Cystoid macular oedema with trypan blue use

We present a large comparative series of trypan blue use in cataract surgery. This series of trypan blue used in all eyes regardless of cataract severity may be unique. We found an apparent increased rate of cystoid macular oedema (CMO) associated with trypan blue use.

Melles et al.'s report on the use of trypan blue in cataract extraction in 1999 combined with Apple et al.'s series on dye enhanced cataract surgery facilitated widespread acceptance of this technique. The dye has been shown to stain basement membrane of lens capsule. Trypan blue is now widely used to assist in cataract extraction when visualisation of the anterior capsule is poor because of loss of red reflex. Trypan blue has also been used to improve contrast during cataract extraction in eyes with corneal opacities and to stain internal limiting membrane and epiretinal membrane during vitreoretinal surgery. The safety profile of trypan blue appears good with no adverse effects reported in several large series.

Patients and methods

In this retrospective, comparative study we identified a consecutive series of 75 patients (group A) in whom trypan blue had been used “routinely” regardless of cataract type or density. A consecutive series of 94 patients (group B) who had routine phacoemulsification by the same surgeon were used as a control group.

Apart from the use of trypan blue to facilitate capsulorhexis, standard phacoemulsification techniques were used in both groups.

The data from the two cohorts were compared using mean and standard deviations for continuous variables such as age, and proportions for categorical variables such as sex. For acuity a numeric ordinal score was created from 1 to 10 by placing all the recorded acuities in order. This numeric ordinal score allowed us to plot the data using box plots, and to analyse the data using non-parametric methods to produce p values where necessary.

Table 1: Age and sex distribution and co-morbidity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A (n = 75)</th>
<th>Group B (n = 94)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age</td>
<td>79.4 (9.8)</td>
<td>78.4 (8.5)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>25 (33.3%)</td>
<td>31 (33%)</td>
</tr>
<tr>
<td>Female</td>
<td>50 (66.7%)</td>
<td>63 (67%)</td>
</tr>
<tr>
<td>ARMD</td>
<td>71</td>
<td>71</td>
</tr>
<tr>
<td>CVA</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>ERM</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

ARMD, age related macular degeneration; CVA, cerebrovascular accident; ERM, epiretinal membrane.

Comment

Cystoid macular oedema resulting in visual loss occurs in up to 3.8% of cases following cataract extraction by phacoemulsification without posterior capsule rupture. Trypan blue use, but trypan blue has been shown to inadver-
dently stain posterior capsule and intraocular lens implants. None of the patients in our group who developed CMO had associated posterior capsule rupture or ocular co-morbidity and all of the cases were treated successfully with good visual outcomes. Dada et al. and Sharma et al. suggested the use of trypan blue in routine cases to aid in training of junior surgeons. Our study would suggest some caution with this approach in view of the apparent increase in the incidence of CMO with trypan blue use.
The preoperative best corrected acuity was decreased in the group in which trypan blue was used. This suggests that the cataracts in this group were of greater density, possibly requiring more energy to remove using phacoemulsification. The energy used during surgery however was not recorded. The CMO OCT scanning of the maculas in both groups would give non-invasive objective evidence of CMO.

We suggest the following steps to limit the apparent complication of CMO with trypan blue use:

- Use the smallest amount and lowest concentration of trypan blue possible (trypan blue in concentrations as low as 0.0125% has been shown to effectively stain the anterior capsule).
- Increase postoperative steroid or anti-inflammatory drops prophylactically.
- Use only in appropriate cases—that is, with poor visualisation of the anterior capsule.

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References


Case report

Two sisters, 11 and 3 years old, were referred for evaluation because of intraocular cysts and amblyopic fellow eyes. They were the products of a full term normal pregnancy with an uneventful perinatal history. Their parents were not relatives. Past medical history was unremarkable. The children in the family were reportedly healthy with no ocular pathology but were ineligible for examination.

