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TRADITIONAL MEDICINE

Effect of muscarinic blocker on enhancing the action of fructus aurantii immaturus on intestinal myoelectric activity in dogs

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Abstract

AIM: To investigate the effect of fructus aurantii immaturus (FAI) on small intestinal electrical activity in dogs.

METHODS: The effect of FAI was observed using a computerized electrophysiological method with the migrating myoelectric complex as a criterion. Fasted, healthy, and conscious dogs were given 100% FAI concentrated solution by gastrostogavage, and as soon as the effect on small intestinal electrical activity appeared, atropine was injected intramuscularly.

RESULTS: The enhancing action of FAI was inhibited significantly by atropine, a cholinergic receptor antagonist. Both the number of spike bursts per cluster and the number of spikes per minute in phase Ⅱ and Ⅲ and the general cycle were decreased ($P < 0.01$), although the duration of phase Ⅱ and the general cycle was prolonged.

CONCLUSION: The effect of FAI might be related to the muscarinic receptors.

Key words: Fructus aurantii immaturus; Small intestine; Atropine; Electrophysiology

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INTRODUCTION

Fructus aurantii immaturus (FAI) is a Chinese herb for reestablishing vital energy. It can promote *qi* (vital energy) circulation, disperse the accumulation of evils and phlegm, relieve flatulence, and so on[1]. Based on small intestinal electrical activity as enhanced by FAI concentrated solution, we used the muscarinic blocker atropine to affect the action of FAI to reveal the pharmacological mechanism of FAI.

MATERIALS AND METHODS

Drug administration

FAI concentrated solution was prepared according to the method of Bi *et al*[2]; atropine was manufactured by the Yan Cheng Pharmaceutical Factory.

Laboratory method

Three healthy dogs weighing 10-20 kg were anesthetized using 3% pentobarbital sodium (1 mL/kg *i.v.*). Two pairs of platinum bipolar electrodes were implanted in the subserosa of the duodenum (4 cm below the junction of the stomach and intestine) and jejunum (5 cm below the ligament of Treitz), respectively. The diameter of the electrodes was 0.3 mm and the distance between two electrodes was 1.5 mm; the electrode wires were passed through the abdominal muscle and fixed in the skin between the shoulder blades. A nylon gastrostoma tube was inserted in to the stomach, from which gastric juice was aspirated or drugs injected.

Conscious dogs fasted 12-24 h but with free water-drinking were placed in a shielding facility. All data were handled by a four-channel physiological recorder and a Zijin 2-A computer; the 150-min migrating myoelectric complex (MMC) cycle, number of spikes per minute, spike bursts per cluster, and histograms were printed immediately.

After recording one MMC cycle, 100% FAI concentrated solution (1 mL/kg) was administered by gastrostogavage in the fifth minute of phase Ⅰ of the second cycle. As soon as the spike decreased, 0.5 mg atropine was administered intramuscularly and one or two MMC cycles were recorded again.

Table 1 Comparison of the duration of each phase and the general cycle before and after administration of drugs ($\bar{x} \pm s$)

	No.	Migrating Myoelectric Complex				General cycle
		Phase Ⅰ	Phase Ⅱ	Phase Ⅲ	Phase Ⅳ	
Control	13	31.92 ± 17.21	53.46 ± 28.90	5.38 ± 1.85	11.38 ± 10.80	104.85 ± 12.85
FAI + atropine	13	26.85 ± 18.22	102.23 ± 51.41[b]	4.38 ± 2.02	9.00 ± 9.18	148.38 ± 45.87[b]

[b]$P < 0.01$ compared with control group. FAI: Fructus aurantii immaturus.

Table 2 Comparison of the number of spikes per cluster before and after administration of drugs (number/cluster, min, $\bar{x} \pm s$)

	No.	Migrating Myoelectric Complex				General cycle
		Phase Ⅰ	Phase Ⅱ	Phase Ⅲ	Phase Ⅳ	
Control	13		3.55 ± 2.23	4.55 ± 2.40	4.55 ± 2.40	4.14 ± 2.10
FAI + atropine	13	Some cycles had no spike	2.45 ± 1.18[b]	4.66 ± 2.11[b]	3.92 ± 2.46	2.79 ± 1.21[b]

[b]$P < 0.01$ compared with control group. FAI: Fructus aurantii immaturus.

