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Effects of Trigger Sensitivity Adjustment as Translational Mechanisms on Weaning Outcomes in Ventilator Induced Diaphragmatic Dysfunction of Critically Ill Patients

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Abstract:

Objectives: This study aimed to assess the efficiency of trigger sensitivity adjustment in patients with ventilator-induced diaphragmatic dysfunction (VIDD).

Patients and methods: This was a prospective observational study based on data collected by observation of 60 MV patients with planned extubation in the Critical Care Department in Qasr Al-Ainy. (1) Vital signs: heart rate, respiratory rate, blood pressure, and oxygen saturation (So2), (2) Arterial blood gases (ABG): PH, PCO2 mmHg, HCO3 mEQ/Liter, PO2 mmHg, (3) Ventilator parameters: minute ventilation and tidal volume were measured pre- and after the sessions.

Result: The mean ± SD of all outcomes (PH, Pco2, Hco3, Sao2, minute ventilation [MV], tidal volume [TV], respiratory rate [RR], heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP]) pre- and post-intervention in both groups were presented in table 2. There were significant increases in Sao2 (Cohen d effect size =1.5) and TV (d=1.06), and significant decreases in Pco2 (d=2.5), MV (d=0.65), RR (d=1.43), HR (d=2.29), SBP (d=1.43), and DBP (d=2.04) within the sample group (p-value <0.05), whereas there were significant increases in Sao2 (d=1.94) and significant decreases in Pco2 (d=1.85), Hco3 (d=0.85), HR (d=1.67), SBP (d=1.76), and DBP (d=2.69) within the controlled group (p-value <0.05). **Conclusion:** We concluded that trigger sensitivity adjustment could be used as a translational mechanism for weaning from MV in ventilator-induced diaphragm dysfunction critically ill patients.

Keywords: Trigger Sensitivity, Ventilator-Induced Diaphragm Dysfunction, weaning from Mechanical ventilation, Vital signs, Ventilator Parameters, minute Ventilation, and tidal volume.

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1. Introduction:

The diaphragm, a primary respiratory muscle, is responsible for generating 70% of the tidal volume during normal respiration. Beyond its respiratory function, the diaphragm plays a vital role in various physiological activities, including coughing, expectorating, vomiting, swallowing, and excretory processes. Additionally, it facilitates blood and lymph flow while influencing visceral actions through pressure changes in the abdominal and thoracic cavities (4).

In intensive care units (ICUs), mechanical ventilation (MV) serves as a crucial life-support measure to ensure sufficient oxygenation and alveolar gas exchange in critically ill patients unable to breathe effectively due to severe illness or respiratory dysfunction. The invasive nature of MV carries the risk of substantial iatrogenic effects. Even short-term MV can lead to ventilator-induced diaphragm dysfunction (VIDD), a condition characterized by diaphragmatic weakness, potentially complicating ventilator weaning and affecting survival rates (24). VIDD's influence on clinical practice may be substantial, given its potential to deteriorate patient outcomes and increase healthcare resource demands. Current research reveals that VIDD primarily manifests as a significant reduction in diaphragmatic contractility coupled with muscle fiber atrophy (17).

In 2008, Levine, S et al. 2008 (11), revealed that VIDD manifests in critically ill patients through substantial wasting of slow and fast-twitch diaphragm fibers, coupled with oxidative damage and increased protein degradation. Diaphragm muscle fiber mechanics are defined by the interplay of free calcium in the cytosol, attachment and cycling rate of the cross-bridges, and the length of sarcomeres (10). Any change in these critical parameters can lead to variations in diaphragmatic muscle force generation. However, the etiology of MV-induced diaphragm contractile impairment appears to be multifaceted, involving oxidative damage to contractile proteins, reduced calcium sensitivity, protease-mediated sarcomere damage, and myosin heavy chain depletion (20).

