The Effects of rapid increases in desflurane and sevoflurane concentrations on spirometry in humans during balanced anesthesia with remifentanil.

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Introduction

High concentrations of desflurane may irritate the airway and consequently increase airway resistance (1). The aim of this study was to assess the effects of rapid increases of desflurane and sevoflurane concentrations during bolus administration on spirometry parameters in humans during balanced anesthesia with remifentanil.

Methods

After Institutional Ethics Committee approval, written informed consent was obtained from 30 ASA I patients scheduled for minor non-laparoscopic surgery. Induction was with TCI remifentanil, propofol and rocuronium (0.9 mg.kg^{-1} IBW). TCI remifentanil was initiated at 4 ng/ml and increased by 25% or decreased by 25% in order to maintain mean arterial pressure and heart rate within 20% of baseline during anesthesia. Patients were randomized into 2 groups (n = 15) to receive BIS-guided desflurane or sevoflurane targeted to maintain a BIS-value between 45 and 55. When BIS $\geq$ 55 for more than 30 s, an inhalation bolus was administered. The bolus administration of desflurane and sevoflurane was performed by setting the vaporizer to maximum output in a fresh gas flow of 4 L/min during 15 s. Afterwards, fresh gas flow was returned to baseline 2 L/min with a 25% increased vaporizer setting. This resulted in 45 boli desflurane and 58 boli sevoflurane. To assess the differences between spirometric data in a period 30 s before and 300 s afterwards, all data were synchronized on the start of the inhalation bolus. At every second the difference between the mean value and the 95% confidence intervals (CI) were calculated (group desflurane – group sevoflurane). Significance is reached when zero is not included in the 95% confidence intervals.

Results

Patient characteristics were comparable for the two groups. Mean end-tidal concentrations before bolus were 0.5 (0.12) MAC desflurane and 0.49 (0.18) MAC sevoflurane($p = 0.46$). Bolus administration resulted in mean peak inspiratory concentrations of 10.8 (1.6) vol% desflurane and 5.4 (1.2) vol% sevoflurane ($p < 0.05$). Pplateau was significantly lower during the 15 s bolus administration of desflurane. Airway resistance was lower in the desflurane group and did not differ significantly from the sevoflurane group (Fig. 1).

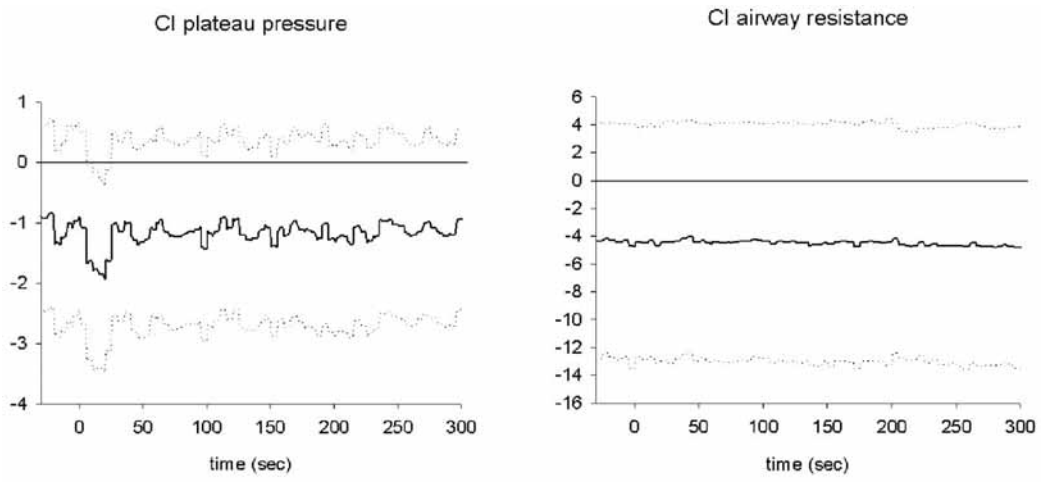

Fig. 1. — Time synchronised analysis of the differences in plateau pressure and airway resistance between groups. The difference of the means is plotted as continous line; the differnce in upper and lower 95% CI as dotted lines.
Significance is reached when zero is not included between the dotted lines.

Discussion

Our results are in conflict with the work of Goff and colleagues (1). When combined with remifentanil, high inspired concentration of desflurane did not provoke bronchoconstriction. Compared to sevoflurane, desflurane does have bronchodilating properties (2). Repetitive increases in desflurane concentrations might blunt airway responses (3).

Conclusions

Repetitive increases in inspiratory and end-tidal concentrations during inhalation boli of desflurane and sevoflurane did not result in increased airway resistance.

References

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