Tracheal Dilatation by Halothane and Enflurane in Man

Isamu YASUDA*, Mihoko IRIMADA*, Toshio HIRANO*,
Hitoshi ISHII** and Tsukasa TAJIMA**

The effect of halothane and enflurane on tracheal tone were studied in 21 patients during the induction of anesthesia. Endotracheal tube cuff pressure was used to measure tracheal tone. Anesthesia, maintained by nitrous oxide 70% in oxygen, was supplimented with succinylcholine drip infusion to immobilize the patient. Ventilation was controlled by a Volume-preset ventilator. In the halothane group, the initial cuff pressure was 14.8 ± 1.3 (mean ± SE) cmH_2O but 10 min after 0.15 mg/kg of pancuronium injection, it increased to 21.7 ± 2.3 cmH_2O (control). Ten min after inhalation of 0.75 % of halothane, cuff pressure decreased to 14.7 ± 2.3 cmH_2O (34 ± 11 % decrease from the control value). In the enflurane group, the initial cuff pressure was 17.6 ± 1.8 cmH_2O and it increased to 21.0 ± 1.7 cmH_2O (control) 10 min after pancuronium injection. Ten min after 1.7 % of enflurane inhalation, cuff pressure decreased to 17.1 ± 2.3 cmH_2O (23.9 ± 6 % decrease from the control value). Halothane and enflurane produced similar tracheal dilatation in healthy individuals. (Key words: halothane, enflurane, pancuronium, trachea)

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The bronchodilating effects of halothane on asthmatic patients have been well recognized clinically. In animal studies, halothane, enflurane[1] and isoflurane[2] decreased pulmonary resistance of the airway previously constricted by ascaris antigen. These observations indicated that inhalation anesthetics had bronchodilating effects on the stimulated airway[1,2]. On the other hand, studies of pulmonary mechanics on the unstimulated airway failed to demonstrate a decrease in the airway resistance by anesthetics[3,4]. In animal experiment, there were several reports which demonstrated dilatory effect of halothane, by using either an intrabronchial probe[3] or bronchograms[4]. In human studies, however, Patterson et al[5] and Brakensiek and Bergman[6] could not show alterations of airway resistance, and they concluded that, in healthy individuals, halothane had no primary effect upon bronchial tone[6]. But there were some possibilities that their methods to measure bronchial tone were not sensitive enough to detect a delicate change. Accordingly, we have undertaken a study to evaluate the effects of halothane and enflurane on the tracheal tone using endotracheal tube cuff pressure in human[7,8].

Methods

Twenty-one adult patients, ranging in age from 25 to 75 years and without pulmonary or cardiac diseases, were studied during the induction of anesthesia for elective surgical

*Department of Anesthesia, Tohoku Rosai Hospital, Miyagi, Japan

**Department of Anesthesiology, Tohoku University School of Medicine, Miyagi, Japan

Address reprint requests to Dr. Yasuda: Department of Anesthesia Tohoku Rosai Hospital, 3-21 Dainohara 4 Chome Sendai, Miyagi, 980 Japan

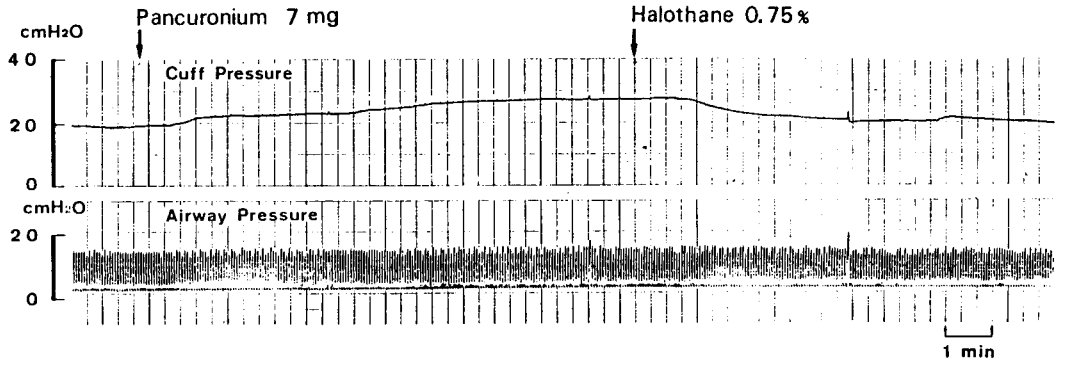

Fig. 1. Representative recording of tracheal tone using endotracheal cuff pressure in one patient. Pancuronium, 7 mg, produced tracheal constriction, and inhalation of halothane 0.75% caused tracheal dilatation. Alteration of tracheal tone did not accompany change of airway pressure.

