

Incidence of aphakic macular oedema

A prospective study

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Irvine (1953) emphasized that macular degeneration was a major complication in cataract extraction. Gass and Norton (1966) described the appearance of aphakic cystoid macular oedema as shown by fluorescein angiography in patients who experienced a fall in visual acuity 4 to 12 weeks after cataract extraction. Gass and Norton (1969) showed that, while the oedema settled in 6 months in 50 per cent of the eyes involved, it persisted for over 3 years in 8 per cent. Tolentino and Schepens (1965) and Maumenee (1967) thought that 1 per cent of newly aphakic patients developed macular oedema, their views being based on the results of slit-lamp examination. By contrast, in a prospective study using fluorescein angiography, Irvine, Bresky, Crowder, Forster, Hunter, and Kulvin (1971) showed the much higher incidence of cystoid macular oedema of 40 per cent 6 to 8 weeks after cataract extraction. In this last series the oedema was transient in many cases and in only a few was the visual acuity severely affected.

In order to establish the incidence of macular oedema after cataract extraction and to assess the factors implicated in the pathogenesis of this condition, a prospective study was carried out on 100 consecutive patients undergoing cataract extraction who were cared for by us between October 1972 and June 1973.

Material and methods

Fluorescein angiography was carried out 6 weeks after cataract extraction, and those patients who had macular oedema at the time of the first study were asked to return 3 to 12 months after the initial angiogram. The angiographic technique used for all these studies was standard to the Retinal Diagnostic Department at Moorfields Eye Hospital, City Road (Teeters and Bird, 1973). Altogether 100 patients were asked to participate, of whom 29 either failed to attend for their appointment or had unsatisfactory photographs. As a result 71 patients, of whom 65 had unilateral and six bilateral extractions,

were analysed so that angiographic studies were available on 77 newly aphakic eyes.

Results

Of the 77 eyes studied 6 to 7 weeks after operation, 36 (46.7 per cent) showed cystoid oedema. The pattern of dye leakage varied from a classical petalloid appearance to a series of fluorescent spots around the foveal region (Figs 1a-d and 2a, b, overleaf). Disc oedema was noted in eight out of the 36 eyes with macular oedema.

VISUAL ACUITY

The mean visual acuity in the group without oedema, excluding the five patients mentioned below, was 6/6 while for those with oedema it was 6/9 (Fig. 3). There were five patients in the group without oedema who had a visual acuity of 6/18 or less, each of whom had a pre-existing ocular disease (two eyes myopic macular chorioretinal degeneration, one senile macular degeneration, and one retinoschisis affecting the macula; one eye was amblyopic). Cystoid oedema appeared to be the only cause for the poor vision in the group showing intraretinal dye accumulation.

PRE-EXISTING OCULAR AND SYSTEMIC DISEASE

At the time of the preoperative assessment, note was made of any disease, either ocular or systemic, that might affect the visual acuity after surgery. Table I shows the visual outcome of those patients with pre-existent ocular disease and Table II that of those patients with co-existent systemic disease.

OPERATIVE TECHNIQUE

Alpha chymotrypsin was used in 33 out of the 36 eyes (91.6 per cent) that showed leakage of fluorescein and in 37 out of the 41 (90.2 per cent) of those with no dye leakage ($P = > 0.10$). Cryoextraction was used in 29 out of the 36 eyes (80.5 per cent) that showed leakage and 31 out of the 41 (75.6 per cent) that did not ($P = > 0.10$).

OPERATIVE COMPLICATIONS

Table III shows the operative complications occurring in the two groups together with the visual acuity at 6 weeks.

