With conservative management his double vision improved gradually as the hematoma, documented by CT, resolved. His double vision had disappeared completely by seven months.

COMMENT

A number of mechanisms other than direct orbital injury have been suggested to explain the fourth nerve palsy that may follow severe head injury. The mechanisms include brain-stem contusion or hemorrhage affecting the fourth nerve nucleus or fascicle and compression or stretching of the nerve at the tentorium or at other vulnerable locations along its subarachnoid course.

In this patient, the fourth nerve palsy resulted from minor head trauma. The delayed onset of double vision implies that the fourth nerve was not injured initially, but that a small blood vessel was probably damaged by the tentorium near the posterolateral aspect of the midbrain. The injured blood vessel may have bled slowly or precipitously, causing the "severe headache" that awakened him later. The development of the hematoma may have been exacerbated by the anticoagulants, since previous episodes of spontaneous hemorrhage had occurred when the clotting profile was in the therapeutic range. The hematoma probably compressed the fourth nerve, which winds around the brain stem in this area, without disrupting or severing the fibers, allowing neural function to recover fully as the hematoma resorbed. Such events reemphasize the clinical rule that an ocular motor palsy occurring as a result of "trivial head injury" should alert the clinician to the possibility of a structural lesion. In this case the documentation of a hematoma provides a definitive explanation for posttraumatic fourth nerve in a patient with mild head trauma.

References

1. Rucker CW: The causes of paralysis of the third, fourth and sixth cranial nerves. *Am J Ophthalmol* 1966;61:1293-1298.
2. Khawam E, Scott AB, Jampolsky A: Acquired superior oblique palsy. *Arch Opthalmol* 1967;77:761-768.
3. Burger LJ, Kalvin NH, Smith JL: Acquired lesions of the fourth cranial nerve. *Brain* 1970; 93:567-574.
4. Younge BR: Analysis of trochlear nerve palsies: Diagnosis, etiology and treatment. *Mayo Clinic Proc* 1977;52:11-18.
5. Jefferson A: Ocular complications of head injuries. *Trans Ophthalmol Soc UK* 1961;81:595-612.
6. Neetens A: Extraocular muscle palsy from minor head trauma: Initial sign of intracranial tumor. *Neuroophthalmology* 1983;3:43-48.
7. Walsh FB, Hoyt WF: *Clinical Neuroophthalmology.* Baltimore, Williams & Wilkins Co, 1969, pp 23-90.

Auditory-Visual Synesthesia

Report of a Case With Intact Visual Pathways

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Transformation of a sound stimulus to a visual experience, auditory-visual synesthesia, is a curious phenomenon reported in patients with acquired visual loss involving the anterior visual pathways. We describe a patient in whom a striking auditory-visual synesthesia developed ipsilateral to a large mass involving the medial temporal lobe and the adjacent midbrain. This patient's neuro-ophthalmologic and neurophysiologic examinations did not disclose any evidence of visual dysfunction. The synesthesia disappeared after removal of the mass.

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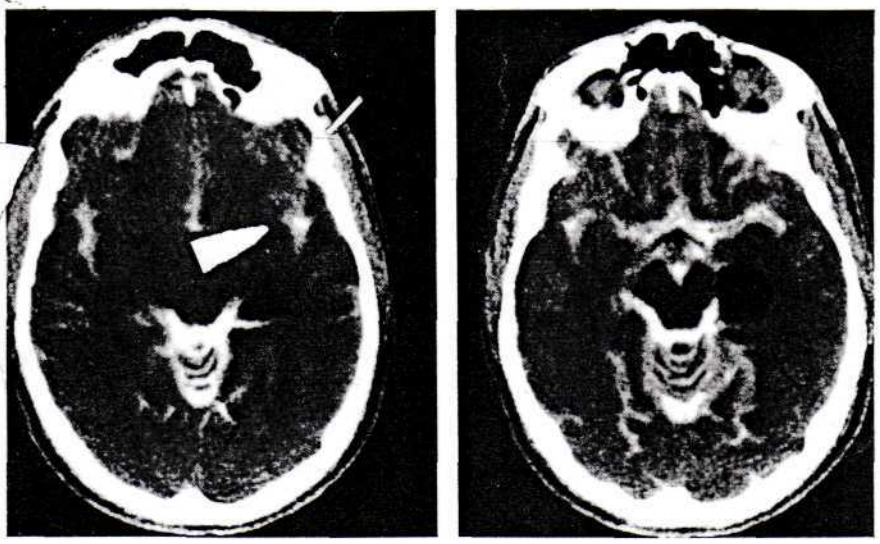
REPORT OF A CASE

