REFERENCES

BECKETT, A. H., VAUGHAN, D. P. & ESSIEN, E. E. (1972). *J. Pharm. Pharmac.*, **24**, 244.
BECKETT, A. H., MITCHARD, M. & SHIHAB, A. A. (1971). *Ibid.*, **23**, 347–352.
POHLAND, A., BOAZ, H. E. & SULLIVAN, H. R. (1971). *J. medl Chem.*, **14**, 194–197.
SULLIVAN, H. R. & BLAKE D. A. (1972). *Res. Comm. Chem. Path. Pharmac.* **3**, 467–478.
SULLIVAN, H. R. & DUE, S. L. (1973). *J. medl Chem.*, in the press.
SULLIVAN, H. R., DUE, S. L. & MCMAHON, R. E. (1972). *J. Am. chem. Soc.*, **94**, 4050–4051.
SULLIVAN, H. R., SMITS, S. E., DUE, S. L., BOOHER, R. E. & MCMAHON, R. E. (1972). *Life Sci.*, Part I, **11**, 1093–1104.

Post-synaptic blockade of dopaminergic transmission by 6-hydroxydopamine

6-Hydroxydopamine selectively destroys adrenergic nerve terminals and may also block peripheral α-adrenoceptors (e.g. Furness, 1971). Any post-synaptic effects of 6-hydroxydopamine on dopaminergic transmission are likely to be difficult to study because of the mainly central localization of dopamine in vertebrates. The effects of 6-hydroxydopamine reported here were studied on synaptic transmission from a giant dopamine-containing cell in the left pedal ganglion of the water snail *Planorbis corneus*. This cell, discovered in freeze-dried preparations (Marsden & Kerkut, 1970) and shown to contain dopamine by biochemical methods (Berry, Cottrell, Pentreath & Powell, unpublished observations), was identified in living ganglia (Berry & Cottrell, 1973), and offers a unique opportunity to study the transmitter function of dopamine. Post-synaptic responses to stimulation of the giant cell occur in at least 20 neurons in the visceral and left parietal ganglia. Responses are excitatory, inhibitory or biphasic, and appear to result from release of dopamine (Berry & Cottrell, 1973).

When 6-hydroxydopamine ($2{\cdot}5 \times 10^{-4}$ M) was tested on inhibitory post-synaptic potentials (IPSPs) produced in post-synaptic cells by stimulation of the cell, there was a total abolition of response. The effect, which was reversible, was found to be due to a blockade of dopamine receptors; the hyperpolarizing response to applied dopamine was abolished. After the preparation had been washed for 10–60 min, there was a gradual recovery of IPSPs and concomitant recovery of the response to dopamine. There was no effect of 6-hydroxydopamine on the hyperpolarizing response to glutamate shown by the post-synaptic cells, or on depolarizing responses to 5-HT and acetylcholine. A check on the specificity of the responses was made by observing the action on spontaneous inhibitory input and on IPSPs produced by stimulating a nerve trunk. There is evidence that these IPSPs are not produced by dopamine but possibly by glutamate. They were only slightly reduced by 6-hydroxydopamine. Excitatory responses of cells in the parietal ganglion to stimulation of the giant cell were abolished by 6-hydroxydopamine together with the depolarizing response to applied dopamine. The results indicate that 6-hydroxydopamine produces specific abolition of dopaminergic transmission by blocking dopamine receptors in this snail.

Wellcome Laboratories of Pharmacology,
University of St. Andrews,
St. Andrews, U.K.

M. S. BERRY
G. A. COTTRELL

October 2, 1973

REFERENCES

BERRY, M. S. & COTTRELL, G. A. (1973). *Nature, New Biology*, **242**, 250–253.
FURNESS, J. B. (1971). In: *6-Hydroxydopamine and Catecholamine Neurons.* Editors: Malmfors, T. & Thoenen, H. pp. 205–214. Amsterdam & London: North-Holland Publishing Company.
MARSDEN, C. & KERKUT, G. A. (1970). *Comp. Gen. Pharmac.*, **1**, 101–116.