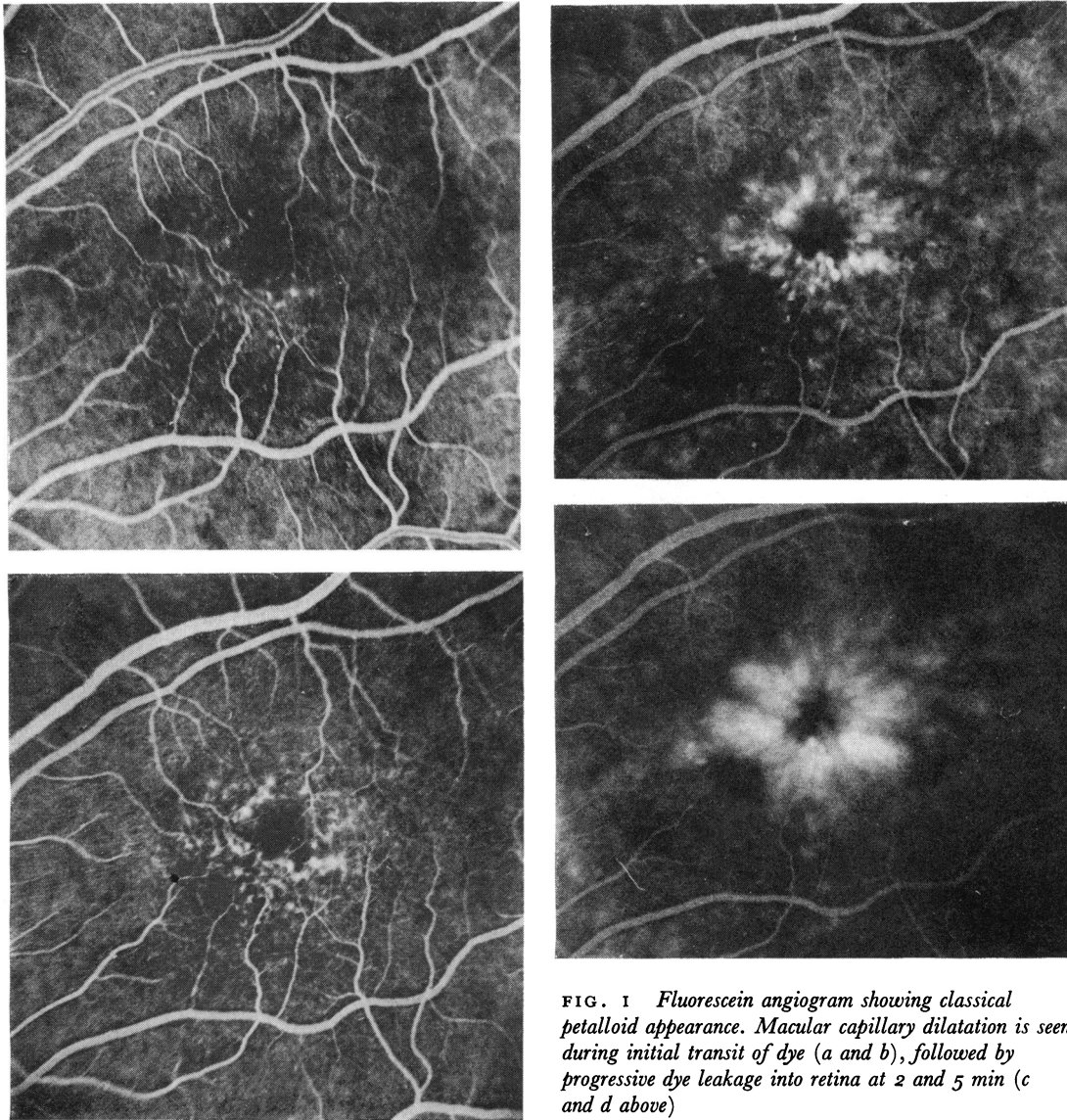


FIG. 1 Fluorescein angiogram showing classical petalloid appearance. Macular capillary dilatation is seen during initial transit of dye (a and b), followed by progressive dye leakage into retina at 2 and 5 min (c and d above)

Table I Pre-existent ocular disease and visual acuity at 6 weeks

<i>Oedema absent</i>	<i>Visual acuity</i>	<i>Oedema present</i>	<i>Visual acuity</i>
Subluxed lens	6/6	Heterochromic uveitis	6/6
Chronic simple glaucoma	6/12	Heterochromic uveitis	6/9
Senile macular degeneration	6/18		
Myopic chorio-retinal degeneration	6/18		
Retinoschisis affecting macula	6/24		
Amblyopia	6/24		
Myopic chorio-retinal degeneration	6/24		

