

Sevoflurane Induction in the Elderly

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Summary

The effects of 2% and 4% sevoflurane in oxygen and nitrous oxide for induction of anaesthesia in 60 unpremedicated elderly patients was compared to those obtained during an intravenous Thiopentone induction. Intravenous induction induced anaesthesia in 27 ± 5 seconds, significantly faster than a 2% or 4% sevoflurane induction (109 ± 36 and 71 ± 24 seconds respectively). One patient in both the thiopentone and 2% sevoflurane groups, and 2 patients in the 4% sevoflurane group coughed during induction. The postinduction reduction in mean arterial pressure was greatest in the thiopentone group followed by the 4% and the 2% sevoflurane groups. Heart rate changes were minimal in all groups. We conclude that 2% or 4% sevoflurane offered suitable conditions for induction of anaesthesia in the elderly with minimal cardiovascular derangement.

Key Words: Anaesthetics volatile, Sevoflurane, Anaesthesia, Geriatric, Anaesthetic techniques, Induction

Introduction

The use of sevoflurane for inhalational induction has been extensively studied in the paediatric population. The low blood gas solubility coefficient and the lack of pungency are ideal properties that facilitate induction of anaesthesia. The effects of its use in the elderly is however, not well defined. The physiological effects of aging may alter the pharmacokinetics and pharmacodynamics of many anaesthetic drugs and result in undesirable cardiorespiratory side effects. The aim of our study is to evaluate the induction characteristics and cardiovascular parameters in the elderly patients during an inhaled induction with tidal volume breathing of sevoflurane 2% or 4% and compared to the conditions obtained during a conventional intravenous thiopentone induction.

Materials and Methods

Ethics committee approval and informed consent was obtained. Sixty unpremedicated ASA I-II two patients aged 60 years and above who were scheduled for elective surgery were randomly assigned into three groups using sealed envelopes. Patients were excluded if they have known sensitivity to any of the drugs used in the study, a difficult airway is anticipated, or a history of gastroesophageal reflux is present.

Patients were induced with sevoflurane 2% and 4% in 67% nitrous oxide and oxygen, or intravenous thiopentone 5mg/kg. For the patients who were to receive inhalational induction, the anaesthetic circuit was primed with a fresh gas flow of 6L/min (4 L/min nitrous oxide and 2L/min oxygen) with the patient end of the circuit connected to a reservoir bag. Sevoflurane was

added and the gases circulated using the ventilator (Narkomed 4™, North American Drager) until the desired sevoflurane concentration was achieved, the sampling being done at the mask elbow connector. The patients were breathing room air before induction of anaesthesia. Patients who received the inhaled induction and were instructed to maintain their tidal volume and respiratory rate and breathe the gaseous mixture with tidal breaths till anaesthetised (loss of eyelash reflexes).

Monitors included an automatic blood pressure monitor, an infrared gas monitor, electrocardiography, pulse oximetry, and capnography. Hemodynamic parameters were monitored at baseline and subsequently every minute for the first 5 minutes during the induction period. The induction time, presence of "induction events" such as coughing, breath holding, laryngospasm, movement and secretions were recorded.

Demographic data were compared between the groups using Student t-test and chi-square test where appropriate. The haemodynamic parameters between the groups were compared using analysis of variance for repeated measurements. A p value <0.05 was considered statistically significant. Sample size was based on an one-tailed alpha value of 0.05, with a 90% power of study to detect a 30 seconds difference

in the time to loss of eyelash reflex between 2- and 4% sevoflurane, with the standard deviation of 30% of population mean. This yielded 19 patients per group.

Results

The 3 groups were similar in demographic data (Table I). Intravenous thiopentone reliably induced anaesthesia in 27±5 seconds, significantly faster than the induction time obtained from a 2% or 4% sevoflurane induction (109±36, 71±24 seconds respectively) (p<0.001). The induction times between the 2- and 4% groups were also statistically significant (p<0.01). One patient in both the thiopentone and 2% sevoflurane groups, and 2 patients in the 4% sevoflurane group had mild cough during induction. Oxygen saturation as measured by pulse oximetry remained at 98% to 100% throughout the study period in all patients. Continuous electrocardiographic monitoring did not show any arrhythmia or ST segment changes during the induction period.

