

Macular abnormalities in the reattached retina

P. E. CLEARY AND P. K. LEAVER

From Moorfields Eye Hospital and the Institute of Ophthalmology, London

SUMMARY Sixty-six patients in whom the macula was detached before surgery were observed for at least 1 year after retinal reattachment. Macular abnormalities were recognised clinically in most patients with severely reduced vision. Failure of retinal receptor regeneration or receptor misalignment may account for visual reduction in a minority of patients but may be less important as a cause of reduced visual acuity than was previously supposed. This study confirms that the visual prognosis is related to the duration of the detachment before surgery, and patients with a macular detachment for 2 months or longer are likely to suffer persistently reduced vision.

Visual recovery is frequently incomplete after surgical reattachment of the retina. The visual acuity after successful reattachment has been documented by several workers (Jay, 1965; Hudson, 1968; Gundry and Davies, 1974; Grupposo, 1975), but the precise causes of reduced vision in the patients studied were not identified. The recovery of visual acuity for up to 2 years after retinal reattachment and the improvement in hue discrimination for as long as 4 years have been attributed to regeneration of retinal receptor outer segments (Foulds and Ikeda, 1966; Chisholm *et al.*, 1973; Gundry and Davies, 1974). Such regeneration has been demonstrated in the owl and rhesus monkey after repositioning of experimentally induced retinal detachments (Machemer, 1968b; Kroll and Machemer, 1969). Conversely, a failure of visual recovery has been accounted for by incomplete receptor regeneration or by receptor misalignment (Machemer, 1968b; Aaberg and Machemer, 1970; Enoch *et al.*, 1973). It is acknowledged that the visual outcome after successful retinal detachment surgery may also be prejudiced by the presence of cystoid degeneration at the macula (Reese, 1937), pigment epithelial changes at the macula (Reese, 1937; Hudson, 1968), macular pucker (Hudson, 1968; Tanenbaum and Schepens, 1969; Gundry and Davies, 1974), or macular oedema (Ryan, 1973), although the precise incidence of detectable macular abnormalities in the reattached retina is unknown. We undertook this study to document the morphological abnormalities observed clinically at the macula after surgical reattachment of the retina in

an attempt to assess the importance of detectable macular abnormalities at the macula relative to recovery of visual acuity.

Patients and methods

A total of 75 patients were entered in this study, but 3 were withdrawn because of increasing lens opacities and 6 failed to attend for follow-up examination. We are reporting on 66 who had been observed for at least 1 year. Consecutive patients were selected in whom the macula was detached preoperatively and reattached postoperatively. Patients were excluded when there was a recognised pre-existing macular disease, opacities in the ocular media, giant retinal tears, and when silicone oil injection had been used to flatten the retina.

Examinations were carried out at intervals of 2 weeks, 4 weeks, 3 months, 6 months, 1 year, and 2 years after surgery. At each examination, the following assessments were made: corrected visual acuity, biomicroscopy of the anterior segment and vitreous, fundus examination by binocular indirect ophthalmoscopy, and a detailed clinical examination of the macula using the Hruby lens. The appearance of the macula was recorded by stereoscopic colour photography and fluorescein fundus angiography at each visit.

Results

VISUAL ACUITIES

At 1 year after surgery the visual acuity had improved in 54 (82%) patients, remained the same in 7 (11%), and worsened in 5 (7%) (Fig. 1). A visual acuity of 6/60 or less was recorded in 47 patients

(71%) preoperatively but in only 15 patients (22%) at one year after surgery. At 1 year 25 patients (37%) had recovered to a visual acuity of 6/12 or better, but only 2 patients had a visual acuity of 6/6 (Fig. 2).

MORPHOLOGICAL APPEARANCES

The macula appeared normal, both on clinical examination and on fluorescein angiography, in 16 patients. Only 3 patients with a normal macular appearance had a visual acuity of less than 6/12

(Fig. 3). In 1 of these amblyopia was suspected; in another, a high myope, the best vision in the unoperated eye was 6/18; and the 1 other patient had a normal macular appearance with a visual acuity of 6/24 which was unexplained.

