tion. It remains to be seen whether the extracapsular technique will 'protect' the retina and show a lower incidence of retinal tear and detachment.

Vascular complications such as arterial or venous occlusion occurred in 4 eyes (and in a slightly greater number in the unselected plain extractions). This must be expected as an occasional ocular complication in elderly patients.

Removal of 8 iris clip lenses has been carried out (all in earlier cases). Three lenses had dislocated, 2 patients had iris prolapse, 2 had updrawn pupils and one had persistent pain and keratitis. One of these eyes (in a 90-year-old) was subsequently lost due to gross infection.

What is the Future of Iris Clip Lenses?

The patient-satisfaction following successful implant surgery is striking. The absence of aniseikonia and the good binocular function contribute to this, and are superior to the results obtained with contact lenses. Only the incidence of macular œdema inhibits the wider use of these clip lenses. There is now strong evidence, however, that an extracapsular technique confers immunity to the macular region. Future cataract management may be recommended as either (1) extracapsular extraction and primary iridocapsular implant; or (2) phakoemulsification followed by elective implant, contact lens trial in younger patients, with secondary implant if contact lenses are not successful.

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Cystoid Macular Œdema

One avenue of the continuous drive towards perfection in cataract surgery is the development of the intraocular acrylic lens by Ridley in 1952. The problems arising in this refinement have been contained by surgical technique and controlled by adept medical care with steroids and other preparations. One complication so far eluding control is that of aphakic cystoid œdema. First described by Irvine (1953), the year after intraocular lenses were reported, it was subsequently reported by Nicholls (1954), Chandler (1954), Gartner (1964), and Tolentino & Schepens (1965); and Gass & Norton (1966) clarified the subject and gave clear angiographic criteria for the diagnosis. Subsequent reports, particularly that of Maumenee (1967), have drawn attention to the role of the vitreous. It is, however, clear from the reports of Satake (1971), Hitchins, Chisholm & Bird(1974), Gass & Norton (1969) and Hitchins and Chisholm (1975), that macular œdema is a transient episode in about 40% of cataract extractions. All authors seem to cite local vitreous changes and inflammation, and systemic problems, hypertension and diabetes, as basic precursors.

The original syndrome, however, was that of œdema occurring late after an initial period of recovery with normal vision. It occurred between four weeks and five years after the cataract extraction and was associated with a reduction in the visual acuity, which subsequently recovered. An incidence rate of between 2 and 2½% was the accepted norm and our review of 750 cases of classical cataract extraction gave a rate of 2.1%. Recovery of the vision may be, in part, responsible for the low reported incidence of bilaterality. François, De Laey & Verbraeken (1972) have reported a high incidence of bilaterality. Personal experience would tend to support this observation. The same report has suggested adrenaline as a possible contributory cause in addition to the usual ones of incarceration of the vitreous and late rupture of the hyaloid face. Inflammatory changes have been difficult to substantiate.

The role of macular traction is unclear. Those cases which show œdema in this situation fall into the syndrome of Irvine and Gass. Those without œdema but with definite cystoid changes are classified as Jaffe's syndrome (Jaffe 1967), which is divisible into three stages and is regarded as a relatively rare occurrence in classical aphakia (Bonnet 1973). The incidence of cystoid œdema in pseudophakia is variously reported as 5% by Pearce (1972) and 12% by Binkhorst and Leonard (1967) and Jardine & Sandford-Smith (1974). A significant feature of the visual change is the greater degree of loss and the failure to recover as fully. The incidence of micropsiæ and changes on the Amsler chart is considerably higher as in Jaffe's syndrome.

The presence of an implant may provide the nidus for the vitreal changes leading to posterior hyaloid detachment with traction, and accounts for the greater degree of visual loss. The fine retinal fold and vitreous traction band and, later, a preretinal membrane, can be identified more readily with the Goldmann contact lens and the Hruby lens. The use of intravenous fluorescein at this stage of the examination is more revealing than an attempted angiography with a camera.

A differentiation between the Irvine-Gass and Jaffe types is important, as the recovery of visual acuity is better in the first than in the second type. Personal observations suggest that in the Irvine-Gass type cystoid change accounts for the majority of cases in classical aphakia with, perhaps, 1% of the Jaffe type. In pseudophakia, however, the

incidence of the two types is about equal and this may account, in part, for the greater loss of visual acuity associated with cystoid changes in the implant series.

It will be argued by some that better binocular function is worth a line or two of acuity. Until the diminished acuity and the higher incidence of this complication in pseudophakia is reduced, the prudent physician will eschew the implant where changes in the vitreous or experience with the other eye indicate a need for caution.

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The Latest Facts and Figures on Anterior Chamber Lens Implants

In March 1965 I was privileged to present a paper to this Section on the development of the Choyce Mark VI, VII and VIII anterior chamber implants (Choyce 1965), and I have been asked to update this by reference to the latest facts and figures available. To some extent I am dependent on information published by or communicated to me by colleagues in different parts of the world, to whom I am grateful.

Fixation

The Mark VIII implant introduced in 1963 has four feet in the horizontal plane. This quadripedal fixation makes it very stable. If an implant is fitted which is 1 mm longer than the horizontal corneal diameter each foot rests snugly, just touching the sclera *immediately behind the scleral spur* (Fig 1), i.e. avoiding the trabecular meshwork and the endothelium. The iris is not interfered with and the pupil can contract or dilate normally. The stability of the implant is unaffected by the status of the posterior lens capsule. No intraocular suturing is required. The position of the lens is of course unaffected by head posture or physical activity.

Complications

The data in Tables 1 & 2 are derived from my own series of implants except where otherwise stated. The Mark VIII implants were all supplied by Rayners except for those used by Bedilo & Nedospasov (1975), who used Soviet brand ST-1 polymethylmethacrylate for making the implants in their own department in Archangel.

Table 2 shows the average anterior chamber depths. The mean depth in phakic eyes is 2.85 mm (Törnquist 1953), so that it is not surprising that the introduction of the Mark VIII implant reduced the incidence of endothelial corneal dystrophy (ECD) (the complication which led

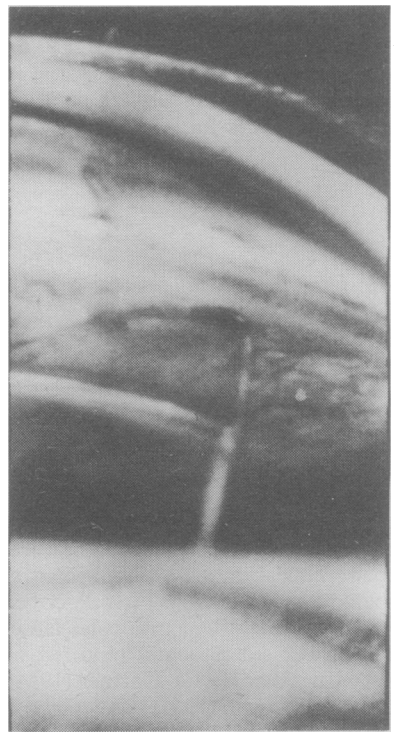

Fig 1 *Gonioscopic view of foot of implant resting against sclera just behind scleral spur, i.e. avoiding trabecular meshwork and endothelium (by courtesy of J L Tennant)*