In the presence of tetrodotoxin (100 ng/ml) the electrical and mechanical responses to low frequency stimulation were abolished. The responses to high frequency stimulation were greatly reduced.

Hexamethonium (100 μg/ml) had little effect upon the electrical or mechanical responses at any stimulation frequency.

In the presence of eserine (2·8 μg/ml) the mechanical responses to stimulation were increased in amplitude and duration. These effects were associated with more prolonged spike activity which at high stimulation frequencies tended to give way to large, slow waves of depolarization.

The effects of these and other agents known to influence the activity of cholinergic neurones provide information about the transmission process and its relatively long latency.

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Augmentation of atropine resistant spasms in plexus-containing guinea-pig longitudinal muscle by ganglionic action of the acetylcholinesterase inhibitor BW 284C51 (1:5 bis(*p*-allyldimethylammonium phenyl)-pentan-3-one dibromide)

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In atropinized preparations (Ambache, Verney & Zar, 1970) field stimulation excites two sets of non-cholinergic neurones in Auerbach's plexus producing 'tetanic spasms' consisting of two pharmacologically distinguishable components, exemplified by response A at 50 Hz and B at 5 Hz (both abolished by tetrodotoxin but unaffected by hexamethonium or nicotine). Response B consisted of one, and A of both components.

BW 284C51 selectively augments component B; this potentiation is abolished by 10^{-4} g/ml pentolinium or hexamethonium, and is therefore due to excitation of 'B' neurones by intra-ganglionic accumulation of acetylcholine.

Responses A and B (Fig. 1) were elicited alternately at 1 min intervals by trains of ten 0·2 ms pulses; B was invariably smaller than A. Brief exposures to BW 284C51, $1{\cdot}8–3{\cdot}6 \times 10^{-7}$ M, increased B well above the height of A; the effect on response A varied from inhibition to augmentation, depending upon the degree of potentiation of its B component.

Mipafox, 4×10^{-6} M, or DFP, 5×10^{-8} M, (dosages specific for butyrylcholinesterase inhibition) did not potentiate either response, but irreversibly facilitated potentiation of component B by BW 284C51; for this reason there was now substantial potentiation also of response A by BW 284C51. Mipafox and DFP, in 1,000× higher dosages

(known to inhibit acetylcholinesterase as well) potentiated response B, as did the non-specific anticholinesterases, eserine and neostigmine.

These results agree with the findings of Ambache, Freeman & Hobbiger (1971) that acetylcholinesterase is localized predominantly in Auerbach's plexus (and is selectively inhibited by BW 284C51) and butyrylcholinesterase, in the longitudinal muscle fibres.

The results also indicate that drugs facilitating ganglionic transmission augment responses which are normally due to excitation of postganglionic fibres. The fact that such augmentation is confined to component B suggests that the 'A' ganglion cells are either not cholinoceptive or are located in Meissner's plexus and therefore lost in this preparation.

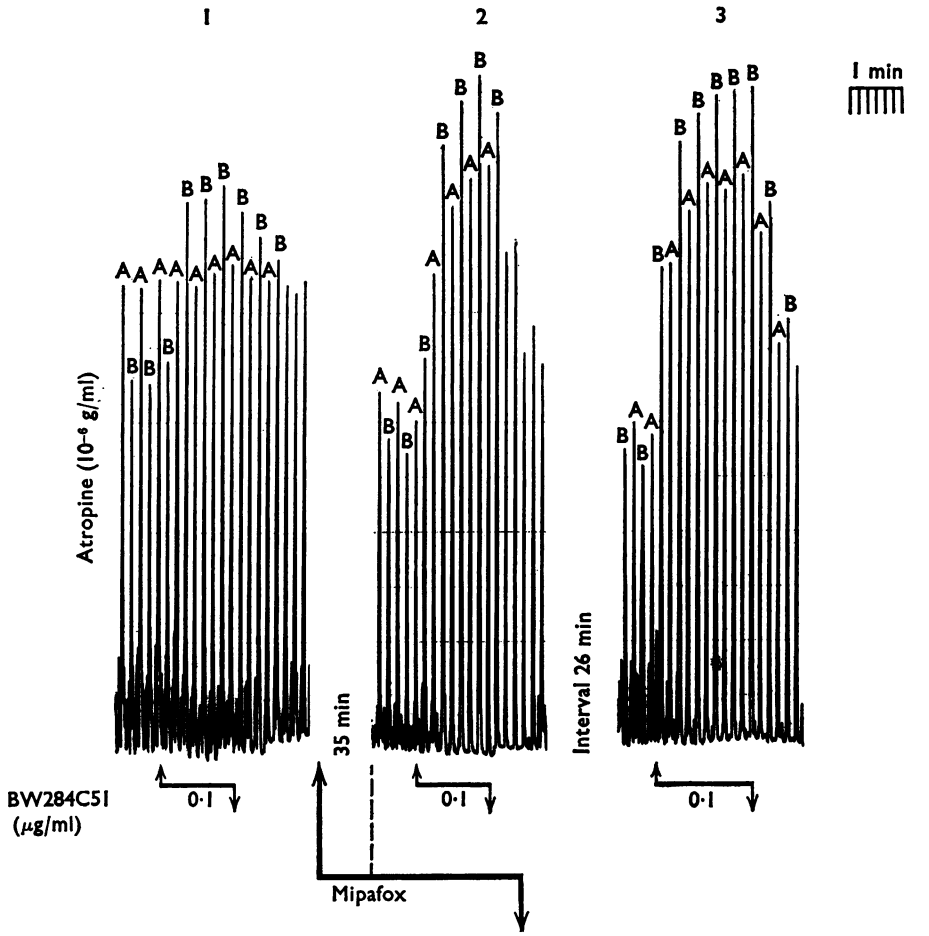

FIG. 1.

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