A disturbance of the surface reflex at the macula with or without recognisable preretinal fibrous tissue, but not associated with distortion of the retinal vessels (cellophane maculopathy), was observed in 6 patients (Fig. 4a). The visual acuity was 6/12 or better in 4 of these and 6/18 in the

Fig. 1 Scattergram showing change in visual acuity in 66 eyes 1 year after retinal reattachment (PL = perception of light. HM = hand movements. CF = counting fingers)

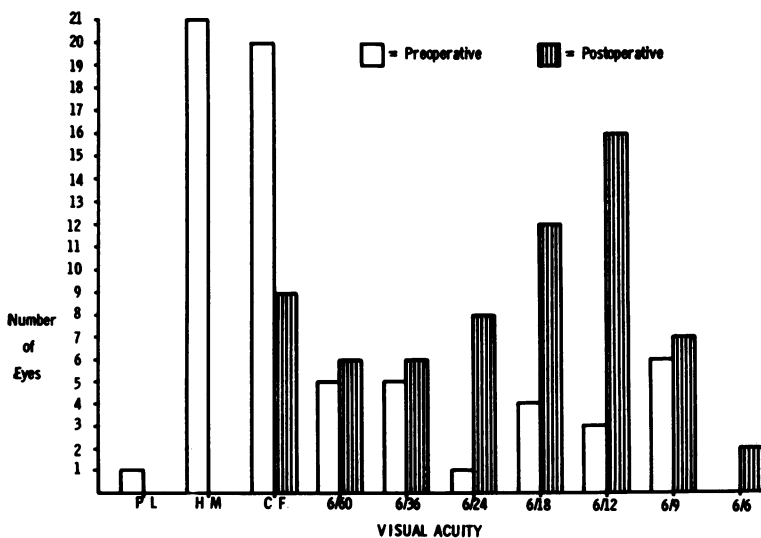
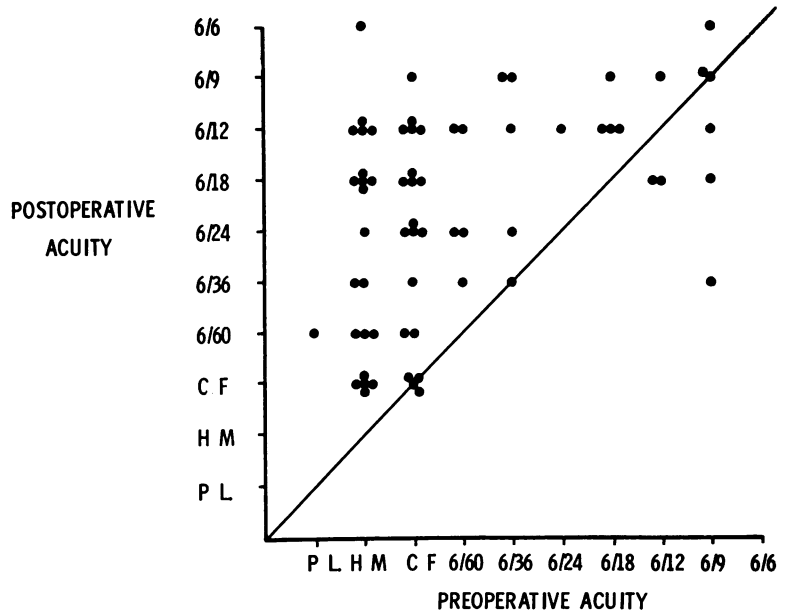


Fig. 2 Histogram showing the change in distribution of visual acuity in 66 eyes 1 year after retinal reattachment

