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Monitoring of vecuronium-induced neuromuscular blockade during one-lung ventilation

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Abstract
Purpose. We investigated the monitoring of neuromuscular blockade caused by vecuronium in patients receiving one-lung ventilation (OLV) anesthesia for lung surgery.
Methods. Eighteen adult patients requiring OLV for lung surgery (OLV group) and 18 undergoing two-lung ventilation (TLV) for colon surgery (control group) were enrolled in this study. In the two groups, anesthesia was maintained with sevoflurane, fentanyl, and epidural lidocaine. Time from vecuronium 0.1 mg·kg^{-1} to the onset of neuromuscular blockade; times to the return of T1, T2, T3, or T4 (the first, second, third, or fourth response of the train-of-four [TOF]); and recovery of T1/control or TOF ratio (T4/T1) were compared between the two groups.
Results. Time to the onset of neuromuscular blockade in the OLV group was similar to that in the control group (289 ± 74 vs 270 ± 85 s [mean ± SD]; $P = 0.482$). Times from vecuronium to the return of T1, T2, T3, or T4 in the OLV group did not significantly differ from those in the control group (21.9 ± 7.0 vs 25.8 ± 6.7 min for T1; $P = 0.099$). T1/control in the OLV group was significantly higher than that in the control group 50–120 min after vecuronium ($P < 0.05$). The TOF ratio did not differ significantly between the two groups.
Conclusion. During OLV for lung surgery, recovery of T1/control is accelerated in anesthetized patients receiving vecuronium.

Key words One-lung ventilation · Train-of-four · Vecuronium

Introduction

It is necessary for anesthetists to isolate and protect the lungs during lung resection. One-lung ventilation (OLV), using a double-lumen tube, is routinely applied for lung resection. During OLV, one lung is deflated and the other is ventilated. The deflation of the lung elicits discharge in the pulmonary vagal afferent nerve [1,2], which increases the activity of the skeletal muscles [3–6]. On the other hand, OLV causes hyperinflation of the ventilated lung [7,8]. It has been shown that when the lung is hyperinflated, the pulmonary vagal afferent nerves are stimulated [3–6], which induces an increase in the activity of the skeletal muscles [3–6]. Consequently, both deflation and hyperinflation of the lung are thought to strengthen the contraction of the skeletal muscles. Also, the airflow resistance is higher for the double-lumen tube compared with a single-lumen tube [9], and the magnitude of autopositive end-expiratory pressure (auto-PEEP) is higher during OLV than during two-lung ventilation (TLV) [7,8,10,11]. Such types of ventilatory loading markedly increase spontaneous activity in the pulmonary vagal afferent nerves [12], which, as noted above, enhances the activity of the skeletal muscles. Based on these previous findings, it appears that the action of neuromuscular relaxants may be affected during lung surgery requiring OLV. However, no previous study has investigated the action of neuromuscular blocking drugs during OLV. We studied the monitoring of neuromuscular blockade caused by vecuronium in anesthetized patients requiring OLV for lung resection, compared with that in anesthetized patients undergoing TLV for colon surgery.

Methods

The protocol of this study was approved by our local ethics committee. Written informed consent was obtained from each patient. Eighteen adult patients, American Society of Anesthesiologists (ASA) physical status I–II, undergoing elective one-lung ventilation (OLV) anesthesia were enrolled in the OLV group. The patients in the OLV group were scheduled for video-assisted thoracoscopic surgery (VATS) for pulmonary

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resection or bullectomy in the lateral decubitus position. Eighteen adult patients, ASA physical status I–II, scheduled for elective two-lung ventilation (TLV) anesthesia for colon surgery in the supine position were also studied (control group). No patient in either group had neuromuscular, hepatic, renal, metabolic, or cardiac disorders, or was receiving any drugs known to affect the action of neuromuscular blocking drugs.

Sample sizes were based upon an ability to detect a difference of 0.2 or more in the mean T1/control or train-of-four (TOF) ratio between groups, with an SD of approximately 0.2. Assuming an α of 5% and with 90% power, 18 patients per group were required [13]. Accordingly, a total of 18 patients were enrolled in each group.