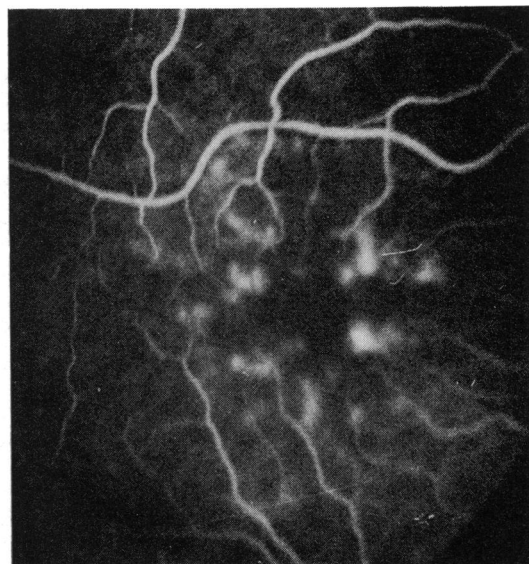
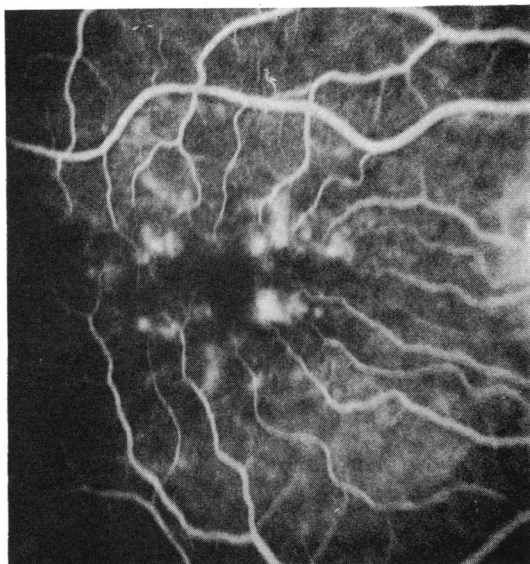


FIG. 2 A run for comparison with that in Fig. 1 showing series of fluorescent spots around foveal region (a) which are not followed by progressive dye leakage (b) in contrast to classical petalloid appearance

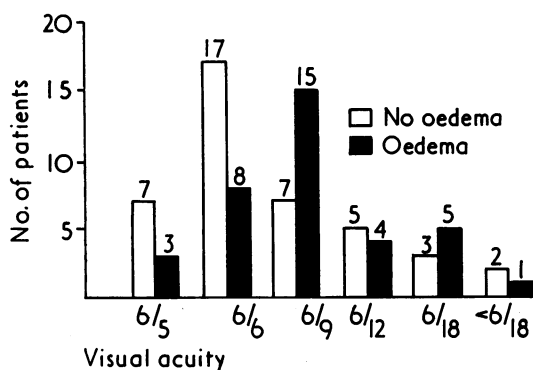


FIG. 3 Visual acuity at 6 weeks

Vitreous loss occurred in four patients, only two of whom developed macular oedema. In each case the other operative complications (two ruptured capsules, one operative hyphaema, and one persistent flat anterior chamber) were followed by macular oedema.

INTRAOCULAR PRESSURE

There were 16 eyes which had an intraocular pressure of 8 mm Hg or less lasting for 1 day or more. Hypotony was not significantly more frequent in those patients with macular oedema (eight out of 36 (22.2 per cent)) when compared with those without oedema (eight out of 41 (19.5 per cent)) ($P = > 0.10$). Similarly, ocular hypertension did not appear to be related to the incidence of macular oedema. A pressure of 25 mm Hg or greater, lasting a day or more, was recorded in six out of the 36 with oedema (16.6 per cent) and six out of the 41 without oedema (14.6 per cent) ($P = > 0.10$).

MEDICATION

Topical mydriatics and antibiotics were used routinely in all patients and steroids were used in 54 eyes. Oedema occurred in 23 out of the 54 who were given steroids (44.2 per cent) compared with 13 out of the 23 who were not given steroids (56.5 per cent) ($P = > 0.10$). No patient was given topical adrenergic drops in the postoperative period.

Table II Co-existent systemic disease and visual acuity at 6 weeks

Oedema absent	Visual acuity	Oedema present	Visual acuity
Rheumatoid arthritis on steroids	6/6	Hypertension	6/6
Rheumatoid arthritis on steroids	6/24	Hypertension	6/9
Ischaemic disease, previous coronary	6/9	Diabetes mellitus	6/6
Ischaemic disease, claudication	6/9	Diabetes mellitus	6/18

Table III Operative complications and visual acuity at 6 weeks

<i>Oedema absent</i>	<i>Visual acuity</i>	<i>Oedema present</i>	<i>Visual acuity</i>
Vitreous loss	6/6	Vitreous loss	6/9
Vitreous loss (high myope with chorio-retinal degeneration)	6/18	Vitreous loss	6/9
		Lens capsule rupture	6/6
		Lens capsule rupture	6/6
		Massive hyphaema	6/9
		Persistent flat anterior chamber	6/18

AGE AND SEX

There was no difference in the mean age or sex distribution of the two groups.

