augmentation and was rapidly discontinued as a result. It is however highly misleading to use this analogy in any discussion regarding silicone oil. Silicone is not a mineral oil; it is not derived from petroleum and is never likely to be.

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Sir,

Reply to 'Silicone oil migration causing increasing proptosis 13 years after retinal surgery'

We thank Snead *et al* for their comments in relation to our recent paper, which enable us to explain elements of the report, which may require further clarification.

The indication we have that this condition was progressive was from the patient himself. He had failed to attend for follow-up appointments following his original surgery. However, he stated that his eye had started to become more prominent approximately 6 months before he reattended despite relative stability in the preceding decade.

The CT scan shows enhancement of the soft tissues, mainly around the medial rectus, but there are changes extending behind the globe medially. This mass in itself would not cause an axial proptosis, rather a non-axial proptosis. We agree that the CT scan shows an enlarged right eye and axial length measurements were specifically obtained as this could have been considered a compounding factor in the apparent prominence of the right eye. However, the relatively rapid change over 6 months reported by the patient suggests that factors other than axial elongation were involved. We are aware that A-scan ultrasound is attenuated in the presence of silicone oil owing to the lower sound velocity. The sound velocity in silicone oil depends on the viscosity of the oil used. In this case, the oil was 1000 centistokes and therefore the velocity used was 980 min/s and this was taken into account when performing the applanation A-scan biometry. Notwithstanding the accuracy of A-scanning in silicone oil, the authors agree that the CT scan does show an enlarged eye.

Although the patient did not report pain from the eye at any stage, we agree that the corneal changes are consistent with old decompensation. Whether this was due to exposure or to complete corneal decompensation as a result of the initial trauma, surgery, or silicone oil contact with the corneal endothelium, it is difficult to determine. It is likely that all played a part. We have no way of determining whether he had developed a secondary glaucoma, as he had not attended for any follow-ups. However, it is highly likely that at some stage he developed glaucoma as this is a known complication of silicone oil. However, as regards the possibility of (relatively rapid) enlargement of the globe from glaucoma, we feel this is unlikely. Certainly, if glaucoma develops in a child the globe will enlarge as a result of the elasticity of the scleral and corneal tissue. The cornea can enlarge up until about age 3 years, but the sclera can continue to deform until about age 10 years.[1] In an adult with normal sclera, this scenario typically does not occur as the adult globe is no longer distensible because of crosslinking of the scleral collagens. The eye wall can stretch with raised intraocular pressure but scleral stretch is minimal and totally reversible without permanent globe enlargement.[2]

We confirm that at the time of enucleation there was clear evidence of large glistening globules of silicone oil throughout the peri- and retro-orbital tissues particularly medially and the conjunctival and tenons layers were extremely adherent together. Therefore, clinically there was no doubt that silicone oil had been actively leaking around the globe. We described the inflammation around the silicone as 'mild inflammation'. We did not describe the inflammation as granulomatous, as we agree it is not 'granulomatous inflammation' in the strict histopathological use of the term. However, the term 'granuloma' is used by pathologists to describe any small nodular delimited aggregation of mononuclear cells and the definition of granuloma is therefore appropriately used when describing 'oil granuloma' and 'silicone granuloma'.

We had also not previously seen such an inflamed eye from silicone oil leakage before and felt that this case was worth reporting, as it was an unusual occurrence. We believe that 'corneal exposure' alone cannot account for the widespread changes reported. The vascularised corneal changes were long-standing and there was no associated epithelial defect or evidence of exposure. The large gelatinous subconjunctival mass seen in the clinical photograph was highly unusual in appearance—prompting the biopsy. Our initial clinical diagnosis included lymphoma but the biopsies were negative for this demonstrating the silicone oil changes only.

In our paper, we make the statement 'Oil granuloma occurs when bulky mineral oils are injected into body tissues'. One widely accepted definition of 'mineral' is any inorganic substance, and of 'oil' is a greasy liquid. By

this definition, we consider silicone oil to be a 'mineral oil' and never intended to suggest that silicone oil is derived from petroleum. We apologise if this was not clear to the readers. Mineral oil is a nonspecific term used for a variety of oils and our comments in relation to breast augmentation were specific for silicone oil.

We thank the authors for their interesting and informed comments about this case and acknowledge their considerable knowledge of the long-term complications of silicone oil internal tamponade. We hope our comments help clarify the key message of our recent case report.

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Sir,

Reply to Ghazawy *et al*

We are flattered to have attracted the kind of high-quality, considered response above. Ghazawy, Saldana and McKibbin have provided original data and in many ways their article surpasses our own in its contribution to the debate for this reason. Their study examined an innovative and potentially sustainable model for fast tracking suspected choroidal neovascular membrane (CNV) referrals and found that 42% of cases with distortion on Amsler grid testing had neovascular macular degeneration. Faced with a confirmed pathology in less than half of those referred, some disappointment is implicit in their use of the word 'very' in their subsequent statement; '(there were) a *very* high number of false positives'. It is possible to draw precisely the opposite conclusion; namely that for so simple and inexpensive a test, the proportion with genuine pathology in this group is remarkably high.

This proportion represents the positive predictive value (PPV) of the Amsler test. Unlike the sensitivity and specificity of the test, which are entirely independent of the amount of pathology in the community, the PPV is profoundly affected by the prevalence of the pathology being sought. A PPV of 42% (38% for CNV) compares favourably with the PPV of screening programmes already widely accepted, for example, 9% in breast screening for women aged between 50 and 59 years,[1] 1% in cervical screening of postmenopausal women on hormone replacement,[2] and, closer to home, 0% for the finding of isolated field defect and subsequent confirmation of glaucoma.[3]

The authors comment that when the optometrist examined the fundus the sensitivity fell to 71% (it would have been interesting to know by how much it fell, but they do not give the figure derived without examination). They were able to achieve a sensitivity and specificity 90% or more with their 'fast track and refinement' clinic. This would undoubtedly greatly elevate the PPV of those being sent on to the medical retina specialist, as the prevalence of pathology in this population (those referred to secondary care with abnormal Amsler test results) is so much higher than in the community. We would love to hear a full report of this patient pathway or of its wider adoption and use in larger numbers.

As the gold standard remains fluorescein angiography, the need for this to take place in the hospital ophthalmic care setting is self-evident, and it demonstrates what may be achieved within the constraints of current resources. But most important of all, it achieved its primary goal: it was fast.

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