The success rate of those cases with less than three lashes per lid was 75.6% while the overall success rate was 62.6%. This is in accord with other authors' finding.²⁻⁴ Sharif³ explained this by the number of laser burns per lash. Our finding shows the association between lower recurrence rate and higher number of burns per lash: with no recurrence there were 16.7 burns per lash and with recurrence there were 14.7 burns per lash. However, this association cannot be proved statistically. Another possible explanation is that in those lids with high number of aberrant hairs the disease leading to trichiasis is still active and the hairs regrow after treatment.

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Electroretinogram as indicator of prognosis of central retinal vein occlusion

EDITOR,—Matsui and colleagues¹ report on ERG b/a wave ratio changes in central vein obstruction and conclude that retinal ischaemia in ischaemic central retinal vein occlusion (CRVO) can be reversible. This result is surprising, as the natural history of ischaemic CRVO has been clearly documented² correlating the degree of retinal ischaemia with the development of neovascularisation – a process that may only be reversed by panretinal photocoagulation (PRP). Their findings are based on investigations including fluorescein angiography (FA) and electroretinogram (ERG), both of which appear to require further analysis.

Firstly, with respect to the FAs in the good prognostic groups which supposedly illustrate resolution of retinal ischaemia. In case 3, the initial FA at 1 month was masked by retinal haemorrhages and the degree of ischaemia cannot be determined owing to the absence of peripheral photographs. Therefore, the FA at 5.5 months, said to show significant improvement, merely shows the predictable changes expected in a well perfused CRVO. Despite inaccuracies in statements regarding the timing of treatment and photography in case 1 (Fig 4), we feel there is no evidence of improvement of ischaemia in (according to the caption) the recovery FA at 5.5 months; this clearly demonstrates marked capillary drop out and macular ischaemia. It also demonstrates PRP which was supposedly not performed until 11 months.

Secondly, two different ERG techniques were used with only one set of normal values and no affirmation that each individual patient was always examined using identical techniques.

Thirdly, the ERGs in Figure 2 show a speculative b-wave identification on an ERG with no replication, and result in a markedly longer a-b interval than when initially seen. If the b-wave were taken at the first visible peak, a very different amplitude value would be

obtained. What were the criteria for b-wave identification?

Finally, following PRP, if case 1 stabilised, how do the authors explain such marked trial to trial variations in a- and b-wave amplitude (Table 1)? What is the expected intersession variability for their laboratory (normally about 10%)?

We therefore suggest that Matsui *et al* reconsider their results, or repeat the study with greater scientific stringency, and reassess the validity of their original conclusion.

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Reply

EDITOR,—We appreciate the comments of Cordeiro and others.

Firstly, they questioned our interpretation of the ischaemic CRVO in case 3, whose FA could not be interpreted because of blood covering the retina. Our interpretation of the ischaemic retina came mainly from the ERG findings of low b/a amplitude ratio with normal a-wave. The blood in front of the retina will not decrease the b/a ratio. It will increase the ratio instead. Non-ischaemic CRVO would not show decreased ERG b/a ratio. However, the level of retinal ischaemia in case 3 would be moderate because the decrease of the b/a ratio is not as marked as that usually seen in complete occlusion of the central retinal artery.

The fundus photograph in case 3 shows extremely dilated retinal veins, extensive retinal haemorrhage, and some capillary dropout in the area not covered by the blood in the FA. Cordeiro *et al* mentioned that the clearing of the haemorrhage and normalisation of the veins, as documented in case 3, was a predictable outcome. I disagree. Predicting the final outcome of CRVO from the initial fundus appearance alone is difficult because one cannot judge the degree of retinal ischaemia if the retina is covered by blood. The ERG plays an important role in such cases.

There was an error in the caption for Figure 4. The FA in the middle of Figure 4 was taken after 11 months just before the PRP instead of 1 month. And the FA in the bottom was taken at 62 months instead of at 5.5 months. The captions of Figures 4 and 2 were mixed for which I apologise. Cordeiro *et al* feel that there is no evidence of improvement of the ischaemia after PRP (bottom Fig 4). The FA taken sometime after PRP was magnified in the posterior pole and shown in the bottom of Figure 4. It is clear that the central non-perfused area is decreased after PRP compared with that before the PRP. The spot in the fovea in the bottom of Figure 4 is a pigmented scar spontaneously developed after the macular oedema subsided.

