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Lumbar epidural block reduces cough strength in healthy young subjects

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Abstract: Effects of lumbar epidural block on maximum expiratory strength were studied in 12 healthy volunteers. Subjects performed maximum expiratory effort against occluded airway at functional residual capacity (FRC) and total lung capacity (TLC) while measuring airway pressure and electromyogram of the abdominal muscles (EMGab). Cough strength was assessed by maximum expiratory pressure (PE_{max}) and peak EMGab (peak-EMGab). Following injection of 2% lidocaine 17.8 ± 1.1 ml into the lumbar epidural space (L2.3 ± 0.4), upper levels of analgesia ranged from T11 to T4 (T7.8 ± 1.3). Peak-EMGab and PE_{max} were significantly reduced by lumbar epidural block at both lung volumes. Compared with severe reduction in peak-EMGab, PE_{max} was well maintained at TLC, but changes in PE_{max} were identical to those in peak-EMGab at FRC. When analgesia spread to higher than T6, PE_{max} at TLC decreased considerably. We conclude that lumbar epidural block producing analgesia above T6 paralyzes the abdominal muscles and severely impairs the ability of effective cough in healthy young men.

Key words: Regional anesthesia, Epidural anesthesia, Cough

Introduction

Lumbar epidural block is generally believed to be a suitable technique for anesthetic management of the elderly and patients with chronic obstructive pulmonary disease (COPD), since it does not impair resting pulmonary ventilation to any significant degree [1,2]. However, this technique does induce various extents of abdominal muscle paralysis depending on the dose, volume, and concentration of local anesthetics [3–5]. The abdominal muscles play a major part in the clearance of retained secretions from the airway by increasing intrathoracic pressure in cough mechanisms [6,7]. Therefore, preservation of the abdominal muscle strength might be very important in the early postoperative period when the residual effects of anesthetics and neuromuscular blockades would profoundly influence the ability to cough. Consequently, paralysis of the abdominal muscles induced by lumbar epidural block might result in decreased cough strength leading to retained secretions.

Accordingly, we hypothesized that lumbar epidural block diminishes the ability to produce effective cough because of paralysis of the abdominal muscles. To test this hypothesis, we evaluated the influences of lumbar epidural block on cough strength by measuring maximum expiratory pressure (PE_{max}) and the electromyogram peak of the abdominal muscles (peak-EMGab) when subjects performed maximum expiratory effort against an occluded airway. Furthermore, relationships between the upper levels of analgesia and the strength of cough were analyzed.

Patients and methods

Twelve healthy young volunteers ranging from 17 to 33 years old were studied. The average weights and heights of the subjects were 61.9 ± 3.8 kg and 171 ± 2.6 cm, respectively (Table 1). None of them had clinical evidence of cardiorespiratory disorders. The protocol of the study was approved by our institutional Ethics Committee and informed consent was obtained from each participant.

After insertion of a thin polyethylene catheter into a lumbar epidural space (from the first to the third space, L2.3 ± 0.4 on average), one pair of surface electrodes was attached on the skin of the abdominal wall in the mid-axillary line at the right side of the umbilicus to measure the electromyogram of the abdominal muscles

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Table 1. Anthropometric characteristics and the range of analgesia during lumbar epidural block

	Number of subjects	Age (years)	Height (cm)	Weight (kg)	Dose of 2% lidocaine (ml)	Sites of epidural puncture	Analgesia	
							Upper level	Lower level
Group 1	4	25.5 ± 3.1	172 ± 3.2	61 ± 1.5	19.3 ± 0.9	L2.5 ± 0.3	T10.5 ± 0.3	S0.5 ± 0.5
Group 2	4	28.8 ± 1.8	173 ± 2.0	70 ± 2.7*	17.3 ± 1.3	L2.3 ± 0.5	T8.0 ± 0.4	L5.5 ± 0.5
Group 3	4	27.3 ± 1.3	168 ± 2.6	55 ± 1.8	17.0 ± 1.2	L2.0 ± 0.6	T4.8 ± 0.5	S0.3 ± 0.8

Values are mean ± SEM.
* $P < 0.05$, *vs* group 3.

