Kindness translation made by PREVOR, Valmondois, France

J Fr. Ophtalmol., 2008; 31, 5, 1-12

REVUE GÉNÉRALE FMC

OCULAR BURNS

H. Merle (1), M. Gérard (2), N. Schrage (3)
(1) Service d'Ophtalmologie, Centre Hospitalier Universitaire de Fort de France, Hôpital Pierre Zobda-Quitman, Fort de France, Martinique — France
(French West Indies).
(2) Service d'Ophtalmologie, Centre Hospitalier de Cayenne, Cayenne, Guyane — France.
(3) University Hospital Aachen, Department of Ophthalmology Cologne Merheim, Cologne, Germany.
Correspondance : H. Merle, Service d'Ophtalmologie, Centre Hospitalier Universitaire de Fort de France, Hôpital Pierre Zobda-Quitman, BP632, 97261
Fort-de-France Cedex, Martinique - France (French West Indies). E-mail : harold.merle@chu-fortdefrance.fr
Texte issu de la Conférence-Débat « Traumatismes du globe oculaire » s'étant tenue au 113e congrès de la SFO en mai 2007.
Reçu le 13 décembre 2007. Accepté le 27 mai 2008.

Ocular burns
H. Merle, M. Gérard, N. Schrage
J. Fr. Ophtalmol., 2008; 31, 5: 1-12
Ocular or thermal burns account for 7.7% to18% of ocular trauma.Most victims are young people. Burns occur in accidents on work premises, at home, or during a physical attacks. The most serious injuries are due to chemical burns by strong acids or bases. Associated with the destruction of limbal stem cells, they generate recurrent epithelial ulcerations, chronic stromal ulcers, deep stromal revascularization, conjunctival overlap, or even corneal perforation. In presence of burning symptoms, it is sometimes difficult to perform the initial clinical exam. However, it enables the physician to classify the injury, establish a prognosis, and most importantly, guide the therapeutic management. The Roper-Hall modification of the Hughes classification system is the most widely utilized, it is broken down into grades based on the size of the stromal opacity and the extent of possible limbal ischemia. This classification is now favourably supplemented by those proposed by Dua and Wagoner, which are based on the extent of the limbal stem cell deficiency. The prognosis of the more serious forms of ocular burns has markedly improved over the last decade because of a better understanding of the physiology of the corneal epithelium. Surgical techniques aimed at restoring the destroyed limbal stem cells have altered the prognosis of severe corneal burns. In order to decrease the incidence of burns, prevention, particularly in industry, is essential.
Key-words:
Ocular burns, chemical burns, ocular wash, ocular irrigation, ocular surface, limbal transplant, amniotic membrane transplant.

INTRODUCTION

Ocular or thermal burns account for 7.7% to18% of ocular trauma[1-3]. In general burns are bilateral and hurt young males [4]. Accidents on work premises, at home or in leisure time cause most of burns. Among burns, the chemical ones are from far the most common. The severity of lesions depends on the causal agent. Chemical burns are feared by ophtalmologists because despite a correctly driven treatment, they may lead to a functional or even anatomic loss of the ocular globe. Thermal burns or burns by radiation usually generate superficial lesions.

Kindness translation made by PREVOR, Valmondois, France

OCULAR CHEMICAL BURNS

Epidemiology

Conditions of occurrence

In France, the knowledge of the epidemiology of burns is rather superficial and the only available data are about burns in general (about 500 000 burns/year), for which home and leisure accidents account for the majority of conditions of occurrence. Most are due to hot liquids or flames.

In one third of all cases cephalic region is altered [5]. There is no data about the ocular alteration alone. However the epidemiologic characteristics of ocular burns show as radically different because of most of them being chemical burns, whether they are caused by acids or bases. They also generate the most severe lesions with serious psychological, social and sometimes legal consequences. Chemical ocular burns account for 10 % of all ocular trauma [6,7]. In the United States, 5.2 % of the 2.5 million accidental exposures to a chemical show an ocular alteration [8]. In England, ocular burns account for 5 % of all burns, with an average 3 day stay in hospital [9]. In 1999 and 2000, ocular burns constituted 6.4 % of all emergencies in a hospital from the area of Paris where ophtalmological emergencies are treated [10].
In most cases ocular burns occur in the setting of industrial or home accidents. Their number varies alongside with the level of industrialization of a given area. In Germany, 73 % of ocular burns are connected to work accidents and come in equal proportions from agriculture, chemical and mechanical industries [11]. In Australia, work accidents account for 71 %, home accidents for 23 % and physical attacks for 2.5 % [12]. The share of home accidents due to DIY or gardening increases constantly and it accounts for more than one third in some series [4]. The share of physical attacks varies alongside with geographic areas and social and economical conditions. In London, there are 24 physical attacks out of 30 burns by bases reported by Davis *et al.* [13]. Such attacks account for more than one third in Martinique, Jamaica and Nigeria [14-16]. Most burns alter the face. Eyes and eyelids are altered in 19 % of cases. In Jamaica, as well as in Hong-Kong, attackers aim to disfigure victims [17]. In most cases, attackers use ammonia and consequences are tragic. Much less severe burns due to tear gases can account for more than a quarter of all ocular burns [10].

Chemical agents involved

Acids and bases are the most frequently involved chemical agents. Burns by acids and bases respectively account for 1.6 % and 0.6 % of all ocular trauma [7]. The gravity of lesions is influenced by the nature, concentration, quantity, time of exposure and ph of the chemical. Mainly bases are: ammonia (NH_3) used as cleaning, freezing or fertilizing agent, bleach (sodium hypochlorite), soda ($NaOH$) used as home detergent, potassium hydroxide (KOH) use as a fertilizer and lime ($Ca(OH)_2$) used as cement. Particles of soda and lime particularly stick to the conjunctiva, and doing so constitute a stock of toxic product.
Among all acids, sulfuric acid also called vitriol (H_2SO_4) causes the most serious accidents. It is used in textile industry and is a component of the liquid in car batteries. Like sulfuric acid, hydrofluoric acid (HF) that is used as a solvent onto rust, has got oxidative effects with very high toxicity. Hydrofluoric acid is used in glass industry, specifically for engraving. Chromic acid (Cr_2O_3) is used in metallurgy. Chlorohydric acid (HCl) is used in the production of organic compounds (vinyl chlorure…), metal scrapping, and even also for home cleaning. Highly concentrated acetic acid (CH_3COOH) causes severe lesions.
Other substances used in chemical industry are irritant or corrosive ; those are the compounds of D'autres substances utilisées dans l'industrie chimique sont irritantes ou corrosives ; ce sont : les composés du sosulphur, of chlore (swimming pool disinfection), solvents, detergents, pesticides …

Latex from plants (Euphorbiacea) is sometimes the origin of impressive burns that can lead to ocular perforation [18, 19].
Tear gases lead to a tearing that stops within a few hours.