No premedication was given to the patients in either group. Before the induction of general anesthesia, an 18-gauge multiorifice epidural catheter (Perifix catheter; B. Braun, Melsungen, Germany) was placed through the Th3/4 or Th4/5 and Th10/11 or Th11/12 intervertebral space using the paramedian approach and loss-of-resistance technique, with the patient in the lateral decubitus position in the two groups. All epidural catheters were advanced 5 cm beyond the Tuohy needle tip. After placement of the catheter, a test dose of 60 mg lidocaine 2% (3 ml) was given to exclude an intrathecal location. Thereafter, both groups received a continuous infusion of lidocaine 2% at a rate of 6–8 $ml \cdot h^{-1}$ via the epidural catheter.

For the monitoring of neuromuscular blockade, two stimulating electrodes were positioned over the ulnar nerve at the wrist. Two recording electrodes were also attached over the adductor pollicis muscle. Additionally, one ground electrode was attached between the stimulating and recording electrodes. In the OLV and control groups, the monitoring of neuromuscular blockade was performed over the ulnar nerve and adductor pollicis muscle of the forearm of the nonoperated side, and over those of the left forearm, respectively. Anesthesia was induced with propofol 1.5 $mg \cdot kg^{-1}$ and fentanyl 2 $\mu g \cdot kg^{-1}$ in the two groups. After loss of the eyelid reflex, TOF stimuli were applied every 20 s, using an electrical nerve stimulator of a neuromuscular transmission module (M-NMT Module; Datex-Ohmeda, Helsinki, Finland). For TOF stimulation, four single-twitch stimuli consisting of 0.2-ms-duration square waves were delivered at 2 Hz. The corresponding electromyographic amplitudes were quantified using the neuromuscular transmission module, and were displayed on an anesthetic monitoring system (Anaesthetic Monitoring System A/S3; Datex-Ohmeda). In each patient, the monitoring system searched automatically for the stimulus current needed to achieve the maximal response of the adductor pollicis muscle. The search began with 10-mA single-twitch stimuli of 0.2-ms duration applied every 1 s. The stimulating current was increased in steps of 5 mA until the increase in current no longer increased the electromyographic response. The stimulating current was then automatically increased by 15%, to produce a supramaximal current. If the supramaximal current was not found, or if the response was too weak to determine the current, the current was set at 70 mA.

Once the supramaximal current had been established, the electromyographic amplitude of T1 was considered to be the control value. During the determination of the supramaximal current and recording of the control value, the patients' lungs were ventilated, using a face mask, with oxygen 6 $l \cdot min^{-1}$ and sevoflurane 2.0% inspired concentration. Thereafter, vecuronium 0.1 $mg \cdot kg^{-1}$ was given intravenously and the trachea was intubated. In the OLV group, a left-sided double-lumen tube (Broncho-Cath; Mallinckrodt Medical, St. Louis, MO, USA) was introduced into the glottis via direct laryngoscopy. The double-lumen tubes varied in size from 35 to 37 Fr in females and from 37 to 39 Fr in males. After clinical confirmation of correct double-lumen tube placement by inspection and auscultation, OLV was controlled by using 100% oxygen and a tidal volume of 8–10 $ml \cdot kg^{-1}$ at a rate of 12–16 min^{-1}. In the OLV group, after the placement of the double-lumen tube, a fiberoptic bronchoscope was passed via the endotracheal lumen, and the proper placement of the double-lumen tube was confirmed. Once the proper intubation of the double-lumen tube had been confirmed, the lumen of the nonventilated side was left open to the atmosphere to allow the nonventilated lung to deflate spontaneously. The dependent lung was ventilated with air 1–3 $l \cdot min^{-1}$, oxygen 2–3 $l \cdot min^{-1}$, and sevoflurane 1.7% end-tidal concentration. Fractional concentration of oxygen in inspired gas ($F_{I_{O_2}}$) was kept at 0.52–0.8. The tidal volume and respiratory rate were 8–10 $ml \cdot kg^{-1}$ and 12–16 min^{-1}, respectively. After the patients were settled in the lateral decubitus position, tube placement was again checked by fiberoptic bronchoscopy. OLV was continued until the end of the surgical procedure.

