Table 1 Effect of potassium depolarization on [^{3}H]-choline uptake by rat sympathetic ganglia

Composition of medium for preliminary incubation and incubation	*Choline uptake* 'High affinity uptake' T/M	% control	'Low affinity uptake' T/M	% control
Control (Krebs bicarbonate Ringer)	6.9 ± 0.4	100	2.68 ± 0.14	100
KCl (40 mM)	9.9* ± 0.8	142*	2.12 ± 0.10*	73*
KCl, Ca free medium	5.1* ± 0.5	68*		
KCl + $MgSO_4$ (20 mM)	5.2* ± 0.2	64*		
KCl, Na free medium	3.5* ± 0.3	51*		
KCl, denervated ganglia	4.5* ± 0.3	54*		
Denervated ganglia (controls)	6.0 ± 0.3	112	2.70 ± 0.27	108

The high and low affinity uptake of choline was estimated by incubating ganglia in a low (0.1 μM) or high (100 μM) concentration of [^{3}H]-choline respectively. The results are expressed as both the tissue : medium ratio (T/M = dpm.g^{-1} wet weight/dpm.ml^{-1}) and as the percentage of uptake in control ganglia not exposed to KCl (% control). The 'T/M column' does not necessarily correspond exactly with the '% control column' since only the latter results are calculated from paired controls. The results (T/M) are the mean ± s.e. mean of 6 to 18 determinations.

* Significantly different from controls ($P < 0.0.5$ and > 0.001).

ACh from nerve terminals, which then results in activation of a high affinity, sodium dependent, uptake process for choline.

A.J. Higgins is an MRC student.

Reference

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Differentiation of neurogenic inhibition from ATP-responses in guinea-pig taenia caeci

N. AMBACHE, SUSAN DALY, S.W. KILLICK & JANETTE P. WOODLEY

Medical Research Council, Department of Physiology, Royal College of Surgeons of England, Lincoln's Inn Fields, London, WC2A 3PN

Twin 3–4 cm preparations were suspended in Krebs-Henseleit (1.5 mM Mg^{++}; 30°C, to reduce spontaneous tone-fluctuations) and contracted by carbachol, 27–164 nM. ATP-tachyphylactic muscles were discarded. ATP-relaxations persisted after phenoxybenzamine (2.3 μM) and pindolol (4–40 μM) but were reversibly antagonized by phentolamine, (2.6–13 μM) acting by an unknown mechanism not involving classical adrenoceptors of this muscle.

Neurogenic inhibition. To eliminate the participation of adrenergic fibres and to single out non-adrenergic inhibitory transmission, unatropinized preparations were rendered insensitive to the relaxing effect of phenylephrine, isoprenaline or noradrenaline by combined α-+β-adrenoceptor block following phenoxybenzamine-treatment (2.3 μM; 15 min) and the introduction of pindolol (40.3 μM). Tetrodotoxin-susceptible inhibitions induced by single 0.1–0.5 ms pulses were compared with relaxations induced by low ATP-concentrations (0.14–1.44 μM for 30 s). In all of 11 experiments, phentolamine (13 μM) greatly reduced ATP-relaxations but left neurogenic inhibitions usually unaltered (Figure 1), or potentiated or slightly reduced.

These results confirm in a different way that illustrated in a 'short report' by Rikimaru, Fukushi & Suzuki (1971), who used 10 times more phentolamine (135 μM); and atropine but no phenoxybenzamine or pindolol. Satchell, Burnstock & Dann (1973) (using guanethidine and hyoscine) failed to confirm this in 3 of 5 experiments with phentolamine (45 μM) and in order to achieve ATP-block raised phentolamine concentration to 180 μM, which then depressed transmission as well. But, under the present conditions, phentolamine in the much lower concentration of 13 μM has regularly differentiated neurogenic from ATP-responses. This, and the frequent occurrence of marked tachyphylaxis to ATP suggest that the non-adrenergic inhibitory transmission is not purinergic and may be akin to autonomic inhibitory transmission elsewhere, e.g. retractor penis, where ATP is excludable because it contracts whereas

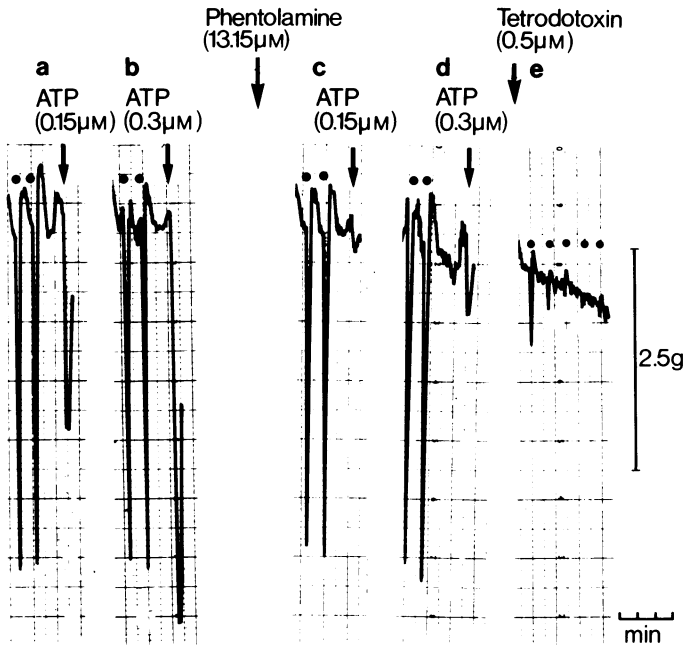

Figure 1 Unatropinized guinea-pig taenia caeci, 30°C; isometric recording. α- and β-adrenoceptors were blocked *ab initio* by phenoxybenzamine (2.3 μM given for 15 min, previously) and pindolol (40.3 μM) in the reservoir. Tone was produced by carbachol (131 nM) renewed before each panel. Panels a–d each show, first, two consecutive inhibitory responses to transmural stimulation with single 0.5 ms pulses of constant voltage, delivered at 1 min intervals, followed, at the arrows, by ATP given for 30 s; the pen recorder was lifted off the paper when ATP was washed out. Phentolamine, (13.15 μM) was introduced 0.5 h before panel c. Panel e shows the extinction of responses to transmural stimulation by tetrodotoxin (0.5 μM). Stimulus deliveries indicated by the dots.

the sacral nerves relax. This effect of phentolamine (13 μmol) was absent in guinea pig vasa or detruser strips (ATP-contractions not antagonized).

References

RIKIMARU, A., FUKUSHI, Y. & SUZUKI, T. (1971). Effects of imidazole and phentolamine on the relaxant responses of guinea-pig taenia coli to transmural stimulation and to adenosine triphosphate. *Tohoku J. exp. Med.*, **105**, 199–200.

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