Sir,

I thank Dr Weale for his comments[1] regarding our article 'Tono-Pen tonometer and corneal thickness'.[2] I was very sorry that I made the typing error on the regression line equation in Fig. 1. It should read $y = 0.87\ x + 1.50$, instead of $y = 1.5\ x + 0.87$ (where y is the measurement at the central cornea and x the measurement at the mid-peripheral cornea). Although the peripheral measurement was significantly larger than the central ($p < 0.01$), the difference was about 0.4–1.2 mmHg when the central reading was 12–20 mmHg. Thus we suggested that 'no *clinically* significant difference was observed between the intraocular pressure (IOP) readings of central and mid-peripheral cornea measured by the Tono-Pen'. In addition, although the intercept is 1.5, it is not significantly different from zero ($p = 0.19$). Corneal curvature has been suggested to negatively affect the IOP measurement, in that more fluid should be displaced under a steep cornea than under a flat one, which increases the ocular rigidity in overestimating the IOP.[3] However, this notion was not supported by our other clinical study which observed no correlation between corneal curvature and IOP of 323 subjects.[4] Thus the factor of corneal curvature has not been considered in the article. Our subjects' ages were between 45 and 65 years and all were free of any corneal disease. Since we just compared the IOP at two different *corneal* points, only about 3 mm apart, they are assumed to be similar in tissue structure, except the thickness and curvature.

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Sir,

A marked pigment dispersion is regularly observed in eyes with exfoliation syndrome or capsular glaucoma. This is why many authors have asserted that pigment liberation to the aqueous humour with subsequent clogging of the trabecular meshwork plays a key role in the pathogenesis of capsular glaucoma.[1–3] However, no definitive conclusion can be drawn, since the exfoliative process without melanosome release from the posterior chamber is extremely rare. In an attempt to separate the two conditions, an interesting case has recently been presented in *Eye* by Tarkkanen and Kivelä in which pigment dispersion syndrome occurred in both eyes, followed by exfoliation syndrome together with capsular glaucoma development in one eye only.[4] Since the two syndromes occurred sequentially, it was concluded that the 'development of exfoliation syndrome may take place irrespective of pigment dispersion'.

It is unlikely that the synthesis of exfoliation material is pigment-dependent in remote tissues usually devoid of melanosomes. Suggesting a similar exfoliation pathogenesis throughout the body, a pigment-independent exfoliation production intraocularly is a consequence, as also indicated by the authors.[4] This is apparently not a controversial conclusion. However, why not include capsular glaucoma in the conclusion as well?

In contrast to exfoliation syndrome, capsular glaucoma is a strictly ocular disease, and we know that a massive temporary pigment release to the aqueous is followed by a marked intraocular pressure peak irrespective of exfoliation syndrome.[5] The question is whether melanosomes are needed to trigger the development of capsular glaucoma. To me it seems that the presented case allows the following extension of the authors' conclusion: Neither exfoliation syndrome nor capsular glaucoma is dependent on the occurrence of pigment dispersion.

Another observation supporting this view is a patient with general albinism who presented with a classical capsular glaucoma, including advanced disc cupping, visual field loss and intraocular pressure increase. There is no reason to believe that Tarkkanen and Kivelä were aware of this case, because it was mentioned along with some other prior information.[6] However, in this connection it has to be mentioned, because it goes right to the heart of the discussion. It shows that the presence of melanosomes is at least not decisive in the pathogenesis of capsular glaucoma, and that the synthesis of exfoliation material is not linked to melanin metabolism.

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Sir,

We read with interest the report of three cases of early onset surgically induced necrosis (SINS) by Mansour and Bashshur[1] and would like to expand on the proposed mechanisms and their implications, comment on the presentation of disease and place the treatment in the context of our own experience.

