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REVIEW

Monitoring the depth of neuromuscular blockade in the intensive care unit

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ABSTRACT:

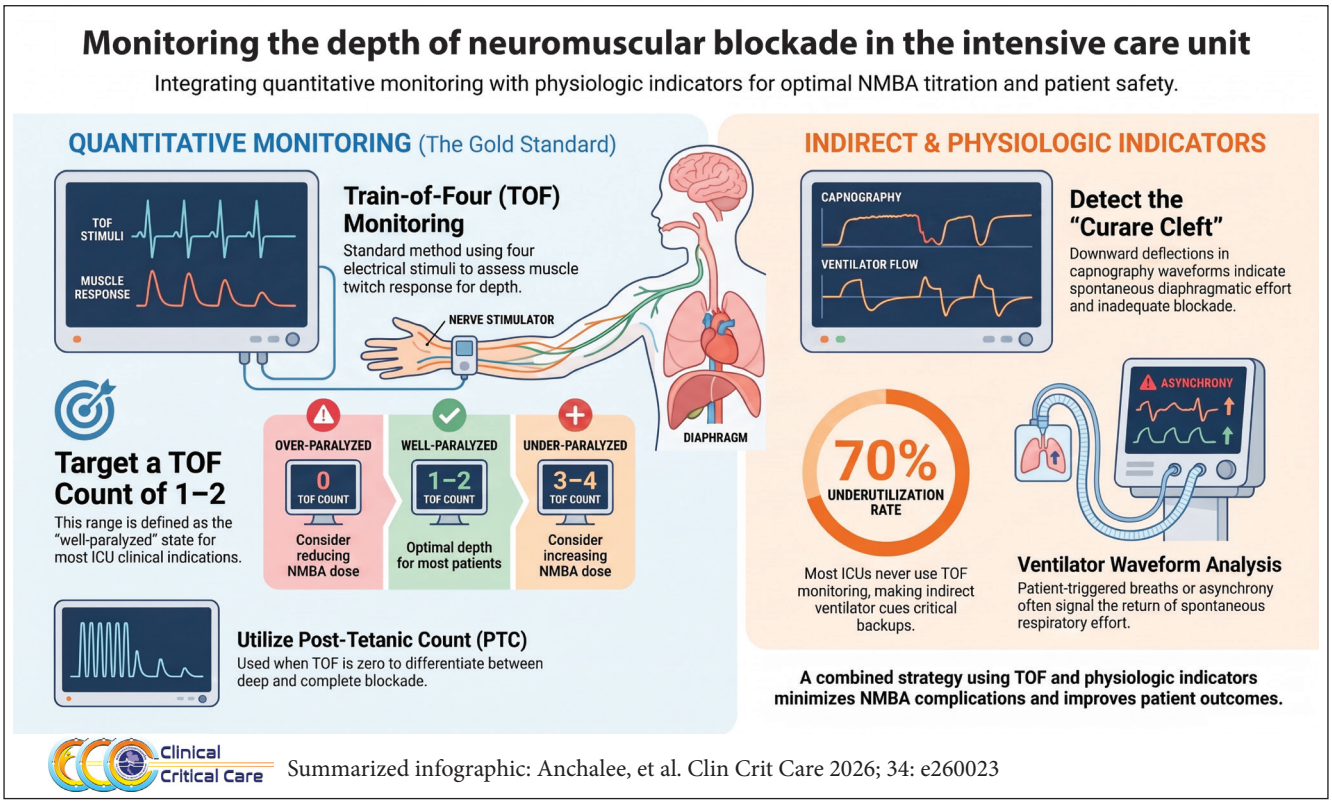
Neuromuscular blocking agents (NMBAs) are widely used in the intensive care unit (ICU) to facilitate mechanical ventilation; however, excessive or prolonged blockade increases the risk of complications such as ICU-acquired weakness. Quantitative train-of-four (TOF) monitoring remains the standard for assessing depth of neuromuscular blockade, although it is often underutilized in ICU practice. Indirect methods including capnography, ventilator waveform analysis, and respiratory effort indices provide complementary but nonspecific information. Integration of quantitative and physiologic monitoring supports safer and more individualized NMBAs management in critically ill patients. This review summarizes current neuromuscular monitoring strategies in the ICU and discusses the clinical utility, limitations, and practical integration of quantitative and indirect monitoring modalities during NMBAs administration.

Keywords: Neuromuscular blocking agents; Neuromuscular monitoring; Train-of-Four stimulation; Critical care; Intensive care units

INTRODUCTION

Neuromuscular blocking agents (NMBAs) are medications that inhibit neuromuscular transmission at the neuromuscular junction, resulting in skeletal muscle paralysis while providing no sedative or analgesic effects. In the intensive care unit (ICU), NMBAs are frequently used to facilitate mechanical ventilation, optimize oxygenation, reduce patient-ventilator asynchrony, and control intracranial or intra-abdominal pressure [1-3] (Table 1). Commonly used nondepolarizing NMBAs in ICU practice include cisatracurium, atracurium, rocuronium, and vecuronium. These agents competitively antagonize acetylcholine at postsynaptic nicotinic receptors on the motor endplate, thereby preventing depolarization and skeletal muscle contraction.

NMBAs differ in their pharmacokinetic profiles, duration of action, and elimination pathways. Rocuronium and vecuronium are aminosteroid compounds that primarily undergo hepatic metabolism and biliary or renal excretion, whereas cisatracurium and atracurium are benzylisoquinolinium compounds that undergo organ-independent Hofmann elimination. Depending on the agent and patient condition, the duration of action may range from approximately 20–80 minutes after intermittent bolus administration, while continuous infusion may produce substantially prolonged recovery in critically ill patients with organ dysfunction. A concise summary of the pharmacology of commonly used NMBAs is presented in Table 2.