Recent insights into the molecular mechanisms underlying VIDD in critically ill individuals reveal complex pathways (21). Diaphragm atrophy results from diverse conditions disrupting the balance between protein breakdown and synthesis, while oxidative stress and dephosphorylation compromise remaining muscle proteins. These detrimental changes are primarily attributed to inflammatory processes and oxidative stress (8). The hallmark of VIDD is a disturbance in protein balance, characterized by diminished synthesis and increased degradation of diaphragm proteins. MV rapidly triggers catabolic processes in the diaphragm, particularly under controlled ventilation modes, resulting in accelerated protein breakdown and subsequent atrophy (7).

Research observations indicate that diaphragm wasting is more severe in patients necessitating intensive ventilatory support, particularly mandatory and support modes (14). Within 24 hours of initiating continuous mandatory ventilation (CMV), the diaphragm undergoes rapid structural and functional changes, coupled with oxidative injury, leading to muscle atrophy and diminished strength (3). Research on organ donors revealed significant diaphragm thinning occurring as early as 18-69 hours following the initiation of mandatory ventilation. Patient prognosis deteriorates with VIDD, as it extends MV time, raises the probability of unsuccessful extubation, and ultimately increases mortality risk (18).

Diaphragmatic ultrasound serves as a diagnostic tool for evaluating diaphragm performance across various respiratory disorders like asthma, cystic fibrosis, chronic obstructive pulmonary disease, interstitial lung disease, and diaphragmatic paralysis. It outperforms alternative methods due to its non-invasive nature, absence of ionizing radiation, feasibility, reproducibility, repeatability, cost-effectiveness, and high interobserver consistency (17). It demonstrates excellent diagnostic accuracy for diaphragmatic neuromuscular disorders, with 93% sensitivity and 100% specificity. The potential of diaphragm ultrasound is expected to grow, offering innovative approaches to disease diagnosis, intervention tracking, and rehabilitation assessment in the upcoming years. In mechanically ventilated patients, diaphragm ultrasound emerges as a valuable tool for assessing diaphragmatic health, with progressive thinning potentially indicating atrophy (13).

Early rehabilitation in ICUs has been shown to reduce hospital stays, enhance patient outcomes, and facilitate discharge. Extended MV is linked to higher costs, poorer prognosis, and decreased quality of life post-ventilation and post-discharge. It also increases complication and mortality risks. Thus, critical care physical therapists should prioritize the prevention and treatment of VIDD in early rehabilitation programs (2).

Mechanical ventilation (MV) weaning is a gradual process of patient-ventilator separation. This critical phase requires precise timing; premature extubation risks re-intubation and nosocomial pneumonia, while delayed weaning exposes patients to prolonged ventilation risks. Careful planning and patient preparation are essential. Studies show successful weaning correlates with maximal inspiratory pressure (MIP) values of -30 cmH2O or lower, while values above -20 cmH2O often indicate unsuccessful weaning (6).

Parameters for weaning encompass lung oxygenation capacity, respiratory muscle strength and endurance, respiratory drive, breathing work, and

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integrative indices (15). The rapid shallow breathing index (RSBI), combining respiratory rate and tidal volume (F/Vt), offers enhanced predictive power. Maximum inspiratory pressure (MIP) or negative inspiratory force (NIF), ranging from -50 to 0 cm H2O, is a key predictor of extubation success, reflecting the crucial role of inspiratory muscles, particularly the diaphragm, in ventilatory work (16).

2 Materials and Methods:

2.1. Study Design and Participants

The study is a prospective observational study on adult patients who were admitted to the Critical Care Department in Qasr Al-Ainy. This study is conducted prospectively to assess the efficiency of trigger sensitivity adjustment on patients with VIDD. We used the Echo to assess diaphragm thickening and excursion. The RSBI and NIF, also known as MIP (less than 30 cm H_2O), are assessed by the mechanical ventilator and observing the weaning outcome. Firstly, we increased the flow trigger gradually by 30% of the initial reading of the NIF for 5 minutes in the 1st session. Then, we increased the time for each session to reach 30 minutes. Afterward, the flow trigger sensitivity is increased to 40% of the initial reading of NIF for 5 minutes, and finally, the time of the session is increased till reaching also 30 minutes. Re-assessment is performed after one week.

