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Emergency treatment of chemical and thermal eye burns

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ABSTRACT.
Chemical and thermal eye burns account for a small but significant fraction of ocular trauma. The speed at which initial irrigation of the eye begins, has the greatest influence on the prognosis and outcome of eye burns. Water is commonly recommended as an irrigation fluid. However, water is hypotonic to the corneal stroma. The osmolarity gradient causes an increased water influx into the cornea and the invasion of the corrosive substance into deeper corneal structures. We therefore recommend higher osmolarities for the initial rinsing to mobilize water and the dissolved corrosives out of the burnt tissue. Universal systems such as amphoteric solutions, which have an unspecific binding with bases and acids, provide a convenient solution for emergency neutralisation. Both conservative anti-inflammatory therapy and early surgical intervention are important to reduce the inflammatory response of the burnt tissue. In most severe eye burns, tenonplasty re-establishes the conjunctival surface and limbal vascularity and prevents anterior segment necrosis.

Key words: cornea – emergency treatment – eye burns – irrigation fluid – reconstructive surgery.

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Recent studies put the incidence of chemical and thermal injuries to the eye at 7.7%–18% of all ocular trauma (Watz & Reim 1973; Pfister et al. 1984; Liggett 1989; MacEwen 1989; Zagelbaum et al. 1993). Most of these injuries are trivial and do not cause any lasting lesions, others result in permanent unilateral or bilateral visual impairment and a life of dependency (Kuckelkorn et al. 1993). The majority of victims are young and exposure occurs at home, work and in association with criminal assaults (Keeney 1974; Morris et al. 1987; Thielsch et al. 1989). Alkali injuries occur more frequently than acid injuries (Pfister 1983; Morgan 1987), eye burns caused by detergents and thermal agents being less frequent again (Kuckelkorn et al. 1995). The most common agents causing alkali burns are ammonia (NH_3), lye (NaOH), potassium hydroxide (KOH) and lime (CaOH2). Sulfuric (H_2SO_4), sulfurous (H2SO_3), hydrofluoric (HF) and hydrochloric (HCL) acids are the most common causes of acid burns. Table 1 lists the data from 191 patients with 260 severely burnt eyes who were treated in the eye center of the RWTH Aachen between 1980 and 1995. There was a high incidence of bilateral injuries and most of the injuries occured at home or during leisure activities.

Action of alkalis and acids

The severity of ocular injury is related to the type of chemical, the volume and concentration (pH) of the solution and the duration of exposure (Hughes 1946). Alkalis penetrate more rapidly than acids. The hydroxylion (OH) saponifies the fatty acid components of the cell membranes with consecutive cell disruption and cell death, while the cation is responsible for the penetration process of the specific alkali (McCulley 1987). The penetration rate increases from calcium hydroxide (slowest), potassium hydroxide (faster), sodium hydroxide (even faster) to ammonium hydroxide (fastest; Grant 1974). Depending on the degree of penetration, there is a loss of corneal and conjunctival epithelium, stromal keratocytes and endothelium. Hydration of the glycosaminoglycans results in loss of clarity of the stroma (Grant & Kern 1955). Damage to the vascular endothelium of conjunctival and episcleral vessels leads to thrombosis of the episleral vessels.

The stronger the alkali, the faster its penetration. Irreversible damage occurs at a pH above 11.5 (Friedenwald et al. 1944). The pH in the aqueous humour rises within a few seconds of contact with ammonium hydroxide (Graupner & Hausmann 1970). Intraocular structures such as the iris, lens and ciliary body are rapidly damaged.

Table 1. Severe chemical and thermal eye burns in the Department of Ophthalmology of the RWTH Aachen (1985–1995): 191 patients (260 eyes).

	Number of patients	Per cent
Unilateral	122	64
Bilateral	69	36
	Eyes	
Occupational injuries	177	68.1
Private injuries	63	24.1
Others	20	7.8
	Eyes	
Alkalis	151	58.1
Acids	37	14.1
Thermal	42	16.2
Others	30	11.6

Apart from hydrofluoric acid and, to a lesser extent, sulfurous acid, acids penetrate the corneal stroma much less readily than alkalis (Grant 1974). The hydrogen ion causes damage due to pH alteration, while the anion produces protein precipitation and denaturation in the corneal epithelium and superficial stroma (Friedenwald et al. 1946). Precipitation of the epithelial proteins offers some protection to the corneal stroma and intraocular structures. However, very strong acids penetrate just quickly as alkalis. No statistical difference between strong alkali and acids burns was discovered in the clinical course and prognosis of such eyes (Kuckelkorn 1996).

