ripheral airway narrowing commonly observed in individuals with asthma (*12*). The paroxysmal cough response to LTC in two subjects (*7*) is inexplicable in view of the lack of cough in the five subjects reported here.

When prostaglandin $F_{2\alpha}$ was administered to normal subjects by means of an aerosol delivery system similar to that used in this study, a dose of 1200 μg was required to produce an 18 percent decrease in the forced expiratory volume in the first second and symptoms of cough and throat irritation; but no normal subject experienced wheezing or shortness of breath (*13*). In contrast, each of our five subjects experienced a 40 to 50 percent decrease in $\dot{V}_{30}$ and audible wheezing after exposure to less than 20 μg of LTC and were free of cough. Thus, greater potency, a slow and prolonged time-course of action, and a marked peripheral airway effect without signs of upper airway irritation, distinguish LTC from histamine and prostaglandin $F_{2\alpha}$ and are consistent with a role for LTC as a major mediator of allergic airway constriction.

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References and Notes

1. D. Webb-Johnson and J. C. Andrews, Jr., *N. Engl. J. Med.* **297**, 476 (1977); *ibid.*, p. 758.
2. W. E. Brocklehurst, *J. Physiol. (London)* **151**, 416 (1960); *ibid.* **129**, 16P (1953); G. K. Adams III and L. M. Lichtenstein, *J. Immunol.* **122**, 555 (1979); K. F. Austen and R. P. Orange, *Am. Rev. Respir. Dis.* **112**, 423 (1975).
3. R. C. Murphy, S. Hammarstrom, B. Samuelsson, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 4275 (1979).
4. E. J. Corey, D. A. Clark, G. Goto, A. Marfat, C. Mioskowski, B. Samuelsson, S. Hammarstrom, *J. Am. Chem. Soc.* **102**, 1436 (1980).
5. R. A. Lewis, K. F. Austen, J. M. Drazen, D. A. Clark, A. Marfat, E. J. Corey, *Proc. Natl. Acad. Sci. U.S.A.* **77**, 3710 (1980); L. Orning, S. Hammarstrom, B. Samuelsson, *ibid.*, p. 2014; R. A. Lewis, J. M. Drazen, K. F. Austen, D. A. Clark, E. J. Corey, *Biochem. Biophys. Res. Commun.* **96**, 271 (1980); C. W. Parker, D. Koch, M. M. Huber, S. F. Falkenhein, *ibid.* **97**, 1038 (1980).
6. J. M. Drazen, K. F. Austen, R. A. Lewis, D. A. Clark, G. Goto, A. Marfat, E. J. Corey, *Proc. Natl. Acad. Sci. U.S.A.* **77**, 4354 (1980); P. Hedqvist, S.-E. Dahlen, L. Gustafsson, S. Hammarstrom, B. Samuelsson. *Acta Physiol. Scand.* **110**, 331 (1980).
7. M. C. Holroyde, R. E. C. Altounyan, M. Cole, M. Dixion, E. V. Elliott, *Lancet* **1981-II**, 17 (1981).
8. J. A. Nadel and D. F. Tierney, *J. Appl. Physiol.* **16**, 717 (1961).
9. P. J. Despas, M. Leroux, P. T. Macklem, *J. Clin. Invest.* **51**, 3235 (1972).
10. R. R. Rosenthal, J. E. Fish, S. Permutt, H. Menkes, P. S. Norman, *J. Allergy Clin. Immunol.* **57**, 220 (1976); C. J. Kim, *J. Allergy* **36**, 353 (1965).
11. N. E. Brown, E. R. McFadden, Jr., R. H. Ingram, Jr., *J. Appl. Physiol. Respir. Environ. Exercise Physiol.* **42**, 508 (1977); T. Higgenbottam, *ibid.* **49**, 403 (1980).
12. E. R. McFadden, Jr., *J. Allergy Clin. Immunol.* **56**, 18 (1975).
13. H. H. Newball and C. J. Lenfant, *Respir. Physiol.* **30**, 125 (1977).
14. This work was supported in part by grants AI-00399, AI-07722, AI-10356, AM-05577, HL-00549, HL-17382, HL-19777, and RR-05699 from the National Institutes of Health; in part by a grant from the Lillia Babbitt Hyde Foundation; and in part by the National Science Foundation. J.W.W. is a recipient of a fellowship from the Parker B. Francis Foundation.
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Oculoparalytic Illusion: Visual-Field Dependent Spatial Mislocalizations by Humans Partially Paralyzed with Curare