Our study was performed on sixty patients with VIDD from both genders. All patients were referred to by a physician. All patients were randomly assigned into two groups equal in number as follows:

Group (A) (study group) included 30 patients who practiced flow trigger sensitivity adjustment on MV for 1 week twice daily in addition to their plan of treatment. Trigger sensitivity refers to the level of effort or inspiratory effort required from the patient to initiate a breath from the ventilator. It is an important setting that determines how easily the ventilator responds to the patient's respiratory efforts. The trigger sensitivity setting on a ventilator determines how sensitive the ventilator is to the patient's respiratory efforts; higher trigger sensitivity means that the ventilator will respond to even small changes in flow or pressure, making it easier for the patient to trigger a breath. On the other hand, lower trigger sensitivity requires the patient to generate a larger effort before the ventilator initiates a breath. Adjusting the trigger sensitivity is important to ensure that the ventilator is synchronized with the patient's breathing pattern. If the trigger sensitivity is set too high, the ventilator may deliver breaths when the patient is not trying to breathe, leading to patient-ventilator asynchrony. Conversely, if the trigger sensitivity is set too low, the patient may have difficulty triggering a breath, leading to increased work of breathing and discomfort. Optimizing trigger sensitivity based on the patient's condition and respiratory effort is important to provide effective and comfortable mechanical ventilation. It is typically adjusted by healthcare providers based on the patient's individual needs and response to ventilation.

Group (B) (controlled group) included 30 patients who received their own routine plan of weaning of the mechanical ventilator (controlled mode, then spontaneous mode, and finally spontaneous breathing trial).

Patients were invited to participate in the research, with the therapist providing comprehensive information about risks, benefits, voluntary participation, and procedures. Both patients and their families were given sufficient time to consider the details, ask questions, and provide informed and voluntary consent. The rights and confidentiality of the patients were strictly upheld.

2.2. Procedures:

Methods of assessment:

All patients were subjected to the following:

1- Detailed history taking.

2- Full physical examination: A detailed clinical examination was done for all patients, including assessment of the vital signs [blood pressure (BP), heart rate (HR), respiratory rate (RR), and oxygen saturation (So2)] for each session.

3- Laboratory investigations:

Arterial blood gases (ABG): [PH, PCO2 mmHg, HCO3 mEQ/Liter, and PO2 mmHg]

4- Ventilator Parameters:

a) Minute Ventilation (MV)

- Is the volume of gas inhaled or exhaled from a person’s lungs per minute.
- Minute Ventilation = Tidal Volume * Respiratory Rate.

b) Tidal Volume (TV)

- Is the air volume inhaled during a normal, quiet breath.

Treatment Procedures:

All patients were investigated by routine investigation (vital signs, arterial blood gas, ventilator mode, and the parameters). Two sessions were implemented per day for the study group. The trial of trigger sensitivity adjustment begins with the application of a load of 30% of the first recorded NIF. In the first session, trigger sensitivity adjustment was set for a maximum of 5 minutes. This duration was then incrementally increased by 5 minutes per session until reaching 30 minutes. If a

patient tolerated 30 minutes, the following session would increase the NIF by 10% of the initial value, resulting in a total increase of 40%. Once we reached 40%, the training recurred starting from 5 minutes and increased every session by 5 minutes till reaching 30 minutes. Patients who were unable to tolerate a 30% NIF load for 5 minutes were trained with a 20% NIF load instead (1).

Days	Sessions	%of NIF	Time
1st Day	1st	30%	5 min.
	2nd	30%	10 min.
2nd Day	1st	30%	15 min
	2nd	30%	20 min.
3rd Day	1st	30%	25 min.
	2nd	30%	30 min.
4th Day	1st	40%	5 min.
	2nd	40%	10 min.
5th Day	1st	40%	15 min
	2nd	40%	20 min.
6th Day	1st	40%	25 min.
	2nd	40%	30 min.
7th Day	1st	50%	5 min.
	2nd	50%	10 min.

