

complete only for an hour or so, thereafter wearing away to a certain residual amount. In strong solutions, e.g., N/10 NaCN, the inhibition is practically constant from the first and amounts to 80 to 95 per cent. The explanation of the escape from inhibition is to be found, perhaps, in the expulsion of HCN from the inner fibers by the lactic acid as a result of the anoxemia produced since lactic acid is stronger than HCN by some six orders; the effect may also be due somewhat to a relatively slow outward diffusion of the lactic acid.

Similarly it was found that nerve respiration may be inhibited fairly completely by carbon monoxide in the dark. The constant expressing the relative affinities of the carbon monoxide and oxygen for the iron catalyst represented by the equation $n/1 - n \cdot \text{CO}/\text{O}_2 = k$ was found to approximate closely to the value of 10 as was found for other cells by Warburg. Furthermore, illuminating the nerve causes a marked decrease of the carbon monoxide inhibition of resting metabolism.

By far the most striking results were obtained when the effect of carbon monoxide on the action potential was studied by means of the cathode ray oscillograph. In mixtures containing from 1 to 3 per cent. of oxygen in carbon monoxide it was found that the height of the action potential decreases progressively to extinction, this decrease being considerably faster than in a similar mixture of oxygen with nitrogen. If during this decline in carbon monoxide the nerve be illuminated by means of an arc-light, the height of the action potential rises immediately and may return to, or even exceed somewhat, the original value. It is important to note that the potential rises immediately with illumination but does not drop at once when the illumination is turned off; the return to the original extinction curve usually takes from 20 to 30 minutes. That the effect is not one of temperature rise or of photo-oxidation is shown by the fact that a companion nerve in nitrogen failing along a similar curve is quite unaffected by the illumination. There is some evidence of small rises in potential in illuminated nerves in presumably pure carbon monoxide; the explanation of this is at present not yet clear.

The work is not sufficiently far along to warrant any sweeping generalizations, but it seems clear that the action potential is produced by an oxidation or oxygenation of a substance or substances in nerve, and that for this purpose, activation of the oxygen by a respiratory enzyme similar to that of Warburg's is essential. Since nerves usually do not fail in pure carbon monoxide any faster than in pure nitrogen it appears that the function of the iron catalyst is chiefly to make active oxygen available to the irritable mechanism which when stimulated is then capable of produc-

ing the action potential. For the further elucidation of the rôle of the iron catalyst and of the oxidations required for the production of the action potential I am attempting to bring together two distinct lines of research: that of manometric measurement of metabolism, and that of the measurement of the electric potential of nerve. Indeed, some progress has already been made in this direction; I refer to the fact that it is now possible in our hands to obtain accurate records of the height, shape, duration, etc., of the action potential of nerves with the cathode ray oscillograph whilst measuring simultaneously their metabolism manometrically. Only by such a union of methods will the questions raised in this report be adequately answered.

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