THE CORRELATION BETWEEN VIP AND NON-ADRENERGIC, NON-CHOLINERGIC INHIBITORY RESPONSES IN THE ANTRAL CIRCULAR MUSCLE OF THE RAT STOMACH

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The purpose of the present experiment is to investigate the electrical and mechanical responses to stimulation of non-adrenergic, non-cholinergic (NANC) inhibitory nerves of antral circular muscles of the rat stomach with special reference to the properties of the transmitter.

1. Electrical and mechanical activities of the smooth muscles were monitored with microelectrode and sucrose-gap methods. Spontaneous slow waves with or without superimposed spike potentials were recorded from the circular muscles of the antrum every 20 to 30 sec. Contractions appeared synchronously with the slow waves.
2. In the presence of atropine (10^{-6}M) and guanethidine (10^{-6}M), transmural stimulation (TMS) with a single pulse evoked an inhibitory junction potential (I. J. P.). TMS with a frequency of less than 1Hz produced I. J. P. s, and those with a frequency of over 1Hz resulted in hyperpolarization. Both I. J. P. and hyperpolarization were accompanied by the inhibition of slow waves and spike potentials. TMS also evoked frequency-dependent relaxation and inhibition of contractions of the smooth muscle.
3. Apamin (10^{-7}M) inhibited both I. J. P. and hyperpolarization evoked by TMS. In the presence of apamin (10^{-7}M), however, TMS still caused the inhibition of slow waves and spike potentials, which were followed by a relaxation and inhibition of spontaneous contractions of the smooth muscle. Apamin-resistant inhibitory responses to TMS were completely abolished by tetrodotoxin.
4. In the presence of atropine and guanethidine, VIP caused a dose-dependent relaxation and inhibition of spontaneous contractions of the smooth muscle. VIP reduced the amplitude of slow waves and abolished spike potentials, but did not cause hyperpolarization. Apamin had no effect on VIP-induced inhibition of mechanical activities.
5. VIP-like immunoreactive substance (VIPLI) released from the antral smooth muscle in response to a continuous TMS for 5min, was measured by radioimmunoassay. TMS evoked VIPLI release, the amount of which was not affected by apamin.
6. These results indicate that VIP mimics the apamin-resistant electrical and mechanical responses of antral smooth muscles to stimulation of NANC inhibitory nerves, and that endogeneous VIPLI was released in the presence of apamin. It is suggested that VIP is partly involved in NANC inhibitory responses in the antral circular muscle of the rat stomach.