Clinical Critical Care Summarized infographic: Anchalee, et al. Clin Crit Care 2026; 34: e260023

In ICU practice, NMBAs are typically administered intravenously either as intermittent bolus doses or continuous infusions, with dosage individualized according to the clinical indication and the desired depth of neuromuscular blockade. However, landmark randomized trials in patients with acute respiratory distress syndrome (ARDS) employed standardized fixed-dose cisatracurium regimens that were generally higher than conventional dosing recommendations, including a 50-mg intravenous bolus followed by a continuous infusion of 5 mcg/kg/min [4] or a 15-mg intravenous bolus followed by a continuous infusion of 37.5 mg/h for 48 hours [5–7] (Table 3).

Although NMBAs provide important clinical benefits, excessive or prolonged neuromuscular blockade is associated with adverse outcomes, including ICU-acquired weakness, prolonged mechanical ventilation, and delayed recovery [8-11] (Table 4). The use of NMBAs in critically ill patients differs substantially from anesthetic practice in the operating room. ICU patients often have altered pharmacokinetics and pharmacodynamics due to sepsis, organ dysfunction, hypothermia, and drug interactions [12-13]. Continuous or prolonged NMBAs infusion further increases the risk of accumulation and unrecognized residual paralysis [14-15]. Prolonged exposure to NMBAs generally refers to continuous infusion extending beyond the 24–48-hour period commonly recommended in current critical care practice, particularly in patients with ARDS, where neuromuscular blockade is typically limited to 48 hours [16]. In patients with profound neuromuscular blockade, conventional Train-of-Four (TOF) monitoring may demonstrate a TOF count of zero, limiting the clinician's ability to distinguish between deep and complete blockade. In such situations, post-tetanic count (PTC) monitoring provides additional clinical value by helping estimate blockade depth and guiding NMBAs titration [17].

KEY MESSAGES:

- Quantitative train-of-four (TOF) monitoring remains the standard method for assessing depth of neuromuscular blockade in the ICU, yet it is frequently underutilized in clinical practice.
- Indirect modalities such as capnography, ventilator waveform analysis, and respiratory effort indices provide complementary information but cannot replace direct neuromuscular monitoring.
- A multimodal monitoring strategy integrating quantitative and physiologic parameters supports individualized NMBAs titration and may reduce NMBAs-related complications in critically ill patients.

Despite these risks and the recognized benefits of objective monitoring, neuromuscular monitoring remains underutilized in many ICUs. A review article reported that only 4% of ICUs routinely used TOF monitoring, where as approximately 70% never used TOF monitoring in patients receiving continuous neuromuscular blockade [18]. In 2016, the Society of Critical Care Medicine (SCCM) published clinical practice guidelines for sustained neuromuscular blockade in critically ill adults. Regarding the monitoring of neuromuscular blockade, the guideline made three key recommendations: (1) peripheral nerve stimulation using Train-of-Four (TOF) monitoring may be a useful tool for assessing the depth of neuromuscular

blockade when incorporated into a comprehensive patient assessment that includes clinical evaluation; (2) TOF monitoring alone should not be used as the sole method for monitoring the depth of neuromuscular blockade in patients receiving continuous infusions of NMBAs; and (3) patients receiving continuous NMBAs infusions should receive a structured physiotherapy regimen to reduce complications associated with prolonged paralysis[19].

Neuromuscular monitoring can be broadly classified into direct (standard) monitoring using peripheral nerve stimulation [17-18,20] and indirect monitoring based on respiratory mechanics, ventilator waveforms [21], capnography, and physiologic surrogates of respiratory muscle activity. Understanding the principles, strengths, and limitations of each modality is essential for safe and individualized NMBAs titration in critically ill patients.

STANDARD NEUROMUSCULAR MONITORING

Train-of-Four (TOF) monitoring

Quantitative or semi-quantitative peripheral nerve stimulation using the Train-of-Four (TOF) technique is considered the standard method for assessing the depth of neuromuscular blockade (Table 5). TOF stimulation consists of four supramaximal electrical stimuli delivered at a frequency of 2 Hz to a peripheral nerve, most commonly the ulnar (Figure 1), facial, or posterior tibial nerve [22-23]. The TOF response is evaluated by counting the number of visible or measurable muscle twitches (TOF count) or by calculating the ratio of the amplitude of the fourth twitch to the first (TOF ratio). In the ICU, TOF monitoring is typically used to guide continuous NMBAs infusions, with the target depth of blockade determined according to the underlying clinical indication. A TOF count of 1–2 twitches is commonly defined as a well-paralyzed state [23].



Figure 1. Placement of a train-of-four (TOF) monitor at the adductor pollicis muscle following ulnar nerve stimulation. Surface electrodes are positioned along the course of the ulnar nerve at the wrist, and the acceleromyography sensor is attached to the thumb to measure contraction of the adductor pollicis muscle during neuromuscular monitoring.

Different muscle groups demonstrate variable sensitivity to NMBAs. Respiratory muscles, including the diaphragm and laryngeal muscles, are relatively resistant to neuromuscular blockade and typically recover more rapidly than peripheral muscles such as the adductor pollicis [24-25]. The diaphragm exhibits relative resistance to nondepolarizing NMBAs compared with the adductor pollicis muscle. Experimental studies have shown that approximately 1.5- to 2-fold higher doses are required to achieve an equivalent level of diaphragmatic blockade. As a result, complete neuromuscular blockade at the adductor pollicis does not necessarily indicate complete paralysis of the diaphragm [26]. Consequently, some patients may continue to exhibit spontaneous respiratory effort or patient–ventilator dyssynchrony despite a TOF count of 1–2 at the ulnar nerve. In this context, facial nerve monitoring, particularly assessment of the orbicularis oculi muscles, may better reflect diaphragmatic and respiratory muscle activity than ulnar nerve stimulation [24,26]. Nevertheless, ulnar nerve monitoring remains useful for evaluating recovery from neuromuscular blockade because the adductor pollicis is more sensitive to residual neuromuscular weakness and recovers later than respiratory muscles.