The training trial was terminated if any of the following criteria were met:

(1) Respiratory distress (RR exceeding 35 breaths/min or paradoxical breathing).
(2) Desaturation, defined as SpO2 < 90%, or significant changes in HR (> 140 beats/min or > 20% increase from baseline) or SBP (> 180 mmHg or < 90 mmHg).
(3) The presence of alarming signs such as diaphoresis, irritability, depression, arrhythmia, convulsions, or sweating, at which point the trial was stopped immediately.

2.3. Statistical analysis:

Statistical analysis was conducted using SPSS for Windows, version 24 (SPSS, Inc., Chicago, IL). Data were normally distributed using the Shapiro-Wilk test (*p*-value > 0.05) and descriptive measures with no extreme scores. Data were homogenous regarding variances using Levene's test (*p*-value > 0.05) except for some variables (PH pre, Sao_2 pre, Hco3 post, MV pre, HR pre) in which the corrected values were reported. Comparison between mean values of the different variables at pre- and post-intervention between the two groups was performed using an independent t-test. Analysis of covariance (ANCOVA) was used for between-group differences post-intervention when there were significant differences pre-intervention (e.g. PH, Pco2, and HR). ANCOVA was sued as there were only two times (pre and post) and controlling for the pre-test limits the comparison to only one estimate: post-test differences. In addition, independent-t test is not valid when pre-test differences (between groups) are significant unless correction is used (e.g. Bonferroni), therefore, ANCOVA was used instead. Moreover, a dependent t-test was used to determine the significant differences within both groups between pre- and post-intervention. Furthermore, differences between groups in categorical baseline data were analyzed using the Chi-square test. *P-value* was set at < 0.05. Descriptive statistics were expressed as mean ± SD for the continuous demographic data of the participants and dependent variables, whereas categorical data were expressed as frequency (percentage). Chi-Square test was used for testing the differences in distribution of gender, causes, and other medical problems between groups.

3. Results:

Descriptive statistics of the patients in the two groups:

Baseline characteristics of all patients in both groups were presented in **Table 1.** There were non-significant differences between both groups in all these baseline data ($p > 0.05$) except in other medical problems ($p = 0.01$), as shown in **Table 1.** Presence or absence of "other medical problems) did not seem to affect the outcomes as there were no significant correlations between "other medical problems) and all outcomes (post-treatment) ($p>0.05$).

Within-group comparisons:

The mean ± SD of all outcomes (PH, Pco_2, Hco_3, Sao_2, minute ventilation [MV], tidal volume [TV], respiratory rate [RR], heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP]) pre- and post-intervention in both groups were presented in **table 2.** There were significant increases in Sao_2 (Cohen *d* effect size=1.5) and TV (*d*=1.06), and significant decreases in Pco_2 (*d*=2.5), MV (*d*=0.65), RR (*d*=1.43), HR (*d*=2.29), SBP (*d*=1.43), and DBP (*d*=2.04) within the sample group (*p*-value<0.05), whereas there were significant increase in Sao_2 (*d*=1.94) and significant decreases in Pco_2 (*d*=1.85), Hco_3 (*d*=0.85), HR (*d*=1.67), SBP (*d*=1.76), and DBP (*d*=2.69) within the controlled group (*p*-value<0.05). **(Table 2)**

Table 1: Descriptive statistics for the demographic variables in both groups

Variable	Mean ± SD		t value	*P*-value
	Sample group N = 30	Controlled group N = 30		
Age (years)	60.97 ± 15.94	65.53 ± 11.43	-1.28	0.21
Weight (kg)	74.4 ± 6.6	74.77 ± 5.61	-0.23	0.82
Height (cm)	160.23 ± 5.94	160.47 ± 5.74	-0.16	0.88
BMI (kg/m^2)	29.1 ± 3.4	29.2 ± 3.5	-0.13	0.9
Sex distribution[b]				
Male	19 (63.3)	17 (56.7)	0.28[d]	0.6
Female	11 (36.7)	13 (43.3)		
Causes of mechanical ventilation[b]				
Pulmonary problems	17	23	3.5[c]	0.32
Post-operative complications	6	2		
Cardiac problems	2	2		
Others	5	3		
Other medical problems[b]				
Yes	21	11	6.7[d]	0.01*
No	9	19		

SD= Standard deviation, P =probability, *=significant, [b]: data were presented as frequency (percentage), [c]: Likelihood ratio, [d]: Chi-square test

Between-group comparisons:

There were non-significant differences between groups at pre-intervention (p>0.05) except Sao2 (p=0.03). Furthermore, there were only significant differences (p<0.05) between groups at post-intervention-after controlling for the significance at pre-intervention in PH, Pco_2, and HR, which were significantly higher in the controlled group (**Table 2**).

