Krebs-Henseleit solution at 37°C in an isolated organ bath. The preparations were gassed with a mixture of 95% oxygen and 5% carbon dioxide and contractions were recorded isotonically using a linear motion transducer and pen recorder. Drugs remained in contact with preparations of trachealis and pulmonary artery for 5–10 min with a rest period of 15–20 min between each dose. Bronchiolar preparations responded more slowly and drugs were allowed to remain in contact for 15 min with a 20 min rest period.

Tracheal muscle contracted in response to acetylcholine (0·002–1·0 μg/ml), 5-hydroxytryptamine (0·01–2·0 μg/ml), bradykinin (0·01–0·1 μg/ml) and histamine (0·05–5·0 μg/ml). Contractions in response to acetylcholine, 5-hydroxytryptamine and bradykinin were antagonized by atropine, methysergide and sodium meclofenamate respectively. Antagonism between 5-hydroxytryptamine and methysergide was seen in every preparation in which this was tested (seven) but atropine was only effective as an antagonist to 5-hydroxytryptamine in concentrations one hundred times those which antagonized an equi-effective dose of acetylcholine. These findings do not support the suggestion of Offermeier & Ariëns (1966) that 5-hydroxytryptamine might act in this tissue by releasing acetylcholine.

Bronchiolar muscle also contracted in reponse to acetylcholine, 5-hydroxytryptamine and histamine but this tissue was less sensitive than tracheal muscle, requiring 10–100 times greater concentrations to produce a response. Pulmonary artery contracted in response to 5-hydroxytryptamine (0·005–0·2 μg/ml), histamine (0·02–2·0 μg/ml), bradykinin (0·1–20 μg/ml) and adrenaline (0·005–1·0 μg/ml).

The lung has been found to be the organ principally involved in acute systemic anaphylaxis in cattle (Aitken & Sanford, 1969a), but the mediators involved have not yet been identified. *In vivo* protection against anaphylaxis in cattle has been achieved by administration of sodium meclofenamate (Aitken & Sanford, 1969b) as in the guinea-pig (Collier & James, 1967).

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A sensitive method for the assay of oxytocin in blood.

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A search has been made for an assay tissue selectively sensitive to oxytocin and suitable for use as a blood-bathed organ (Vane, 1964). Intestinal smooth muscle preparations from chicks, rats, desert rats, cats, dogs, rabbits all proved too insensitive or too unspecific. Only the uterus of the desert rat responded to oxytocin at concentrations less than 1 m-u./ml, but this often exhibited spontaneous contractions and was too unspecific.

The sensitivity of the vasculature of the fowl to oxytocin has been exploited as an assay for many years (Coon, 1939) and pulmonary vasculature is particularly sensitive (Somlyo, Somlyo & Woo, 1967). We have confirmed these observations; when superfused with Krebs solution at 5–10 ml/min, spiral strips of pulmonary artery from the hen were contracted by oxytocin (1×10^{-5} u./ml). Preparations of pulmonary artery from pigeons showed a similar sensitivity to those from chickens, but preparations from the duck were 4–5 times more sensitive. The longitudinal muscle of the pulmonary vein of the duck was as sensitive as the spiral arterial strips and more suitable as an assay organ because a plateau response was obtained far more quickly than with arterial strips.

Tension changes were recorded isometrically; the duck pulmonary vein was sensitive to oxytocin (2×10^{-6} u./ml). The preparation was 10–15 times less sensitive to vasopressin than oxytocin and was insensitive to prostaglandins A_1, E_1, E_2 and $F_{2\alpha}$, angiotensin II, bradykinin, substance P, SRS-A, pentagastrin and acetylcholine (all at 10 ng/ml). At this concentration histamine, 5-hydroxytryptamine, noradrenaline and adrenaline contracted the preparation. Exposure of the strips to phenoxybenzamine (1 μg/ml) for 30 min before use abolished the actions of adrenaline and noradrenaline and reduced that of histamine, without affecting the response to oxytocin.

Dogs were anaesthetized with chloralose (100 mg/kg intravenously) and given heparin (1,000 i.u./kg intravenously). Blood from a femoral artery was superfused at 10 ml/min over the duck pulmonary vein preparation (previously treated with phenoxybenzamine). There was a transient contraction, after which the specificity of the preparation was similar to that found in Krebs solution, but the sensitivity was decreased (2–3 fold).

Possible inactivation of oxytocin in the pulmonary vascular bed was tested by comparing the effects of infusions into the vena cava with those into the aorta. No inactivation occurred at infusion rates of 2·8 and 5·6 m-u./kg per min, confirming the results of Biron & Boileau (1969).

Disappearance of oxytocin in one complete circulation was studied by comparing the effects of intravenous infusions with those of infusions through a co-axial intra-aortic catheter (Ferreira & Vane, 1967). From an intra-arterial infusion of oxytocin (5·7–11·4 m-u./kg per min) 14–23% disappeared in one passage through the general circulation.

To detect release of endogenous oxytocin, strips of duck pulmonary vein were bathed in internal maxillary vein blood and in femoral arterial blood. A rabbit rectum preparation (Gilmore & Vane, 1968) was also used to detect vasopressin. Vagotomy or bilateral carotid occlusion after vagotomy gave a release of oxytocin-like material from the head in six out of ten experiments.

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