was related to the birth weight and gestational age in the multiple gestation group. Interestingly the percentage of neonates reaching threshold was smaller in the multiple gestation group than the single gestation group. Using risk-adjusted outcomes for very low birth weight (VLBW = between 401 and 1500 g) neonates it has been shown that twins and singletons have similar morbidity and mortality outcomes.[7] In addition it has been reported that the more severe ROP may be observed in the higher birth weight twin.[8] We therefore do not accept that the only reason for these children reaching threshold disease is age and birth weight.

Excellent obstetric and neonatal care is indeed having an impact on ROP screening, with the evidence suggesting a reduction in severe retinopathy except in the extremely premature neonates of less than 25 weeks gestation.[10–12] The increasing survival of VLBW infants is possibly a direct result of good obstetric and neonatal care,[9] with the mothers of twins more likely to receive prenatal care, have caesarian delivery and receive antenatal glucocorticoids than those with singleton pregnancies.[7] Hence assisted conceptions are likely to receive 'good' obstetric care and the development of ROP would appear not to be a direct result of their plural gestation or solely a function of their gestational age and birth weight. Our study suggests that the method of conception is an important risk factor for the development of severe ROP.

We continue to exercise and advise extreme caution in all assisted conceptions reaching stage 3 disease, as the experience in our cohort demonstrates that they are likely to progress to threshold disease.

References

1. McKibbin M, Dabbs TR. Assisted conception and retinopathy of prematurity. Eye 1996;10:476–8.
2. Watts P, Adams GGW. *In vitro* fertilisation and stage 3 retinopathy. Eye 2000;14:330–3.
3. Luke B. The changing pattern of multiple births in the United States: maternal and infant characteristics 1973 and 1990. Obstet Gynecol 1994;84:101–6.
4. MRC working party on children conceived by *in vitro* fertilisation. Births in Great Britain resulting from assisted conception, 1978–87. BMJ 1990;300:1229–33.
5. McFaul PB, Patel N, Mills J. An audit of obstetric outcome of 148 consecutive pregnancies from assisted conception: implication for neonatal services. Br J Obstet Gynaecol 1993;100:820–5
6. Blumenfeld LC, Siatkowski M, Johnson RA, Feuer WJ, Flynn JT. Retinopathy of prematurity in multiple gestation pregnancies. Am J Ophthalmol 1998;125:197–203.
7. Donovan EF, Ehrenkranz RA, Shankaran S, *et al.*, for the National Institute of Child Health and Human Development Neonatal Research Network. Outcomes of very low birth weight twins cared for in the National Institute of Child Health and Human Development Neonatal Research Network's intensive care units. Am J Obstet Gynecol 1998;179:742–9.
8. Fellows RR, McGregor ML, Bremen DL, *et al.* Retinopathy of prematurity in discordant twins. J Pediatr Ophthalmol Strabismus 1995;32:86–8.
9. Stevenson DK, Wright LL, Lemons JA, *et al.*, for the National Institute of Child Health and Human Development Neonatal Research Network. Very low birth weight outcomes of the National Institute of Child Health and Human Development Neonatal Research Network. January 1993 through December 1994. Am J Obstet Gynecol 1998;179:1632–9.
10. Higgins RD, Mendelsohn AL, DeFeo MJ, *et al.* Antenatal dexamethasone and decreased severity of retinopathy. Arch Ophthalmol 1998;116:601–5.
11. Termote J, Schalij-Delfos NE, Brouwers HAA, Donders ART, Cats BP. New developments in neonatology: less severe retinopathy of prematurity? J Pediatr Ophthalmol Strabismus 2000;37:142–8.
12. Coats DK, Paysse EA, Steinkuller PG. Threshold retinopathy of prematurity in neonates less than 25 weeks estimated gestational age. J AAPOS 2000;4:183–5.