Discussion:

The study is a prospective observational study (clinical trials.gov identifier: NCT06515600), based on data collected by observation of 60 mechanically ventilated patients who were admitted to the Critical Care Department in Qasr Al-Ainy. This study is conducted prospectively to assess the efficiency of trigger sensitivity adjustment on patients with VIDD. The NIF, which measures the patient's maximum inspiratory effort after exhaling, also known as MIP (less than 30 cm H2O), is assessed by the mechanical ventilator and observing the weaning outcome. The RSBI combines respiratory rate and tidal volume (F/Vt), enhancing predictive power. NIF, also known as MIP, is clinically crucial due to the diaphragm's primary role in ventilation. Successful weaning involves extubation with no respiratory distress for 48 hours, while reintubation within this period indicates unsuccessful weaning (12).

The results of thesis showed within-group comparisons: The mean ± SD of all outcomes (PH, Pco2, Hco3, Sao2, minute ventilation [MV], tidal volume [TV], respiratory rate [RR], heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP]) pre- and post-intervention in both groups were presented in **table 2**. There were significant increases in Sao2 (Cohen d effect size=1.5) and TV (d=1.06), and significant decreases in Pco2 (d=2.5), MV (d=0.65), RR (d=1.43), HR (d=2.29), SBP (d=1.43), and DBP (d=2.04) within the sample group (p-value <0.05), whereas there were a significant increase in Sao2 (d=1.94) and significant decreases in Pco2 (d=1.85), Hco3 (d=0.85), HR (d=1.67), SBP (d=1.76), and DBP (d=2.69) within the controlled group (p-value <0.05). (**Table2**). Between-group comparisons: There were non-significant differences between groups at pre-intervention (p>0.05) except Sao2 (p=0.03). Furthermore, there were only significant differences (p<0.05) between groups at post-intervention after controlling for the significance at pre-intervention in PH, Pco_2, and HR, which were significantly higher in the controlled group (**Table2**).

These results coincide with Ahmed M. Ewis 2018. (1) They conducted research on 40 elderly ICU patients, intubated and mechanically ventilated for 48+ hours, and employed progressive inspiratory muscle training (IMT). Starting at 30% MIP, the load increased by 10% daily. Two 5-minute sessions were conducted daily throughout weaning. Results showed significantly increased MIP in the experimental group (mean difference 7.6 cmH2O, 95% CI: 5.8-9.4) and shorter weaning time (mean difference 1.7 days, 95% CI: 0.4-3.0) compared to controls (26).

Similarly, El Naggar et al. (2021) (13) referenced earlier studies showing daily 6-7.5% decreases in end-expiratory diaphragmatic thickness during MV, with diaphragmatic atrophy correlating to ventilator support levels. Their study found a 6% thickness decrease in patients without IMT. Conversely, IMT patients showed an 8.6% increase in end-inspiratory diaphragmatic thickness (Tdi-exp) after three days, suggesting IMT's effectiveness in

preventing diaphragm thinning. The study's limitation was not accounting for PEEP effects on expiratory diaphragm thickness during ultrasound measurements.

Similarly, Tassaux et al. (2005) (22), examined how varying flow thresholds for cycling off affected patient-ventilator interaction. Ten patients with obstructive lung disease on pressure support ventilation were assessed at 10, 25, 50, and 70% of peak inspiratory flow thresholds. Raising thresholds from 10 to 70% minimized expiratory valve opening delays without causing premature opening. Higher thresholds also reduced dynamic hyperinflation and enhanced patient-ventilator synchrony, as evidenced by decreased triggering delays and fewer ineffective efforts.