Physiopathology

The natural development of a burn depends on the chemical involved; however all chemical buns possess some common elements of evolution. Thus, after a phase of initial sideration there is a phase of detersion of the lesions of necrosis, followed by a phase of scarring. A characteristic of detersion is the influx of inflammatory cells that are attracted by the products resulting from the degradation of the cornea and the conjunctiva (prostaglandins, leucotrienes…) [20]. Polynuclear neutrophile leukocytes secrete various detergent enzymes such as matricial metalloproteases (collagenases, gelatinases and stromelysin) which take part into the detersion and aggravate the destruction of ocular structures [21]. Scarring is made possible by the persistence of healthy tissues around the burnt area. Cicatrization applies to the ischemic lesions due to the destruction of the vascular network and to the lesions of corneal and conjunctival cells. Ischemic cells will trigger factors such as VEGF (Vascular Endothelial Growth Factor), TGF (Transforming Growth Factor) and FGF (Fibroblast Growth Factor). Those factors favour the proliferation of blood vessels into burnt tissues. When this neovascularization is a good opportunity for the conjunctiva, it reveals deleterious for both the cornea and the anterior chamber.
The scarring of the cornea and the conjunctiva can occur thanks to the mutation of surviving cells into fibroblasts and the division of stem cells. The appearance of a fibroblastic tissue is the origin of constitution of the symblepharons and opacification of the cornea. The filling of the iridocorneal angle by this tissue is coupled with an ocular hypertonia. Whether they are corneal limbal ones or conjunctival ones from the level of the fornix, stem cells enable the reconstruction of a normal corneal or conjunctival epithelium [22].
Bases quickly penetrate into ocular environments. The anion saponifies fatty acids of cellular membranes and instantly causes the death of epithelial cells. The cation facilitates the intraocular penetration of the base. Ammonium (NH_4^+) has the highest penetration speed [23].Pressure helps penetration too and it is moreover responsible for the mechanical destructuration of tissues. Above a 11.5 pH, ocular lesions due to bases develop quickly and are irreversible. pH of the anterior chamber changes within the seconds following the application of ammoniac. In addition to corneal lesions, iris, iridocorneal angle, ciliary body and crystalline lens may be altered. A complete destruction of the eyeball is possible [24]. Acids penetrate less quickly than bases. Protons (H^+) precipitate and denature proteins. Superficial cells and extracellular matrix are destroyed. The resulting superficial coagulation limits a deeper penetration of acid into the cornea *(fig. 1)*. However lesions due to strong acids are comparable to those due to bases because below a 2.5 pH lesions are deep and necrotizing. They are always coupled with lesions of the vessels of the conjunctiva and of the limbus as well as lesions of the corneal nerve endings which influence the prognosis [25].

Semiology

Ocular burns are emergencies and the initial clinical exam must quickly lead to the first therapeutic measures, and specifically to the ocular wash. It is an agreement to distinguish data from the initial examination and lesions that are observed during the evolution of burns.

Acute phase

Asking the victim a few questions brings accuracy about the conditions of occurrence of the accident, date and time of the trauma, the nature of the substances involved and the actions that have already been performed on the burn patient. Symptomatology may be very expressive (photophobia, tearing…) and pain may be important.

The instillation of anaesthetic collyrium helps to decrease the spasm of eyelids. Most often the eye is red because of a conjunctival hyperhemia, the presence of subconjunctival haemorrhages, of a perikeratic circle or of a chemosis. Burns of small importance only cause a superficial punctuate keratitis located in the opening zone between the eyelids or a wider ulceration of the corneal epithelium. In addition to the destruction of epithelium, the more serious burns are coupled with an edema that may look like porcelain, in the worst cases. *(fig. 2)*. A more important alteration is characterized by the presence of ulcerations, zones of ischemia or necrosis of the limbal region or of the bulbar conjunctiva *(fig. 3)*. Ischemic zones look white and oedematous because of the stopping of blood circulation into the vessels of the episclera and conjunctiva. They are often predominant in the lower part where the chemical concentrate. These severe forms are coupled with an inflammatory reaction of the anterior chamber, an ocular hypertonia and a corneal anesthesia. There are also some palpebral lesions, especially on the eyelid margin and on the lacrimal canaliculi. All of the clinical observations (ulceration of cornea and conjunctiva, zones of ischemia…) are to be reported on a diagram.

Classification of lesions

From the initial clinical data, a classification aims to set up a prognosis and to guide the therapeutic behavior. The most used classification is the one by Hughes, updated by Roper-Hall (*table I*) [26, 27]. It is made of 4 grades and is based on the importance of the stromal opacity and on the extent of the potential limbal ischemia. Grade 1 and 2 burns have a good prognosis when the prognosis of grade 3 and 4 burns is bad.
Roper-Hall classification is not accurate enough about the alteration of limbus for grade 4 burns and then using Wagoner or Dua classification is better (*table II*), both of which are based on the importance of the deficit of limbal stem cells [28, 29]. A Roper-Hall grade 4 burn can have a positive evolution when the limbal alteration rate is below 75 %. On the other hand, a complete destruction of limbus has a very bad prognosis [30, 31]. The new classification by Dua takes account of the alteration of limbus (not only the ischemic part of it) and conjunctiva. The alteration of limbus is measured in clock hours and the alteration of bulbar conjunctiva in percentage of surface. Using an analogical scale, with the number of hours and the percentage of conjunctival surface respectively being the first and second figures, enables an initial classification and the follow-up care of burns. Grades I, II and III have good prognosis. Grade IV burns, with 6 to 9 clock hours of limbal alteration and 50 to 75 % of conjunctival alteration, have a good or reserved prognosis. Grade V burns have a bad prognosis, and grade VI ones have a very bad prognosis. Grades IV, V and VI constitute grade IV in Roper-Hall classification, the prognosis of which is labeled as bad.

Figure 1 : Burn by acid : complete destruction of corneal epithelium, no alteration of corneal stroma and limbus region.
Figure 2 : Burn by base ; complete corneal ulceration, important corneal edema, limbal ischemia over 50 % of circumference.
Figure 3 : Burn by base : complete corneal ulceration, major corneal edema with no visualization of anterior segment, limbal ischemia over 75 % of circumference, necrosis of bulbar conjunctiva in the caruncle region.

