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ORIGINAL RESEARCH

# Low-Dose Neostigmine at 0.7 Train-of-Four Ratio Safely Accelerates Neuromuscular Recovery: A Prospective Observational Study

Chang-Hoon Koo[1,2,*], Chang Ik Choi[3,*], Insun Park[1,2], Sohyun Lee[1], Ah-Young Oh[1,2]

[1]Department of Anesthesiology and Pain Medicine, Seoul National University Bundang Hospital, Seongnam, Korea; [2]Department of Anesthesiology and Pain Medicine, Seoul National University College of Medicine, Seoul, Korea; [3]Department of Anesthesiology and Pain Medicine, Anyang Sam Hospital, Anyang, Korea

*These authors contributed equally to this work

Correspondence: Ah-Young Oh, Department of Anesthesiology and Pain Medicine, Seoul National University College of Medicine, Seoul National University Bundang Hospital, Seongnam, Korea, Tel +82-31-787-7506, Fax +82-31-787-4063, Email ohahyoung@hanmail.net



**Purpose:** Neostigmine is recommended to reverse the minimal depth of the neuromuscular blockade. However, neostigmine administered at a minimal blockade may cause paradoxical muscle weakness. This study aimed to assess the efficacy and safety of neostigmine administered at a train-of-four (TOF) ratio of 0.7 for neuromuscular recovery.

**Patients and methods:** This prospective observational study included 54 patients who underwent thyroid surgery under intravenous anesthesia. Neostigmine 20 mcg/kg was administered when the TOF ratio spontaneously recovered to 0.7 during surgery. The recovery times were recorded based on the time from a TOF ratio of 0.7 to 1.0 and 0.9. Outcomes were compared with those of patients with spontaneous recovery, including recovery times and incidence of adverse events, such as paradoxical muscle weakness.

**Results:** Neostigmine administration significantly reduced recovery time from a TOF ratio of 0.7 to 1.0 (4.9 vs 10.2 min, $P = 0.001$), from 0.7 to 0.9 (3.3 vs 6.7 min, $P = 0.002$), and from 0.9 to 1.0 (1.5 vs 4.3 min, $P = 0.003$), compared with spontaneous recovery. Importantly, no patients exhibited a reduction in the TOF ratio after neostigmine administration, nor did they develop clinical signs or symptoms of paradoxical muscle weakness after surgery.

**Conclusion:** Administering 20 mcg/kg neostigmine at a TOF ratio of 0.7 is effective and safe, facilitating neuromuscular recovery without adverse effects. This treatment regimen can enhance surgical workflow efficiency and reduce patient recovery times while maintaining patient safety.

**Keywords:** neostigmine, neuromuscular blockade, neuromuscular recovery, train-of-four ratio, paradoxical muscle weakness

## Introduction

Neostigmine, an acetylcholinesterase inhibitor, acts as a conventional reversal agent for neuromuscular blockade by preventing the breakdown of acetylcholine, thereby increasing its concentration at the neuromuscular junction.[1] This increased acetylcholine level competes with nondepolarizing neuromuscular blocking agents, facilitating effective recovery of neuromuscular function.[2]

Current guidelines suggest that neostigmine can be administered when the train-of-four (TOF) ratio exceeds 0.2 or 0.4.[3,4] However, the potential development of paradoxical muscle weakness following neostigmine administration is a critical concern. When neostigmine is administered at high doses or under full recovery from neuromuscular blockade, excessive accumulation of acetylcholine can result in sustained depolarization of the postsynaptic nicotinic receptors at the neuromuscular junction.[5] Sustained depolarization impairs normal neuromuscular transmission, leading to muscle weakness.[5] Consequently, neostigmine should be avoided when the TOF ratio exceeds 0.7.[6]

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However, even at a TOF ratio of 0.7–0.9, there is a risk of pharyngeal dysfunction or upper airway obstruction,[7–10] indicating a need for reversal agents. Furthermore, evidence supports the safe use of a small dose of 20 mcg/kg neostigmine at this TOF stage.[11,12]

Therefore, this study aimed to investigate whether neostigmine administered at a TOF ratio of 0.7 can facilitate safe and effective neuromuscular blockade reversal. We hypothesized that this approach would improve recovery from neuromuscular blockade without inducing paradoxical muscle weakness.