Table 2: Within and between-group comparisons for all outcomes.

Variables	Time	Sample ± SD $\overline{X}$	Controlled ± SD $\overline{X}$	t-value (*p*-value)
PH	**Pre-intervention**	7.38 ±0.12	7.42 ±0.09	-1.61 (0.11)
	Post-intervention	7.39± 0.033	7.42 ±0.041	-2.56 (0.01) *
Effect size (Cohen *d*)		0.17	0.04	
t-value (*p*-value)		-0.93 (0.36)	0.22 (0.83)	
Pco_2	**Pre-intervention**	49.53 ±6.96	50.83 ±7.68	-0.69 (0.5)
	Post-intervention	40.63 ±5.04	46.6 ±7.13	-3.74 (<0.001)
Effect size (Cohen *d*)		2.5	1.85	
t-value (*p*-value)		13.8 (<0.001) *	10.15 (<0.001) *	
Hco_3	**Pre-intervention**	25.83 ±4.6	27.57± 4.85	-1.42 (0.16)
	Post-intervention	25.13 ±5.42	24.93 ±3.4	0.17 (0.87)
Effect size (Cohen *d*)		0.24	0.85	
t-value (*p*-value)		1.3 (0.2)	4.67 (<0.001) *	
Sao_2, %	**Pre-intervention**	95.83 ±1.56	94.63 ±2.51	2.23 (0.03) *
	Post-intervention	98.13±1.53	97.47 2.1	0.06[a] (0.81)
Effect size (Cohen *d*)		1.5	1.94	
t-value (*p*-value)		-8.21 (<0.001) *	-10.6 (<0.001) *	
MV	**Pre-intervention**	9.66 ±1.76	9.5 ±2.72	0.27 (0.79)
	Post-intervention	8.75 ±1.68	9.45± 2.2	-1.39 (0.17)
Effect size (Cohen *d*)		0.65	0.03	
t-value (*p*-value)		3.5 (0.001) *	0.18 (0.86)	
TV	**Pre-intervention**	423.4 ±87.49	442.23 ±110.91	-0.73 (0.47)
	Post-intervention	478.83± 86.47	439.40± 91.03	1.72 (0.09)
Effect size (Cohen *d*)		1.06	0.07	
t-value (*p*-value)		-5.8 (<0.001) *	0.38 (0.71)	
RR	**Pre-intervention**	23.9 ±6.8	22.1± 6.52	1.05 (0.3)
	Post-intervention	18.97 ±4.93	22.03 ±5.73	-2.22 (0.03)
Effect size (Cohen *d*)		1.43	0.02	
t-value (*p*-value)		7.8 (<0.001) *	0.11 (0.91)	
HR	**Pre-intervention**	94.37 ±4.46	95.7 ±2.8	-1.39 (0.17)
	Post-intervention	82.33 ±5.31	87.17 ±5.9	-3.34 (0.001) *
Effect size (Cohen *d*)		2.29	1.67	
t-value (*p*-value)		12.6 (<0.001) *	9.16 (<0.001) *	
SBP	**Pre-intervention**	137.33 ±9.07	138.17 ±11.63	-0.31 (0.76)
	Post-intervention	122.07 ±14.3	126.38 ±13.55	-1.31 (0.19)
Effect size (Cohen *d*)		1.43	1.76	
t-value (*p*-value)		7.67 (<0.001) *	9.66 (<0.001) *	
DBP	**Pre-intervention**	86.83 ±5.94	86.33±5.24	0.35 (0.73)
	Post-intervention	72.33 ±9.07	72.67±8.17	-0.15 (0.88)
Effect size (Cohen *d*)		2.04	2.69	
t-value (*p*-value)		11.16 (<0.001) *	14.75 (<0.001) *	

$\overline{X}$: Mean, SD: Standard Deviation, *: Significant, a: ANCOVA, p: probability, MV: Minute ventilation, TV: Tidal volume, RR: Respiratory rate, HH: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure

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On the other hand, Newth et al. (2009) (16), noted that while NIF measurement should indicate respiratory muscle function, its variability and lack of standardization limit its reliability. However, consistently low NIF values (above -15 cmH2O) likely predict weaning failure. Studies correlating thickness fraction (TF) with diaphragm effort suggest that maintaining a 15-30% TF may provide a safe threshold for effort titration during MV.

