more widespread use, for the reasons discussed in their excellent paper.

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Sir,

We thank Claoué and Dorey *et al.* for their interest and their kind remarks.

We accept that a small subconjunctival injection of local anaesthetic means that our technique is not purely topical. We felt able to call it a topical method as we were sure that the topical local anaesthetic was the main conductor to pain relief during surgery. The subconjunctival injection was 0.1 ml only, in contradistinction to most, more appropriately named, subconjunctival techniques which use a much larger volume, and was intended to cover the scleral cautery only. This injection was given just posterior to the limbus under the operating microscope at the commencement of surgery with a 26 gauge needle. We believe, therefore, the risks of globe perforation are negligible. Since publication we have stopped the injection completely together with moving to a routine temporal approach. There has been no noticeable increase in patient discomfort, although this has not as yet been audited.

We do accept that some patients may like to take advantage of sedation if offered, but as Dorey *et al.* state, this does require the presence of an anaesthetist and many hospitals, including ours, do not have this luxury. Our main point is that there are safe and advantageous local anaesthetic techniques that make the presence of an anaesthetist unnecessary. In addition, sedation is time-consuming to administer and has effects that may persist after discharge from a day case unit; also there is little more disturbing for patient and surgeon than if the patient falls asleep and wakes disoriented and confused during surgery. We still feel sedation has little place in routine day case surgery, but *vive la différence*!

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Sir,

We read with interest the article by Anand *et al.*[1] regarding *Escherichia coli* endogenous endophthalmitis. We wish to present the histopathological findings of a patient with *E. coli* endogenous panophthalmitis with orbital cellulitis. We would also like to add a note of caution in the interpretation of gas bubbles seen radiographically in the anterior of the orbit.

Case Report

A 79-year-old hypertensive woman with non-insulin-dependent diabetes mellitus presented with a painful red left eye, proptosis and ptosis accompanied by malaise and acute loss of diabetic control. Initial minor irritation and redness had rapidly progressed to profound visual loss on the second day, but she did not seek advice until day 5. She denied any trauma, and had undergone no previous surgery. The right eye was amblyopic. Past medical history was unremarkable except for 'influenza' 3 weeks earlier.

On examination, visual acuity was 6/24 in the right eye, no perception of light in the left. There was extensive periorbital erythema and oedema. The left eye was proptosed and displaced inferolaterally with florid haemorrhagic chemosis and a sticky discharge. There was complete ophthalmoplegia with ptosis and an afferent pupillary defect. The cornea was slightly oedematous. There was a 2 mm hypopyon and pupillary inflammatory membrane allowing no view of the posterior segment. Intraocular pressure was raised at 36 mmHg. She was mildly pyrexial at 37.1 °C, but systemic examination revealed no evidence of infection or neoplasia.

Initial investigations revealed a haemoglobin level of 11.5 g/dl, neutrophil leucocytosis of 12.75×10^9/l, and erythrocyte sedimentation rate of 90 mm in the first hour. Severe hyperglycaemia required an insulin regime. Ultrasound showed vitreous reflectivity and thickened sclera consistent with panophthalmitis.

Spiral CT scan with contrast enhancement (Fig. 1) confirmed the presence of orbital cellulitis and sinus disease with mucosal thickening in the maxillary antra. Although retrobulbar inflammation and thickening of the coats of the eye itself was also demonstrated, no mass, subperiosteal inflammation or cavernous sinus thrombosis were seen, nor was there evidence of intracranial pathology. A presumptive diagnosis of panophthalmitis with orbital cellulitis was made and treatment was started with intravenous vancomycin, cefuroxime and pulsed methylprednisolone. Some improvement occurred initially, with reduction in the proptosis and partial resolution of the cellulitis. Microbiological investigation of blood, conjunctiva and urine, however, yielded no organisms, although mid-stream urine showed more than 50 white cells/mm^3 and 30 red

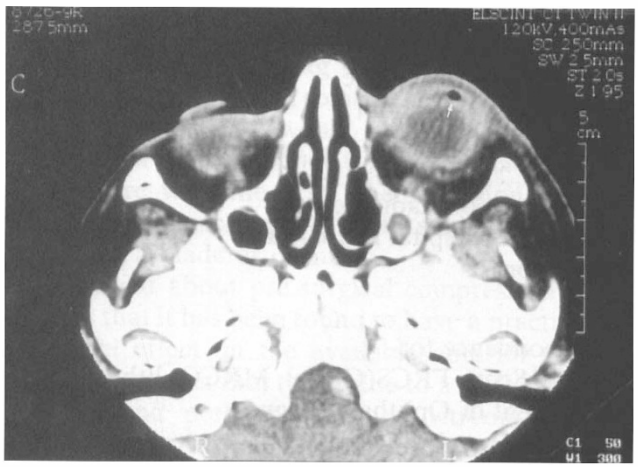
cells/mm^3. Immunological tests, thyroid function tests and chest radiography were normal.

At 10 days after presentation the left eye was enucleated because the patient's general condition had deteriorated with no visual improvement. A vitreous sample at the time of enucleation yielded *E. coli* sensitive to gentamicin, ciprofloxacin and cefotaxime. Subsequent progress has been excellent, following a course of ciprofloxacin, with complete resolution of the orbital cellulitis and restoration of diabetic control. Histopathological examination confirmed panophthalmitis with posterior segment involvement with replacement of the retina, choroid and vitreous with purulent material. There was gross scleral inflammation and thickening. The posterior lens capsule and peripheral cortex of the lens had been destroyed, but there was no histological evidence of inflammatory response to the lens material. The inflammatory changes in the anterior segment were less marked than those in the posterior segment. The pattern of inflammation in the eye was consistent with extension of the infection from posterior to anterior within the globe, and from internally to externally.