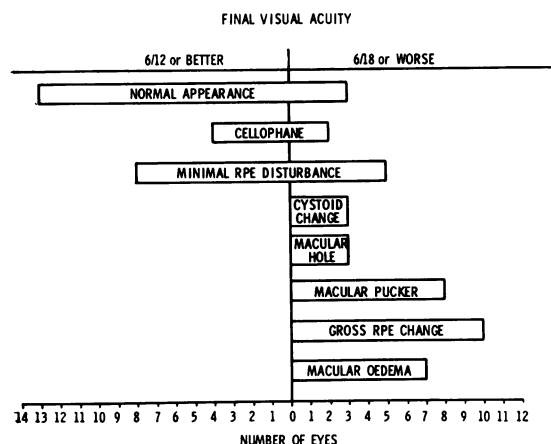


Fig. 3 Bar chart showing the relationship between the morphological appearance of the macula and the visual acuity in 66 eyes 1 year after retinal reattachment (RPE = retinal pigment epithelium)

other 2 (Fig. 3). More severe preretinal fibrosis with opaque fibrous tissue at the macula and distortion of the retina and traction on the surrounding retinal vessels (macular pucker) occurred in 8 patients and was associated with a visual acuity of 6/36 or less in all (Figs. 3 and 4b). Fluorescein angiography showed the marked distortion of the retinal blood vessels at the macula and the accompanying dye leakage.

Minimal disturbance in the retinal pigment epithelium at the macula was diagnosed and confirmed by fluorescein angiography in 13 patients. Of these, 8 had a visual acuity of 6/12 or better, 3 had a visual acuity of 6/18, and 2 had a visual acuity of 6/24 (Fig. 3). Gross disturbance in the retinal pigment epithelium at the macula occurred in 10 patients. In 4 of these, pigment epithelial abnormalities followed the resolution of intraretinal cystoid changes (Fig. 5). One patient had a visual acuity of 6/24, and all the other patients had a visual acuity of 6/36 or worse (Fig. 3).

Cystoid changes were visible at the macula in the early studies of 7 patients, all of whom were phakic. The intraretinal cystoid spaces were seen on biomicroscopic examination and colour photographs, but the angiographic appearance of the macula was unremarkable (Figs. 5 and 6). However, at 6 months after surgery 4 of these patients had gross disturbance of the retinal pigment epithelium at the macula and the cystoid spaces were no longer visible (Fig. 5). The 3 patients with persistent cystoid changes had visual acuities of 6/18, 6/24, and 6/36.

Macular oedema characterised by intraretinal accumulation of dye during fluorescein angiography

occurred in a total of 17 patients (Figs. 7 and 8). Persistent oedema was associated with a visual acuity of 6/18 or worse (Fig. 7). In 2 aphakic patients the oedema resolved in one and persisted in the other. Ten patients had macular oedema at 9 months after surgery, and in 7 it was still present at 18 months (Fig. 9).

A full-thickness macular hole was seen after retinal reattachment in 3 patients, and all 3 patients had a visual acuity of 6/60. Fluorescein angiography showed the typical central transmission defects.

Pigment fallout was observed at the macula in 7 patients. Four had a visual acuity of 6/12 or better. The macula was otherwise normal in 2 of

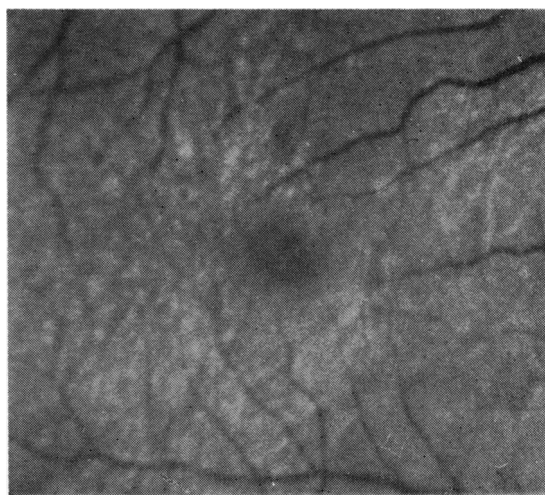


Fig. 4a Red-free photograph showing a mild degree of preretinal fibrosis at the macula (cellophane maculopathy)



Fig. 4b Red-free photograph showing macular pucker

these. One patient had a minor disturbance in the retinal pigment epithelium and one had a cellophane maculopathy. The 3 other patients with pigment fallout had a visual acuity of 6/36 or worse, and all had other changes at the macula—either macular pucker, a gross disturbance in the retinal pigment epithelium, or persistent macular oedema.

