REFERENCES

BURNS, D. B. & PATON, W. D. M. (1951). Depolarisation of the motor end-plate by decamethonium and acetylcholine. *J. Physiol., Lond.*, **115**, 41–73.

DERKX, F. H. M., BONTA, I. L. & LAGENDIJK, A. (1971). Species-dependent effect of neuromuscular blocking agents. *Eur. J. Pharmacol.*, **16**, 105–108.

GALINDO, A. (1971). Depolarising neuromuscular block. *J. Pharmacol. exp. Ther.*, **178**, 339–349.

LU, T. C. (1970). Affinity of curare-like compounds and their potency in blocking neuromuscular transmission. *J. Pharmacol. exp. Ther.*, **174**, 560–566.

PATON, W. D. M. & ZAIMIS, E. J. (1949). Pharmacological actions of polymethylene bistrimethyl ammonium salts. *Br. J. Pharmac. Chemother.*, **4**, 381–400.

Specificity of adenosine on transmitter output at the neuromuscular junction

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It has recently been found that the amount of transmitter released from the phrenic nerve of the rat, as measured by the ratio of the amplitude of evoked to spontaneous end-plate potentials, is reduced in the presence of adenosine, in a concentration of 0·025 mM or above (Ginsborg & Hirst, 1972). Investigations have now been made of the effects of a number of substances which might be expected to share some of the pharmacological properties of adenosine (see Burnstock, 1972). Of these only 5′-adenosine monophosphate (5′-AMP) shared the action of adenosine. The remainder, adenine, inosine, guanosine, cystine and uridine, in concentrations of up to at least 1 mM, did not reduce either the quantal content of end-plate potentials or alternatively the twitch tension of indirectly stimulated rat diaphragms bathed in high Mg^{2+}/low Ca^{2+} solutions: they were thus presumably without effect on transmitter release.

The interest in these results is related to the fact that adenosine and 5′-AMP are known to increase cyclic 3′,5′-adenosine monophosphate (cyclic AMP) in central nervous tissue whereas the remaining substances tested in these experiments are known not to have this effect (Sattin & Rall, 1970). The possibility that cyclic AMP is involved in the effect of adenosine on transmitter release or that the effect of adenosine on transmitter release and cyclic AMP have a common step cannot yet be rejected.

REFERENCES

BURNSTOCK, G. (1972). Purinergic nerves. *Pharmacol. Rev.*, **24**, 509–581

GINSBORG, B. L. & HIRST, G. D. S. (1972). The effect of adenosine on the release of the transmitter from the phrenic nerve of the rat. *J. Physiol., Lond.*, **224**, 629–645.

SATTIN, A. & RALL, T. W. (1970). The effect of adenosine and adenine nucleotides on the cyclic adenosine 3′-5′-phosphate content of guinea pig cerebral cortex slices. *Molec. Pharmac.*, **6**, 13–23

Bicuculline and frog spinal neurones

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Bicuculline has been reported to antagonize selectively the inhibitory effect of γ-aminobutyric acid (GABA) on mammalian spinal neurones (Curtis, Duggan, Felix & Johnston, 1970). Both GABA and glycine depress ventral root responses to dorsal root stimulation in amphibian spinal cord (Curtis, Phillis & Watkins, 1961). In the present experiments some preliminary attempts have been made to see whether these effects show a differential sensitivity to bicuculline.