# Methods

## Ethics Statement

This prospective, single-center observational study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of Seoul National University Bundang Hospital (Approval number: B2209-780-301; Approval date: September 26, 2022). Informed consent was obtained from all patients before the surgery. In addition, anonymized data from a control group that did not receive neostigmine from a previous study were used for comparison.[12]

## Inclusion and Exclusion Criteria

Adult patients (aged ≥ 20 years) with a body mass index of 18.5–25 kg/m$^2$ scheduled for elective thyroid surgery under general anesthesia, between October 2022 and August 2023, were included. Patients were excluded if they refused to participate or had a history of hypersensitivity to acetylcholinesterase inhibitors, neuromuscular disease, or hepatic or renal dysfunction.

## Anesthesia

Upon patient arrival in the operating room, standard monitoring, including electrocardiography, noninvasive blood pressure, bispectral index (BIS), and pulse oximetry, was performed. Anesthesia induction and maintenance were achieved using target-controlled infusion of propofol and remifentanil via Orchestra infusion pumps (Fresenius Vial, Brezins, France). The effect-site concentrations of propofol and remifentanil were adjusted to maintain a BIS of 40–60 and heart rates and blood pressures within 20% of the preoperative values. Central body temperature was monitored with an esophageal temperature probe (Top probe, Meditop Co., Gyeonggi-do, South Korea) and maintained above 35 °C.

Neuromuscular blockade was induced with rocuronium 0.6 mg/kg. Neuromuscular function was assessed using acceleromyography (AMG, TOF-Watch SX®; Organon, Swords, Ireland). Following skin cleansing, two skin electrodes (7–11 mm) were placed over the ulnar nerve proximal to the wrist at an interelectrode distance of 3–6 cm. The monitored forearm was immobilized, and the acceleration transducer was attached to the volar aspect of the thumb's distal phalanx using a small elastic hand adapter (TOF-Watch Hand Adapter®; Schering-Plough, Swords, Ireland). Calibration was automatically performed after 50 Hz tetanic stimulation for 5s. TOF stimulation was subsequently applied every 15s, and a stable baseline (< 5% variation in the TOF ratio) was confirmed for at least 2 min before rocuronium administration.

Patients were intubated when their TOF count was two. After the intubating dose, no additional rocuronium was administered to facilitate intraoperative recurrent laryngeal nerve monitoring. Consequently, spontaneous recovery of neuromuscular function progressed during surgery. When the TOF ratio spontaneously recovered to 0.7, an attending anesthesiologist administered a 20-mcg/kg dose of neostigmine and 4 mcg/kg of glycopyrrolate. If recurrent laryngeal nerve monitoring necessitated neostigmine administration before the TOF ratio reached 0.7, the patient was excluded from the analysis.

The time from neostigmine administration to a TOF ratio of 0.9 and 1.0 was recorded. After surgery, patients were extubated and transferred to a post-anesthesia care unit (PACU). In the PACU, an independent investigator monitored for signs of weakness, including hypoxia (oxygen saturation [$SpO_2$] < 90%), inability to sustain a head lift for 5s, and diplopia.

## Outcomes

The primary outcome of this study was the time from a TOF ratio of 0.7 to 1.0 after neostigmine administration. Secondary outcomes included the time from a TOF ratio of 0.7 to 0.9, the time from a TOF ratio of 0.9 to 1.0, and clinical symptoms or signs of muscle weakness, including hypoxia ($SpO_2$ < 90%), inability to sustain a head lift for 5s, and diplopia.

## Sample Size Calculation

Based on our previous data showing that the time from a TOF ratio of 0.7 to 1.0 was 15.2 (≈ 15) ± 12.5 (≈ 13) min without neostigmine administration,[12] we performed a one-sided, one-sample *t*-test calculation to detect a 30% reduction in recovery time with 80% power at a significance level of 0.05. This yielded a minimum of 53 patients, and after accounting for a 10% dropout rate, 59 patients were required.

## Statistical Analyses

Continuous variables are presented as means with standard deviations or medians with interquartile ranges, with the normality of data distribution assessed using the Shapiro–Wilk test. Categorical variables are presented as numbers and percentages. Comparisons between the groups were conducted using Student's *t*-test for normally distributed continuous variables and the Mann–Whitney *U*-test for non-normally distributed variables. The chi-squared or Fisher's exact tests were used for categorical variables. All statistical analyses were conducted using Jamovi software version 2.4.14 (Jamovi Project, Sydney, Australia). Statistical significance was set at $p < 0.05$.

