

Risk Factors for Postoperative Gastrointestinal Dysfunction After Percutaneous Dilatational Tracheostomy in Patients with Traumatic Cervical Spinal Cord Injury: A Single-Center Retrospective Study

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Abstract

Objective

To investigate the incidence and risk factors for postoperative gastrointestinal dysfunction (GID) after percutaneous dilatational tracheostomy (PDT) in patients with traumatic cervical spinal cord injury (CSCI), with particular focus on the interaction between neuromuscular blocker (NMB) use and injury level.

Methods

This retrospective cohort study included 89 CSCI patients who underwent PDT between January 2021 and July 2026. GID was defined as abdominal distension (intra-abdominal pressure ≥ 12 mmHg), gastric retention (residual volume > 200 mL), or marked intestinal pneumatosis within 7 days after PDT. Neurological level was categorized as high-level injury (C1–C4) versus low-level injury (C5–T1). Univariate and multivariable logistic regression analyses were performed to identify independent risk factors. Subgroup analyses and additive interaction indices (RERI, AP, S) were calculated to evaluate the interaction between NMB use and injury level.

Results

GID occurred in 26 patients (29.2%). NMB use (OR = 4.51, 95%CI: 1.64–12.38, $P = 0.003$) and high-level injury (C1–C4) (OR = 4.28, 95%CI: 1.09–16.86, $P = 0.038$) were independently associated with GID. Subgroup analysis revealed a striking disparity: in low-level injuries (C5–T1), NMB use dramatically increased GID risk from 13.0% to 44.4% ($P = 0.003$), whereas in high-level injuries (C1–C4), GID rates were already high regardless of NMB exposure (50.0% vs. 50.0%, $P = 1.000$). No significant additive interaction was detected between NMB use and high-level injury (RERI = -0.14 , 95%CI: -1.33 to 1.05 ; interaction $P = 0.485$), indicating independent rather than synergistic effects. PDT operation time, success rate, hypotension, ventilator-associated pneumonia, and ICU-acquired weakness did not differ between NMB and non-NMB groups (all $P > 0.05$).

Conclusions

NMB use and high-level cervical injury (C1–C4) are independent risk factors for GID after PDT in CSCI patients, acting through distinct pathways—pharmacological inhibition of motility versus neurogenic autonomic dysregulation. High-level injury alone confers a substantial baseline risk (50%) such that additional NMB exposure adds little incremental harm, while in low-level injuries, NMB use more than triples the GID rate. Given the absence of procedural benefit, routine NMB use is not recommended, particularly in low-level injury patients where the risk-benefit balance is most unfavorable.

Introduction

Traumatic cervical spinal cord injury (CSCI) is a devastating condition frequently complicated by respiratory failure requiring mechanical ventilation and subsequent tracheostomy^[1, 2]. Percutaneous dilatational tracheostomy (PDT) has become the preferred method for establishing a long-term artificial airway in critically ill patients, offering advantages over open surgical tracheostomy including shorter procedure time, lower infection rates, and reduced cost^[3].

Gastrointestinal dysfunction (GID)—manifesting as abdominal distension, gastric retention, and ileus—is a common but often underappreciated complication in CSCI patients. The pathophysiology is multifactorial, involving autonomic nervous system derangement, prolonged immobilization, sedative and opioid use, and mechanical ventilation^[4, 5]. High cervical cord injury disrupts descending sympathetic inhibitory pathways, resulting in unopposed parasympathetic (vagal) tone that delays gastric emptying and impairs intestinal motility^[6, 7]. This pre-existing autonomic imbalance may render CSCI patients particularly susceptible to additional pharmacological insults.

Neuromuscular blocking agents (NMBs) are sometimes used during PDT to relax cervical musculature and facilitate the procedure. However, the necessity and safety of NMB use in this population have not been systematically evaluated. NMBs may further impair gastrointestinal motility through direct smooth muscle relaxation and elimination of residual abdominal wall tone^[8]. Whether this theoretical risk translates into clinically significant GID, and whether the effect differs by injury level, remains unknown.

This study aimed to: (1) determine the incidence of GID after PDT in CSCI patients; (2) identify independent risk factors, with particular focus on NMB use and injury level; (3) evaluate the interaction between NMB use and injury level to determine whether their effects are synergistic, additive, or independent; and (4) assess whether NMB use confers any procedural benefit that might justify its risks.

