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Sir,

We have very mixed feelings about The National Survey of Local Anaesthesia for Ocular Surgery: Early Report from the Royal College of Ophthalmologists.[1]

We are pleased that the survey was able to show (as have others[2]) that 'routine' pre-operative investigations before local anaesthesia are unnecessary, and feel that the Royal College of Ophthalmologists should now revise their original guidelines[3] to reflect this.

We have major reservations about methodology, which is seriously flawed from at least two aspects:

1. The lack of standardised definition of method of anaesthesia, in particular failing to define peribulbar and retrobulbar injections, will have caused confusion. A peribulbar injection is defined as a deliberate extraconal injection[4] and a retrobulbar as a deliberate intraconal injection.[5] We are aware that many doctors who administer ophthalmic local anaesthesia do not follow these definitions, and that much of the outcome data comparing these two techniques is therefore suspect.
2. There is a large variation in the incidence of adverse effects between the two phases of the survey. The survey reports the incidence of systemic adverse events at 0.9% (0.1% severe) in the first week when all cases were to be reported and 0.19% (0.06% severe) over the remainder of the 3 months when only adverse events were to be reported. We feel that this can only be explained on the basis of under-reporting and that the data from the second 3 month period cannot be relied on.

We are most concerned about the results of the survey. If the adverse event data from the first week are accurate then local anaesthesia for ocular surgery as currently practised in the UK is an unsafe procedure. In the first week 3.5% of patients had either an 'orbital' (2.6%) or a 'systemic' (0.9%) adverse effect; 0.28% of patients had a severe adverse event. Taken at face value this 3.5% risk makes local anaesthesia the single highest risk to the patient's health or sight, comparable to the risk of vitreous loss or endophthalmitis, in cataract surgery.[6] A risk of this magnitude must be disclosed and discussed with the patient, and it is our belief that no sensible patient would choose to run this risk unless general anaesthesia was absolutely contraindicated. This being said, these figures do not accord with our own experience, nor with that of many other surgeons who also electively perform cataract surgery under local anaesthesia, nor with other published results.

It is clear that rather than settling issues, the survey may have actually raised more serious issues. Careful thought needs to be given to whether this is a valid survey and ought to be accepted, or repeated if flaws can be identified and addressed. If the survey is valid it is necessary to identify the reasons for and remedy the high rate of adverse effects. We look to the Royal College of Ophthalmologists to undertake this.

There is a further issue, which is probably the main issue in ophthalmic anaesthesia from the patients' perspective. This is 'What is the safest anaesthetic for the procedure?' This can only be answered by comparing local anaesthesia with general anaesthesia and comparing the various techniques of local anaesthesia. We would hope that any future survey could be structured so as to answer this question.

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Sir,

I read with interest the paper by Claoué and colleagues on the relative frequencies of ophthalmic disease in Moorfields Eye Hospital and one of its outreach clinics.[1] Whilst it may be useful for an individual department to examine its referrals in terms of proportion percentages, caution should be used before assuming similar proportions would exist in other hospital populations as there are clearly many factors that play a part in whether a patient is referred to a particular unit.

In order to plan services appropriately for a population, a combination of epidemiological prevalence studies and demand incidence work is required. Such an example of the latter type of study was performed at Nottingham in 1989/90,[2] which included all presentations of eye disease in a balanced population of 36 000 utilising verified data from GP attendances and Eye Casualty.

It is interesting to compare some 'proportions'. In Nottingham, the demand incidence for cataract (at 1.9 per 1000 population per year) was approximately twice that of glaucoma and suspect glaucoma, whereas in the Moorfields series referral for cataract was 3.3 times as common in 1991 and 3.6 times as common in 1993. However, the ratio of glaucoma to age-related macular degeneration was similar in the two studies, at 1.27 and 1.16 in the Moorfields series versus 1.29 in the Nottingham series. This suggests, for whatever reasons, a 'bias' towards cataract in the Moorfields patient population. This bias may be greater than it appears as some of the Nottingham patients presenting to their GPs may not have been referred to the hospital service. Indeed, only 29% had an acuity less than 6/12 in both eyes and 33% had 6/12 or better in both eyes.

The purchaser/provider split renders recent data from many Units suspect and changes in the pattern of disease presenting to ophthalmologists must be identified by the use of the appropriate methodology, i.e. by demand incidence studies.

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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.

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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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