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-