There was a significant decrease in the mean blood pressure postinduction from their respective baseline and this was greatest in the patients who received intravenous thiopentone for induction of anaesthesia followed by those patients who received 4% sevoflurane for induction (maximal decrease by 18%) and least in those who received 2% sevoflurane induction

Table I
Demographics and Induction Time

Group	Thiopentone	Sevoflurane 2%	Sevoflurane 4%
Weight, kg	63.3±8.8	60.0±9.4	59.5±11.4
Height, cm	160.5±7.8	160.5±7.4	163.4±8.1
Age, years	68.5±7	72.3±8.8	70.1±8.9
Sex M/F	10/10	7/13	11/9
Induction time, secs	27±5	109±39*	71±24*+

Values expressed as mean±SD

* p<0.05 compared to Thiopentone group

+ p<0.01 compares to 2% group

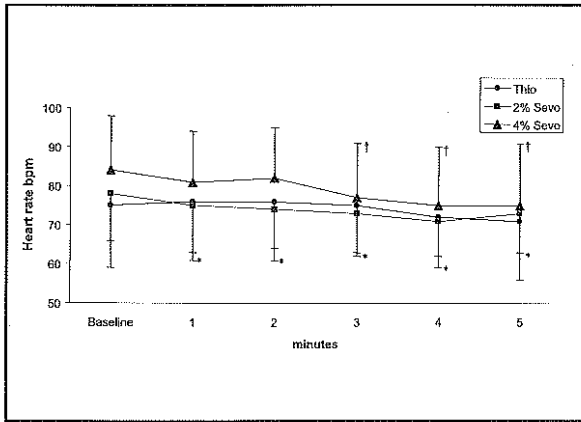


Fig. 1: Changes in mean arterial pressure during induction of anaesthesia. Mean arterial pressure was significantly different from baseline in both Thiopentone and 4% sevoflurane groups from the first minute onwards while it was statistically significant different from baseline from the 2nd minute postinduction onwards in the 2% sevoflurane group.

* $p < 0.05$, compared to baseline

† $p < 0.05$, compared to baseline

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(approximately 10%) (Figure 1). There was an associated reduction in mean heart rate with a 5% decrease in patients who received intravenous induction while those patients who had the gaseous induction had a statistically significant reduction of about 8 and 10% in patients who received 2% and 4% sevoflurane respectively (Figure 2).

Discussion

Our results indicated that an inhalational induction via the tidal breath technique using

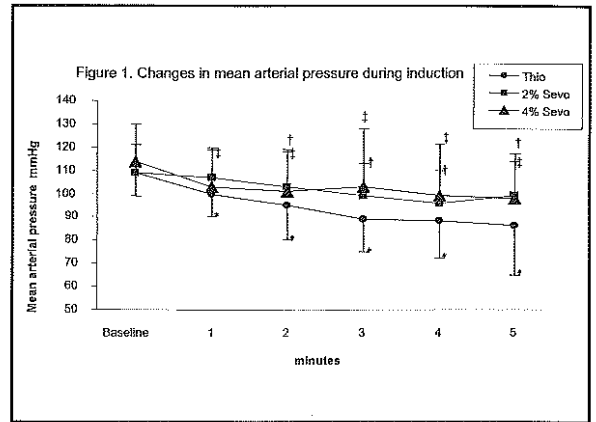


Fig. 2: Changes in mean heart rate during induction of anaesthesia. Heart rate changes were significantly different from baseline from the 1st and 3rd minute postinduction in the 2% and 4% sevoflurane groups respectively. * $p < 0.05$, compared to baseline † $p < 0.05$, compared to baseline