DURATION OF RETINAL DETACHMENT

The duration of retinal detachment varied from less than 1 week in some patients to more than 2 years

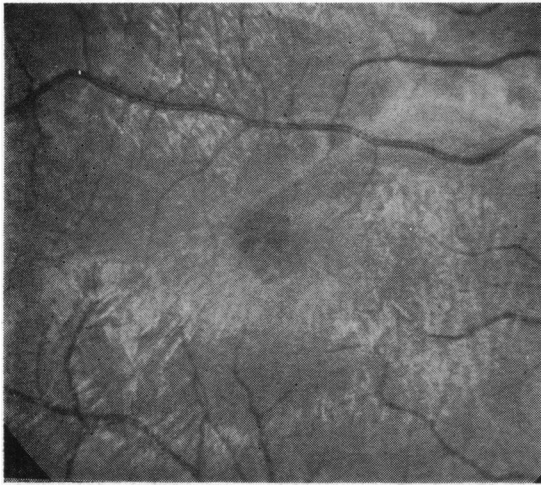


Fig. 5a Red-free photograph showing intraretinal cystoid spaces at the macula 3 months after reattachment. The high watermark of the detachment is visible running obliquely above the superior temporal vessels

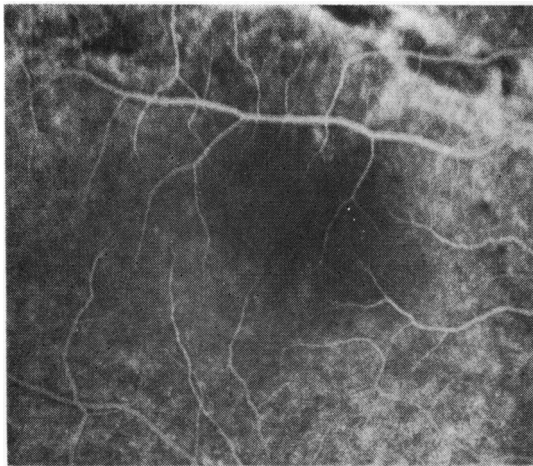


Fig. 5b The fluorescein angiogram at 3 months showing no leakage of dye from perifoveal retinal capillaries. The high watermark is clearly visible



Fig. 5c Red-free photograph 9 months after retinal reattachment. The intraretinal cystoid spaces are no longer visible, but there are retinal pigment epithelial changes at the macula

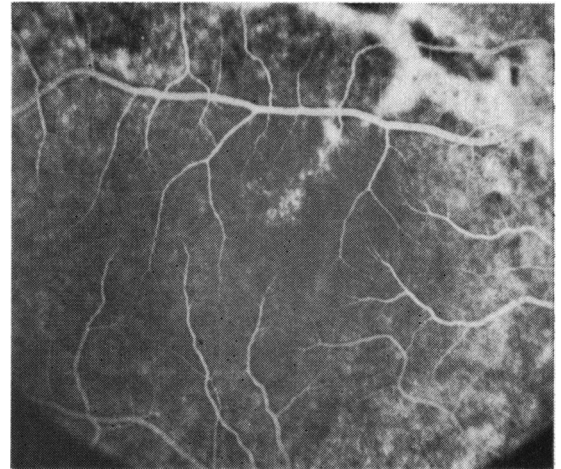


Fig. 5d The fluorescein angiogram now shows transmission defects corresponding with the retinal pigment epithelial changes

in others (Fig. 10). After a retinal detachment of 1 month's duration or less the macula was usually normal or showed pucker, oedema, or minimal retinal pigment epithelial changes. However, a retinal detachment of 2 months' duration or longer was followed by gross retinal pigment epithelial changes at the macula in 8 out of 16 patients (50%) in this series (Fig. 10).