Evolution

When superficial punctuate keratitis quickly heal, the cicatrisation of more severe affects is influenced by the deficit in limbal stem cells [29]. On a histological level, this appears as an invasion of the corneal surface by an epithelium of conjunctival type that is characterized by the presence of caliciform cells (*fig. 4*). The deficiency of limbal stem cells is the origin of recurrent or chronic epithelial ulcerations, a hazing, a neovascularization or even a perforation of cornea.
Conjunctival lesions cause an alteration of the lacrimal film and often lead to a retraction which is the origin of symblapherons. There are frequently intraocular complications in case of severe burns.

They are cataract, superinfection, intraocular inflammation, ocular hypertonia or ocular hypotonia [12, 32, 33], often associated with ocular dryness.
Eyelid lesions may become more complicated because of a districhiasis, an entropion or an ectropion.

Treatment

The therapeutic management of ocular burns has two targets. First it aims to eliminate or to limit the aggressiveness and the penetration of the irritant or corrosive substances into the ocular environment. This is the job of the ocular wash. In second hand it aims to control the inflammatory reaction and to favor cicatrization. This is achieved by medical and surgical treatments which are most of times closely intertwined.

Ocular wash

Although the surgical techniques, aiming to restore destroyed limbal stem cells, have remarkably improved the prognosis of severe corneal burns, ocular wash is still a crucial gesture. The future of the burn depends on its precocity and quality. It must be achieved as soon as possible on the accident scene. [34]. Because of pain or eyelid spasm, a wash carried out by the patient is not always efficient. So the medical management must systematically start the wash again, which is made easier by the instillation of an anesthetic collyrium. A general anaesthesia may be required for children. A wash must last for 15 minutes using about 1.5 liter of solution. It is necessary to make an eversion of the eyelids and to rinse thoroughly the conjunctival sacs. All the foreign bodies have to be removed, by using an operatory microscope.

Table I
Hughes Classification modified by Roper-Hall [26, 27].

Grade	*Prognosis*	*Corneal alteration*	*Limbal ischemia (% limbal circumference)*
1	Excellent	Epithelial alteration no corneal opacity	*0*
2	Good	Oedematous cornea but iris still visible	< 33 %
3	Reserved	Complete loss of corneal epithelium, stromal oedema disturbing visualization of details in iris	33 %-50 %
4	Bad	Opaque cornea, iris and pupil not visible	> 50 %

Table II
Dua classification [28].

Grade	*Prognosis*	*Limbal alteration in clock hours*	*Conjunctival alteration*	*Analogue scale*
I	Very good	0	0 %	0/0 %
II	Good	<3	< 30 %	0,1 à 3/1 à 29,9 %
III	Good	3 à 6	30 à 50 %	3,1 à 6/31 à 50 %
IV	Good to reserved	6 à 9	50 à 75 %	6,1 à 9/51 à 75 %
V	Reserved to bad	9 à 12	75 à 100 %	9,1 à 11,9/75,1 à 99,9 %
VI	Very bad	12	100 %	12/100 %

The examination of the conjunctival sacs must be a meticulous search of solid particles adhering o the conjunctiva (*fig. 5*).
Except the use of ethylenediamine tetraacetic acid (EDTA) to remove the particles of lime or cement, the use of antidotes is not recommended. The realization of a puncture associated with a wash of the anterior chamber is neither advised [1].
Most of times available on the accident scene, water is the most used universal wash solution. In comparison with the intraocular environment, water is hypotonic and then it can, thanks to epithelial lesions, penetrate into the corneal stroma, worsen the edema and drag acid or basic particles into the cornea. Using iso- or hypotonic solutions is better because they create a flow from the inside to the outside of the intraocular environment. Measured through the corneal thickness, the edema of the cornea is inversely proportional to the osmolarity of the rinsing solution [35]. Running water and saline solution have no buffer effect and only act by dilution and mechanical draining [36]. Ringer lactate and BSS (Balanced Saline Solution) are more tolerated than saline solution [7]. Ringer lactate has a very weak buffering potential [35]. BSS is different because of its osmolarity being close to the one of aqueous humor, but it has no buffering effect [20]. Solutions containing buffering phosphates are to be avoided because they may cause some irreversible corneal calcifications in the experimental field as well as in clinical conditions [37, 38]. Moreover, the action of buffering phosphates generates an exothermic reaction. Today there is only one hypertonic solution with a 820 mOms/l. It is an amphoteric solution with a high buffering potential (Diphotérine®,by Prevor laboratories) that has been used for several years in chemical industries. Amphoteric substances such as EDTA act by capture of ions and neutralization due to an amphophilic reaction. They can connect with bases or acids without modifying the environing pH and with no exothermic reaction. Washing a burn by base with an amphoteric solution restores the normal extraocular pH more quickly. The rise in the intraocular pH curve is less important and the decrease faster [35, 36, 39]. Because of their mechanisms of action and results in experimental as well as in clinical conditions, amphoteric solutions seem to be the best options for emergency wash of chemical burns [14].
The wash must be applied in case of thermic burns because it helps to lower the temperature on the surface of the eyeball [40]. In case of a severe burn, ocular wash must be continued during the patient's transfer to hospital. Using a transfusion tubulure kept around 20 centimeters from the eyeball is a better option than setting up an automatic irrigation system. These systems are made of a polyethylene ring or a polymethylmethacrylate scleral lens (Morgan lens). They have the disadvantage of being difficult set up, causing iatrogenic lesions and not guaranteeing a good rinsing of all the ocular surface [41]. The wash of lacrimal ways is not advised, particularly because of the fast appearence of an edema of lacrimal meati, the risk of worsening of meatic lesions and the risk of false passage. Burns of the lacrimal punctum may require successive dilatations, and even the set up of an intubation [29].
pH can be measured by using a paper indicator. Wash is to be continued until pH of the ocular surface gets back to normal.

Figure 4: Burn by base: complete covering of corneal surface by an epithelium of conjunctival type.
Figure 5: Ablation of lime particles adhering to palpebral conjunctiva.