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Sir,

I read with interest the report concerning first day discharge to the optometrist with subsequent communication to the hospital in 77% of cases.[1]

Before we are all swept into a whirlwind of fast track cataract surgery (others, notably in Gloucestershire, have recommended but one post-operative visit[2]), may I make a plea for documentation of visual outcomes.

The national benchmark is for 6/9 with correction after phacoemulsification in 76%.[3] However, this leaves much to be desired: the aim should be for at least 80% seeing 6/12 unaided and with 99% achieving 6/9 with correction when co-morbidity is excluded. How are we therefore to improve standards without careful follow-up and self-audit? It seems that the rush into higher turnover with optometrist communication at best in only 77%, will inevitably lower the standard of patient care.

References

1. Muthucumarana DJ, Rimmer TJ. Cataract surgery and the optometrist. Eye 2000;14:777–8.
2. Moss S. Referring patients to hospital: the Gloucestershire experience. Action on Cataracts, Leeds, 12 July 2000.
3. Desai P, Minassian DC, Reidy A. National Cataract Surgery Survey 1997–8. Br J Ophthalmol 1999;83:1336–40.

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Sir,

Percival is concerned that post-operative arrangements outlined in our paper are inadequate for audit. Concerns of such a leading figure in modern British cataract surgery must be taken very seriously indeed. We all want the highest standard of surgery delivered to as many patients as possible within the confines of available recources.

Despite the absence of feedback in 23%, only 6 eyes were unaccounted for out of 318 cataract operations because of ongoing follow-up for other conditions. Those few patients were probably satisfied because it is inconceivable that a British general practitioner would refer a dissatisfied patient to another eye surgeon without informing the operating surgeon. Thus no patients suffered as a result of the shared care.

High standards of surgery could be ensured without exhaustive post-operative details of every single patient. A successful cataract operation depends on a highly skilled surgeon and an accurate biometrist. A surgeon could be peroperatively assessed as part of the College inspection, in part analogous to the monitoring of airline pilots. This would reveal more of the surgeon's respect for the endothelium than could be detected in clinics, not to mention bedside manner, which is important for an overall success of local anaesthetic cataract surgery as perceived by the patient. In due course artificial, calibrated eyes may be available against which biometrists could be measured.

Shared care of cataract patients now saves our department about a thousand outpatient appointments a year and may contribute to the fact that after the decision in clinic to operate, our cataract patients have about 6 weeks to wait for their operation.

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Sir,

We read with interest the article on the role of IgM isotype anticardiolipin antibodies in ocular vaso-occlusive disease.[1] As the author states, the published data suggest that the isotypes of aCL antibodies (IgM, IgG and IgA) are not homogeneous with respect to ischaemic events. The role of high titre IgG anticardiolipin antibodies in the pathogenesis of vaso-occlusive disease is now well documented, but the role of the other isotypes, IgM in particular, remains controversial.[2,3] The author describe 2 patients with ocular vaso-occlusive disease in whom an isolated, moderately raised titre of IgM aCL antibodies was found. They then conclude that their data provide evidence that the IgM isotype may play an important role in the pathogenesis of aCL antibody-associated thrombosis in patients with the primary antiphospholipid syndrome (PAPS). It is, however, appreciated that both IgG and IgM aCL antibodies can be raised as an epiphenomenon in response to vascular occlusion.[4] Therefore one can only be certain that raised aCL antibodies have a causative role in the pathogenesis of a vaso-occlusive event if they remain persistently elevated after the acute vascular event.

In this report the authors have not made it clear that the cases they describe did have persistently elevated IgM aCL antibodies. The presence of persistently elevated IgM aCL antibodies in an ocular vaso-occlusive disease would be an important contribution to our understanding of the role of the different aCL isotypes in vaso-occlusive disease. However, without the confirmation that the IgM aCL antibody titres were persistently elevated the conclusion that the raised IgM aCL antibodies were pathogenic can only be speculative, and it would be premature to conclude that the moderately raised aCL antibodies were causally related to the vaso-occlusive events described.

