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

S. E. Dorey
H. C. Seward
D. de Alwis

Croydon Eye Unit
33 Mayday Road
Thornton Heath
Surrey CR7 7XN
UK

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*!

T. D. Manners
R. L. Burton

Department of Ophthalmology
West Norwich Hospital
Bowthorpe Road
Norwich
Norfolk NR2 3TU
UK

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