(EMGab) mainly reflecting the activity of the external oblique muscle. The EMGab signal was filtered, amplified, and integrated (bioelectric amplifier AB-621G, integrator EI-601G, Nihon Koden Tokyo, Japan). The patients were placed in the supine position and breathed through a mouthpiece connected to a three-way stopcock where airway pressure was measured using a water-filled pressure transducer (carrier amplifier AP-601G, Nihon Koden). EMGab signals and airway pressure were recorded on an 8-channel thermal recorder (Nihon Koden).

Maximum expiratory effort was performed while the airway was occluded by turning a three-way stopcock at functional residual capacity (FRC) or total lung capacity (TLC). This was accomplished by encouraging the patient to blow as vigorously as possible without using the cheek muscles to avoid the influence of cheek compliance on the measurement of airway pressure. The effort was repeated 3–4 times at each of the two lung volumes. To prevent muscle fatigue, the interval of each trial was set at approximately 1 min. The two trials which showed the greatest airway pressure deflection were chosen as successful trials, and were analyzed. PE_{max} was determined by mean values of peak airway pressure in these trials. The corresponding peak amplitude of EMGab was defined as peak-EMGab. Peak-EMGab at FRC before lumbar epidural block was defined as 100%.

After control measurements of PE_{max} and peak-EMGab, 2% lidocaine was administered into the epidural space (1.78 ± 1.1 ml). Following stabilization of the level of analgesia to pin prick, series of maximum expiratory maneuver determined PE_{max} and peak-EMGab at both lung volumes. Upper and lower dermatome levels of analgesia to pin prick were defined as mean values of before and after trials. When the levels of analgesia were different from the right and the left side, mean values of both sides were calculated.

To assess the relationships between the upper levels of analgesia and the strength of cough, subjects were divided into three groups according to the upper level of analgesia: group 1, lower than T10; group 2, T10 to T6; and group 3, higher than T6.

Statistical analysis was performed using ANOVA and Tukey's test. $P < 0.05$ was considered to be statistically significant. All values are expressed as mean ± SEM.

Results

Following the injection of 2% lidocaine 17.8 ± 1.1 ml on average, the upper level of analgesia ranged from T11 to T4 (T7.8 ± 1.3). The analgesia obtained following lumbar epidural block for each subject was variable and was independent of the amount of lidocaine and the site of epidural puncture, as shown in Table 1. None of them had any clinical evidence of hypotension, bradycardia, and excitation owing to lumbar epidural block. Most of the subjects complained of loss of abdominal muscle strength to some extent.

Figure 1 illustrates representative recordings of airway pressure and EMGab of maximum expiratory maneuver in a subject whose analgesia extended from S1 to T4 segments. Lumbar epidural block considerably reduced peak-EMGab at both FRC and TLC. PE_{max} also decreased at both lung volumes. As the lung volume increased, PE_{max} was augmented. This lung volume dependence of PE_{max} was observed before and following lumbar epidural block while peak-EMGab was independent of lung volume. Compared with the profound decrease in EMGab, the decrease in PE_{max} was small. A discrepancy existed between the changes of peak-EMGab and those of PE_{max}.

The results of all patients are shown in Table 2. Lumbar epidural block reduced peak-EMGab from 100% to 65.2 ± 7.2% at FRC ($P < 0.01$), and from 93.0 ± 3.2% to 57.7 ± 6.2% at TLC ($P < 0.01$). Changes in the lung volume did not influence the values of peak-EMGab. Following lumbar epidural block, PE_{max} decreased from 87.2 ± 6.3 cmH_2O to 59.9 ± 7.4 cmH_2O at FRC ($P < 0.01$), and from 126 ± 6.3 cmH_2O to 97.7 ± 9.5 cmH_2O at TLC ($P < 0.05$). PE_{max} was significantly greater at TLC than that at FRC ($P < 0.01$) indicating the dependence of PE_{max} on the lung volume.