References

1. Dogulu FC, Kansu T, Sibel K. The role of IGM isotype anticardiolipin antibodies in occlusive vascular disease: report of two cases with primary antiphospholipid antibody syndrome. Eye 2000;14:789–90.
2. Asherson RA, Merry P, Acheson JF, Harris EN, Hughes GRV. Antiphospholipid antibodies: a risk factor for occlusive ocular vascular disease in systemic lupus erythematosus and the 'primary' antiphospholipid syndrome. Ann Rheum Dis 1989;48:358–61.
3. Gahravi AE, Harris EN, Asherson RA, Hughes GRV. Anticardiolipin antibodies: isotype distribution and phospholipid specificity. Ann Rheum Dis 1987;46:1–6.
4. Harris EN, Gahravi AE, Hughes GRV. Antiphospholipid antibodies. Clin Rheum Dis 1985;11:591–609.

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Sir,

We thank Mr Puri and Dr Squirrell for their interest in our report.

We agree that raised aCL antibodies have a causative role in the pathogenesis of a vaso-occlusive event if they remain persistently elevated after the acute vascular event. In both our patients we assayed the aCL antibody titres (IgM and IgG) every 6 months and the aCL IgM levels remained persistently elevated. Therefore, we conclude that the persistently elevated IgM isotype aCL antibodies of our patients are causally related to their vaso-occlusive events.

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Sir,

I enjoyed the article by Potamitis *et al.*[1] and the accompanying editorial[2] about driving safety after pupil dilatation, but feel that further comment is warranted.

Crashes are of multifactorial causation and previous investigators have found only minimal or no significant association between visual acuity or contrast sensitivity and crash rates.[3] Given the small differences in visual acuity and contrast sensitivity associated with mydriatic use,[1] if a driver were unfortunate enough to be involved in a crash after pupil dilatation, neither tropicamide nor the administering ophthalmologist should be considered culpable.

In their discussion, Potamitis *et al.* highlight the subject who had a delayed reaction time of 0.62 s following pupil dilatation, which they assume to be an effect of the tropicamide. But differences in reaction time were non-significant, there was not a control group given saline twice, and if tropicamide is responsible for delayed reaction time, how can one explain the other 42% of their subjects who had either no change or an improvement in reaction time (of up to 0.33 s)?

The editorial mentioned that pupil dilatation may exacerbate the adverse effect of cataract on visual function, but equally, many patients with axial lens opacity would in fact be better off driving home with dilated pupils. It was also implied that an insurance company may refuse to support a driver with dilated pupils who crashes. But given the current lack of evidence to support any association between pupil dilatation and crash risk abrogation of the insurance company's responsibility to the victim of any crash would be wholly unjustified and should be roundly condemned by our profession.

The editorial rightly highlighted the ineffectiveness of miotics in reversing the effects of tropicamide. The risk of inducing angle closure glaucoma with miotic use should also be noted,[4] particularly when phenylephrine has been previously administered.[5] Administration of pilocarpine after mydriasis represents a far greater danger to the patient than driving home with dilated pupils.

From literature spanning a century of motoring history I am now aware of a single reported case of a motor vehicle crash attributable to the use of tropicamide. The available evidence permits clear and simple advice to be given: patient may drive home if they feel comfortable to do so and can meet minimum legal visual acuity standard.

References

1. Potamitis T, Slade SV, Fitt AW, McLaughlin J, Mallen E, Auld RJ, *et al.* The effect of pupil dilatation with tropicamide on vision and driving simulator preformance. Eye 2000;14:302–6.
2. Keightley S. Pupil dilation and driving. *Eye* 2000;14:261–2.
3. Owsley C, McGwin G. Vision impairment and driving. Surv Ophthalmol 1999;43:535–50.
4. Gorin G. Angle closure induced by miotics. Am J Ophthalmol 1966;62:1063–7.