Comparison of gastric peristalsis inhibition by scopolamine butylbromide and glucagon: evaluation by electrogastrography and analysis of heart rate variability

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Introduction

Anticholinergics are generally administered during morphological examinations of the gastrointestinal tract, but they sometimes produce side effects such as palpitations, urinary disturbance, and accommodation disorder, particularly in patients with prostatic hypertrophy or heart disease.1,2 Therefore, glucagon is frequently used for such patients because it inhibits peristalsis by acting directly on gastrointestinal smooth muscle.3–6

Unlike scopolamine butylbromide, glucagon may inhibit gastric peristalsis by relaxing the stomach via glucagon receptors on smooth muscles.9,10 However, no studies have been conducted to date evaluating the antigastric peristalsis action of glucagon from the perspective of autonomic nervous activity and electrogastrographic findings. In the present study, we evaluated autonomic nervous activity based on electrogastrograms (EGGs) and analysis of heart rate variability to compare the gastric peristalsis inhibiting action of glucagon and scopolamine butylbromide.
Subjects and methods

Subjects

The subjects were 60 healthy volunteers (41 men, 19 women; mean age 33.9 ± 7.5 years) with no past history of cardiopulmonary disease. These subjects showed normal physical findings and a resting blood pressure of 140/90 mmHg or below. There were no abnormalities on standard 12-lead electrocardiography (ECG), chest radiography, urinalysis, or clinical laboratory findings; and these subjects were clinically diagnosed as healthy. Drinking and eating were prohibited from 9:00 the evening before the examination until the studies, which were performed between 9:00 in the morning and 12:00 noon.

The healthy volunteers were randomly divided into the following three groups: G group consisting of 20 subjects (15 men, 5 women; mean age 33.3 ± 8.9 years) who received an intramuscular injection of glucagon (1 mg); SB group consisting of 20 subjects (13 men, 7 women; mean age 33.9 ± 7.5 years) who received an intramuscular injection of scopolamine butylbromide (20 mg); and C group consisting of 20 subjects who received physiological saline (1 ml) injection alone (13 men, 7 women; mean age 34.5 ± 7.9 years). Holter's ECG and percutaneous EGG were continuously recorded from 1 h before to 2 h after administration of glucagon or scopolamine butylbromide. Written informed consent was obtained from all subjects before examination.

Percutaneous electrogastrography

Percutaneous EGG was performed using an electrogastrograph (Nipro EG; A&D, Tokyo, Japan). As shown in Fig. 1, four surface probe electrodes (channels 1–4) were placed around the stomach, and a central electrode (C) was placed at the center of the line between the navel and the xiphoid process to record EGG patterns by bipolar leads.14 The sampling cycle was 1 s, and the measurement frequency was 2.1–6.0 cycles/min (cpm). In addition, data recording was performed at 13 bits, and the influence of respiration was completely removed by a 10th filter. Using a linear-phase filter, the influence of body movement was minimized to decrease the strain of EGG signals. Data recorded on a portable EGG weighing 300 g were transferred to a personal computer (Windows 98) via RC232C, and fast Fourier transformation (FFT) analysis of 512-point data was performed using EG software (Nipro EG; A&D, Tokyo, Japan). Dominant frequencies and their amplitudes (peak powers) were obtained from four-channel EGGs before and after administration of glucagon, scopolamine butylbromide, or physiological saline; the mean values were calculated.

Analysis of heart rate variability

To remove the influence of postural change on autonomic nerve activity, a two-channel Holter's ECG (CM5 and CC5 leads) was continuously recorded in the decubitus position from 1 h before to 2 h after intramuscular administration of glucagon or scopolamine butylbromide using an SM-50 (Fukuda Denshi, Tokyo, Japan). The magnetic tapes used to record Holter's ECG were replayed and analyzed using a work station (DMW-900H system; Fukuda Denshi). Subsequently, FFT analysis was performed after transferring R-R interval data to the personal computer (Windows 2000) via a communication cable (RS232C) using Holter's ECG data processing software (Fukuda Denshi). R-R interval data before and after the occurrence of a premature beat were excluded from the analysis to avoid the influence of the premature beat. Subsequent data analysis was performed by interpolating data missing due to the exclusion of the R-R interval data before and after a premature beat.14

Heart rate variability analysis was performed using R-R interval data from 256 heartbeats. As heart rate variability power spectra, the low frequency (LF) power (0.04–0.15 Hz) and high frequency (HF)