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# Effects of rapid inhalation induction with sevoflurane-oxygen anesthesia on epidural pressure in humans

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**Abstract:** In this study, we chose sevoflurane as the volatile anesthetic for rapid inhalation induction (RII) and investigated its usefulness. We also assessed how RII with sevoflurane affected epidural pressure, and compared RII with rapid intravenous induction by thiopental on epidural pressure. The results were as follows: RII with 5% sevoflurane had a shorter induction time compared with published results on RII with other volatile anesthetics like halothane and isoflurane, and was accompanied by fewer complications. When RII with sevoflurane was attempted, epidural pressure increased significantly upon exhalation to residual volume just before induction and during laryngoscopy and endotracheal intubation compared with the preinduction value. There was no significant difference in epidural pressure between the two induction methods during laryngoscopy and endotracheal intubation. Epidural pressure measurements are reportedly useful in monitoring intracranial pressure. Consequently, in patients with increased intracranial pressure, exhaling to residual volume and increasing arterial blood pressure during laryngoscopy and endotracheal intubation should be avoided. The results of this study suggest that RII with 5% sevoflurane in itself is safe and useful, and that it is unlikely to increase intracranial pressure as compared with rapid intravenous induction by thiopental.

**Key words:** Rapid inhalation induction, Sevoflurane, Epidural pressure

## Introduction

Rapid inhalation induction (RII) is a rapid induction method that employs high concentrations of volatile anesthetics from the beginning [1–7]. This study was conducted to investigate the usefulness of sevoflurane as a volatile anesthetic for RII. We also assessed how RII with sevoflurane affected epidural pressure, and compared RII with rapid intravenous induction by thiopental on epidural pressure.

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## Materials and methods

Sixteen adult patients (nine men and seven women, ASA physical status I or II) undergoing elective upper-abdominal surgery were selected as subjects. They were free of neurologic and cardiovascular diseases. Their ages ranged from 30 to 64 years (mean ± SD, 45.6 ± 10.5). The patients were randomly assigned to two groups: RII with sevoflurane ($n = 9$) and rapid intravenous induction by thiopental ($n = 7$).

Institutional approval and informed consent from all of the patients were obtained. An epidural catheter was inserted 2 h before starting this study. Following sterile preparation and draping of each patient in the right lateral decubitus position, lidocaine (1%, 3–4 ml) was injected subcutaneously and intradermally at the T9–10 or T10–11 intervertebral space. The epidural space was identified with a 17-gauge Tuohy needle by a paramedian approach. Entry of the needle point into the epidural space was confirmed by the loss-of-resistance technique with a physiological-saline-filled syringe. An 18-gauge epidural catheter (Hakko, Tokyo, Japan) was inserted through the needle and the catheter was positioned 2–3 cm cephalad in the epidural space. Neither blood nor cerebrospinal fluid was aspirated through the catheter in any of the patients. A test dose was not used.

Premedication with hydroxyzine hydrochloride 50 mg and atropine sulfate 0.5 mg was administered by intramuscular injection 30 min before arrival in the operation room. In the operation theater, the patient was placed in the supine position. Before anesthesia

induction, the epidural catheter was filled with physiological saline and connected to a transducer (Transpac, Abbott Critical Care Systems, North Chicago, Illinois, USA). Direct arterial blood pressure (radial artery) was recorded simultaneously on a polygraph (Life Scope 12, Nihon Kohden, Tokyo, Japan). All transducers were calibrated to zero at the level of the patients' external auditory meatus.