Secondly, Cordeiro *et al* asked why two different ERG techniques were used with only one set of normal values. This report is a

retrospective study from data collected over many years. At one point we had to change the ERG system in our laboratory because of new recommended standardisation, and for other reasons. However, the majority (6/8 cases) had ERG recorded with the old system with description of the normal age matched values. Only two cases were recorded with the new system. ERG data used in those two cases were those recorded with bright single flash, strong enough to record oscillatory potential. The intensity was comparable with that of the old system. Only ERG b/a ratio was used in those two cases.

Thirdly, Cordeiro *et al* mentioned that the b-wave identification in Figure 2 was speculative. This criticism would be well taken if we had to measure the b-wave from this recording only. We routinely record ERG on EEG recording paper simultaneously with the cathode oscilloscope. In the reading on the EEG paper, whose paper speed is much slower, identification of the b-wave peak is easy. We recorded the amplitude from both these findings.

Finally, Cordeiro *et al* questioned if case 1 was stabilised, why was there a marked a- and b-wave amplitude variation after PRP. It would be wishful thinking that the retinal function would be stabilised after PRP. Obviously it was not. There was a change in the fundus appearance after PRP besides the scar formation on the laser spots, such as haemorrhage or vascular anastomosis during a long observation period after PRP, as described in the test. Vision fluctuated too. Therefore, it is not surprising to see some fluctuation of the ERG findings. In spite of this fluctuation, there was a trend in which the a-wave declined and the b-wave increased. As a result, the b/a amplitude ratio improved. The suggestion was made to repeat the study with greater scientific stringency. Such a study requires a long time if done by one institute. A nationwide study in CRVO has been recently completed in the United States in which multiple medical centres participated. This study included ERG and FA. The results of the study may disclose a similar case. Namely, ischaemia in CRVO may not be permanent and may be reversible in some.

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Effect of trabeculectomy on pulsatile ocular blood flow

EDITOR,—We read with interest the paper by James.¹

An increase of pulsatile ocular blood flow (POBF) was found in the standing position following trabeculectomy. This was attributed to an increase in perfusion pressure which is expected with reduction of intra-ocular pressure (IOP) assuming autoregulation was absent.

In the lying position, however, POBF was unchanged following trabeculectomy despite similar magnitudes of IOP lowering. It was suggested that in this group, because of the extremely high IOP in the lying position pre-operatively, the POBF was somehow maintained at an elevated level by autoregulatory mechanisms which masked any improvements in POBF after surgery. This was also felt to be responsible for the regaining of the usual postural changes following trabeculectomy.

The suggestion of an autoregulatory mechanism which can prevent further reduction in POBF when the IOP is very high seems plausible but it is curious that this effect was not seen in the standing position preoperatively when IOP was also high.

We agree with the author that changes in ocular rigidity can dampen the ocular pulse, but in this study ocular rigidity was assumed to be unaltered as the usual postural changes in IOP were observed following surgery. We feel that the reduced pulse amplitudes found in both positions following trabeculectomy when systemic pulse pressure was unaltered are worthy of comment. As the ocular pulse is a pressure pulse due to an afferent bolus of blood to the eye, the pulse amplitude is higher when the globe is less distensible.² When ocular rigidity is reduced and the globe is more distensible, the pulse amplitude is reduced as the pressure pulse is converted to a volume pulse. This is seen in high myopia, nearly perforated corneal ulcer, and staphylocoma.³ This fall in pulse amplitude is likely to be due to a reduction in ocular rigidity following trabeculectomy rather than as a consequence of a lowered IOP as pulse amplitude has been found to be unaffected when IOP is lowered in medically treated glaucomatous eyes.⁴ Furthermore, the smaller IOP rise on supine posturing following surgery also suggests a reduced ocular rigidity. In cataract patients, following surgery the magnitudes of the IOP changes on supine posturing are not reduced and the pulse amplitude changes are much less pronounced. This implies that the change in ocular rigidity is likely to be related to the drainage bleb.

It is important to note Krakau's observation that a reduced pulse amplitude does not imply a reduced blood flow; POBF does not depend on the amplitude of the pulse wave but the slope of the steepest part of the pulse volume wave. The pulse volume wave is intrinsic to the POBF calculation and is derived from the pneumotonometrically measured continuous IOP (pressure) wave and predetermined standard data on the pressure/volume relations of intact globes.⁵ POBF calculations in eyes following trabeculectomy must therefore be interpreted with care.

This study shows that trabeculectomy reduces pulse amplitude and IOP without the changes in systemic blood pressure (which can accompany β blockade). Any consequent ocular blood flow changes, however, must be evaluated by a method which does not involve the ocular rigidity factor.

