flask which serves as a vacuum reservoir and waste trap. The side arm of the vacuum flask is connected to a liquid nitrogen vapour trap which in turn leads to a high vacuum pump (Edwards, I.S. 15).

With six wetted Whatman GF/B filters in place, a flow rate of 300 ml/min is achieved through each filter.

Using this apparatus we have found that binding of [^{3}H]-quinuclidinyl benzilate to membrane prepared from rat corpus striatum has a $K_D = 0.43$ nM, and a Scatchard analysis reveals 0.55 pmoles of binding sites/mg protein. These values are in good agreement with those published by Yamamura & Snyder (1974). IC_{50} and RC_{50} values for the inhibition of uptake and release of [^{3}H]-dopamine by (+)-amphetamine in rat striatum synaptosomes were found to be 1×10^{-8}M and 2×10^{-7}M respectively, values in good agreement with those found using centrifugation to separate the synaptosomes (Kruk & Zarrindast, 1976).

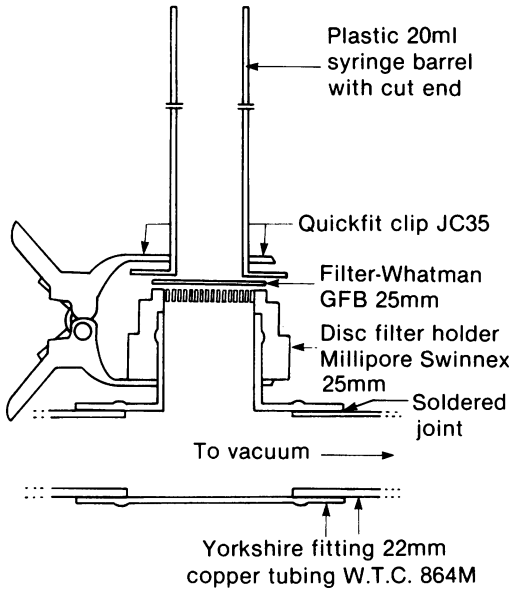

Figure 1 Transverse section of vacuum filtration unit.

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Effect of atropine on prejunctional muscarinic and α-adrenoceptors on postganglionic sympathetic neurones in an isolated blood vessel

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Atropine enhanced the overflow of tritium evoked by electrical-field stimulation of an isolated blood vessel preloaded with [^{3}H]-noradrenaline, which may be due to inhibition of prejunctional α-adrenoceptors (Nedergaard & Schrold, 1975; 1977). Atropine antagonizes the inhibitory effect of acetylcholine on stimulation-evoked tritium release from adrenergic neurones in many tissues (cf. Starke, 1977). The present investigation was undertaken in order to obtain direct evidence for the view that atropine acts on prejunctional α-adrenoceptors and muscarine receptors which both modulate the neuronal transmitter release.

Rabbit isolated pulmonary artery preloaded with [^{3}H]-noradrenaline was used. Atropine (10^{-4}M) and phentolamine (10^{-6}M) increased the stimulation-induced overflow of tritium, while clonidine (10^{-6} to 10^{-5}M) and acetylcholine (10^{-6}M) diminished it. After the overflow had been increased by either atropine (10^{-4}M) or phentolamine (10^{-6}M), clonidine (10^{-6}M) decreased the overflow below the control value. Clonidine (10^{-5}M) prevented the enhancement of tritium overflow evoked by atropine (10^{-4}M). A lower concentration of clonidine (10^{-6}) only caused a partial prevention. Atropine (10^{-7}M), in a concentration which was without any effect on the stimulation-induced tritium overflow, prevented the reduction caused by acetylcholine (10^{-6}M).

We conclude that atropine in a low concentration blocks prejunctional muscarinic receptors; at higher concentrations it blocks in addition prejunctional α-adrenoceptors.

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