Because NMBAs produce paralysis without affecting consciousness, analgesia, or memory, adequate sedation and analgesia must always be ensured before and during NMBAs administration. Critically ill patients receiving continuous neuromuscular blockade are at risk of unintended awareness if sedation is inadequate. Current ICU practice therefore, recommends routine monitoring of sedation depth using validated clinical scales, such as the Richmond Agitation-Sedation Scale (RASS) or Sedation-Agitation Scale (SAS), and, when appropriate, processed electroencephalographic monitoring such as the bispectral index (BIS), particularly in deeply sedated or pharmacologically paralyzed patients [19,27].

The study by Rezaiguia-Delclaux et al. demonstrated that, in patients with ARDS, the addition of TOF monitoring to routine clinical assessment did not improve ICU mortality or reduce the duration of mechanical ventilation but was associated with greater atracurium consumption compared with clinical assessment alone [28]. In contrast, Hraiech et al. reported that a nurse-driven, TOF-guided protocol for NMBAs administration in patients with ARDS reduced cisatracurium consumption without compromising the adequacy of neuromuscular blockade [29]. Furthermore, Rudis et al. showed that peripheral nerve stimulation–guided titration reduced vecuronium requirements while accelerating the recovery of neuromuscular function and spontaneous ventilation in critically ill patients [30]. These conflicting findings should be interpreted cautiously due to differences in their NMBAs protocols and NMBAs used.

Although TOF monitoring is widely accepted, several factors should be considered when interpreting its results in critically ill patients. Heier et al. found during continuous vecuronium infusion, a decrease in adductor pollicis muscle temperature resulting from core hypothermia significantly increases the degree of neuromuscular blockade [31]. In addition, qualitative (visual or tactile) assessment is inherently subjective and may underestimate residual neuromuscular blockade, particularly when the TOF ratio

Table 1. Indications for neuromuscular blocking agents use in the intensive care unit [3].

Indication	Clinical context
1.Orotracheal intubation	Facilitation of endotracheal intubation and optimization of intubating conditions
2.Acute Respiratory Distress Syndrome (ARDS)	Improvement of ventilator synchrony and reduction of oxygen consumption in moderate-to-severe ARDS
3.Status asthmaticus	Reduction of severe bronchospasm and facilitation of mechanical ventilation
4.Elevated intracranial pressure (ICP)	Prevention of coughing, straining, and ventilator dyssynchrony that may increase ICP
5.Intraabdominal hypertension (IAH)	Improvement of abdominal wall compliance and reduction of intra-abdominal pressure
6.Therapeutic hypothermia after cardiac arrest	Prevention of shivering during therapeutic hypothermia

Table 2. Concise pharmacology of commonly used nondepolarizing neuromuscular blocking agents (NMBAs).

NMBAs	Class	IV bolus dose (mg/kg)	Continuous infusion (mcg/kg/min)	Onset of action (min)	Duration of action (min)	Metabolism / Elimination	Clinical consideration
Cisatracurium	Benzylisoquinolinium	0.1-0.2	1-3	2-3	30-60	Organ-independent Hofmann elimination and nonspecific ester hydrolysis; minimal renal elimination	Predictable recovery in organ failure
Atracurium	Benzylisoquinolinium	0.4-0.5	5-20	3-5	20-35	Organ-independent Hofmann elimination and ester hydrolysis	Histamine release
Rocuronium	Aminosteroid	0.6-1.2	8-12	1-2	60-80	Primarily hepatic uptake and biliary excretion with minor renal elimination	Prolonged duration in hepatic dysfunction
Vecuronium	Aminosteroid	0.08-0.1	0.8-1.7	3-4	20-35	Hepatic metabolism with biliary and renal excretion	Prolonged recovery in hepatic/renal dysfunction

Adapted from: Tezcan B, Turan S, Ozgok A. Current Use of Neuromuscular Blocking Agents in Intensive Care Units.Turk J Anaesthesiol Reanim. 2019;47(4):273–281. with additional information synthesized from contemporary pharmacology references.

Table 3. Landmark randomized controlled trials of neuromuscular blocking agents in ARDS.

Authors/Year	Type of study / Population	NMBAs	Monitoring NMBAs	Outcomes
Gainnier et al. [4] 2004	Multicenter RCT (n = 56)	Cisatracurium: 50-mg IV bolus followed by 5 μg/kg/min infusion for 48 h	TOF-guided titration (target: TOF = 0 at orbicularis oculi); TOF assessed every 8 h	Improved oxygenation after 48 h of neuromuscular blockade.
Papazian et al. (ACURASYS trial) [5] 2010	Multicenter RCT (n = 340)	Cisatracurium: 15-mg IV bolus followed by 37.5 mg/h infusion for 48 h	No peripheral nerve stimulation; deep sedation (Ramsay score 6)	Early cisatracurium improved adjusted 90-day survival and increased ventilator-free days without increasing ICU-acquired weakness.
Guervilly et al. [6] 2017	Randomized controlled trial (n = 30)	Cisatracurium: 15-mg IV bolus followed by 37.5 mg/h infusion for 48 h	TOF-guided supplemental boluses when TOF ≥1	Reduced spontaneous expiratory effort and transpulmonary pressure in moderate-to-severe ARDS.
ROSE Trial (PETAL Network) (2019) [7]	Multicenter RCT (n = 1,006)	Cisatracurium: 15-mg IV bolus followed by 37.5 mg/h infusion for 48 h	No TOF monitoring; deep sedation	No significant improvement in 90-day mortality compared with usual care using lighter sedation.