Medical treatment

Control of the inflammatory reaction

Now local corticoids seem to be accepted in the treatment of burns, and particularly chemical burns [12]. Even if it has been discussed for a long time, their use is justified by their capacity to reduce the inflammatory reaction. They restrain stromal invasion by polynuclear neutrophile leukocytes, stabilize cellular and lysosomal membranes and have an anticollagenase action.
In presence of chemical burns, corticoids limit the destruction of mucus cells of the conjunctiva [42]. However, they limit the migrationvof keratocytes, inhibate the synthesis of collagen and delay cicatrization [43]. Donshik *et al.* [44] have shown that for animals an intensive use of local corticoids within the ten first days after burn does not increase the risk of corneal perforation. Therefore they have advised to reintroduce corticoids from the fourth week in case of persistence of an inflamatory reaction. When they are associated with ascorbic acid and introduce by systemic way, Davis *et al.* [13] have stated that corticoids can be prescribed beyond 8 days with beneficial results. The incidence of infectious complications does not seem to increase with local corticotherapy [12, 14, 42].
In association with or in substitution of corticoids, the use of non steroid anti-inflammatory drugs has been suggested. However they should be advised against because they increase the delay of epithelial cicatrization and modify the sensitivity of the cornea [45].
Tetracyclines reduce the activity of collagenases and the appearence of corneal ulcerations in experimental burns. This action is independent from their antimicrobial action and is due to a chelation of zinc that is necessary for the activity of metalloproteinases [46]. They also reduce the activity of polynuclear leukocytes. When administered by systemic route, tetracyclines reduce the incidence of corneal ulceration and facilitate their cicatrization [47].
Cycloplegic collyriums reduce pain and limit the formation of synechiae in iris and lens. They must be systematically prescribed.
Citrate is a calcium chelator. It lowers the chimiotactism of polynuclear neutrophile leukocytes, the adherence as well as the release of lysosomal enzymes. It restrains the activity of collagenases and reduces the incidence of corneal ulcerations in rabbits. Citrate is more efficient when given by local route then by systemic route [48, 49]. Its use in 10 % collyrium associated with 10 % ascorbate would help a faster reepithelialization of severe burns [12]. Citrate is not available in France. Inhibitors of collagenases, such as cysteine, acetylcysteine and synthetic thiols have proved efficient on experimental burns but have been clinically studied [50].

Potentialization of cicatrization

Burns of the conjunctiva are associated with a decrease of the number of mucus cells. The alterations of the mucous layer of the lacrimal film reduce the adherence of the lacrimal film onto the ocular surface and play a part into the weakening of the corneal epithelium. The regular application of free of conservative artificial tears is recommended and can be potentialized by the temporary or permanent obstruction of the lacrimal puncti.
With its rate decreasing in aqueous humor in case of a burn, ascorbatee (or acid ascorbic) is a cofactor of the synthesis of collagen. Pfister and Paterson [51] have shown that a supplementation of ascorbic acid administered by either local or systemic route reduces the incidence of the thinning and of the ulcerations of the cornea in experimental as well as in clinical conditions. The administration of 10 % collyrium would be more efficient because of the potential lesions to the ciliary body that limit the concentration of ascorbic acid into the anterior chamber. For Brodovski *et al.* [12], the addition of ascorbate enables the use of local corticoids beyond the first week. However, it seems to be less efficient in case of an existing ulcer. Ascorbate as a collyrium is no more available in France. It may be prescribed by oral route, between 2 and 3 grams per day [12, 14, 30].
Common clinic does not use growth factors (EGF, FGF…) such as fibronectin and retinoic acid.
Prevention of infections is guaranteed by the administration of a wide spectrum antibiotic collyrium and by tetracyclines given by systemic route.

Contact lenses are little applied because they are difficult to bear and because they would facilitate superinfections.
Antalgic drugs are often prescribed, either by oral or systemic route, because corneal nerve lesions may be associated with violent pain.

Surgical treatment

The prognosis of serious types of ocular burns has remarkably improved during the last decade thanks to a better knowledge of both the physiology of corneal epithelium and the physiopathology of burns. Aiming to restore destroyed limbal stem cells, surgical techniques have notably improved the prognosis of severe corneal burns.

Excision of necrotic tissues

As well as for local corticotherapy, excision aims to reduce the inflamatory reaction due to the degradation products of a necrotic conjunctiva that contribute to the detersion of the area. Thus it restrains the generation of free oxygen cytotoxic radicals. It also enables the removal of the caustic material trapped in these tissues. Excision must be practiced as soon as the rinsing of the eyeball and the ablation of potential foreign bodies are achieved. It consists in the ablation of necrotic tissues from the eyeball surface. Excision of conjunctiva and subconjunctival tissue may be practiced up to superior and inferior fornices, when necessary. Only necrotic and avascular tissues are removed until reaching layers of tissues in which blood traffic has not been altered. Even when ischemic, sclera tissue and cornea must be preserved [52-54].

Tenoplasty

When serious ocular burns are associated with a complete loss of limbal vascularization, in addition to the predictable impossibility of secondary reepithelialization, there is an immediate risk of necrosis of the anterior segment. Tenoplasty is a good option in order to restore a limbal circulation and to prevent the evolution of a necrosis or of an aseptic ulceration. It consists in a limbus based advancing Tenon's flap [52, 54-57]. The intervention must be driven early, just after the ablation of necrotic tissues has been achieved.

Preventing the formation of symblepharons

In all cases of extended burns of the conjunctiva, prevention of the formation of symblepharons is thought about. Several methods can be applied. One is the regular release of adherences from the conjunctival sacs using a glass stick or a swab which is to be realized under local a naesthesia.
Another one is the set up of scleral glasses or of a polymethylmethacrylate ring. Yamada *et al.* [58] suggest the set up of a gelatin sponge in the conjunctival sac.

Limbus transplantation

Suggested by Schermer *et al.* [59] and developed by Tseng [22], the theory of limbus stem cells (LSC) is the base of limbus transplantation.
The corneal epithelium quickly regenerates from undifferentiated LSC that are located in the basal layers of the limbus.
The limbus auto-transplantation is the top technique for the treatment of the destruction of corneal limbus and its complications [60]. The technique of conjunctiva and limbus transplantation was described by Kenyon and Tseng in 1989 [61]. It applies to unilateral limbus deficiencies when the controlateral side offers a safe donor eye. All of the conjunctival pannus which covers the corne is removed beyond the limbus for about 3 mm. Dissection starts from the level of the cornea and follows towards the conjunctiva [62]. The transplant is taken from a corneal incision that is practiced

1 mm before the limbus. Dissection operates a centrifugal tunnelization of about 2 mm at the back of the limbus. In order to generate no limbus deficiency on the donor eye, the transplant must be shorter than 180° [63]. The transplant is sutured to the receiving site onto the cornea using 10/0 nylon separate points and onto the conjunctiva with 8/0 resorbable thread. The limbus autotransplantation enables to get a good quality corneal reepithelialization for 75 to 100 % of cases and the constitution of a barrier preventing cicatricial neovascular events of conjunctival origin [61, 64, 65]. The date of operation after burn is questioned. For most authors, it is a better option to wait several months for the inflammatory reaction to decrease. Other authors suggest an earlier intervention, that is before the appearence of complications due to the LSC deficiency [29, 31].
The limbus allotransplantation has the same target as the autotransplantation [66]. The limbus allotransplantation applies to bilateral extended limbus lesions or to unilateral lesions on a single eye. The tissue is taken from a corneal transplant or an eye that are kept in a tissue bank. The limbus allotransplantation is associated with a major risk of rejection, this requires an extended immunosuppression.