Abstract. *In darkness, observers partially paralyzed with curare make large (> 20 degrees) gaze- and dosage-dependent errors in visually localizing eye-level-horizontal and median planes, in matching the location of a sound to a light, and in pointing at a light. In illuminated, structured visual fields visual localization and pointing are accurate but errors in auditory-to-visual matches remain. Defects in extraretinal eye position information are responsible for all errors. The influence of extraretinal eye position information on visual localization is suppressed by a structured visual field but is crucial both in darkness and for intersensory localization if visual capture is prevented.*

Shifts of the image at the back of the eye are produced either by eye movements or by displacements of the visual field of view. Although displacements of the visual field are normally perceived to be displacements, in the presence of eye movements stationary visual fields continue to appear stationary. The observer's use of extraretinal eye position information (EEPI) (*1*) is somehow involved in the difference. Theoretical treatments of EEPI have been unsuccessful in accounting either for the precision and accuracy of visual localization generally or in predicting localization errors (*1*). To further examine the influence of EEPI on spatial localization we attempted to modify the normal quantitative relations between gaze direction and EEPI (whether derived from motor commands directing gaze or proprioceptive feedback from the orbit) by reversibly weakening the extraocular muscles of five adult male human observers through systemic injection of *d*-tubocurarine (*2*). This produced large errors in visual, intersensory, and sensorimotor localizations with magnitudes that systematically depend on degree of muscular weakness, direction of ocular gaze with respect to the head, and most important, the presence or absence of a structured visual field. We have called this the oculoparalytic illusion (OPI) and describe it below.

When the reclining, partially paralyzed observer (Fig. 1g) fixated a single stationary visual target (*3*) at eye level in normal illumination, the target appeared to lie at eye level as it did to unparalyzed subjects. However, as soon as all room illumination was extinguished, the partially paralyzed observer (but not the unparalyzed observer) saw the fixation target slowly descend to a position near the (invisible) floor. Normal illumination immediately restored the appearance of the target to eye level again. This sequence could be repeated as often as desired. When the fixated light was vertically positioned in darkness to a height which the observer reported to be eye-level-horizontal ("one-light experiment"), the settings (*4*) were more than 0.6 m above true horizontal—more than a 20° error at the viewing distance of 1.83 m. When the observer viewed from a position in which his head-and-body were tilted forward the effect was reduced. In a position of still greater forward tilt the light fixated at eye level appeared to ascend to a position near the (invisible) ceiling when room illumination was extinguished; vertical settings of a fixated target to

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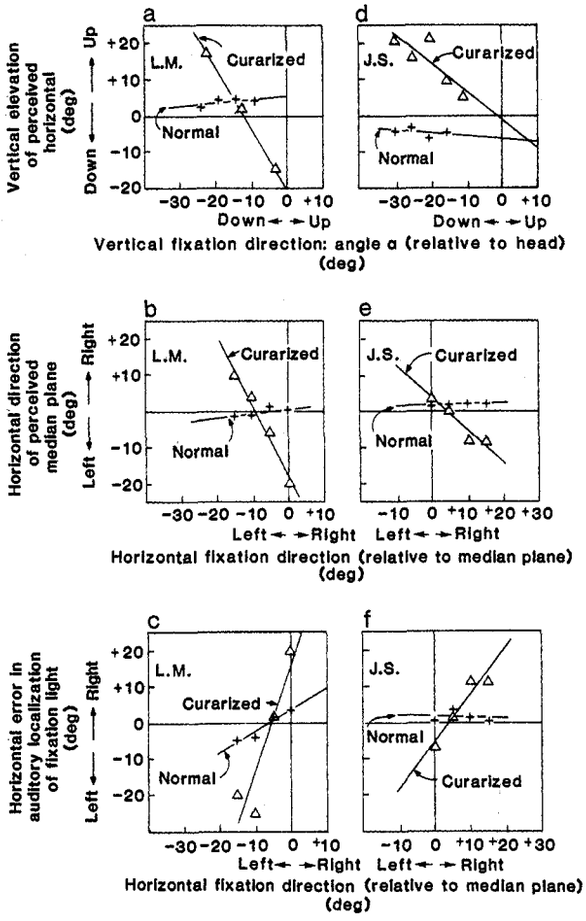
appear at eye-level-horizontal were now more than 0.6 m below true horizontal—again more than 20° error (*5*).

