Sub-Tenon's infiltration of local anaesthetic with hyaluronidase

EDITOR,—Stevens1 has recently described local anaesthetic delivery into the sub-Tenon's space as an alternative to the conventional retrobulbar and peribulbar approaches for ophthalmic anaesthesia. Using a 50:50 mixture of lignocaine 2% and bupivacaine 0.5% without hyaluronidase he obtained effective anaesthesia in all of his 50 patients. However, 46% required an additional Van Lint facial block. 2

I have now used the same method, employing a 21 gauge lacrimal cannula to deliver the anaesthetic in 24 patients undergoing cataract surgery. Lignocaine 2% (5 ml) and bupivacaine (Marcair) 0.5% (5 ml) were mixed with 1500 IU of hyaluronidase (Wyeth). Three of the latter were delivered in the manner Stevens described. All patients had satisfactory analgesia and akinesia, while one patient only required an additional Van Lint facial block.

The results are shown in the Table.

<table>
<thead>
<tr>
<th>Compl.</th>
<th>Incompl.</th>
<th>No effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anesthesia</td>
<td>24</td>
<td>0</td>
</tr>
<tr>
<td>Akinesia</td>
<td>21</td>
<td>3</td>
</tr>
<tr>
<td>Paralysis of orbicularis</td>
<td>14</td>
<td>9</td>
</tr>
</tbody>
</table>

* Proceeded to facial block.

TABLE

Three patients who had previously experienced retrobulbar blocks for contralateral surgery stated that the anaesthesia for the second eye was much less painful. Only one patient complained of pain due to the infiltration of anaesthetic. There were no adverse complications.

This novel method of delivery of local anaesthesia is usually pain free and avoids the range of complications due to needle perforation. The use of hyaluronidase appears to increase the proportion of patients with sufficient facial akinesia to facilitate cataract surgery, without recourse to an additional facial nerve block.

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Reply

EDITOR,—I welcome Mr Dutton's results with the local anaesthetic technique using a lignocaine-bupivacaine mixture with the addition of hyaluronidase. Hyaluronidase has been shown to improve both the speed of onset and the degree of akinesia produced by retrobulbar injection3 but no comparison for sub-Tenon delivery has been published. When I assessed one quadrant sub-Tenon delivery in 1991, hyaluronidase was not used owing to difficulties in supply, though I postulated that its use would be beneficial.4 A recent study involving ultrasound assessment of administered sub-Tenon solution found a rapid diffusion or 'leakage' of fluid from the sub-Tenon space to the anterior retrobulbar compartment.5 This evidence of posterior diffusion from the sub-Tenon space further supports the use of hyaluronidase to improve the efficacy of the sub-Tenon technique.

It is my current practice to perform sub-Tenon delivery for cataract surgery using approximately 3-0 ml of lignocaine 2% with added hyaluronidase and point diathermy applied to the conjunctival site of application. This provides a rapid onset of action with the elimination of haemorrhage from the site of incision in the conjunctiva.

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Development of neovascular glaucoma in the course of interferon alfa therapy for hepatitis type C

EDITOR,—I report a case of neovascular glaucoma which occurred 2 months after initiation of interferon therapy, widely used today to treat hepatitis and malignant diseases.

A 56-year-old man came to my clinic on 23 June 1993 with a complaint of sudden decrease of vision. Both eyes had undergone cataract surgery in January of the same year, and no postoperative complications were identified during postoperative visits. His diabetes was poorly controlled; and he has been treated for hepatitis type C with 6x10⁸ units of interferon alfa subcutaneously three times a week since April. On 23 July, the left eye showed severe ciliary injection and hypaema occupied the bottom half of the anterior chamber. Intraocular pressure was 40 mm Hg. Diabetic retinopathy had also worsened since the initiation of interferon therapy and fluorescein fundus angiography demonstrated an extensive avascular region. The anterior segment of the right eye showed neovascularisation at the anterior chamber angle which was not noted 6 months before. Interferon therapy was discontinued and anterior chamber lavage of the left eye was performed. On the following day, ciliary injection had disappeared, hypaema did not recur, and the intraocular pressure returned to normal. Both uveitis and corneal epithelial defect diminished. Panretinal photocoagulation was performed in both eyes, resulting in remission of all ocular symptoms.

Ocular complications during interferon therapy include retinal haemorrhage and cotton wool patches, and oculomotor palsies.6 Deposition of the immune complexes in the retinal vasculature is considered to be the possible cause for retinopathy. In our patient, diabetic retinopathy worsened and neovascular glaucoma was encountered shortly after initiation of interferon therapy. Although these ocular symptoms are often seen in diabetic patients, it is interesting that they began to improve when interferon was discontinued.

Indocyanine green enhanced diode laser photocoagulation of subretinal neovascular membranes

EDITOR,—It was with interest that I read M W Ulbig and colleagues' excellent article that appeared recently in your journal, concerning diode laser photocoagulation of choroidal neovascular membranes (CNVM).1 It was, however, very surprising that no mention of indocyanine green (ICG) enhanced diode laser of subretinal neovascular membranes was made in this paper.2 This is a very important concept since ICG accumulates in subretinal neovascular membranes after clearance from the surrounding circulation.

This not only aids visualisation and identification of established and occult subretinal neovascular membranes in ICG-enhanced infrared angiography but acts as a chromophore facilitating absorption of 810 nm infrared diode laser energy.3 This potentially enhances thermal damage to CNVM as exemplified by the successful closure of all CNVM treated with ICG enhancement and diode laser (810 nm) at one session, without recurrence.4

Greater energy delivery is required with diode lasers to achieve a given endpoint in retrobulbar photocoagulation since there is greater transmission into the choroid. Subretinal haemorrhage and Bruch's membrane rupture are thus significant considerations.5 Any method that may allow for enhancement of thermal damage to the CNVM with reduced energy delivery to the surrounding choroid may thus be desirable.

