



Relationships between electrogastric dysrhythmias and leucin enkephalin in rats

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Author contributions: All authors contributed equally to the work.

Original title: *China National Journal of New Gastroenterology* (1995-1997) renamed *World Journal of Gastroenterology* (1998-).

Received: December 11, 1995

Revised: January 31, 1996

Accepted: March 1, 1996

Published online: September 15, 1996

Abstract

AIM: Electrogastric dysrhythmias are very common in clinic. It correlates with gastrointestinal diseases, but its pathogenesis is rarely understood. In the present study, we established rat model of electrogastric dysrhythmias and investigated the relationships between dysrhythmias and enkephalin.

METHODS: Sixty wistar rats were randomly divided into 3 groups: (1) control ($n = 20$); (2) test group ($n = 30$). Rats were fed irregularly for 4 wk: fasted for 24-h every other day, drinking water randomly, 1 mL HCl was added to water to disturb the pH of stomach. Then the silver electrodes were implanted on antrum. 7 d after operation, gastric myoelectric activity, the contents of leucin enkephalin (L-ENK) in plasma and tissues of antrum and duodenum were measured by RIA, the distribution and number of LEK immunoreactive nerves in duodenal myenteric plexus were measured by immunochemistry and image analysis system. (3) Drug group ($n = 10$). To investigate the effects of morphine (0.1 mg/kg, ip), naloxone (0.1 mg/kg, ip.) and morphine after pretreatment with naloxone on gastric myoelectric

activity.

RESULTS: (1) A short tachygastria or arrhythmia occurred only in 2 rats of control. Dysrhythmia index was 1.25% in control. Dysrhythmias occurred in 27 rats in test group. Dysrhythmia index was 36.67%. (2) The content of L-ENK in plasma had no significant difference between control (55.49 ± 23.41 ng/g) and test group ($65.38\% \pm 19.37\%$ ng/g) ($P > 0.05$), but the content of L-ENK in the tissues of antrum and duodenum were significantly higher in test group (5.75 ± 2.13 ng/g; 21.64 ± 8.73 ng/g) than in control group (3.62 ± 1.94 ; 12.37 ± 4.93 ng/g) ($P < 0.01$). The number of L-ENK immunoreactive nerves in duodenal myenteric plexus was significantly increased in test group compared with control group ($P < 0.01$). (3) Morphine induced dysrhythmias in all rats after ip.administration, but naloxone didn't. After naloxone pretreatment, morphine didn't induce dysrhythmias in 9 rats, a short bradyrhythmia occurred only in 1 rat.

CONCLUSION: We successfully established rat model of electrogastric dysrhythmias. Morphine could induce electrogastric dysrhythmias, the effect could be reversed by naloxone. The increase of L-ENK in antral, duodenal tissues and myenteric plexus played an important role in electrogastric dysrhythmias.

Key words: Electrogastric dysrhythmias; Morphine; Naloxone

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Zhang Y, Wang ZH. Relationships between electrogastric dysrhythmias and leucin enkephalin in rats. *World J Gastroenterol* 1996; 2(Suppl1): 81 Available from: URL: <http://www.wjgnet.com/1007-9327/full/v2/iSuppl1/81.htm> DOI: <http://dx.doi.org/10.3748/wjg.v2.iSuppl1.81>

E- Editor: Liu WX



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