TYPE OF SURGICAL PROCEDURE

All eyes were treated by scleral buckling and

cryopexy through full-thickness sclera. Local Silastic sponge explants were used in 55 and encircling silicone-rubber bands in 11. In 27 eyes the subretinal fluid was drained, while in 39 it was left to absorb spontaneously.

Of 14 eyes in which preretinal fibrosis occurred at the macula after surgery 9 had local explants and 5 had encircling bands; 8 were drained and 6 were not. In 17 eyes with macular oedema there were 10 local explants and 7 encircling bands; 10 were drained and 7 were not.

Discussion

Macular abnormalities were identified in the majority of patients with severely reduced vision after retinal reattachment. Abnormalities such as cystoid degeneration at the macula, macular hole, macular pucker, a gross retinal pigment epithelial disturbance, or macular oedema appeared easily sufficient to account for a low visual acuity (Fig. 3). However, persistently reduced vision could not be attributed to macular changes in every case. A minority of patients had either a normal macula, cellophane maculopathy, or a minimal disturbance of the retinal pigment epithelium, with low visual acuity, and it is conceivable that visual reduction in these patients is the result of an abnormality or failure of retinal receptor regeneration. Indeed, abnormal or incomplete retinal receptor regeneration may explain why so few patients in this series achieved a visual acuity of 6/6 (Fig. 2).

In the clinical examination of the macula we found the magnification offered by slit-lamp biomicroscopy as opposed to indirect ophthalmoscopy particularly useful. Fluorescein angiography was also helpful in distinguishing macular oedema from cystoid degeneration, in demonstrating macular pucker, and in confirming the presence of macular hole or retinal pigment epithelial disturbance.

Preretinal fibrous tissue at the macula was the most frequently recognised abnormality. It is acknowledged as a common cause of poor vision after successful retinal detachment surgery (Gass, 1972; Gundry and Davies, 1974), varying from a mild disturbance of the surface reflex from the retina (cellophane maculopathy), with minimal visual loss, to severe retinal distortion (macular pucker), with profound loss of central vision. The pathogenesis of preretinal fibrosis is complex (Foos, 1974; Gloor and Daicker, 1975; Bellhorn *et al.*, 1975; Machemer and Laqua, 1975), but in this study it was apparently unrelated to the type, extent, or duration of the retinal detachment, or to

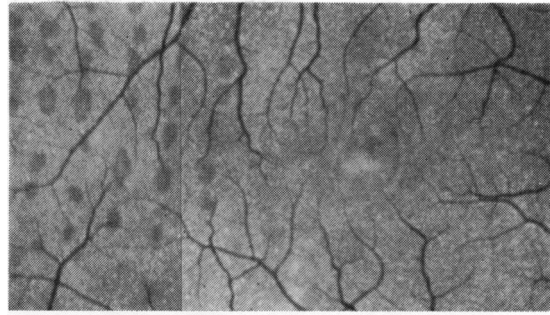


Fig. 6a Red-free photographs at 3 months showing thickened retina and intraretinal cystoid spaces at the macula. Temporal to the macula are multiple round lesions, probably at the level of the retinal pigment epithelium, which may represent resolution of small localised serous retinal detachments