Abbreviations: ARDS: acute respiratory distress syndrome; NMBAs: neuromuscular blocking agents; RCT: randomized controlled trial; TOF: Train-of-Four.

Table 4. Complications of neuromuscular blocking agents (NMBAs) in critically ill patients.

Complication	Details
1.Complication related immobility [3]	Eschar formation; keratitis; corneal abrasion secondary to loss of eyelid closure reflexes
2.ICU-acquired weakness (ICU-AW)	NMBAs use is associated with an increased risk of ICU-AW (odds ratio 2.77) [9]. Both corticosteroids and NMBAs have been identified as pharmacologic risk factors [11].
3.Muscle atrophy and inflammation [3]	Prolonged neuromuscular blockade may contribute to skeletal muscle atrophy and inflammatory myopathy.
4.Awareness during paralysis	Inadequate sedation and analgesia during NMBAs administration may result in unintended awareness.
5.Increased long-term mortality	NMBAs exposure has been associated with increased long-term mortality (adjusted hazard ratio 1.26; 95% CI 1.07–1.49) [12].
6.Residual neuromuscular blockade	Residual paralysis is common, occurring in 37% of non-postoperative and 44% of postoperative patients [14]. Rocuronium demonstrates prolonged recovery in ICU patients with organ failure (10 vs. 4.6 hours) [15]. Preferential use of cisatracurium has been recommended in critically ill patients because it may reduce the risk of drug accumulation and persistent neuromuscular blockade. Unlike steroid-based neuromuscular blocking agents, cisatracurium undergoes organ-independent metabolism through Hofmann elimination, resulting in more predictable pharmacokinetics in the setting of multiorgan dysfunction [10].

Table 5. Quantitative neuromuscular monitoring: Depth of neuromuscular blockade and nerve stimulation sites [17,20,23].

A. Depth of Neuromuscular Blockade (Quantitative monitoring) [17]	
Depth of block	**Monitoring criteria**
Complete block	PTC = 0
Deep block	PTC > 1, TOF count = 0
Moderate block	TOF count = 1-3
Shallow block	TOF ratio < 0.4
Minimal block	TOF ratio = 0.4-0.9
Acceptable recovery	TOF ratio > 0.9
B. Three levels of neuromuscular blockade [23]	
Clinical interpretation	**TOF count**
Over-paralyzed	TOF count = 0
Well-paralyzed	TOF count = 1-2
Under-paralyzed	TOF count = 3-4
C. Nerve stimulation sites and corresponding motor responses [20]	
Nerve stimulated	**Expected motor response**
Ulnar nerve	Contraction of adductor pollicis muscle
Facial nerve	Contraction of orbicularis oculi muscle
Posterior tibial nerve	Plantar flexion of the great toe
Peroneal nerve	Dorsiflexion of the foot

Abbreviations: PTC: Post-tetanic count; TOF: Train-of-four; TOF ratio (TOFR): ratio of the fourth to the first twitch response.

exceeds 0.4–0.6. Quantitative monitoring devices such as acceleromyography and electromyography provide improved sensitivity and reproducibility for assessing the depth of neuromuscular blockade and recovery [32].

Indirect neuromuscular monitoring

Indirect monitoring techniques do not directly measure neuromuscular transmission but infer the presence or absence of neuromuscular blockade from respiratory or ventilatory parameters. These methods are particularly useful when standard TOF monitoring is unavailable or unreliable.

End-tidal CO_2 monitoring and curare drift

End-tidal carbon dioxide ($EtCO_2$) monitoring can provide indirect evidence of respiratory muscle activity. The phenomenon known as the curare cleft refers to a transient downward deflection in the alveolar plateau of the capnogram during controlled mechanical ventilation, reflecting spontaneous diaphragmatic effort due to inadequate depth of neuromuscular blockade or partial recovery from paralysis [33] (Figure 2).

In patients receiving NMBAs, unexpected fluctuations in end-tidal carbon dioxide ($EtCO_2$) despite stable ventilator settings may suggest the presence of spontaneous respiratory effort, inadequate depth of neuromuscular blockade, or changes in ventilatory mechanics. Conversely, stable $EtCO_2$ values during controlled mechanical ventilation may be consistent with adequate paralysis; however, this finding is nonspecific and cannot reliably confirm the absence of diaphragmatic activity.

Ventilator waveform and mode analysis

Modern ventilators provide continuous information on patient–ventilator interaction. The appearance of assisted breaths instead of fully controlled breaths may indicate the return of spontaneous respiratory effort and incomplete neuromuscular blockade [34,35] (Figure 3).

Key ventilator indicators include triggering of breaths in controlled modes, negative deflection in airway pressure or flow preceding ventilator-delivered breaths, and changes in tidal volume or airway pressure that are inconsistent with preset parameters [36-38], although these findings should be interpreted cautiously because ventilator auto-triggering and circuit leaks may mimic true patient effort.