Given the fact that the half the patients in Ulbig's study required further photocoagulation presumably either for incomplete treatment, failure to identify the extent of the CNVM, or precipitation of a further membrane, I am surprised that no reference to this important concept was made in their publication.

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EDITOR,—We read with interest the letter of A P Moriarty concerning ICG enhanced treatment using diode laser for choroidal neovascularisation. We are aware of the literature on this matter and the significance for the potential of this treatment mode. However, our pilot study was aimed at assessing whether the near infrared diode laser was as effective as the other laser wavelengths. Clearly, there is much future work to be done on the effect of indocyanine green and the role of laser treatment of occult neovascular membranes. As yet we are not aware of any clinical study which confirms improved efficacy of combined dye enhanced diode laser treatment compared with non-enhanced diode treatment of choroidal neovascularisation. However, the first clinical results of ICG enhanced diode laser treatment, as now presented by Carmen Puliafito at the Annual Meeting of the American Academy of Ophthalmology in Chicago, did not show significant advantages over non-enhanced treatment. Clearly Puliafito’s post-treatment slides showed marked areas of choroidal and retinal pigment epithelium atrophy. This may reflect no lesser damage of the retina as in our study with non-enhanced infrared treatment. In conclusion, a comparative study will be required.

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Ophthalmic complications after bungee jumping

EDITOR,—I would like to report a case of ophthalmic complications after bungee jumping. A 20-year-old healthy male presented to the eye casualty department with a history of transient visual loss in both eyes and gradual onset of bilateral red eyes after the jump. No neurological symptoms were noted and he did not recall any history of ocular or head injury during the whole event. On examination, with myopic correction, his visual acuity was 6/5 in both eyes. Pupillary reflexes, extraocular eye movements, and visual fields (by confrontation) were all normal. Anterior segment examination with slit-lamp revealed bilateral nasal and temporal subconjunctival haemorrhages (Fig 1). Anterior chamber, intraocular pressure, and vitreous examinations were normal. Funduscopy with cycloplegia showed bilateral multiple parafoveal dot and blot haemorrhages (Fig 2).

Fig 1
Bilateral subconjunctival haemorrhage.

Investigations included full blood count, urea and electrolytes, liver function test, serum glucose, coagulation screen, and skull x ray which was normal. Fluorescein angiogram showed no leakage of dye. He was treated conservatively and followed up in clinic with no sequelae.

Bungee jumping is becoming more popular and there is increasing evidence of injuries related to this pursuit. A case of quadriplegia resulting from unilateral locked facet and another case of non-fatal hanging injury were reported by Hite et al.1 Periorbital bruising has also been reported by Harries.2 Hanbury reported one case of bilateral subconjunctival chemosis and haemorrhages.

In this case, the manifestations of gross bilateral subconjunctival and retinal haemorrhages could be due to the sudden change in the hydrostatic pressure in the ciliary and retinal circulation respectively, in relation to the wide swing of change of the gravitational force during the dive. The potential ocular complications of this sport should not be ignored.

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Fig 2
Bilateral macular dot and blot haemorrhages.


BOOK REVIEW


There is a continuing need for good solid textbooks of ophthalmic pathology. Such a book is the recently published Ophthalmic Histopathology by Professor W R Lee.

Professor Lee is professor of ophthalmic pathology at the Tennent Institute of Ophthalmology, University of Glasgow and has amassed a wide experience of all aspects of ophthalmic pathology. His approach, however, has not simply been to document pathological changes in eye diseases but has been to attempt to understand the underlying mechanisms of the pathology and to demonstrate to his students and colleagues that without understanding mechanisms, ophthalmologists will have great difficulty in addressing many of the problems that they face. This approach undoubtedly shines through in his textbook.

The text is logically subdivided into chapters addressing common pathological groupings rather than simply taking an anatomical approach to the descriptive pathology. The first chapter is an essential one describing the technical aspects of examination of the globe and indeed should form an excellent introduction to any pathologist who wishes to undertake ophthalmic pathology. The difficulties in obtaining good morphology to correlate with fixation and staining techniques which, of necessity, alter that morphology is well described. In addition there is passing reference to aspects of immunohistochemistry and morphological techniques associated with molecular biology including in situ hybridisation. These, however, are not dealt with to any great extent further in the text.

The remaining chapters deal with specific aspects. Chapter two covers ocular injuries and has some valuable information on the sort of injury that will render an eye irreparable. The difficulties of differentiating post-traumatic lens induced uveitis and sympathetic ophthalmia on histopathology are also addressed. An important section dealing with the pathology of post-traumatic phthisis is described and should be of considerable value to ophthalmologists, since this is an area where there is some confusion concerning the precise mechanisms.

Further chapters deal with glaucoma, retinal vascular disease, intraocular tumours, ocular inflammation, failed treatment of retinal detachment, the malformed eye, the eye and systemic disease, biopsy of the adnexa, the conjunctiva and the orbit, and finally the histopathology of the corneal disc and the lens. Particularly impressive are the chapters on intraocular tumours and retinal vascular disease where the author has particular experience. The difficulties of defining malignant melanomas using the Callander classification are addressed and departures from this histological grading are well recognised. The chapter on retinal vascular diseases contains some beautiful low power magnifications following vessels throughout the course of their pathology and showing additional features relating to abnormal vascular formations.

Finally the chapters describing biopsies of various adnexal and orbital tissues are of considerable value in their descriptions of the wide range of pathologies that can occur in these tissues.

My only gripe regarding this volume is that, presumably for reasons of cost, the extremely high quality illustrations were not...