CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187
http://doi.org/10.5281/zenodo.4430138

Avalable online at: http://www.iajps.com *Research Article*

MECHANICAL IMPACT OF AIRWAY HUMIDIFICATION DEVICES IN DIFFICULT TO WEAN PATIENTS

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Article Received: November 2020 **Accepted:** December 2020 **Published:** January 2021

Abstract:

Aim: *To evaluate the impact of airway humidification devices on the effectiveness of ventilation in difficult to wean patients.*

Study Design: *This prospective, randomized, controlled study was conducted in a 22-bed intensive care unit at the Central ICU in collaboration with the Pharmacology department of Nishtar Hospital Multan for six-months duration from July 2020 to December 2020.*

Interventions: *22-bed intensive care unit Patients with chronic respiratory failure were included. The effects of a heated humidifier and heat and moisture exchanger on diaphragm muscle activity, breathing pattern, gas exchange, and breathing comfort were assessed when disconnected from mechanical ventilation using supportive ventilation. Eleven patients with chronic respiratory failure received four consecutive pressure support ventilations at two different levels of pressure support ventilation (7 and 15 cm H2O) using a heated humidifier and a heat and moisture exchanger.*

Results: *Compared to the heated humidifier and regardless of the level of pressure assisted ventilation applied, the heat and moisture exchanger significantly increased all the inspiratory effort variables (inspiratory work of the breath expressed in J / L and J / min, pressure-time product, esophageal pressure and changes diaphragm pressure; p <0.05) and dynamic internal positive end-expiratory pressure (p <0.05). Similarly, the heat-moisture exchanger caused a significant increase in PaCO2 (p <0.01) responsible for severe respiratory acidosis (p <0.05), which was poorly compensated for despite a significant increase in minute ventilation (p <0.05). 05). This resulted in respiratory failure in all patients with heat and moisture exchangers (p <0.01). The negative effects were partially mitigated by increasing the level of pressure assisted ventilation by> 8 cm H2O using a heat-moisture exchanger.*

Conclusions: *The type of respiratory humidifier used may adversely affect the mechanical efficiency of ventilation and the use of a heat exchanger should not be recommended, unless the level of pressure support ventilation increases significantly. and moisture in patients requiring or potentially difficult to wean. chronic respiratory failure. (Crit Care Med 2003; 31: 1306-1311)*

Keywords: *heat and moisture exchanger; heated humidifier; respiratory work; pressure assisted ventilation; weaning*

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Please cite this article in press Tayyaba Roshni et al, ***Mechanical Impact Of Airway Humidification Devices In Difficult To Wean Patients.,*** *Indo Am. J. P. Sci, 2021; 08(1).*

INTRODUCTION:

Two devices are currently available for clinicians to heat and humidify inspired gases during mechanical intratracheal ventilation (MV): Heated Humidifiers (HH) and Heat Moisture Exchangers (HME). Various studies comparing the wetting, heating and inhibition of bacterial colonization capacity between the two systems. However, its effect on ventilation efficiency is less known. Airway humidifiers (AHD) behave differently due to their design and location in the ventilator circuit. HMEs are placed between the endotracheal tube and the Y portion of the circuit, while HHs are placed in the inspiratory circuit. Therefore, both systems can increase the circuit resistance to the flow of blown gases, but the effect appears to be relatively limited. In particular, unlike the HH, the HME can increase instrumental dead space depending on the model and reduce alveolar ventilation for a given level of minute ventilation (VE). Therefore, this HME effect can impair ventilation efficiency, especially during Spontaneous Breathing (SB) modes. One study also suggested that an increase in VV due to extra dead space could cause inspiratory work of respiratory overload (Wi) in 15 weaned patients. However, this study did not measure Wi, and no study has measured inspiratory effort with different AHD in difficult to wean patients. Therefore, the aim of this study was to evaluate the effect of HME on diaphragmatic activity, breathing pattern, gas exchange and breathing comfort during weaning from MV with support ventilation compared to HH. pressure (PSV) in chronic respiratory failure (CRF).