# Results

A total of 59 patients were enrolled in the study from October 2022 to August 2023, and five were excluded due to neostigmine administration before achieving a TOF ratio of 0.7 (n = 3) or technical errors with neuromuscular monitoring (n = 2). Ultimately, 54 patients were included in the final analysis (Figure 1). The patient characteristics are shown in Table 1. There were no significant differences between patients who received neostigmine and those who did not.

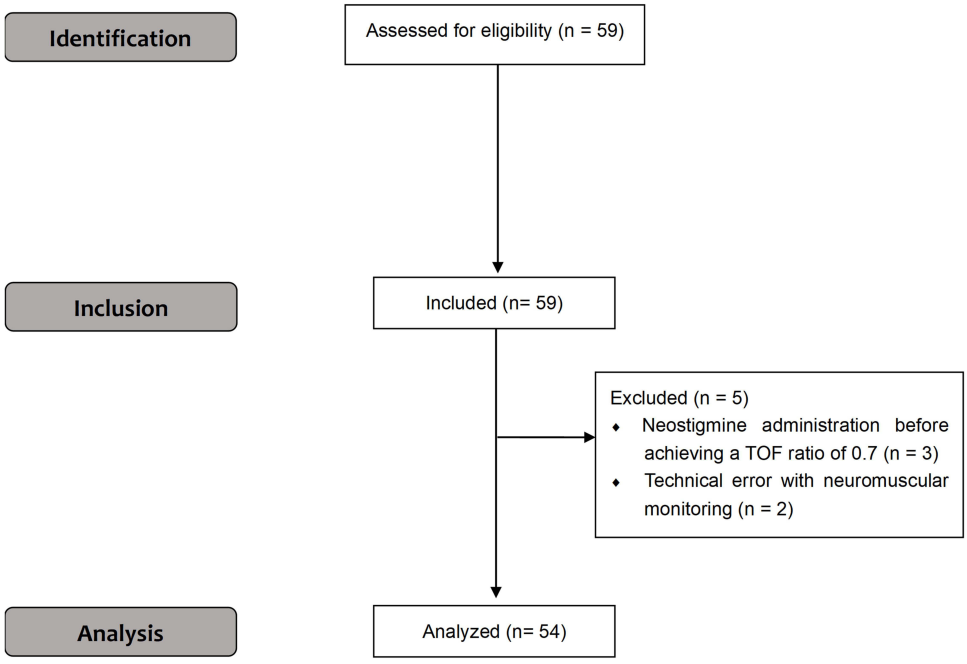


**Figure 1** Flow diagram of patient inclusion and exclusion.
**Abbreviation**: TOF, train-of-four.


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**Table 1** Demographic Variables in the Neostigmine and Control Groups

| Variable | Neostigmine Group | Control Group | p-value |
|---|---|---|---|
| **Age (years), mean ± SD** | 42.3 ± 9.8 | 49.0 ± 11.1 | 0.083[a] |
| **Male/female, n (%)** | 10 (18.5)/44 (81.5) | 4 (50.0)/4 (50.0) | 0.069[b] |
| **Height (cm), mean ± SD** | 165.0 ± 6.6 | 161.7 ± 9.8 | 0.226[a] |
| **Weight (kg), median [Q1, Q3]** | 59.0 [56.0, 68.0] | 60.8 [53.3, 67.0] | 0.508[c] |
| **BMI (kg/m$^2$), median [Q1, Q3]** | 22.9 [21.2, 24.0] | 22.3 [22.0, 23.2] | 0.933[c] |
| **ASA I/II, n (%)** | 49 (90.7)/5 (9.3) | 6 (75.0)/2 (25.0) | 0.220[b] |

**Notes**: [a]Student's *t*-test. [b]Fisher's exact test. [c]Mann–Whitney *U*-test.
**Abbreviations**: SD, standard deviation; BMI, body mass index; ASA, American Society of Anesthesiologists.

