primary angle closure (PAC) is not independently associated with cataract progression.

The authors mention that cataract surgery may be an alternative treatment for occludable angles, potentially addressing both PAC and cataract blindness with one procedure. The potential complications from intraocular surgery, though, are greater than those from LPI. However, LPI has complications such as intraocular haemorrhage and inflammation, intraocular pressure (IOP) spikes, glare, diplopia, and corneal damage. These are primarily not sight threatening, but have to be always taken into consideration.

Another potential complication is cataract formation, and this has been extensively reviewed by Yip *et al*. Despite their conclusion, there is still some controversy on this matter, with some authors supporting the opposite.[2,3] Thus, one must always be aware of such a theoretical risk after LPI. Except for the disturbances in aqueous flow in patients undergoing LPI, we suggest that, using higher-energy settings, inaccurate focusing of the laser beam, excessive or undertreated post-LPI uveitis, previous intermittent angle-closure episodes with IOP elevation, and other anatomical parameters, yet to be recognised, could be considered as possible stimuli of crystalline lens disturbance with consequent opacification.

A potential complication of Nd:YAG LPI was reported by us recently.[4] This involves damage to the zonules with subsequent dehiscence during routine phacoemulsification cataract surgery, affecting an otherwise healthy female with narrow angles. Our paper includes reports suggesting the same effect of LPI (both with Nd:YAG and with argon lasers), resulting in spontaneous dislocation of the crystalline lens.[4] We suggested that Nd:YAG LPI may be regarded as an isolated risk factor for structural zonular damage and instability of the crystalline lens, and appropriate precautions should be taken during intraocular surgery. Regardless of the opacification being the result of the LPI, age-related or of any other cause, zonular damage could have considerable implications in subsequent cataract surgery, especially in cases where the zonules are already compromised, such as in pseudoexfoliation syndrome, previous ocular trauma, and congenital systemic diseases like Marfan's syndrome.[5]

Considering the large number of patients who would potentially benefit from prophylactic LPI, potential adverse sequelae of such a procedure must not be underestimated. More specifically, the possibility of cataract progression and zonular instability after LPI has important implications for patients at risk of angle closure. Choosing between primary cataract surgery and LPI is the main consideration in such cases. The therapeutic approach should be individualised and treatment benefits must always be balanced against eventual complications.

Finally, we would like to congratulate the authors for their excellent contribution on a very important field of ophthalmology.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Reply to Athanasiadis *et al*

We are grateful to Dr Athanasiadis *et al*[1] for their interest in our manuscript,[2] and for this opportunity to reiterate the points made therein.

Zonular disruption during laser peripheral iridotomy (LPI) can occur if sufficient energy is applied or if there is pre-existing zonular weakness. Indeed, Nd:YAG laser zonulotomy and hyaloidotomy are used in the management of some cases of aqueous misdirection syndrome. However, in our experience from the specialist angle-closure clinic at Moorfields City Road, the Zhongshan Angle-closure Prophylaxis (ZAP) study in Guangzhou, China (ISRCTN45213099), and our research programme in Mongolia, culminating in over 4500 LPIs and 800 phacoemulsification procedures in the same pool of patients, we have not encountered this problem with LPI.

Dr Athanasiadis's case report[3] omits to mention where the initial phaco wound was (ie superior or temporal). This may have some bearing on the location of the dehiscence. The report also does not mention the power and number of shots during LPI. We were puzzled as to why two iridotomies were performed in each eye of the patient reported in this case. One adequately sized iridotomy is sufficient in management of angle closure.

Angle closure is known to affect people with a variety of genetic mutations that cause zonular abnormalities and weakness as part of their phenotype: PXF, FBN1 (Marfan and Weill Marchesani syndromes), lysyl hydroxylase (Ehler Danlos VI), MTHFR

(homocystinuria), and ADAMTS4 and ADAMTS10 (spherophakia). As per the principles of Occam's razor, we would suggest that the case Dr Athanasiadis *et al* report had a pre-existing zonular weakness and/or received higher-than-usual amounts of laser energy. It is important to emphasise that LPI should be performed by a skilled, experienced operator, using the lowest possible power to achieve a satisfactory iridotomy. We would advocate the use of sequential argon/YAG iridotomy in patients with thick, dark brown irides.[4]

Regarding the risk of cataract formation/progression after PI, similar principles to those outlined above apply. With excess power or inappropriately applied laser treatment, it is possible to induce lens opacities, but this can be avoided with careful and precise treatment. Studies suggesting that LPI accelerates the formation of age-related cataract are exclusively retrospective studies, or individual case reports. Some have used surrogate outcome measures, such as reduction in visual acuity, rather than lens opacity grading. We believe that our study,[2] which was carried out prospectively, in the largest number of treated cases so far studied, with a control group selected from the community, using a standardized objective assessment of lens opacity (LOCS III system), currently constitutes the most robust scientific assessment of the risk of lens opacity after laser iridotomy.

The choice of either laser iridotomy or lens extraction for management of angle-closure glaucoma should be informed by the ongoing MRC EAGLE trial (https://viis.abdn.ac.uk/HSRU/eagle/).

We are grateful to the journal for giving us the opportunity to reiterate these points.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Screening for childhood blindness and visual impairment in a secondary school in rural Malawi

Childhood blindness is one of five areas of disease control in Vision2020: The Right to Sight,[1] and it is estimated that there are 300 000 blind children in Africa.[2] However, data on the epidemiology of childhood blindness in sub-Saharan Africa is scant, as children are rarely included in blindness surveys. Historically, information has been obtained from schools for the blind and, more recently, the key informant method,[3] but this does not address children with milder forms of visual impairment. Screening for visual impairment in regular schools may also yield useful information, although children with debilitating visual impairment may be less likely to attend, and secondary school attendance is not universal in Africa.

We examined 1000 children (aged 11–19 years) attending secondary school in Malamulo in rural Malawi. Presenting visual acuity (VA, with spectacles if owned, but uncorrected otherwise) was assessed with Snellen Chart at 6 metres. If presenting VA was $<6/18$ ('visual impairment'), they were invited to attend Malamulo Hospital Eye Department for formal refraction and slit-lamp examination, following suitable permission. Spectacles were dispensed if necessary.

There were 39 students (3.9%) with presenting VA $<6/18$ in one ($N=20$) or both ($N=19$) eyes. Among them 20 (51.2%) were male. One student (0.1%) was blind (VA $<3/60$) bilaterally, due to high myopia (-16.0 dioptres), and one had unilateral blindness from amblyopia (due to strabismus). Causes of visual impairment are presented (Table 1). In all, 29 (14 in bilateral group and 15 in unilateral group) attended the

Table 1 Aetiology of visual impairment in a secondary school in rural Malawi

Aetiology	*Bilateral vision impairment N (%)*	*Unilateral vision impairment N (%)*
Myopia	12 (85.7)	1 (6.7)
Cataract	1 (7.1)	2 (13.3)
Corneal scar	1 (7.1)	4 (26.7)
Trauma	—	2 (13.3)
Keratitis	—	1 (6.7)
Amblyopia (due to strabismus)	—	2 (13.3)
Other refractive error	—	3 (20.0)
Total	14 (100.0)	15 (100.0)