Subsequent experiments separated the influence of the angle of the eye in the head (angle α in Fig. 1g) from the angular relation of the head-and-body to gravity (angle β), and demonstrated that the illusion was determined by α only. In those experiments the observer set a movable, peripherally viewed visual target (*3*) to the perceived eye-level-horizontal with α established by the vertical position of a separate foveally fixated visual target ("two-light experiment"). That only α and not β determined the sign and magnitude of the illusion was demonstrated as follows: (i) The error in setting the second light to the perceived eye-level-horizontal in darkness was linearly related to α (Fig. 1, a and d); this relation did not change with different settings of β. These systematic errors were not made by the partially paralyzed observer in normal room illumination or by the unparalyzed observer either in illumination or in darkness. (ii) When the results from the one-light experiment were transformed to the coordinates of Fig. 1a they were indistinguishable from those of the two-light experiment at the same level of paralysis. (iii) The no-illusion direction (NID)—the angle of the eye in the head for which no drop or rise of a fixated visual target was perceived when illumination was extinguished—was also the direction of gaze for which the setting of the visual target to the perceived eye-level-horizontal was most accurate for both one- and two-light experiments. (iv) The NID was the same for all head-and-body tilts. (v) Partially paralyzed subjects pointed accurately to the proprioceptive horizontal in darkness or with eyes closed; they also pointed accurately at the horizontal direction when pointing toward a visual target located at the physical eye-level-horizontal in normal room illumination (*6*), but they pointed more than 20° downward (or upward) when pointing at a visually fixated target viewed in darkness which was physically at eye level but appeared to lie near the floor (or the ceiling); pointing errors were less for gaze directions at which visual errors were smaller (*7*). Thus, we conclude that although perception of the eye-level-horizontal must involve information about both angles α and β, the paralyzing procedure only distorts EEPI about α (*8*).

Similar results were obtained when we measured the influence of muscle weakening on visual spatial localization for horizontal changes in gaze direction in a two-light experiment. The subject set a movable, peripherally viewed visual target to the position along a horizontal arc that appeared to intersect the median plane of his body while his horizontal direction of gaze was fixed by the horizontal position of the foveally fixated target. As with the perceived eye-level-horizontal, neither paralyzed nor unparalyzed subjects made errors in setting the visual target to the perceived median plane when fixating at different eccentricities of gaze in normal illumination.