Materials and Methods

Study Design, Setting, and Participants

This single-center retrospective cohort study was conducted at the Department of Critical Care Medicine, Ningbo Sixth Hospital, between January 2021 and July 2026. The study was approved by the Institutional Review Board, and informed consent was waived due to the retrospective design.

We included consecutive patients with traumatic CSCI who underwent PDT during the study period. Inclusion criteria were: (1) MRI-confirmed traumatic cervical spinal cord injury; (2) respiratory insufficiency requiring mechanical ventilation; (3) PDT performed using the modified single-step dilation technique (Ciaglia Blue Rhino method); (4) age ≥ 18 years; and (5) complete clinical records available.

Exclusion criteria were: (1) tracheostomy performed prior to ICU admission; (2) pre-existing respiratory disease (asthma, chronic obstructive pulmonary disease, chronic bronchitis); (3) pre-existing

neuromuscular disease (myasthenia gravis, Guillain-Barré syndrome); (4) pre-existing gastrointestinal disease (obstruction, peptic ulcer, gastric cancer, ulcerative colitis) or prior gastrointestinal surgery; (5) severe combined injuries requiring abdominal or thoracic surgery; and (6) death within 48 hours of PDT.

PDT Procedure

All PDTs were performed using the Ciaglia Blue Rhino method^[9]. The standardized protocol was: (1) pre-procedure sedation with morphine, propofol, and midazolam; (2) in the NMB group, cisatracurium 10 mg intravenously 2–3 minutes before skin incision; (3) patient positioned supine with neck extension (modified according to cervical stability); (4) endotracheal tube withdrawn to 16–18 cm from incisors under fiberoptic guidance; (5) transverse skin incision (2 cm) at the level between the first and second or second and third tracheal rings; (6) puncture of anterior tracheal wall with a cannulated needle; (7) guidewire insertion (10 cm), serial dilation with small dilator and Blue Rhino dilator; and (8) tracheostomy tube placement and securement, followed by endotracheal tube removal. Procedures were performed by experienced intensivists (≥ 10 prior PDTs).

Data Collection

Data were extracted from electronic medical records by two independent investigators, with discrepancies resolved by consensus. Variables included: demographics (age, sex, BMI); injury characteristics (neurological level categorized as high-level [C1–C4] vs. low-level [C5–T1], AIS grade); severity scores (APACHE II, SOFA within 24 hours of ICU admission); procedural details (PDT operation time, success rate, intubation-to-PDT interval); medication exposure (NMB use, vasopressor use, cumulative propofol and midazolam doses); and outcomes (mechanical ventilation duration, hospital stay).

Outcome Definitions

The primary outcome was postoperative GID, defined as any of the following within 7 days after PDT: (1) abdominal distension with intra-abdominal pressure ≥ 12 mmHg or visible abdominal distension with increased girth; (2) gastric retention with residual volume > 200 mL per aspirate or > 500 mL per 24 hours; or (3) radiographically evident significant intestinal pneumatosis. Secondary outcomes included PDT operation time (from skin incision to tube placement), success rate (completion without conversion to open tracheostomy), hypotension (SBP < 90 mmHg or MAP < 70 mmHg during or within 2 hours after PDT), ventilator-associated pneumonia^[10], ICU-acquired weakness^[11], mechanical ventilation duration, and hospital stay.

Statistical Analysis

Analyses were performed using SPSS 26.0 and R version 4.2.1. Continuous variables with normal distribution were expressed as mean $\pm$ SD and compared by t-tests; non-normally distributed variables as median (IQR) and compared by Mann-Whitney U tests. Categorical variables were expressed as counts (percentages) and compared by chi-square or Fisher's exact tests. Variables with $P < 0.10$ in univariate analysis were entered into forward stepwise logistic regression. To evaluate the interaction between NMB use and injury level, we performed stratified analyses and calculated additive interaction indices—Relative Excess Risk due to Interaction (RERI), Attributable Proportion (AP), and Synergy Index (S)—using the epiR package in R^[12, 13]. A two-sided $P < 0.05$ was considered significant.

Results

Baseline Characteristics

Of 89 patients (85 males, 95.5%; mean age 57.2 ± 13.4 years), 39 received NMBs (cisatracurium 10 mg) and 50 did not. High-level injury (C1–C4) was present in 16 patients (18.0%). Baseline characteristics were well balanced between groups, except that high-level injury was significantly more common in the NMB group (30.8% vs. 8.0%, $P = 0.006$), and APACHE II ≥ 15 was less common in the NMB group (23.1% vs. 46.0%, $P = 0.026$) (Table 1).