The older sister was known to have worn glasses since the age of 7 years. She complained of intermittent obscurations of vision in her right eye. Her vision was 6/12, J1 right eye and counting fingers at 1 metre with J14 left eye. By indirect ophtalmoscopy of her right eye, a round, pigmented cystic structure was observed in the vitreous cavity (fig 1). The left fundus showed myopic chorioretinal changes with a tilted optic disc. Retinoscopy showed marked myopic anisometropia of +1.00 –1.25 × 75° right eye and –10.50 –2.00 × 95° left eye. Ultrasoundography disclosed a 3.65 mm cystic, round, hypoechoic vitreal structure. (fig 1). It was partially mobile with vitreal after-movements and was tethered to fine vitreal strands. Its walls showed internal reflectivity of 60%, whereas its content had very low (<5%) reflectivity. The younger sister was fitted with spectacles at the age of 7 months because of anisometropic myopia. Her visual acuity (picture cube) was 0.03 right eye with unsteady fixation and 0.2 left eye. In her left eye a cystic, pigmented lesion was attached to the posterior iris surface and extended into the anterior vitreous (fig 2). It was located in the superotemporal quadrant causing adja
cent lenticial cortical opacities. Indirect ophtalmoscopy revealed bilateral mild retinal myopic changes. High frequency echo
graphy of the iris lesion disclosed a cyst with hypoechoic content measuring 3.68 mm in diameter (fig 2). Cycloplegic refraction showed anisometropic myopia of –7.5 –1.00 × 90° right eye and –0.5 –3.00 × 80° left eye.

Familial intraocular cysts in association with anisometropia

Vitreous cysts are rare. Their origin is postulated to be a congenital remnant of the primary hyaloid system or ciliary body pigment epithelium. Although most vitreal cysts are asymptomatic, some may cause intermittent visual field defects. Treatment is seldom indicated, though laser photocoagulation or vitrectomy have been suggested. Iris cysts include iris pigment epithelial cysts and stromal cysts. The former may get dislodged into either the anterior chamber or into the vitreous chamber. They become symptomatic when they enlarge and occlude the visual axis. Treatment includes aspiration or surgical excision of the cyst.

Both vitreous and iris cysts have been previously reported as sporadic findings. In this report, we present the clinical and echoraphic manifestations of intraocular cysts in two siblings.

Figure 1 (A) Homogeneously pigmented vitreous cyst. (B) B-scan echography that demonstrates a round, echolucent vitreous cyst bound by fine vitreal strands.
with anisometric amblyopia look normal to the family, leading to delay in detection and treatment. The physical characteristics of the vitreous cyst we described, including its confinement to the region of Cloquet’s canal, are similar to those reported by others. This suggests that the cysts may be remnants of the persistent fetal vasculature, though this manifestation was not included in Goldberg’s description of this disease. However, since no surgical excision was performed, we may postulate regarding their cellular origin. Nork and Millechica suggested after histological studies, that the cyst origin was pigment epithelial-type cells. In our study, indirect evidence that the cysts originated from pigment epithelium include their homogeneous brown pigmentation, medium reflectivity, and continuation of iris cyst with the posterior iris surface. The cellular origin of the vitreous cyst is less obvious. It can either be a primary congenital hyaloidal remnant or a cyst that detached from the iris during childhood. Only few have reported on vitreous cysts jarring loose from the ciliary body pigment epithelium.

The physical characteristics of the vitreous cyst we described, including its confinement to the region of Cloquet’s canal, are similar to those reported by others. This suggests that the cysts may be remnants of the persistent fetal vasculature, though this manifestation was not included in Goldberg’s description of this disease. However, since no surgical excision was performed, we may postulate regarding their cellular origin. Nork and Millechica suggested after histological studies, that the cyst origin was pigment epithelial-type cells. In our study, indirect evidence that the cysts originated from pigment epithelium include their homogeneous brown pigmentation, medium reflectivity, and continuation of iris cyst with the posterior iris surface. The cellular origin of the vitreous cyst is less obvious. It can either be a primary congenital hyaloidal remnant or a cyst that detached from the iris during childhood. Only few have reported on vitreous cysts jarring loose from the ciliary body pigment epithelium.

Central retinal artery occlusion and ophthalmoplegia following spinal surgery

Visual loss and ophthalmoplegia are very infrequent complications after spinal surgery. Visual loss may be caused by ischaemic optic neuropathy, central retinal artery or vein occlusion, or ocular stroke. Previous reports have attributed this complication to patient positioning, intraoperative blood loss, and controlled hypotension or shock. Associated risk factors include anaemia, prolonged surgical time, bradyarrhythmia, hypertension, diabetes, smoking, vascular disease, and increased blood viscosity. Ophthalmoplegia after spinal surgery is even more unusual than visual loss, and only few reports exist in the literature.

