those required to produce hyperoxic seizures, but it is conceivable that conduction deficiencies may develop more quickly and with lower pressures of oxygen in tissue with a circulation.

Oxygen at high pressure (12 atm) produces conduction block in frog peripheral nerve. The block is partially reversible if it is not maintained more than a few minutes. The block caused by 5-percent CO_2–95-percent O_2 has a shorter latency, and its reversibility is more pronounced than that of the block produced by oxygen alone. The presence or absence of activity in nerve caused by continuous stimulation does not affect the time course of hyperoxic conduction block (*9*).

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References and Notes

1. P. Bert, *La pression barométrique* (Paris, 1878), translated by M. A. and F. A. Hitchcock (College Book Co., Columbus, Ohio, 1943), p. 840.
2. L. Hill and J. J. R. Macleod, *J. Hyg.* 3, 401 (1903).
3. J. W. Bean and D. F. Bohr, *Am. J. Physiol.* 124, 576 (1938).
4. R. Lorente de Nó, *A Study of Nerve Physiology* (Rockefeller Institute for Medical Research, New York, 1947), p. 17 (Corrected by personal communication). Buffer formula: Ringer stock: 65 g NaCl, 1 g KCl, 12 ml 10-percent $CaCl_2$ solution, and sufficient distilled water to make 1 lit. Buffer stock: 0.92 g $NaH_2PO_4 \cdot H_2O$, 3.765 g Na_2HPO_4, and sufficient distilled water to make 500 ml. Use 10 ml of Ringer stock to 100 ml of distilled H_2O for normal Ringer. Use 5 ml of buffer stock to 100 ml of normal Ringer for *p*H 7.3.
5. G. W. Snedecor, *Statistical Methods* (Iowa State College Press, Ames, ed. 4, 1948), p. 81.
6. F. Dickens, *Biochem. J. London* 40, 145 (1946).
7. R. Gerschman *et al.*, *Science* 119, 623 (1954).
8. L. A. Shaw *et al.*, *Am. J. Physiol.* 108, 652 (1934).
9. Opinions and assertions in this report are the author's private ones and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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Basis for After-Discharge in the Median Giant Axon of the Earthworm

The ventral nerve cord of the earthworm (*Lumbricus terrestris*) contains three giant axons, many smaller nerves, and a complex neuropile. The giant axons are endowed with many synapses that range from endings of smaller nerves on the giant axons to the segmental transverse septa that interrupt the substance of the latter (*1*). Because of this abundance, the giant axons are interesting material for the study of some fundamental properties of interneuronal relationship (*2*).

Isolated ventral nerve cords were immersed in an earthworm saline (*3*) and stimulated with a pair of surface electrodes. Responses were simultaneously recorded with a pair of electrodes on the entire nerve cord and one or two prefilled capillary microelectrodes (*4*) in the median giant axon. Action potentials recorded from the intracellular loci ranged from 80 to 100 mv; these were 25 to 35 mv beyond the resting potential of 50 to 70 mv. Svaetichin (*5*) has reported the same limits for the resting potential obtained with an axially inserted microelectrode.

At room temperature of 19° to 24°C, the internally recorded spike lasted 1 msec, but it was followed by a slight negativity that persisted for about 4 msec. Occasionally, with one brief stimulus, two or more responses occurred. The first direct response to the shock rose rapidly, while the later ones were often preceded by a prepotential that rose more slowly. The nature of the repetitive discharge could be resolved by applying two brief shocks in rapid succession. The second response elicited in the relative refractory period was followed by a sequence of small and additive potentials. These were highly variable in number, amplitude, frequency, and time and site of occurrence.

Figure 1 illustrates a case in which activity was recorded from three sites of a preparation. The entire nerve cord was stimulated at one end, and impulses were recorded from the other end with external electrodes. Two microelectrodes, 11.3 mm apart, were placed in the median giant axon in a stretch between the surface electrodes. Response to the first stimulus, which was threshold for the median giant axon alone, was recorded earliest at the two microelectrodes and, after the appropriate conduction time, on the external leads. The strong second stimulus that was applied in the relative refractory period reexcited the axon, but the internally recorded responses were smaller and the externally recorded response was markedly delayed. The external trace, but neither internal trace, also registered the spike of the lateral giants evoked by the strong stimulus. The trace of one of the microelectrodes, however, remained elevated above the base line and carried numerous small peaks. The trace of the other had only a brief additional potential soon after the spike. About 7 msec after the second stimulus another spike developed in the giant axon. Since the small potentials began during the absolute refractory period of the giant axon, they could not be produced by the same mechanism responsible for the electrically excitable spike. Owing to their local nonpropagating nature, low amplitude, frequency of repetition, and additiveness, these potentials are probably synaptic, developing at the terminations of small nerve fibers on the giant axon. This junctionally

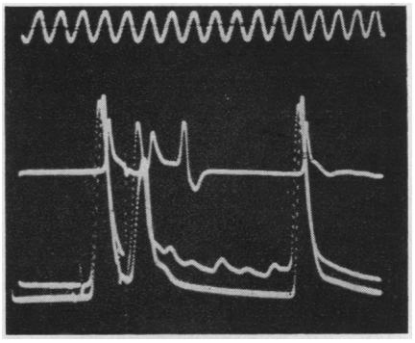

Fig. 1. Simultaneous recording from three sites of the nerve cord (external leads) and median giant axon (internal microelectrodes). The upper trace is the external recording, which is also the base line for the resting potentials. The latter are 72 and 74 mv; spike heights are 98 and 110 mv. The distance between the stimulating cathode and proximal microelectrode was 8.5 mm; between the microelectrodes, 11.3 mm; and between the distal microelectrode and the first external lead, 3.5 mm. The two directly coupled beams are not coincident. Time 1000 cycles.

elicited postsynaptic activity of the median giant axon is then responsible for reexciting the fiber.

Other evidence indicates that the activity of the small nerve fibers that excite the giant axon synaptically are themselves initiated, at least in part, by previous activity of the giant axon. Thus, small fiber activity, as well as synaptic potentials and late all-or-none discharges, disappears when the second stimulus is applied during the absolute refractory period of the giant axon. The evidence, therefore, suggests the presence of a closed circuit containing efferents from and afferents into the median giant axon. It also indicates that "reentry" is an important factor in the after-discharge following a brief stimulus to this preparation.

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References and Notes

1. H. B. Stough, *J. Comp. Neurol.* 40, 409 (1926); W. M. Smallwood and M. T. Holmes, *ibid.* 43, 327 (1927); T. H. Bullock, *J. Neurophysiol.* 8, 55 (1945); J. Stephenson, *The Oligochaeta* (Oxford Univ. Press, Oxford, 1930).
2. I am deeply indebted to H. Grundfest of the department of neurology, College of Physicians and Surgeons, Columbia University, for a generous loan of instruments. To him, and to C. McC. Brooks, I am grateful for many stimulating discussions.
3. W. A. H. Rushton, *Proc. Roy. Soc. London* B132, 423 (1945).
4. G. Ling and R. W. Gerard, *J. Cellular Comp. Physiol.* 34, 383 (1949); W. L. Nastuk and A. L. Hodgkin, *ibid.* 35, 39 (1950); C. Y. Kao, *Science* 119, 846 (1954).
5. G. Svaetichin, *Acta Physiol. Scand.* 24, Suppl. 80, 50 (1951).
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