and thickness, such as 12-in. lengths of Whatman No. 1 paper, the differences are virtually negligible. Furthermore, the deviation is not cumulative, and it is no greater for a single one than for any one of a number of similar strips taped together end to end.

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References and Notes

1. This work was supported by grants from the U.S. Public Health Service and the National Science Foundation. The help of Carl F. Schuster of the technical apparatus shop is gratefully acknowledged.
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12 October 1959

Luminosity Losses in Deuteranopes

In 1958 Heath (*1*) published some luminosity curves for normal and dichromatic subjects. The luminosity values are given as the reciprocals of relative energies of different wavelength bands required to provide a critical flicker frequency of 20, 25, 30, or 40 flashes per second.

Heath found the usual luminosity loss for protanopic subjects as contrasted with normal subjects. However, his findings for deuteranopes were unusual. Instead of a luminosity loss in the blue and green regions of the spectrum, he found a luminosity gain for deuteranopes in the spectral region from about 520 mμ into the red beyond 700 mμ.

The results obtained on deuteranopes by Heath with the critical flicker measurements are not in accord with some recent foveal luminosity measures based on foveal threshold determinations (*2*) and on the determination of luminosities for different levels of visual acuity (*3*) [see also the data obtained by Boynton *et al.* (*4*) on single dichromats by a rapid chromatic adaptation method].

The fact that Heath's results may not be in accord with those obtained by the other methods may be due to a number of factors, including the possibilities (i) that the method involving equality of flicker may provide conditions that give rise to special wavelength effects, and (ii) that Heath's criteria for the selection of deuteranopes were different from those used by the other investigators. We have found a heightened sensitivity comparable to that reported by Heath for wavelengths longer than about 500 mμ in two subjects, one protanomolous, the other deuteranomolous. Such cases merit further study, and flicker criterion data should be correlated with data obtained by other methods.

Heath's conclusions, taken at face value, seem to bolster a position upheld by Walls (*5*), among others—that is, that luminosity losses do not occur in deuteranopes. The evidence cited here indicates that deuteranopes can, and that many do, lose luminosity in the green and blue regions of the spectrum.

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2. S. Hecht and Y. Hsia, *J. Gen. Physiol.* **31**, 141 (1947); E. N. Willmer, *J. Physiol. (London)* **110**, 422 (1949); *Documenta Ophthalmol.* **3**, 194 (194→ Y. Hsia and C. H. Graham, *Proc. Natl. Acad. Sci. U.S.* **43**, 1011 (1957); C. H. Graham and Y. Hsia, *Science* **127**, 675 (1958); J. Zanen, R. Wibail, A. Meunier, *Bull. mém. soc. franç. ophtalmol.* **70**, 81 (1957); W. E. Collins, *J. Psychol.* **48**, 285 (1959).
3. G. Verriest, *Ann. optique oculaire* **4**, 53 (1958).
4. R. M. Boynton, G. Kandel, J. W. Onley, *J. Opt. Soc. Am.* **49**, 654 (1959).
5. G. L. Walls has argued [*A.M.A. Arch. Ophthalmol.* **62**, 13 (1959); *Am. J. Ophthalmol. Monograph No. 233* (1958), p. 1] on the basis of this belief that the unilaterally colorblind subject described by us [*Science* **127**, 675 (1958)] who has normal vision in her right eye and dichromatic vision in her left and shows in the left eye a considerable loss of luminosity in the blue and green, could not be a deuteranope.

   We do not say in any of our more complete accounts that her left eye is unqualifiedly deuteranopic; her hue discrimination is, in fact, atypical in the blue and green. [We did refer to her "deuteranopic" eye in two early abstracts: *Science* **120**, 780 (1954) and *J. Opt. Soc. Am.* **45**, 407 (1955).] We do, however, emphasize the fact that her dichromatic eye shows many of the characteristics of deuteranopia (including possible luminosity loss), and we have thought it useful to consider principles based on her case that might apply to this form of color blindness.

24 October 1959

The question of whether deuteranopes show losses or gains in luminosity, quite apart from the two possibilities suggested by Graham and Hsia, may lie in one's definition of "luminosity." Curves of reciprocals of foveal threshold energies—that is, cone sensitivity curves—representing the *envelope* of the individual sensitivity curves of only the most dark-adaptable of the several receptor types, cannot be regarded as "luminosity" curves in the same sense as curves of reciprocals of energies required for a given photopic effect [for example, a constant brightness, or a particular critical frequency of flicker fusion (CFF)] where interaction, summation, inhibition, and adaptive effects may markedly alter the respective contributions of each type of receptor (*1*). The "special wavelength effects" of the tremendous difference in adaptation levels between threshold and photopia are by no means yet fully known, but interpretation of threshold data in terms of luminosity losses or gains would imply that, whatever the adaptive effects, they must be independent of wavelength, so that an observer's threshold sensitivity curve would be identical in shape to his photopic luminosity curve. A mass of evidence to the contrary exists (*2*). Moreover, the threshold method requires the further assumption that the relationship between the thresholds of normal, protanope, and deuteranope subjects is the same as the relationship beween their photopic luminosities—that is, that the rate of increase in subjective brightness with increased stimulus intensity is identical for all observers as well as for all wavelengths. Figure 1 demonstrates the changing relations found among our observers at photopic levels with the CFF method, and the results of such changes if extrapolated to the "cone threshold" level—results which resemble those found by Hecht and Hsia (*3*).