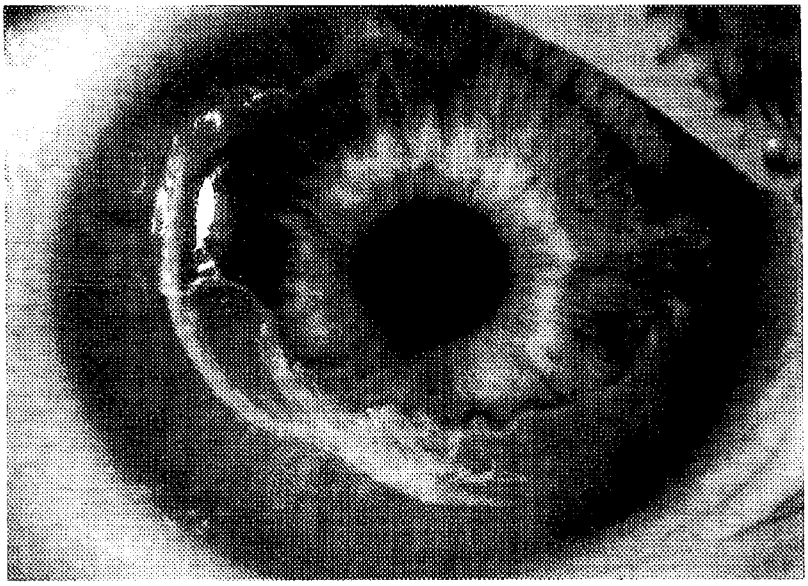

Fig. 1. Grade I chemical injury: hydrochloric acid (HCl). Burn of the cornea only. Coagulated corneal epithelium with 'ground glass' appereance. Partial removal of the epithelium, clear corenal stroma.

Clinical classification of chemical and thermal burns

Eye burns are classified in 4 grades (Reim 1987, 1990). The clinical course and ultimate prognosis correlates with the extent of limbal ischemia (Hughes 1946; Ballen 1963; Roper-Hall 1965). The prognosis also depends on the extent of damage to conjunctival and episcleral tissue, severity of lid burn and damage to intraocular structures (Table 2).

Mild burns of grades I and II are associated with hyperemia, small conjunctival ecchymosis and chemosis as well as erosion of the corneal epithelium (Figs 1 and 2). In mild acid burns, the coagulated corneal epithelium often has a 'ground-glass' appearance. After removal of the epithelium, the clear corneal stroma is visible.

Grade III, and especially grade IV, burns are accompanied by extensive and deep damage to the tissue (Figs 3 and 4). Typically, large areas of the conjunctival and subconjunctival tissue are involved. The visible blood vessels are thrombosed and appear dark. The corneal keratocytes are lost and hydration of the denatured proteins results in corneal opacification. Chemical injury to the iris and crystalline lens may produce mydriasis, a greyish appearance of the iris and the fast development of a cataract. The lysis of cells of the anterior chamber destroys the blood aqueous barrier and leads to iridocyclitis and fibrinous exsudation.

Toxic substances such as prostaglandines, superoxide radicals, and presumably histamine, angiotensin, leukotriens and others are released from the burnt cells of the necrotic tissue (Eakins & Bhattacherjee 1977; Kulkarni & Srinivasan 1993; Rochels et al. 1982). An inflammatory response is initiated, when they diffuse into surviving tissues. In mild burns this reaction resolves quickly, while in severe burns a severe and long-term inflammatory process is initiated, determining the clinical course of the burnt eyes (Reim 1982, 1987, 1992; Williams et al. 1983; Struck et al. 1991; Reim & Leber 1993; Reim et al. 1993).