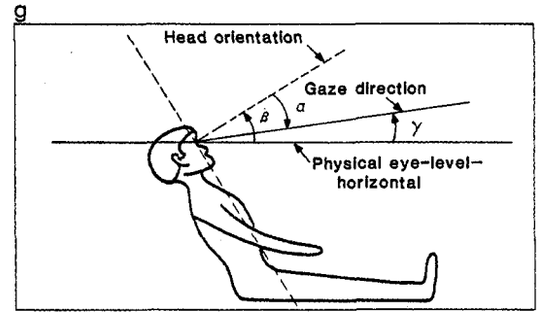


Fig. 1. Psychophysical localization measurements by observers L.M. and J.S. of perceived eye-level-horizontal [(a) and (d)], perceived median plane [(b) and (e)], and auditory-to-visual matches [(c) and (f)]. Crosses show control measurements obtained before curare administration; triangles show measurements during paralysis. Each point is an average of two or three settings. The two lines through the data in each graph are least-squares fits. (a) and (d) In complete darkness the observer fixated a small visible target whose angular elevation with respect to the transverse plane through the head is plotted on the abscissa: this elevation defined the vertical angle of the eye in the head. The transverse plane through the head was itself above the physical horizontal by about 25° for L.M. and 30° for J.S. While maintaining fixation on this first light the subject set a second peripherally viewed light (which was movable in the same vertical meridian as the first target) to a height that appeared to be at his eye-level-horizontal; this last height is plotted on the ordinate as vertical elevation of perceived horizontal. (b) and (e) The observer fixated a small visible target in complete darkness whose horizontal deviation from the physical median plane through his body is plotted on the abscissa. This angle defined the horizontal angle of gaze. While maintaining fixation on this first light, the subject instructed the experimenter to set a second visual target (which was movable in the same horizontal plane as the first target) to the perceived median plane. This latter setting is plotted as the ordinate. (c) and (f) The observer matched the perceived horizontal location of a sound to that of a fixated light. The abscissa is the physical location of the fixated light with respect to the median plane. The ordinate is the error in the auditory localization of the fixated light—the difference between the point of subjective equality of the auditory localization of the fixated light and the physical location of that light. For both (c) and (f) room illumination was left on. The results are indistinguishable when the experiment is carried out in total darkness. (g) The relation between the vertical fixation direction (gaze direction) relative to the head (angle α), the orientation of the head with respect to the physical horizontal (angle β), and the direction of gaze with respect to the physical horizontal (angle γ).

Table 1. Dependence of localization errors of oculoparalytic illusion (OPI) on presence or absence of visual field for the partially paralyzed observer.

Condition of room illumination	Visually perceived median plane setting	Visually perceived eye-level-horizontal setting	Auditory-to-visual match of localizations	Naming auditory directions*	Pointing to proprioceptive horizontal	Pointing to a visual target
Total darkness	Gaze-dependent errors	Gaze-dependent errors	Gaze-dependent errors	Accurate	Accurate	Gaze-dependent errors
Structured visual field	Accurate	Accurate	Gaze-dependent errors	Accurate	Accurate	Accurate†

*See (*9*) †See (*6*).

However, in total darkness partially paralyzed subjects (but not unparalyzed subjects) made errors that increased linearly with horizontal eccentricity of gaze (Fig. 1, b and e). Increased rightward deviation of the direction of fixation resulted in settings that were increasingly to the left of the true median plane, and vice versa. In contrast to the specification of the eye-level-horizontal by its relation to the direction of gravity, the median plane is defined only with respect to body-centered coordinates and not with respect to an external direction such as is defined by gravity. Thus there can be no variable analagous to body tilt involved in determination of the visually perceived median plane, and the median plane settings show in isolation the consequences for perception of the modification of EEPI produced by partial paralysis.

Auditory localization is not influenced by systemic curare (*8–10*) and thus provides an undisturbed pointer with which to measure the curare-induced disturbances in visual localization. The observers matched the horizontal location of a sound to that of a fixated visual target (*4, 11*). The systematic departure of the matches of the partially paralyzed observers from baseline measurements without paralysis are shown in Fig. 1, e and f, where the error in setting the light to the sound is plotted against gaze eccentricity. These errors varied linearly with eccentricity of gaze; when the measurements are recalculated as a deviation from the NID, the eccentricity of the loudspeaker exceeds the eccentricity of the visual target to which it is matched. For a given eccentricity of gaze, the errors in matching sound and light were roughly the same as the errors in setting a visual target to the median plane. This is consistent with the interpretation that the entire systematic error in the auditory-to-visual match is a consequence of a paralysis-induced change in localization of the visual target and that the auditory localization of the loudspeaker is itself unaffected.