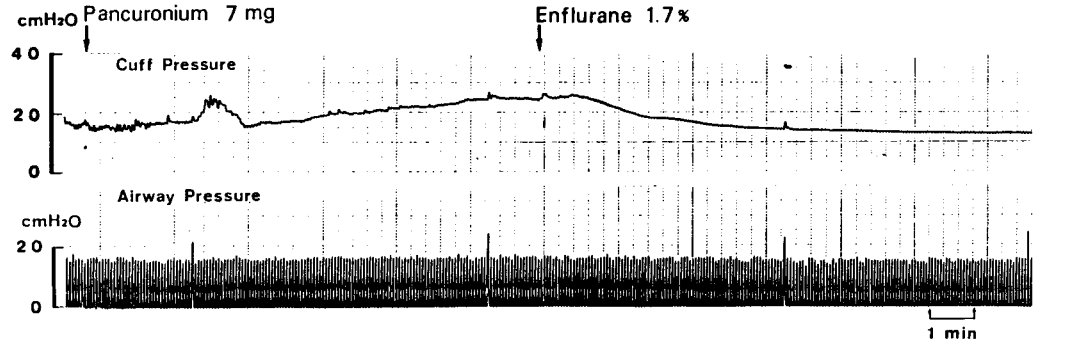

Fig. 2. Representative recording of tracheal tone. Pancuronium, 7 mg, produced tracheal constriction, and inhalation of enflurane 1.7% caused tracheal dilatation. Alteration of tracheal tone also did not accompany change of airway pressure.

procedures. The patients recieved 0.5 mg of atropine and 50 mg of meperidine intramuscularly, 90 min before the operation. Anesthesia was induced with 5 mg/kg of thiamyral sodium and 1 mg/kg of SCC. The trachea was intubated by a tube with a floppy rubber cuff (Igarashi B type) filled with sterilized water. Initially, the patients were anesthetized with 70 % of nitrous oxide and SCC drip infusion (ranged 60–80 mg) was used to immobilize the patients. The proxymal end of cuff catheter was connected to a pressure transducer (Statham P24BB) and a recorder (San-ei Instrument 1410–6). The initial cuff pressure was adjusted to 10–20 cmH_2O. Ventilation was controlled by a Simens Servo B ventilator, and endotidal CO_2 concentration was monitored. After completion of these preparations, 0.15 mg/kg of pancuronium was injected intravenously, and the changes of cuff pressure were observed for the following 10 min. Ten patients recieved 0.75 % (1MAC) of halothane and eleven patients recieved 1.7 % (1MAC) of enflurane. Ten min after initiation of inhalation anesthesia, changes in the cuff pressure were again measured. Airway pressure was simultaniously recorded from the output of ventilator.

Alterations in cuff pressure after injection of pancuronium and inhalation of halothane or enflurane were statistically analyzed using Student's t test for paired data. Cuff pressures during halothane and enflurane inhalation were compared using Student's t test for unpaired data. Differences were

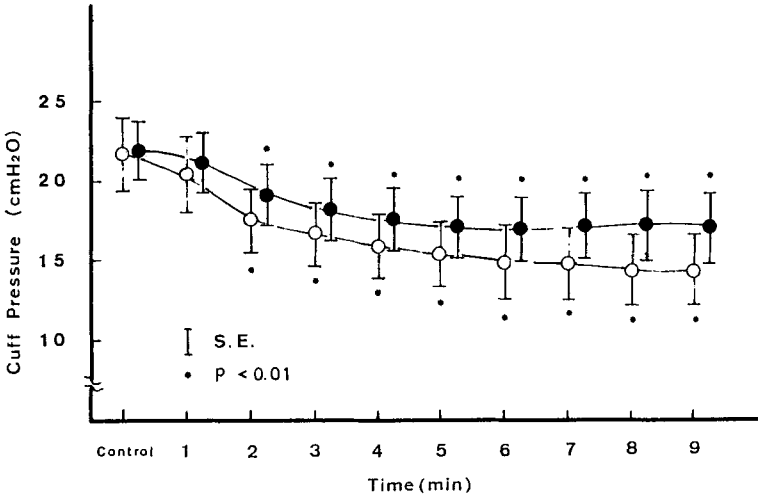

Fig. 3. Effects of halothane (open circle) and enflurane (solid circle) on cuff pressure. Mean value for 10 patients inhaling halothane and 11 patients inhaling enflurane.

considered significant when P values were less than 0.05.