MATERIALS AND METHODS:

This prospective, randomized, controlled study was conducted in a 22-bed intensive care unit at the Central ICU in collaboration with the Pharmacology department of Nishtar Hospital Multan for six-months duration from July 2020 to December 2020. Patients were selected who were ventilated for acute, extreme chronic respiratory failure and were considered difficult to wean after meeting simple weaning criteria: 150 mm Hg PaO2 / FIO2 ratio, positive external end expiratory pressure (PEEP) 5 cm HO2, tidal volume respiratory ratio 105 beats • min 1 • L 1, no sedatives or vasoactive substances. However, they failed the SB weaning test, which lasted up to 2 hours using a T-piece or HH technique with a PSV level of 7 cm H2O (Table 1). Chronic respiratory failure was based on a prior medical history, chest X-ray, and / or lung function tests, and written informed consent was obtained for inclusion. Non-cooperative patients and those who had undergone gastrointestinal surgery or intestinal obstruction who had recent contraindications for the insertion of an esophageal tube were not included. Patients were examined within 24 hours of BS discontinuation failure. They were subjected to four PSV cross-over sequences lasting 20 minutes and assessed for HH and HME at two different PSV levels, 7 cm H2O (HH-7 / HME-7) and 15 cm H2O (HH-15 / HME-15). The order of administration of the AHD and PSV sequences was randomized for each patient and separated by 20-minute MV baseline periods. Patients were not clearly informed about the change in AHD or the PSV level used. The booster mode settings are the settings used for each patient before the test. During the clinical follow-up and testing period, respiration and heart rate, blood pressure, and arterial oxygen saturation were continuously monitored by percutaneous oximetry. The study was discontinued in the event of clinical intolerance (agitation, sweating, respiratory rate of 35 breaths / min, asynchronous thoracoabdominal pain), altered consciousness or agitation. All patients were ventilated using the NPB 840 ventilator (Mallinckrodt, Carlsbad, CA) and tested in a semi-sitting position by the bed. The respiratory variables recorded at the end of each PSV sequence were: esophageal pressure change (Pes), gastric pressure, diaphragmatic pressure (Pdi, intragastric pressure Pes) and change (Pdi), product of pressure over time, Wi J / L and J / min, Pdi 'Dynamic internal PEEP, breathing pattern, gas exchange and breathing comfort measured from. Prior to the examination, a radial artery catheter and a double esophageal balloon catheter were inserted and checked. The calculated signal analysis was performed with the Resdiag software. Variables recorded over 30 seconds were measured or calculated on average from five to ten respiratory cycles. Wi was calculated using the Pdi tidal volume curve according to a Campbell diagram, integrating the area drawn between the pressure-volume curves (Pditidal volume) and the relaxation curve of the chest wall. The product of pressure against time was obtained from the product of the inspiratory time and the area under the Pdi curve corresponding to the inspiratory time. Breathing comfort was assessed using a 100-millimeter visual analog scale, in which patients rated their feelings from 0 as "very uncomfortable" to 100 as "very comfortable" (15). The primary endpoint of the study was to compare the diaphragm muscle variables (Wi, pressure time product, Pdi, Pes) between HME and HH according to the PSV level used. For secondary endpoints, the other variables tested under these conditions were compared using the two AHDs. The results between the two different sequences were compared using the Wilcoxon test for paired runs and expressed as mean standard deviation. A statistically significant difference was assumed for $P < 0.05$.

RESULTS:

Fifteen additional patients were studied, but four patients had to discontinue early due to clinical intolerance to HME (n = 3) or refusal to continue the study (n = 1). The clinical features of the 11 evaluable patients are summarized in Table 1.

Table 1. Clinical characteristics of patients on admission and at study entry

Patient	Gender	Age, yrs	Type of CRF	SAPS II	Cause of Intubation	MV Time, days	Pao_2/Fio_2, mmHg	SB Trial Failure
1	M	51	COPD	53	Exacerb.	2	240	T tube
2	M	62	Mixed	40	Superinf.	7	157	T tube
3	M	68	COPD	41	Exacerb.	2	198	PSV 7
4	M	73	COPD	45	Exacerb.	5	285	T tube
5	F	75	Mixed	44	Exacerb.	2	190	PSV 7
6	M	62	Mixed	27	Superinf.	3	184	PSV 7
7	M	73	COPD	42	Shock	7	156	T tube
8	M	68	COPD	46	Superinf.	12	182	T tube
9	M	75	COPD	31	Superinf.	4	236	T tube
10	F	78	Mixed	72	Shock	6	370	T tube
11	F	69	COPD	40	Exacerb.	4	203	T tube

Patients were examined on average after 5 days of MV. The effect of two AHDs on diaphragm function depending on the PSV level used is shown in Figure 1 and Table 2. Regardless of AHD, functional variables of the respiratory muscles increased as opposed to a decrease in PSV. used (Table 2).