FOLLOW-UP STUDY

Of the 36 patients (29 eyes) who had macular oedema 6 weeks after the operation 28 were re-studied. Examination of the visual acuity at 6 weeks suggests that those patients who returned were representative of the group as a whole (mean visual acuity at 6 weeks of those who later returned 6/9 and of those who did not 6/7.5). In 16 eyes the oedema had resolved, in eight eyes it had improved showing minimal leakage, in two eyes there was no change, and in three the leakage was worse (Figs 2, 4a, b and 5a, b, c). The mean visual acuity of this group was now 6/7.5 (Fig. 6) whereas it had previously been 6/9. Nine patients showed an improvement of two or more lines on the Snellen test types, while four patients showed a deterioration of two or more lines. Nine patients still had a visual acuity of 6/12 or worse, of whom seven had persistent oedema and only one of these had had a spontaneous

rupture of the vitreous face. In two, poor vision persisted despite resolution of the oedema.

Discussion

This study shows that cystoid oedema is a common sequel to cataract extraction; it occurred in 46.7 per cent of the cases reported here which is very close to the figure of 40 per cent reported by Irvine and others (1971). The angiograms were performed 6-7 weeks postoperatively at a time when the incidence of oedema was thought to be highest (Irvine and others, 1971). Therefore it is likely that most of those patients who were destined to develop oedema were identified. A few cases showed increased leakage of fluorescein at the time of the second angiogram as compared with the first, so that some who did not have oedema initially may have developed oedema later. Identification of these would have given an even higher incidence of this complication.

There was only slight reduction in the mean visual

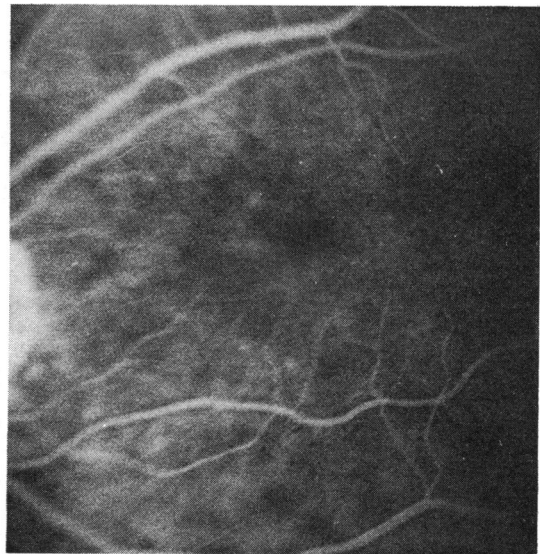
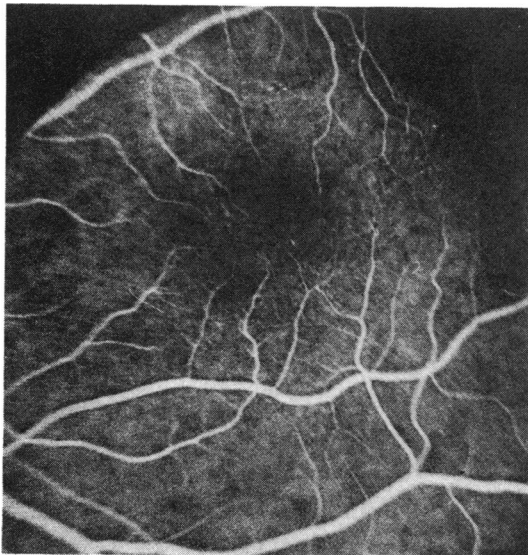


FIG. 4 Repeat angiograms on the patient shown in Fig. 1 to show resolution of oedema (early picture a, late picture b)