The auditory-to-visual matches were entirely unaffected by the presence or absence of illumination, a result markedly different from the measurements of the perceived eye-level-horizontal and median planes (Table 1). This provided the following paradox: In normal illumination the paralyzed observer correctly set a visual target to his median plane regardless of gaze eccentricity, suggesting that visual localization is correct within the horizontal plane; yet in matching the horizontal location of a sound to a light he made large errors that depended on horizontal gaze direction. A coherent explanation of the results—including the paradox—is provided by the following three points.

1) The gaze-dependent mislocalizations in darkness are a consequence of curare-induced errors in EEPI as established above. However, settings to the median plane and to the eye-level-horizontal were both accurate in the presence of a visual field; thus we conclude that EEPI is not involved in visual localization there (*12*). Assuming that a structured, normally illuminated visual field suppresses EEPI (while providing the basis itself for visual localization) explains the elimination of visual mislocalizations by the illuminated field but fails to explain the continued existence of errors in matching locations of auditory and visual targets in normal illumination. Interpretations based on errors in processing the auditory stimulus per se are ruled out (*8–10*). The paradox is resolved if EEPI itself is not suppressed, but instead its utilization is suppressed by the visual field for only some classes of perceptual judgments (*13*).

2) The slopes of the linear functions relating the three kinds of localization errors (Fig. 1) to gaze eccentricity are indistinguishable at a given level of paralysis. These slopes all increase in the same way with level of paralysis, supporting the conclusion that all three kinds of errors are mediated by the same curare-induced deficits in EEPI. This also suggests that at a given level of paralysis the EEPI signaling a given eccentricity of gaze has been scaled up by a factor k from the unparalyzed state, where the slope of the function relating error to gaze eccentricity is equal to $k - 1$ and its value increases with level of paralysis (when $k = 1$, slope $= 0$, and errors do not increase with eyeturn). This is consistent with the existence of a linear relation between visual localization and extent of the intended eyeturn. It is also consistent with our observations (i) that the range of gaze directions over which subjects could turn their eyes decreased with increase in level of paralysis, and (ii) that although the limits of gaze eccentricity are increasingly reduced with increasing levels of paralysis, the subjects' impressions were that the maximum effort exerted at these limits was the same at all levels of paralysis.

3) We were able to use audition to uncover the presence of deficits in EEPI in normal illumination only because we eliminated the influences on auditory localization arising from the visual field which are normally very strong determinants of auditory localization. The elimination was effected by presenting the auditory stimulus on each trial from one of 25 loudspeakers in an array that provided no visual clues to indicate which loudspeaker emitted the sound (*14*).

The oculoparalytic illusion is consistent with either outflow or hybrid theory, but not inflow theory because, although curare affects the cholinergic mechanism for mediation of efferent control of muscle spindles as well as for extrafusal muscle, it does not directly affect sensory receptors or nerve fibers. Under total extraocular muscle paralysis observers in three laboratories (*15*) have reported no visually perceived changes, when they attempted to turn their eyes, a result which appears to contradict outflow theory. However, those observers only viewed in well-illuminated visual fields, the condition that we have shown suppresses the influence of EEPI on visual localization. The present results show that it will now be necessary to reexam-

ine the total paralysis situation in darkness as well as in well-illuminated visual fields in order to decide on the source of EEPI.

