PHAKIC
 AVERAGE VALUE : 21.72 mm SD : 0.09
 AXIAL LENGTH : 21.67 mm
 1532m/s 1641m/s 1532m/s
 AC = 2.76 L = 4.55 V = 14.36

Fig. 1. Sound speed of vitreous internal set at 1532 m/s.

example). Fig. 2 shows the same scan now with the calculation for the vitreous cavity set at 987 m/s. The ACD and lens thickness are still the same but the vitreous cavity is now measured at 9.25 mm (obviously incorrect as in this eye the normal vitreous was present). If this eye were filled with silicone oil the correct vitreous interval would have been calculated. In Murray *et al.*'s example illustrated in fig. 1 of their article,¹ simply recalculating the vitreous length by dividing it by 1550 to get back the length of time it took the sound wave to traverse it and then multiplying it by 987 we get 14.84 mm (or 15.02 mm if we use 1532 m/s), which is very close to what they got after the oil was removed. However, there is an unexplained discrepancy in their measurements of the ACD and lens thickness in their example. These should be the same before and after silicone oil removal. In their pre-operative scan (fig. 1a) the ACD is 2.06 mm while it is 4.13 mm in their post-operative scan (fig. 1b). Similarly the lens is 6.15 mm in their pre-operative scan and 3.91 mm in their post-operative scan. These discrepancies are confirmed in their table 4, which shows large variations in what should be static measurements of ACD and LT. Unless the authors can explain these different measurements in the same eyes, doubt is cast on all measurements used in the paper. Furthermore, because of the large variations in the total length of ACD

PHAKIC
 AVERAGE VALUE : 16.59 mm SD : 0.08
 AXIAL LENGTH : 16.56 mm
 1532m/s 1641m/s 987 m/s
 AC = 2.76 L = 4.55 V = 9.25

Fig. 2. Sound speed of vitreous internal set at 987 m/s.

plus LT in these four patients (from 0.13 to 2.65 mm, average 0.94 mm), calculation of any ratio for the vitreous cannot be reliable. Even if the measurement of the vitreous cavity were accurate the authors have the potential for an average 4 D error in the intraocular lens (IOL) calculation in the anterior segment alone.

The authors also calculate IOL power using the two sets of measurements derived from their method. As the conversion factor is derived from the same set of figures, i.e. the apparent change in axial length following oil removal, the two sets of figures are dependent on each other. Therefore to point to the similarity of the results of IOL power calculation is no proof of the accuracy of their work.

Moving to the issue of IOL calculation in these cases, I believe it is important to take account of whether the oil is to be removed or not. If it is to be left behind then IOL calculation is even more difficult as the oil has a refractive index very different from vitreous and therefore the IOL power must be modified to take account of this. Holladay² suggests that one may need to add up to 3 D to the IOL power in such cases. Additionally the contour of the back surface of the IOL affects what refractive effect the IOL/oil interface will have, so that the calculation will change for different lenses.

Patients who require IOL power calculation in an eye with silicone oil in the vitreous cavity should have their eye measured by an experienced biometrist using a machine in which sound speeds can be varied and the gates marking the intraocular anatomy moved. The biometrist must be able to identify each acoustic interface and adjust the gates manually if required. Even then the surgeon must be aware that a successful refractive outcome may well entail lens exchange in the first week following surgery and must be prepared for this. Unfortunately not all the maculae in such patients who have needed silicone oil to reposition their retinae are capable of good visual acuity.

Biometry is set to assume its rightful place in cataract surgery. This group of patients present special difficulties which push current technology and expertise to its limit.

References

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Sir,

We thank Mr Bolger for his comments on our paper.¹ The aim of the study was to determine whether, in the silicone oil-filled eye (viscosity 1300 centistokes), measured axial length (AL) varied from true AL by a constant factor. We found a remarkably consistent variation in this ratio, ranging from 0.70 to 0.73 in seven eyes (mean 0.71, SD 0.01). This could enable eyes undergoing combined phacoemulsification of cataract, lens implantation and removal of silicone oil to have desired lens power determined, since true AL can be estimated by simply multiplying measured AL of the silicone oil-filled eye by 0.71.

We accept that there are limitations with this method, not least of which is the fact that true AL may be underestimated in very long eyes. We are of the opinion, however, that possible errors are likely to be less than those incurred by using measurements based on the fellow eye, or by implanting an 'average' power or 'best guess' intraocular lens, as is common practice at present. Table 5 in our paper¹ shows that true AL and AL calculated using the conversion factor of 0.71 are quite similar, differences ranging from -0.61 to +0.39 mm, with mean percentage error being only -0.57%.

