impairment of the primary visual cortex.[4] Whooshing sounds were likely hallucination originating from auditory cortex. The vivid combination of visual and auditory disturbances has been found to explain alleged haunted houses.[5]

The lesson is that CO poisoning can mimic GCA and should form part of the differential. Focused social history and carboxyhaemoglobin level can help exclude the condition.

Conflict of interest
The authors declare no conflict of interest.

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K Xue[1], M Mota[1], K Rahman[1], M Magliano[2] and K Manuchehri[1]

[1]Department of Ophthalmology, Stoke Mandeville Hospital, Buckinghamshire, UK
[2]Department of Rheumatology, Stoke Mandeville Hospital, Buckinghamshire, UK
E-mail: kanminxue@doctors.org.uk

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Sir,

'Non-steroidal drug-induced glaucoma' by MR Razeghinejad, MJ Pro and LJ Katz

In this review,[1] the authors conclude that 'the majority of cases of drug induced CAG are of the pupillary block closed angle type and preventable …'—a claim unsupported by any evidence other than anecdotal case reports. In the elderly, a significant number of patients will have narrow angles,[2] with a prevalence of CAG in Caucasian eyes as high as 0.6%.[3] The authors have not shown that systemic anticholinergic or sympathomimetic drugs are more important than other known precipitating factors of CAG, such as emotional upset, dim illumination, reading or the prone position.[2]

The reference for their figure that 'at least one third of acute closed angle glaucoma (ACAG) cases are related to over-the-counter or prescription drugs' is a paper discussing risk factors in open-angle glaucoma.[4]

The authors' view that systemic anticholinergic or sympathomimetic medication can cause pupillary dilatation, which precipitates pupillary block CAG, is unsubstantiated. The risk of inducing ACAG has been shown to be zero with tropicamide and between 1 in 4000, and 1 in 20 000, when using long-acting or combined agents.[5] It is unlikely, therefore, that the minimal degree of pupil dilatation produced by systemic medications could induce CAG.

The authors warn that 'Mapstone reported severe IOP elevation, following tropicamide dilation in 19 out of 58 patients …'.[6] These 58 eyes in Mapstone's study were all eyes with untreated angle-closure glaucoma and were dilated with tropicamide as part of a study of agents to use in provocative testing. The pressure rise in the 19 responding eyes was a mean pressure rise from 18 to 30 mm Hg, and all pressures returned to normal within 2 h of installation of the topical pilocarpine and systemic acetazolamide.

As idiopathic ACAG is almost always unilateral,[2] the unilateral nature of ACAG in the case reports would suggest that the ACAG is idiopathic. CAG arises from an anatomical pre-disposition in the ageing eye,[2] and contrary to the conclusions of Razeghinejad *et al*,[1] there is no evidence that the use of systemic medications increases the risk of the development of pupillary block CAG.

Conflict of interest
The author declares no conflict of interest.

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PK Wishart
St Pauls Glaucoma Unit, Royal Liverpool University Hospital, Liverpool, UK.
E-mail: peter.wishart@rlbuht.nhs.uk

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