



Effect of endogenous nitric oxide on gastrointestinal propulsive motility inhibited by hyperglycemia in rats

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Abstract

AIM: Hyperglycemia inhibited antral motility and gastric emptying. Motilin increased gastric motility. Hyperglycemia also inhibited the release of motilin. And motilin reduced the release of endogenous nitric oxide inhibited gastrointestinal motility. The propose of this study was to investigate the effect of endogenous nitric oxide on gastrointestinal propulsive motility by intravenous high concentration of glucose in rats.

METHODS: 27 rats (154-308 g) were studied. Group 1 ($n = 8$): First, intravenous injection of NS (0.1 mL/100 g body weight) was done; Second, after 15 min. NS (0.1 mL/100 g body weight) was injected intravenously ; Third after 15 min, the toner (5% toner plus 10% Gum Acacia Power, 1 mL/200 g body weight) was introduced intragastrically; Fourth 30 min. Later, after de capitation, the abdomen was opened and then the distance traveled by toner meal through the gastrointestinal tract (from cardia to the end of rectum),

measured in terms of percentage of its total length, was recorded as the index of propulsive motility. Group 2 ($n = 7$): In second step, 20% glucose (0.1 mL/100 g body weight) was injected intravenously and other steps were the same as group 1. Group 3 ($n = 6$): In first step, L-nitro-arginine methyl ester (L-NAME, 0.3 mg/100 g body weight) was injected intravenously and the other were the same as group 2. Group 4 ($n = 6$): Pretreatment with L-arginine (L-ARG, 30 mg/100 g body weight) intravenously 15 min later, then the operation was the same as group 3.

RESULTS: (1) The percentages of distances from group 1 to group 4 were 78.36 ± 10.34 , 58.44 ± 3.98 , 79.04 ± 13.10 , 62.49 ± 3.96 . (2) 20% glucose significantly inhibited the gastrointestinal propulsive motility ($P < 0.01$). (3) L-NAME significantly increased the gastrointestinal propulsive motility against 20% glucose ($P < 0.01$). (4) The excitatory effect of L-NAM E on gastrointestinal propulsive motility could be reversed by pretreatment with L-ARG ($P < 0.05$).

CONCLUSION: (1) Hyperglycemia significantly inhibited gastrointestinal propulsive motility in rats. (2) Endogenous nitric oxide, at least partly, mediated this inhibitory effect of hyperglycemia.

Key words: Hyperglycemia; Gastrointestinal motility; Nitric oxide

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