Amniotic membrane transplantation

Used as soon as 1947 by Sorsby and Simmonds [67] for ocular burns, amniotic membrane is a tissue located at the interface between placenta and amniotic fluid. It is made of a unistratified epithelium, a basal strip and an avascular mesenchyma.
Amniotic membrane facilitates reepithelialization by reducing the inflammatory and cicatricial reaction [68]. It favors the migration of epithelial cells and the adherence of basal cells [69]. It behaves like an actual substitution basal membrane and favors the epithelial phenotypic expression. As it has no class II HL-A antigen, amniotic membrane has no rejection reaction. With its epithelial side upwards, the piece of amniotic membrane is sutured to the cornea with 10/0 nylon separate points. Several layers can be put one on each other. The amniotic membrane is covered by the corneal epithelium, then integrated to the stroma and absorbed. It may be applied in order to restore the conjunctival sacs after exeresis of the symblepharons [69, 70].
The current trend is the early realization of the amniotic membrane transplantation, in the early phase of the burn. Reepithelialization is said to be over 80 % within 15 days, visual acuity improved in 77 % of cases and symblepharons rare [71]. When it is realized later, there are still good results [72].
The amniotic membrane transplantation does not suffice for the treatment of severe LSC deficiencies due to burns [73]. In such a case, it must be associated with an LSC transplantation. The amniotic membrane is first sutured to the surface of the deepithelialized cornea and the limbus transplant is sutured onto the edge of the circumference of the amniotic membrane [74].

Keratoplasties

An 11 to 12 mm diameter transfixing keratoplasty (TK) provides a double advantage. It is an architectonic or optic TK and it gives some LSC [75, 76]. However it is associated with a high risk of rejection which alters its experimental results. Instead, it is better to operate a preliminary LSC transplantation to be followed by a classic diameter TK. The rate of TK rejection is usually about 10 %. It is bigger for chemical burns, because of the frequency and importance of the stromal neovascularization of the receiving cornea [77-79]. TK do not bring LSC. Therefore it does not suffice for the treatment of extended limbal ischemia. It must be associated with a limbus transplantation [80]. TK can be operated at the same operatory time as limbus allotransplantation [81]. However epithelial cicatrisation and corneal transparency are better when the TK is realized in a second time (between 1 and 13 months after burn) [60].
An auto-TK associated with a limbus autotransplantation can be exceptionally realized as shown on *figure 6*. It shows a left eye monophtalmic patient with a Grade IV burn due to an alkali. Two TK had resulted in a failure because a successive rejection. The left eye wisual acuity was restrained to a good light localization. Cornea was white, neovascularized and ulcerated. There was a complete limbal deficiency. The right has not been working since the patient was a child because of a closed

contusion. In the same operatory time, both the cornea (8 mm trepanation) and the limbus (on 360 °) have been taken from the right eye. After ablation of the conjunctival pannus that was covering the left eye limbus and cornea, the cornea and limbus sampled from the right eye have been transplantanted.
Deep lamellar keratoplasties (DLP) consist in transplanting the stroma and epithelium of the transplant while respecting the Desecmet's membrane and endothelium of the receiver. It applies to corneal burns with no alteration of the Descemet's membrane and endothelium. The rate of rejection is less with DLP than with TK, even when the cornea is very neovascularized [82]. Vajpayee *et al.* suggested big diameter LK in 2000 [83]. Big diameter LP give LSC and enables the reepithelialized ovular surface to get steady. It is recommended when burns have not altered the deep layers of cornea. Keratoprosthesis surgeries are the last surgical treatment of bilateral corneal blindness, when TK and LSC transplantations cannot be performed anymore. Although they are difficult to set up, they are still performed car their results are sometimes very encoraging [84].

Transplantation of cultivated epithelial limbus cells

The autotransplantation of cultivated epithelial limbus cells onto an amniotic membrane is a recent technique. It has been developed in Taiwan byb Tsai *et al.* [85]. A 1 x 2 ml fragment of limbal epithelium is sampled from the safe eye. Then it is cultivated for 3 weeks on an amniotic membrane. The epithelial tissue is transplanted with amniotic membrane onto the receiving cornea, after excision of the fibrovascular tissue. The same culture technique has been used by Shimazaki *et al.* [86], but only for allotransplantations. The short term prognosis of a TK after transplantation of epithelial limbus cells is said to be good [87].

Figure 6 : (a) Right eye. Preoperatory look: clear cornea, quiet anterior segment, ancient posterior synechiae, surgical extracapsular aphakia. No light perception. Normal ocular tonicity. (b) Left eye. Preoperatory look : white oedematous, ulcerated and neovascularized cornea. Limbus deficiency due to a destruction of limbus over 360°. Visual acuity limited to a good orientat ion towards light. (c) Riht eye. One month after operation. Dystrophic cornea from left eye sutured by 16 10/0 nylon separate points. (d) Left eye. One month after operation. Clear cornea from right eye sutured by 16 10/0 nylon separate points. 360° limbus transplan tation, with a sample from right eye, sutured to the corneal side by 8 10/0 nylon separate points and to the conjunctival side by 8 8/0 vicryl separate points.

Conjunctival transplantation, using nasal or buccal mucous membrane samples

Initially developed by Thoft [88], conjunctival transplantation does not achieve the cicatrisation of corneal epithelium, for which it has been supplanted by LSC transplantations. However, it is still recommended for the restoration of conjunctival sacs altered by cicatricial fibrosis. The buccal mucous membrane transplant is usually sampled from the posterior side of the upper or lower lip. The buccal mucous membrane may be used for the treatment of symblepharon, trichiasis, distichiasis, entropion or keratinized zone of conjunctiva or palpebral margin [89]. The transplantation of nasal mucous membrane has been first suggested by Naumann *et al.* [90] in the treatment of bilateral severe conjunctival mucous deficiencies. The transplant of nasal mucous membrane is sampled from the septum, the inferior or middle nasalis concha. The advantage of the nasal mucous membrane transplantation is the possibility to sample big sized transplants and the transplantation of intraepithelial mucus cells.