With regard to the selection of sub-

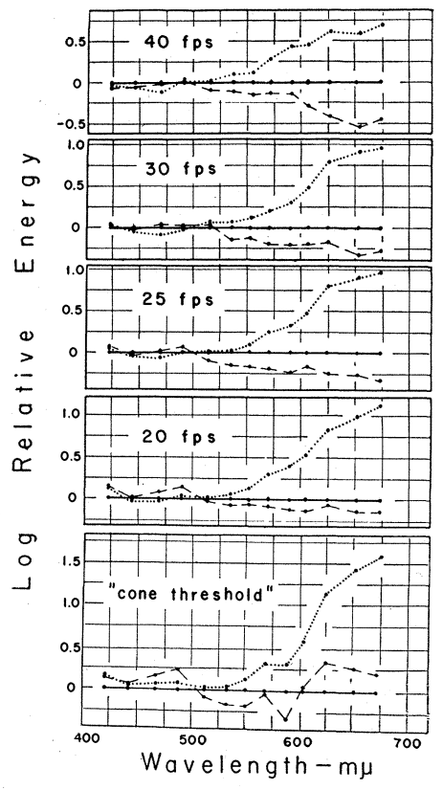

Fig. 1. Logarithms of the ratios of spectral energy requirements of protanopes (dotted lines), normal subjects (solid lines), and deuteranopes (dashed lines), to the normal requirements for four flicker-fusion frequencies. "Cone threshold" data were derived by linear extrapolation to the zero CFF level.

jects, the criteria (*4*) used were perhaps more rigorous than those employed by some other investigators (*5*). Concordance of the results of the selection tests could not, of course, rule out the possibility that the six deuteranopes and nine normal subjects were not truly representative of their respective populations in sensitivity and luminosity. In this regard it is of interest to note that Boynton's (*6*) normal subject was quite unlike Hsia and Graham's normal subjects and that his deuteranope and protanope showed virtually identical foveal thresholds, from 410 to 540 mμ, in marked contrast to the loss of sensitivity of deuteranopes relative to protanopes in this spectral region found by Hsia and Graham, but in excellent agreement with our (photopically determined) identity of this section for all subjects at all brightness levels.

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References and Notes

1. The intensitive variations of receptor contributions are evident in the drastic color changes of lights of high brightness [E. Auerbach and G. Wald, *Science* **120**, 401 (1954)] and in the not-so-drastic color changes of the Bezold-Brücke phenomenon. This was also the explanation advanced by W. T. M. Forbes [*Am. J. Psychol.* 41, 517 (1929)] to account for L. L. Sloan's [*Psychol. Monograph No. 38*, (1928), p. 7] discovery of a hump which appeared on the red side of the luminosity curve at reduced intensities, a hump also found on foveal luminosity curves by H. V. Walters and W. D. Wright [*Proc. Roy. Soc. (London)* **B131**, 340 (1943)] and appearing as a prominent feature of our CFF-determined curves [G. G. Heath, *Science* **128**, 775 (1958)].
2. In addition to the evidence cited in (*1*), studies of dark adaptation with colored lights [A. Kohlrausch, *Arch. ges. Physiol. Pflüger's* **196**, 113 (1922); A. Chapanis, *Am. J. Physiol.* **146**, 489 (1946); ———, *J. Gen. Physiol.* **30**, 423 (1947); E. Auerbach and G. Wald, *Science* **120**, 401 (1954)] have provided direct evidence of the inapplicability of cone threshold data to studies of photopic luminosity curves, since differently colored test lights equated for brightness at photopic levels required quite different attenuations to reduce them to cone threshold.
3. S. Hecht and Y. Hsia, *J. Gen. Physiol.* **31**, 141 (1947); S. Hecht, *Documenta Ophthalmol.* **3**, 289 (1949).
4. In addition to appropriate performance on AO H–R–R pseudoisochromatic plates, tests and criteria were those described by G. L. Walls and G. G. Heath [*J. Opt. Soc. Am.* **46**, 640 (1956)], which included the Nagel anomaloscope, a neutral-point determination, and the Walls-Mathews three-light and RDP tests [G. L. Walls and R. W. Mathews, *Univ. Calif. (Berkeley) Publs. Psychol.* **7**, 1 (1952)].
5. For example, at least three of the seven "deuteranopes" and three of the seven "protanopes" reported by Zanen [J. Zanen, R. Wibail, A. Meunier, *Bull. mém. soc. franç. Ophtalmol.* **70**, 81 (1957)] were obviously no more than anomalous trichromates, as shown by their performances on the Farnsworth Dichotomous and the Farnsworth-Munsell 100-hue tests, and perhaps also on the anomaloscope, though their test procedure with the latter is not clear but seems unconventional. These faults, and the failure to determine neutral points, were pointed out by Dubois-Poulsen, and the possibility of misdiagnoses was admitted by Zanen in the discussion at the end of the paper.
6. R. M. Boynton, G. Kandel, J. W. Onley, *J. Opt. Soc. Am.* **49**, 654 (1959).