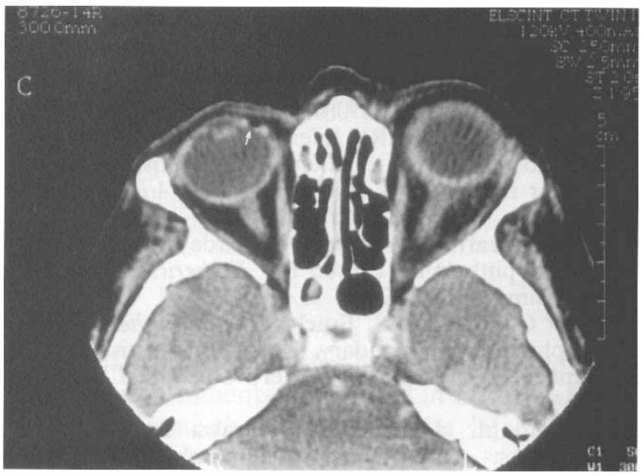
Comment

We concur with Anand *et al.* that *E. coli* is being recognised increasingly as a cause of endogenous endophthalmitis, especially in diabetic patients with urinary tract infections.[1–8] Our experience is in keeping with the ocular findings and poor prognosis in previous cases, and in view of the severe visual loss at presentation and the poor general condition, it became necessary to remove the eye, with benefit of removal of the focus of infection from the orbit, and identification of the causative organism from a vitreous biopsy. The source of the infection was never identified, although urinary tract infection is suspected from urine microscopy. Although sinus disease was present (Fig. 1) there was no radiographic evidence of subperiosteal reaction, nor was bony destruction seen with bone windows. An otorhinolaryngological opinion had been sought, but it was felt intervention was inappropriate.

The presence of gas within the globe leads to the suspicion of an anaerobic organism. We read with interest that Anand *et al.*[1] found *intraocular* gas and attributed this to the *E. coli* infection. We would be interested to know whether this gas bubble was seen clinically. We also noted gas in the area of the anterior chamber on the CT scan of our patient (Fig. 1a), and in a number of cuts more than 2.5 mm apart. That this represented a true intraocular gas bubble is unlikely, and we would be cautious in interpreting this as intraocular gas within the anterior chamber. The focus of the infection was the posterior segment of a phakic eye with extensive posterior synechiae, and even with the posterior lenticular destruction and the patient supine, we feel that the gas would not traverse the cilio-lenticular barrier. No gas bubble was seen at any time in the anterior chamber at daily slit lamp biomicroscopy, nor was crepitus detected in orbital tissues which might have suggested tissue emphysema. It may be that some atmospheric gas was present in the conjunctival fornices causing an artefact. The scan cut is 2.5 mm thick, and with gas in contact with the outer curvature of the cornea, this may produce the appearance of gas within the eye, when in truth it overlies it. A similar bubble is seen anterior to the cornea in the *normal* fellow eye of our patient (Fig. 1b).



(a)



(b)

Fig. 1. *(a) Axial spiral CT scan (2.5 mm slice) showing proptosis of the left eye with retrobulbar streaky shadows, thickened sclera and optic nerve. There is peribulbar inflammation. A gas bubble is noted (arrow) overlying the anterior chamber. The left maxillary sinus shows mucosal thickening, but there is no subperiosteal inflammation. (b) Axial spiral CT scan (2.5 mm slice) showing proptosis of the left eye with retrobulbar streaky shadows, thickened sclera and optic nerve. A gas bubble (arrow) is noted anterior to the right globe in the conjunctival fornix.*

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Sir,

The experience of Brian Leatherbarrow's Manchester team shows that, even in the best hands, early dehiscence of the conjunctival wound over a hydroxyapatite implant occurs in up to 11% of cases.[1] Unfortunately, while many re-epithelialise spontaneously, a number do not, either requiring further early intervention, or progressing to further dehiscence and ultimate chronic exposure. We have recently dealt with a case which illustrates a hitherto unrecognised cause of failure of re-epithelialisation following early conjunctival wound dehiscence.

Case Report

A 37-year-old man suffered a penetrating injury to his left eye, resulting in a blind, painful eye which required enucleation and insertion of an orbital implant 2 months following injury. Surgical technique was identical to that described by Ashworth *et al.*[1]

It needs to be emphasised that, since the scleral shell is simply fitted round the implant, there is a defect where the cornea has been removed. The wrapped implant is simply turned 'back to front' with the exposed implant facing the orbital apex and, significantly, the optic nerve 'stump', cut flush with the sclera, facing anteriorly.

At first post-operative visit at 5 days, the socket was settling well. The patient was reviewed 1 month later. At that time, a tiny central defect in the wound was apparent, plugged by loose, white tissue. This tissue lifted off easily, revealing the 'optic nerve window' with exposed implant underneath. Closer inspection revealed that the conjunctival wound had in fact dehisced more extensively. The exposed sclera had subsequently re-epithelialised almost entirely but had failed to do so over the optic nerve stump.

Over the following weeks, the defect showed no signs of spontaneous resolution and, in fact, increased slightly to 3 mm in diameter. The patient therefore required repair of the defect, with insertion of an additional scleral patch and mobilisation and suturing of the conjunctiva. This anteriorly placed defect in the scleral covering at the site of the optic nerve is an Achilles heel. It should be covered by an additional patch of sclera – a technique used routinely by the Manchester group now (personal communication) – or the implant should be inserted 'off centre', so that the area of concern is covered by one of the two horizontal recti. Early dehiscence of the conjunctiva is probably a fairly common occurrence but, as long as the implant is fully covered by sclera, re-epithelialisation will occur across the defect spontaneously.

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