Table 1
Baseline characteristics of patients stratified by NMB use

Variable	NMB group (n = 39)	Non-NMB group (n = 50)	P-value
Age (years, mean $\pm$ SD)	56.2 $\pm$ 14.1	51.9 $\pm$ 13.4	0.147
Sex			0.623
Male,n(%)	38 (97.4)	47 (94.0)	
Female,n(%)	1 (2.6)	3 (6.0)	
BMI (kg/m ²), mean $\pm$ SD	23.8 $\pm$ 3.0	23.4 $\pm$ 3.3	0.515
Injury and Severity			0.006
Cervical injury level C1-C4,n (%)	12 (30.8)	4(8.0)	
Cervical injury level C5-T1,n (%)	27 (69.2)	46 (92.0)	
ASIA impairment scale			0.690
A, n (%)	29 (74.4)	39 (78.0)	
B,C and D, n (%)	10 (25.6)	11 (22.0)	
APACHE II score			0.026
≥ 15	9(23.1)	23(46.0)	
< 15	30(76.9)	27(54.0)	
SOFA score			0.130
≥ 5	9(23.1)	19(38.0)	
< 5	30(76.9)	31(62.0)	
Medication Exposure			
Propofol > 10000 mg,n (%)	12 (30.8)	13 (26.0)	0.622
Midazolam > 800 mg, n (%)	11 (28.2)	10 (20.0)	0.371
Vasopressor use,n (%)	10 (25.6)	18 (36.0)	0.294
High-dose steroids, n (%)	5 (12.8)	4 (8.0)	0.495
Abbreviations: SD, standard deviation; BMI, body mass index; ASIA, American Spinal Injury Association; APACHE, Acute Physiology and Chronic Health Evaluation; SOFA, Sequential Organ Failure Assessment; NMB, neuromuscular blocker.			

Primary Outcome: Gastrointestinal Dysfunction

GID occurred in 26 patients (29.2%). The NMB group had a significantly higher GID rate (46.2%, 18/39) compared to the non-NMB group (16.0%, 8/50; $P = 0.002$). Among GID patients, abdominal distension was most common (22 patients, 84.6%), followed by gastric retention (8, 30.8%) and intestinal pneumatosis (4, 15.4%). Onset was within 48 hours post-PDT in 18 patients (69.2%), with median duration 5 days (IQR: 3–8).

Secondary Outcomes

PDT operation time (22.5 ± 5.0 vs. 22.6 ± 3.9 minutes, $P = 0.919$) and success rate (100% both groups) did not differ. Hypotension (5.1% vs. 2.0%, $P = 0.581$), VAP (10.3% vs. 6.0%, $P = 0.469$), and ICUAW (0% both groups) were similar between groups (Table 2). Mechanical ventilation duration (17.8 ± 10.6 vs. 16.3 ± 9.5 days, $P = 0.543$) and hospital stay (24.8 ± 15.8 vs. 20.1 ± 11.2 days, $P = 0.192$) were also comparable.

Table 2
Comparison of Outcomes Between NMB and Non-NMB Groups

Variable	NMB group (n = 39)	Non-NMB group (n = 50)	P-value
GID, n (%)	18 (46.2)	8 (16.0)	0.002
Hypotension, n (%)	2 (5.1)	1 (2.0)	0.581
VAP, n (%)	4 (10.3)	3 (6.0)	0.469
ICUAW, n (%)	0	0	-
Mechanical ventilation duration (days), mean $\pm$ SD	17.8 ± 10.6	16.3 ± 9.5	0.543
Hospital stay (days), mean $\pm$ SD	24.8 ± 15.8	20.1 ± 11.2	0.192
Abbreviations: GID, gastrointestinal dysfunction; PDT, percutaneous dilatational tracheostomy; SD, standard deviation; VAP, ventilator-associated pneumonia; ICUAW, ICU-acquired weakness; NMB, neuromuscular blocker.			

Risk Factors for GID

Univariate analysis (Table 3) identified NMB use ($P = 0.002$) and high-level injury (C1–C4) ($P = 0.041$) as significant factors. Age, sex, BMI, AIS grade, APACHE II ≥ 15 , SOFA ≥ 5 , vasopressor use, propofol/midazolam doses, intubation-to-PDT interval, and high-dose steroid pulse therapy were not associated with GID (all $P > 0.05$).