Table 3 Variation of the number of spikes per minute before and after the use of drugs (number/min, $\bar{x} \pm s$)

	No.	Migrating Myoelectric Complex				General cycle
		Phase Ⅰ	Phase Ⅱ	Phase Ⅲ	Phase Ⅳ	
Control	13		26.82 ± 17.86	117.02 ± 46.60	42.44 ± 30.78	27.58 ± 15.90
FAI+atropine	13	Same as in Table 2	15.15 ± 9.55[b]	89.40 ± 45.93[b]	36.11 ± 35.97	15.35 ± 9.82[b]

[b]$P < 0.01$ compared with control group. FAI: Fructus aurantii immaturus.

Thirteen recordings in all were made before and after the administration of FAI and atropine.

RESULTS

The comparison of the duration of each phase and the general cycle before and after FAI and atropine administration is shown in Table 1. Atropine was injected in to the dogs as soon as the spike burst was induced by FAI. The duration of phase Ⅰ shortened from 31.92 min ± 17.21 to 26.85 min ± 18.22. The duration of phase Ⅱ and the general cycle was prolonged ($P < 0.01$), indicating that FAI can induce spike burst and enhance intestinal electrical activity. The shortened duration of phase Ⅲ may have been related to the effect of atropine in inhibiting the spike burst, as in phase Ⅲ, the spike loading slow wave encompassed over 95% of the slow wave. The reduced spike decreased the spike loading slow waves.

The comparison of the number of spikes per cluster before and after FAI and atropine administration is shown in Table 2. The mean number of spikes per cluster in phase Ⅱ and Ⅲ and the general cycle was decreased ($P < 0.01$). The results reveal that the spike induced by FAI was inhibited significantly by atropine.

The changes in the number of spikes per minute before and after FAI and atropine administration are shown in Table 3.

After atropine was administered, the number of spikes per minute in phase Ⅱ, phase Ⅲ, and the general cycle was decreased ($P < 0.05$), suggesting that the spike induced by FAI was inhibited significantly by atropine.

DISCUSSION

MMC is a well-known sensitive index for evaluating gastrointestinal motion and regularity[3]. Gastrointestinal contractile activities are identical to the MMC. Four sequential phases (Ⅰ-Ⅳ) of MMC, defined in terms of action potential activity, appear to correspond to the resting (quiescence), preceding irregular, strong, and subsiding contractions[4].

Smooth muscle cells may suddenly discharge one or more action potentials under the condition of depolarization. These spikes usually accompany the contraction of smooth muscle. In many slow waves, spikes appear continuously. Smooth muscle cells produce action potential and contract correspondingly with the periods of depolarization and hyperpolarization[5].

In phase Ⅱ, if stronger spikes continuously occur on three or four basic electric rhythms, the corresponding segment of the small intestine will undergo peristalsis. In phase Ⅲ, if 95% basic electric rhythm loads high-frequency and -amplitude spikes, intense metameric activity takes place in that intestinal segment. There are many more spikes in each spike cluster in phase Ⅲ than in phase Ⅱ. It manifests in strengthened movement of the small intestine with the increase of spikes in each spike cluster[6].

The intestinal tract is mainly governed by parasympathetic nerves[7]. The small intestine has a complex enteric nervous system. Its terminal neuron belongs to the cholinergic neurons. Its role is mainly related to motion that can be blocked by atropine.

FAI can strengthen the phasic contraction and tension of intestinal smooth muscle, shorten the duration of phase Ⅰ and the general cycle, and prolong the duration of phase Ⅱ. When small intestinal electrical activity began to be enhanced by FAI, atropine inhibited the enhancing effect of FAI, prolonging the duration of phase Ⅱ and the general cycle; decreasing the number of spikes per cluster and the number of spikes per minute in phase Ⅱ, phase Ⅲ, and the general cycle; and shortening phase Ⅳ. Atropine can inhibit the spike induced by FAI, suggesting that the enhancing action of FAI on small intestinal electrical activity may be related to the muscarinic receptor.

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