Preventive treatment

Preventive treatment mainly applies to any field of industry. Chemical industry is a particularly hazardous environment because of the handling of very concentrated substances, with an accident risk present from the reception of raw materials up to the expedition of manufactured products. In addition to perfect ventilation of work premises and well planned installation of machines, protection of individuals is crucial. It is based on the training about chemical hazards, on the elaboration of a

standard protocole for the management of victims which is known by all staff, on the wearing of protection glasses, on the generalization of installation of eye rinsing solutions in hazardous areas, and even on the distribution and use of an individual rinsing solution.
Labeling of hazardous chemical products and preparations is often the first available information. It is crucial because it displays information about hazards that might occur as well as about precautions to be taken. Many burns are caused by the manipulation of packaging and bottles, and specifically by their opening. The packaging is often a soft pack or a plastic bottle which are easy to squeeze and have no opening safety system. As already highlighted by Pouliquen in 1972 [91], this kind of packaging is not suitable at all to the hazard potential of such products.

THERMAL AND RADIATION BURNS

Thermal burns

There are only 1 to 5 % cases of ocular alteration out of all thermal burns. Most of them are burns by flames or hot liquids occurring in home accidents. The severity depends on both the temperature and time of exposure. Thanks to the winking speed and to the Charles Bell's phenomenon, the eyeball is protected and so burns by flames only alter eyelashes, eyebrows and eyelids. However around the palpebral opening there is an ulceration of cornea and conjunctiva. Sometimes it is associated with an opacification of stroma and some sings of limbal stem cell deficiency. Considering burns by contact, solids, that keep the heat in, and bodies with a high melting point cause deep lesions, and sometimes even lead to the loss of eyeball. Most severe ocular lesions come from patients with Grade III cutaneous burns [92]. The treatment of superficial lesions is a combination of local antibiotherapy, instillation of artificial tears, occlusive dressing and sometimes a cycloplegy.
There is often complication of the retractile palpebral scars because of the appearence of trichiasis, entropion or ectropion.

Radiation burns

The most frequent are burns by ultraviolet rays. The range of sources is wide: extended exposure to very reflected sunbeams (snow, sea, desert), arc welding, disinfecting or tanning devices. Ultraviolet rays are almost completely absorbed by the cornea in which they cause an epithelial cell detachment and a stromal oedema. It is around 12 hours after exposure that pain, blepharospasm, tearing and photophobia appear. There is a superficial punctuate keratitis and a hyperhemia of conjunctiva. Healing is achieved in 48 hours and is made easier by the ocular occlusion. In order to prevent secondary infection, a local antibiotherapy must be prescribed.
Burns by infrared radiation (explosions, solar eclipses…) only generate a supeficial punctuate keratitis to the cornea, but may lead to cataract or chorioretinitis.

CONCLUSION

Chemical burns may cause a severe and irreversible bilateral alteration of the visual function. It is sometimes difficult to perform the initial clinical examination because of the presence of a noisy symptomatology. However it enables the classification of lesions, the elaboration of a prognosis and the guiding of the therapeutic management. The prognosis of severe ocular burns has remarkably improved in last decade thanks to a better knowledge of the physiology of the corneal epithelium. Surgical techniques aiming to restore the destroyed limbal stem cells have modified the prognosis of severe corneal burns. In spite of this, in order to restrain the incidence of burns, prevention, particularly in the industrial environment, is crucial because many tragic cases could be avoided thanks to basic information, training and regulation.

Kindness translation made by PREVOR, Valmondois, France

REFERENCES

1. Pfister RR. Chemical injuries of the eye. Ophthalmology 1983;90:1246-53.
2. Liggett P. Ocular trauma in an urban population. Ophthalmology 1989;97: 581-4.
3. Zagelbaum B, Tostanowski J, Kerner D, Hersh PS. Urban eye trauma. Ophthalmology 1993;100:851-6.
4. Morgan SJ. Chemical burns of the eye: causes and management. Br J Ophthalmol 1987;71:854-7.
5. Wasserman D. Épidémiologie et organisation de la prise en charge des brûlés en France. Brûlures 2000;4:194-200.
6. Jones NP, Hayward JM, Khaw PT, Claoue CM, Elkington AR. Function of an ophthalmic accident and emergency department: results of a six months survey. Br Med J 1986;292:188-90.
7. Herr RD, White GL, Bernhisel K, Mamalis N, Swanson E. Clinical comparison of ocular irrigation fluids following chemical injury. Am J Emerg Med 1991; 9:228-31.
8. Lai MW, Klein-Schwartz W, Rodgers GC, Abrams JY, Haber DA, Bronstein AC et al. 2005 Annual Report of the American Association of Poison Control Centers' National Poisoning and Exposure Database. Clin Toxicol 2006;44:803-932.
9. Wilkinson E. The epidemiology of burns in secondary care, in a population of 2.6 million people. Burns 1998;24:139-43.
10. Girard B, Bourcier-Bareil F, Agdabede I, Laroche L. Activité et épidémiologie d'un centre d'urgence en ophtalmologie. J Fr Ophtalmol 2002;25:701-11.
11. Kuckelkorn R, Makropoulos W, Kottek A, Reim M. Retrospektive betrachtung von schweren alkaliverätzungen der Augen. Klin Monatsbl Augenheilkd 1993;203: 397-402.
12. Brodovsky SC, McCarty CA, Snibson G, Loughnan M, Sullivan L, Daniell M et al. Management of Alkali burns. An 11-year retrospective review. Ophthalmology 2000;107:1829-35.
13. Davis AR, Ali QH, Aclimandos WA, Hunter PA. Topical steroid use in treatment of ocular alkali burns. Br J Ophthalmol 1997;81:732-4.
14. Merle H, Donnio A, Ayéboua L, Michel F, Thomas F, Ketterle J et al. Alkali ocular burns in Martinique (French West Indies). Evaluation of the use of an amphoteric solution as the rinsing product. Burns 2005;2:205-11.
15. Branday J, Arscott GDL, Smoot EC, Williams GD, Fletcher PR. Chemical burns as assault injuries in Jamaica. Burns 1996; 22:154-155.
16. Ukponmwan CU. Chemical injuries to the eye in Benin City, Nigeria. West Afr J Med 2000;19:71-6.
17. Young RC, Ho WS, Burd YA. Chemical assaults in Hong Kong: a 10-year review. Burns 2002;28:651-3.
18. Merle H, Trode M, Richer R, Ayéboua L, Jallot Sainte-Rose N. Brûlures oculaires par le latex de mancenillier. J Fr Ophtalmol 1995;18:461-7.
19. Sofat BK, Sood GC, Chandel RD, Mehrotra SK. Euphorbia royleana latex keratitis. Am J Ophthalmol 1972;74:634-7.
20. Kuckelkorn R, Keller G, Redbrake C. Emergency treatment of chemical and thermal eye burns. Acta Ophthalmol Scand 2002;80:4-10.
21. Schultz GS, Strelow S, Stern GA, Chegini N, Grant MB, Gallardy RE et al. Treatment of alkali-injured rabbit corneas with a synthetic inhibitor of matrix metalloproteinases. Invest Ophthalmol Vis Sci 1992;33:3325-31.
22. Tseng SC. Concept and application of limbal stem cells. Eye 1989;3:141-57.
23. Mc Culley J. Chemical injuries. In: Smolin G, Thoft RA. The cornea. Boston: Little, Brown and Co., 1983: p. 422-35.
24. Gérard M, Merle H, Ayéboua L, Richer R. Étude prospective sur les brûlures par bases au CHU de Fort de France. J Fr Ophtalmol 1999;22:834-47.
25. Kuckelkorn R, Schrage N, Reim M. Poor prognosis of severe chemical and thermal burns. The need for adequate emergency care and primary prevention. Int Arch Occup Environ Health 1995;67:281-4.
26. Hughes WF. Alkali burns of the cornea. I. Rewiew of the literature and summary of present knowledge. Arch Ophthalmol 1946;35:423-36.
27. Roper-Hall MJ. Thermal and chemical burns of the eye. Trans Ophthalmol Soc UK 1965;85:631-46.
28. Dua HS, King AJ, Joseph A. A new classification of ocular surface burns. Br J Ophthalmol 2001;85:1379-83.
29. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. Surv Ophthalmol 1997;41: 275-313.
30. Gérard M, Merle H, Chiambaretta F, Rigal D, Schrage N. An amphoteric rince used in the emergency treatment of a serious ocular burn. Burns 2002;7:670-3.
31. Morgan SJ, Murray A. Limbal autotransplantion in the acute and chronic phases of severe chemical injuries. Eye 1996;10: 349-54.
32. Beare JDL. Eye injuries from assault with chemicals. Br J Ophthalmol 1990;74: 514-8.
33. Kuckelkorn R, Kottek A, Reim M. Intraocular complications after severe chemical burns: incidence and surgical treatment. Klin Monatsbl Augenheilkd 1994;205: 86-92.

