Changes in Breathing Patterns after Lung Operations

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To assess the effects of thoracic surgery on breathing patterns without conscious intervention, we analyzed and compared the breathing patterns observed immediately before and after surgical procedure while anesthesia was maintained with enflurane and nitrous oxide at a constant level in 15 patients undergoing thoracic surgery. Our results showed that immediately after the surgical procedure, respiratory frequency (f) was significantly higher and tidal volume (V_T) was significantly smaller than those observed immediately before the surgical procedure. The increased f during the period immediately after the surgical procedure was due to shortening of both inspiratory time (T_I) and expiratory time (T_E) without changing the ratio of T_I to the total breath time (T_{tot}). Despite the significant decrease in V_T observed immediately after the surgical procedure, the value of negative pressure during the occluded inspiration (P_{max}) was significantly higher than that observed immediately before the surgical procedure. These results indicate that a rapid, shallow type of breathing pattern can occur after thoracic surgery without conscious intervention. (Key words: breathing pattern, thoracic surgery, enflurane anesthesia)

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Major thoracic surgery can affect respiratory mechanics and performance, which in turn may cause abnormal breathing patterns that predispose a patient to respiratory complications in the postoperative period. Although it has been reported that the breathing pattern after thoracic surgery is characterized by shallow, rapid breathing with an absence of large breaths and sighs[1–4], mechanisms and factors that produce such types of breathing are unknown. Obviously, the presence of anxiety and/or discomfort during a postoperative period affects the breathing patterns. Although anesthesia can eliminate the possible effects of conscious intervention on the breathing patterns, no information is available about the postoperative breathing patterns before awakening from anesthesia. Accordingly, in the present study we analyzed the differences of breathing patterns observed immediately before and after a surgical procedure in patients undergoing thoracic surgery while anesthesia was maintained at a constant depth.

Methods

The studies were performed on fifteen patients (nine men and six women) scheduled for lobectomy of the lung. Their ages ranged from 23 to 66. None had a history or clinical evidence of cardiovascular disease. This study was approved by our Institutional Ethics Committee, and informed consent was obtained from all patients. Patients were premedicated with atropine (0.5 mg, i.m.) and hydroxyzine (50 mg, i.m.) 45 min before the induction of anesthesia. Anesthesia was induced with thiopentone (4–5 mg·kg^{-1},

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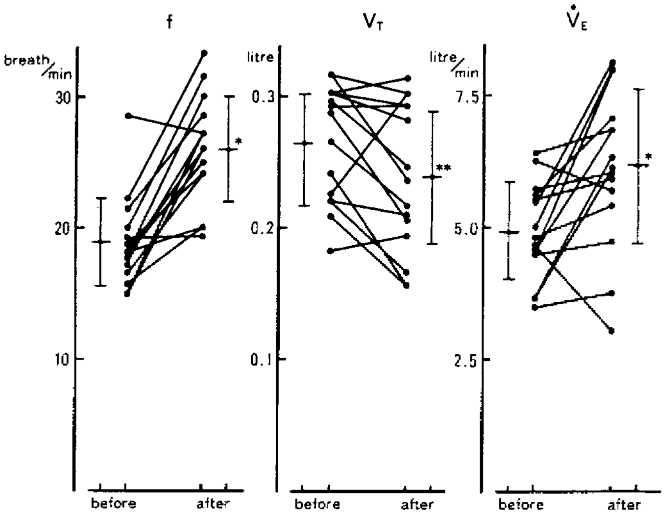

Fig. 1. Changes in respiratory frequency (f), minute ventilation ($\dot{V}_E$), and tidal volume (V_T) immediately after the surgical procedure. Mean ± SD values for the whole group are also shown. Both individual values and mean ± SD values are shown. $*P < 0.05$, significantly different from the values obtained immediately before the surgical procedure.

i.v.), and succinylcholine (1 $mg \cdot kg^{-1}$, i.v.) was given to facilitate endotracheal intubation. Anesthesia was then maintained with enflurane (end-tidal concentration 0.8%) in 50% N_2O-O_2, and spontaneous respiration was allowed to resume. After spontaneous respiration had returned with complete recovery from the effect of succinylcholine, the endotracheal tube of each patient was connected to an experimental apparatus incorporated in an anesthetic circuit. The details of the experimental apparatus and the method of measuring respiratory variables have been reported elsewhere[1]. In brief, tidal volume (V_T) was measured by electronically integrating the flow signal obtained from a pneumotachograph (Fleisch #2). Tracheal pressure (P_{tr}) was measured with a pressure transducer (Toyo Baldwin, DDL-0.05). End-tidal CO_2 and enflurane concentrations were continuously measured with Datex Normocap and Datex Normac, respectively. In order to assess the breathing pattern immediately before surgery, airflow, V_T, P_{tr}, and P_{ETCO_2} all were measured continuously and recorded at least 10 min after the patients had established a steady spontaneous breathing pattern. In addition to the recording of the spontaneous breathing pattern, a threeway stopcock in the experimental apparatus was abruptly turned during expiration so that the airway was obstructed during the subsequent inspiration at end-expiratory lung volume. During the occluded inspiration, the peak of negative pressure (P_{max}) was determined.