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References and Notes

1. Extraretinal eye position information (EEPI) refers to any information which the observer has about the orientation of his eye in the orbit that does not arise from visual stimulation of the retina by light. EEPI has been considered to originate in (i) neural centers generating the command to turn the eyes (outflow theory), (ii) sense organs in extraocular musculature or retrobulbar tissue (inflow theory), or (iii) sense organs in extraocular musculature where the afferent signals are modulated by efferent signals or from other more central regions generating feedback regarding the efficacy of the command (hybrid theory) [for reviews, see L. Matin, in *Handbook of Sensory Physiology*, vol 7, part 4, *Visual Psychophysics*, D. Jameson and L. M. Hurvich, Eds. (Springer-Verlag, Heidelberg, 1972), pp. 331–380; *Perception* **5**, 233 (1976); in *Tutorials on Motion Perception*, A. Wertheim, W. A. Wagenaar, H. W. Leibowitz, Eds. (Plenum, New York, in press)].
2. A total of ten sessions was run on the five subjects: four on L.M., two each on J.S. and D.Y., and one each on E.P. and R.M. *d*-Tubocurarine was given in doses of 0.05 mg/kg every 5 minutes until the desired level of paralysis was achieved and then continuously infused at a rate of about 9 mg/hour for a total of 20 to 30 mg during the 2- to 4-hour session. At the level of paralysis used during quantitative psychophysical work speech was barely audible, the arm could barely be raised from the lap, expiratory vital capacity was decreased to about 85 percent of normal, and heart rate and blood pressure were unchanged from baseline. Breathing was entirely unassisted. Fluctuations in level of paralysis during a session produced influences on our results that were small relative to the size of the main effects. Glycopyrrolate (0.4 mg) was administered before curare to reduce autonomic effects; neostigmine was used to reverse the paralysis. Ptosis and diplopia were present throughout and subjects only viewed monocularly (the other eye was occluded by an eyepatch). Human Subject committees at both Columbia University and the University of Pennsylvania approved the protocols and procedures. The experiments were carried out at the anesthesiology department of the University of Pennsylvania Medical School.
3. The fixation target used in a number of experiments was a transilluminated "E" (24 by 24 minutes of arc, visual angle) that appeared indistinct or totally unresolvable when viewed with peripheral vision at more than 2° to 4° off central fovea. Thus, although we did not monitor eye position directly, we were well informed regarding whether or not foveal fixation was at least roughly maintained when required. Good foveal fixation was maintained throughout all measurements reported here. Each of the five other movable visual targets was a transilluminated circular disk, 9 minutes of arc in diameter. All targets were viewed from a distance of 1.83 m.
4. Settings were made by the experimenter positioning a visual target on oral command (grunts) of the subject. In experiments involving auditory targets an adaptive psychophysical procedure was used in which the experimenter switch-controlled the loudspeaker location from an adjacent room in response to pushbutton signals by the subject until the subject indicated that a sound-to-light localization match was reached (reliability of the match setting was 2°). When the subject was too weak to depress the pushbuttons, a second experimenter (always in the room with the subject) depressed the pushbuttons in response to oral commands by the subject.
5. Each of the sets of measurements described herein were carried out on at least the two observers L.M. and J.S.; in most cases all five observers were examined. The main features described for each type of measurement were consistent for all observers.
6. When the visual field was illuminated, the subject viewed the target, then closed his eyes, and finally pointed at the previously viewed target with eyes closed. Although our procedure for measuring the direction of finger pointing was not extremely accurate, it is clear that errors of the subject in this condition were no more than 2° to 3°.
7. Since the muscles guiding the finger were substantially weakened, the essential accuracy with which observers pointed to the horizontal in darkness implies that an inflow source is involved in sensing finger orientation; this must be in addition to whatever contribution is made by the vestibular organs.
8. Any small contribution to our results due to modification of neck and trunk proprioception by curare is below the level of precision of our measurements. It is unlikely that either the vestibular system or the inner ear (efferents as well as afferents)—both lying within the blood-brain barrier—were affected at all [see R. Klinke and N. Galley, *Physiol. Rev.* **54**, 316 (1974); P. S. Guth, C. H. Norris, R. P. Bobbin, *Pharmacol. Rev.* **28**, 95 (1976)]. In a study in which an increase in the sensitivity for detecting curare in cerebrospinal fluid was achieved by radioimmunoassay, only infinitesimal amounts were reported to pass the blood-brain barrier [R. S. Matteo, E. K. Pua, H. J. Khambatta, S. Spector, *Anesthesiology* **46**, 396 (1977)]; previous less sensitive methods had not detected any passage at all.
9. A separate psychophysical procedure (name-the-speaker) provides strong evidence that curare does not induce errors in auditory localization: The 25 loudspeakers were sequentially numbered and the observer in total darkness reported the speaker number on a series of trials in which order of presentation of the speakers was random. The observer's accuracy was not influenced by paralysis.
10. Although curare does not reach the inner ear or higher reaches of the auditory nervous system [see (*8*)], it does reach the tympanic and stapedius muscles in the middle ear (which lie outside the blood-brain barrier) and produces a diminution of the acoustic reflex [R. Smith, M. Loeb, J. L. Fletcher, D. M. Thomas, *Acta. Oto-Laryngol.* **62**, 101 (1966); R. A. Ruth, M. E. Johns, T. J. Gal, *Ann. Otol. Rhinol. Laryngol.* **89**, 188 (1980)], a result that is likely to be bilaterally symmetric and hence unimportant for the present purposes.
11. The auditory stimulus was a 64-cycle segment of a sine wave periodically repeated three times a second. To eliminate any possible localization cues that might originate in differences between loudspeakers, the frequency of the sine wave was quasi-randomly varied from trial to trial between 2 and 4.5 kHz (no significant effects of frequency on the localization match occur in this range); the duration of each burst was thus quasi-randomly varied between 14 and 32 msec.
12. This conclusion is most clearly drawn by noting that for two different gaze directions the error in auditory-to-visual matches may differ by 50°, implying a difference in error of EEPI of about 50°; nevertheless, in the presence of a structured visual field for both gaze directions visual localization (that is, perceived median plane and eye-level-horizontal settings) is accurate.
13. For a detailed discussion of the paradox, see L. Matin, J. Stevens, E. Picoult, in *Spatially Oriented Behavior*, A. Hein and M. Jeannerod, Eds. (Springer-Verlag, New York, 1982).
14. Under curare visual capture continued to function normally for other auditory stimuli (for example, when the experimenter spoke, the partially paralyzed subject who viewed him with eccentric gaze heard the speech as emanating from the experimenter's mouth). Thus it was our procedure for presenting stimuli that eliminated visual capture of the auditory stimuli emanating from the loudspeakers, not the curare.
15. J. K. Stevens, R. C. Emerson, G. L. Gerstein, T. Kallos, G. P. Neufeld, C. W. Nichols, A. C. Rosenquist, *Vision Res.* **16**, 93 (1976); G. S. Brindley, G. M. Goodwin, J. J. Kulikowski, D. Leighton, *J. Physiol. (London)* **258**, 65P (1976); R. Siebeck, *V. Graefes Archiv. Ophthalmol.* **155**, 26 (1954).
16. Supported by NIH grant EY 03198 and award N62269-80C-0296 from the Naval Air Development Center. A description of these experiments was presented at the 1980 meeting of the Association for Research in Vision and Ophthalmology in Orlando, Fla. [April Supplement to *Invest. Ophthalmol. Vis. Sci.* **19**, 81 (Abstr.) (1980)].

