References

1. Claoué C, Foss A, Daniel R, Cooling B. Why are new patients coming to the Eye Clinic? An analysis of the relative frequencies of ophthalmic disease amongst new patients attending hospital eye clinics in two separate locations. Eye 1997;11:865–68.
2. Sheldrick JH, Vernon SA, Wilson A, Reid SJ. Demand incidence and episode rates of ophthalmic disease in a defined urban population. BMJ 1992;305:933–6.

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Sir,

We read with interest the pilot study by Tufail and co-workers[1] on the role of prophylactic argon laser retinopexy prior to the removal of silicone oil.

At the Taunton & Somerset Hospital we compared patients who had silicone oil removal (SOR) from January 1994 onwards (all of whom had prophylactic 360° peripheral indirect laser) with a similar number of patients prior to this date who had SOR without prophylactic laser, as was the policy then. Details are shown in Table 1.

Prophylactic 360° peripheral laser prior to SOR significantly reduced the rate of retinal re-detachment in our study, as shown in Table 2. This was comparable to the data published by Tufail *et al.*

Though the sample sizes in both studies were small it would be reasonable to infer that prophylactic 360° laser prior to SOR may have a role in reducing the incidence of retinal re-detachment, and we would recommend it for all patients requiring silicone oil removal.

Reference

1. Tufail A, Schwartz SD, Gregor ZJ. Prophylactic argon laser retinopexy prior to removal of silicone oil: a pilot study. Eye 1997;11:328–30.

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Sir,

Rundle and colleagues recently described a family presenting with iris degeneration, associated with sensorineural deafness/tinnitus and glaucoma, which appeared to be inherited as an autosomal dominant trait.[1] They discussed a number of conditions in relation to this family including the mesodermal dysgeneses, aniridia, the irido-corneal endothelial syndromes, iridoschisis and Waardenburg's syndrome. I would like to suggest a number of other important conditions that may underlie the features described in their family.

Table 1. *Study details*

	Study group (with laser)	Control group (without laser)
Period of study	Jan. 1994–Feb. 1997	Jan. 1990–Dec. 1993
No. of eyes	9	9
Follow-up (months)		
Mean	21.7	24.1
Range	7–31	1–70

Table 2. *Retinal detachments after silicone oil removal*

	Study group (with laser)	Control group (without laser)
Study at Taunton	1 (11.2%)	4 (44.44%)
Tufail *et al.*[1]	6.7%	25%

The condition iridogoniodysgenesis anomaly (IGDA) shows a number of striking similarities to the described family. It is an autosomal dominant condition, characterised by iris hypoplasia, goniodysgenesis and glaucoma. The typical iris appearance is that of a slate grey or chocolate-brown iris due to iris pigment epithelium showing through a hypoplastic iris stroma. The iris sphincter stands out strikingly against this featureless background. The iris abnormalities typically predate the development of glaucoma and have therefore been used clinically to predict those at risk of glaucoma.[2] Iris stromal atrophy and iris changes predating any rise in intraocular pressure are both also features of the family described by Rundle *et al.* IGDA is believed to result from the aberrant migration or terminal induction of the neural crest cells involved in the formation of the anterior segment of the eye – a pathology also suggested for the described family. It has recently been mapped to chromosome 6p25.[3]

Iridogoniodysgenesis syndrome is an autosomal dominant condition similar to IGDA, but in addition to the ocular features, non-ocular features exist such as maxillary hypoplasia and dental anomalies. It has been mapped to chromosome 4q25 and may therefore be allelic with Rieger's syndrome.[4]

The SHORT syndrome is characterised by short stature, hyperextensibility of joints and/or hernia, ocular depression, Rieger's anomaly and teething delay.[5] Two patients with the SHORT syndrome have been described who, in addition to Rieger's anomaly, suffered from glaucoma and sensorineural deafness.[5,6] The genetic basis of the SHORT syndrome is unknown. It has been suggested that, as in Rundle *et al.*'s family, it is due to an autosomal dominant gene with a variable expression.[7] It is possible, therefore, that their family may have a mild form of this syndrome.

Iris malformation, glaucoma and sensorineural deafness, amongst other defects, have also been reported in two children of a consanguineous couple. No underlying genetic defect was, however, ascribed to this family.[8]

I would suggest, therefore, that there are a number of very important conditions that should be considered in relation to the family reported by Rundle and colleagues in addition to those that they discuss in their paper. Consideration of these conditions may help in their attempt to determine the underlying genetic defect in this family.

References

1. Rundle P, Lotery AJ, Archer DB, McGinnity FG. Familial deafness associated with iris degeneration and glaucoma. Eye 1997;11:476–8.
2. Martin JP, Zorab EC. Familial glaucoma in nine generations of a South Hampshire family. Br J Ophthalmol 1974;58:536–42.
3. Mears AJ, Mirzayans F, Gould DB, Pearce WG, Walter MA. Autosomal dominant iridogoniodysgenesis anomaly maps to 6p25. Am J Hum Genet 1996;59:1321–7.
4. Héon E, Sheth BP, Kalenak JW, Sunden SLF, Streb LM, Taylor CM, *et al.* Linkage of autosomal dominant iris hypoplasia to the region of the Rieger syndrome locus (4q25). Hum Mol Genet 1995;4:1435–9.
5. Brodsky MC, Whiteside-Michel J, Merin LM. Rieger anomaly and congenital glaucoma in the SHORT syndrome. Arch Ophthalmol 1996;114:1146–7.