The RII method was carried out as follows: first, using a Jackson-Rees breathing circuit, the patients were preoxygenated with 100% oxygen at a flow rate of 8 L/min for 5 min using a mask. Induction was started with a full 4 L reservoir bag to allow for inspiration of a full vital-capacity breath. Mixtures of 5% sevoflurane and oxygen were delivered by a vaporizer ($VIP_{\Sigma}$, Penlon, UK) into the circle system of an anesthesia machine (Vip-202, IMI, Saitama, Japan). The pop-off valve was open at all times. Respiratory gases were sampled into the multi-gas monitor (ULTIMA, Datex, Helsinki, Finland). Following preoxygenation, the patients were instructed to breathe out to residual volume and hold their breath at residual volume while the circuit was replaced with the preprimed circle system. They were then instructed to take a vital capacity breath and attempt to hold this breath as long as they could, preferably until loss of consciousness ensued. Following initial vital capacity breathing, patients were allowed to resume spontaneous respiration. Loss of consciousness was defined as the loss of both the eyelash reflex and the response to the verbal command, "Open your eyes". These were repeated at 5-s intervals until the patients failed to respond. Just after loss of consciousness, the trachea was intubated orally with intravenous succinylcholine at 1 $mg \cdot kg^{-1}$.

The time when patients resumed spontaneous breathing after holding their breath, and the time of the onset of unconsciousness were recorded. The five complications of induction of anesthesia, coughing, laryngospasm, breath holding, movements, and secretions, demonstrated by Lamberty et al. [5], were recorded. Systolic and diastolic blood pressure and heart rate were measured before induction as the control, 30, 60, and 90 sec after starting induction, and when laryngoscopy and endotracheal intubation were being attempted. Epidural pressure was also recorded throughout this study.

The rapid intravenous induction method was carried out as follows: thiopental at 5 $mg \cdot kg^{-1}$ was administered intravenously, and just after loss of the eyelash reflex, the trachea was intubated orally with intravenous succinylcholine at 1 $mg \cdot kg^{-1}$. Systolic blood pressure, diastolic blood pressure, heart rate, and epidural pressure were measured before the start of induction as the control, and during laryngoscopy and endotracheal intubation.

Statistical analysis of the data obtained was performed using two-way analysis of variance followed by the Dunnett procedure, and the paired and unpaired *t*-test. $P < 0.05$ was considered statistically significant. Values are shown as mean ± SD.

## Results

There were no significant differences in age, body weight, height, systolic blood pressure, diastolic blood pressure, heart rate and epidural pressure between the two groups before induction (Table 1).

In the RII group, the time until patients resumed spontaneous breathing after holding their breath was 45.6 (± 16.7) s. The onset of unconsciousness occurred at 84.3 (± 18.6) s. During induction, complications such as coughing, laryngospasm, breath holding, and secretions were not observed, but one patient exhibited movements.

The traces of systolic and diastolic blood pressure, and epidural pressure before and after the start of induction, are shown in Fig. 1.

Systolic blood pressure decreased gradually after the start of induction. At 90 s, systolic blood pressure was

**Table 1.** Patient characteristics

| | Rapid inhalation induction group | Rapid intravenous induction group |
|---|---|---|
| *n* | 9 | 7 |
| Sex (M/F) | 4/5 | 5/2 |
| Age (years) | 45.6 ± 10.5 | 49.9 ± 13.4 |
| Body weight (kg) | 56.1 ± 3.6 | 63.5 ± 11.3 |
| Height (cm) | 158.2 ± 8.2 | 158.0 ± 9.7 |
| Systolic blood pressure (mmHg) | 149.4 ± 18.4 | 144.1 ± 13.8 |
| Diastolic blood pressure (mmHg) | 76.1 ± 7.9 | 67.7 ± 8.1 |
| Heart rate ($beat \cdot min^{-1}$) | 81.9 ± 14.5 | 73.0 ± 20.1 |
| Epidural pressure (mmHg) | 10.9 ± 3.1 | 11.3 ± 2.4 |

Values are expressed as mean ± SD.

