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Complex Visual Hallucinations in Partial Blindness due to Eye Disease

By N. J. WHITE

SUMMARY Three patients experienced complex formed hallucinations during progressive visual failure from eye disease. The hallucinations began abruptly, were brightly coloured stereotyped figures, animals or objects, and appeared to be provoked by light.

As blindness progressed the clarity, frequency and duration of the hallucinations faded. The patients had no abnormalities other than their eye disease, which in two cases was macula degeneration, and choroideraemia in the third.

Visual hallucinations may arise as a result of a disorder at any level from the peripheral end organ to the highest level of intellectual function. (Duke-Elder and Scott, 1974; Posey and Spiller, 1906). Visual hallucinations related to primary eye disease are usually unformed and consist of simple flashes of light or zigzag lines, whereas more complex visual hallucinations are associated with diseases of the cerebral peduncles (L'Hermitte, 1922), cerebral hemispheres (Russell and Whitty, 1955) and possibly the optic chiasm (Weinberger and Grant, 1940). The following three patients described complex, formed visual hallucinations arising spontaneously as they gradually lost vision secondary to eye disease.

Case 1 (W.S.)

A 64-year-old man presented with a six-month history of complex visual hallucinations. His eyesight had gradually deteriorated since the age of 21 due to choroideraemia. At the time of admission, he was able only to distinguish light from dark. Ophthalmological examination revealed bilateral cataract, some band keratopathy and the fundal appearances of choroideraemia. There were no abnormal neurological signs and he was otherwise well.

The hallucinations had begun abruptly while he was listening to music; he suddenly saw a brightly coloured circus troupe burst through the window. Thereafter, he would see similar vivid animated figures on most days. The hallucinations were provoked by light and abolished by darkness or eye closure. They were unaffected by phenothiazines, but gradually became less vivid and less frequent during a three-year period. All investigations, including skull X-rays and EEG were normal; the CT scan showed mild atrophic changes, but was otherwise normal.

Case 2 (A.T.)

A 75-year-old lady presented with a 12-month history of deteriorating vision. Acuity was assessed as: Right eye 6/9. Left eye 3/12. Examination revealed extensive macula degeneration with mild bilateral cataract, and her vision gradually deteriorated thereafter. Two and a half years later, she suddenly began to see brightly coloured children; animals, trees and houses. The hallucinations occupied the entire visual field and occurred predominantly in the daytime. They were provoked by bright light, but would persist with the eyes closed.

She was admitted for investigation six months later. Neurological examination revealed conductive deafness of the left ear, but was otherwise normal. Her visual acuity was 6/60 right eye, whereas with the left eye she could only count fingers at one metre. Skull x-rays, brain scan and EEG were normal.

In recent months, as her sight has continued to fail, the hallucinations have decreased in frequency and clarity.

Case 3 (E.M.)

A 72-year-old lady presented with a history of deteriorating vision in both eyes over several years. Her acuity at this time was 6/60 Right eye, 6/18 Left eye. She was diagnosed as having macula degeneration, and her eyesight steadily deteriorated thereafter. Four years later, she suddenly began to see brightly coloured trees, plants, and on one occasion gnomes in her garden. There was no overt distortion, but the images would rapidly change their shape and size, and could be abolished by closing the eyes. She was immediately aware that they were unreal.

Eighteen months later, she was admitted for investigation of a mild anaemia, at which time the hallucinations were less prominent. There were no abnormal neurological

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signs; visual acuity was 3/60 right eye, 6/60 left eye. EEG and skull X-rays were normal, while the CT scan showed moderate cerebral atrophy.

Discussion

These three cases demonstrate that visual hallucinations may arise in patients who have impaired vision secondary to eye disease. In each case, the hallucinations were complex, formed, brightly coloured and brilliantly clear—in sharp distinction from the muted indistinct content of those patients' normal visual experience. There did not appear to be any lateralization or localization of the hallucinations to particular areas of the visual field—a feature noted in some hemisphere lesions. (Lance, 1976; Weisman and Hackett, 1958). Each patient's hallucinations were stereotyped, and although familiar in form were not recognized from past experience. Two of the patients became sufficiently familiar with the recurring images of certain human figures to apply nick-names to them.