After these measurements the patients received the scheduled operations. Operations were the standard ones for lung cancer in our hospital. In brief, thoracotomy was performed by means of costectomy of the fifth rib followed by lobectomy and lymphnode dissection. The operations lasted for 3–4 hr with a total blood loss of less than 300 ml in all the cases. During the operations anesthesia was maintained with enflurane (end-tidal concentration 1–2%) and nitrous oxide (30–60%), and pancuronium for muscle relaxation (6–8 mg). At the end of the operation, intrathoracic catheters were introduced into the pleural space and attached to a water trap which was attached to a suction pump with a vacuum of −11 cm of water. After the completion of the operation, when neuromuscular block was reversed with 1.0 mg atropine (i.v.) and 2.0 mg of neostigmine (i.v.) and spontaneous breathing was steady, the same measurement as in the period immediately before the surgical procedure was repeated while anesthesia was maintained with enflurane (0.8% end-tidal) in 50% N_2O-O_2. Breathing patterns were analyzed in terms of changes in minute ventilation ($\dot{V}_E$), inspiratory time (T_I), expiratory

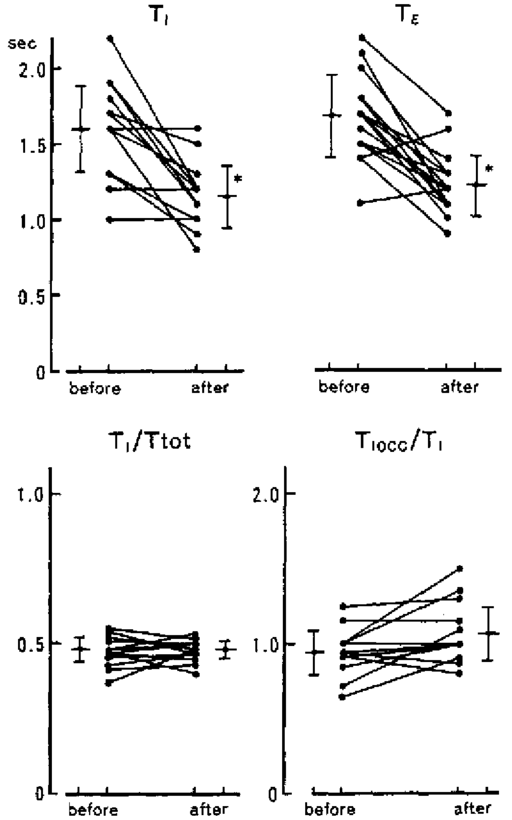

Fig. 2. Comparisons of T_I, T_E, T_I/T_{tot}, and T_{Iocc}/T_I immediately before and after the surgical procedure. $^*P < 0.05$, $^{**}P < 0.01$.

time (T_E), respiratory frequency (f), P_{max}, and V_T. Statistical analysis of the data was performed using the paired t-test.

Results

Figure 1 shows mean values of f, V_T, and $\dot{V}_E$ obtained from all the patients. These results show that both $\dot{V}_E$ and f significantly increased while V_T significantly decreased in the period immediately after the surgical procedure. The increase in $\dot{V}_E$ observed immediately after the surgical procedure was due exclusively to an increase in f, and the increase in f was due to decreases in both T_I and T_E (fig. 2). No significant differences in the values of T_I/T_{tot} and T_{Iocc}/T_I were observed between before and after the surgical procedure (fig. 2). Despite the significant decrease in V_T in the period immediately after the surgical procedure, P_{max} did not decrease but rather significantly increased, causing a significant increase in effective elastance (E'rs) which is defined as the ratio of P_{max} and V_T (E'rs = P_{max}/V_T) (fig. 3). The values of P_{ETCO_2} before and after the operation were 45.7 ± 4.0 and 43.8 ± 3.7 mmHg, respectively and there was a small but significant difference ($^*P < 0.05$, paired t-test) between the two values. Monitoring of arterial oxygenation with a pulse oximeter showed that there was no evidence of hypoxemia both before and after the surgical procedure in all patients.