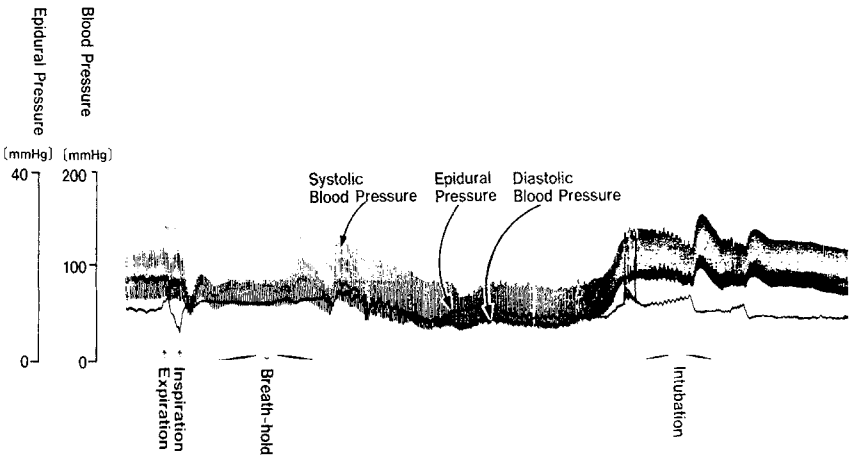


**Fig. 1.** Traces of systolic and diastolic blood pressure, and epidural pressure before and after induction in the rapid inhalation induction group

significantly lower than the control value. Systolic blood pressure reached its highest value during laryngoscopy and endotracheal intubation, and it was significantly higher than the control value (Table 2).

Diastolic blood pressure also decreased gradually after the start of induction, and at 90 s diastolic blood pressure was significantly lower than the control value. Diastolic blood pressure reached its highest value during laryngoscopy and endotracheal intubation, and it was significantly higher than the control value (Table 2).

Heart rate did not change significantly until 90 s after the start of induction. Heart rate during laryngoscopy and endotracheal intubation was the highest, and it was significantly higher than the control value (Table 2).

Epidural pressure increased significantly upon expiration to residual volume just before induction, and during laryngoscopy and intubation (Table 3).

In the rapid intravenous induction group, the onset of unconsciousness occurred at 20.3 (± 6.5) s. Systolic blood pressure, diastolic blood pressure, heart rate, and epidural pressure during laryngoscopy and endotracheal intubation were significantly higher than the control values (Table 4).

There were no significant differences between the two groups in these parameters concerning the control values and the values at laryngoscopy and intubation.

## Discussion

RII is a rapid induction method in which a high concentration of volatile anesthetic is used from the beginning [1–7]. Ruffle et al. [1] first described the technique of RII: A vital capacity of 4% halothane was inhaled and held in the lungs for 30–90 s, and this was followed by tidal breathing until consciousness was lost. In 15 of the 16 subjects, unresponsiveness to command was shown after 2 min of induction. Wilton and Thomas [2] used a single breath of 4% halothane in 67% nitrous oxide to induce anesthesia in 100 outpatients by a similar procedure: vital capacity inspiration followed by a breath-hold. Apnea was present for approximately 30 s, and the eyelash reflex was lost after almost 80 s. Seven patients coughed and six moved during induction. Loper et al. [3] showed that in patients pretreated with fentanyl at 5 $\mu g \cdot kg^{-1}$ to attenuate the cough reflex, time

**Table 2.** Systolic blood pressure, diastolic blood pressure, and heart rate before and after the start of induction in the rapid inhalation induction (RII) group

| | Before the start of induction (control) | After the start of induction | | | |
|---|---|---|---|---|---|
| | | 30 s | 60 s | 90 s | Laryngoscopy and endotracheal intubation |
| Systolic blood pressure (mmHg) | 149.4 ± 18.4 | 146.6 ± 24.9 | 139.4 ± 21.3 | 121.9 ± 21.3* | 186.9 ± 26.3* |
| Diastolic blood pressure (mmHg) | 76.1 ± 7.9 | 74.3 ± 12.1 | 73.1 ± 10.7 | 64.4 ± 14.3* | 109.1 ± 22.0* |
| Heart rate ($beat \cdot min^{-1}$) | 81.9 ± 14.5 | 85.6 ± 18.4 | 83.4 ± 17.8 | 84.3 ± 18.2 | 96.0 ± 11.2* |

Values are expressed as mean ± SD.
* $P < 0.01$ vs. control.