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Site and Mechanism of Tick Paralysis

Abstract. A neurophysiological investigation indicates that the paralysis produced in the dog by the wood tick, *Dermacentor andersoni* Stiles, is due to failure in the liberation of acetylcholine at the neuromuscular junction because of a conduction block in the somatic motor fibers produced by the tick "toxin."

Tick paralysis, an acute ascending flaccid paralysis which may terminate in respiratory failure, affects animals and humans. It is caused by the feeding of the female tick, which is believed to secrete a neurotoxin in its salivary glands (*1*). As the common wood tick, *Dermacentor andersoni* Stiles, has been responsible for most of the cases in North America, the disease has, accordingly, been largely confined to the northwestern United States and the adjoining southwestern region of Canada. Human cases have been recorded recently in the eastern and southern United States, due largely to the common dog tick *D. variabilis* Say (*2*). This indicates a more widespread distribution of the disease than is generally recognized. Over 300 cases of tick paralysis in humans have been recorded on this continent, with a mortality of approximately 12 percent (*3*). As removal of the tick ensures recovery except when the patient is moribund, early diagnosis of the disease is desirable to avoid fatalities.

In order to determine the site and the pathological mechanism of the paralysis, a neurophysiological study was carried out on mongrel dogs which were paralyzed by applying the ticks *D. andersoni* Stiles. The disease produced in the dog closely resembles that in the human; in the severely paralyzed animal the muscles are flaccid and the tendon reflexes are absent.

The anterior tibial muscle was selected for study because it was found to be involved early in the paralysis. The first significant observation was that this muscle responded to direct electrical stimulation but failed to contract when stimulated through the peroneal nerve (*4*). This finding indicated either that the motor nerve fibers could not conduct a nerve impulse or that there was a block at the neuromuscular junction. When the sixth lumbar ventral root was stimulated, an action potential was recorded from the peroneal nerve, indicating that motor nerve fibers were conducting an impulse (*5*). The muscle, which failed to respond to nerve stimulation, contracted when acetylcholine was injected intra-arterially directed into the muscle. Indeed, the paralyzed muscle exhibited an increased sensitivity to acetylcholine (*6*), a condition which occurs in denervated muscle. The response to acetylcholine, to antagonists of blocking agents, and to repetitive stimulation indicated that the paralysis did not resemble that produced by the known blocking agents —for example, curare or decamethonium—or by excessive doses of anticholinesterases, but was similar to that in botulinum toxin poisoning (*5*). The latter has been shown to be probably due to a block in the small terminal motor nerve fibers (*7*).

When a paralyzing dose of succinylcholine was injected into normal and tick-paralyzed anesthetized dogs and a wick electrode was swept across the under surface of the anterior tibial muscle, small areas of depolarization could be consistently detected and located; these represented depolarized end-plate regions. When the peroneal nerve was stimulated intermittently in normal dogs, each nerve impulse produced a transient potential (end-plate potential) which could be recorded from each located end-plate region; in tick-paralyzed dogs, however, end-plate potentials could not be detected at located end-plate regions during nerve stimulation, which suggested that acetylcholine was not being liberated at the nerve terminal (*6*).

In order to prove conclusively that tick paralysis is due to failure in the liberation of acetylcholine, paralyzed and normal anterior tibial muscles were perfused with Ringer's solution and the acetylcholine liberated into the perfusate was estimated. While acetylcholine was liberated by the normal muscle (6×10^{-12} gm per nerve volley) during nerve stimulation, none was liberated by the paralyzed muscle on stimulating either the nerve or the muscle directly (*8*). Emmons and McLennan (*9*) have shown recently that the muscles of the perfused hind leg of the tick-paralyzed ground hog also fail to liberate acetylcholine when the sciatic nerve is stimulated.

The inability of nerve or direct muscle stimulation to liberate acetylcholine could be due to (i) failure of the terminal motor nerve fibers to conduct the nerve impulse, or (ii) defective storage, synthesis, or release of acetylcholine at the nerve terminals. When the paralyzed muscle was perfused with Ringer's solution containing four times the normal concentration of potassium, acetylcholine was liberated, indicating that it is available at the nerve terminals. Recent evidence indicates that choline acetylase, the enzyme required for acetylcholine synthesis (*10*), and probably acetylcholine (*11*) are produced in the nerve cell and migrate down the axon. In five normal dogs, the mean number of milligrams of acetycholine liberated per gram of acetone powder per hour