Case 1

A 62 year old male ex-smoker underwent a L2–L3 posterior spinal decompression and segmental instrumentation for lumbar spondylosis and scoliosis, in prone position with ocular protection (gauze swab and tape). The surgery lasted 2 hours and 45 minutes. Before the procedure blood pressure was 140/60 mm Hg and during operation it was maintained at 90/60 mm Hg. Just after surgery he complained of visual loss and left ocular and nasal pain. Examination revealed left palpebral oedema, local erythema, blindness, and total ophthalmoplegia of the left eye. Left pupil was dilated and fixed. The funduscopic examination showed retinal oedema, a central cherry-red spot at the macula, and attenuated arteries. The rest of his neurological examination was normal. The haematomatice dropped from 43% to 34%. The brain MRI was normal and the orbit MRI revealed enlargement and hyperintensity of left ocular muscles in T2 weighted images sparing their tendons (fig 1). Ocular motility recovered in 4 weeks but visual loss persisted until the last follow up at 7 months.

Case 2

A 23 year old man with a history of tobacco abuse and asthma underwent a prolonged cervical arthrodesis in prone position caused by C7 vertebral collapse with spinal contusion. Immediately after surgery he complained of left visual loss and he was referred to our hospital. Details of duration of surgery, ocular protection, intraoperative blood pressure, and haematomatice were unavailable. Upon examination he showed blindness of the left eye with palpebral oedema, orbital pain, and total external ophthalmoplegia. The funduscopic examination revealed a pale retina with a macular cherry-red spot. The pupil was dilated and fixed. The MRI studies showed a normal brain, but swelling of the left extraocular muscles; MRI angiography and ophthalmalic echo Doppler were normal. After 3 months

The physical characteristics of the vitreous cyst we described, including its confinement to the region of Cloquet’s canal, are similar to those reported by others. This suggests that the cysts may be remnants of the persistent fetal vasculature, though this manifestation was not included in Goldberg’s description of this disease. However, since no surgical excision was performed, we may postulate regarding their cellular origin. Nork and Millechica suggested after histological studies, that the cyst origin was pigment epithelial-type cells. In our study, indirect evidence that the cysts originated from pigment epithelium include their homogeneous brown pigmentation, medium reflectivity, and continuation of iris cyst with the posterior iris surface. The cellular origin of the vitreous cyst is less obvious. It can either be a primary congenital hyaloidal remnant or a cyst that detached from the iris during childhood. Only few have reported on vitreous cysts jarring loose from the ciliary body pigment epithelium.

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Reference

Figure 1
MRI of the orbit. T2 weighed image shows proptosis and oedema of extraocular muscles in the left eye, sparing their tendons.

Figure 2
(A) Cystic structure extending from the posterior layer of the iris in the superotemporal quadrant. (B) High frequency ultrasound echography that shows a cystic structure attached to the posterior iris.

Comment
This reports the unusual association between vitreous and iris cysts found in two siblings. The familial clustering of pigmented intraocular cysts suggests a hereditary aetiology in these sisters. Sallo et al reported an association between vitreous cyst in a patient and corneal dermoid in her son. A familial association between vitreous cyst and iris cyst was not previously reported. Our study, in accordance with previous reports, emphasises the need for examination of family members once an intraocular cyst has been diagnosed in a young child. The second unusual association described in these siblings is the deeply amblyopic fellow eye with high anisometropic myopic astigmatism. It is not clear whether axial myopia developed because of the amblyopia or whether the amblyopia is secondary to anisometropia. Amblyopia in anisometropia results from the suppression of cortical vision centres that receive inputs from the chronically defocused eye. Also, the eyes of a child

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Comment

Our two patients developed complete ophthalmoplegia and CRAO after spinal surgery. Intraoperative ocular protection was used at least in the first patient. Common features included prone position and postoperative signs of orbital swelling. Imaging studies revealed signs of oedema in extraocular muscles sparing their tendons. To our knowledge, extraocular muscles abnormalities in MRI have not been previously reported. Ophthalmoplegia partially improved in one patient and fully recovered in the other within a few weeks, but visual loss persisted in both cases.

