correlated with the corresponding values of the respiratory quotient, it was found that high values of the latter were accompanied by low oxygen consumptions and vice versa. It follows that different substrates occurring in the liver of the frog are oxidized at different rates.

A more detailed account of the results will shortly be published elsewhere.

P. E. LINDAHL.
CHR. WERNSTEDT.

Wenner-Gren's Institute for Experimental Biology, University of Stockholm.

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Effect of Anoxia on Excitation and Impulse Propagation in Isolated Motor Nerve Fibres

ALTHOUGH the paralysing action of anoxia on peripheral nerve has been frequently studied in previous investigations, so far as we know, no attempt has as yet been made to examine whether excitability and propagation of the nervous impulse react identically to lack of oxygen.

Monophasic action potentials of isolated motor nerve fibres (sciatic nerve of *Rana esculenta*) were used as indicators. The nerve was placed in a moist chamber consisting of two separate sections, one containing two platinum electrodes for stimulation with condenser discharges, the other two silver–silver chloride electrodes for leading-off action potentials. Care was taken to avoid stimulus escape and to provide constant resistance between the leading-off electrodes. Anoxia was produced by passing a moist stream of purified oxygen-free hydrogen through each section of the chamber.

When anoxia is applied to both the stimulated and the conducting region of the nerve fibre, the average time necessary to suppress activity is 34 minutes, while it takes more than 70 minutes when anoxia is limited to the leading-off section of the chamber. This difference is statistically highly significant. The action potential reappears immediately after readmission of oxygen with reduced amplitude and without a positive after-potential. After 15 minutes in oxygen it has regained its original shape and amplitude. The stimulated region of a peripheral nerve fibre is thus far more sensitive to anoxia than its purely conducting parts, a difference which might be of interest in the interpretation of the mechanisms of excitation and propagation.

A further discussion on the matter is to be published elsewhere.

FRITZ BUCHTHAL.
HELGE HERTZ.

Physiological Institute, University of Lund.
Nov. 16.

Toxicity of Adrenaline

UNHEATED solutions of adrenaline quickly acquire a coloration if exposed to air or oxygen for a short period of time. Sodium or potassium metabisulphite has been proposed as an antioxidant for such solutions[1], and the U.S. Pharmacopeia XII permits the use of reducing agents such as sodium bisulphite up to concentrations of 0·5 per cent. The effect of this substance on the toxicity of adrenaline was investigated in the United States[2], and an increase of more than 100 per cent subcutaneously and more than three times intramuscularly was shown when tests were made on mammals. But no reference was made to the toxicity of adrenaline solutions containing metabisulphite after heat treatment, for example, autoclaving. These heated solutions, provided the *p*H is adjusted, have already been found to have lost very little activity, and to be sterile and colourless[1].

Solutions of adrenaline (1/1,000) in hydrochloric acid, with a final *p*H of about 3·0, were therefore prepared with and without 0·1 per cent of potassium metabisulphite, and their toxicities were tested on rats and frogs. The solutions containing metabisulphite were divided into two parts, and one was put in an ampoule and heated at 115° C. for 30 minutes. The rats were injected subcutaneously, the solutions containing 0·9 per cent sodium chloride; the frogs received their doses into the lymph sacs, the solutions containing 0·6 per cent sodium chloride. The approximate values for *LD* 50 (in mgm. per kgm.) were as follows: (*a*) rats—subcutaneously; adrenaline 12, adrenaline with metabisulphite 6, adrenaline with metabisulphite heated 14; (*b*) frogs—lymph sac; adrenaline 75, adrenaline with metabisulphite 30, adrenaline with metabisulphite heated 60.

Totals of sixty frogs (both sexes) and a hundred rats (all male) were used to obtain these values. These are relatively small numbers, but the results are important and more detailed work is in progress. Metabisulphite more than doubled the toxicity of adrenaline, thus confirming previous results[2]; but, on heating these solutions in 10-ml. ampoules, the tendency was to return to the toxicity figures of the plain solution. Other experiments have been carried out using metabisulphite solutions (0·1 per cent in 0·9 per cent sodium chloride), heated adrenaline solutions, and heated metabisulphite solutions, but no significant results were obtained.

G. B. WEST.

College of the Pharmaceutical Society, London, W.C.1.
Nov. 16.

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Absolute Scotopic Sensitivity of the Eye in the Ultra-violet and in the Visible Spectrum

IN a paper published in 1941[1] measurements were given of scotopic and photopic sensitivity, for which nine observers were used. These measurements were made in the range between the mercury lines 709 and 302 mμ. In the paper by Goodeve, Lythgoe and Schneider published in 1942[2] measurements were given of scotopic sensitivity, for which six observers were used, including one with an aphakic eye. Their measurements were made at the mercury lines at 365 and 546 mμ. It is of interest to compare the results obtained after dark adaptation of one hour, with the results of Goodeve *et al.*, obtained after dark adaptation for 10 minutes.

The absolute scotopic sensitivity, S_λ, in terms of (quanta/sec. sq. mm.)$^{-1}$ is related to the illumination of the pupil, E_λ (the latter being expressed in terms of erg./sec. sq. cm.), by the equation

$$S_\lambda = \left[\frac{E_\lambda \times 10^{-3}\,(1-r)}{h\nu} \cdot \frac{\sigma}{a}\right]^{-1},$$