will increase the sensitivity of the technique which is what is required of a screening method. Once again underestimation is only in relation to autopsy findings and not to the actual clinical findings where the points are evenly distributed each side of the regression line.

(5) With regard to the third observation: this observation agrees with what is the only real punchline of the whole paper and the proposed use of the technique described.

(6) With regard to the fourth observation: this is a different interpretation of the second observation. The same conclusion as in (4) above therefore applies.

The criticism regarding application of the technique to measurement of neuroretinal rim area is unjustified and unsupported. Furthermore such a use of the technique has not been postulated. It is merely pointed out that large discs have larger neuroretinal rim areas regardless of cup/disc ratio as long as there are no abnormal features on clinical examination.

Nowhere is it proposed that this technique can be used for measuring optic disc dimensions accurately. On the contrary, it is clearly stated to be a quick and easy clinical method of estimating (defined by the *Oxford English Dictionary* as 'an approximate judgment') disc size which may be of help in patients with large physiological cups or asymmetric cups.

This letter has correctly stated the formulas for linear magnification that can be found in any textbook of physiological optics. It has not come to any conclusions about that effect nor does it contradict the purpose for which the technique was described and is in fact in agreement with the conclusions in the paper.

To further illustrate how little the variables mentioned will actually alter the use of this technique the following examples have been calculated (Colin Fowler, Department of Vision Sciences, Aston University, Birmingham, personal communication).

LENS MAGNIFICATION

Assume the Volk 90 D lens to be a 'thin' lens, place at 20 mm from the eye.

Using the simplified schematic eye, standard power +60 D for emmetropia, refractive index 1·333, axial length 22·22 mm. For axial ametropia, change the length of the eye, for refractive ametropes change power.

(1) Axial hypermetropia

Assuming an axial length of 20 mm, equivalent to +6·5 D hypermetropia. Magnification produced by 90 D lens: 0·7×.

(2) Axial myopia

Assuming an axial length of 24 mm, equivalent to −4·46 D myopia. Magnification produced by 90 D lens: 0·64×.

(3) Refractive myopia

Assuming power of eye is +65 D, giving −5D myopia. Magnification with 90 D lens: 0·7×.

(4) Refractive hypermetropia

Assuming power of eye is +55 D, giving +5D hypermetropia. Magnification with 90 D lens: 0·63×.

(5) Emmetrope

Magnification produced by volk lens 0·67× if power of eye remains at 60 D.

CHANGE IN POSITION OF LENS

In the case of an emmetrope magnification will be constant.

In ametropia, consider the following example of a 24 mm axial myope, as in (2) earlier:

Position of 90 D lens from eye:	20	30	40 (mm)
Magnification:	0·64	0·67	0·71

It can be seen from these examples that the range of magnification in emmetropia is much less than suggested in Barr's letter. Also that to alter significantly the image size the lens would have to be moved a considerable distance and this does not occur in clinical practice.

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Laser flare intensity in diabetics

EDITOR,—We congratulate Ino-ue and colleagues on their article recently published in the *BJO*.[1] Many of their findings agree with our previously published article on this subject.[2] The controlled groups in our study[2] had similar flare values to those reported[1] and we conclude similarly that patients with more severe proliferative or regressed proliferative retinopathy have greater flare values than those without retinopathy. However, our results differ since in our study diabetics, even without diabetic retinopathy, had significantly greater flare values than normal controls, as did all patients with background diabetic retinopathy. Ino-ue *et al* find no difference between diabetics without retinopathy and normal subjects at any stage and a significant difference between background retinopathy and normals only after five decades.

Our findings of increased breakdown of the blood-aqueous barrier in diabetes without coexistent retinopathy, or preceding its development, is borne out by two other studies. Fluorescein angiographic changes in iris vasculature occur before breakdown of the blood-retinal barrier.[3] In addition, the well known association of uveitis with diabetes consistently precedes the development of retinopathy.[4]