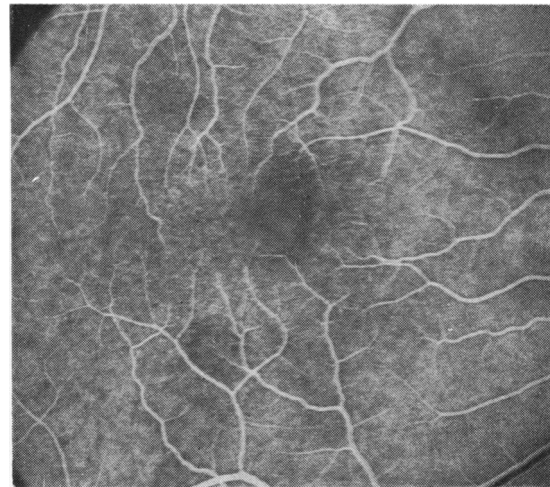


Fig. 6b Fluorescein angiogram shows no leakage from perifoveal retinal capillaries. The multiple round lesions show faintly as masking the background choroidal fluorescence

the type of surgical procedure used to reattach the retina.

Pigmentary changes in the reattached retina can be attributed to degeneration and loss of retinal pigment epithelial cells (Hogan and Zimmerman, 1962; Machemer, 1968a). Typically, gross disturbance of the retinal pigment epithelium followed long-standing retinal detachments, where atrophy of the retinal receptor layer could also have accounted for poor vision (Hogan and Zimmerman, 1962; Aaberg and Machemer, 1970). Pigmentary changes have also been attributed to choroidal infarction as a result of scleral buckling (Foulds *et al.*, 1971), and in this study a single patient had

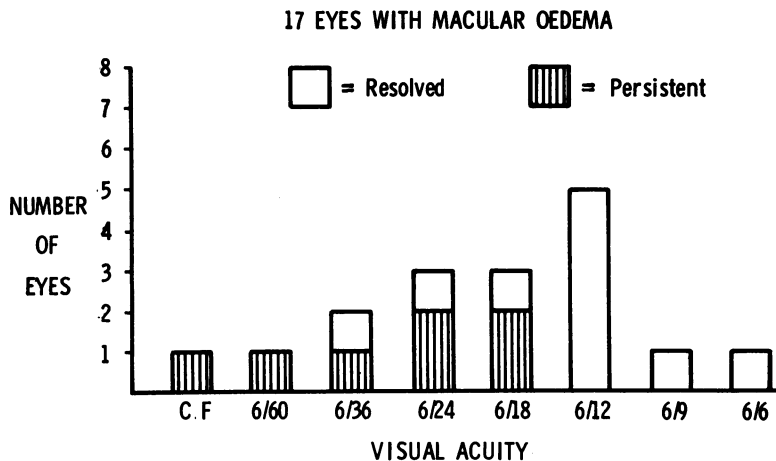


Fig. 7 Histogram showing visual outcome at 1 year after retinal reattachment in 17 eyes with macular oedema

grossly delayed choroidal perfusion on fluorescein angiography which corresponded with an area of retinal pigment epithelial disturbance involving the macula. However, no other patient had impaired choroidal perfusion in the macular or paramacular areas, although this does not exclude ischaemic episodes at the time of surgery.

Cystoid macular oedema characterised by leakage from retinal vessels has been recognised as a cause of reduced vision after retinal detachment surgery in both aphakic and phakic eyes (Ryan, 1973). This finding was supported by our observation that persistent macular oedema was associated with a visual acuity of 6/18 or worse, and resolution of oedema was accompanied by an improvement in

visual acuity (Fig. 7). Macular oedema was less common than after cataract extraction, but its course was more prolonged and it persisted more often (Hitchings *et al.*, 1975; Meredith *et al.*, 1976). The presence of oedema did not correlate with the age of the patient, with the apparent degree of intraocular inflammation, or with the type of surgical procedure used to reattach the retina.