Stevens et al., in a retrospective review of 3450 spinal surgeries, identified seven patients (incidence 0.2%) with visual loss. No details of surgical positioning or postoperative signs of orbital swelling were given. Since the original report of Slocum et al., it has been emphasised that CRAO may be the result of extrinsic ocular pressure caused by head rest or anaesthetic mask malposition in the presence of hypotension, shock, and prolonged anaesthesia.²³⁻²⁵

Ophthalmoplegia related to spinal surgery is also an exceptional complication. West et al. described a patient who developed unilateral total extraocular muscle oedema and unilateral visual loss following scoliosis surgery. An orbit computed tomography scan performed 1 week later showed left proptosis and swelling of the left medial rectus and inferior oblique muscles. The authors suggested that perioperative ocular compression might be the cause. Wolfe et al. reported a young woman who suffered a right CRAO after an instrumented spinal surgery. Postoperatively, the patient had blindness, moderate ptosis, restriction of the extraocular movements, and paraesthesias in the supraorbital region of the same eye. A CT scan of the brain and orbits showed only mild oedema of the right optic nerve. Ocular motility improved but the visual loss did not. Although prognosis is usually very poor, a recent patient described in Japan was treated successfully with urokinase and PG-E1, stellate ganglion block, and hyperbaric oxygen therapy.¹

Hollenhorst et al. reported eight cases of unilateral visual loss after inadvertent orbital pressure during general anaesthesia for neurosurgical procedures. The most severe cases had proptosis, ptosis, and paralysis of extraocular muscle musculature with no recovery. Moreover, they provoked visual loss and ophthalmoplegia by orbital compression for 60 minutes in seven rhesus monkeys, in the setting of hypovolaemia and hypotension. They proposed that partial or complete collapse of the arterial and venous channels of the orbit, occurred as a result of a tamponade action of the ocular contents. When the external pressure is released, the ischaemic vascular channels dilate and there is a transudation of fluid through the permeable walls into the tissue spaces. This results in orbital oedema, proptosis, paresis of ocular movement, and massive oedema of the retina. Based on findings from this animal model, it is likely that external pressure during the surgical procedure induces oclusion of the arterial and venous orbital vessels. The increased MRI signal in extraocular muscles is probably the expression of post-ischaemic oedema facilitated by the richer vascular supply and the more prominent extravascular space characteristic of these muscles.²⁶ Reversibility of ophthalmoplegia will probably depend on the degree of ischaemia suffered by both the extraocular muscles and the III, IV, and VI cranial nerves.

CRAO and ophthalmoplegia are unusual, but severe, complications after spinal surgery. Postoperative signs of orbital swelling, only in the affected eyes, were clear evidence of intraoperative compression in our patients.

The increase in intraorbital pressure associated with hypotension, shock, anaemia, prolonged operative time, and bradycardia/hyptothermia are considered to be the main risk factors for developing CRAO and ophthalmoplegia. Adequate eye protection during surgery, and meticulous attention to keep the eyes free from pressure, can reduce the risk of these potentially avoidable serious complications.

Acknowledgement

We thank Dr John Stewart for his critical reading of the manuscript.

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References

8 Hollenhorst RW. Swien HJ, Benoit CF. Unilateral blindness occurring during anesthesia for

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**Table 1** Reported patients with postoperative CRAO, ophthalmoplegia, or both

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex/age</th>
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<th>Position</th>
<th>Instrumentation</th>
<th>Operating time (minutes)</th>
<th>Blood pressure (mm Hg)</th>
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<th>Ophthalmoplegia</th>
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<td>Slocum 1948</td>
<td>Female</td>
<td>Neurosurgical</td>
<td>Prone</td>
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<td>80/60</td>
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<tr>
<td>Givner 1950</td>
<td>Male</td>
<td>Abdominal</td>
<td>Supine</td>
<td>No</td>
<td>265</td>
<td>Shock</td>
<td>Right</td>
<td>No</td>
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<tr>
<td>Gillan 1953</td>
<td>Male</td>
<td>Abdominal</td>
<td>Supine</td>
<td>No</td>
<td>85</td>
<td>Mild shock</td>
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<tr>
<td>Hollenhorst 1954</td>
<td>Male</td>
<td>Spine</td>
<td>Prone</td>
<td>Yes</td>
<td>120</td>
<td>Normal</td>
<td>Left</td>
<td>No</td>
</tr>
<tr>
<td>West 1990</td>
<td>Female</td>
<td>Spine</td>
<td>Prone</td>
<td>Yes</td>
<td>270</td>
<td>Systolic 70–90</td>
<td>Left</td>
<td>Yes</td>
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<tr>
<td>Wolfe 1992</td>
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<td>Spine</td>
<td>Prone</td>
<td>Yes</td>
<td>135</td>
<td>90/60</td>
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<tr>
<td>Givner 1993</td>
<td>Male</td>
<td>Spine</td>
<td>Prone</td>
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<tr>
<td>Hollon 2004</td>
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<td>Spine</td>
<td>Prone</td>
<td>Yes</td>
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</tbody>
</table>