No statement on control of diabetes is made in the patient data analyses reported.[1] Hypoglycaemic therapy may have had a significant effect on these results, since initiation of treatment is known to affect and delay the development of retinopathy and reduce blood-retinal barrier dysfunction in animals.[5] It is unknown whether this can be extrapolated to humans. Use of insulin in 25% of our patients with no retinopathy[2] may have normalised blood-retinal barrier dysfunction and hence prevented or delayed the appearance of diabetic retinopathy, but did not affect blood-aqueous barrier dysfunction to the same extent. This may account for a higher flare value in diabetics, even without retinopathy, compared with normals. This merits consideration when all diabetics undergo anterior segment surgical procedures or laser photocoagulation.[6]

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Reply

EDITOR,—The laser flare intensity is a quantitative indicator in the evaluation of diabetic dysfunction of the blood-ocular barrier without tracers. The laser flare intensity correlated with the degree of retinopathy.[1–3] However, a recent report[4] has indicated a significantly higher flare intensity in eyes without retinopathy. Fluorescein iris angiography[5] and fluorophotometry[6] have demonstrated that the blood-aqueous barrier dysfunction precedes breakdown of the blood-retinal barrier. Moriarty *et al*[4] have discussed a greater tendency of insulin to produce blood-aqueous barrier dysfunction. In our study most of patients without retinopathy was non-insulin dependent. The effect of insulin on the blood-aqueous barrier and the laser flare intensity should be evaluated. Whereas, a tracer of fluorescein has a small molecular size, the laser flare intensity reflects the larger molecular size of proteins. Leakage of various materials depends on the degree of the dysfunction of barrier. Even if fluorescein leakage has been observed, the laser flare intensity may not be elevated. In young diabetic patients without retinopathy, fluorescein iris angiography revealed a higher incidence of dye leakage than normal controls, but no difference in the laser intensity was observed.[7] Shah *et al*[8] has suggested that fluorophotometry and laser flare cell metry may measure different variables of the blood-aqueous barrier. Blood-aqueous barrier function at the early stage of diabetes should be simultaneously evaluated with iris angiography, fluorophotometry, and laser flare cell metry.

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Relation between blood glucose control over 3 months and colour discrimination in insulin dependent diabetic patients without retinopathy

EDITOR,—Visual pathway function may be abnormal in patients with insulin dependent diabetes (IDDM) with angiographically normal retinas.[1] This could be a result of reversible changes in visual pathway function, dependent upon blood glucose control. When blood glucose was fixed for several hours at predetermined levels (2·5–14 mmol/l) using a hyperinsulinaemic glucose clamp, no difference in colour discrimination in non-retinopathic IDDM patients was found.[2] We have therefore conducted a preliminary study to investigate the relation between changes in intermediate term (over 3 months) blood glucose control and colour discrimination.

Eleven clinically non-complicated IDDM patients of median age 35 (interquartile range 27–37) years and with diabetes duration 8 (6–16) years, without evidence of retinopathy on single (45°) field fundus photography participated in the study. Most (8/11) had also had fluorescein angiography (which was normal) within 1 year. None was receiving medication other than insulin. They were assessed at 0 and 3 months, with measurement of glycated haemoglobin (HbA_1, reference range 5·5–8·2%) (Corning Medical, Corning, NY, USA). Colour discrimination was assessed by means of the Farnsworth-Munsell 100-Hue test as described previously.[1] Neither patient nor examiner was aware of the patient's HbA_1 at colour vision assessment.

Patients were divided into two groups on the basis of 3 month HbA_1 results: in the first group (n=6) diabetes control had improved over 3 months (HbA_1 11·5% (9·8–12·6%) to 9·4% (7·6–11·0%), $p<0·05$), in the second group (n=5) diabetes control had deteriorated over 3 months (HbA_1 9·8% (8·9–10·4%) to 11·2% (10·4–12·9%), $p<0·05$). Baseline age, diabetes duration, and 100-Hue error score were similar in the two groups.

Comparisons between groups were by Mann-Whitney U test and within group comparisons at 0 and 3 months by Wilcoxon's signed rank test. The 100-Hue error scores were normalised by square root transformation[3] and were compared by paired *t* test.

In the group in which glycaemic control improved, colour discrimination improved significantly over 3 months: mean (SD) square root 100-Hue error score 7·5 (2·7) to 5·9 (2·2) at 3 months, $p<0·05$. By contrast, when glycaemic control worsened, colour discrimination worsened 6·6 (4·3) to 8·3 (3·9) at 3 months, $p<0·05$. No patient had a specific axis of colour discrimination loss.

