Visual Hallucinations: More Diagnoses

To the Editor.—In the discussion of visual disorientation of short duration in a patient with right-sided hemiplegia and hemianopsia,[1] consideration of hemodynamics at the level of the circle of Willis was omitted. If the patient had occlusion of the left internal carotid artery with a patent left posterior communicating artery, transient occipital lobe ischemia may have been produced by a transient reduction of blood flow in the dominant vertebral artery. This may have been due either to mechanical obstruction of the vertebral artery (eg, as a function of head position) or to a reduction in cardiac output (eg, as a result of the upright position, Valsalva's maneuver, or cardiac dysrhythmia).

If the internal carotid artery is occluded and if vertebral angiography shows filling of both the anterior and posterior circulation, brain ischemia with functional impairment is common. Blood flow in the vertebral artery can be maximized by a vein bypass to the ver-

tebral artery at the skull base.[2,3] Under these conditions, vertebral artery reconstruction has improved forebrain and hindbrain perfusion and restored sensory function, including visual perceptive function.

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1. Newmark ME: Visual hallucinations. *JAMA* 1987;257:82.
2. Carney AL, Anderson EM: Carotid distal vertebral bypass for carotid occlusion: Case report and technique. *Clin Electroencephalogr* 1978;9:105-109.
3. Carney AL, Anderson EM: Blood flow to the brain—the direction of change, in Robicsek F: *Extracranial Cerebrovascular Disease: Diagnosis and Management*. New York, Macmillan Publishing Co Inc, 1986, chap 5.

To the Editor.—We must disagree with the answer given in response to Dr Babcock's inquiry to the QUESTIONS AND ANSWERS section about an 80-year-old man with visual hallucinations,[1] or at least suggest another alternative that should be strongly considered.

We believe Dr Babcock's patient probably has the Charles Bonnet syndrome.[2,3] This is a constellation of formed visual hallucinations with visual loss, usually seen in elderly people, characterized by several diagnostic features that seem applicable to the patient under discussion: (1) The hallucinations are exclusively visual and are well formed, such as people and places. (2) They occur with insight and a clear consciousness—the patient is sane and aware he is hallucinating. The hallucinations are fairly devoid of emotional content and not sinister or threatening. (3) They are superimposed on or occur in combination with normal perceptions. The patient can still perceive events around him. (4) They are brief, lasting a few minutes at most. (5) The syndrome is much more common in the elderly. (6) The final and key point is that they occur in the setting of visual loss. Most patients have a visual field defect, and may also have macular degeneration (this patient had both), cataracts, glaucoma, or other causes of poor visual acuity. Although the mechanism of visual hallucinations accompanying visual loss is poorly understood, the phenomenon resembles the hallucinations seen with sensory deprivation.

Education and reassurance, rather than the anticonvulsants suggested, are usually adequate treatments. The hallucinations disappear if visual acuity is improved (eg, by cataract surgery, corrective lenses, or even turning up the lights in the room). It is important to realize that visual hallucinations can occur in elderly patients with poor visual acuity as part of a benign, self-limited syndrome.

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1. Newmark ME: Visual hallucinations. *JAMA* 1987;257:82.
2. Rosenbaum F, Harati Y, Rolak L, et al: Visual hallucinations in sane people: Charles Bonnet syndrome. *J Am Geriatr Soc*, in press.
3. Berrios GE, Brook P: The Charles Bonnet syndrome and the problem of visual perceptual disorders in the elderly. *Age Ageing* 1982;11:17.

In Reply.—The carefully written letters by Dr Carney and Drs Rolak and Baram are appreciated. Concerning the letter by Dr Carney, vascular disease may well be the cause of this man's hemiparesis and symptoms, and was originally mentioned in the discussion. However, with an 80-year-old man with a severe fixed deficit, many physicians would not subject the man to an arteriogram and few would go further and attempt vertebral artery surgery. The value of vascular surgery for patients with cerebrovascular disease has always been speculative; with the combination of age and a severe deficit, I would think that further vascular evaluation would not be rewarding.

Concerning the letter by Drs Rolak and Baram, the Charles Bonnet syndrome is indeed a possibility. With the patient of Dr Babcock's, however, the episodes of visual hallucinations were constant, brief, and stereotyped; they apparently did not vary from episode to episode. In my opinion, this type of attack fits better with a partial seizure than with the type of release phenomenon after sensory deprivation that can be vaguely described and rarely is stereotyped. Reassurance and education alone as suggested by Drs Rolak and Baram might be inappropriate for this man with a severe neurological deficit and repetitive attacks.

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The following letter was received after the author had written the above response.
—ED.

To the Editor.—While Dr Newmark's answer[1] offered a good discussion, I would like to add some comments. Peduncular hallucinations are not synonymous with "release" phenomena. The latter occur with any focal lesion along the visual pathways, and as such have little localizing value, although they are most often placed in the temporal, parietal, and occipital lobes; the release hallucinations are formed, variable in content, and modified by opening or closing the eyes and are seen within the visual field defect.[2-4] Peduncular hallucinations are vivid, animated, short-lived, and often recognized as unreal; most of the patients reported have had signs of brain-stem dysfunction pointing to lesions in this region. As noted, a sleep abnormality is prominent and the hallucinations often occur at a specific time, usually in late daytime or evening. One patient was treated successfully when 5-hydroxytryptophan restored normal sleep.[4,5]

Ictal visual hallucinations are brief, stereotyped, and rarely localized to a portion of the visual field and are not necessarily associated with a field defect. Those occurring in the primary and associative cortex are unformed, while those in more anterior (temporal) areas are more commonly formed remembered scenes. With the latter, in particular, consciousness may be disturbed during or after the ictus and motor phenomena (head or eye deviation). A wide variety of visual distortions are often associated with the seizures. Both ictal and release hallucinations are more common with non–dominant hemisphere foci.[5]

In addition to these entities, ophthalmologic disorders that reduce vision—including cataracts and macular, retinal, or choroidal disease—may produce hallucinations. Although there is some controversy over the use of the term *Charles Bonnet syndrome* to cover these conditions, the onset of hallucinations in the aged should provoke a search for ocular pathology.[4]

In this patient, the phenomena reported probably originate from a combination of the release hallucinations and macular degeneration. As well as the electroencephalogram and computed tomographic scan of the head, neurological and ophthalmologic follow-up would be useful.

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2. Lance JW: Simple formed hallucinations confined to the area of a specific visual field defect. *Brain* 1976;99:719-734.
3. Brust JCM, Behrens MM: 'Release hallucinations' as the major symptom of posterior cerebral artery occlusion: A report of two cases. *Ann Neurol* 1977;2:432-436.
4. Cummings JL: *Clinical Neuropsychiatry*. Orlando, Fla, Grune & Stratton, 1985.
5. Caplan LR: 'Top of the basilar' syndrome. *Neurology* 1980;30:72-79.