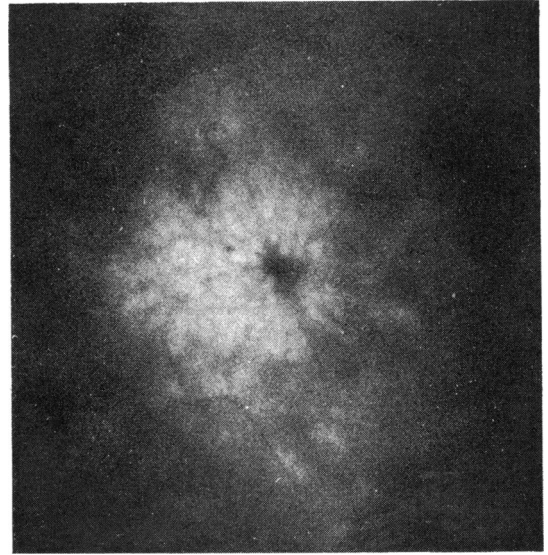
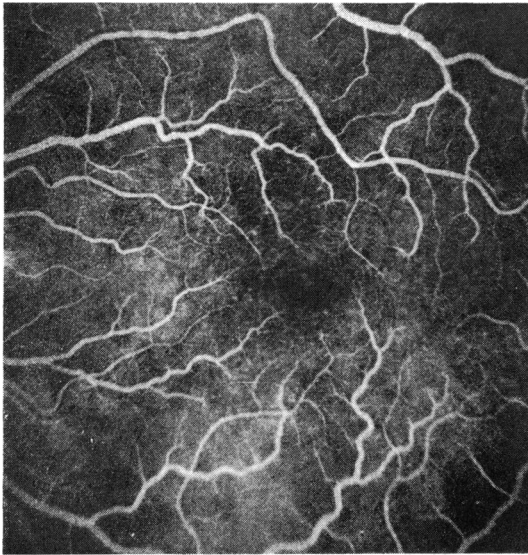


FIG. 5 *Fluorescein angiogram showing diffuse fluorescence in initial study (a, b) and no petalloid appearance. The repeat angiogram shows marked petalloid oedema at 5 min (c) (above)*

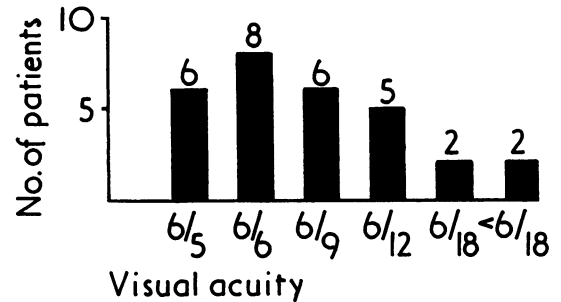
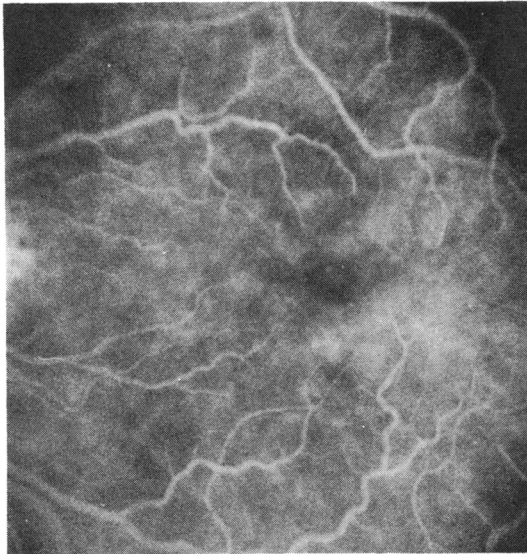


FIG. 6 *Visual acuity at time of follow-up study*

acuity in the patients with oedema at 6 weeks, 6/9 compared with 6/6 for those without oedema. Some eyes with oedema preserved excellent visual acuity while others showed marked visual loss. There was no apparent correlation between degree of dye leakage and visual loss. Some patients had a visual acuity of 6/6 despite marked oedema, and others had poor acuity in the presence of relatively mild changes.

At the time of the follow-up examination, the mean visual acuity had improved but, at 6/7.5, it was still worse than that of the eyes without oedema. Three out of the four patients whose visual acuity fell between the time of their first and second examina-

tions showed an increase in the extent of the cystoid oedema, and conversely a reduction in the fluorescein leakage was accompanied by an improvement of visual acuity in all but two patients. In these two patients the resolution of oedema was not associated with recovery of vision, and, as no other cause could be found for the lack of improvement at the time of the follow-up study, it was presumed that the visual loss was the result of structural damage to the retina caused by the oedema. We concluded that, while there was no apparent correlation between the extent of retinal oedema and visual acuity at the time of the initial study, macular oedema was responsible for the visual loss in these patients.