sevoflurane in nitrous oxide and oxygen provided a reliable induction that was safe, practical with acceptable cardiovascular changes. The suitability of sevoflurane induction in elderly patients via the vital capacity technique was shown by Walpole and Logan and separately in another study by Gravel *et al*^{1,2}. Walpole and Logan found that a 4% or 8% sevoflurane in 50% nitrous oxide induced patients in about 2 minutes. The maximal reduction in mean arterial pressure in their patients was about 30% from baseline values while heart rate remained unchanged in the patients who received the higher concentration of sevoflurane. The incidence of "induction events" was however, high, with almost 50% of all patients studied developing one or more complications¹. Gravel and associates compared the haemodynamic effects of 4% sevoflurane in oxygen to a total intravenous technique using midazolam and propofol in premedicated patients

scheduled for elective coronary artery bypass graft procedures². None of the patients who received the inhaled induction via the vital capacity breath technique developed "induction incidents". The authors observed a reduction in heart rate, cardiac index and arterial pressure during the induction period. The average systolic pressure in the patients who received sevoflurane induction was significantly lower than those who received intravenous induction (102 Vs 120mmHg). In addition, more patients in the sevoflurane-induced group required glycopyrrolate to treat heart rate <80% baseline. We had chosen to study the lower range of sevoflurane-concentration induction (2- and 4%) in this group of elderly patients to determine the efficacy and the haemodynamic changes, which had not been previously reported. Consistent with these two studies, there was a reduction in arterial pressure and mean heart rate postinduction in our patients. We observed that in his group of elderly patients, the greatest decrease in mean blood pressure occurred in the patients who received intravenous induction, followed not unexpectedly in the group of patients who received the higher concentration of sevoflurane. We are not aware of any studied comparing the cardiovascular effects of thiopentone with sevoflurane in the elderly, but the dose-dependent cardiovascular effects of the two concentrations of sevoflurane is evident from our data. However, the lowest mean arterial pressure observed in the 4% sevoflurane group patients of 85mmHg were within clinically acceptable limits and none of the patients required vasopressor therapy. In contrast to the significant number of patients in Gravel's group that required rescue therapy as a result of bradycardia, the minimal mean heart rate obtained in all 3 groups were also within clinically acceptable limits. These differences are probably related to the use of premedication and the presence of coronary artery disease in Gravel's study.

The main methodological issue that is relevant when comparing the effects of an inhalational with an intravenous induction pertains to the

equability of the agents used. The MAC equivalents in 60% nitrous oxide and oxygen for 2% and 4% sevoflurane in an elderly population with mean age of 60 years was about 2.3 MAC and 4.5 MAC respectively³. Equivalence of the two techniques used cannot be confirmed, as there was no monitoring of the depth of anaesthesia. We have based the doses used in this study on our clinical experience and the results obtained from our pilot study. As such, the haemodynamic changes may be related to the difference in the equivalence of the two techniques. In addition, we have chosen to use the tidal breathing rather than the vital capacity technique as our elderly population cannot achieve or hold onto a vital capacity breath effectively. Even though the vital capacity technique has been shown to induce patients in a slightly faster time and is associated with less "induction events"⁴, we felt that the variability that exists in our elderly population is too great despite coaching that the results that may be obtained would be questionable. Oxygenation was well maintained in other studies employing the vital capacity technique for inhalational induction, with or without preoxygenation⁵⁻⁷. This is probably related to the vital capacity volume of an enriched oxygen-air mixture in the circuit and the lungs when the induction began¹. Similar to the other studies employing the tidal breathing technique, none of our patients who received inhalational induction had any episodes of desaturation during the induction process^{4,8}. One must however consider the slower induction times of 1 to 2 minutes as demonstrated in our studies against the potential greater haemodynamic changes associated with the use of higher concentrations of sevoflurane.

In conclusion, while an intravenous induction with thiopentone predictably induced patients more rapidly, inhalation induction with 2% or 4% sevoflurane is a safe and viable option to the conventional intravenous induction. In addition, the use of 4% sevoflurane resulted in a faster induction time with similar haemodynamic profile and no increase in complications during induction when compared to 2% sevoflurane.

References

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