A 25-year-old man complained of dull left-sided headaches, weakness and numbness of the left side, and intermittent double vision, which had increased in intensity during a four- to five-week period. During the past two years, he had experienced several episodes during which he saw moving spots of light "like comets" in front of both eyes. General physical examination revealed no abnormality. The neurological examination showed a diminished corneal reflex on the left side, a left-sided peripheral facial weakness, decreased pain sensation on the left side of the face and body, and a mild left hemiparesis. He performed tandem gait poorly and displayed appendicular ataxia that was more pronounced on the left. His visual acuity was 20/15 OU. No afferent pupillary defect was seen. Color plate discrimination and results of the Farnsworth 100-hue test were normal. Direct ophthalmoscopy demonstrated full nerve-fiber bundles. The optic discs and macular reflexes were normal. Tangent screen examination with multiple white and red test objects and Goldmann perimetry showed full visual fields. Saccadic and pursuit eye movements appeared normal. Slit-lamp examination with a Hruby lens showed no evidence of vitreous detachment.

Pattern-reversal visual evoked potentials (VEPs) and brain-stem auditory evoked potentials (BAEPs) were normal. During the BAEP study, at intensities above a 65-dB sensation level (SL), the patient noted a pronounced visual experi-

Visual Experiences Reported by Patient When 65-dB (Sensation Level) Click Was Delivered at Different Rates to Left Ear

Stimulus	Rate, No./s	Visual Phenomena
Click	1.1	Kaleidoscopic image changing with each click, left eye
Click	11.1	Spiraling image: lines, squares converging, in front of left eye.
Click	31.1	Bright lights spiraling "like crazy" in front of left eye
Click	81.1	"Explosion of sunny spots"; "lights coming at 1,000 miles per hour" toward me, left eye, occasionally less intense in right eye



Metrizamide-enhanced computed tomographic scans showing large cystic mass on left side extending from medial temporal region to midbrain.

ence in the left eye. It occurred only with sound presentation to the left ear and increased in intensity with an increasing click rate (Table). The patient ... no demonstrable startle response during the BAEP study. There was no notable deviation of the eyes toward the auditory stimulus, and eye movements in the absence of sound produced no visual experience. We later exposed him to different types of loud sound. He reported a less intense but somewhat similar experience in the left eye after left ear stimulation. An audiogram was normal. A computed tomographic scan showed a large, radiolucent mass within the medial temporal lobe on the left side extending to the midbrain (Figure). A left temporal craniotomy exposed a cystic tumor, and the mass was excised. Histological examination of the tissue disclosed gliosis but no evidence of malignancy. Results of repeated BAEP study one month after surgery were essentially unchanged. The patient did not experience auditory-visual synesthesia during repeated testing.

COMMENT

Auditory-visual synesthesia can occur in normal persons during hypnagogic states.[1] Bender[2] drew attention to the pathological importance of this phenomenon. His patient, a 53-year-old man with a nine-month history of optic neuritis and central scotoma, saw blue lights in the shape of the scotoma each time he heard a loud sound. Lessell and Cohen[3] reported the occurrence of sound-induced phosphenes in optic neuropathy, optic nerve compression and following keratoplasty. They attributed this phenomenon to either postdenervation supersensitivity of bimodal neurons or ephaptic transmission of neural impulses between auditory and visual axons. A more detailed report[4] described nine patients with auditory-visual synesthesia: seven with optic neuropathy and two with chiasmal lesions. All patients displayed evidence of visual field defect on perimetric examination and had abnormal VEPs. Jacobs et al[4] believed synesthesia resulted from postdenervation hypersensitivity of lateral geniculate neurons.

