Although the mode of action of cyclic AMP in mediating the natriferic effects of vasopressin is unknown, it is of interest that the effects of frusemide on cyclic AMP binding are seen at concentrations which inhibit natriferic effects of vasopressin in the intact tissue.

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Release by kinin of a substance contracting rabbit aorta (RCS) from guinea-pig lung

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Rabbit-aorta contracting substance (RCS) is released from chopped guinea-pig lung by stirring with a blunt nylon rod (Palmer, Piper & Vane, 1970). Its release is inhibited by low concentrations of anti-inflammatory drugs such as aspirin. These drugs have no effect when superfused over the rabbit aortic strip and thus modify the release of RCS from the lung (Piper & Vane, 1969).

Piper & Vane (1969) showed that bradykinin released RCS from perfused unsensitized guinea-pig lung and this led Collier (1969) to suggest that such an action occurred *in vivo* especially as anti-inflammatory agents had been shown to antagonize some of the *in vitro* actions of bradykinin. The present study was designed to test and extend this proposal.

Krebs solution containing hyoscine, phenoxybenzamine and mepyramine (each at 0·5 μg/ml) with propranolol and cyproheptadine (each at 2 μg/ml) was passed at a rate of 10 ml/min at 34°C through a perspex vessel containing chopped guinea-pig lung. The effluent was made to superfuse a rat stomach strip (to detect prostaglandin-like activity), a rabbit aortic strip (to detect RCS), and a rat duodenum (to detect kinin).

When the lung tissue was stirred for 30 s with a blunt nylon rod, RCS was released and the rabbit aortic strip contracted. It was possible to obtain up to 8 such responses from a single preparation. When aspirin (2–20 μg) was administered to the lung tissue 5 min before stirring, the release of RCS was inhibited (5 experiments). However, a complete inhibition of the release of RACF was obtained (5 experiments) with aprotinin (500–1,000 K.I.U.) and with soya bean trypsin inhibitor (50–100 μg), and this lasted for more than 20 min. When bradykinin (10–100 μg) was given to the lung tissue, stirring resulted in an increased output of RCS which was dose-dependent. The release of prostaglandin, as measured on the rat stomach strip, was not significantly altered during these procedures, and stirring failed to release detectable amounts of bradykinin.

Thus, the potentiation of the RCS release from lung tissue by bradykinin is consistent with the hypothesis that kinins are involved in this process. Furthermore, inhibition of the release of RCS by aprotinin and soya bean trypsin in indicative of the involvement of proteases.

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