34. Burns FR, Paterson CA. Prompt irrigation of chemical eye injuries may avert severe damage. Occup Health Saf 1989;58:33-6.

35. Kompa S, Schareck B, Tympner J, Wüstemeyer H, Schrage N. Comparison of emergency eye-wash products in burned porcine eyes. Graefe's Arch Clin Exp Ophthalmol 2002;240:308-13.

36. Schrage NF, Kompa S, Haller W, Langefeld S. Use of an amphoteric lavage solution for emergency treatment of eye burns. First animal type experimental clinical considerations. Burns 2002;28: 782-6.

37. Schrage NF, Schlossmacher B, Aschenbernner W, Langefeld S. Phosphate buffer in alkali eye burns as an inducer of experimental corneal calcification. Burns 2001;27:459-64.

38. Kompa S, Redbrake C, Dunkel B, Weber A, Schrage N. Corneal calcification after chemical eye burns caused by eye drops containing phosphate buffer. Burns 2006;32:744-7.

39. Gérard M, Josset P, Louis V, Ménérath JM, Blomet J, Merle H. Existe-il un délai pour le lavage oculaire externe dans le traitement d'une brûlure oculaire par l'ammoniaque ? Comparaison de deux solutions de lavage : sérum physiologique et Diphotérine*. J Fr Ophtalmol 2000;5:449-58.

40. Schrage NF, Flick S, Von Fischern T, Reim M, Wenzel M. Temperaturveränderungen der Hornhaut durch Anlegen eines Augenverbandes. Ophthalmologe 1997;94:492-95.

41. Morgan SJ. A new drug delivery system for the eye. Industrial Med 1971;40:11-3.

42. Brent BD, Karcioglu ZA. Effect of topical corticosteroids on goblet-cell density in an alkali-burn model. Ann Ophthalmol 1991;23:221-3.

43. Chung JH, Kang YG, Kim HJ. Effects of 0.1% dexamethasone on epithelial healing in experimental corneal alkali wounds: morphological changes during the repair process. Graefe's Arch Clin Exp Ophthalmol 1998;236:537-45.

44. Donshik PC, Berman MB, Dohlman CH, Gage J, Rose J. Effect of topical corticosteroids on ulceration in alkali-burned corneas. Arch Ophthalmol 1978;96: 117-21.

45. Struck HG, Geiser H, Block HU, Mest HJ, Tost M. Leukotriene antagonist S872419 for early-phase treatment of chemical burn in the rabbit eye. Eur J Ophthalmol 1991;1:137-41.

46. Perry HD, Hodes LW, Seedor JA, Donnenfeld ED, McNamara TF, Golub LM. Effect of doxycycline hyclate on corneal

epithelial wound healing in the rabbit alkali-burn model. Preliminary observations. Cornea 1993;12:379-82.

47. Seedor JA, Perry HD, Mc Namara TF, Golub LM. Systemic tetracycline treatment of alkali induced corneal ulceration. Arch Ophthalmol 1987;105:268-72.

48. Pfister RR, Haddox JL, Paterson CA. The efficacy of sodium citrate in the treatment of severe alkali burns of the eye is influenced by the route of administration. Cornea 1982;1:205-11.

49. Pfister RR, Haddox JL, Snyder TL. Topical citrate inhibits the adherence of neutrophils in postcapillary venules. Cornea 1990; 9:238-45.

50. Burns FR, Gray RD, Paterson CA. Inhibition of alkali-induced corneal ulceration and perforation by a thiol peptide. Invest Ophthalmol Vis Sci 1990;31:107-14.

51. Pfister RR, Paterson CA. Ascorbic acid in the treatment of alkali burns of the eye. Ophthalmology 1980;87:1050-7.

52. Reim M, Overkamping B, Kuckelkorn R. Two-year experience with Tenon-plasty. Ophthalmologe 1992;89:534-40.

53. Reim M, Kuckelkorn R. Chemical and thermal lesions in the orbital region. Bull Soc belge Ophtalmol 1992;245:21-8.

54. Reim M, Teping C. Surgical procedures in the treatment of most severe eye burns. Acta Ophthalmol Scand 1989;67:47-54.

55. Teping C, Reim M. Tenoplasty as a new surgical principle in the early treatment of the most severe chemical eye burns. Klin Monatsbl Augenheilkd 1989;194:1-5.

56. Kuckelkorn R, Redbrake C, Reim M. Tenoplasty: a new surgical approach fort the treatment of severe eye burns. Ophthalmic Surg Lasers 1997;28:105-10.

