Atropine, in the concentration used does not affect the inactivation of ACh by uptake processes (Polak, 1969) nor does tetrodotoxin alter the potentiating effect of atropine on ACh release *in vitro* (Molenaar & Polak, 1970). Therefore, the observed

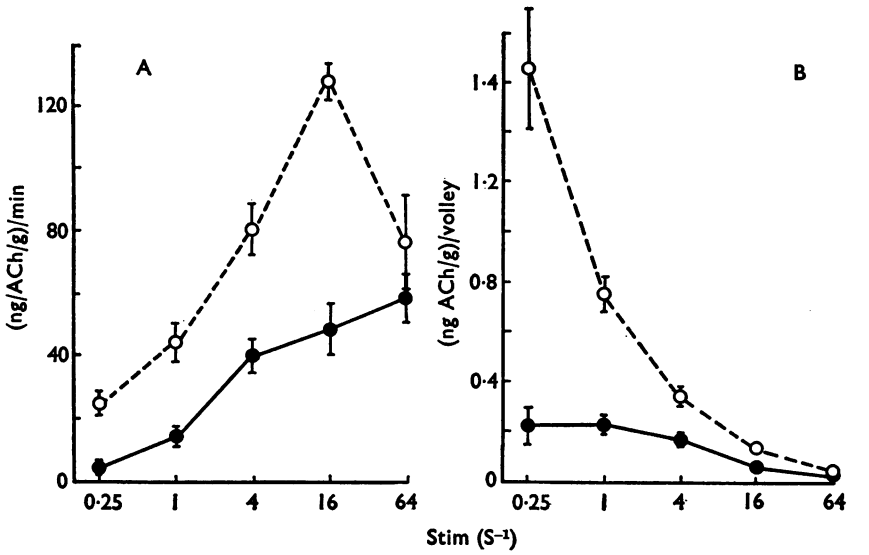

FIG. 1. Release of ACh from cortical slices stimulated at different frequencies. A, Increase in minute output (average minute output during 10 min stimulation minus average minute output during 10 min prestimulation period). B, Volley output (increase in minute output/stimuli per stimulus) ●—● without, ○—○ with 3×10^{-7} M atropine sulphate. Each point is the average ± S.E.M. of three experiments.

potentiation of ACh release by atropine is likely to be due to a presynaptic action which could be an antagonism of a presynaptic inhibitory effect mediated by released ACh.

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Neurally evoked release of noradrenaline from the olfactory bulb

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Previous experiments on the olfactory bulb *in vivo* by Chase & Kopin (1968) have failed to show any specific release of noradrenaline (NA) in response to nervous

stimulation. The problem has been re-examined using a modification of the cortical cup technique (Mitchell, 1963) since it seemed likely that previous failure was due to artifacts associated with the use of push–pull cannulae.

Adult rabbits were anaesthetized with urethane, the frontal cortex removed to facilitate the placement of bipolar stimulating electrodes on the olfactory tract, and both olfactory bulbs exposed. A Perspex cup (7 mm i.d.) containing 0·25 ml Krebs-phosphate solution at 37°C was then applied to both bulbs, and the system made leakproof by the infiltration of paraffin wax (M.P. 37°C) around the cup. Radioactive (±)-noradrenaline (7-^{3}H or methylene-^{14}C, 5 μCi) with either ^{14}C-urea (20 μCi) or ^{3}H-γ-aminobutyric acid (2,3-^{3}H, 5 μCi) (^{3}H–GABA) was then added to the cup fluid and left for 1 hour. Thereafter the Krebs solution contained metaraminol (10^{-5} M). The bulbs were washed twice, and successive 10 min samples taken for estimation of activity of the two differently labelled substances by liquid scintillation counting. The spontaneous and evoked electrical activity in the bulbs was monitored with a unipolar electrode inside the cup.

Initially there was a large spontaneous efflux of the labelled compounds which declined rapidly over 1·5 hours. Thereafter the radioactivity declined more slowly and stimulation studies were made in this latter period. Continuous stimulation of one medial olfactory tract (0·5 ms duration pulses, 6 mA, 15–25 Hz) or direct stimulation of the bulbs for periods of 7 min caused an increase in the efflux, attributable to labelled NA, to levels about 50% greater than in the preceding period. Characteristically these increases did not coincide with the period of stimulation, but followed immediately after. The neurally evoked release of ^{3}H–NA appeared to be calcium dependent and was not accompanied by an increase in the efflux of the marker substance ^{14}C-urea. It could be repeated 3 or 4 times within an experiment, and the size of the release was related to the intensity and rate of stimulation. In preliminary experiments the increased release of ^{14}C-NA, following stimulation of one olfactory tract, was invariably accompanied by an increased release of ^{3}H–GABA.

These results suggest that a neurally evoked specific release of labelled NA from the olfactory bulb can be achieved *in vivo*, and this technique may be of use in analysing the role of NA in synaptic inhibition in the olfactory bulb.

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Effects of an anticonvulsant, acetazolamide, on the sodium gradient and xylose uptake of cerebral cortex slices

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The kinetics of uptake of D-(+)-xylose by the non-raffinose compartment of guinea-pig cerebral cortex slices incubated in a bicarbonate medium suggest that xylose interacts with a membrane component (carrier) during its transport (Gilbert, 1966). The