The complexity of the imagery would suggest origins in the cortical or subcortical regions of the brain. In each case, bright light appeared to trigger the phenomena. The onset of the hallucinations was abrupt, but thereafter they became less prominent as the patients' sight further deteriorated. The patients were elderly, their mental function was good, and each was completely aware of the unreality of this aspect of their visual experience. There was no associated disorder of thought or personality and no abnormality in the other sensory modalities.

Complex visual hallucinations are not uncommon in the partially blind following cataract extraction (Weisman and Hackett, 1958) or corneal grafting when the eyes are covered. The perception of sparkling lights (phosphenes) can result from a variety of stimuli (Duke-Elder and Scott, 1974) ranging from pressure on the lobe to optic nerve demyelination (Earl, 1964). Irritative stimuli within the eye such as retinal detatchment may provoke a variety of brightly coloured photopsiae which are simple in form and content, although more complex hallucinations have been described in retinal haemorrhage (Ormond, 1925). Lesions from the optic chiasm to the cortex may provoke complex visual hallucinations. The images described in these three cases are reminiscent of those described with lesions of the visual association areas (Lance, 1976). It would be reasonable to postulate that the hallucinations arose in these regions, rendered supersensitive by the reduced visual input, and somehow triggered or derepressed by light (Horowitz, 1964). The relationship to light, as well as the tendency to fade as sight was lost, separate these cases from the hallucinations associated with sensory deprivation (Flynn, 1962), where intermittent visual stimulation has not been shown to facilitate 'reported visual sensations'. The hallucinations produced by sensory deprivation and also by drugs tend to begin with non-complex, unstructured forms and then progress to more complex, structured and meaningful imagery (Zuckerman, 1969). Complex imagery was present from the onset of the hallucinations in these three patients.

These phenomena may be more common in the elderly age-group than has been previously appreciated; people are not unnaturally reluctant to recount such experiences for fear of being considered mad. Complex visual hallucinations in the partially blind should not be confused with those arising from a psychotic process or from organic intracerebral disease. They are harmless and appear to fade as sight is finally lost.

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References

Duke-Elder, S. & Scott, G. I. (1974) *System of Ophthalmology*, Vol. XII. London: Henry Kimpton.

Earl, C. J. (1964) Some aspects of optic atrophy. *Transactions of Ophthalmological Societies of the United Kingdom*, **84**, 215–26.

Flynn, W. R. (1962) Visual hallucinations in sensory deprivation. *Psychiatric Quarterly*, **36**, 55–65.

Horowitz, M. J. (1964) The imagery of visual hallucinations. *Journal of Nervous and Mental Disease*, **138**, 513–23.

Lance, J. W. (1976) Simple formed hallucinations confined to the area of a specific visual field defect. *Brain*, **99**, 719.

L'Hermitte, J. (1922) Syndrome de la calotte du pédoncule cérébral (les troubles psychosensoriels dans les lésions du mésencéphale. *Revue Neurologique*, **29**, 1359–65.

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ORMOND, A. W. (1925) Visual hallucinations in sane people. *British Medical Journal*, *iii*, 376–8.

POSEY, W. C. & SPILLER, W. G. (1906) *The Eye and the Nervous System, Their Diagnostic Relations*. Philadelphia: J. P. Lipincott Co.

RUSSELL, W. R. & WHITTY, C. W. M. (1955) Studies in traumatic epilepsy, 3 visual fits. *Journal of Neurology, Neurosurgery and Psychiatry*, **18**, 79–96.

WEINBERGER, L. M. & GRANT, F. C. (1940) Visual hallucinations and their neuro-optical correlates. *Archives of Ophthalmology*, **23**, 166–79.

WEISMAN, A. D. & HACKETT, T. P. (1958) Psychosis after eye surgery. *New England Journal of Medicine*, **258**, 1284–9.

ZUCKERMAN, A. (1969) *Sensory Deprivation. Fifteen Years of Research* (ed. J. P. Zubek). New York: Appleton Century Crofts.

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