Discussion

In the present study we have shown that the breathing pattern observed immediately after the surgical procedure was considerably different from that observed immediately before the surgical procedure, despite a constant level of anesthesia. The breathing pattern observed immediately after the surgical procedure in the present study was characterized by a decreased tidal volume and an increased respiratory frequency. This pattern is similar to the postoperative breathing pattern observed in awake patients undergoing major thoracic surgery[2–5], indicating that a shallow rapid .type of breathing pattern in the postoperative period can occur without conscious intervention.

There are a number of factors that might cause the rapid shallow type of breathing after thoracic surgery. Obviously, mechanical factors play the most important role in determining the breathing pattern in the postoperative period. Considerable decreases in lung tissue as well as deformation of the chest wall following thoracotomy and lobectomy might well restrict a normal expansion of the thoracic cage and the normal motion of the diaphragm. The mean tissue loss in our patients was approximately 4 segments. Proprioceptive stimuli from the lungs or respiratory muscles might reflexively affect respiratory rate and depth and produce the restrictive type of breathing. In this regard, there is evidence to suggest that chest wall compression in humans causes a rise in res-

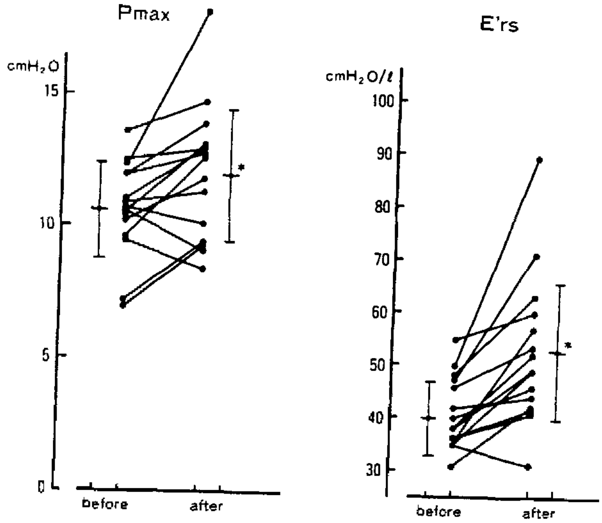

Fig. 3. Comparisons of P_{max} and E'rs before and after the surgical procedure. $^*P < 0.05$.

piratory frequency and a fall in tidal volume and results in rapid, shallow breathing[6], although it has been suggested that both lung irritant receptors and chest wall mechanoreceptors are responsible for this response. Thus, the rapid, shallow breathing observed in our patients may be explained by activation of these receptors. Also, the decreased tidal volume after thoracic surgery may partly be due to reflex inhibition of the diaphragm. However, the possibility of reflex inhibition of the diaphragm is unlikely, since the occlusion pressure obtained immediately after the surgical procedure was considerably higher than that obtained immediately before the surgical procedure, indicating the increased activity of the diaphragm in the postsurgical period. The mechanism which caused the increase in occlusion pressure is unclear. However, since the length-tension characteristics of the inspiratory muscles depend on lung volume, it is possible that the increased activity of inspiratory muscles was due solely to a decrease in lung volume after operation. It is also possible that the increased activity of the inspiratory muscles might be associated with increased central inspiratory activity, since it is conceivable that various surgical stimuli can facilitate the activity of the respiratory centers even in the state of anesthesia.

The finding that V_T decreased despite the increased inspiratory activity indicates that increases in mechanical impedance might occur after surgery. Indeed, our study showed that E'rs which represents the stiffness of the respiratory system[7] significantly increased postoperatively. Changes in chemical ventilatory factors such as pH, P_{CO_2}, and Pa_{O_2} might affect the breathing patterns. However, the values of PET_{CO_2} in the postsurgical period varied by less than 2 mmHg from those obtained immediately before the surgical procedure. There was no evidence of hypoxemia both before and after surgical procedure. Therefore, it is unlikely that changes in ventilatory chemical drive are responsible for the changes in breathing patterns observed in the present study. The data presented in this present study are compatible with the hypothesis that respiratory rate and depth are regulated to minimize the amount of work or force[8]. Mechanisms responsible for such a regulation are not totally clarified. However, there is no doubt that the mechanisms regulating respiratory rate and depth play a major role in determining the breathing pattern in the postoperative period, regardless of the presence or absence of consciousness.

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