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Plant Phenols Utilized as Nutrients by a Phytophagous Insect

Abstract. *Phenols are commonly regarded as feeding deterrents for phytophagous insects, but the tree locust* Anacridium melanorhodon *survives better and grows faster when certain phenols are added to a food plant that is relatively low in both protein and phenols. The phenols are at high concentration in the common host plants. Much of the phenol retained by the insect becomes bound in the cuticle where it probably stabilizes the protein.*

Plant phenols are commonly regarded as allelochemicals that are deterrent or deleterious to phytophagous insects (*1*). Tannic acid, however, has been shown to improve growth of the tree locust *Anacridium melanorhodon* (Walker) (*2*). Since tannic acid is hydrolyzed in the gut of locusts to gallic acid (*3*), the growth of nymphs was examined by feeding lettuce with and without the addition of gallic acid or related phenols found in host plants.

The lettuce leaves contained approximately 20 percent protein and 0.2 percent phenols (dry weight) (*4*), and the major phenols present were caffeic, protocatechuic, and gentisic acids. Phenols were added by dipping lettuce in alcoholic solutions of protocatechuic acid, gallic acid, ferulic acid, or caffeic acid. After evaporation of the solvent, measured quantities of leaf material with known weights of added phenol were fed to insects for the last two nymphal instars. Addition of protocatechuic, gallic, or caffeic acids significantly increased the survival and growth rates above that of the controls, whereas addition of ferulic

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