We appreciate the concern expressed about the differences in anterior chamber depth (ACD) and lens thickness (LT) in four patients prior to and following the removal of silicone oil. However, measurements were taken several weeks apart and this may account for the variation in ACD and LT in these four patients. There is inevitably inter-test variability due to variation in biometry parameters. This is especially true of ACD measurements, which can vary with the extent of corneal compression from forward or backward movement of the patient. ACD was found to have increased in three of four patients following removal of silicone oil. Backward displacement of the lens-iris diaphragm with secondary deepening of the anterior chamber in the vitrectomised eye may have contributed to this increase. In addition, rapid-onset nuclear sclerosis is a well-recognised complication of silicone oil use.² This may well account for the increase in LT in three of four patients. This, in combination with intra- and inter-observer variability, most likely explains the difference in ACD and LT prior to and following removal of silicone oil and further validates the use of an overall constant which encompasses ACD, LT and vitreous cavity depth (VCD) measurements. Mr Bolger

demonstrates identical measurements in ACD and LT in one patient on two separate scans. It would interest us to know the time between scans and whether or not vitrectomy had been done between measurements.

Whilst we agree that the ideal scenario would be to have AL measured by an experienced biometrist using a machine in which sound speeds can be varied, Mr Bolger has pointed out that even in the most experienced hands lens exchange may still be necessary. In addition, the speed of sound through silicone oil of viscosity 1300 centistokes is unknown (987 m/s is actually sound speed through silicone oil of viscosity 1000 centistokes³). In any event, not all units are equipped with biometry machines with which ACD, LT and VCD can be measured individually and in which sound speeds can be varied. Instead, many older machines give one measurement of AL which incorporates ACD, LT and VCD and/or have the velocities preset into the instrument.

The conversion factor of 0.71 used to estimate true AL allows estimation of desired intraocular lens power which may otherwise not be possible. Prospective evaluation of this conversion factor will be the subject of a future report.

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Desirée Murray ✉

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Sir,

The propriety of a domiciliary nurse for first dressings after cataract surgery (Willins *et al.*, *Eye* 1999; 13: 336–8) is perhaps not so much in question, as the actual instructions given to a well-trained specialist nurse. The empirical use of 2-hourly steroid or acetazolamide for a hazy cornea, a florid uveitis, or undue pain is surely poor practice.

Should not *all* such patients be referred immediately to hospital for slit-lamp examination? How else can intense

anterior chamber activity with or without exudate possibly indicating early endophthalmitis, be excluded?

In the last four and a half years at Brecon, in a multi-purpose theatre without phaco facilities, 225 extra-capsular procedures have been carried out. All but a few (social reasons) have been day case procedures in this very rural area. There has been a zero incidence of intraocular infection. No antibiotics have been used in perfusion fluids.

A hypopyon incidence of more than 1 per 100 procedures as in the paper by Willins *et al.* poses the question, 'Why?'

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Sir,

We thank Mr Karseras for his interest in our paper and his comments.

We believe that an experienced ophthalmic nurse is able to make a sensible determination as to whether the eye is more inflamed than normal, and we fully support them using their discretion in applying more intensive steroids for a few days. We believe that this has been normal practice for many years amongst ophthalmologists, and we are simply allowing our nursing colleagues to exercise the same discretion.

The signs and symptoms of endophthalmitis do not begin to manifest until day three or four post-operatively and therefore any such increased inflammation at day one cannot be taken as an indication of impending endophthalmitis.

We would agree entirely that the key to early detection and treatment of post-operative endophthalmitis is patient awareness of the condition and of the importance of reporting promptly. The close contact of our nurses with patients before, during and after surgery reinforces this.

The two hypopyons in our series were in cases of sterile uveitis which responded to intensive steroid treatment and were not cases of endophthalmitis.

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Sir,

In his excellent review of the management of monocular congenital cataracts¹ Scott Lambert refers to pupil

trap as a complication which can be avoided by careful attention to surgical technique and lens design.

Having seen a number of cases of pupil trap occurring 1 or 2 weeks after paediatric cataract surgery we hypothesised that eye rubbing could be the cause, particularly at night when pupil dilatation increases the possibility that the iris could be 'tyre-levered' behind the lenticulus when endocapsular placement has not been possible. During the last 4 years we have prescribed pilocarpine 1% morning and evening for 2 weeks post-operatively and have arranged for children to wear an eye shield, especially at night during this period. During this time, we have seen no cases of pupil trap.

The validation of our hypothesis would require a clinical trial; however, the combination of pupillary constriction and prevention of eye rubbing, particularly at night, appears, in our practice at least, to have eliminated the problem of pupil trap occurring during the weeks following cataract surgery in children.

Reference

1. Lambert SR. Management of monocular congenital cataracts. *Eye* 1999;13:474–9.

Gordon N. Dutton ✉

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Sir,

I agree with Drs Dutton and Fahad that pupillary capture can be a problem in children if both haptics are not placed in the capsular bag. Careful attention to wound closure in a child is also critical because of the possibility of eye rubbing or ocular trauma.

I also recommend an eye shield be worn at night, but do not favour the use of miotics. In fact, I typically prescribe cyclopentolate 1% to be used once a day in an attempt to maintain the normal function of the pupil. I have not had a child develop pupillary capture over many years using this regimen.

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Erratum

Eye 1999;13:391–2

The name of Richard A Harrad, first author of the letter regarding the paper by Inkster *et al.*, was accidentally omitted.