These preliminary data support an association between colour discrimination and intermediate term glycaemic control and confirm prospectively the correlation between glycated haemoglobin and colour discrimination observed in the cross sectional study of Muntoni *et al.*[4] That colour discrimination changes reflect changes in visual pathway function rather than alterations in concentration or cerebral function is supported by the observation that other tests of visual pathway function have improved with better glycaemic control.[5 6]

The mechanism of the relation between glycaemic control and changes in visual function is unclear. It may be that excess or deficiency of some product of intermediary metabolism affects cone pathway function, or that the function of some key visual pathway enzyme is affected by abnormal glycosylation. It may be that hyperglycaemia induced alterations in retinal blood flow affect delivery of oxygen and nutrients to cells of the visual pathways. These issues remain unresolved.

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1 Hardy KJ, Lipton J, Scase MO, Foster DH, Scarpello JHB. Detection of colour vision abnormalities in uncomplicated type 1 diabetic patients with angiographically normal retinas. *Br J Ophthalmol* 1992; **76:** 461–4.
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Mapstone's hypothesis confirmed

EDITOR,—In 1979, Mapstone[1] suggested that angle closure could begin with iridocorneal apposition to the angle wall at the level of Schwalbe's line before the development of iridotrabecular contact. He presented clinical evidence obtained at the slit-lamp to show that, since the peripheral curvatures of cornea and iris are different, there is a finite distance between the trabecular meshwork and iris root anterior to the iris insertion. In this situation, intraocular pressure (IOP) would remain normal until the aqueous humour present in the space between the iris root and trabecular meshwork had exited the eye. Increasing aqueous humour volume in the posterior chamber and increasing iris apposition to the meshwork as the aqueous between the meshwork and iris root diminished, would then lead to angle closure glaucoma with elevated IOP.

Using high frequency, high resolution, anterior segment ultrasound biomicroscopy (UBM)[2 3] we have been able to show that the iris configuration hypothesised by Mapstone does occur. An 82-year-old man was referred with a diagnosis of closed angles and normal IOP. Gonioscopy revealed slit to grade I angles in bright light which, when the room was darkened and a small, square slit beam used to illuminate the angle, became appositionally closed.

UBM imaging revealed iridolenticular apposition and a convex iris configuration diagnostic of pupillary block (Fig 1). The ciliary body (*) was positioned somewhat anteriorly, suggesting a component of plateau iris.[4 5] A triangular space was present at the angle recess (arrow), which narrowed to the point at which the iris was apposed to the external wall of the angle.

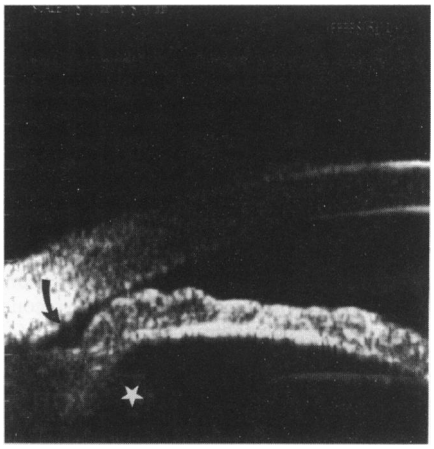

Figure 1 Appositional angle closure (see text for details).

Mapstone[1] had suggested that closure of the angle recess and obliteration of the remaining space would occur after intraocular pressure rose sufficiently so as to push the iris against the trabecular meshwork. The image depicted suggests the possibility of an alternative sequence of events in the initiation of angle closure glaucoma. It is possible that, once iridocorneal apposition is of sufficient duration (for example, prolonged exposure in a darkened room), aqueous present within the angle recess continues to exit through the trabecular meshwork, but cannot be replaced by aqueous from the anterior chamber because of the apposition. As a result, the iris moves naturally to the trabecular meshwork as the volume of aqueous decreases, and when this apposition is increased, then intraocular pressure rises.

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NOTICES

British College of Optometrists

The centenary conference of the British College of Optometrists will be held at Churchill College, Cambridge on 5–8 April 1995. Further details: BCO Conference Secretariat, Conference Contact, 42 Devonshire Road, Cambridge CB1 2BL. (Tel: 01223 323437; Fax: 01223 460396.)