The authors proposed that scleritis occurred due to dellen formation where the conjunctiva lay retracted post-

operatively. If this mechanism is correct, there are implications for wound closure in ophthalmic surgery. Over recent years it has become more common to leave conjunctiva to grow back over a scleral wound or blow it back with an injection of subconjunctival antibiotics rather than suture it carefully. An alternative pathogenesis is that local ischaemia caused by suture or cautery[2] could have caused conjunctival necrosis resulting in the appearance of conjunctival retraction and dellen formation. This may have encouraged tear- or blood-derived leucocytes to produce a pool of immune mediators such as matrix metalloproteases (MMP 8 and 9) released from leucocytes causing necrotising scleritis.

The early presentation of these cases at 1, 3 and 4 weeks post-operatively supports the influence of such peri-operative surgical factors. In our own experience, and those of others, the majority of such cases of SINS present months if not years after surgery, suggesting other aetiologies such as molecular mimicry/cross-reactivity between ocular antigens and remote tissue or microbial antigens.[2] The patients described in this paper may represent a subgroup of SINS with an earlier presentation and a good response to local surgical treatment.[1] However, we believe that this group should be placed in the context of later-onset SINS where early systemic immunotherapy with prednisolone and cyclophosphamide remains the mainstay of treatment.[2] Systemic treatment not only rapidly alters the course of ocular disease but may save life when the scleritis is part of a systemic vasculitis.

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Sir,

The comments of Ramsay and Dart are very well taken. They differentiate early-onset scleral melt due to conjunctival retraction from the late-onset vasculitic scleritis. It is of note that the entity described in our article is rare. More common causes of scleral melt include forceps-induced scleromalacia due to non-gentle holding of the eyeball (especially in beginning trainees) and keratoconjunctivitis sicca. Causes of scleral melt in a teaching university setting are, in order of decreasing frequency: (1) trauma, (2) keratoconjunctivitis sicca, (3) vasculitis, (4) conjunctival retraction – dellen complex.

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Sir,

We read with interest the article in the August 1999 issue of *Eye*, 'Peroperative retinoscopy as a predictor of final post-operative refraction'.[1] It has close parallels with a study we published in the *British Journal of Ophthalmology* in July 1998.[2] We feel that 'immediate post-operative' is a more accurate description than 'per-operative': retinoscopy was undertaken immediately after the operation in both studies.

We also found that following phacoemulsification surgery, immediate post-operative objective refraction can be performed satisfactorily in most cases. Whereas Tappin and Ferguson compared the accuracy in prediction of post-operative refraction by immediate post-operative retinoscopy and biometry and found the former to be significantly better, we looked at the refractive change from the immediate post-operative period to the final refraction. For the particular implant type used in the study (Chiron C10UB) we found a statistically significant refractive change with a mean hypermetropic shift of 1.11 D which can then be taken into account if immediate implant exchange is considered.

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Sir,

We thank Tu *et al.* for their comments on our paper.

We agree that in some respects the two papers are similar; however, we attempted to compare per-operative retinoscopy with the refraction predicted by pre-operative biometry. Retinoscopy was thus used as a predictor of immediate post-operative refraction, rather than for recording the change in post-operative refraction during the first 6 weeks after surgery. We think that per-operative rather than post-operative refraction is a more accurate term owing to the fact that the retinoscopy was performed while the patient was still draped and a sterile surgical field was maintained. If the retinoscopy indicated a very different refraction to that expected from the biometry then an implant exchange could be carried out immediately.

We note that Tu *et al.* found a hyperopic shift of 1.1 D from the time of surgery (in which a plate haptic implant was used) until 6 weeks post-operatively. In our study with flexible haptics there was also a change in refraction during the 6 week period following surgery; there was a mean error of +0.55 D. We think this was due either to a systematic error in the retinoscopy or to a change in the position of the lens.

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Sir,

We much appreciated Choong *et al.*'s clinical study,[1] introducing a new protocol for the management of acute angle closure glaucoma. Nevertheless there is an apparent discrepancy as the authors suggest commencing stage 2 treatment (osmotic agents) one and a half hours after the administration of the stage 1 treatment (includes oral Diamox), while declaring that the maximum effect of the latter is exhibited in 2 h.

As the protocol is based on theoretical considerations rather than good randomised controlled trials of glaucoma treatment, would it be best to treat according to the known effects of the drugs concerned?