CRAO, central retinal artery occlusion.
I read with great interest the article by neonatorum
Isenberg et al. I note the following reason.


Prophylaxis of ophthalmia neonatorum

The authors deserve to be commended for their pioneering interest in childhood blindness. There are certain points that I would like to clarify and supplement with regard to their study.

1. The authors have mentioned that only babies born by a vaginal delivery were studied, since the eyes of babies delivered by caesarean section were previously proved to be nearly always sterile. This would result in a gross understatement of the incidence of ophthalmia neonatorum in this study, for the following reason.

By convention, ophthalmia neonatorum is defined as conjunctivitis arising within 1 month after birth. Hence, some of these conjunctival infections could originate from sources other than the maternal vaginal and cervical flora. In fact, some cases of ophthalmia neonatorum, especially those caused by Staphylococcus aureus, could have originated at home, as previously reported by the authors themselves. In the same study, no significant difference in the frequency or type of infection was noted among the infants delivered vaginally or by caesarean section.

2. Other authors have made similar observations. Krohn et al. have found some cases of ophthalmia neonatorum to have been caused by the infants' nasogastroental passage or from their caregivers after delivery. Verma et al. in a prospective study from India, found no correlation between the microbiology of the conjunctival swabs of the infected eyes (Staph aureus was the commonest isolate) and the vaginal and cervical swabs of the mothers (Escherichia coli was the commonest isolate). They concluded that most of the cases of ophthalmia neonatorum were acquired postnatally. In the light of these previously reported studies, I feel that exclusion of cases that were delivered by caesarean section was not warranted and weakens the power of this study. The efficacy of the second drop of povidone-iodine was not tested on a significant proportion of the cases that were delivered by caesarean section.

3. While he is correct regarding the definition of ophthalmia neonatorum including all infections acquired by an infant during the first 30 days of life, for the purposes of our study, we were primarily interested in those cases resulting from neonatal exposure in the birth canal. This source of ophthalmia neonatorum is the one that would be influenced by this study. Indeed, Dr Vendantham's definition of "ophthalmia neonatorum," would not be impacted by this second drop and therefore would not be directly affected by this study. Indeed, Dr Vendantham's interest in neonatal dacryocystitis would also fall within the same question since the reflux from the tear duct causing this infection generally does not arise until well after the first day of life.

4. The proportion of ophthalmia neonatorum cases acquired postnatally compared with those acquired during the birth process probably differs by country. In India, for example, the incidence of ophthalmia neonatorum is the one that would be influenced by this study. Indeed, Dr Vendantham's definition of "ophthalmia neonatorum," would not be impacted by this second drop and therefore would not be directly affected by this study. Indeed, Dr Vendantham's interest in neonatal dacryocystitis would also fall within the same question since the reflux from the tear duct causing this infection generally does not arise until well after the first day of life.

References


Authors' reply

We greatly appreciate the inquiry of Dr Vendantham and are happy to reply to his questions. While he is correct regarding the definition of ophthalmia neonatorum including all infections acquired by an infant during the first 30 days of life, for the purposes of our study, we were primarily interested in those cases resulting from neonatal exposure in the birth canal. This source of ophthalmia neonatorum is the one that would be influenced by this study. Indeed, Dr Vendantham's definition of "ophthalmia neonatorum," would not be impacted by this second drop and therefore would not be directly affected by this study. Indeed, Dr Vendantham's interest in neonatal dacryocystitis would also fall within the same question since the reflux from the tear duct causing this infection generally does not arise until well after the first day of life.

