Responses of the isolated sphincter of Oddi from the guinea-pig to field stimulation

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Recent reviews (Persson, 1972; Sarles, 1974; Tansy, Innes, Martin & Kendall, 1974) have highlighted the uncertainties concerning the control of the terminal portion of the common bile duct. The present study was undertaken to investigate the neurally mediated responses of the guinea-pig sphincter of Oddi to field stimulation and their modification by drugs.

The choledochoduodenal junction of the guineapig is anatomically noteworthy. The lower end of the common bile duct expands into a large oval pouch lying on the serosal surface of the duodenum. From the caudal end of the pouch a small duct passes directly into the duodenum (Higgins, 1927).

Male guinea-pigs (200 to 1,000 g) were stunned, bled and the terminal portion of the common bile duct and surrounding duodenal areas were removed. A polyethylene cannula (1 mm i.d.) was passed, via the common bile duct, into the pouch and ligated. The duodenum was trimmed to the limits of the pouch. The cannula was attached to a Y-tube one arm of which was connected to a pressure transducer linked to a polygraph, the other was perfused with Krebs solution at the rate of 0.024 ml/min. The preparation was then placed in a bath of Krebs solution maintained at 37°C and bubbled with 5% CO₂ in oxygen. Field stimulation was applied via two platinum loop electrodes (2 cm apart at 50 V, 40 Hz and 0.2 ms pulse width.

The preparations exhibited resting perfusion pressures of 1 to 6 cm H_2O with approximately one third showing spontaneous activity. Field

stimulation resulted in changes in perfusion pressure of up to 25 cm H₂O. The increase in resistance to perfusion with field stimulation was abolished by tetrodotoxin (1 µg/ml) and lignocaine (117 μ g/ml). This stimulation response was also much reduced in 30 of 44 preparations by atropine (28 ng/ml) or hyoscine (152 ng/ml). Acetylcholine (0.05 to 50 μ g/ml) and carbachol (1 to $5 \mu g/ml$) produced increases in perfusion pressure whilst neostigmine (10 to 100 µg/ml) caused an increase in spontaneous activity and a sustained increase in perfusion pressure. Propranolol (5 µg/ml), phentolamine (140 ng/ml) or guanethidine $(1.9 \,\mu g/ml)$ did not affect the response to field stimulation. Noradrenaline (0.5 to $2 \mu g/ml$) in a single instance increased perfusion pressure. Isoprenaline (0.5 to $2 \mu g/ml$) consistently inhibited activity in spontaneously preparations. Nicotine (0.5 to 16 µg/ml) caused a small rise in perfusion pressure in 3 out of 11 preparations. This response was blocked by hexamethonium (10 μ g/ml).

In this preparation, therefore, there is evidence of cholinergic neurones having a motor function, but little evidence for significant adrenergic motor control.

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Peptide hormones and the extinction of conditioned taste aversion

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When a rat is subjected to the unpleasant effects of a drug contingent upon drinking a distinctively flavoured novel solution, it acquires a strong aversion to the taste of that solution (Rozin & Kalat, 1971). This phenomenon is commonly called 'conditioned taste aversion'.

Rats, which were deprived of water for 23.30 h, were allowed to drink a 5% glucose solution ('sugar water') in a single 15 min session. Lithium chloride (LiCl, 0.15 M 10 ml/kg i.p.) injected 30, 60 or 120 min after the conclusion of the drinking session induced strong avoidance of the sugar water at subsequent sessions. Avoidance was less as the interval between drinking and drug treatment