TABLE 1. *Effect of DHG on the resistance and capacitance vessels in the denervated vascular bed of cat skeletal muscle (calf)*

Dose of DHG μg/kg intra-arterially	Capacitance response expressed as a % of the response to supramaximal sympathetic nerve stimulation*	Mean ± S.E.	Resistance response expressed as a % of the response to supramaximal sympathetic nerve stimulation†	Mean ± S.E.
1·67	0, 0, 0, 0, 0	0± 0	−1, 2, 0, 0, −1	0±0·5
5	65, 0, 0, 0, 0	13±13	1, 2, 1, 0, 0	1±0·5
15	76, 51, 58, 51, 40	55± 6	−1, 0, 2, 1, 1	1±0·5
45	54, 91, 43, 94, 62	69±10	5, 2, 2, 2, 2	3±0·5
135	101, 112, 76, 120, 83	98± 8	7, 17, 5, 6, 6	8±2
405	94, 109, 167, 60, 91	104±18	15, 6, 16, 5, 3	9±2·5

*100% capacitance response to supramaximal sympathetic nerve stimulation mobilization of 27·6% of tissue blood content.

†100% resistance response to sympathetic nerve stimulation 643% increase in resistance.

The results reported here confirm the findings of Mellander & Nordenfelt (1970) and establish the reported selectivity of DHG for the capacitance vessels over a wide dose range in which there is a straight line dose-response relationship.

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Transmission from cholinergic neurones to circular smooth muscle obtained from the rabbit caecum

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The rabbit caecum has a spirally arranged constriction which denotes the position of a fold of the mucous layer known as the spiral valve. In this region there is a thickening of the circular smooth muscle layer. The electrical and mechanical properties of preparations of circular smooth muscle from this region have been studied using the sucrose gap technique of Bülbring & Burnstock (1960).

When bathed by Krebs solution at 37·5°C most preparations were quiescent, both electrically and mechanically. Single pulses of 50V (0·5 ms duration) delivered via two ring electrodes around the live side of the tissue elicited depolarization, a burst of spike activity and the development of tension. The first indication of depolarization followed the stimulus artifact after a latent period of 176±8 ms (mean±standard error, n=30). This relatively long latent period was unaffected by stimulus strength and is comparable to that observed at other cholinergic neuro-effector junctions (Burnstock, Campbell, Bennett & Holman, 1964; Ohashi & Ohga, 1967; Creed & Wilson, 1969).

A train of stimuli was applied for 1 s every 100 seconds. The tension response increased with stimulation frequency up to a maximum at 64 Hz. The effects of increasing the stimulation frequency upon the electrical response were complex but generally included an increase in spike activity or a more prolonged period of depolarization.

Atropine (100 ng/ml) abolished the mechanical response of the tissue to stimulation and the electrical response was reversed in sign. Single pulses in the presence of atropine generated small hyperpolarizations which summated at frequencies over 4 Hz.

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