57. Kuckelkorn R, Schrage N, Reim M. Treatment of severe eyes burns by tenoplasty. Lancet 1995;345:657-8.

58. Yamada M, Sano Y, Watanabe A, Mashima Y. Preventing symblepharon formation with a gelatine sponge in the eye of a patient with an alkali burn. Am J Ophthalmol 1997;123:552-4.

59. Schermer A, Galvin S, Sun TT. Differentiation-related expression of a major 64K corneal keratin *in vivo* and in culture suggests limbal location of corneal epithelial stem cells. J Cell Biol 1986;103: 49-62.

60. Shimazaki J, Shimmura S, Tsubota K. Donor source affects the outcome of ocular surface reconstruction in chemical or thermal burns of the cornea. Ophthalmology 2004;111:38-44.

61. Kenyon KR, Tseng SCG. Limbal autograft transplantation for ocular surface disorders. Ophthalmology 1989;96:709-23.

62. Gérard M, Merle H, Chiambaretta F, Louis V, Richer R, Rigal D. Technique chirurgicale de l'autotransplantation limbique dans les brûlures oculaires graves et récentes. J Fr Ophtalmol 1999;22:502-6.
63. Holland JH, Schwartz GS. The evolution of epithelial transplantation for severe ocular surface disease and a proposed classification system. Cornea 1996;15: 549-56.
64. Rao SK, Rajagopal R, Sitalakshmi G, Padmanabhan P. Limbal allografting from related live donors for corneal surface reconstruction. Ophthalmology 1999; 106:822-28.
65. Frucht-Pery J, Siganos CS, Solomon A, Scheman L, Brautbar C, Zauberman H. Limbal cell autograft transplantation for severe ocular surface disorders. Graefes Arch Clin Exp Ophthalmol 1998;236: 582-7.
66. Tsai RJ, Tseng SC. Human allograft limbal transplantation for corneal surface reconstruction. Cornea 1994;13:389-400.
67. Sorsby A. Simmonds H. Amniotic membrane graft in caustic burns of the eye (burns of second grade). Br J Ophthalmol 1947;31:409-18.
68. Hao Y, Ma DH, Hwang DG, Kim WS, Zhang F. Identification of antiangiogenic and antiinflammatory proteins in human amniotic memnbrane. Cornea 2000;19: 348-52.
69. Tseng SC, Prabhasawat P, Lee SH. Amniotic membrane transplantation for conjunctival surface reconstruction. Am J Ophthalmol 1997;124:765-74.
70. Solomon A, Pires RT, Tseng SC. Amniotic membrane transplantation after extensive removal of primary and recurrent pterygia. Ophthalmology 2001;108:449-60.
71. Meller D, Pires RT, Mack RJ, Figueiredo F, Heiligenhaus A, Park WC et al. Amniotic membrane transplantation for acute chemical or thermal burns. Ophthalmology 2000;107:980-90.
72. Ucakhan OO, Koklu G, Firat E. Nonpreserved human amniotic membrane transplantation in acute and chronic chemical eye injuries. Cornea 2002;21:169-72.
73. Hanada K, Shimazaki J, Shimurra S, Tsubota K. Multilayered amniotic membrane transplantation for severe ulceration of the cornea and sclera. Am J Ophthalmol 2001;131:324-31.
74. Stoiber J, Muss WH, Pohla-Gubo G, Ruckhofer J, Grabner G. Histopathology of human corneas after amniotic membrane and limbal stem cell transplantation for severe chemical burn. Cornea 2002;21: 482-9.
75. Redbrake C, Buchal V, Reim M. Keratoplasty with a scleral rim after most severe eye burns. Klin Monastsbl Augenheilkd 1996;208:145-51.
76. Kuckelkorn R, Keller G, Redbrake C. Long-term results of large diameter keratoplasties in the treatment of severe chemical and thermal eye burns. Klin Monatsbl Augenheilkd 2001;218:542-52.
77. Beekhuis WH. Current clinician's opinions on risk factors in corneal grafting. Cornea 1995;14:39-42.
78. Mattax JB, Mc Culley JP. Corneal surgery following corneal burns. Int Ophthalmol Clin 1988;28:76-82.
79. Brown SI, Bloomfield SE, Pearce DB. Follow-up report on transplantation of the alkali burned cornea. Am J Ophthalmol 1974;77:538-42.
80. Zieske JD. Perpetuation of stem cells in the eye. Eye 1994;8:163-9.
81. Tsubota K, Toda I, Saito H, Shinozaki N, Shimazaki J. Reconstruction of the corneal epithelium by limbal allograft transplantation for severe ocular surface disorders. Ophthalmology 1995;102:1486-96.
82. Fogla R, Padmanabhan P. Deep anterior lamellar keratoplasty combined with autologous limbal stem cell transplantation in unilateral severe chemical injury. Cornea 2005;24:421-5.
83. Vajpayee RB, Thomas S, Sharma N, Dada T, Tabin GC. Large-diameter lamellar keratoplasty in severe ocular alkali burns. Ophthalmology 2000;107:1765-8.
84. Stoiber J, Forstner R, Csaky DC, Ruckhofer J, Grabner G. Evaluation of bone reduction in osteo-odontokeratoprosthesis by three-dimensional computed tomography. Cornea 2003;22:126-30.
85. Tsai RJ, Li LM, Chen JK. Reconstruction of damaged corneas by transplantation of autologous limbal epithelial cells. N Engl J Med 2000;343:86-93.
86. Shimazaki J, Aiba M, Goto E, Kato N, Shimmura S, Tsubota K. Transplantation of human limbal epithelium cultivated on amniotic membrane for the treatment of severe ocular surface disorders. Ophthalmology 2002;109:1285-90.
87. Sangwan VS, Matalia HP, Vemuganti GK, Ifthekar G, Fatima A, Singh S et al. Early results of penetrating keratoplasty after cultivated limbal epithelium transplantation. Arch Ophthalmol 2005;123:334-40.
88. Thoft RA. Conjunctival transplantation. Arch Ophthalmol 1977;95:1425-7.
89. Shore JW, Foster CS, Westfall CT, Rubin PA. Results of buccal mucosal grafting for patients with medically controlled ocular cicatricial pemphigoid. Ophthalmology 1992;99:383-95.
90. Naumann GO, Lang GK, Rummelt V, Wigand ME. Autologous nasal mucosa transplantation in severe bilateral conjunctival mucus deficiency syndrome. Ophthalmology 1990;97:1011-7.

91. Pouliquen Y. Les brûlures de la cornée.
Clin Ophtalmol 1972;5:21.
92. Bouchard CS, Morno K, Perkins J, McDonnell
JF, Dicken R. Ocular complications of
thermal injury: a 3-year retrospective. J
Trauma 2001;50:79-82.