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.

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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.

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