**Table 2** Time to Recovery and Postoperative Weakness

| Variable | Neostigmine Group | Control Group | p-value |
|---|---|---|---|
| **Time from rocuronium administration to a TOF ratio of 0.7 (min), median [Q1, Q3]** | 48.0 [40.5, 53.2] | 45.0 [38.7, 61.6] | 0.745[a] |
| **Mean ± SD** | 49.6 ± 11.9 | 48.1 ± 15.4 | |
| **Time from a TOF ratio of 0.7 to 1.0 (min), median [Q1, Q3]** | 4.9 [4.3, 5.7] | 10.2 [8.0, 17.8] | 0.001[a] |
| **Mean ± SD** | 5.1 ± 1.5 | 12.5 ± 7.3 | |
| **Time from a TOF ratio of 0.7 to 0.9 (min), median [Q1, Q3]** | 3.3 [2.7, 4.1] | 6.7 [5.4, 10.9] | 0.002[a] |
| **Mean ± SD** | 3.6 ± 1.3 | 7.7 ± 4.1 | |
| **Time from a TOF ratio of 0.9 to 1.0 (min), median [Q1, Q3]** | 1.5 [1.3, 1.8] | 4.3 [2.0, 7.1] | 0.003[a] |
| **Mean ± SD** | 1.6 ± 0.5 | 4.9 ± 3.5 | |
| **PACU event, n** | 0 | 0 | 1.000 |

**Note**: [a]Mann–Whitney *U*-test.
**Abbreviations**: TOF, train-of-four; SD, standard deviation; PACU, post-anesthesia care unit.

The median time from a TOF ratio of 0.7 to 1.0 was significantly lower in the neostigmine than control group (4.9 vs 10.2 min, p = 0.001). Similarly, the median time from a TOF ratio of 0.7 to 0.9 and from 0.9 to 1.0 was significantly shorter in the neostigmine than control group (3.3 vs 6.7 min, p = 0.002; 1.5 vs 4.3 min, p = 0.003, respectively; Table 2).

A post-hoc power analysis was performed. From the one-sample perspective — with a population mean of 15.2 (SD, 12.5) min from prior data, an observed mean of 5.1 min in the neostigmine group, and a one-sided significance level of 0.05 — the effect size was 0.82, yielding a post-hoc power of 100%. From the two-sample perspective, with means of 12.5 (SD, 7.3) min and 5.1 (SD, 1.5) min in the control and neostigmine groups, respectively, the post-hoc power was 81.5% at a significance level of 0.05.

In the neostigmine group, we did not observe any cases of a return to a decrease in the TOF ratio after neostigmine administration. None of the patients in either group experienced clinical symptoms or signs of muscle weakness, dyspnea, or oxygen desaturation in the PACU.

## Discussion

This study demonstrated that administering 20 mcg/kg of neostigmine at a TOF ratio of 0.7 significantly reduced the time to achieve a TOF ratio of 1.0, without causing paradoxical muscle weakness. Additionally, the times from a TOF ratio of 0.7 to 0.9 and from 0.9 to 1.0 were significantly reduced by > 50%, compared with the time required for spontaneous recovery, with no adverse clinical symptoms or signs.

Several recent studies have shown results similar to ours regarding the efficacy and safety of low-dose neostigmine. Fuchs–Buder et al[13] conducted a study involving 48 patients under desflurane anesthesia who received either saline or 10, 20, or 30 mcg/kg of neostigmine at a TOF ratio of 0.6. Data demonstrated that a 20-mcg/kg dose of neostigmine significantly reduced the time to achieve a TOF ratio of more than 0.9, compared with saline, without adverse effects, indicating its efficacy in reversing minimal neuromuscular blockade. Two randomized controlled trials reported that administering neostigmine at minimal blockade was not associated with clinical evidence of neostigmine-induced muscle weakness.[14,15] Interestingly, the incidence of diplopia was significantly higher in the group that did not receive

neostigmine,[14] suggesting that neostigmine administration at a minimal blockade reduces residual neuromuscular blockade.

