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Book Review

Keratoprostheses. By S N FYODOROV, Z I MOROZ, V K ZUEV. Pp. 143. £29·95. Churchill Livingstone: Edinburgh. 1987.

In 1877 von Hippel reported on the use of keratoprostheses in seven patients. He and others of his time had almost 100% failure owing to extrusion, fistulisation, infection, aseptic corneal necrosis, and retroprosthetic membranes. Cynics would say that there has been little progress since.

In this small book Fyodorov reviews the history of keratoprostheses and describes his own modified design. He uses a two-stage procedure. Firstly, a perforated titanium supporting plate is inserted between corneal lamellae without entering the anterior chamber. Three months later a full thickness central plug of cornea is trephined out and an optical cylinder of polymethyl methacrylate is screwed into the supporting plate.

Fyodorov's indications for surgery are interesting. As well as treating chemical burns and vascularised corneas, where penetrating keratoplasty is relatively contraindicated, he also elects to perform keratoprosthesis in patients with 'endothelial-epithelial dystrophies' such as aphakic and pseudophakic bullous keratopathy. The reason for this is that penetrating keratoplasties in such patients 'are almost always diminished by opacification of the transplant.'

His results are better than may be imagined. Although the percentage of postoperative complications 'remains high,' only about 10% of prostheses in vascularised corneas extrude (over an average follow-up period of 4·4 years). Visual acuity of better than 6/12 (with limited field) occurs in 30% of his patients at some time postoperatively, though many of these subsequently lose vision secondary to complications.

Unfortunately this book suffers from poor English, muddled layout, and almost anecdotal reporting of clinical results. Fyodorov's showing no awareness of the success of modern penetrating keratoplasty techniques for bullous keratopathy is astonishing. His keratoprosthesis probably represents an improvement on other designs. Perhaps the technique should be performed more often on carefully selected cases. Read this book for an insight into Soviet ophthalmology.

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Sudden visual loss associated with clostridial bacteraemia

SIR, I was interested to learn that only two cases of endogenous clostridial panopthalmitis had been published prior to the report by Cannistra *et al.*,[1] as a similar case was seen at our hospital. The patient, a 38-year-old lorry driver, presented on 22 January 1987 with sudden onset of pain in the left eye associated with headache, nausea, and vomiting. The previous September his optician had reported that the vision in the left eye had decreased from 6/6 to 6/12, but could find no ocular abnormality, and from that time the patient had also noted diplopia with torsion. The left vision had deteriorated markedly one month prior to his presentation.

On examination the patient was afebrile with no evidence of systemic disease. The visual acuities were 6/5 right with myopic correction and no perception of light in the left. The right eye was normal. On the left there was marked conjunctival congestion and corneal epithelial and stromal oedema. The anterior chamber was almost flat, the lens hazy, and the fundus could not be visualised. Intraocular pressures were 18 mm Hg right and 70+ mm Hg left. Treatment for acute glaucoma with intravenous acetazolamide and intensive pilocarpine was commenced.

The next day the patient was mildly pyrexic. The eye remained painful and injected, and the corneal oedema was unchanged. The anterior chamber was deeper but there was a 3-4 mm hyphaema. Peripheral anterior synechiae were noted inferiorly, and the intraocular pressure was 40 mm Hg. Signs of orbital cellulitis developed over the next 24 hours, with proptosis, severe periorbital oedema, and gross restriction of ocular movement. Blood cultures were taken, and treatment with intravenous gentamicin and ampicillin was commenced, along with topical chloramphenicol and oral prednisolone. Full blood examination revealed a neutrophil leucocytosis of $18{\cdot}8\times10^{9}$/l. The findings on CT scan were consistent with an orbital cellulitis, and no space occupying lesion was identified. Sinus *x*-rays were normal.

By the following day (three days after presentation) the pain had decreased and the lid oedema had lessened, but there was still marked conjunctival chemosis, corneal oedema, and hyphaema. *Clostridium perfringens* was grown from the blood culture, and intravenous therapy was changed to penicillin and cephalexin. The orbital cellulitis settled rapidly, but the hyphaema was replaced by an obvious hypopyon. Within two months the eye became phithisical. A hyphaema recurred 12 months after presentation, and the patient continues to suffer from recurrent episodes of chemosis and pain in the left eye.

The case was similar in many ways to the one presented by Cannistra *et al.*, but the history of visual loss and diplopia over several months was suggestive of a low grade infection that suddenly took a fulminant course.

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Reference

1 Cannistra. *Br J Ophthalmol* 1988; **72:** 380–5.

Note

International Strabismological Association

The Sixth Congress will be held at Surfers Paradise, Gold Coast, Queensland, Australia, on 11-16 March 1990.