LETTERS TO THE EDITOR

Sub-Tenon's infiltration of local anaesthetic with hyaluronidase

EDITOR,—Stevens' 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.

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 (Marcain) 0.5% (5 ml) were mixed with 1500 IU of hyaluronidase (Wytel). Three were delivered in the manner Stevens describes. All patients had satisfactory analgesia and akinesia, while one patient only required an additional Van Lint facial block.

The results obtained are shown in the Table.

Table
<table>
<thead>
<tr>
<th></th>
<th>Complete effect</th>
<th>Incomplete effect</th>
<th>No effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anesthesia</td>
<td>24</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Akinesia</td>
<td>21</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Paralysis of orbicularis</td>
<td>14</td>
<td>9</td>
<td>1*</td>
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* Proceeded to facial block.

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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Developement 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 epibulbar or conjunctival irritation was identified during postoperative visits. His diabetes was poorly controlled; and he has been treated for hepatitis type C with 6·10⁹ units of interferon alfa subcutaneously three times a week since April. On 23 June, 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 ocular motor palsy.1 Deposition of the immune complexes in the vitreous 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 We found, however, very surprised that no mention of indocyanine green (ICG) enhanced diode laser of subretinal neovascular membranes was made in this paper. 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 infrared vitreous angiography but acts as a chromophore facilitating absorption of 810 nm infrared diode laser energy.2 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.3 Greater energy delivery is required with diode lasers to achieve a given endpoint in retinal photocoagulation since the ICG enhances thermal energy delivery to the surrounding choroid. Subretinal haemorrhage and Bruch's membrane rupture are thus significant considerations.4 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 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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