Figure 2 demonstrates the relationships between the upper levels of analgesia and changes in peak-EMGab and PE_{max}. All values were normalized as percentages

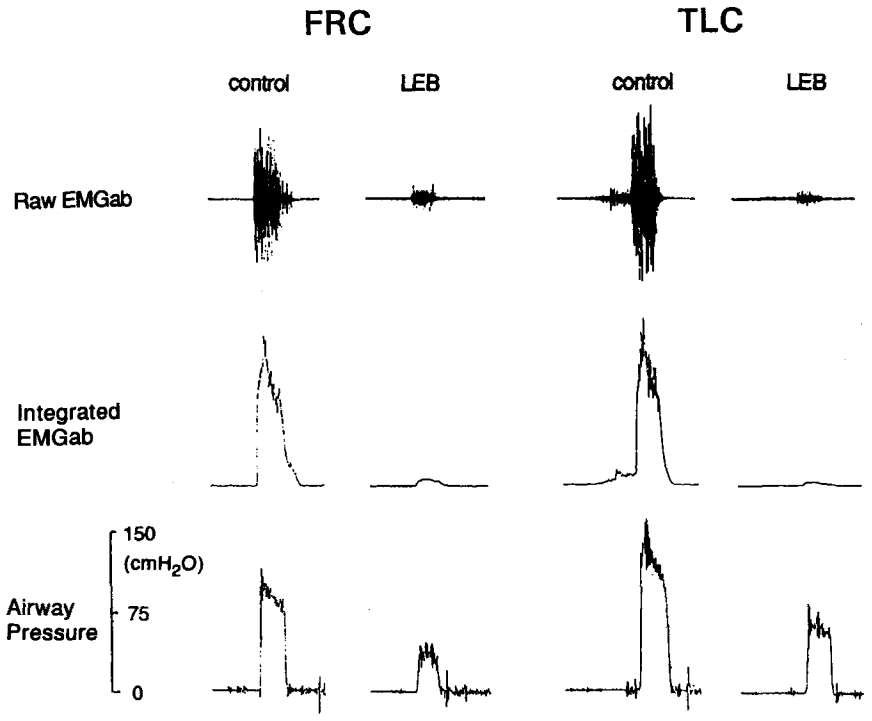

Fig. 1. Representative recordings of airway pressure and electromyogram activity of the external oblique muscle (*EMGab*) when a patient performed maximum expiratory effort against occluded airway. Analgesia was obtained from S1 to T4 by lumbar epidural block (*LEB*) in this patient. *FRC*, functional residual capacity; *TLC*, total lung capacity

Table 2. Effects of lumbar epidural block (LEB) on maximum expiratory pressure (PE_{max}) and peak activity of electromyogram of the abdominal muscle (peak-EMGab) at functional residual capacity (FRC) and total lung capacity (TLC)

	FRC		TLC	
	peak-EMGab (%)	PE_{max} (cmH_2O)	peak-EMGab (%)	PE_{max} (cmH_2O)
Group 1				
Control[a]	100	88.2 ± 10.5	85.8 ± 2.3	125 ± 8.4^{++}
LEB[b]	88.6 ± 4.9	79.8 ± 10.5**	74.0 ± 5.0	118 ± 11.6^{++}
Group 2				
Control	100	79.8 ± 11.6	103 ± 5.6	125 ± 15.8^{++}
LEB	66.7 ± 10.6	54.6 ± 12.6	66.9 ± 4.0*	106 ± 16.8^{++}
Group 3				
Control	100	92.4 ± 13.7	89.9 ± 4.0	127 ± 10.5^{++}
LEB	40.2 ± 6.4**	45.2 ± 9.5*	32.1 ± 6.4**	69.3 ± 12.6*$^{+}$
Total				
Control	100	87.2 ± 6.3	93.0 ± 3.2	126 ± 6.3^{++}
LEB	65.2 ± 7.2**	60.0 ± 7.4**	57.7 ± 6.2**	97.7 ± 9.5*$^{++}$

