TITLE: EFFECT OF I-653(DESFLURANE) ON SOMATOSENSORY EVOKED POTENTIALS

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This study evaluated the effect of I-653 on somatosensory evoked potentials (SSEPs).

After IRB approval and obtaining informed consent, 5 adult patients scheduled for elective abdominal aortic aneurysm resection were induced with thiopental and maintained with I-653 (up to 1.75 MAC) in O_2, fentanyl ($\leq$ 600mcg), midazolam ($\leq$ 2 mg), and vecuronium. In all patients, ECG, O_2 saturation, temperature, end expired O_2, CO_2, and I-653, MAP, PAP, PCWP, and RAP were monitored. Normocarbia and normothermia were maintained. For SSEP recording a multichannel signal averager (Nicolet Pathfinder I) was used. Median nerve or posterior tibial nerve stimulation was used. Stimulus duration was 200 msec, intensity 10 MA for upper extremity stimulation and 20 MA for lower extremity and stimulation rate 7.3/s for upper extremity and 4.3/s for lower extremity with band pass filters of 30 to 1000 Hz. SSEPs were recorded prior to placement of the aortic crossclamp during periods of hemodynamic stability. SSEPs were recorded when end tidal I-653 was zero (control) and at multiple end expired I-653 concentrations following establishment of a steady state at that level. Amplitude and latency of the first cortical potential at each anesthetic level were compared to control for that patient.

Increasing concentrations of I-653 resulted in no consistent changes in latency of cortical SSEPs, but a dose dependent decrease in the amplitude of the cortical SSEP was noted(TABLE). In no patients were the SSEPs obliterated at any concentration of I-653.

As with other volatile anesthetics I-653 results in dose dependent decreases in amplitude in cortical SSEP, but unlike the other volatile anesthetics no consistent increases in latency were noted.[1,2] At up to 1.75 MAC I-653 the ability to monitor reliable SSEPs was preserved.

References

1. Anesthesiology 70: 207-212, 1989.
2. Anesthesiology 62: 626-633, 1985.

TABLE

% DECREASE IN CORTICAL AMPLITUDE VS MAC

MAC	% Change (+/- SD)
0.25	-7.2 (12.2)
0.50	5.6 (47.7)
1.0	37.1 (22.5)
1.5	45.0 (20.3)
>1.5	61.9 (17.8)

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TITLE: EFFECT OF PROSTAGLANDIN E_1 ON THE GASTRIC SECRETION IN HUMANS DURING ANESTHESIA

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Introduction. Prostaglandin E_1 (PGE_1) has not only a hypotensive effect but also various other physiological actions. Classen et al[1] reported that intravenous administration of PGE_1 reduces the volume and acidity of gastric juice in humans. However, the effects of PGE_1 on the gastric secretion during general anesthesia have not been clarified. In this study, changes in the gastric secretion were evaluated in patients who underwent contiuous infusion of PGE_1 for the control of blood pressure during general anesthesia.

Methods. Thirty-three patients scheduled for elective surgery of ASA physical status 1-2 were studied. Anesthesia was induced with thiamylal and maintained with N_2O(66%) and enflurane (1-2%). PGE_1 was administered by continuous infusion at a rate of 50-200 $ng.kg^{-1}.min^{-1}$ in 16 patients who required hypotensive medication. In the PGE_1 group, gastric juice was collected serially 3 times before and during administration and 1 hour after discontinuation of PGE_1. In the control group (17 patients), gastric juice was collected at times corresponding to those in the PGE_1 group. The pH, acidity and pepsin activity of gastric juice were measured.

Results. Figure depicts change in the pH of gastric juice under general anesthesia. The change in acidity was similar to that in the pH. The pepsin activity was significantly decreased during and after infusion of PGE_1.

Discussion. The pH, acidity and pepsin activity of gastric juice were all suppressed by PGE_1, indicating that PGE_1 inhibits gastric secretion at doses that produce a sufficient hypotensive effect under general anesthesia. The action of PGE_1 to reduce the pH of gastric juice was considerably strong, being comparable to that of 20 mg famotidine, a H_2 antagonist[2]. The gastric antisecretory effect of PGE_1 is very unique pharmacological features, and may be promising for the prevention of aspiration pneumonia and gastric stress ulcer in perioperative period.

References. 1)Digestion 4:333-344, 1971
2)Anesth Analg 68:541-544, 1989

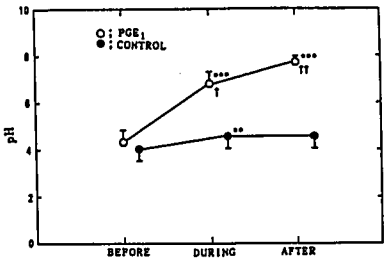

Figure: Changes in the pH of gastric juce. Mean± S.D. **p<0.01 vs pre-infusion. ***p<0.001 vs pre-infusion. +p<0.05 vs control value. ++p<0.01 vs control value.