We have distinguished between cystoid degeneration, without leakage from retinal vessels on fluorescein angiography, and cystoid macular oedema. Typically, cystoid degeneration occurs after long-standing retinal detachments. It may be accompanied by loss of neural tissue in the retina and atrophy of the receptors (Hogan and Zimmerman, 1962:

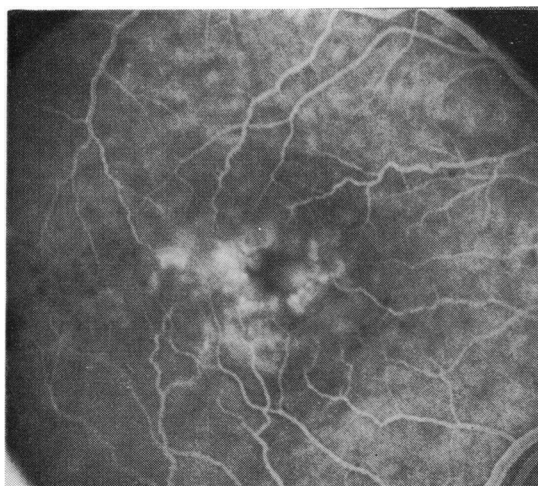


Fig. 8a Fluorescein angiogram at 6 months showing macular oedema

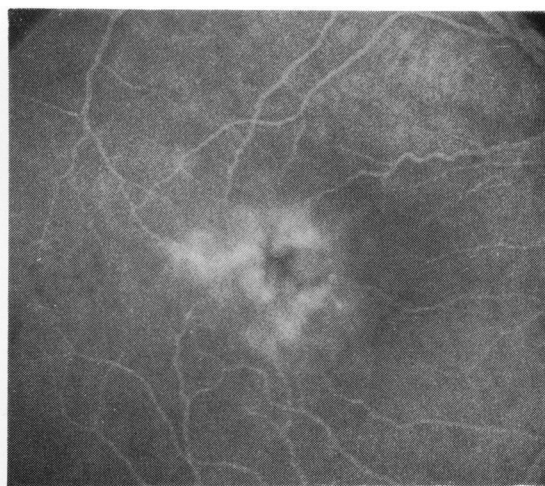


Fig. 8b Fluorescein angiogram (same patient as in 8a) at 15 months showing persistence of macular oedema

DURATION OF MACULAR OEDEMA
(17 Eyes)

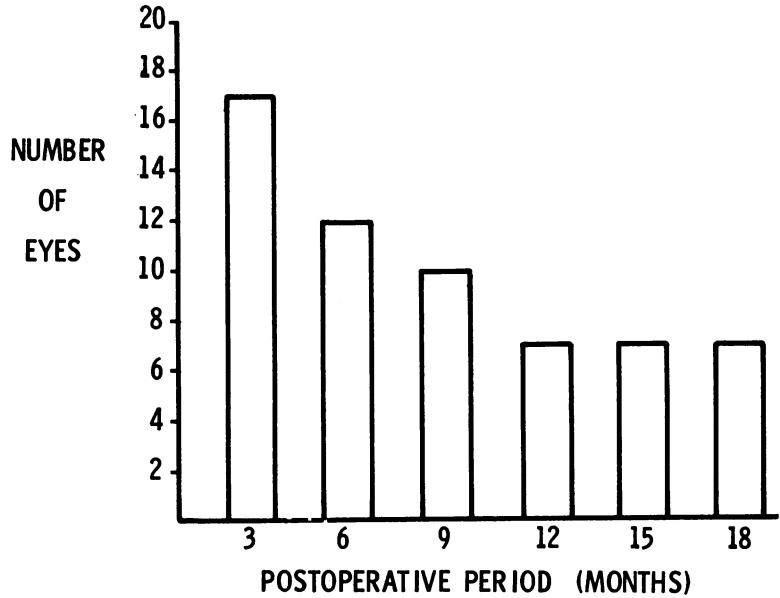


Fig. 9 Histogram showing the duration of macular oedema in 17 eyes up to 18 months after retinal reattachment

Machemer, 1968a), which could certainly account for persistently reduced vision. When the cystoid degeneration resolved, it was often followed by retinal pigment epithelial changes at the macula as noted previously (Reese, 1937), but the visual acuity did not improve. The development of these

cystoid changes, which are unrelated to retinal capillary permeability, may be the result of a metabolic abnormality in the detached retina (Aaberg and Machemer, 1970) and may resemble the cystoid degeneration seen at the macula in patients with juvenile X-linked retinoschisis (Deutman, 1971)