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Reply

EDITOR,—I thank Yang and Hubert for the interest that they have shown in this paper and reply to the points they have raised. It

was hypothesised that the absence of the postural change in pulsatile ocular blood flow before surgery, previously reported in glaucoma and other conditions,¹ may at these high pressures be due to autoregulation. The study does not enable me to comment on whether or not autoregulation was present in the standing position before surgery.

The ocular pulse amplitude will be influenced in an individual by, among other factors, changes in scleral rigidity and ocular volume.² Changes in scleral rigidity following surgery were of obvious concern in this study. Surgery itself did not appear to increase or decrease rigidity in the cataract group in terms of the calculation of the pulsatile ocular blood flow. A reduction in scleral rigidity following trabeculectomy may, however, occur as the authors suggest. Such a reduction would reduce the amplitude of the ocular pressure pulse at a given pressure thereby underestimating the pulsatile ocular blood flow derived from it. None the less, in the standing posture following trabeculectomy pulsatile ocular blood flow was found to increase.

The authors comment that the postural change in intraocular pressure was maintained in cataract patients following surgery whereas it was reduced in the patients undergoing trabeculectomy. This difference may be explained by the large reduction in intraocular pressure following trabeculectomy and not necessarily an independent change in scleral rigidity.

In the paper by Bøsem *et al*³ there was no change in the ocular pulse volume (not amplitude) despite a reduction in intraocular pressure. Pulsatile ocular blood flow did increase. The results on pulse amplitude are not given.

I agree with the authors that even in a longitudinal study an alteration in the ocular pressure pulse amplitude does not imply a change in blood flow. This may also be affected by, for example, alterations in heart rate and intraocular pressure.⁴ It is for this reason that the pressure pulse must be converted into a volume pulse with the inevitable assumptions that this conversion requires. It would indeed be extremely useful to have a technique of clinical ocular blood flow assessment that was free of these and other problems. In the context of glaucoma, one that enabled the anterior optic nerve-head blood flow to be measured. As yet such a technique awaits development.

The ocular pressure pulse undoubtedly results from the flow of blood into the eye during cardiac systole and its interpretation may thus yield information about ocular blood flow. The results of this and other techniques of determining ocular blood flow must always be interpreted with caution and knowledge of the limitations of the methods employed.

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NOTICES

Wellcome General Overseas Travelling Research Fellowships 1994-95

The purpose of these fellowships is to allow postdoctoral scientists and medical graduates to gain further research experience by working in leading laboratories in the UK or the Republic of Ireland. Applications are invited from such workers who wish to undertake a research project in any branch of the natural or clinical sciences, which has a bearing on human or veterinary medicine, with the exception of cancer.

Applicants may be from any country outside Europe, with the exception of New Zealand and the USA for whom special schemes are available. Awards will be made on the basis of the research proposal. The research proposed should be relevant to the research interests of the candidate in his/her own country. Awards are made for one year in the first instance, although requests for an extension may be considered. Fellowships provide a stipend within the range from £13 941 to £27 869 per annum, depending on age and experience. They also include the cost of research, attendance at scientific meetings, and return travel.

Candidates must be nominated by a sponsor in the UK or the Republic of Ireland, through whom all initial inquiries should be made. A preliminary proposal should include a one or two page outline of the research proposed, the curriculum vitae of the candidate, and a letter indicating that he/she has a position to return to at the end of the fellowship. There are no special deadlines for this scheme and applications may be submitted at any time during the year.

Requests for application forms should be addressed to: Dr J M Wilkinson, The Wellcome Trust, 183 Euston Road, London NW1 2BE. Tel: 0171-611 8407.

Candidates from New Zealand and the USA should contact the Health Research Council of New Zealand, Auckland, NZ or the Burroughs Wellcome Fund, Morrisville, NC 27560, USA, respectively, for details of appropriate schemes.

Association for Research in Vision and Ophthalmology

The annual meeting of the Association for Research in Vision and Ophthalmology (ARVO) will be held on 14-19 May 1995 at the Fort Lauderdale/Broward County Convention Center, Fort Lauderdale, Florida, USA. Further details: Anne Meltzer, the ARVO Central Office, 9650 Rockville Pike, Bethesda, MD 20814-3998, USA. (Tel: (301) 571-1844; Fax: (301) 571-8311.)

4th International Symposium on Ocular Circulation and Neovascularisation

The 4th International Symposium on Ocular Circulation and neovascularisation - in memory of Professor Michaelson - will be held in Budapest, Hungary on 22-26 May 1995. Further details: Congress Bureau Motesz, Budapest, Hungary, PO Box 145, H-1443.