A 2-inch, 20-gauge Teflon catheter was placed in the radial artery of the nonoperated side forearm by direct arterial cannulation. Occasionally, blood samples were obtained via the cannula.

In the control group, a single-lumen endotracheal tube (Sheridan/HVT; Hudson RCI, Temecula, CA, USA) was intubated into the glottis by direct laryngoscopy, and the lungs were ventilated with 100% oxygen and a tidal volume of 8–10 $ml \cdot kg^{-1}$ at a rate of 8–10 min^{-1}. After confirmation of successful tracheal intubation, anesthesia was maintained with air 3 $l \cdot min^{-1}$, oxygen 1–2 $l \cdot min^{-1}$, and sevoflurane 1.7% end-tidal concentration. In the control group, $F_{I_{O_2}}$ was kept at

0.4–0.52. Additionally, in the two groups, peak inspiratory airway pressures were measured using a pressure gauge of the anesthetic machine after successful intubation was confirmed, and the peak inspiratory airway pressures were compared between the two groups.

TOF stimuli were continuously applied every 20 s. The supramaximal stimulating current; times from vecuronium to the onset of neuromuscular blockade (times from vecuronium to the disappearance of the TOF response); and times from vecuronium to the return of T1, T2, T3, or T4 (the first, second, third, or fourth response of the TOF) were compared between the two groups. After vecuronium, T1/control or T4/T1 (TOF ratio) was compared every 10 min between the two groups.

A bolus dose of fentanyl 2 $\mu g \cdot kg^{-1}$ was administered intravenously before skin incision in the two groups. When patients exhibited systolic hypertension (systolic arterial pressure > 150 mmHg) or tachycardia (heart rate > 100 bpm), a supplemental bolus of fentanyl 2 $\mu g \cdot kg^{-1}$ was given. The end-tidal concentrations of anesthetic and Pa_{CO_2} were measured continuously using a multiple gas monitor (AS/3 ADU Anaesthetic Work Station; Datex, Helsinki, Finland). In the OLV group, patients were settled in the lateral decubitus position throughout the surgical procedure. Times from vecuronium to the beginning of the surgical procedure and the operative times were compared between the two groups. Esophageal temperature was monitored continuously. Also, the surface skin temperature over the adductor pollicis muscle was measured using a thermometer probe (Terumo-Finer; Terumo, Tokyo, Japan).

If the TOF ratio did not return to a value of 0.9 at the end of the surgical procedure, atropine 0.02 $mg \cdot kg^{-1}$ and neostigmine 0.04 $mg \cdot kg^{-1}$ were administered to antagonize residual neuromuscular blockade.

Patient characteristics were compared between the two groups using an unpaired *t*-test or χ^2 statistic. The time from vecuronium to the beginning of the surgical procedure; operative times; peak inspiratory airway pressures; supramaximal stimulating currents; times to the onset of neuromuscular blockade; and times to the return of T1, T2, T3, or T4 were compared between the two groups using unpaired *t*-tests. Comparison of T1/control or the TOF ratio between the two groups during recovery from neuromuscular blockade was done using analysis of variance (ANOVA) and unpaired *t*-tests with Bonferroni's adjustment. Values for all results are expressed as numbers or means ± SD (percentages). A P value of less than 0.05 was considered statistically significant. Statistical analyses were performed using a statistical package (SYSTAT 8.0; SPSS, Chicago, IL, USA) running on a personal computer.

Results

As noted in Table 1, patient characteristics did not significantly differ between the two groups. The time from vecuronium to the beginning of the surgical procedure was significantly longer in the OLV group than that in the control group ($P < 0.001$). Operative times were comparable in the two groups.

The double-lumen and single-lumen endotracheal tubes were successfully placed in all patients in the OLV and control groups, respectively.

Surgical procedures in the OLV and control groups are listed in Table 2.

Peak inspiratory airway pressure in the OLV group was significantly higher than that in the control group (32.3 ± 2.7 vs 16.1 ± 3.6 cmH_2O; $P < 0.001$).

