficient patient numbers to show harm. Waldman et al. randomized 116 patients to receive 1% tetracaine hydrochloride or placebo (saline) every 30 minutes while awake for 24 hours. There were no complications and no significant difference in corneal healing as measured by number of patients with persistent fluorescein uptake at 48 hours (23.9 percent versus 21.3 percent). Although the treatment group failed to demonstrate a significant difference in pain scores taken every 2 hours, they scored better on perceived overall effectiveness of pain control after 48 hours.

Topical anesthetics are extremely effective in relieving ocular pain; however, this also makes them prone to misuse. Case reports of corneal epithelial injury progressing to ulceration and infection because of prolonged use (days to weeks) of topical anesthetics can be found throughout the literature. These cases underscore the danger of masking pain. As stated previously, persistent or worsening pain is atypical for corneal abrasion and requires immediate consultation. Limiting use of topical anesthetics to the first 24 hours allows adequate monitoring of symptoms.

Other topical analgesic options have been used in place of topical anesthetics, such as topical nonsteroidal antiinflammatory drugs (e.g., diclofenac 0.1%, ketorolac 0.4%) and cycloplegics (e.g., homatropine 5%, cyclopentolate 1%). Topical nonsteroidal antiinflammatory drugs, although longer acting and shown to relieve pain, are weaker and more expensive than anesthetics. Moreover, prolonged use is associated with corneal toxicity, limiting use of these agents to 1 to 2 days. Unless the patient has an allergy to topical anesthetic, topical nonsteroidal antiinflammatory drugs should not be considered a first-line analgesic for corneal abrasion. Topical cycloplegics can relieve pain secondary to ciliary spasm from traumatic iritis; however, they do not appear beneficial in uncomplicated corneal abrasion.

Eye patching has been used to improve patient discomfort; however, clinical studies suggest no benefit and potential harm with patching. Turner and Rabiu conducted
a meta-analysis of 11 randomized and quasi-randomized studies comparing patching and no patching. None of the included studies demonstrated significant pain reduction in patch groups (two actually found significantly more pain with patching); and combined data from seven studies demonstrated significantly less healing on the first day of follow-up with patching. Delayed healing may be attributable to compression of choroidal vessels and/or reduced atmospheric oxygen diffusion, both of which diminish oxygenation. Eye patching does not appear to improve patient discomfort and may provide a suboptimal environment for corneal healing by diminishing oxygenation; therefore, it is not recommended.

A prophylactic topical antibiotic should be prescribed for all corneal abrasions to prevent secondary infection and ulceration.\(^7^4\) Although prospective comparative studies are lacking, antibiotics carry little downside (the most common side effect is contact hypersensitivity), whereas the consequence of infection can be disastrous. Antibiotics can be given as solution (drops) or ointment. Contact lens wearers are frequently colonized with Gram-negative organisms; therefore, an antibiotic with antipseudomonal activity is recommended for these patients.\(^7^2\)\(^7^3\) Antibiotics are prescribed for 2 to 3 days. Preparations containing steroids are contraindicated because they can delay healing and increase risk of infection.\(^7^4\) Table 11 provides a list of recommended antibiotics.

In addition to topical anesthetic and prophylactic antibiotics, lubricant drops should be given to support ocular surface moisture and provide comfort. Preservative-free lubricant drops, such as Systane PF (Alcon, Forth Worth, Texas) or Refresh (Allergan, Inc., Irvine, Calif.), are preferred because they stabilize the tear film and have minimal side effects. Symptoms should be monitored closely until completely resolved. Outpatient treatment is acceptable if the patient is reliable for daily follow-up. Table 12 summarizes the updated treatment guidelines for perioperative corneal abrasion.\(^7^5\)\(^7^6\) If there is any evidence of deviation from the expected healing course during treatment (symptoms worsen, fail to improve, or persist), immediate ophthalmologic consultation is required.

### Table 11. Prophylactic Antibiotic Choices

<table>
<thead>
<tr>
<th>Category</th>
<th>Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-line</td>
<td>Erythromycin 0.5% ointment</td>
</tr>
<tr>
<td></td>
<td>Bacitracin ophthalmic ointment</td>
</tr>
<tr>
<td></td>
<td>Polymyxin B/trimethoprim (Polytrim; Allergan) solution</td>
</tr>
<tr>
<td></td>
<td>Sulfacetamide 10% (Bleph-10; Allergan) solution</td>
</tr>
<tr>
<td></td>
<td>Antipseudomonal coverage</td>
</tr>
<tr>
<td></td>
<td>Ofloxacin 0.3% (Ocuflox; Allergan) solution</td>
</tr>
<tr>
<td></td>
<td>Moxifloxacin hydrochloride 0.5% (Vigamox; Alcon) solution</td>
</tr>
<tr>
<td></td>
<td>Gentamicin 0.3% ointment or solution</td>
</tr>
</tbody>
</table>

### Table 12. Treatment Guidelines

**Recommended**

- Oral analgesic (i.e., narcotics, acetaminophen, nonsteroidal antiinflammatory drugs) as needed for pain
- Topical anesthetic (proparacaine hydrochloride 0.1–0.5% or tetracaine hydrochloride 1%)\(^*\) every 30 min as needed for pain during first 24 hr\(^†\)
- Prophylactic topical antibiotic (antipseudomonal coverage if patient wears contact lenses) for 2–3 days
- Preservative-free 0.5% methylcellulose lubricant drops as needed for comfort
- Daily symptom monitoring until resolution or referral to specialist care

**Recommended against**

- Eye patching
- Topical steroids

\(^*\)Proparacaine may be less cytotoxic than tetracaine (Moreira LB, Kasetsuwan N, Sanchez D, Shah SS, LaBree L, McDonnell PJ. Toxicity of topical anesthetic agents to human keratocytes in vivo. J Cataract Refract Surg. 1999;25:975–980). Proparacaine is typically available in 0.5% concentration. It can be diluted with sterile balanced salt solution. Proparacaine concentration should be at least 0.1% to achieve complete corneal anesthesia (Shahinian J Jr, Jain S, Jager RD, Lin DT, Sanislo SS, Miller JF. Dilute topical proparacaine for pain relief after photorefractive keratectomy. Ophthalmology 1997;104:1327–1332).

\(^†\)Alternatively, topical nonsteroidal antiinflammatory drugs can be used (e.g., in patients with allergy to topical anesthetic).

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**CONCLUSIONS**

Understanding the pathophysiology of perioperative corneal abrasion is the first step in preventing this unfortunate complication. The surgical team should appreciate the surgery- and patient-specific risk factors that predispose to ocular surface injury and apply appropriate preventative measures. When a perioperative abrasion occurs, discomfort and morbidity are minimized with appropriate treatment, symptom monitoring, and ophthalmologic referral when indicated.

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REFERENCES


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