Goligher et al., 2018 (7) suggested that end-expiratory diaphragmatic thickness reflects atrophy more than weakness, noting that thickness reduction correlates with diminished inspiratory effort and heightened risks of complications and weaning difficulties.

Venkataraman et al. (2002) (23) highlighted NIF measurement as the most clinically relevant respiratory muscle assessment, given the diaphragm's primary role in ventilation. In PICUs, true maximal NIF requires inspiration from the residual volume, rarely achievable in intubated patients. NIF values of at least -30 cmH2O predict extubation success in children and adults, either standalone or within the CROP index. However, PICU measurements often use uncalibrated manometers and obstruct both inspiration and exhalation, compromising validity.

Gupta et al. (2013) (9), identified post-extubation respiratory rate and oxygen saturation as indicators of extubation failure, they evaluated NIV's effectiveness in averting extubation failure in pediatric cardiac patients. Predictors of NIV success encompassed lower risk-adjusted congenital heart surgery scores, preserved left ventricular function, normal respiratory rate, and adequate oxygen saturation.

Despite promising integrated weaning indices, no single index has proven ideal. Before Yang and Tobin's 1991 (24) introduction of the Rapid Shallow Breathing Index (RSBI), clinicians relied on vital capacity, MIP, and minute ventilation. RSBI, the ratio of respiratory rate to tidal volume, with a threshold value of >105 breaths/min/L, is strongly associated with weaning failure, whereas RSBI <105 breaths/min/L is linked to weaning success. Later studies, including Frutos-Vivar et al. (2006) (5), have validated the use of RSBI in predicting extubation failure, along with other factors such as positive fluid balance and pneumonia. Minor asynchronies (cycling delay, early cycling and trigger delay) are more frequent than and predispose to major ones and require higher expertise to be detected at the bedside (25).

Schwake et al. (2011) (19) demonstrated that 73 of 74 patients successfully underwent NIV, with blood gas improvements noted within 1-2 hours in successful cases. Consistent with the findings of Aziz et al. (2019) (15), it was determined that in both the RSBI and NIF groups, decreased oxygen saturation post-extubation was significantly associated with re-intubation (p-value <0.001 for RSBI, 0.003 for NIF). Additionally, in the RSBI group, blood gas measurements showed that re-intubated patients had a significantly lower pH (p-value 0.013) and a significantly higher PaCO2 (p-value 0.003). For the NIF group, similar findings were observed with lower pH (p-value 0.004) and higher PaCO2 (p-value 0.01) in those needing re-intubation. The respiratory rate post-extubation was also significantly higher in re-intubated patients in both groups (p-value 0.006 for RSBI and <0.001 for NIF). Furthermore, the study highlighted that hypocalcemia in the RSBI group was a significant factor contributing to re-intubation (p-value 0.034), underscoring the importance of respiratory rate, oxygen desaturation, blood gases, and hypocalcemia in predicting extubation outcomes.

Conclusion:

We concluded that both groups weaned on either pre- or post-intervention. There were significant increases in Sao2 and TV, and significant decreases in Pco2, MV, RR, HR, SBP, and DBP within the sample group (p-value <0.05), whereas there were significant increases in Sao2 and significant decreases in Pco2, Hco3, HR, SBP, and DBP within the controlled group (p-value <0.05). Between-group comparisons: There were non-significant differences between groups at pre-intervention (p>0.05) except Sao2 (p=0.03). Furthermore, there were significant differences (p<0.05) between groups at post-intervention-after controlling for the significance at pre-intervention in PH, Pco2, and HR. So, these results recommended that trigger sensitivity adjustment could be used as a translational mechanism for weaning from MV in VIDD critically ill patients.

Conflict of interest:

The corresponding author, on behalf of all authors, declares that there are no conflicts of interest related to this work.

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