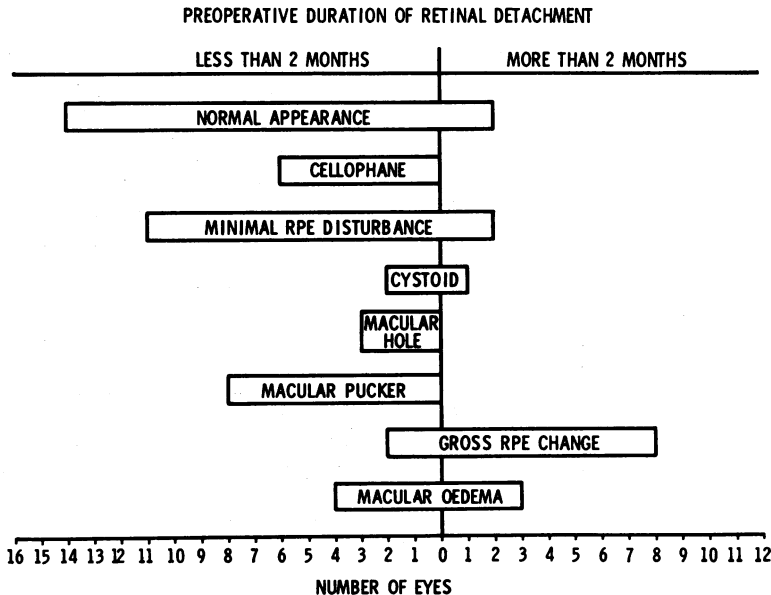


Fig. 10 Bar chart showing the relationship between the morphological appearance at the macula 1 year after surgery and the preoperative duration of retinal detachment

and in some patients with retinitis pigmentosa (Gass, 1970; ffytche, 1972; Fishman *et al.*, 1977) and in nicotinic acid maculopathy (Gass, 1973).

Reduced vision after retinal reattachment has also been attributed to subretinal pigment migration and accumulation at the macula as a result of cryotherapy (Shea, 1968; Abraham and Shea, 1969; Sudarsky and Yanuzzi, 1970). We observed 3 patients with pigment fallout at the macula and reduced vision, but all 3 had other macular abnormalities which were certainly sufficient to account for a low visual acuity. By contrast the 4 patients with only pigment fallout at the macula had good vision, supporting the assertion that subretinal pigment migration does not of itself result in visual loss or diminished retinal sensitivity (Hilton, 1974).

After surgical reattachment of experimentally induced retinal detachments in owl monkeys multiple small localised retinal detachments were demonstrated by fluorescein angiography and by histopathology (Machemer, 1968b). Such small localised detachments have not been documented in human eyes and were not identified in this series. However, a single patient showed an appearance suggestive of small localised serous detachments of the retina which had resolved and no longer filled with dye on the fluorescein angiogram (Fig. 5).

The results in this study suggest that severely reduced vision after retinal reattachment may be accounted for by morphological abnormalities at the macula which are easily recognised by slit-lamp biomicroscopy. It is confirmed that the visual prognosis is related to the duration of the detachment before surgery and that patients with a macular detachment for 2 months or longer are likely to suffer persistently reduced vision. Cystoid degeneration and gross retinal pigment epithelial changes follow long-standing retinal detachments and are largely predictable. However, macular pucker as a result of preretinal fibrosis and cystoid macular oedema due to leakage from retinal capillaries emerge as serious and unpredictable causes of severely reduced vision after retinal reattachment. Both conditions are responsible for low vision after a wide range of ophthalmic surgical procedures, their pathogenesis is ill understood, and at present both are difficult to treat. The prevention or successful treatment of these 2 complications could alter radically the visual prognosis for retinal detachment surgery.

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