Many factors have been implicated in the causation of this syndrome which is presumed to be the result of leakage from the macular capillaries. Rupture of

the anterior hyaloid face, considered to be contributory by several authors (Irvine, 1953; Gass and Norton, 1966, 1969) was found in only one patient in the present series at the time of the follow-up study. Direct vitreo-macular traction has also been implicated (Irvine, 1953; Tolentino and Schepens, 1965; Reese, Jones, and Cooper, 1967) but several authors have failed to observe this (Gass and Norton, 1966; Maumenee, 1967; Irvine and others, 1971). Furthermore, Jaffe (1967) demonstrated that macular oedema due to vitreous traction is quite different morphologically from cystoid macular oedema occurring after cataract extraction. Posterior vitreous detachments were noted to occur very commonly in these patients (Gass and Norton, 1966, 1969). Inflammatory cells were seen in the vitreous in most of our cases confirming the observation of several previous authors (Gass and Norton, 1966; Maumenee, 1967; Irvine and others, 1971). However, the syndrome has been seen to occur when these cells are absent and conversely vitreous inflammation is not always associated with cystoid macular oedema (Gass and Norton, 1969). Because of the frequent occurrence of vitreous cells after cataract extraction it is difficult to assess the importance of inflammation in the pathogenesis of cystoid oedema. If intraocular inflammation were a major factor, then topical steroids might be expected to reduce the incidence of the oedema. While the incidence of oedema in patients who were given topical steroids (23 out of 54) was less than in those who were not given steroids (13 out of 21), the difference was not significant at the 0.10 level. The use of topical steroids was dependent on the preference of the consultant surgeon rather than the degree of postoperative inflammation. Both the eyes with pre-existing intraocular inflammatory disease (heterochromic uveitis) developed cystoid oedema.

Gass and Norton (1969) found a high incidence of cardiovascular disease in patients who developed cystoid oedema. In the present series both the patients whose hypertension was severe enough to require treatment developed cystoid macular oedema, but the two patients with evidence of ischaemic vascular disease did not.

Postoperative hypotony has been suggested by some as a cause of this problem (Dellaporta, 1955; Chandler, 1954; Welch and Cooper, 1958; Satake, 1971) but is rejected by others (Gass and Norton, 1966, 1969; Irvine and others, 1971; Jaffe, 1967). The syndrome of hypotonic maculopathy was described by Gass (1972) who noted that macular oedema and small retinal vessel leakage did not occur. We found that the incidence of oedema was not higher in eyes with either hypotension or hypertension.

Postoperative medication with topical adrenaline has been suggested as a factor (Kolker and Becker,

1968; Gass and Norton, 1969). None of our patients received topical adrenaline.

There was a higher rate of operative complications in the group that leaked fluorescein, although vitreous loss, which occurred in four patients, was equally divided between those who had oedema and those who had not. A higher proportion of those cases in which alpha chymotrypsin was used showed leakage of fluorescein at 6 weeks compared with those who showed no leakage, but the difference was not significant ($P = > 0.10$).

In the follow-up group of patients no factor appeared to be especially prominent as a cause of persistent leakage of fluorescein.

Cystoid macular oedema may result from a number of contributory factors among which must be considered operative complications, pre-existing vascular disease, and past inflammatory eye disease. In the present series we were unable to identify a positive relationship between the occurrence of macular oedema and the incidence of vitreous face rupture, vitreous loss, the use of alpha chymotrypsin, or the occurrence of post-operative hypotony. Vascular disease was identified in too few cases for any conclusions to be drawn. Postoperative inflammation may account for the higher incidence of oedema in those patients who suffered operative complications. It is possible that postoperative inflammation plays an important role, since there was a lower incidence of oedema in patients receiving topical steroids compared with those who did not, although the level of significance was low.

Summary

A prospective angiographic study of 77 aphakic eyes demonstrated cystoid macular oedema in 36 eyes (46.7 per cent) 6 to 7 weeks postoperatively. This subsequently resolved in 16 eyes, improved in eight, and was no better or worse in five (eight patients failed to return for follow-up). Of those patients with cystoid oedema, 16.6 per cent had a visual acuity of less than 6/12 at the time of the original examination; this was reduced to 13.8 per cent at the time of the follow-up study. The mean visual acuity of the group with oedema improved from 6/9 to 6/7.5 between the two examinations compared with 6/6 in the remaining 53.3 per cent without oedema. Preoperative ocular or general disease, specific surgical technique, and vitreous loss were not shown to be associated with macular oedema. However, there was some indication that intraocular inflammation predisposed to macular oedema.

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