However, other studies have reported conflicting results. Kent et al[16] administered neostigmine at a mean dose of 35 mcg/kg in awake healthy volunteers. The authors found that neostigmine significantly decreased handgrip strength, forced expiratory volume, and forced vital capacity. They stated that neostigmine could elicit excessive concentrations of acetylcholine, leading to a depolarizing neuromuscular blockade and unintended adverse symptoms in awake patients, suggesting that careful monitoring is essential when administering neostigmine. Similarly, Payne et al[17] used two doses (2.5 mg) or a single dose (5 mg) of neostigmine in anesthetized patients and observed decremental tetanic responses. The authors emphasized the potential of neostigmine to cause a neuromuscular blockade when administered at higher doses, especially without adequate neuromuscular monitoring. However, in our study, we used a lower neostigmine dose (20 mcg/kg) with strict, advanced neuromuscular monitoring. It is plausible that these procedures contributed to the absence of neostigmine-induced muscle weakness.

The mechanism underlying the absence of neostigmine-induced muscle weakness may be explained by the abundance of nicotinic acetylcholine receptors (nAChRs) present at the neuromuscular junction, which greatly surpasses the number required for effective neuromuscular transmission.[18] This receptor reserve ensures that, even with substantial receptor occupation by neuromuscular blocking agents, the twitch response is not suppressed until more than 70% of the nAChRs are occupied. In other words, even if neuromuscular function appears to be fully recovered, approximately 70% of nAChRs may still be bound by neuromuscular-blocking agents. Therefore, increased acetylcholine levels, induced by a 20-mcg/kg dose of neostigmine at a TOF ratio of 0.7, still seem to compete with the remaining neuromuscular blocking agents, thereby promoting neuromuscular recovery.

Although sugammadex provides faster and more predictable reversal of rocuronium-induced neuromuscular blockade than neostigmine, its availability remains limited in many healthcare settings worldwide, and its substantially higher cost continues to be a barrier to routine use. A global survey reported that sugammadex was available to only 46% of anesthesia providers across 183 countries.[19] Furthermore, the cost-effectiveness of sugammadex over neostigmine remains controversial and appears to depend on patient risk profiles and local healthcare economics.[20,21] This study suggests that low-dose neostigmine may offer a safe, effective, and cost-efficient alternative for reversing minimal neuromuscular blockade. Investigating the efficacy and safety of neostigmine therefore has practical relevance for a substantial proportion of clinical settings worldwide.

This study had some limitations. First, the control group data were derived from a previous study conducted at our institution.[12] Owing to ethical concerns regarding withholding neostigmine administration in patients with incomplete neuromuscular recovery, prospective enrollment of a concurrent control group was not feasible, and a historical control design was adopted. The nonrandomized design and small control group size preclude definitive exclusion of confounding. However, the time from rocuronium administration to a TOF ratio of 0.7 (which reflects baseline neuromuscular recovery) was comparable between groups, and both groups were managed with the same anesthetic technique, monitoring equipment, and rocuronium dosing protocol. Second, this study was conducted in patients undergoing thyroidectomy with a single intubating dose of rocuronium (0.6 mg/kg) under total intravenous anesthesia. The results may not be directly applicable to longer surgical procedures requiring repeated or higher doses of neuromuscular blocking agents, or to cases performed under volatile anesthesia, which is known to potentiate neuromuscular blockade.[22] Third, this was a single-center study with a relatively homogenous population, which limits the generalizability of our findings to patients of different ethnicities, body compositions, or comorbidity profiles. However, the single-center design ensured uniformity in anesthetic protocols, surgical techniques, and neuromuscular monitoring practices. Fourth, paradoxical muscle weakness was assessed based only on clinical symptoms or signs in the PACU. Measuring the TOF ratio could provide more accurate, objective evidence. However, TOF stimulation can cause discomfort in awake patients.

The strength of our study was that the endpoint for neuromuscular recovery was defined as a TOF ratio of 1.0, which is recommended for non-normalized TOF ratios with acceleromyography.[22] Furthermore, we enrolled patients who underwent surgery under intravenous rather than inhalational anesthesia, which minimized confounding factors related to the anesthetic technique.[22]

## Conclusion

In conclusion, administering 20 mcg/kg of neostigmine at a TOF ratio of 0.7 significantly reduced neuromuscular recovery time by >50% compared with spontaneous recovery, without causing paradoxical muscle weakness or adverse clinical events. These findings suggest that low-dose neostigmine can be a safe and effective option for reversing minimal neuromuscular blockade. Further randomized controlled trials are warranted to confirm these results.

## Data Sharing Statement

Deidentified data can be made available upon reasonable request to the corresponding author.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article, gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The author(s) report no conflicts of interest in this work.

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