Emergency treatment

Immediate irrigation is of paramount importance after chemical or thermal burns (Lubeck & Greene 1988; Cohen & Hynduik 1978; Rodeheaver et al. 1982). In most cases the victims are disabled by severe reflectory blepharospasm with ensuing disorientation. In this situation the victims are unlikely to be capable of reaching the nearest body or eye shower and need res-

Table 2. Clinical classification and prognosis of eye burns

Grade	I	II	III	IV
Appearence	Erosio	Erosio	Erosio	Erosio
Limbal ischemia >3/4	Hyperemia	Limbal ischemia >1/3	Limbal ischemia >1/2	
		Chemosis	Chemosis	Chemosis
Opacification	Opacification			Extensive necrosis
Clinical outcome	Regeneration	Recirculation	Vascularisation	Ulceration
		Regeneration	Ulceration	Iris atrophy
			Proliferaton	Cataract
			Cicatrization	Glaucoma
Prognosis	Complete restitution	Complete restitution	Scars	
Prevention of the globe				
Multiple operations for cosmetic rehabilitation		Slight scars	Multiple operations for limited visual rehabilitation	

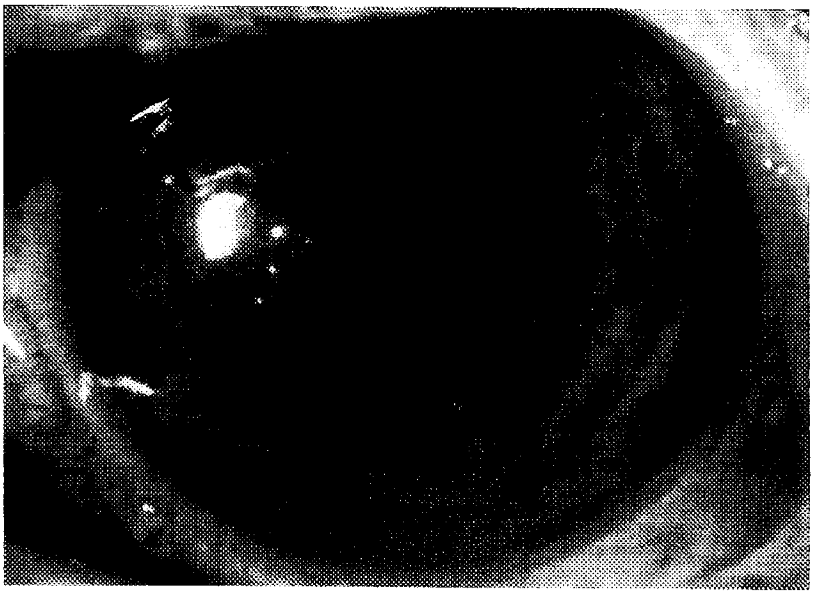

Fig. 2. Grade II chemical injury: lime (CaOH). Central epithelial defect, partial limbal ischemia in the nasal inferior quadrant.

cuers who remove them from dangerous areas and apply fast and efficient help to their eyes and body (Morgan 1987).

Effective first aid involves knowing how to overcome blepharospasm by a passive opening of the lids and how to perform effective irrigation of the eye. All aspects of the conjunctiva and cornea should be irrigated, and the patient should be asked to look in all directions (Tannen & Marsden 1991). Topical anesthetic drops may be applied to reduce the pain and to facilitate irrigation. According to the American National Standards Institute (ANSI) standard (Z358.1–1990) severe eye burns have to be rinsed for 15 min. At least 500–1000 mL of irrigation fluid are thus necessary. Amphoteric or buffered solutions can normalize the pH of the anterior chamber within that time (Schrage et al. 1996). Particles are sometimes trapped in the fornizes or under the upper lid. Therefore, ectropinisation and intensive cleaning of the cul-de-sac are mandatory after every burn. Materials containing calcium oxid (lime, cement dust) react avidly with water to produce a calcium hydroxide solution with a pH of 12.4 (Moon & Robertson 1983). A cotton-tipped applicator soaked in EDTA 1% (EDTA, di-sodium-ethylendiamintetra-acetat) can be used to facilitate cleaning of the cul-de-sac from calcium hydroxide (Pfister 1983). Immediate irrigation is also important in thermal burns, because this cools the ocular surface (Schrage et al. 1997). Continuous irrigation also removes inflammatory substances from the ocular surface (Reim 1990; Reim & Kuckelkorn 1995).

First aid with intensive irrigation immediately after the injury has a decisive influence on the clinical course and prognosis of such eyes (Saari & Parvi 1984; Burns & Paterson 1989). A comparison between visual outcome of better than 1/50 with that of less than 1/50 revealed a highly significant difference, with significantly better results after immediate irrigation. Visual acuity of >1/50 enables the patient to move unaided. The number of operations and the length of stay on the ward are significantly reduced for eyes that received prompt irrigation (Table 3; Kuckelkorn et al. 1995).

