

Effect of monoamine oxidase (MAO) inhibition upon the N, P and LN potentials of the rabbit superior cervical ganglia

G. HENDERSON (introduced by A. G. H. BLAKELEY)

Department of Pharmacology, Glasgow University, Glasgow W2

It has been postulated that an adrenergic component of ganglionic transmission is responsible for the generation of the P potential (Eccles & Libet, 1961). Inhibition of MAO might affect such a mechanism since such inhibition increases the noradrenaline content of ganglia (Reinert, 1963). Some MAO inhibitors block ganglionic transmission by a mechanism unrelated to their inhibition of MAO (Gertner, 1961; Levine, 1962). Da Costa & Goldberg (1961) have demonstrated that in the case of pargyline this block is reversible. Inhibition of MAO by pargyline is not reversible.

In these experiments the effect of MAO inhibition by pargyline upon the N, P and LN potentials has been investigated during the onset and recovery from ganglionic block by this drug. Ganglionic potentials have been recorded from rabbit superior cervical ganglion *in vitro*. Pargyline added to the bathing solution in a concentration of 10^{-3} M blocked ganglionic transmission in a reversible manner. This treatment also reduced MAO activity in these ganglia by $98 \pm 1.4\%$ S.E. of mean ($n=5$) as determined by the method of Otsuka & Kobayashi (1964). This inhibition of MAO was not reversible during the period of the experiments (3 h).

To investigate the effects of pargyline on the postulated adrenergic process, the N, P, and LN potentials were produced by addition of tubocurarine to the bathing solution to a concentration of 5×10^{-6} M. Pargyline reduced the size of each potential similarly. The P potential was reduced by $95.2 \pm 3.6\%$ ($n=5$), the N potential by $93.42 \pm 5.3\%$ ($n=5$) and the LN by $91.77 \pm 6.0\%$ ($n=5$). When the ganglia were replaced in the pargyline-free solution the potentials recovered. The P potential recovered to $96.5 \pm 6.1\%$ ($n=4$) in a manner no different from the N and LN potentials which recovered to $98 \pm 4.2\%$ ($n=4$) and $100 \pm 2.2\%$ ($n=4$) respectively. There was no evidence of a selective enhancement, which might be expected if MAO was important in the termination of the process responsible for the P potential.

Therefore, inhibition of MAO alone without the ganglion blocking action of the MAO inhibitor has no selective action on the ganglionic P potential in these experiments. The action of pargyline in blocking ganglia is not related to its inhibition of MAO or to an effect on the postulated adrenergic component of transmission.

REFERENCES

- ECCLES, R. M. & LIBET, B. (1961). The origin and blockade of the synaptic responses of curarized sympathetic ganglia. *J. Physiol., Lond.*, **157**, 484–503.
- DA COSTA, F. M. & GOLDBERG, L. I. (1961). Depression of ganglionic transmission by MO-911. *Fedn Proc. Am. Soc. exp. Biol.*, **20**, 318.
- GERTNER, S. B. (1961). The effects of MAO inhibitors on ganglionic transmission. *J. Pharmac. exp. Ther.*, **131**, 223–230.
- LEVINE, J. (1962). Inhibition of MAO activity in sympathetic ganglia of the cat. *Biochem. Pharmac.*, **11**, 395–396.
- OTSUKA, S. & KOBAYASHI, Y. (1964). A radioisotopic assay for MAO determinations in human plasma. *Biochem. Pharmac.*, **13**, 995–1006.
- REINERT, H. (1963). The role and origin of noradrenaline in the superior cervical ganglion. *J. Physiol., Lond.*, **167**, 18–29.