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References


by “disease oriented evidence” (DOE) like intraocular pressure and retinal attachment, which are surrogate outcomes.1 These DOE may not correlate well with the patients’ symptoms and it would be valuable if the authors can include the level of pain and discomfort as other outcome measures for the study.

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References


Blue light and the circadian clock

Dr S Mainster and Sparrow have provided an excellent perspective on the relative merits and difficulties of extending intraocular lens (IOL) absorption into the blue portion of the spectrum.1 However, they have not considered an unintentional consequence of blocking some of the blue portion of the spectrum—reducing the activity of intrinsically photosensitive retinal ganglion cells.2–4 These cells subserve several non-visual ocular photoreceptive tasks, most prominently the entrainment of the circadian clock to external light-dark cycles.4 Pupillary light responses in mice are also at least partially controlled by this system, which appears to use a novel opsin (melanopsin)5 and possibly also a flavoprotein (cryptochrome)6 as photopigments. Experiments in mice have suggested that the action spectrum for these photopigments peak in the blue, at approximately 480 nm, but with substantial sensitivity to blue light to 430 nm.7 This system appears to be functional in humans as documented by the action spectrum for light suppression of the pineal hormone, melatonin.8 9

The clinical importance of these photoreceptors is presently unknown, although it appears that loss of retinal ganglion cells predisposes children and young adults to disorders of sleep timing that outer retinal disease does not.10 While, as the authors note, there may be substantial benefit in blocking blue light phototoxicity, particularly for patients with pre-existing outer retinal degeneration, these lenses may have unintended consequences with respect to the timing of sleep and wakefulness or levels of certain neurohormones.

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References


Author’s reply

I appreciate Van Gelder’s thoughtful comments regarding the potential consequences of a ultraviolet + blue light absorbing intraocular lens (IOL) on circadian rhythmicity. I agree that the clinical importance of retinal ganglion photoreceptors is currently unknown and that decreasing the amount of blue light reaching them might affect their function. Conversely, if photosensitive ganglia respond to circadian changes in their blue light exposure rather than just the magnitude of that exposure, a ultraviolet + blue light absorbing IOL may not impair ganglion function.

Van Gelder re-emphasises our finding that IOL chromophore selection balances the potential loss of useful visual function against a reduction in the risk of acute ultraviolet-blue phototoxicity. Our paper did not state, however, that ultraviolet + blue absorbing IOLs were desirable for people with outer retinal degeneration. Indeed, blue light is more important in scotopic than photopic vision. Individuals with age related macular degeneration have greater night-time visual problems than their peers without it, and these scotopic problems may be exacerbated if a significant amount of blue light is blocked by an IOL.
one third of the total conjunctival surface respectively. Using conjunctival fluorescein staining as an indicator of the extent of conjunctival damage, the area of involvement can be based on the fraction of the third involved, limiting any division into not less than sixths—that is, the tarsal surfaces together comprise a third of conjunctiva (see fig 1). This includes the issue that a vertical distribution of conjunctival injury is as important as a horizontal distribution.

Corneal involvement in terms of prognosis remains an area of difficulty. Although it may be assumed that limbal and conjunctival damage implies a worse prognosis than isolated corneal damage, this has yet to be shown. In addition, a severe chemical injury involving the cornea but not the limbus, or vice versa, would be expected to be an uncommon event. We therefore propose to retain the degree of corneal damage (as proposed by Roper-Hall and Ballen1) in grading of the injury (see table 1).

Thus, grade I is identified by any isolated corneal epithelial injury. Grade II includes limbal or conjunctival involvement, but involves less than one third of the area involved. Grade III includes either a hazy cornea, defined as obscurity of the iris or pupil details (as per Roper-Hall’s and Ballen’s original descriptions), and/or greater than one third of limbal or conjunctival damage. With the advent of recent surgical techniques such as amniotic membrane transplants and limbal allografts, the prognosis of more severe ocular chemical injuries previously classified as Roper-Hall grade IV have improved and no longer carry a uniformly poor prognosis.2 Therefore, we reason that these cases can be included in grade III of our proposed classification.

