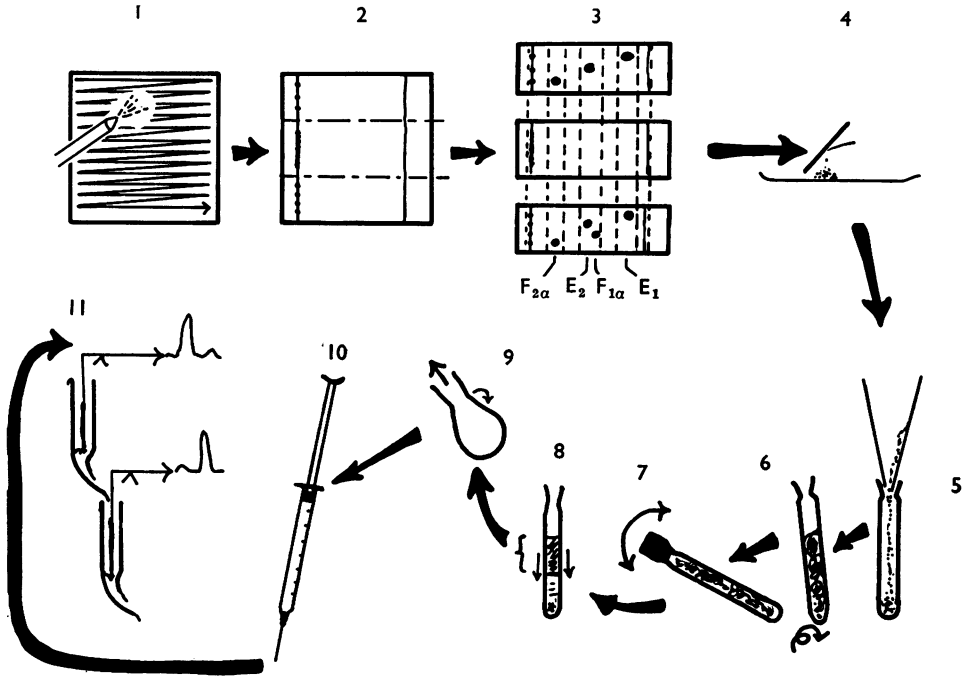

FIG. 1. Separation in the AII system. Using a glass spray (Aimer), the plate was sprayed to saturation with a solution of 10 g of $AgNO_3$ in 100 ml of 86% aqueous ethanol (1). This solution must be used fresh, otherwise substances are formed (probably ethanol oxidation products) which interfere with the separation. The plate was thoroughly dried with a hair drier, followed by 3 h in an oven at 60° C. Marker PGs (5 μg) were applied as single spots of 1 mg/ml solution in ethanol or ethanolic Na_2CO_3 solution. Biological extract was dissolved in 0·5 ml of ethanol and a measured volume applied to the plate as a 5 cm band using a stream of *cool* air to evaporate the ethanol (2). After development, the plate was dried, cut and marker PGs visualized with phosphomolybdic acid (3). Zones of silica gel (-------) were scraped from the extract portion of the plate onto glossy paper (4), transferred into glass-stoppered tubes (5) and eluted into aqueous saline solution (6) which precipitated silver ions as AgCl. After adjusting to pH 3 with HCl, PGs in the eluent were extracted into ethyl acetate mixing the two phases by inversion of the tubes (7). After centrifugation (8), the ethyl acetate extracts were dried on a rotary evaporator at 50° C (9) and dissolved in 1 ml of Krebs solution (10) for bioassay (11).

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The Vidian nerve and the sphenopalatine ganglion in relation to lacrimation in the cat

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The secretory fibres to the lacrimal gland are conveyed by the lacrimal branch of the fifth cranial nerve (Botelho, Hisada & Fuenmayor, 1966; Elsby & Wilson, 1967) but they are believed to leave the brain stem with the seventh nerve (Landolt, 1903). Transfer of these secretory fibres from the Vidian branch of the seventh to the fifth

nerve is thought to take place in the region of the sphenopalatine ganglion (Jendrassik, 1894). Recent observations (Arenson & Wilson, 1970) have shown that in the cat the secretory fibres do not run in the sphenopalatine and infra-orbital nerves to reach the lacrimal gland, as previously thought. In the current investigation, the role of the Vidian nerve and of the sphenopalatine ganglion has been examined in the anaesthetized cat to elucidate the parasympathetic pathway.

After bilateral cervical sympathectomy and removal of the nictitating membrane by cauterization, the brain stem was exposed by removing the occipital bone and cerebellum.

Supramaximal stimulation of the brain stem induced a secretion which was collected from the superior conjunctival fornix by sheathed filter paper strips. This secretion did not occur after removal of the lacrimal gland, showing that under the experimental conditions used, secretion was produced entirely by this gland. In further experiments, section of the Vidian nerve with the lacrimal gland intact reduced the secretion to amounts which were similar to the resting values. The secretion induced by brain stem stimulation could also be reduced or abolished by painting the sphenopalatine ganglion with a solution of nicotine (2%).

The results of this study show that the Vidian nerve contains secretory fibres which relay in the sphenopalatine ganglion. Since it has been shown that the Vidian nerve must be stimulated in a central direction to activate the lacrimal gland (Arenson & Wilson, 1970), it is concluded that the secretory fibres in this nerve must be postganglionic. The site at which these fibres transfer to the fifth nerve is central to the sphenopalatine ganglion. Experiments are in progress in an attempt to determine whether the site is intra- or extracranial.

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The effect of isoprenaline on the responses of the guinea-pig isolated heart to vagal stimulation and to acetylcholine

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Guinea-pig isolated hearts with or without the right vagus nerve were used to study the effect of isoprenaline on the bradycardia elicited by vagal stimulation or by the addition of acetylcholine to the perfusate. The coronary vessels were perfused with a constant volume (7·0 ml/min) of McEwen solution at 29° C. The vagus nerve was stimulated supra-maximally with rectangular pulses of 1 ms duration at the following frequencies: 1, 2, 5, 10 and 20 Hz for 30 s. The heart rate was measured by recording the electrical activity of the sinus node through electrodes attached to the left atrium. Acetylcholine in increasing doses (0·2, 0·4, 0·8, 1·6 and 3·2 μg) was injected into the perfusion cannula.