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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

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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.

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