Results

The initial cuff pressur of the halothane group was 14.8 ± 1.3 (mean ± SE) cmH_2O and it increased to 21.7 ± 2.3 cmH_2O ($P<0.001$) 10 min after pancuronium injection (fig. 1). This increased cuff pressure was assumed as the control value. Ten min after inhalatin of halothane the cuff pressure decreased to 14.7 ± 2.3 cmH_2O, which was 34 ± 11% decrease from the control value and statistically significant ($P<0.001$) (figs. 1, 3).

In the enflurane group, the initial cuff pressure of 17.6 ± 1.8 cmH_2O increased to 21.0 ± 1.7 cmH_2O ($P<0.001$) 10 min after pancuronium injection (fig. 2), and 10 min after inhalation of enflurane, it decreased to 17.1 ± 2.3 cmH_2O ($P<0.01$) (23.9 ± 6 % decrease from the control) (figs. 2, 3).

The control values of both groups were almost identical but halothane caused a slightly greater tracheal dilatation than enflurane.

Both halothane and enflurane induced a progressive decrease of cuff pressure during the observation period, but the difference between them was not statistically significant (fig. 3).

Discussion

The simple cuff pressure method utilized in this study to measure tracheal tone in vivo demonstrated that the increases and decreases of cuff pressure were induced by tracheal constriction and dilatation, respectively[7,8]. The present study is the first report to show that in healthy individuals pancuronium increases cuff pressure and produces tracheal constriction, while halothane and enflurane decrease the pressure and cause tracheal dilatation.

Hirshman and Bergman reported that halothane and enflurane were equally effective in decreasing pulmonary resistance in an ascaris antigen stimulated bronchoconstriction[1]. Our results in human showed similar changes of the tracheal tone, although the extent of tracheal dilatation by halothane was slightly greater than that of enflurane.

As shown in figure 1 and 2, changes of tracheal tone in our study were not accompanied by alterations in the airway pressure. Accordingly, pressure tests of the lung mechanics were insensitive to minor changes of the airway tone. Brakensiek and Bergman tested the lung mechanics but failed to demonstrate discernible change of bronchial tone by halothane[6]. Only direct measurement by an intrabronchial probe[3] or bronchograms[4] can detect dilatations of the

airway.

Several mechanisms of bronchial dilatation by halothane have been proposed[9]. These are 1) blocking airway reflexes; 2) directly relaxing airway smooth muscle; 3) inhibiting mediator release; 4) augmenting beta adrenergic tone. We presume that these mechanisms are based on tracheal dilatation induced by inhalation anesthetics. Recently Shah and Hirshman pointed out that major action of halothane in attenuating histamine-induced bronchoconstriction is primary due to the block of the vagal reflex[10]. Korenaga et al demonstrated the direct effect of high concentration of halothane on the airways[11]. It has been known that there is a well developed parasympathetic nervous system in the airway which promotes constriction of airway smooth muscle and this nervous system appears to mediate the rapid reflex by the presence of the endotracheal tube[9]. Tracheal dilatation by inhalation anesthetics may be partly due to blocking of the airway reflex and direct action on tracheal smooth muscle. The other two mechanisms however cannot be ruled out.

The present study has shown that pancuronium increased cuff presure in man and produced tracheal constriction in healthy individuals. The reports of pancuronium induced airway constriction have been rare. Brauer and Ananthanarayan reported anaphylactoid reaction due to release of histamine by pancuronium[12]. On the contrary, Dobkin et al., studying 270 patients, found no change in plasma histamine level after an injection of pancuronium[13]. The mechanism of tracheal constriction caused by pancuronium is still unknown.

In summary, the present study showed that: 1) tracheal smooth muscle tone was increased by pancuronium, and 2) equipotent concentrations of halothane and enflurane produced similar tracheal dilatation.

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