

Action Potentials of Single Muscle Fibres evoked by Stimulation via the Recording Micro-electrode

Most experiments for recording the action potentials of striated muscle by means of intracellular electrodes are carried out with isolated curarized muscles. If non-curarized muscles *in situ* are used, it is very difficult to immobilize them so that artefacts appear¹ and the electrode can easily break. To overcome these difficulties the stimulation of only one fibre through the intracellular recording electrode was tried. Where the stimulus was applied intracellularly, a positive stimulation pulse was used. The action potential then appeared during the pulse^{2,3}. The amplitude of the recorded action potential during such a positive pulse, however, decreases with increase in the stimulation pulse (Fig. 1) because of the decrease of the impedance of the membrane during the action potential⁴. This decrease of the impedance produces a decrease of the recorded stimulation voltage (which is proportional to the strength of the stimulus), because the stimulus is a constant current. The recorded action potential is a summation of the true action potential and this fall in voltage. A higher stimulus will therefore cause a smaller recorded action potential.

This deformation made it desirable to evoke potentials after cessation of the stimulus. On the analogy of extracellular anodal break stimulation—where the action appears also after the stimulus—we have tried using a negative pulse for stimulation so that the action potential appears after cessation of the pulse.

Depending on the strength or duration of the pulse, one or more action potentials will appear. An increase of this strength or duration causes an increase in the number of action potentials (Fig. 2). The time interval between these action potentials increases progressively and an increase of strength or duration of the stimulus causes a decrease of the time intervals (Fig. 2).

We have checked the nature of the action potential of these recordings using the property of action potentials to propagate along the muscle fibre. We have therefore used two electrodes (one stimulating and recording electrode and one recording electrode) in one fibre. It appeared that there was a propagation along the muscle fibre in our experimental set-up.

The experiments as they are described here can be performed on animals as well as on human subjects *in situ*. In our opinion this is a useful addition to the more conventional methods for studying the effect of pharmacology and diseases on the electrical properties of striated muscle.

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¹ Den Hertog, A., *et al.*, *Acta Physiol. Pharmacol. Neerl.* (in the press).

² Araki, T., and Otani, T., *J. Neurophysiol.*, **18**, 472 (1955).

³ Beranek, R., *E.E.G. Clin. Neurophysiol.*, **16**, 301 (1964).

⁴ Cole, U. S., and Curtiss, H. J., *J. Gen. Physiol.*, **22**, 37 (1938).

Transport of Ammonia by the Small Intestine of the Golden Hamster

HYPERAMMONAEMIA is thought to be a cause of hepatic coma. Ammonium which is contributed to the general circulation by the alimentary tract is derived from the degradation of protein and urea¹. Ammonium chloride is absorbed readily from the small intestine *in vivo*, but a more precise characterization of ammonium transport can come only from *in vitro* investigations. Our report

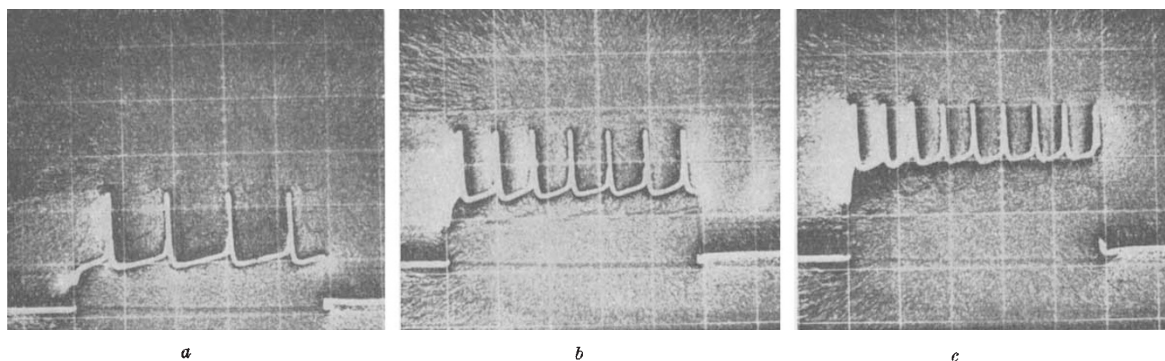


Fig. 1. Muscle fibre response after positive intracellular stimulation. *a*, Stimulation current = 1.0×10^{-7} amp; *b*, stimulation current = 1.5×10^{-7} amp; *c*, stimulation current = 2.0×10^{-7} amp. Ordinate, 10 msec/division; abscissa, 50 mV/division.

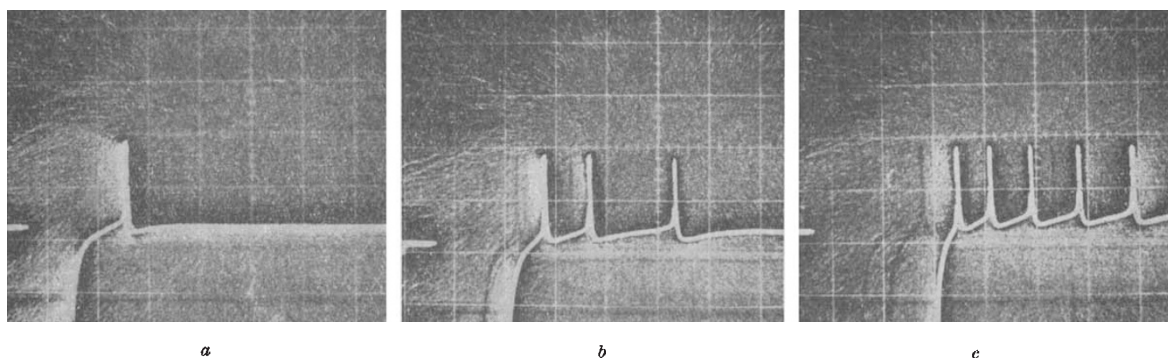


Fig. 2. Muscle fibre response after negative intracellular stimulation. *a*, Stimulation current = 6×10^{-7} amp; *b*, stimulation current = 8×10^{-7} amp; *c*, stimulation current = 1×10^{-6} amp. Ordinate, 10 msec/division; abscissa, 50 mV/division.