Eur Arch Psychiatry Clin Neurosci (2006) 256 : 62–63 DOI 10.1007/s00406-005-0594-6

LETTER TO THE EDITORS

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Dynamic changes in visual acuity as the pathophysiologic mechanism in Charles Bonnet syndrome (visual hallucinations)

Received: 23 February 2005 / Accepted: 18 March 2005 / Published online: 4 August 2005

We read with interest Shiraishi and coworkers' report of a patient who developed Charles Bonnet syndrome (CBS) during two distinct periods when his visual acuity was declining (Shiraishi et al. 2004). These were followed by hallucination-free periods when his visual acuity was static; in the first case, when visual acuity improved to 0.6 and, later, when the patient became blind. Therefore, it is interesting to note that although CBS is associated with poor visual acuity (Teunisse et al. 1995), the patient experienced hallucinations when his visual acuity was dropping to hand movement, yet the hallucinations ceased when he was blind. We agree with the authors that it may be the dynamic change in visual acuity and not its chronic or static level that is important in the pathogenesis of CBS (Shiraishi et al. 2004). We would now like to propose a possible pathophysiological explanation for these interesting observations, and support this proposal by discussing other patients who have exhibited a similar temporal sequence of events.

It is well known that deafferentation in the brain leads to hyperexcitability in the deafferented brain regions (denervation supersensitivity). In the acute period following deafferentation caused by retinal or visual pathway lesions, there are surviving neurons within the deafferented region and its penumbra with potential for recovery. These neurons attempt to adapt to the visual loss by reorganization of their receptive field and alteration of their sensitivity to incoming stimuli (Eysel et al. 1999). During this period of visual system plasticity, the surviving neurons are hypersensitive to residual visual stimuli and even normal levels of stimuli may be sufficient to trigger visual hallucinations. Once the recovery process is complete or has ceased, the sensitivity of the remaining neurons returns to a normal steady state and therefore does not respond to the same (normal) level of visual stimuli. At this time, the hallucinations cease to occur.

During the patient's initial visual loss, it is possible that because of the hyperexcitability of the visual cortex, normal visual input from surviving areas of functioning retina was sufficient to trigger the visual hallucinations. The improvement of his vision following surgical treatment represents a period when the recovery process was stable. During this time, the sensitivity of the neurons returned to normal, and no hallucinations occurred. It is interesting to note that the patient had one episode of visual hallucination after the surgery, possibly because the visual system was still in the process of recovery and hence hypersensitive to stimuli. When the patient's visual acuity declined a second time following retinal detachment, the visual hallucinations recurred due to renewed attempts at recovery or compensation by the visual system. Once the patient became blind, attempts at recovery were no longer viable, hence no hallucinations were experienced.

In another report, two patients experienced visual hallucinations immediately after macular translocation for choroidal neovascularization (Au Eong et al. 2001). The hallucinations occurred during the postoperative period when visual acuity was poor due to a partial gas bubble and residual inferior retinal detachment. They ceased 3 and 7 days after the surgery, when the retina became reattached. The temporal sequence of visual hallucinations when the visual acuity was poor, and its cessation when the visual acuity improved, further supports the plasticity mechanism we have proposed.

Besides retinal pathology, the mechanism we propose may also apply to lesions in the visual pathways, as evidenced by four patients who experienced visual halluci-

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nations within areas of visual field defect which developed after neurosurgery (Freiman et al. 2004). Similar to the cases described above, the visual hallucinations ceased within days to 6 months after their onset. Although the visual acuities and change in topography of visual fields of these patients were not specified, it is possible that the same pathophysiological mechanisms may apply to these patients.

The temporal sequence of the above patients' symptoms, where hallucinations occurred during periods of dynamic change in visual acuity, indicate that CBS may in fact be a good thing since hallucinations may correlate with adaptive neuroplasticity and periods when visual functions are recovering. This is an important area which requires further study.

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