6. Schwingshandl J, Mache CJ, Rath K, Borkenstein MH. SHORT syndrome and insulin resistance. Am J Med Genet 1993;47:907–9.
7. Sorge G, Ruggieri M, Polizzi A, Scuderi A, Di Pietro M. SHORT syndrome: a new case with probable autosomal dominant inheritance. Am J Med Genet 1996;61:178–81.
8. de Chadaremian JP, Kaplan P, Vekunas M, Kaplan BS. Aniridia/glaucoma and Wilms tumour in a sibship with renal tubular acidosis and sensorineural deafness. Am J Hum Genet 1987;Suppl 3:323–8.

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Sir,

We welcome the comments made by Mr Adam Booth regarding the family that we recently described.[1] We became aware of the autosomal dominant iridogoniodysgeneses only after submission of our own manuscript, hence their omission from our differential diagnosis. As suggested by Mr Booth, we hope to use the known loci for these conditions as a starting point for our own investigations.

Reference

1. Rundle P, Lotery A, Archer DB, McGinnity FG. Familial deafness associated with iris deneration and glaucoma. Eye 1997;11:476–8.

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Sir,

We were interested to read the paper by Zambarakji *et al.* on the interesting complication of anterior capsular shrinkage following phacoemulsification surgery.[1] We would argue with their conclusion that silicone folding lenses are at greater risk of this complication, as this is neither supported by the literature nor proven by their data.

The capsule contraction syndrome (CCS) is an incompletely understood phenomenon, but is likely to involve many factors besides optic material. Those implicated in the literature to date include: (1) factors relating to the patient (as summarised by Zambarakji *et al.*); (2) factors relating to surgical technique, including degree of capsular polishing[2] and capsulorhexis size;[3] and (3) factors relating to intraocular lens (IOL) design including both optic materials[1] and haptic materials,[3] as well as design of haptic (loop vs plate).[4]

Whilst the authors dealt with the patient risk factors by excluding subjects with ocular co-morbidity, they failed to deal adequately with the surgical risk factors. Two surgeons performed the surgeries and the subjects were not randomly allocated to lens type or surgeon. It is likely that the two surgeons involved have slightly different techniques and differ in their preference for IOL design. When investigating a phenomenon such as the interaction of the CCS with so many putative risk factors there is no substitute for randomisation in order to eliminate conscious or unconscious bias. Ideally randomisation of IOL should occur after cortical aspiration is completed in order to ensure that IOL design does not influence the preceding steps of surgical technique. A further step in the elimination of bias would have been the use of an objective system of capsulorhexis measurement as used by Gonvers *et al.*,[4] or masking the observer to the date of surgery and previous measurements.

The importance of randomisation in a prospective study of this sort is not mere academic pedantry. The two groups in a study such as this must be identical apart from the variable under investigation. In this paper they clearly were not identical. A striking difference is that the mean capsulorhexis sizes in the two groups were unequal at 20.43 mm^2 in the PMMA group and 16.05 mm^2 in the silicone group on day 1. It is not surprising that the silicone IOL group had smaller capsulorhexes at 6 weeks when they started off smaller!

The data presented in the paper could easily be used to argue that a small capsulorhexis is more liable to the CCS. There is a sound pathophysiological basis for this theory in that a 5.5 mm capsulectomy removes twice as many lens epithelial cells as a 4.0 mm capsulectomy;[5] contact between lens epithelial cells and an optic causes proliferation and metaplasia;[6] and finally the centripetal force required to close a smaller opening is less than for a larger opening.

A further potential reason for the apparent shrinkage of the capsulorhexis in the silicone IOL group is the nature of the haptic on the particular IOL chosen. Polypropylene haptics are recognised to provide less resistance to deforming forces and to be more prone to decentration.[7] If one were to suspect that the optic material influences the development of the CCS then a more rational comparison would be between a PMMA lens and a silicone lens with PMMA haptics. We appreciate that these lenses may not have been available at the time of the study. A recent study by Gonvers *et al.*[4] found that capsular shrinkage was not statistically significantly different between one-piece PMMA lenses and silicone lenses with PMMA haptics.

In conclusion we feel that Zambarakji's study cannot be considered strong enough evidence to justify their condemnation of silicone lenses. It is possible that silicone lenses are a factor, but without meticulous attention to experimental methodology it is premature to make that judgement. We thank Zambarakji and colleagues for their thought-provoking study of this increasingly common problem. None of the authors of this letter have any proprietary interest in any form of lens manufacture.

References

1. Zambarakji HJ, Rauz S, Reynolds A, Joshi N, Simcock PR, Kinnear PE. Capsulorhexis phymosis following uncomplicated phacoemulsification surgery. Eye 1997;11:635–8.
2. Joo CK, Shin JA, Kim JH. Capsular opening contraction after continuous curvilinear capsulorhexis and intraocular lens implantation. J Cataract Refract Surg 1996;22:585–90.
3. Hansen SO, Crandall AS, Olson RJ. Progressive constriction of the anterior capsular opening following intact capsulorhexis. J Cataract Refract Surg 1993;19:77–82.
4. Gonvers M, Sickenberg M, van Melle G. Change in capsulorhexis size after implantation of three types of intraocular lens. J Cataract Refract Surg 1997;23:231–8.
5. Davison JA. Capsule contraction syndrome. J Cataract Refract Surg 1993;19:582–9.
6. Hara T, Hara T, Kojima M, *et al.* Specular microscopy of the anterior lens capsule after endocapsular lens implantation. J Cataract Refract Surg 1988;14:533–40.
7. Apple DJ, Soloman KD, Tetz MR, *et al.* Posterior capsule opacification. Surv Ophthalmol 1992;37:73–116.

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Sir,

We thank Wong *et al.* for their comments on our paper[1] discussing capsular phymosis following uncomplicated phacoemulsification surgery.

Although the starting capsular diameters in our study were smaller in the foldable intraocular lens (IOL) group, we do not consider that the observed greater reduction in anterior capsular areas is solely due to the initial capsular diameters. We found no significant difference between the