Table 1. Patient characteristics, time from vecuronium to the beginning of the surgical procedure, and operative time in the OLV and control groups

	OLV ($n = 18$)	Control ($n = 18$)	P value
Sex (female/male)	9/9	9/9	
Age (years)	48.2 ± 19.4	53.8 ± 8.6	0.274
Height (cm)	161.8 ± 11.5	159.1 ± 6.0	0.379
Weight (kg)	56.8 ± 8.7	58.0 ± 7.4	0.913
ASA physical status			
1	9 (50%)	12 (67%)	
2	9 (50%)	6 (33%)	0.310
Time from vecuronium to the beginning of the surgical procedure (min)	44.2 ± 7.2	21.7 ± 5.4	<0.001
Operative time (min)	151.5 ± 30.7	155.4 ± 25.1	0.680

Values are numbers or means ± SD (percentages)
Sex, age, height, weight, ASA physical status, and operative time did not differ significantly between the two groups. Time from vecuronium to the beginning of the surgical procedure was significantly longer in the OLV group than in the control group ($P < 0.001$)
OLV, one-lung ventilation

The supramaximal stimulating currents; the time to the onset of neuromuscular blockade; and the time from vecuronium to the return of T1, T2, T3, or T4 were similar in the two groups (Table 3).

T1/control in the OLV group was significantly higher than that in the control group 50–120 min after vecuronium ($P < 0.05$; Fig. 1). The TOF ratio did not differ significantly between the two groups (Fig. 2).

In no patient did the esophageal or peripheral temperature over the adductor pollicis muscle decrease to less than 35.5°C or 32.0°C, respectively.

Arterial blood gas was occasionally analyzed, and it was confirmed that pH, Pa_{O_2}, Pa_{CO_2}, and base excess were maintained at 7.37–7.44, 100–200 mmHg, 34–42 mmHg, and –3.0 to +3.0 $mEq \cdot l^{-1}$ in the two groups, respectively.

At the end of the surgical procedure, the TOF ratio did not return to a value of 0.9 in four patients in the OLV group and eight patients in the control group. In these patients, atropine 0.02 $mg \cdot kg^{-1}$ and neostigmine 0.04 $mg \cdot kg^{-1}$ were administered, and the recovery of the TOF ratio to a value of more than 0.9 was ensured before tracheal extubation.

Table 2. Surgical procedures requiring one-lung ventilation in the OLV group and two-lung ventilation in the control group

	OLV	Control
Wedge resection of lung	3	
Segmentectomy	2	
Lobectomy	8	
RUL	2	
RML	1	
RLL	2	
LUL	2	
LLL	1	
Bullectomy	5	
Right hemicolectomy		10
Left hemicolectomy		3
Transverse colectomy		5

Values are numbers
OLV, one-lung ventilation; RUL, right upper lobectomy; RML, right middle lobectomy; RLL, right lower lobectomy; LUL, left upper lobectomy; LLL, left lower lobectomy

Discussion

The present study showed that the supramaximal stimulating currents; the time to onset of neuromuscular blockade caused by vecuronium; and time from vecuronium to the return of T1, T2, T3, or T4 were comparable in anesthetized patients during OLV and in those during TLV. After the administration of vecuronium, T1/control was higher during OLV than during TLV; however, the TOF ratio during OLV did not differ from that during TLV.

During OLV, the nonventilated lung is deflated and the contralateral dependent lung is ventilated. Badier et al. [1] reported that lung deflation produced a marked tonic discharge in the phrenic nerve and intercostal muscle in the rabbit during mechanical ventilation. Collapse of the lung also increased the discharge in the vagal afferent nerve in anesthetized rabbits, cats, and monkeys during spontaneous breathing [2]. Homma et al. [3] noted that lung deflation increased vagal afferent impulses via rapidly adapting receptors in the rabbit during spontaneous breathing. The discharge in the vagal nerve increased the activity of the skeletal muscles, i.e., the intercostal muscles, in the rabbit during mechanical ventilation [1], the levator veli palatini muscle in the dog during spontaneous respiration [5], and abdominal muscles in the cat during mechanical ventilation [6]. Therefore, contraction of the skeletal muscle is assumed to be enhanced during OLV.

