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Effects of nitrergic nerves on development of electrogastric dysrhythmias in rats

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Abstract

AIM: To investigate the effects of nitrergic nerves on occurring of electrogastric dysrhythmia, the rat model of electrogastric dysrhythmia was used.

METHODS: 70 adult Wistar rats were randomly divided into three groups. (1) Control group (20 rats). (2) Test group (40 rats), gastric myoelectric activities were recorded after the rats were fed irregularly for 4 wk; The distribution of nitrergic, cholinergic (SS-IR) and substance P immunoreactive (SP-IP) nerves were shown in antral and proximal duodenal myenteric plexus in the rats with histochemistry or immunohistochemistry technique, then their contents and interrelationships were investigated. (3) Drug group (10 rats), gastric myoelectric activities were recorded followed by injecting intraperitoneally sodium nitroprusside (NaNP), L-NAME, substance P (SP), sandostatin and atropine respectively.

RESULTS: (1) The abnormal rhythm index (38.50%) and the coefficient of variation (cv) of slow wave frequency (23.91%) were significantly increased compared with control ($P < 0.01$), fast waves were also abnormal, *i.e.* their frequencies and amplitudes were significantly decreased compared with control ($P < 0.01$). (2) Compared with control, the contents of nitrergic nerves and SS-IR nerves in myenteric plexus in test group rats were significantly increased ($P < 0.05$), and the contents of SS-IR were significantly correlative with the contents of notrergic nerves ($P < 0.01$); The contents of cholinergic and SP-IR nerves were significantly decreased ($P < 0.05$), and were not significantly correlative with the contents of nitrergic nerves. (3) The electrogastric dysrhythmias were induced by injecting NaNP, atropine or somatostatin analogue sandostatin respectively, while the electrogastric rhythm was not varied significantly by injection of L-NAME might block the effects of sandostatin on electrogastric rhythm, while SP might inhibit the effects of NaNP on electrogastric rhythm.

CONCLUSION: (1) The increase of release of NO or exogenous NO can induce electrogastric dysrhythmias. (2) The effects of peptidergic nerves on gastric myoelectric activities are mediated by nitrergic and/or cholinergic nerves. (3) The abnormal proportion of nitrergic nerves to cholinergic nerves plays the most important role in the occurring of electrogastric dysrhythmias.

Key words: Electrogastrography; Electrogastric dysrhythmia; Nitrergic nerve

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