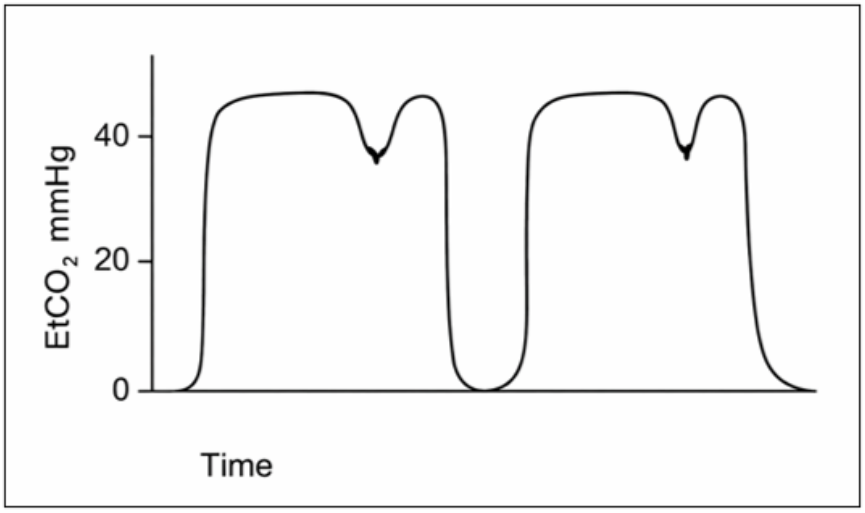


Figure 2. Capnogram demonstrating a curare cleft pattern. The downward deflection during the expiratory plateau represents spontaneous inspiratory effort occurring during the expiratory phase, indicating inadequate neuromuscular blockade.

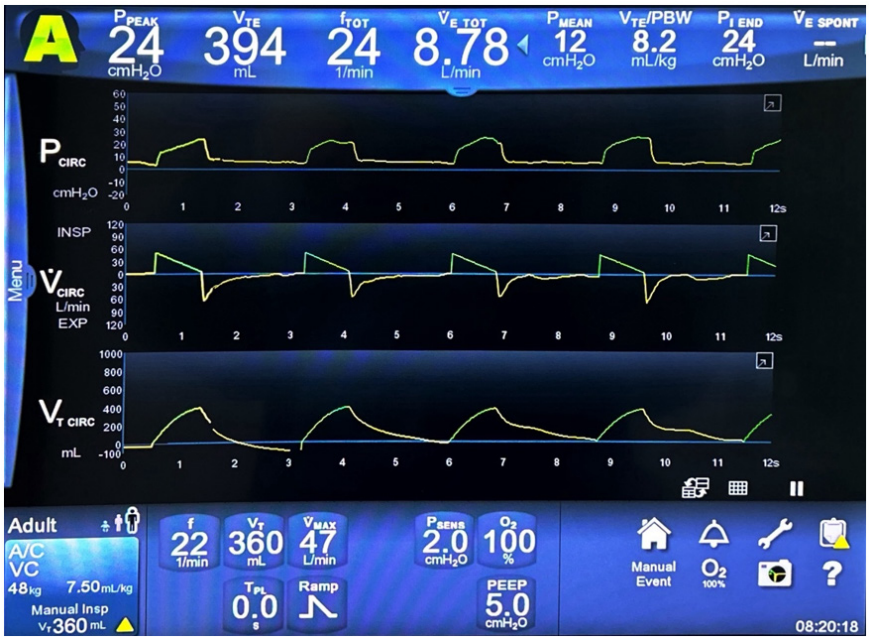


Figure 3. Ventilator waveforms demonstrating inadequate neuromuscular blockade. In assist-control ventilation mode, the set respiratory rate was 22 breaths/min; however, the observed total respiratory rate increased to 24 breaths/min due to patient-triggered breaths, suggesting inadequate depth of neuromuscular blockade with persistent spontaneous respiratory effort. RR: respiratory rate.

Airway occlusion pressure ($P_{0.1}$), Pocc, NIF, and Pmus

Ventilator-derived indices of respiratory drive and inspiratory muscle effort are increasingly utilized in the ICU. These include $P_{0.1}$ (airway occlusion pressure measured 100 ms after the onset of inspiration), which reflects central respiratory drive and inspiratory muscle activity [39]; Pocc (airway occlusion pressure measured during an end-expiratory occlusion), which provides an estimate of inspiratory effort [40]; negative inspiratory force (NIF), which assesses maximal inspiratory muscle strength [41]; and Pmus, a calculated measure of inspiratory muscle pressure derived from airway pressure and respiratory mechanics [42].

In the context of neuromuscular blockade, near-zero or absent values suggest profound suppression of respiratory muscle activity, whereas increasing values indicate recovery of diaphragmatic or accessory muscle activity. These parameters are particularly useful during partial paralysis or during weaning phases [5,43]. However, the reliability is significantly limited during deep neuromuscular blockade, particularly in the early management phase of severe ARDS. Under these conditions, ventilator trigger sensitivity, circuit artifacts, reverse triggering, or auto-triggering may generate misleading signals despite profound respiratory muscle paralysis [16,44]. Consequently, these parameters should not be used as the primary method for titration of NMBAs during deep blockade. Instead, they should be interpreted alongside established neuromuscular monitoring techniques, including Train-of-Four (TOF) and post-tetanic count (PTC) assessments [16,23].

Esophageal balloon monitoring

Esophageal balloon catheters allow direct measurement of esophageal pressure as a surrogate for pleural pressure, enabling accurate assessment of respiratory muscle effort and patient–ventilator interaction. In patients receiving NMBAs, esophageal pressure monitoring can confirm the absence of inspiratory effort and detect subtle recovery of diaphragmatic activity before it becomes apparent on ventilator waveforms. This technique is considered the most physiologically precise indirect method for evaluating neuromuscular blockade but requires expertise, appropriate equipment, and correct catheter positioning [45-47].

Advantages include high sensitivity for detecting respiratory effort and improved assessment of patient–ventilator synchrony. Limitations include invasiveness, technical complexity, and limited availability in routine ICU practice.