**Table 3.** Epidural pressure before and after the start of induction in the RII group

| | Before the start of induction | | | After the start of induction | | | |
|---|---|---|---|---|---|---|---|
| | Control | Expiration to residual volume | Inspiration in deeply | 30 s | 60 s | 90 s | Laryngoscopy and intubation |
| Epidural pressure (mmHg) | 10.9 ± 3.1 | 16.0 ± 4.7* | 9.1 ± 3.1 | 12.4 ± 2.6 | 12.0 ± 3.7 | 10.6 ± 3.7 | 17.9 ± 5.8* |

Values are expressed as mean ± SD.
* $P < 0.01$ vs. control.

**Table 4.** Systolic blood pressure, diastolic blood pressure, heart rate, and epidural pressure before the start of induction (control) and during laryngoscopy and endotracheal intubation in rapid intravenous induction group

| | Before the start of induction (control) | Laryngoscopy and endotracheal intubation | Significance |
|---|---|---|---|
| Systolic blood pressure (mmHg) | 144.1 ± 13.8 | 207.9 ± 22.3 | $P < 0.001$ |
| Diastolic blood pressure (mmHg) | 67.7 ± 8.1 | 116.4 ± 10.3 | $P < 0.0005$ |
| Heart rate (beat·min$^{-1}$) | 73.0 ± 20.1 | 109.6 ± 15.7 | $P < 0.005$ |
| Epidural pressure (mmHg) | 11.3 ± 2.4 | 15.1 ± 4.1 | $P < 0.05$ |

Values are expressed as mean ± SD.

to loss of consciousness was significantly longer with 3.5% halothane than with 5% isoflurane (vs 86 s, 38 s respectively). Lamberty and Wilson [5] used 2% isoflurane with 66% nitrous oxide in oxygen for induction, and 19% of the patients exhibited complications such as coughing, laryngospasm, breath holding, movements, and secretions. Yurino and Kimura [6] compared vital capacity rapid inhalation induction of anesthesia with 3% sevoflurane and 2% isoflurane in oxygen. The mean time for induction of anesthesia with sevoflurane (120 s) was significantly shorter than that with isoflurane (145 s). They tried induction using 4.5% sevoflurane with 66.6% nitrous oxide in oxygen [7]. The mean time required for induction was 53.8 s, and coughing or movements occurred in two of the 32 subjects. In our study, we also chose sevoflurane as the volatile anesthetic for RII. In comparison with previously published results, sevoflurane is the most useful volatile anesthetic for RII because it is associated with fewer complications, and because supplemental agents such as fentanyl are not always needed. Furthermore, it takes less time for induction even if nitrous oxide is not added since sevoflurane has lower blood-gas solubility. If very rapid induction is needed, higher concentrations of sevoflurane with nitrous oxide in oxygen are strongly recommended.

While induction was being attempted, patients were instructed to try to hold their breath as long as they could. The reason is as follows: an anxious patient who is mildly hypocapnic tends to remain apneic for a longer period after the onset of anesthesia until sufficient carbon dioxide is retained to provide a stimulus to substitute for conscious anxiety as a drive to breathing. The breath-holding part of this induction technique appears to be useful because carbon dioxide is retained, and provides an alternative stimulus to ventilation when consciousness is lost [8].

In this study, there were no significant differences between the two groups in epidural pressure during laryngoscopy and intubation. In the RII group, it was found to be especially high during expiration to residual volume just before induction and during laryngoscopy and intubation. Sevoflurane reportedly increases intracranial pressure [9–11]. Epidural pressure measurements seem to be valuable for estimating cerebrospinal fluid pressure [12–13]. Intraspinal epidural pressure measurements are useful in monitoring intracranial pressure in patients with increased intracranial pressure. In addition, the procedure is simple and relatively noninvasive [14]. Thus, exhalation to residual volume and increases in arterial blood pressure during laryngoscopy and endotracheal intubation should be

avoided in patients with increased intracranial pressure. The results of this study suggest that RII with 5% sevoflurane is safe and useful, and is unlikely to increase intracranial pressure, as compared with rapid intravenous induction with thiopental which decreases intracranial pressure [15].

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