choline output for at least 5 s after the conditioning pulse. After 10 s the test pulse had only a very small effect, after 20 s the acetylcholine output was about 50% of the normal output and after 60 s there was no longer any interaction between pulses. When the responses of the longitudinal muscle were recorded in the absence of eserine, no such interaction was found even at pulse intervals as short as 2 s. These observations suggest that the suppression of the acetylcholine output from the nerve terminals occurs in the presence but not in the absence of eserine. A poststimulatory inhibition of acetylcholine release in the presence of eserine was also found at higher frequencies (0·1 and 0·5 Hz), but it was not so marked.

Supported by U.S. Public Health Service Grant NB 03026.

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## The release of prostaglandin $E_2$ from the bovine iris

J. POSNER (introduced by G. PATERSON), *Department of Pharmacology, King's College, London, W.C.*2

Ambache (1957, 1959) extracted from iris tissue a pharmacologically active material which he called irin. This material is now thought to contain prostaglandin (Waitzman, Bailey & Kirby, 1967). Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) has been isolated from the sheep iris (Änggård & Samuelsson, 1964) and irides of cat and rabbit are thought to contain $PGE_2$ and $PGF_{2\alpha}$ (Ambache, Brummer, Rose & Whiting, 1966). Mechanical stimulation of the rabbit eye causes the release into anterior chamber perfusates of an active substance (Ambache, Kavanagh & Whiting, 1965). The present experiments investigate the release from the bovine isolated iris of a substance tentatively identified as $PGE_2$.

A loop of iris was cut 3–4 mm from the pupillary margin and suspended in Krebs solution at 37° C and bubbled with 5% $CO_2$ in $O_2$. The tissue was left for 2 h and slowly acquired a constant level of tone; the bath fluid was then collected at hourly intervals, and tested on isolated preparations. The rat fundus, chick rectum and rabbit duodenum responded with contractions resistant to hyoscine, mepyramine and bromolysergic acid diethylamide. The active material partitioned into ether from an acidic aqueous phase and was unaffected by incubation with chymotrypsin. Assay after paper chromatography showed 5-hydroxytryptamine and related substances to be absent from ether extracts. Preparative thin-layer chromatography using the A I and A II systems of Gréen & Samuelsson (1964) indicated the presence of $PGE_2$. Eluates relaxed guinea-pig colon spiral strips which relax to E-type prostaglandins and contract to F-type compounds (Fleshler & Bennett, 1969). Loss of activity of both $PGE_2$ and the extract occurred on mild alkaline hydrolysis whilst that of $PGF_{1\alpha}$ and $PGF_{2\alpha}$ was unaffected. The prostaglandin antagonist SC 19220 (Sanner, 1969) specifically inhibited responses of rat fundus to $PGE_2$ and to the bath fluid extract.

Resting release (mean$\pm$S.E. of mean) from twenty-nine irides assayed on rat fundic strips as $PGE_2$ was (0·91$\pm$0·09 $\mu$g)/g. This release did not appear to be neurogenic in origin since output was unaffected by tetrodotoxin ($2\times10^{-8}$ g/ml) or

procaine ($2\times10^{-4}$ g/ml) or by transmural stimulation at 10 Hz and 0·3 ms for up to 60 s or by storage at 4° C for 48 h.

Release of the material was related to sphincter tone. Incubation with amyl nitrite, 2,4-dinitrophenol, $Ca^{2+}$ free Krebs solution, anoxia or stimulation with high pulse widths for long periods reduced tone and inhibited release of the active material. Output increased as tone increased after first mounting the tissue.

Estimates of prostaglandin content of the bovine iris give a very low level. The results, therefore, suggest that release of the prostaglandin-like substance in the present experiments is not a passive process.

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## Effects of decaborane on gastric secretion in the Shay rat

F. D. HENMAN*, K. H. MOLE and D. M. SHEPHERD, *Department of Pharmacology and Therapeutics, University of Dundee*

Decaborane ($B_{10}H_{14}$) was shown by Merritt & Sulkowski (1967) to be a potent inhibitor of aromatic L-amino-acid decarboxylase. More recently Medina, Londez & Foster (1969) showed that it is also a potent inhibitor of the specific histidine decarboxylase of the rat stomach, causing a reduction in histamine concentration in the stomach.

Rats were pylorus-ligated under ether 12 h after receiving decaborane (0, 15, 30, 45 or 60 mg/kg in vegetable oil 1 ml/kg intraperitoneally). Decaborane produced a dose-related reduction in the concentrations of total acid, free acid and $K^+$ ions and in the volume of gastric secretion. $Na^+$ ion concentrations were increased dramatically. All these effects were statistically highly significant ($P<0.01$). Sodium output was unchanged at 45 mg/kg. Pepsin concentration was unaffected. Gastric histidine decarboxylase activity was virtually abolished. Severe toxicity was seen with the two highest doses.

*In vitro* decaborane inhibited specific histidine decarboxylase and aromatic L-amino-acid decarboxylase, but it had no effect on peptic activity. Decaborane reacted rapidly with pyridoxal phosphate.

The results suggest that this compound preferentially inhibits the secretory mechanisms of the parietal cell in the rat.