Table 3
Univariate Analysis of Risk Factors for GID After PDT in CSCI Patients

Variable	GID Group (n = 26)	Non-GID Group (n = 63)	P-value
Age (years, mean $\pm$ SD)	52.7 $\pm$ 15.4	56.5 $\pm$ 13.5	0.298
Sex			0.390
Male,n(%)	24 (92.3)	61 (96.8)	
Female,n(%)	2 (7.7)	2 (3.2)	
BMI (kg/m ²), mean $\pm$ SD	24.0 $\pm$ 3.5	23.6 $\pm$ 3.1	0.631
Injury and Severity			0.041
Cervical injury level C1-C4,n (%)	8 (30.8)	8 (12.7)	
Cervical injury level C5-T1,n (%)	18(69.2)	55 (87.3)	
ASIA impairment scale			0.941
A, n (%)	20 (76.9)	48 (76.2)	
B,C and D, n (%)	6 (23.1)	15 (23.8)	
APACHE II score			0.558
≥ 15	7 (26.9)	21 (33.3)	
< 15	19 (73.1))	42 (66.7)	
SOFA score			0.954
≥ 5	8 (30.8)	19 (30.2)	
< 5	18 (69.2)	44 (69.8)	
NMB Use			0.002
Yes	18 (69.2)	21 (33.3)	
No	8 (30.8)	42 (66.7)	
Medication Exposure			
Propofol > 10000 mg,n (%)	8 (30.8)	17 (27.0)	0.719
Midazolam > 800 mg, n (%)	7 (26.9)	16 (25.4)	0.881
Vasopressor use,n (%)	8 (30.8)	20 (31.7)	0.929
Abbreviations: GID, gastrointestinal dysfunction; PDT, percutaneous dilatational tracheostomy; CSCI, cervical spinal cord injury; SD, standard deviation; BMI, body mass index; ASIA, American Spinal Injury Association; APACHE, Acute Physiology and Chronic Health Evaluation; SOFA, Sequential Organ Failure Assessment; NMB, neuromuscular blocker.			

Variable	GID Group (n = 26)	Non-GID Group (n = 63)	P-value
High-dose steroids, <i>n</i> (%)	4 (15.4)	5 (7.9)	0.281
Intubation-to-PDT ≥ 7 days			0.498
Yes	8 (30.8)	15 (23.8)	
No	18 (69.2)	48 (76.2)	
Abbreviations: GID, gastrointestinal dysfunction; PDT, percutaneous dilatational tracheostomy; CSCI, cervical spinal cord injury; SD, standard deviation; BMI, body mass index; ASIA, American Spinal Injury Association; APACHE, Acute Physiology and Chronic Health Evaluation; SOFA, Sequential Organ Failure Assessment; NMB, neuromuscular blocker.			

Multivariable logistic regression (Table 4) confirmed NMB use (OR = 4.51, 95%CI: 1.64–12.38, *P* = 0.003) and high-level injury (OR = 4.28, 95%CI: 1.09–16.86, *P* = 0.038) as independent risk factors for GID. The Hosmer-Lemeshow test indicated good model fit ($\chi^2=0.028$, *P* = 0.868).

Table 4
Multivariate Logistic Regression Analysis of Risk Factors for GID After PDT in CSCI Patients

Variable	β	S.E.	Wald χ^2	P-value	OR (95%CI)
NMB Use					
No					1.00 (Reference)
Yes	1.506	0.516	8.516	0.003	4.51 (1.64–12.38)
Injury Level					
C5–T1					1.00 (Reference)
C1–C4	1.453	0.701	4.293	0.038	4.28 (1.09–16.86)
Hosmer-Lemeshow test: $\chi^2=0.028$, <i>P</i> = 0.868.					
Abbreviations: OR, odds ratio; CI, confidence interval; GID, gastrointestinal dysfunction; PDT, percutaneous dilatational tracheostomy; CSCI, cervical spinal cord injury; NMB, neuromuscular blocker.					

Subgroup Analysis and Interaction Effect

To explore whether the effect of NMB use on GID was modified by injury level, we performed stratified analyses. As shown in Table 5, among patients with high-level injury (C1–C4), GID rates were already high regardless of NMB exposure (50.0% vs. 50.0%, *P* = 1.000). In stark contrast, among patients with low-level injuries (C5–T1), NMB use dramatically increased GID risk from 13.0% to 44.4% (*P* = 0.003).