Fig. 3. Grade III chemical injury: sodium hydroxide (NaOH). Complete corneal and proximal conjunctival epithelial defect with loss of corneal stromal clarity. Limbal ischemia in theinferior quadrants.

Choice of irrigation fluid

Burns are accompanied by a loss of the corneal epithelium within a few seconds. The acutely burnt cornea takes up the burning substance by osmolar forces resulting in a high osmolarity. One of the aims of rinsing therapy is to remove this chemical burden.

Water is commonly recommended as an irrigation fluid. It is available almost everywhere, and copious amounts of water have a dilutive effect. However, water is hypotonic to the corneal stroma and intraocular milieu. In measurements of osmolarity, Schrage et al. (unpublished) found the corneal stroma to have an osmolarity of 420 mOsm/L. The corneal tissue is diluted by rinsing with water and this is accompanied by an increased uptake of additional water and diffusion of the corrosive into the deeper layers of the cornea. We thus recommend the use of irrigation fluids with higher osmolari-

Table 3. Value of immediate rinsing therapy *versus* clinical results in severe eye burns (101 patients, 131 eyes).

Rinsing	Number of operations	Hospitalization (months)	Visual acuity
Immediate	6.5 ± 4.6	4.2 ± 2.8	45 (76%)
Delayed or no	10.4 ± 10	6.0 ± 4.5	22 (55%)

Unpaired *t*-test, $P < 0.05$.

ties for initial rinsing in order to prevent water influx into the cornea and to enable the mobilisation of water and the dissolved corrosives out of the burnt tissue.

Normal saline (NS), which is often recommended as irrigation fluid, also has a lower osmolarity than tear fluid. It fails to normalize the pH of the anterior chamber even after prolonged irrigation (Table 4).

Phosphate buffer is often cited as an ideal buffer solution (Thiel 1965; Laux et al. 1975; Poser 1983; Roth 1993). For this reason, there is widespread use of this buffer in many factories. However, in an experimental study complete calcification of the superficial stroma occured in 100% of all animals after burning with 1 n NaOH for 30 s and immediate rinsing with 500 mL phosphate buffer. (Schrage et al., unpublished). We suggest that exogenously applied phosphate reacts with endogenous calcium released from ruptured cells to produce calcium-phophate complexes.

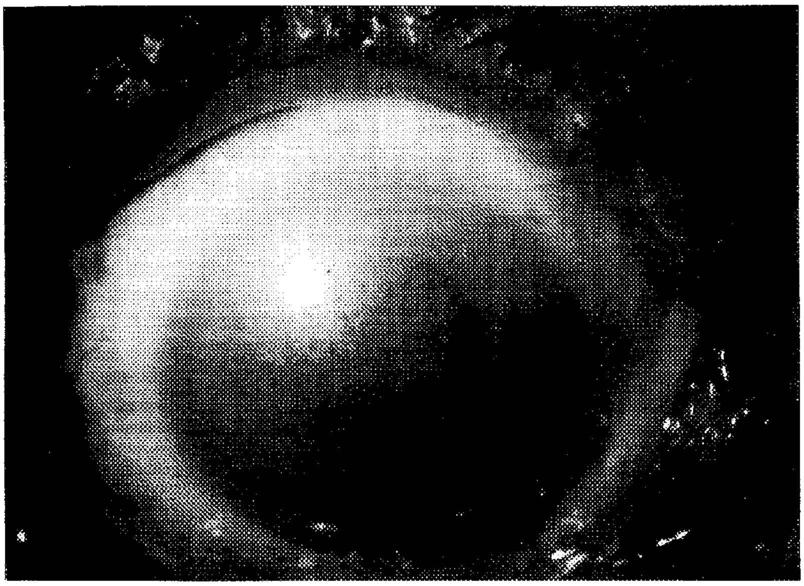

Fig. 4. Grade IV chemical injury: sodium hydroxide (NaOH). Loss of corneal transparency, ectropion uveae and cataract formation, circular loss of conjunctival and episcleral tissue down to the fornices. The sclera is ischemic.

