tions, such as bronchiectasis, pulmonary abscess, unresolved pneumonia, typhoid fever, cardiovascular renal disease, and carcinoma, when no blood loss was associated, was found to be of this type.

IV. The fourth class is distinguished by a marked reduction in the volume of the red corpuscles and a decrease in their hemoglobin content which is even more marked than the decrease in size. Even when the number of red corpuscles is not lowered a distinct reduction in their volume has been found in this group of anemias. The reduction in size in this class is even more marked than that observed in class III. The lowest values for mean corpuscular volume have been found in this group.

Even more characteristic than the reduction in the size of the cells in this type of anemia is the alteration in their hemoglobin content. The reduction in corpuscular hemoglobin is even more marked than the diminution in volume with the result that in this group, and apparently in this group alone, corpuscular concentration values significantly and constantly lower than normal are found. For this reason I have called this type of microcytic anemia *"hypochromic."*

The anemias which fall into this hypochronic, microcytic group are largely composed of those resulting from chronic blood loss. Since the cause of the anemia resulting from hookworm infestation is still disputed, it is of especial interest that the anemia observed in hookworm disease has been of this hypochromic type.

Further observations are being made with a view to determining the accuracy and usefulness of this classification, the relationship of the 4 classes of anemia to one another, and the effects of various forms of therapy on the size and hemoglobin content of the red corpuscles.

5120

Mechanism of Nerve Asphyxiation: With a Note on the Nerve Sheath as a Diffusion Barrier.

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In the absence of oxygen nerve conduction is suspended in several hours, the activity of each fibre becoming gradually less until it blocks. Conduction has been shown to depend on oxidations, and the long persistence of activity with outside oxygen excluded to depend on the existence in nerve of an oxidizing reserve. The rapid recov-

ery from anoxia when oxygen is again admitted suggested that the nerve might similarly recover when some other reducible substance, such as methylene blue, were made available.

To study this, a double chamber was constructed to accommodate the 2 sciatics of one frog. Each nerve was stimulated at one end and the action potentials led from the other crushed end and the side. Between stimulating and lead electrodes the nerve traversed a sealed compartment, through which nitrogen could be passed, and opening through its floor to a bulb containing fluid. During the asphyxiation nitrogen entered the bulb from the bottom and, after bubbling through the liquid, swept through the nerve chamber. This rendered the liquid oxygen-free, while the nerve asphyxiated, and compression of the bulb then bathed the nerve with this liquid. One side contained Ringer solution buffered at pH 7 or 8, the other a similar solution plus 0.5% methylene blue or 1-naphthol 2-sulphonate indophenol. It was regularly found that neither solution brought about recovery. (The action potentials are led from nerve in air below the exposed region and are an index, therefore, of the number of fibre-impulses getting through, not of the impulse intensity in the experimental stretch.)

We then observed that methylene blue, though staining the nerve sheath deeply, hardly penetrated to the bundle of axones. It is not difficult to split the sheath longitudinally with little injury to the conducting elements, and in subsequent experiments this was done. The asphyxiated nerve then showed a definite recovery in methylene blue and also in Ringer solution, the action potentials returning to about 1/3 their original value. (This does not mean that each fibre recovered from zero activity to 1/3 normal.)

The great importance of the perineurium as a diffusion barrier is seen when the time to block by various agents (KCl, $CaCl_2$, NaCN, glucose) is determined for the nerves with intact and split sheaths (Table I). It may be noted that the agents producing most rapid block are least rapidly recovered from in Ringer.

TABLE I.

Nerve in	Intact Nerve		Sheath Split	
	Block	Maximum recovery in Ringer	Block	Maximum recovery in Ringer
	min.	min.	min.	min.
Isotonic glucose	None in 3 hrs.	<1	17	<1
Isotonic $CaCl_2$	50+	20	<2	9
Isotonic KCl	15	50	<2	20
N/150 NaCN in Ringer	80	—	10	—

Since even Ringer led to some recovery from asphyxial block a new method was used to determine the methylene blue effect. The stretch of nerve was asphyxiated in Ringer solution by bubbling nitrogen through and then solid methylene blue, lying on a plate at the top of the compartment, was tipped in. No recovery was obtained with this dye or the indophenol. A final series of experiments, with oxidations of a stretch of nerve interfered with by cyanide, also yielded no difference when methylene blue was present.

