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Reversal of devazepide on antagonistic effect of CCK-8 on morphine on electrical and mechanical activities of rat duodenum *in vitro*

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Abstract

AIM: To study the antagonistic effect of cholecystokinin octapeptide (CCK-8) on morphine and its mechanism.

METHODS: The method of simultaneously recording the electrical and mechanical activities of rat duodenum *in vitro* was adopted.

RESULTS: The results showed that the average amplitude and num ber of spike potentials (SPA and SPN), after injection of acetylcholine (Ach, 300 nmol/L) of 22 duodenal segments *in vitro* respectively increased from 0.70 ± 0.07 mV ($\bar{x}$ ± s) before injection to 0.94 ± 0.09 mV, and 2.71 ± 0.23 to 3.88 ± 0.15, followed by the increase of the average amplitude of contractions (CA) from 16.40 ± 1.00 mm to 24.44 ± 1.63 mm. This SPA, SPN and CA were reduced to 0.59 ± 0.06 mV, 2.71 ± 0.09 and 13.54 ± 1.04 mm after administration of morphine (330 nmol/L), respectively. But the SAP, SPN and CA were respectively increased to 0.81 ± 0.07 mV, 3.52 ± 0.13 and 22.73 ± 1.00 mm after injection of CCK-8 (0.7 nmol/L). Using CCK-A receptor antagonist (Devazepide, 10 nmol/L), the SPA, SPN and CA were reduced to 0.54 ± 0.05 mV, 2.66 ± 0.18 and 13.67 ± 0.66 mm, respectively. Moreover, all of them showed extremely significant differences ($P < 0.001$) when the latter was compared with the corresponding item of the former.

CONCLUSION: The results firstly demonstrated that CCK-8 could antagonize the effect of morphine which inhibited the potentiation of Ach on the electrical and mechanical activities of rat duodenum *in vitro*, whereas deva-zepide could reverse the anti-morphine effect of CCK-8. It is suggested that the antagonistic effect of CCK-8 on morphine was mainly mediated by CCK-A receptor, and thus provide a new clue for the clinical treatment of disturbances in intestinal movement function.

Key words: Duodenum/Physiology; Duodenum/drug effects; Cholecystokinin/Pharmacology; Morphine/Pharmacology; Devazepide/Pharmacology

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