Hyperinflation of the ventilated lung often occurs during OLV [7,8]. When the lung is inflated, the pulmonary vagal receptors are stimulated [3,5,14], causing an increase in the activity of the levator veli palatini muscle

Table 3. Supramaximal stimulating current; time to onset of neuromuscular blockade caused by vecuronium 0.1 $mg \cdot kg^{-1}$, and time from vecuronium 0.1 $mg \cdot kg^{-1}$ to the return of T1, T2, T3, or T4 in the OLV and control groups

	OLV	Control	*P* value
Supramaximal stimulating current (mA)	35.1 ± 13.6	31.8 ± 8.9	0.235
Time to onset of neuromuscular blockade (s)	288.9 ± 74.3	270.0 ± 84.6	0.482
Time (min) to return of			
T1	21.9 ± 7.0	25.8 ± 6.7	0.099
T2	33.3 ± 8.5	34.3 ± 8.0	0.720
T3	38.4 ± 9.8	40.6 ± 10.7	0.541
T4	40.6 ± 9.9	42.8 ± 11.4	0.537

Values are means ± SD
The supramaximal stimulating current; onset of neuromuscular blockade; and time to the return of T1, T2, T3, or T4 did not differ significantly between the two groups
OLV, one-lung ventilation

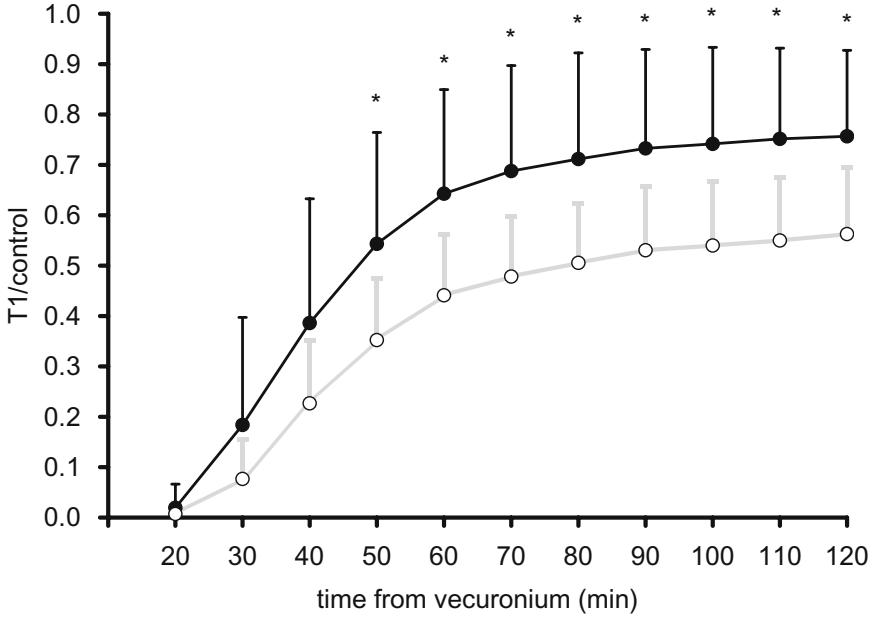

Fig. 1. Recoveries of the first response of the train-of-four (TOF) (*T*1)/control after administration of vecuronium 0.1 mg·kg^{-1} in the one-lung ventilation (OLV; *closed circles*) and control (*open circles*) groups. Values are means ± SD. * $P < 0.05$ between the two groups