The negative results with methylene blue cannot be due to lack of penetration—the axones were well stained, nor too low an oxidizing potential—the nerve does reduce methylene blue and the more powerful indophenol is not more effective. Probably the energy available is not sufficient or is liberated largely at a stage in the chain of reactions or a region of the cytoplasm where it cannot be utilized.

Asphyxial block of nerve must represent a suspension or diminution of energy yielding reactions (oxidations). This might be the result of (1) exhaustion of reactants (oxidizing reserve depleted), (2) accumulation of end products, or, (3) alteration of the mileau so that the conditions permitting these reactions at a necessary rate no longer obtain. Of course all factors may contribute to lowering below a critical value for conduction. When a nerve first blocks in nitrogen it cannot be entirely due to (1) or bathing in Ringer would be ineffectual. The oxidizing reserve is not fully exhausted until after 10 to 16 or more hours anoxia (at 20-23°C.), at which time Ringer no longer gives any recovery though oxygen is still able to restore the nerve almost completely.

Earlier workers noting the action of Ringer on asphyxiated nerve (with intact sheath!) have attributed this to a washing out of metabolites, (2) above; but possibly (3) an alteration of the nerve condition must also be considered. The addition of considerable sodium lactate to Ringer does not lessen its restorative action but a much smaller quantity of lactic acid, due to the resultant acidity, does. Carbon dioxide appears to slow the reactions of nerve. Resting nerve produces lactic acid (and no other acid in significant amounts) during anoxia; but this cannot be removed in oxygen, whereas a nerve may fully recover from asphyxia in oxygen alone. Against an outdiffusion of metabolites is the effectiveness of very small quantities of Ringer, just enough to remoisten the nerve surface. On the other hand, the action of Ringer cannot be due to some specific ion, other than Na^+ or Cl^-, since 0.2% NaCl in isotonic glucose is effective. Some ion effect is suggested by analogy with the phenomenon of reversible inexcitability in muscle, and the bene-

ficial action of Ringer on a nerve that has failed to recover from cold block. Finally nerves soaked in isotonic NaCl for 40 minutes before the start of asphyxia, though still fully recovering in oxygen, no longer show any recovery in Ringer. Nerves soaked in NaCl followed by Ringer behave as if they had been in Ringer throughout.

5121

A Note on the Propagation of the Virus in Experimental Poliomyelitis.*

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While there is good evidence to show that the virus travels along the axis cylinder of the nerve fibers of the spinal cord, particularly from recent work of Fairbrother and Hurst,[1] dissemination by the cerebrospinal fluid has been invoked in explanation by other authors. (Flexner and associates.[2, 3]) A crucial experiment to settle the question would be found in an attempt to recover virus from the cervical cord and from the lumbar cord of a monkey, inoculated intracerebrally, after complete transsection of the spinal cord above the lumbar region. Incidentally, further information might be adduced by histological examination of the 2 separate cord sections for the presence or absence of specific lesions.

Accordingly, we have carried out a complete transsection of the spinal cord in 2 monkeys, severing the structure at the level of the first lumbar vertebra. In the first animal (Monkey 144), 2 silk ligatures were tied tightly around the unopened dural sac, crushing the cord. The whole structure was then cut between the ligatures, the stumps retracting about 1 cm. apart. In the second animal (Monkey 165), the dural sac was opened, but not severed, and the cord was totally divided, leaving a gap of about 3 mm. The operative procedure in the first animal was adopted in order to test the possibility of dissemination by routes other than either the nervous substance or the cerebrospinal fluid, such as the general circulation or the lymphatics. In the second animal our main interest lay in dis-

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1 Fairbrother, R. W., and Hurst, E. W., *J. Exp. Path. a. Bact.*, 1930, xxxii, 17.

2 Flexner, S., and Amoss, H. L., *J. Exp. Med.*, 1914, xx, 249.

3 Amoss, H. L., in Rivers, Th.: Filterable Viruses, Baltimore, 1928.