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Relationship between respiratory pattern and work of breathing during halothane anesthesia with spontaneous ventilation

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Introduction

Halothane is an inhalational anesthetic known to change the respiratory pattern. Because of its preferential suppression of intercostal muscle function and relative sparing of diaphragmatic activity [1,2], halothane decreases the rib cage contribution to ventilation (%RC). Inhalational anesthetics also reduce functional residual capacity and decrease the compliance of both the thorax [4] and the lung [5]. For these reasons, an increase in the work of breathing of patients (WOBp) during halothane anesthesia might be expected.

To determine the relationship between the respiratory pattern and its mechanics during halothane anesthesia with spontaneous ventilation, we compared measurements obtained using a respiratory inductive plethysmograph and a pulmonary function monitor with an esophageal catheter.

Patients and methods

This study was approved by the Sapporo Medical University Committee on Human Research and informed consent was obtained from each patient. Fifteen ASA physical status I or II adult male patients (48.2 ± 6.2 years old, mean ± SD) who required general anesthesia for minor surgery on their body surfaces were studied. Patients with a history of smoking, pulmonary dysfunction, or extreme obesity (body mass index >30) were excluded from this study.

The respiratory pattern was measured with a respiratory inductive plethysmograph (Respigraph, NIMS, Miami Beach, FL, USA). This device simultaneously measures changes in the rib cage and in abdominal movements, using two belts (respibands) that contain inductance coils. The change in the coil's inductance is proportional to its cross-sectional area. The %RC was measured as the ratio of the rib cage's change to the total change (rib cage and abdomen) [6]; the phase shift between the rib cage and abdominal movements (PSrc-ab) was also measured by this plethysmograph. WOBp and airway resistance (Raw) were measured using a pulmonary function monitor (CP-100, Bicore, Irvine, CA, USA), which operates with integral transducers. WOBp was measured by integrating the area defined by the changes in the esophageal pressure against the inspired volume [7]. The measurement of Raw is a calculation based on pressure measurements in the esophagus in conjunction with airway flow and pressure measurements at the mouth. To exclude the elastic component of the overall resistance to airflow, we measured Raw when the inspired volumes and the expired volumes were identical and flow rates were approximately maximal in both the inspiratory and expiratory phases of a respiratory cycle [8].

The patients were premedicated with atropine 0.5 mg i.m. 1 h before operation, but no other premedication was given. In the operating room, respibands were applied, one attached to the patient's rib cage and the other to the abdomen, and then both %RC and PSrc-ab were measured for 5 min as controls.

After anesthesia was induced with thiamylal 4 mg/kg i.v., an esophageal catheter and a laryngeal mask (LM) were inserted. An air-filled balloon with the esophageal catheter was placed in the lower third of the esophagus, approximately 30–40 cm from the nose to

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the lower tip of the balloon. To measure esophageal pressure, the catheter balloon had been partially inflated to a small pre-determined volume of approximately 0.6 ml. A flow transducer and a sampling tube of an anesthetic gas monitor (5250 RGM, Ohmeda, Louisville, MO, USA) were connected to the LM, and anesthesia was maintained with 0.74% (1 MAC) halothane in oxygen under spontaneous ventilation via the LM. All patients were supine while being studied.

The %RC, PSrc-ab, WOBp, and Raw were measured at least 30 min after administration of thiamylal, and at least 15 min after the end-tidal halothane concentration exhibited approximately 0.74%. Values of %RC and PSrc-ab were averaged for 5 min, whereas those of WOBp and Raw were averaged for 10 cycles of breathing.

Student's *t*-test was used to compare, respectively, %RC and PSrc-ab between during wakefulness and during halothane anesthesia. Comparisons during halothane anesthesia were made between %RC and WOBp, between %RC and Raw, and between Raw and WOBp; for these three comparisons, we used linear regression analysis. In all comparisons, $P < 0.05$ was considered statistically significant. The data are expressed as scatter diagrams in the figures and as mean ± SD in the text.

Results

In wakefulness before anesthetic induction, %RC was 33.6 ± 8.2%. The phasing of inspiration and expiration was identical in timing for rib cage and abdomen, and PSrc-ab was 1.00 in all cases.

After induction of halothane anesthesia, %RC significantly decreased to 10.0 ± 14.1% ($P < 0.01$), and PSrc-ab significantly increased to 1.07 ± 0.09, indicating that the movement of the rib cage lagged behind that of the abdomen. WOBp averaged 1.23 ± 0.39 joules/l (range 0.46 to 1.75) during halothane anesthesia. When WOBp was compared with %RC, there was a significant negative correlation between them ($r = -0.87$, $P < 0.01$, Fig. 1A). Raw averaged 11.1 ± 3.9 $cmH_2O \cdot s^{-1} \cdot l^{-1}$ (range 4.8 to 17.5) during halothane anesthesia. When Raw was compared with %RC, there was a significant negative correlation between them ($r = -0.90$, $P < 0.01$, Fig. 1B). When WOBp was compared with Raw, there was a significant positive correlation between them ($r = 0.76$, $P < 0.01$, Fig. 1C).