Values are mean ± SEM.
* $P < 0.05$, ** $P < 0.01$ *vs* control; $^{+}$ $P < 0.05$, $^{++}$ $P < 0.01$ *vs* FRC.
[a] Before LEB, [b] following LEB.

from those of control state in each subject. As the upper levels of analgesia extended to higher thoracic segments, peak-EMGab and PE_{max} decreased progressively at both lung volumes. When analgesia spread to higher than T6 (group 3), PE_{max} ($P < 0.05$ *vs* group 1) and peak-EMGab ($P < 0.01$ *vs* group 1) were profoundly depressed at both lung volumes. However, a clear discrepancy was observed between changes in both variables at TLC ($P < 0.05$) whereas changes in PE_{max} were identical to those of peak-EMGab at FRC.

Discussion

In the present study, we demonstrated that: (1) lumbar epidural block significantly reduced PE_{max} and peak-EMGab, (2) when analgesia spread to higher than T6, PE_{max} was seriously reduced at both lung volumes, and (3) a discrepancy existed between changes in PE_{max} and those in peak-EMGab at TLC.

We used EMG activity of the external oblique muscle as an index of electromyogram activity of all abdominal

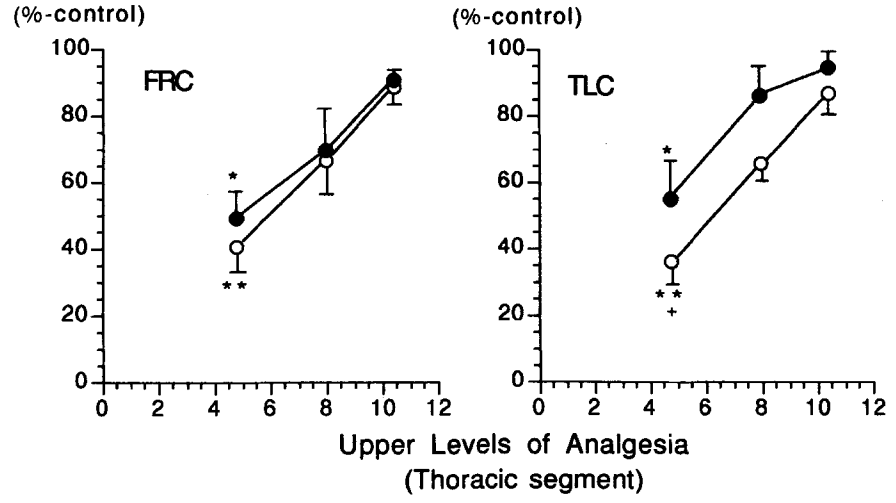

Fig. 2. Effects of upper levels of analgesia on changes in peak electromyogram activity of the abdominal muscles (peak-EMGab) (*open circles*) and maximum expiratory pressure (PE_{max}) (*closed circles*) at FRC and TLC. Peak-EMGab and PE_{max} following lumbar epidural block were expressed as a percentage of the control values in each patient. Data were plotted in each group. *Bars* represent ranges of SEM. $^{*}P < 0.05$, $^{**}P < 0.01$ *vs* group 1; $^{+}P < 0.05$ *vs* group 2

muscles. The external oblique muscle and the rectus abdominis muscle are innervated by T8–T11 and T5–T11, respectively. The muscle fibers of the latter receive segmental innervation while those of the former do not. Accordingly, measurement of EMG activity of the external oblique muscle would have a certain advantage in determining the behavior of whole abdominal muscles' contraction, whereas segmental measurements of EMG activity of the rectus abdominis muscle determine the levels of motor paralysis [3–5,8].