At present, there is ongoing experimental research to find irrigation fluids with an osmolarity similar to the corneal stroma. Currently available fluids which are suitable for irrigation are sterile, lactated Ringer's (LR) and balanced saline solution (BSS; Herr et al. 1991). Lactated Ringer is a buffered solution and may be more effective than normal saline. The osmolarity of BSS is similar to that of aqueous humour; its pH is neutral and it contains sodium acetat and citrat (McDermott et al. 1988). According to Pfister, isotonic citrate buffer initiates chelat-complexes and binds unspecific metal-ions derived from the corrosive (Pfister et al. 1981; Pfister et al. 1984). Balanced saline solution has an enhanced buffering capacity; it prevents the cornea from swelling and preserves the corneal endothelium (McNamara et al. 1987).

The pH, osmolarity and buffer capacity of the aqueous humour, corneal stroma and some irrigations fluids are listed in Table 5.

A new amphoteric solution which is suitable for irrigation is Diphoterine (Previn®, Fa. Prevor). This newly synthetisized fluid is able to bind both alkalis and acids. 0.4% Diphoterinehas a pH of 7.4 and an osmolarity of 820 mosml/L. The pH in the conjunctival sac and in the corneal stroma is reduced as rapidly as after irrigation with phosphate buffer. The constituents of Diphoterine are listed in Table 6.

Transport problems

As strong acids and alkalis penetrate within seconds or minutes and remain for hours (Grant & Kern 1955), irrigation should not be interrupted during transport to a professional eye-care unit. The recommendations for minimum irrigation times range from 15 min (ANSI standard; Lubeck & Greene 1988) to 2–4

Table 4. pH on the corneal surface and in the anterior chamber after rinsing with different irrigation fluids (experimental eye burn for 30 s/1n NaOH).

	pH corneal surface	anterior chamber
Directly after burn	13 ± 0	10 ± 0
5 minutes after rinsing with 500 mL normal saline	9 ± 0	10 ± 0
5 minutes after rinsing with 500 mL phosphat buffer	7.5 ± 0	9.25 ± 0.44
5 minutes after rinsing with 500 mL diphoterine	7.5 ± 0	9.34 ± 0.59

Concentration of phosphat buffer: 440 mg sodiumdihydrogenphosphat and 4040 mg sodiummonohydrogenphosphat in 100 mL H_2O.

Table 5. pH, osmolarity, constituents and buffer capacity of the aqueous humour, corneal stroma and different irrigation fluids.

	pH	osmolarity	constituents	buffer capacity
Aqueous humour	7.4	304	Na, K, Cl, Ca, PO_4, proteins	0.0008
Stroma	7.4	420	Na, K, Cl, Ca, S, SO_4, PO_4, proteins, lipids, glycosaminoglycans	0.0004
Normal saline	7.0	290	Na, Cl	0.0002
Phosphate buffer	7.4	260	Na, K, PO_4	0.00625
Lactated Ringer's	5.0–7.5	280–309	Na, K, Ca, Cl, lactat	0.00069
BSS	7.2	310	Na, K, Ca, Cl, citrat, acetat	0.001
Diphoterine	7.4	820	Diphoterine, Na, Cl, glycin	0.02

h (Pfister 1983; Saari et al. 1984). We recommend the use of an intravenous infusion set to supply at least 500–1000 mL of irrigation fluid. As described above, one of the rescuers should hold the eyelids open while a second rescuer flushes the eye with a mild, directable and controllable stream of fluid. Local anesthetic drops should be administered repeatedly if necessary to relieve the patient from pain and to facilitate irrigation.

Some authors favor the use of specially designed irrigation systems (Naumann 1964; Girard & Soper 1966; Schulze & Tost 1967; Tan 1970; Morgan 1971; Lau 1979). Whereas these systems provide continuous irrigation of the eye, they fail to flush the ocular surface homogenously and appropiately, especially the cul-de-sac. A further risk with lime or cement burnsis that particles retained under the eye lids are not detected and removed once the loop or lens has been applied. Moreover, rescuers unfamiliar with the handling of these systems will lose precious time when installing the slings or lenses, which may cause additional damage to the eye.

The effectiveness of rinsing therapy can be assessed by using universal indicator paper to determine the pH of the external eye. Irrigation must be continued as long as the pH remains outside the normal range. If prolonged irrigation does not achieve normalization of the pH, one must consider the possibility that there are still particles in the superior or inferior cul-de-sac.

Table 6.

