Estimation of optic disc size

EDITOR,—In a recent paper,1 Ruben described a technique for estimating the real vertical optic disc diameter using the slit beam height of a Haag-Streit slit-lamp with a +90 D condensing lens. It is concluded that a slit beam height of greater than 1.4 mm indicates a vertical disc diameter of greater than 1.88 mm.

Linear magnification is the ratio of image height (h') (for example, slit beam height) and object height (h) (for example, real vertical disc diameter). Where the total linear magnification of the system is m (comprising the optical elements of the patient’s eye and the condensing lens), the image height in emmetropia may be calculated according to:

\[ h' = m \cdot \frac{F_0}{P} \]

where \( F_0 \) is the equivalent power of the eye and \( P \) the power of the condensing lens. The minus sign indicates an inverted image. The equivalent power of the eye in emmetropia varies between +53 D and +64 D2,3 and is normally distributed. The total linear magnification in emmetropia therefore varies between -0.59 and -0.71 with the +90 D lens.

The relation between real vertical disc diameter and slit beam height in emmetropia is shown in Figure 1. Ruben’s regression line (Y = 1.17 X + 0.24) is superimposed.

In ametropia, linear magnification must also take into account the ocular refraction (K), the position of the first principal plane of the eye (e) and the working distance of the condensing lens (q). The image height may be calculated according to:

\[ h' = m \cdot \frac{F_0 + K}{P - K\left(1 - (P\varepsilon + Pq)\right)} \]

By considering the normal distributions of the ocular elements, the total linear magnification in ametropia (–5.00 D to +5.00 D) varies between -0.55 and -0.74 (Fig 1).

The following observations can be made with regard to Ruben’s method:

1. The regression line, \( Y = 1.17 X + 0.24 \), is lower than one would expect from theoretical considerations. This may be due to error in image height estimation (SD 0.25 mm) and error due to Littmann’s method.

2. The vertical disc diameter of most optic discs is underestimated, the effect being more pronounced with larger discs (Fig 2) and less pronounced overall in hypermetropia (Fig 2C).

3. In agreement with Ruben, should the slit beam height be greater than 1.4 mm, the vertical disc diameter is almost certain to be greater than 1.88 mm.

4. The vertical disc diameter, when measured at the dissecting microscope, varies between 1.4 mm and 2.4 mm (mean 1.88 mm), is normally distributed4 and not correlated with low ametropia.5 Fifty per cent of vertical optic disc diameters will therefore be greater than 1.88 mm. The shaded area in Figure 1 represents those optic discs estimated at less than, but actually greater than, 1.88 mm, comprising approximately half of such optic discs. Therefore, when the image height is between 1.2 mm and 1.4 mm, a significant number of optic discs will be greater than 1.88 mm in diameter.

The optimal quantitative optic disc predictor in glaucoma is the neuroretinal rim area.6 Ruben’s method cannot be adapted to perform this measurement: magnification due to the optical components of the patient’s eye is not considered, it is not possible to estimate the real dimension of cups whose image with

\[ m = -\left[F_0 + K\left(1 - (P\varepsilon + Pq)\right)\right] \]

because a high power condensing lens is being used (90 D) and the variation in e and q is measured in metres the difference is in fact quite small. Furthermore, the variation in magnification will depend on whether the emmetropia is axial or refractive in nature (see below). It is clearly stated in the original paper that refractive error will influence the image size which is why the method is confined to low emmetropia and why the technique cannot be used as an accurate measure of dimensions. However, should the refraction, keratometry, and axial length of the eye be known then a more exact measurement could be made.

(3) With regard to the first observation the regression line is different because it is based on clinical methods and not purely on theoretical grounds. It must be stated that all optic discs studied in this paper were clinically within normal limits and the conversion factor obtained relates to the image size estimated using a planimetric device and not that found at autopsy.

(4) With regard to the second observation: because this technique is designed as an aid to clinical assessment and not as a method for exact measurement, underestimation of size.
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.

(3) 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.

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 scheme, 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: 0.7x.

(2) **Axial myopia**
Assuming an axial length of 24 mm, equivalent to -4.46 D myopia. Magnification produced by 90 D lens: 0.64x.

(3) **Refractive myopia**
Assuming power of eye = +65 D, giving -5 D myopia. Magnification with 90 D lens: 0.7x.

(4) **Refractive hypermetropia**
Assuming power of eye = +55 D, giving +5 D hypermetropia. Magnification with 90 D lens: 0.63x.

(5) ** Emmetropia**
Magnification produced by Volk lens 0.67x 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 appearance of the lens would have to be moved a considerable distance and this does not occur in clinical practice.

**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 previous observations on this subject.2 The controlled groups in our study2 had similar flare values to those reported1 and we conclude similarly that patients with more severe proliferative or nascent 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. found 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 between diabetes and cardiovascular disease 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 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 retinopathy2 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

**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 report7 has indicated a significantly higher flare intensity in eyes without retinopathy. Fluorescein angiography3 and fluorophotometry6 have demonstrated that the blood-aqueous barrier dysfunction precedes breakdown of the blood-retinal barrier. Moriarty et al.8 have discussed a greater tendency of insulin to produce blood-aqueous barrier dysfunction. In our study most 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 angiography revealed a higher incidence of dye leakage than normal controls, but no difference in the laser intensity was observed.9 Shah et al.10 has suggested that fluorophotometry and laser flare cellmetry could be used to evaluate the 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 cellmetry.

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