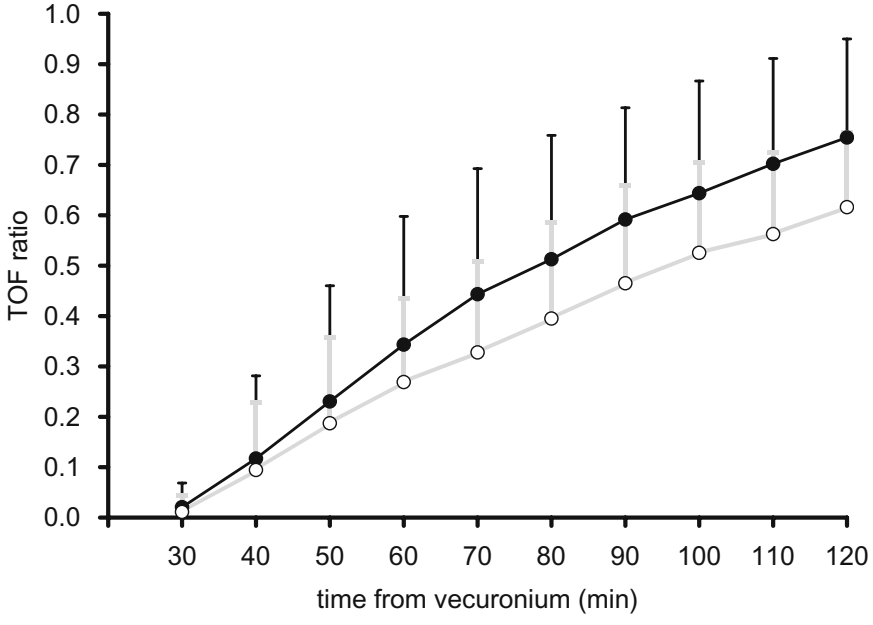

Fig. 2. Recoveries of train-of-four (*TOF*) ratio after administration of vecuronium 0.1 mg·kg^{-1} in the OLV (*closed circles*) and control (*open circles*) groups. Values are means ± SD. No significant difference was found between the two groups

in the dog during spontaneous respiration [5], abdominal muscles in the cat during mechanical ventilation [6], and upper airway muscles in the cat during mechanical ventilation [15]. Further, stimulation of the pulmonary vagal nerves enhances the contraction of the adductor pollicis muscle in humans during spontaneous respiration [4]. Hence, not only deflation of the lung but also hyperinflation of the lung may accelerate recovery from neuromuscular blockade.

It has been reported that the magnitude of auto-positive end-expiratory pressure (auto-PEEP) is higher during OLV than during TLV [7,8,10,11]. Inomata et al. [10] noted that airway resistance increased because of a decrease in the compliance of the dependent lung during OLV, while they found that auto-PEEP was most likely to exist when airway resistance was increased. They also postulated that auto-PEEP tended to appear in the dependent lung during OLV, primarily because of high airway resistance from a relatively small tracheal tube for OLV. Pepe and Marini [16] showed that expiratory flow was impeded because of the resistance of a double-lumen tube, which may have caused dynamic pulmonary hyperinflation and auto-PEEP. Thus, expiratory airway resistance increases during OLV.

Moreover, inspiratory airway pressures were increased in anesthetized patients when they were switched from TLV to OLV with unchanged ventilatory settings [17]. Szegedi et al. [17] demonstrated that when patients were switched from TLV to OLV, baseline peak and plateau inspiratory pressures increased by a mean of 55.1% and 41.9%, respectively. Also, in their study, peak inspiratory pressure was significantly higher during OLV than during TLV. Thus, inspiratory as well as expiratory airway resistance would increase during OLV. Consequently, ventilatory loading would be apparent during OLV. As noted above, ventilatory loading activates the pulmonary vagal afferent nerves, resulting in an increase in the activity of the skeletal muscles [1,4–6,15]. The ventilatory loading-induced activation of the pulmonary vagal afferent nerves may be related to the accelerated recovery of T1/control during OLV.