Discussion

In the present study, %RC decreased and the movement of the rib cage lagged behind that of the abdomen as a result of halothane anesthesia. The chest wall is subdivided into the rib cage and abdomen/diaphragm, both of which have supportive and ventilatory functions [9]. Therefore, when the movement of the upper rib cage is more suppressed than that of the abdomen/diaphragm by inhalational anesthetics, the movement of the rib cage might become passive and be controlled by that of the abdomen/diaphragm. This would account in part for the phase shift between the rib cage and abdominal movements in this study.

Another explanation for the shift includes upper airway obstruction. The resistance of breathing via an LM, *per se*, can be excluded as the reason because of its low resistance [10]. We also ascertained the absence of the epiglottis downfolding by using a fiberoptic laryngoscope in the patients whose %RC exhibited negative values. However, it does not seem reasonable to

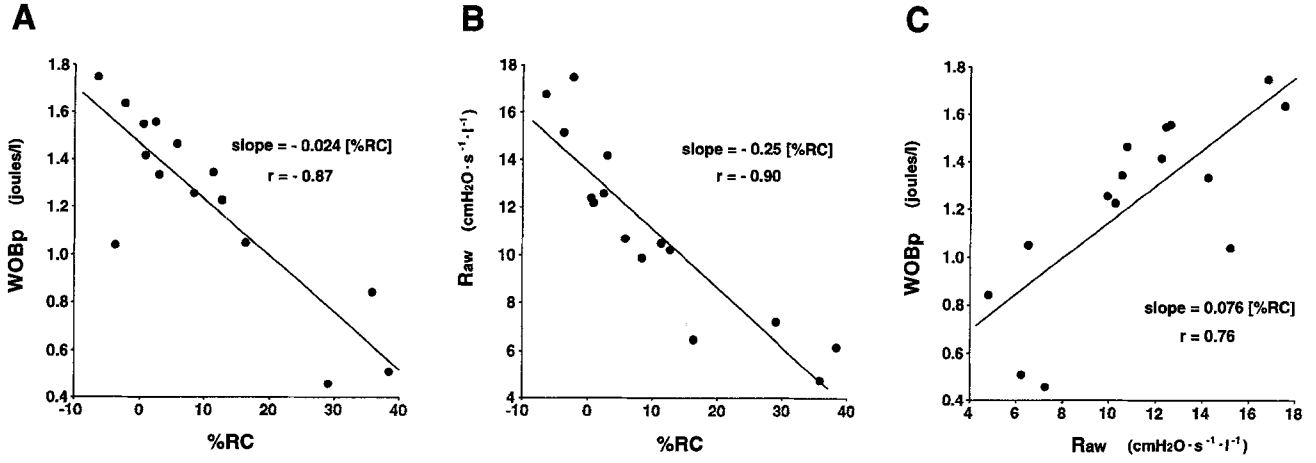

Fig. 1A–C. Relationships between respiratory pattern and its mechanics during halothane anesthesia with spontaneous ventilation. **A** Relationship between %RC and WOBp. **B** Relationship between %RC and Raw. **C** Relationship between Raw and WOBp. %RC, rib cage contribution to ventilation; Raw, airway resistance; WOBp, work of breathing of patients

exclude entirely the possibility of the upper airway obstruction, because a rather high Raw (11.1 ± 3.9 $cmH_2O \cdot s^{-1} \cdot l^{-1}$) and the negative correlation between %RC and Raw were observed in this study (Fig. 1B). One possible explanation for the phase shift is that the larynx may somehow be affected by insertion of the LM, perhaps due to compression, angulation, or laryngeal edema formation. In addition, the larynx may not be in a truly neutral open position during anesthesia via an LM. Some investigators have demonstrated that the vocal cords moved toward the midline during expiration and the resistance of the respiratory system rose above inspiratory level during sleep and eupnea [11,12]. The possible participation of the vocal cords, however, is only speculative since it is not yet known whether they function normally during halothane anesthesia.

The present study also revealed that WOBp exhibited a rather high value of 1.23 ± 0.39 joules/l during halothane anesthesia. We cannot conclude that the halothane anesthesia in this study could have induced the high WOBp because we did not measure the value in awake patients due to the discomfort of esophageal catheter placement. We do know, however, that the normal values of WOBp for an adult range from 0.45 to 0.65 joules/l [13], and therefore we might conclude that the high WOBp during halothane anesthesia could have been partly attributable to the high Raw as shown in Fig. 1C. Fiastro et al. [13] have also reported that 0.75 joules/l is the critical point for weaning from mechanical ventilation. Taking these into account, the patients, especially those with depressive chest wall movements, seemed to breathe with high WOBp even under respiratory suppressive halothane anesthesia (Fig. 1A).

In conclusion, this study demonstrated that the chest wall movement was inhibited by inducing halothane anesthesia, and that WOBp under halothane anesthesia exhibited rather high values with a significantly negative correlation with %RC.

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