CLINICAL INTEGRATION AND PRACTICAL CONSIDERATIONS

No single monitoring modality is sufficient for all ICU patients. Optimal neuromuscular monitoring requires integration of quantitative train-of-four (TOF) assessment with indirect clinical, ventilatory, and physiologic parameters. A multimodal approach enhances the detection of inadequate neuromuscular blockade (under-paralysis) and excessive blockade (over-paralysis), thereby supporting individualized NMBAs titration. However, indirect monitoring methods primarily identify insufficient blockade and are limited in their ability to detect excessive neuromuscular paralysis.

In clinical practice, a stepwise monitoring strategy may be useful. Quantitative TOF monitoring should remain the primary method for assessing the depth of neuromuscular blockade [17,48]. When available, post-tetanic count (PTC) assessment may further differentiate deep from complete blockade in patients with absent TOF responses [48]. Additional bedside indicators, including curare clefts on capnography, spontaneous respiratory effort, and patient–ventilator asynchrony, may suggest inadequate paralysis or recovery of respiratory muscle activity [44]. Ventilator-derived indices such as P0.1, Pocc, NIF, and Pmus may provide complementary information during partial blockade or the weaning phase but should not replace standard neuromuscular monitoring techniques during deep paralysis [44,49].

Recent regional studies published in Thai journals have similarly emphasized the importance of objective neuromuscular monitoring. A study by Werawatganon et al. demonstrated good agreement between a locally developed peripheral nerve stimulator and the TOF-Watch® SX system, supporting the feasibility of quantitative TOF monitoring in routine clinical practice [50]. This locally developed device may promote and increase the utilization of neuromuscular blockade monitoring in Thailand. In addition, a recent study by Chanthawong et al. reported a high incidence of postoperative residual neuromuscular blockade, further highlighting the clinical importance of standardized neuromuscular monitoring strategies [51]. Both studies suggest that the use of neuromuscular monitoring in both ICU and operating room settings should be increased in Thailand.

Education of ICU staff, standardized protocols, and increased availability of quantitative neuromuscular monitors may improve patient outcomes and reduce NMBAs-related complications [52].

CONCLUSION

Neuromuscular monitoring in the ICU is an important yet frequently underutilized component of patient management during NMBAs therapy. Quantitative TOF monitoring remains the standard method for assessing depth of neuromuscular blockade, whereas indirect approaches including clinical assessment, capnography ($EtCO_2$ patterns), ventilator waveforms, respiratory drive indices, and esophageal pressure measurements may provide complementary physiologic information but cannot replace direct neuromuscular assessment. A multimodal approach that integrates quantitative monitoring with clinical and ventilatory parameters may enhance the safe and individualized use of NMBAs in critically ill patients.

CONFIDENTIALITY

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AUTHORS' CONTRIBUTIONS

S.A.: Concept, Literature search, Data synthesis, Manuscript writing, Critical revision; W.J.: Data synthesis, Manuscript writing, Critical revision, Supervision.