In the present study, T1/control was significantly higher in the OLV group than in the control group during recovery from vecuronium-induced neuromuscular blockade. Although the TOF ratio was higher in the OLV group than in the control group, the difference did not reach statistical significance. T1/control represents the neuromuscular blocking effect at the postjunctional region of the neuromuscular junction, i.e., the muscular membrane of the skeletal muscle, while the TOF ratio indicates the neuromuscular blocking effect at the prejunctional region of the neuromuscular junction, i.e., motor nerve endings [18]. Therefore, the present study revealed that the recovery of neuromuscular blockade was accelerated mainly at the skeletal muscle rather than that at the nerve endings. Again, as described above, many previous reports support the present results. That is to say, deflation of the lung [1,5,6], hyperinflation of the lung [4–6,15], and an increase in ventilatory loading [16] may enhance the contraction of skeletal muscle. In contrast, only one previous study suggested that activation of the motor nerves occurred during OLV. Specifically, Badier et al. [1] noted that lung deflation elicited tonic discharge in the phrenic nerve in rabbits. So it may not be surprising that the recovery of T1/control was accelerated, while that of the TOF ratio was not. Additionally, although the time from vecuronium to the return of T1, T2, T3, or T4 is also indicative of the postjunctional effect of vecuronium [18], the time to the return of T1, T2, T3, or T4 in the present study did not significantly differ between the OLV and control groups. We are not able to explain this result clearly. However, in the present study, the mean time from vecuronium to the return of T1, T2, T3, or T4 was less than 42.8 min. In contrast, T1/control was higher in the OLV group than in the control group 50–120 min after vecuronium. Based on this finding, it appears that the accelerating effect of OLV on the recovery from neuromuscular blockade may become apparent more than 50 min after vecuronium.

In the patients in our OLV group, an epidural catheter was placed through the Th3/4 or Th4/5 intervertebral space. The patients received a continuous infusion of lidocaine 2% at a rate of 6–8 $ml \cdot h^{-1}$ via the epidural catheter. Sundberg et al. [19] noted that after the administration of bupivacaine 0.5% via an epidural catheter inserted at the Th4 level, motor blockade of the intercostal muscles was produced. In the present study also, motor blockade of the adductor pollicis muscle may have been produced due to the epidural blockade. To our knowledge, the effect of epidural blockade on the monitoring of neuromuscular blockade at the adductor pollicis muscle has not yet been clarified. However, in the present study, the thoracic epidural anesthesia may have affected the TOF response measured at the adductor pollicis muscle.

We presumed that the vagal nerve would be stimulated during lung surgery. No previous study has shown vagal nerve stimulation during lung surgery. However, Yu et al. [20], in a study in rats, described that the lungs were richly innervated by vagal nerves, and when hypertonic saline was injected into the periphery of the lungs, the vagal nerves were activated. Consequently, it may be possible that in our OLV group, the surgical procedure itself activated the vagal nerve, resulting in a quick recovery from neuromuscular blockade.

On the other hand, Park et al. [21] reported that the vagal reflex was potentiated by thoracic epidural anesthesia. They also noted that the vagal reflex was especially apparent when the sensory level was above Th6 in patients receiving epidural anesthesia [21]. Accordingly, in our OLV group, an epidural anesthesia-induced vagal reflex may have accelerated the recovery of neuromuscular blockade.

It has been reported that when arterial blood pressure fell, sympathetic activity increased [22]. Although severe hypotension did not occur in our present study, a decrease in arterial blood pressure could have suppressed vagal nerve activity, resulting in delayed recovery from neuromuscular blockade.

In our OLV and control groups, the patients were in the lateral and supine positions, respectively. Miralles et al. [23] reported that, in healthy subjects, significantly higher electromyographic activities were recorded in the sternocleidomastoid muscle in the lateral position than in the supine position. Such body-position effects may have accelerated the recovery of T1/control in our study. In addition, Kamenik [24] showed that cardiac output decreased significantly after a shift from the supine to the left lateral position in humans. It has also been noted that the rapidity of the onset time of vecuronium-induced neuromuscular blockade in the adductor pollicis muscle was clearly related to cardiac

output [25]. Based on this, it is possible that the onset of neuromuscular blockade may be delayed in patients in the left lateral position.

In conclusion, we studied the recovery of neuromuscular blockade in patients receiving OLV for lung surgery under general anesthesia combined with epidural anesthesia, performed via the Th3/4 or Th4/5 intervertebral space, comparing the recovery with that in patients receiving TLV for colon surgery under general anesthesia and epidural anesthesia, performed via Th10/11 or Th11/12. Recovery of T1/control was hastened in patients undergoing OLV for lung surgery as compared with that in patients receiving TLV for colon surgery. Although the time to the return of T1, T2, T3, or T4 and the recovery of the TOF ratio were not affected during OLV, it is noteworthy that recovery from vecuronium-induced neuromuscular blockade may be accelerated during OLV for lung surgery.

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