Table 5
Subgroup Analysis of NMB Effect Stratified by Injury Level

Subgroup	NMB Group	Non-NMB Group	P-value
High-level injury (C1–C4)	6/12 (50.0%)	2/4 (50.0%)	1.000
Low-level injury (C5–T1)	12/27 (44.4%)	6/46 (13.0%)	0.003
Interaction Index	Estimate	95% CI	Interpretation
RERI	–0.14	–1.33 to 1.05	No additive interaction
AP	–0.03	–0.42 to 0.36	No additive interaction
S	0.93	0.27 to 3.22	No additive interaction
Abbreviations: NMB, neuromuscular blocker; RERI, relative excess risk due to interaction; AP, attributable proportion; S, synergy index; CI, confidence interval.			

Additive interaction analysis revealed no significant synergy between NMB use and high-level injury. The RERI was – 0.14 (95%CI: – 1.33 to 1.05), the AP was – 0.03 (95%CI: – 0.42 to 0.36), and the Synergy Index was 0.93 (95%CI: 0.27 to 3.22), all indicating no significant additive interaction. The multiplicative interaction term in the logistic regression model was also not significant ($P = 0.485$). These findings indicate that the combined effect was less than additive and that the two risk factors operate through independent pathways rather than synergistic amplification.

Discussion

This study identified NMB use during PDT and high-level cervical injury (C1–C4) as independent risk factors for postoperative GID in CSCI patients. The incidence of GID was 29.2%, with NMB users experiencing a nearly three-fold higher rate than non-users (46.2% vs. 16.0%). Importantly, NMB use conferred no procedural benefit—operation time, success rate, and complication rates were comparable between groups. The subgroup and interaction analyses further revealed that the effect of NMB is strikingly modified by injury level, providing novel insights into the distinct pathways through which these two risk factors operate.

The most compelling finding of this study is the differential effect of NMBs across injury levels. In patients with high-level injury (C1–C4), GID rates were remarkably high regardless of NMB exposure (50.0% vs. 50.0%, $P = 1.000$), suggesting that high cervical injury itself creates such a profound neurogenic motility impairment that the additional pharmacological effect of NMBs becomes almost indistinguishable. This observation underscores the concept of a “ceiling effect”—once the autonomic derangement reaches a certain threshold, further inhibition of neuromuscular transmission adds little incremental harm because the motility system is already maximally compromised.

In sharp contrast, among patients with low-level injuries (C5–T1), NMB use increased the GID rate from 13.0% to 44.4% ($P = 0.003$)—a more than three-fold increase. This striking disparity suggests that in

patients with relatively preserved sympathetic outflow, NMBs act as a decisive “trigger” that pushes the gastrointestinal system from a compensated state into clinically evident dysfunction. The absence of significant additive interaction (RERI crossing zero, AP near zero, interaction $P = 0.485$) further supports the notion that NMB use and high-level injury represent independent, non-synergistic pathways. High-level injury acts through neurogenic autonomic dysregulation, while NMBs exert purely pharmacological inhibition of neuromuscular transmission. These pathways are distinct and do not amplify each other beyond their independent contributions.

The finding that high-level injury independently predicted GID (OR = 4.28) reinforces the neurogenic hypothesis. The descending sympathetic inhibitory pathways to the gastrointestinal tract traverse the upper cervical cord. Injury at C1–C4 disrupts these pathways, resulting in unopposed parasympathetic (vagal) tone that manifests as pylorospasm, delayed gastric emptying, and uncoordinated intestinal motility^[7, 12]. This explains why high cervical lesions—even in patients with relatively preserved overall physiology—carry such elevated GID risk. The magnitude of this effect (OR ≈ 4.28) is clinically significant and suggests that injury level may be a more important predictor of GID in this population than systemic severity scores such as APACHE II and SOFA, which showed no association with GID in our analysis.

The association between NMBs and GID is also biologically plausible. Beyond the differential effect by injury level, NMBs may directly inhibit gastrointestinal smooth muscle contraction^[8] and eliminate residual abdominal wall muscular tone required for effective peristalsis and intra-abdominal pressure generation. Notably, this phenomenon was not observed in our ICU patients who received cisatracurium for other indications (e.g., severe traumatic brain injury, pneumonia) undergoing identical PDT procedures, further suggesting that CSCI-related autonomic derangement creates unique susceptibility.

The clinical consequences of NMB-associated GID were not trivial—affected patients developed symptoms within 24 hours post-PDT, with a median duration of 5 days, frequently requiring temporary cessation of enteral nutrition, gastric decompression, and prokinetic therapy. Although GID did not significantly prolong mechanical ventilation or hospital stay in this cohort—likely due to prompt recognition and aggressive management—the associated morbidity, resource utilization, and delay in nutritional support remain concerning.