In conclusion, in the absence of good evidence for re-classifying ocular surface injuries, it would seem reasonable to keep the Roper-Hall/Ballen classification and to move it forward by addressing the weaknesses of that system. We hope that the proposed grading system improves the consistency with which chemical injuries are reported in the literature, serves as a basis for controlled comparative evaluation of modern treatment, and stimulates further work in this area.

Figure 1 Estimation of conjunctival injury. For example, 1/6th + 1/6th = 1/3rd.

Table 1 Modified classification of ocular chemical injuries

<table>
<thead>
<tr>
<th>Grade</th>
<th>Cornea</th>
<th>Limbal ischaemia</th>
<th>Conjunctival involvement</th>
<th>Prognosis</th>
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<td>I</td>
<td>Clear epithelial damage only</td>
<td>None</td>
<td>None</td>
<td>Good</td>
</tr>
<tr>
<td>II</td>
<td>Clear epithelial damage only</td>
<td>&lt;1/3</td>
<td>&lt;1/3</td>
<td>Good</td>
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<tr>
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<td>Hazy cornea</td>
<td>&gt;1/3</td>
<td>OR &gt;1/3</td>
<td>Guarded</td>
</tr>
</tbody>
</table>

References

Author’s response
The response by Harun et al is to be commended in so far as it highlights the problems with the current Roper-Hall classification system and the difficulties it poses in evaluating outcome and efficacy of treatments in ocular surface burns. As a proposed modification, however, it is a retrograde step.

The three major issues with the Roper-Hall classification were that it lumped all injuries with 50% or more of limbal involvement into one category, did not take into account conjunctival involvement in the actual classification, and placed undue emphasis on the degree of corneal haze.

The proposed modification by Harun et al goes a step backwards by grouping all injuries with more than 33% limbal involvement into one category. The grading of a patient with all 12 clock hours of limbus involvement would then be the same as one with just over 11 clock hours of limbus involvement. Conversely, a patient presenting with less than one third limbal involvement does not necessarily come with less than one third conjunctival involvement, which could be much more. The proposed modification does not allow for such variances, which are frequent. The Dua, King, and Joseph classification considered limbal involvement (to encompass ischaemia as well) rather than limbal ischaemia alone.

The point about conjunctival involvement is well made in the proposed modification. This does not alter significantly from the Dua, King, and Joseph classification. The latter was the first to take this aspect of burns into account in determining severity and prognosis. The authors mention the importance of tarsal conjunctival involvement. This is a valid though often an impractical consideration. Associated swelling, induration, thickening, shrinkage and the like, of the lids make tarsal conjunctival examination impractical if not impossible in some cases, in the immediate post-injury period. It was for this practical consideration that the Dua, King, and Joseph classification included only the extent of bulbar conjunctival involvement in determining the grade. It is interesting to note that the authors disregard limbal fluorescein staining as an indicator of limbal damage (as proposed in the Dua, King, and Joseph classification) but propose fluorescein staining as an indicator despite the difficulty in evaluating extent of conjunctival damage. This implies that fluorescein staining is appropriate to evaluate both conjunctival epithelial damage and conjunctival ischaemia but not limbal epithelial damage and limbal ischaemia. There is no rationale for this.

Corneal haze can be an indicator of the offending chemical rather than the severity of the insult. It is not uncommon to find a clear and transparent cornea, which is totally denuded of its epithelium, immediately after a chemical injury. This can stay so for a few days before becoming rapidly hazy or opaque, or remain clear and become re-epithelialised. Corneal endothelial damage leading to corneal oedema and haze can occur later in the course of an acute chemical injury. Conversely, a hazy cornea with a resultant scar could do well following a corneal graft procedure if the limbal involvement is minimal. The proposed modification retains corneal haze as a grading parameter and includes a hazy cornea in grade 3 only.

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problem in the Roper-Hall classification and its proposed modification.

Most important of all, the proposed classification is purely theoretical and has not been validated. The Dua, King, and Joseph classification is based on several years of clinical experience of managing burns including more than 67 patients. It is simple and easy to use (clock hours of limbus involvement and percentage of conjunctival involvement), flexible, and allows for all combinations of different extents of involvement of the two structures. It is validated as a prognostic indicator and allows for accurate comparison of cases. The proposed new classification/ modification fails on all these counts.

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Further communications regarding this subject can be found at eLetters on the BJO website (www.bjophthalmol.com).