REFERENCES

1. Iavarone IG, Al-Husinat L, Vélez-Páez JL, Robba C, Silva PL, Rocco PRM, et al. Management of neuromuscular blocking agents in critically ill patients with lung diseases. JCM. 2024;13(4):1182. doi:10.3390/jcm13041182
2. Tezcan B, Turan S, Özgök A. Current use of neuromuscular blocking agents in intensive care units. Turk J Anaesthesiol Reanim. 2019;47(4):273-281. doi: 10.5152/TJAR.2019.33269
3. Rodríguez-Blanco J, Rodríguez-Yanez T, Rodríguez-Blanco JD, Almanza-Hurtado AJ, Martínez-Ávila MC, Borré-Naranjo D, et al. Neuromuscular blocking agents in the intensive care unit. J Int Med Res. 2022;50(9):03000605221128148. doi:10.1177/03000605221128148
4. Gainnier M, Roch A, Forel JM, Thirion X, Arnal JM, Donati S, et al. Effect of neuromuscular blocking agents on gas exchange in patients presenting with acute respiratory distress syndrome. Critical Care Medicine. 2004;32(1):113–9. doi:10.1097/01.CCM.0000104114.72614.BC
5. Papazian L, Forel JM, Gacouin A, Penot-Ragon C, Perrin G, Loundou A, et al. Neuromuscular Blockers in Early Acute Respiratory Distress Syndrome. N Engl J Med. 2010;363(12):1107–16. doi:10.1056/NEJMoa1005372
6. Guervilly C, Bisbal M, Forel JM, Mechati M, Lehingue S, Bourenne J, et al. Effects of neuromuscular blockers on transpulmonary pressures in moderate to severe acute respiratory distress syndrome. Intensive Care Med. 2017;43(3):408–18. doi:10.1007/s00134-016-4653-4
7. The National Heart, Lung, and Blood Institute PETAL Clinical Trials Network. Early neuromuscular blockade in the acute respiratory distress syndrome. N Engl J Med. 2019;380(21):1997–2008. doi:10.1056/NEJMoa1901686
8. Lin C, Chao WC, Pai KC, Yang TY, Wu CL, Chan MC. Prolonged use of neuromuscular blocking agents is associated with increased long-term mortality in mechanically ventilated medical ICU patients: a retrospective cohort study. J Intensive Care. 2023;11(1):55. doi:10.1186/s40560-023-00696-x
9. Bellaver P, Schaeffer AF, Leitao CB, Rech TH, Nedel WL. Association between neuromuscular blocking agents and the development of intensive care unit-acquired weakness (ICU-AW): A systematic review with meta-analysis and trial sequential analysis. Anaesthesia Critical Care & Pain Medicine. 2023;42(3):101202. doi:10.1016/j.accpm.2023.101202
10. Workum JD, Janssen SHV, Touw HRW. Considerations in neuromuscular blockade in the ICU: A case report and review of the literature. Case Reports in Critical Care. 2020;2020:1–4. doi:10.1155/2020/8780979
11. Schweickert WD, Hall J. ICU-acquired weakness. Chest. 2007;131(5):1541–9. doi:10.1378/chest.06-2065
12. Morales Castro D, Dresser L, Granton J, Fan E. Pharmacokinetic alterations associated with critical illness. Clin Pharmacokinet. 2023;62(2):209–20. doi:10.1007/s40262-023-01213-x
13. Zhou J, Poloyac SM. The effect of therapeutic hypothermia on drug metabolism and response: cellular mechanisms to organ function. Expert Opinion on Drug Metabolism & Toxicology. 2011;7(7):803–16. doi:10.1517/17425255.2011.574127
14. Ross J, Ramsay DP, Sutton-Smith LJ, Willink RD, Moore JE. Residual neuromuscular blockade in the ICU : a prospective observational study and national survey. Anaesthesia. 2022;77(9):991–8. doi:10.1111/anae.15789
15. Groetzinger LM, Hutchins AT, Rivosecchi RM. An evaluation of continuous infusion rocuronium for sustained neuromuscular blockade in critically ill adults. Ann Pharmacother. 2021;55(6):732–7. doi:10.1177/1060028020966731
16. Alhazzani W, Belley-Cote E, Møller MH, Angus DC, Papazian L, Arabi YM, et al. Neuromuscular blockade in patients with ARDS: a rapid practice guideline. Intensive Care Med. 2020;46(11):1977–86. doi:10.1007/s00134-020-06227-8
17. Naguib M, Brull SJ, Kopman AF, Hunter JM, Fülesdi B, Arkes HR, et al. Consensus statement on perioperative use of neuromuscular monitoring. Anesthesia & Analgesia. 2018;127(1):71–80. doi:10.1213/ANE.0000000000002670
18. Jones SK. An Algorithm for Train-of-Four Monitoring in Patients Receiving Continuous Neuromuscular Blocking Agents: Dimensions of Critical Care Nursing. 2003;22(2):50–7. doi:10.1097/00003465-200303000-00001
19. Murray MJ, DeBlock H, Erstad B, Gray A, Jacobi J, Jordan C, et al. Clinical practice guidelines for sustained neuromuscular blockade in the adult critically ill patient. Critical Care Medicine. 2016;44(11):2079–103. doi:10.1097/CCM.0000000000002027
20. Rowlee SC. Monitoring neuromuscular blockade in the intensive care unit: The peripheral nerve stimulator. Heart & Lung. 1999;28(5):352–64. doi:10.1053/hl.1999.v28.a99734
21. Warnaar RSP, Cornet AD, Beishuizen A, Moore CM, Donker DW, Oppersma E. Advanced waveform analysis of diaphragm surface EMG allows for continuous non-invasive assessment of respiratory effort in critically ill patients at different PEEP levels. Crit Care. 2024;28(1):195. doi:10.1186/s13054-024-04978-0
22. Radkowski P, Ruść J, Kęska M. Evaluation of Neuromuscular Blockade: A Comparative Study of TOF-Cuff® on the Lower Leg and TOF-Scan® on the Ulnar Nerve During Mivacurium Anesthesia. Med Sci Monit. 2024;30. doi:10.12659/MSM.945227
23. Bouju P, Tadié JM, Barbarot N, Letheulle J, Uhel F, Fillatre P, et al. Clinical assessment and train-of-four measurements in critically ill patients treated with recommended doses of cisatracurium or atracurium for neuromuscular blockade: a prospective descriptive study. Ann Intensive Care. 2017;7(1):10. doi:10.1186/s13613-017-0234-0
24. Donati F, Antzaka C, Bevan DR. Potency of pancuronium at the diaphragm and the adductor pollicis muscle in humans. Anesthesiology. 1986;65(1):1–5. doi:10.1097/00000542-198607000-00001
25. Cantineau JP, Porte F, d'Honneur G, Duvaldestin P. Neuromuscular effects of rocuronium on the diaphragm and adductor pollicis muscles in anesthetized patients. Anesthesiology. 1994;81(3):585–90. doi:10.1097/00000542-199409000-00010
26. Donati F, Meistelman C, Plaud B. Vecuronium neuromuscular blockade at the diaphragm, the orbicularis oculi, and adductor pollicis muscles. Anesthesiology. 1990;73(5):870–5. doi:10.1097/00000542-199011000-00013
27. Devlin JW, Skrobik Y, Gélinas C, Needham DM, Slooter AJC, Pandharipande PP, et al. Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. Critical Care Medicine. 2018;46(9):e825–73. doi:10.1097/CCM.0000000000003299
28. Rezaiguia-Delclaux S, Laverdure F, Genty T, Imbert A, Pilorge C, Amaru P, et al. Neuromuscular blockade monitoring in acute respiratory distress syndrome: randomized controlled trial of clinical assessment alone or with peripheral nerve stimulation. Anesthesia & Analgesia. 2021;132(4):1051–9. doi:10.1213/ANE.0000000000005174
29. Hraiech S, Forel JM, Guervilly C, Rambaud R, Lehingue S, Adda M, et al. How to reduce cisatracurium consumption in ARDS patients: the TOF-ARDS study. Ann Intensive Care. 2017;7(1):79. doi:10.1186/s13613-017-0305-2
30. Rudis MI, Sikora CA, Angus E, Peterson E, Popovich J, Hyzy R, et al. A prospective, randomized, controlled evaluation of peripheral nerve stimulation versus standard clinical dosing of neuromuscular blocking agents in critically ill patients. Critical Care Medicine. 1997;25(4):575–83. doi:10.1097/00003246-199704000-00005
31. Heier T, Caldwell JE, Eriksson LI, Sessler DI, Miller RD. The effect of hypothermia on adductor pollicis twitch tension during continuous infusion of vecuronium in isoflurane-anesthetized humans. Anesthesia & Analgesia. 1994;78(2):312–7. doi:10.1213/00000539-199402000-00019
32. Renew JR, Ratzlaff R, Hernandez-Torres V, Brull SJ, Prielipp RC. Neuromuscular blockade management in the critically Ill patient. J Intensive Care. 2020;8(1):37. doi:10.1186/s40560-020-00455-2
33. Ward KR, Yealy DM. End-tidal carbon dioxide monitoring in emergency medicine, Part 1: Basic Principles*. Academic Emergency Medicine. 1998;5(6):628–36. doi:10.1111/j.1553-2712.1998.tb02473.x
34. Emrath E. The Basics of ventilator waveforms. Curr Pediatr Rep. 2021;9(1):11–9. doi:10.1007/s40124-020-00235-4
35. Mojoli F, Pozzi M, Orlando A, Bianchi IM, Arisi E, Iotti GA, et al. Timing of inspiratory muscle activity detected from airway pressure and flow during pressure support ventilation: the waveform method. Crit Care. 2022;26(1):32. doi:10.1186/s13054-022-03895-4
36. De Oliveira B, Aljaberi N, Taha A, Abduljawad B, Hamed F, Rahman N, et al. Patient–ventilator dyssynchrony in critically ill patients. JCM. 2021;10(19):4550. doi:10.3390/jcm10194550
37. Holanda MA, Vasconcelos RDS, Ferreira JC, Pinheiro BV. Patient-ventilator asynchrony. J bras pneumol. 2018;44(4):321–33. doi:10.1590/s1806-37562017000000185
38. Flynn BC, Miranda HG, Mittel AM, Moitra VK. Stepwise ventilator waveform assessment to diagnose pulmonary pathophysiology. Anesthesiology. 2022;137(1):85–92. doi:10.1097/ALN.0000000000004220
39. Jonkman AH, De Vries HJ, Heunks LMA. Physiology of the respiratory drive in ICU patients: Implications for diagnosis and treatment. Crit Care. 2020;24(1):104. doi:10.1186/s13054-020-2776-z
40. Van Oosten JP, Akoumianaki E, Jonkman AH. Monitoring respiratory muscles effort during mechanical ventilation. Current Opinion in Critical Care. 2024. doi:10.1097/MCC.0000000000001229
41. Vu PH, Tran VD, Duong MC, Cong QT, Nguyen T. Predictive value of the negative inspiratory force index as a predictor of weaning success: a crosssectional study. Acute Crit Care. 2020;35(4):279–85. doi:10.4266/acc.2020.00598
42. Kondili E, Alexopoulou C, Xirouchaki N, Vaporidi K, Georgopoulos D. Estimation of inspiratory muscle pressure in critically ill patients. Intensive Care Med. 2010;36(4):648–55. doi:10.1007/s00134-010-1753-4
43. Akoumianaki E, Maggiore SM, Valenza F, Bellani G, Jubran A, Loring SH, et al. The application of esophageal pressure measurement in patients with respiratory failure. Am J Respir Crit Care Med. 2014;189(5):520–31. doi:10.1164/rccm.201312-2193CI

