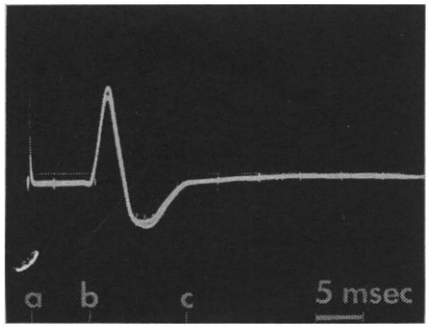

Fig. 2. Tracing obtained when ulnar nerve was stimulated for 0.1 msec at 150 v in a normal patient.

form that is recorded in normal patients.

The "H" reflexes were first described by Magladery and McDougall (*8*). They represent the response in muscle recordings when the afferent fibers from muscle are stimulated by low-threshold nerve stimulation. They usually follow the direct motor efferent response of nerve stimulation and have a latent period of the order of 30 to 40 msec. The afferent impulse is presumed to travel to the spinal cord and then both monosynaptic and polysynaptic connections cause a discharge over the lower motor neuron which is then recorded at the periphery as an "H" response. In a series of extensive and thorough investigations, Magladery and his coworkers (*8, 9*) demonstrated that these responses are not usually seen in normal persons but are seen predominantly in patients who have cerebral, brain stem, and spinal lesions. They postulated that to observe these reflexes, some release of inhibitory influences on the final lower motor neuron was necessary. The closer the lesion was to the segmental reflex studies, the clearer was the phenomena. The "H" reflexes have also been demonstrated in tetanus intoxication (*10*), where there is a depression of certain inhibitory actions of interneurons upon the motor neuron (*11*).

The prominence of the "H" reflexes in almost all the records of nerve conduction strongly suggests some effect of the botulinus toxin at the spinal level. Since both tetanus and botulinus toxin are clostridial toxins, it is possible that botulinus toxin may have an effect on inhibitory actions of some interneurons similar to that reported for tetanus. A direct action upon the cholinergically innervated inhibitory neurons in the cord (Renshaw cells) is also possible. A central action of the toxin could explain phenomena such as tonic spasm of the hand (*12*) and occasional respiratory or cardiac irregularities seen in these patients. These phenomena are difficult to understand if the action of botulinus toxin is solely limited to the periphery (*13*).

H. RICHARD TYLER
Peter Bent Brigham Hospital and
Department of Neurology,
Harvard Medical School,
Boston, Massachusetts

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Rhodopsin: An Enzyme

Electron histochemical evidence indicates that a nucleoside triphosphatase is present in the membranes of the outer segments of frog retinal receptors (*1*). On the possibility that this enzyme is actually rhodopsin, we examined the adenosine triphosphatase activity of digitonin extracts of frog rhodopsin.

These extracts exhibit substantial adenosine triphosphatase activity, and the activity is twofold greater in bright tungsten light than it is in the dark. The activity of the enzyme is virtually eliminated by hydroxylamine, which presumably traps retinene away from the opsin. Furthermore, when the light is passed through different colored filters which transmit equal absolute energies (as measured by a thermopile), a filter with maximum transmission in the neighborhood of frog rhodopsin's maximum absorption —502 mμ—is the most effective in accelerating enzyme activity.

Finally, when solutions of either frog or cattle opsin, obtained by extraction of bleached retinas with digitonin, are incubated with a solution of all-trans retinene$_1$ which has been partially converted to the cis-isomer by irradiation with tungsten light, the activity of the enzyme increases in the same manner in which rhodopsin recovers its absorption peak.

We infer that rhodopsin is an adenosine triphosphatase which requires the attachment of its retinene chromophore for activity either in the light or dark. The retinene may act both as a light trap and as a cofactor for the enzyme.

George Wald recently wrote that all the best discoveries should have been anticipated (*2*). That rhodopsin is in fact an enzyme was anticipated no later than a dozen years ago—by George Wald.

D. G. MCCONNELL
D. G. SCARPELLI
Institute for Research in Vision and
Laboratory of Cellular Pathology,
Ohio State University, Columbus 12

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