BOOK REVIEW

Complications in Ophthalmic Plastic Surgery

There is no doubt that complications in surgery are an inevitable fact. Nevertheless, the unforeseen surgical outcomes always play a fundamental part in the self improving process. Certainly, the experience provides the safest way, for both the patient and the surgeon, to prevent harm and smooth the final result.

This book represents one of the most serious, and not very common, works focusing on the complications in ophthalmic plastic surgery. It is a considerably well organised book, which apparently requires some basic knowledge of ocuoplastics and facial aesthetic surgery. The format is based on three distinguished parts: cosmetic surgery, ptosis, and lower eyelid malposition. A very competent number of contributors cover the topics of their specific interest. In the first part the authors are dealing with the blepharoplasties, the laser resurfacing, and the forehead lift. The ptosis chapter is referred to the most common ptosis techniques but brow suspension is remarkably absent. The third part, although it is entitled “Lower eyelid malposition,” includes and some unrelated, though welcomed topics, like DCR, enucleation, and orbital fractures.

The necessity of the communication between the surgeon and the patient is vigorously emphasised and didactically analysed in every single chapter. Deep understanding of the patient’s expectations as well as detailed information about the pragmatic results is recommended throughout the chapters of the book. There is quite a sufficient reference to preoperative evaluation of the patient regarding measurements, anesthesia, and surgical preparation.

Although the covered operations are extensively described, a countable number of other surgical techniques, and their possible complications, are not mentioned. The latter is probably related to the editor’s orientation to aesthetic oculoplastic surgery.

The anatomical and pathophysiologic mechanisms of the most common complications are thoroughly explained. At the same time, the authors give many enlightening tips, based on their broad experience, for preventing the problems, and meticulously describe the management of the intraoperative and postoperative complications. The number of the illustrations do not adequately correspond to the addressed complications and the quality of the pictures varies, depending on the author’s collection. Additionally, the shortage of references in some of the most interesting chapters (ptosis, enucleation) should certainly not be overlooked, for the magnitude of such a book.

Every attempt to give precious advice about the frustrating and unavoidable surgical complications is always warmly welcomed. Brian Brazzo’s book is predominately a useful guide to the understanding, prevention, and management of the commonest problems in oculoplastic surgery. Despite the expected problems of every first edition this generally represents a meticulous work on specific issues and thus is recommended for the ophthalmic surgeon and especially for surgeons who are chiefly interested in oculoplastics and cosmetic surgery.

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CORRECTION

In the letter titled Sequential treatment of central retinal vein occlusion with intravitreal tissue plasminogen activator and intravitreal triamcinolone (Br J Ophthalmol 2004;88:1100-1101) the authors were listed incorrectly. The correct listing is as follows: J M Lahey, J J Kearney, M C Cheung. The journal apologises for this error.

It has come to our attention that, owing to a production error, the letter by N Islam, K Mireskandari, and G E Rose has been published twice, in the June issue (Br J Ophthalmol 2004;88:833-834) and the August issue (Br J Ophthalmol 2004;88:1092-1093). The initial publication in the June issue should be taken as the article of record. We apologise to the authors and readers for any confusion this accidental duplicate publication may have caused.

NOTICES

Childhood cataract
The latest issue of Community Eye Health (No 50) deals with the manitude, management, economics and impact of childhood cataract. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shah@lshtm.ac.uk; online edition: www.jcheb.co.uk). Annual subscription (4 issues) UK £28/US$45. Free to developing country applicants.

Ophthalmic Anesthesia Society
The 18th annual meeting of the Ophthalmic Anesthesia Society will be held on 1–3 October 2004 in Chicago, USA. For further details: Ophthalmic Anesthesia Society (OAS), 793-A Foothill Blvd, PMB #119, San Luis Obispo, CA 93405, USA (tel: 001 805 534 0300; fax: 001 805 534 9030; email: info@eyeanaesthesia.org; website: www.eyeanaesthesia.org).

4th International Congress on Autoimmunity
The 4th International Congress on Autoimmunity will take place 3–7 November 2004 in Budapest, Hungary. Further details: Ophthalmic Anesthesia Society (OAS), 793-A Foothill Blvd, PMB #119, 17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland (tel: +41 22 908 0488; fax: +41 22 732 2850; email: autoimm04@kenes.com; website: www.kenes.com/autoimm04).