Expiratory flow limitation occurs when forced expiration is performed. Flow through a collapsible tube is used as an analogy of forced expiration [7]. In this analogy, upstream pressure is a significant determinant of maximum flow through the collapsible tube. In fact, there is a linear relationship between upstream pressure and maximum flow [9]. PE_{max} measured in the present study reflects upstream pressure in forced expiration although the measurement was performed under static conditions. Accordingly, a decrease in PE_{max} must result in a decrease in maximum expiratory flow. High intrathoracic pressure generated by expiratory muscles during cough serves to induce high gas velocities within the airway which supply the shearing forces necessary to dislodge materials adherent to airway walls [6,7]. Taking the mechanisms of cough into account, the method in the present study is apparently suitable to evaluate the effects of lumbar epidural block on cough strength.

Unlike the results of Moir [1] and Freund et al. [8], we found significant impairment of maximum expiratory force following lumbar epidural block. Freund et al. measured expiratory reserve volume when lumbar epidural block with 2% lidocaine achieved analgesia up to 2.3 thoracic segments. They found 21% reduction in expiratory reserve volume concluding no significant influence of lumbar epidural block on cough ability [8]. It may not be sufficient to assess cough strength based solely on lung volume changes although expiratory force depends on lung volume. Moir concluded that the ability of cough was not significantly impaired based on the finding that peak expiratory flow rate was reduced by only 5% following lumbar epidural block with 1.5% lidocaine in patients without respiratory disease [1]. The use of lower concentrations of lidocaine may have produced minimal motor block although upper levels of analgesia were higher than our study.

A discrepancy existed between changes in PE_{max} and those in peak-EMGab at TLC although both variables proportionally decreased in response to lumbar epidural block. Because this discrepancy was only prominent in the measurements at TLC, alterations in force-length relationships of the abdominal muscles between the two lung volumes may account for this finding. Another possible explanation for the difference is that other expiratory muscles might be recruited at the higher lung volume which compensate for the impairment of the abdominal muscles resulting in the maintenance of PE_{max}. Although we have no evidence to verify this, the study of De Troyer et al. may support our speculation [10]. They found that the pectoralis major played an important role in active expiration in tetraplegic patients by compensating for the diminished expiratory forces of the abdominal muscles.

PE_{max} increased with the increase in lung volume. Presumably, this well-documented phenomenon is due to an increase in muscle length or recruitment of the other expiratory muscles at higher lung volume [7,11]. Regardless of the mechanisms, postoperative patients should be encouraged to cough voluntarily at higher lung volumes. Following lumbar epidural block, PE_{max} at TLC decreased markedly when analgesia spread above T6, while it did not significantly change below that segment. This indicates that lumbar epidural block which produces analgesia above T6 results in impaired cough strength, leading to reduced clearance of secretions from the airway. Severe paralysis of the abdominal

muscles indicated by profound decrease in peak-EMGab apparently caused the reduction in PE_{max} at TLC. Because of the dependence of maximum expiratory force on the lung volume, decrease in vital capacity following lumbar epidural block [1] may, in part, be responsible for the severe impairment of expiratory strength. Reduction of lung volume after abdominal or thoracic surgery would further impair cough strength.

Clinically, our observation may have a significant implication in the anesthetic management of COPD patients and elderly patients who chronically retain secretions in the airway. Ventilatory reserve is markedly reduced and the ability to produce effective cough is already disturbed in these patients before surgery [12]. The effects of local anesthetics administered into a lumbar epidural space tend to be underestimated especially in these patients. Accordingly, the adverse effects of lumbar epidural block might be more prominent and severe than in healthy young patients.

In conclusion, lumbar epidural blocks which produced analgesia above T6 paralyzed the abdominal muscles seriously, and it impaired the ability of effective cough.

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