## Effect of rubidium on responses of rabbit vas deferens to transmural stimulation

A. JOHNS\* & D.M. PATON

Department of Pharmacology, University of Alberta, Edmonton, Alberta, Canada

Transmitter release is known to be very sensitive to changes in the duration of the action potential in nerve terminals (Katz & Miledi, 1967). In the present study, we have examined the influence of rubidium on adrenergic transmission in rabbit vas deferens as rubidium increased the duration of the action potential in squid giant axon (Baker, Hodgkin & Shaw, 1962).

New Zealand rabbits were killed by air embolism. Their vasa deferentia were removed and mounted longitudinally in 5 ml organ baths containing modified Krebs solution at 37°C. The medium contained 3 mM KCl and was equilibrated with 95% 0<sub>2</sub>/5% CO<sub>2</sub>. Contractions were recorded isometrically with strain gauges. Tissues were stimulated transmurally with biphasic pulses of 1 msec duration, supramaximal voltage and 0.5-16 Hz for 60 s every 4 minutes. Transmural stimulation resulted in a rapid twitch response followed by a maintained contraction. This secondary, maintained response resulted from noradrenaline release as it was abolished by phentolamine and guanethidine. In subsequent experiments, the magnitude of the secondary response was determined.

Addition of 2 mm RbCl caused a markedly potentiated response to transmural stimulation at 2-14 Hz both in the presence and absence of 3 mm KCl in the medium. This effect was not due to an impairment of re-uptake of released noradrenaline as the 30 min uptake of  $[^3H](\pm)$ metaraminol was not altered by 2 mm RbCl (with 3 mm KCl, uptake was  $175.7 \pm 11.2$  pmol/g; with 5 mM KCl,  $164.8 \pm 17.1 \text{ pmol/g}$ ; with 3 mM KCland 2 mM RbCl,  $179.9 \pm 12.8$  pmol/g; mean  $\pm$  s.e. of 10 observations). The responses of vasa deferentia to exogenous (-)-noradrenaline were not altered by 2 mm RbCl thus excluding an effect at receptor level or at subsequent steps in excitation-contraction coupling.

In order to determine the effect of RbCl on transmitter release, tissues were exposed to 10<sup>-7</sup>M [<sup>3</sup>H](±)-metaraminol for 60 minutes. After 35 min in an amine-free medium, subsequent transmural stimulation at 5 Hz for 2 min resulted in the release of  $[^3H](\pm)$ -metaraminol and this release was very significantly increased by addition of 2 mm RbCl to the medium.

These studies suggest that rubidium may potentiate adrenergic transmission by increasing the release of noradrenaline as has also been reported to occur with caesium (Johns & Paton, 1974).

Supported by a grant from the Medical Research Council of Canada (MT2472).

## References

BAKER, P.F., HODGKIN, A.L. & SHAW, T.I. (1962). The effects of changes in ionic concentrations on the electrical properties of perfused giant axons. J. Physiol., 164, 355-374.

JOHNS, A. & PATON, D.M. (1974). Potentiating effects of caesium on transmural stimulation of rabit vas deferens. IRCS (Med. Sci.)., 2, 1434.

KATZ, B. & MILEDI, R. (1967). The release of acetylcholine from nerve endings by graded electric pulses. Proc. R. Soc. Lond. Ser. B, 167, 23-28.

## Effects of prostaglandins in calves

MAUREEN M. AITKEN\*1 & J. SANFORD2

Department of Veterinary Pharmacology, University of

The protective effect of sodium meclofenamate against bovine anaphylaxis (Aitken & Sanford, 1972) indicated that prostaglandins might be important mediators in this species. Indomethacin prevents the systemic hypotension, pulmonary hypertension and apnoea characteristic of anaphylaxis and inhibits Schultz-Dale contractions of isolated bovine pulmonary artery and bronchiole (Aitken & Sanford, unpublished observation).

However, Lewis & Eyre (1972) found that bovine bronchial ring failed to respond to PGF<sub>2</sub>α and was relaxed by PGE<sub>1</sub>. Effects of other prostaglandins on this tissue have not been

<sup>&</sup>lt;sup>1</sup> Present address, A.R.C. Institute of Research in Animal Diseases, Compton, Newbury, Berks.

<sup>&</sup>lt;sup>2</sup> Present address, Wyeth Institute of Medical Research, Taplow, Maidenhead, Berkshire.