Constituents of diphoterine.	
Amphoter: Diphoterine	3.8/100 mL
NaCl:	1.8/100 mL
Glycin:	0.75/100 mL
Preservative:	0.05/100 mL
Aqua destillata:	ad 100 mL

Subsequent care

The subsequent care of eye burns is dependent on the severity of the injury. Further therapeutical procedures are applied according to the extent of the damage. If the injury is mild (grades I and II) and irrigation began immediately, most eyes will heal without permanent damage within a few days (Moon & Robertson 1983; Morgan 1987; Beare 1990; Kuckelkorn et al. 1993). Topical steroid/antibiotic drops and ointment plus padding may suffice for the treatment of these mild burns. Follow-up treatment within 24 h is mandatory.

Severe ocular eye burns (grades III and IV) are difficult to treat and the course of healing often takes several months. In these cases, accurate classification with regard to the extend of (limbal) ischemia and depth of tissue destruction is essential. An examination with the operating microscope is thus mandatory. Parabulbar or general anesthesia are sometimes needed if the patient suffers pain and local anesthetic drops are not sufficient.

Less severe eye burns (grade III) are characterized by superficial ischemia of the conjunctival tissue. In these cases where regular anterior chamber structures are preserved and there is no damage to the iris, ectropium uveae or fibrinous exsudation, subsequent management takes the form of a more conservative therapy. Admission to and treatment in a local eye clinic are thus sufficient (Reim & Kuckelkorn 1995).

Most severe eye burns (grade IV) lead to significant limbal ischemia and necrosis of the bulbar and tarsal conjunctiva as well as of the episcleral tissue down to the fornices. In cases with superficial necrosis, the deep episcleral vessels are still perfused while necrosis of the deeper episcleral tissue is associated with thrombosis of the episcleral vessels. In these severe cases, opacification of the cornea is common and the anterior chamber structures are obscured. A greyish aspect of the iris, ectropium uveae and the breakdown of the blood aqueous barrier with fibrinous exsudation into the anterior chamber confirm the destruction of the deep anterior segment. The lids and aspects of the tarsal conjunctiva are often involved. Many problems arise in the acute phase of the burn. The most delicate problem is preventing the eye from early melting.

Necrosis of the conjunctiva and subconjunctival tissue is accompanied by a considerable exsudation of leucozytes (PMN's). These leucozytes release large amounts of lysosomal enzymes. The matrix metalloproteinases (MMP); collagenase (MMP-1 and MMP-8; Itoi et al. 1969; Hook et al. 1971; Newsome & Gross 1977; Johnson-Muller & Gross 1978; Kuter et al. 1989; Fini & Girard 1990), gelatinase (MMP-2, MMP-9) (Collier et al. 1988; Fini & Girard 1990; Huhtala et al. 1990), and stromelysin (MMP-3; Collier et al. 1988; Chin et al. 1995) in particular are responsible for the splitting of the collagen molecules and the development of corneoscleral and corneal ulceration, characteristically 4–6 weeks after the accident.

The basic principle in the treatment of these eyes is to reduce the inflammatory response caused by the necrotic tissue. The traditional mainstay of therapy is the early and intensive application of corticosteroids (Donshik et al. 1978; Leibowitz 1980; Reim & Schmidt-Martens 1982; Kenyon 1985; Reim 1987). Additionally, local antibiotics are necessary to prevent microbiobal infections until the ocular surface has reepithelialized (Girard et al. 1970; Kuckelkorn et al. 1987; Beare 1990). Tetracycline derivates play an important role because they have been shown to inhibit metallo-proteinases (Brion et al. 1985; Golup et al. 1987; Seedor et al. 1987; Burns et al. 1989; Perry et al. 1993) independently of their anti-microbial properties.

Besides conservative therapy, active surgical intervention with the débridement of necrotic conjunctival and subconjunctival tissue is necessary in order to remove a nidus of continued inflammation from retained caustic materials, although also any accumulation of PMN and to prevent the sustained release of their destructive enzymes. Special reconstructive approaches such as tenonplasty allow the denuded avascular sclera to be covered with vital connective tissue prepared from the equator of the globe (Reim & Teping 1989; Reim 1992; Reim & Kuckelkorn 1992, 1995; Kuckelkorn & Reim 1993; Reim & Leber 1993; Kuckelkorn et al. 1995). The main advantage of this tissue is that it enables the reconstruction of the conjunctival matrix and of limbal vascularity. These interventions prevent anterior segment necrosis and/or sterile ulceration and the eye is preserved. It is advisable to admit these cases to an eye clinic specialized in the treatment of these eyes and familiar with specialized procedures in plastic reconstruction.

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