44. Clement KC. Ventilator triggering. J Pediatr intensive care. 2015 Jul 28;02(01):011–7. doi:10.3233/PIC-13043
45. Jonkman AH, Telias I, Spinelli E, Akoumianaki E, Piquilloud L. The oesophageal balloon for respiratory monitoring in ventilated patients: updated clinical review and practical aspects. Eur Respir Rev. 2023;32(168):220186. doi:10.1183/16000617.0186-2022
46. Pham T, Telias I, Beitler JR. Esophageal manometry. respiratory care. 2020;65(6):772–92. doi:10.4187/respcare.07425
47. Yoshida T, Brochard L. Esophageal pressure monitoring: why, when and how? Current Opinion in Critical Care. 2018;24(3):216–22. doi:10.1097/MCC.0000000000000494
48. Fuchs-Buder T, Claudius C, Skovgaard LT, Eriksson LI, Mirakhur RK, Viby-Mogensen J. Good clinical research practice in pharmacodynamic studies of neuromuscular blocking agents II: the Stockholm revision. Acta Anaesthesiol Scand. 2007;51(7):789–808. doi:10.1111/j.1399-6576.2007.01352.x
49. Telias I, Junhasavasdikul D, Rittayamai N, Piquilloud L, Chen L, Ferguson ND, et al. Airway occlusion pressure as an estimate of respiratory drive and inspiratory effort during assisted ventilation. american journal of respiratory and critical care medicine. 2020;201(9):1086–98. doi:10.1164/rccm.201907-1425OC
50. Werawatganon T, Desudchit T, Pirompanich K, Tailanan K, Hamta-nont P. Agreement of Train-of-Four count by Thai peripheral nerve stimulator compare with TOF-Watch® SX. Thai J Anesthesiol. 2023;49(3):243-250.
51. Chanthawong S, Apithambundit T, Chaimala N, Saras S, Yotepanya T, Nonphiaraj S. Incidence of postoperative residual neuromuscular blockade at the postanesthesia care unit following general anesthesia. Siriraj Med J. 2025;77(7):496–504. doi:10.33192/smj.v77i7.272875
52. Hadique S, Badami V, Forte M, Kovacic N, Umer A, Shigle A, et al. The Implementation of protocol-based utilization of neuromuscular blocking agent using clinical variables in acute respiratory distress syndrome patients. Critical Care Explorations. 2021;3(3):e0371. doi:10.1097/CCE.0000000000000371

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