known disease are hypothesis-driven, where many scientists have contributed a great deal in terms of thought and experimental data. It is important not to let one scientist's undoubtedly questionable data blind us in our attempts to understand more about glaucoma and so realise more appropriate therapies. If the trend is to encourage the argument that a role for glutamate in glaucoma is fictional, then it may not be to the benefit of future glaucoma patients.

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Sir,
**Charles Bonnet syndrome (visual hallucinations)
following enucleation**

We read with interest the excellent article by Drs Ross and Rahman, describing visual hallucinations in a patient following enucleation.¹ These hallucinations were characteristic of Charles Bonnet syndrome (CBS) and, interestingly, disappeared with eye closure. We would like to propose a possible pathophysiologic mechanism to explain this observation.

A visual acuity of 6/6 in the patient's other eye is not incompatible with the diagnosis of CBS, which has been described both in people with good visual acuity in the fellow eye^{2,3} and in patients who have visual field defects and good central visual acuity in the affected eye.^{2,4,5} Shiraishi *et al*⁶ proposed that it is the dynamic reduction in visual acuity, rather than the actual visual acuity, that has a greater impact on CBS.^{2,7}

This case is intriguing because the hallucinations ceased when the patient's eyes were closed, only to return when he opened his eyes. Although it is well known that eye closure may terminate hallucinations in patients with CBS, this is the first case in which it appears that transient reduction of light perception on closure of the fellow eye is associated with its cessation. The enucleated eye constantly has no light perception and lid closure will not have any additional effect. It is possible, as the authors suggest, that closing the eyes results in secondary normalization of sensory input, thus abolishing the abnormal independent impulses and resultant complex imagery.¹ Another possibility might be

that deafferentation induced changes in the cortical neurons, resulting in reorganization of the receptive field and increased sensitivity to sensory input.⁸ Stimulation of these hypersensitive areas by normal sensory impulses (in this case from the left eye) may trigger visual hallucinations.^{7,9,10} However, a minimum amount of sensory input is required in order to trigger the hallucinations. Therefore, when the patient closes both his eyes, normal input is abolished and the hallucinations cease, only to return when he opens his eyes. This theory would also explain why some hallucinations cease when patients eventually lose all light perception. This possibility is illustrated in another patient who experienced CBS following cortical resection for cortical dysplasia.⁵ In this patient, the hallucinations diminished with eye closure, and varied in intensity with blinking, light intensity, and the sight of moving objects—factors that vary the intensity of the visual stimulation.

Regardless of the mechanism, we agree with the authors that it is important to recognize CBS and its possible occurrence following sudden loss of vision.

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Sir,
Reply to Tan *et al*

We welcome and thank Tan *et al*¹ for their interest and
observations regarding our previously published case
report.

We are in agreement regarding Snellen acuity not
being a diagnostic criteria for Charles–Bonnet Syndrome
(CBS). Numerous reports have highlighted the presence
of CBS-type hallucinations in individuals with good
Snellen acuity with restricted visual fields. It appears,
therefore, that the quality of visual function and its
deviation from the previous norm for the individual are
key elements in production of CBS hallucinations. It is for
this reason that we agree with the observation by

Holroyd *et al*² that sudden and abrupt reduction in visual
function may trigger CBS symptoms.

Tan *et al*¹ comment on reduction of light perception in
the fellow eye terminating CBS symptoms in our case.
We agree that the proposed theory could account for
resolution of CBS on eye closure. This theory, however, is
similar to the other commonly perceived theories of
deafferentation described by Bartlett³ and West.⁴ The
visual sensory cortex, when deprived of normal afferent
input may exhibit spontaneous independent activity
with resultant conscious images. This hypothesis is
supported by the observation that such hallucinations
may be abolished by normal or excessive visual
stimulation.³ The perceptual theory of West describes
afferent input reduction below a threshold level (such as
in disease of the eyes or visual pathway), resulting in the
brain allowing previously registered subconscious
perceptions to emerge into consciousness, resulting in a
hallucinatory experience.⁴

Similarly, as the hallucinations of CBS have only been
documented in acquired visual deprivation, it is possible
that in-built higher centre mechanisms are in place so
that eye closure may trigger intracerebral activity
suggesting to the sensory visual cortex that visual
stimulation has ended, rather than sensory input itself
being detected by sensory mechanisms to be decreased.
This may explain why darkness with the eyes open may
still result in CBS but darkness as a result of eye closure
may obliterate CBS hallucinations.

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