Our patient described his visual experience as always in the eye ipsilateral to the ear that received the auditory stimulus. This relationship was puzzling, since present anatomic knowledge dictates extensive crossing of the auditory fibers in the brain stem and arrangement of postchiasmatic visual pathways by field rather than eye. We noted, however, that 11 of 12 patients of the last two previously mentioned communications[3,4] described their visual experiences similarly. In fact, as with our patient, Jacobs et al[4] experimentally reproduced the ear-eye ipsilaterality in two of their patients.

The data derived from the BAEP test during the past decade indicated existence of an ipsilateral auditory pathway in the human brain stem. Unilateral BAEP abnormalities frequently represent dysfunction of brain stem on the side of the ear being stimulated.[5,6] We believe that the sound stimulus responsible for auditory-visual synesthesia reaches the upper brain stem through the same ipsilateral pathway. There, through intercollicular or intergeniculate connections, it contacts the visual system, where it stimulates the bimodal neurons.[7,8] Those bimodal neurons receiving primarily this ipsilateral auditory input may receive their major visual input from the ipsilateral retina. Under pathological conditions, direct irritation of these neurons or their supersensitivity due to deafferentation results in ipsilateral auditory-visual synesthesia.

Our patient demonstrated the following three special features:

1. The patient's auditory-visual synesthesia was caused by a tumor on the same side. This finding supports the notion of ipsilateral convergence of auditory and visual stimuli at the mesencephalic-diencephalic region.

2. Unlike previously described patients, he showed no evidence of visual system dysfunction on neuro-ophthalmologic testing.

3. Increasing the rate of auditory stimulus clearly increased the intensity of his visual experience, a finding that indicates presence of "rate responsive" auditory-visual units in human beings. Our patient's earlier visual experiences can be best explained as spontaneous photisms, resulting from either disturbance of temporal cortex[9] or mesencephalic-diencephalic regions.[10]

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References

1. Edbecke U: Uber ein entoptisenes Phänomen bei Schreck. *Klin Monatsbl Augenheilkd* 1943;109:190-193.

2. Bender MB, Rudolph SH, Stacey CB: The neurology of visual and oculomotor systems, in Baker AB, Baker LH (eds): *Clinical Neurology*. Hagerstown, Md, Harper & Row Publishers Inc, 1982, chap 4, vol 1, p 37.

3. Lessell S, Cohen MM: Phosphenes induced by sound. *Neurology* 1979;38:1524-1527.

4. Jacobs L, Karpik A, Bozian D, et al: Auditory-visual synesthesia: Sound-induced photisms. *Arch Neurol* 1981;38:211-216.

5. Stockard JJ, Stockard JE, Sharbrough FW: Brain stem auditory evoked responses in neurology, in Aminoff MJ (ed): *Electrodiagnosis in Clinical Neurology*. New York, Churchill Livingstone, 1980, pp 370-413.

6. Chiappa KH: Brain stem auditory evoked potentials, in Stalberg E, Young R (eds): *Clinical Neurophysiology*, ed 1. Washington, DC, Butterworths Inc, 1981, pp 259-277.

7. Stein BC, Arigbede MO: Unimodal and multimodal response properties of neurons in the cat's superior colliculus. *Neurology* 1972;36:179-196.

8. Beteleva TG: Responses of the rabbit's lateral-geniculate body to sound stimuli and to electrical stimulation of the reticular formation of the brain stem, in Sikolou ER, Vinogradova OS (eds): *Neuronal Mechanisms in the Orienting Reflex*. Hillsdale, NJ, Lawrence Erlbaum Associates Inc Publishers, 1975.

9. Van Boagaert L: Sur les hallucinations visuelles au cours des affections organiques du cerveau (contributions a l étude du syndrome des hallucinations lilliputiennes). *Encephale* 1926;21:657-679.

10. Dunn DW, Weisberg LA, Nadell J: Peduncular hallucinations caused by brainstem compression. *Neurology* 1983;33:1360-1361.