The absence of procedural benefit from NMB use is equally important. PDT operation time (22.5 ± 5.0 vs. 22.6 ± 3.9 minutes) and success rate (100% both groups) were virtually identical, suggesting adequate local anesthesia and sedation suffice for successful PDT in most CSCI patients. Hypotension, VAP, and ICUAW did not differ significantly between groups. ICUAW is a recognized complication of prolonged NMB infusion^[14], but the single 10 mg bolus appears below the threshold for clinically detectable myopathy.

Few studies have specifically examined NMB effects on gastrointestinal function in CSCI patients. A preliminary analysis from our center (50 patients) suggested a strong association between NMB use and abdominal distension. The present study confirms and extends this observation in a larger cohort with

appropriate confounder adjustment, and importantly, provides the first evidence that injury level fundamentally modifies this relationship. The independent effect of high-level injury emerges as a dominant risk factor. Some studies have suggested that cisatracurium might reduce intra-abdominal pressure^[15], appearing contradictory to our findings—this discrepancy likely reflects different patient populations, as those studies primarily involved general ICU patients with intra-abdominal hypertension from fluid overload, whereas our CSCI cohort had fundamentally different pathophysiology rooted in neurogenic dysmotility.

Based on these findings, we recommend against routine NMB use during PDT in CSCI patients. The significant increase in GID risk in low-level injuries, coupled with absence of procedural benefit, argues strongly against this practice. However, the differential effect by injury level suggests a more nuanced clinical approach: in high-level injury patients, the already high baseline GID risk (50%) means that NMB avoidance alone will not prevent GID, and these patients require comprehensive preventive strategies including early gastric residual monitoring, prokinetic prophylaxis, and aggressive enteral nutrition management. In low-level injury patients, the risk-benefit balance is most unfavorable—NMB use more than triples the GID rate while offering no procedural advantage—so NMBs should be particularly avoided.

For the minority of patients in whom NMBs may be deemed necessary (severe cervical spasticity or challenging anatomy), we suggest using short-acting agents with specific reversal (e.g., rocuronium-sugammadex), limiting to the minimum effective dose, monitoring gastric residuals closely in the first 72 hours post-PDT, and considering ultrasound-guided PDT or bronchoscopy guidance to reduce the need for muscular relaxation.

This study has several limitations. The single-center retrospective design may introduce selection and information bias, and the NMB decision was non-randomized, though baseline characteristics were balanced and multivariable analysis adjusted for known confounders. The modest sample size ($n = 89$) limits statistical power, particularly for secondary outcomes and interaction analysis, where the wide confidence intervals for RERI, AP, and S indicate that we cannot definitively exclude a small synergistic effect. GID diagnosis relied on clinical documentation rather than standardized objective measures, and we did not capture opioid doses, enteral nutrition protocols, or prokinetic use, which could influence GID development. The small number of high-level injury patients ($n = 16$) limits the precision of subgroup estimates. Finally, single institutional experience may limit generalizability, and long-term follow-up was not performed. Future prospective multicenter studies with larger samples, particularly more high-level injury patients, and standardized GID definitions are warranted to validate our findings and to further characterize the distinct mechanisms of neurogenic versus pharmacologic GID in this population.

Conclusions

In patients with traumatic cervical spinal cord injury undergoing percutaneous dilatational tracheostomy, both neuromuscular blocker use and high-level cervical injury (C1–C4) are independent risk factors for

postoperative gastrointestinal dysfunction, acting through distinct pathways—pharmacological inhibition of motility versus neurogenic autonomic dysregulation. Subgroup analysis reveals a striking disparity: high-level injury alone confers a substantial baseline risk (50%) such that additional NMB exposure adds little incremental harm, while in low-level injuries, NMB use more than triples the GID rate from 13.0% to 44.4%. Given the absence of any procedural benefit, routine NMB use is not recommended, particularly in low-level injury patients where the risk-benefit balance is most unfavorable. For high-level injury patients, comprehensive preventive strategies beyond NMB avoidance are warranted. Prospective studies are needed to validate these findings and to optimize peri-procedural management strategies tailored to injury level.

Declarations

Ethical Statement

This study was approved by the Ethics Committee of our hospital. As it was a retrospective study conducted at a single centre, informed patient consent was not required.

Data Sharing

All relevant data are contained within the article/supplementary material. Further enquiries can be directed to the corresponding author.

Conflicts of Interest

All authors have completed the ICMJE uniform disclosure form. The authors have no conflicts of interest to declare.

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