Table 2. Effects of the two humidification devices on respiratory muscle function variables according to pressure support level

	HH-7	HME-7	HH-15	HME-15	*p*
Wi, J/L	1.35 ± 0.80	1.71 ± 0.97[a]	0.93 ± 0.84	1.40 ± 89[a]	NS
Wi, J/min[b]	14.44 ± 11.03	20.19 ± 15.21[c]	9.86 ± 10.23	16.50 ± 14.98[a]	NS
PTPdi, cm H_2O-sec/min	9.20 ± 4.52	12.40 ± 5.59[c]	7.48 ± 4.39	9.16 ± 5.10[c]	NS
ΔPdi, cm H_2O	14.18 ± 7.06	19.45 ± 9.16[a]	9.86 ± 6.52	13.64 ± 8.19[a]	NS
ΔPes, cm H_2O	18.57 ± 9.41	22.38 ± 11.14[c]	12.82 ± 8.20	19.88 ± 11.48[a]	NS

The inspiratory effort for a given PSV level was significantly higher for the HME compared to the HH (Wi, J / min, pressure product over time, Pdi or Pes in J / L) regardless of the variable used or how it was expressed. These indicators did not differ significantly when comparing HH-7 with HME15. The effects of HME and HH on the mechanical and respiratory pattern are shown in Table 3.

Table 3. Effects of the two humidification devices on breathing pattern according to pressure support level

	HH-7	HME-7	HH-15	HME-15	*p*
Vte, mL	468 ± 143	457 ± 143	516 ± 126	516 ± 150	NS
f, beats/min	28 ± 7	29 ± 7	26 ± 7	28 ± 7	NS
$\dot{V}_E$, L/min	12.4 ± 3.5	13.4 ± 3.5[a]	12.9 ± 2.9	13.9 ± 3.6[a]	.005
Ti, secs	0.89 ± 0.36	0.84 ± 0.29	0.95 ± 0.32	0.92 ± 0.38	NS
Te, secs	1.52 ± 0.76	1.39 ± 0.52	1.56 ± 0.62	1.43 ± 0.63	NS
Ti/Ttot	0.38 ± 0.04	0.38 ± 0.04	0.39 ± 0.05	0.39 ± 0.04	NS
Vt/Ti, L/sec	0.54 ± 0.13	0.58 ± 0.14	0.55 ± 0.10	0.60 ± 0.15	NS
Paw, cm H_2O[b]	2.8 ± 2.9	1.5 ± 3.6[a]	8.4 ± 4.1	7.2 ± 4.0[a]	.007
PEEPi, cm H_2O	4.5 ± 3.1	6.5 ± 3.8[a]	4.9 ± 3.3	5.6 ± 4.2	.032

Regardless of the PSV level used, VE increased significantly with HME versus HH ($p < 0.05$) and this difference remained significant. HH-7 and HME-15 ($p < 0.01$). The increase in VE appears to be related to the increase in respiratory rate in HME, but this was not significant, but tidal volume remained similar for the two AHDs. HME also significantly increased dynamic intrinsic PEEP compared to HH for a given PSV level ($p < 0.05$). Although this increased the PSV level to 15 cm H2O with the use of HME compared to the use of HH-7, an increase in intrinsic

PEEP was also found ($p < 0.05$; Table 3). It was found that the mean airway pressure was significantly lower with HME than with HH for both PSV levels used ($p < 0.05$). Oxygenation variables did not change depending on the AHD and PSV levels used (Table 4).

Table 4. Effects of the two humidification devices on gas exchange according to pressure support

	HH-7	HME-7	HH-15	HME-15	*p*
Pa_{O_2}, kPa	12.7 ± 1.4	13.5 ± 8.3	14.9 ± 3.3	12.9 ± 2.4	NS
Pa_{O_2}/Fi_{O_2}, mm Hg	223 ± 36	235 ± 127	266 ± 70	229 ± 62	NS
Sa_{O_2}, %	97 ± 1	96 ± 1	97 ± 1	95 ± 5	NS
pH	7.35 ± 0.05	7.30 ± 0.09[a]	7.38 ± 0.06	7.29 ± 0.09[b]	.020
Pa_{CO_2}, kPa	7.0 ± 1.5	8.5 ± 2.7[b]	6.5 ± 1.7	8.4 ± 2.5[b]	.025
HCO_3^-, mmol/L	28.1 ± 3.6	28.9 ± 4.4	27.2 ± 4.2	28.3 ± 3.3	NS
Fet_{CO_2}, %	5.7 ± 1.2	6.9 ± 1.7[b]	5.3 ± 1.4	6.5 ± 1.8[b]	.016

In contrast, regardless of PSV levels, HME induced respiratory acidosis with increases in PaCO2 and FetCO2 and a much more severe decrease in pH than HH (Table 4). HME also caused significant respiratory failure compared to HH in all patients using both PSV levels ($p < 0.01$; Fig. 2). This discomfort was also observed when we compared HME-15 with HH-7 ($p < 0.01$).

DISCUSSION:

The study shows that discontinuation of MV with PSV may be significantly influenced by the type of AHD used in difficult-to-wean CKD patients. Regardless of the PSV level used (7 or 15 cm H2O) and compared to HH, HME produces Wi overload with an increase in dynamic spontaneous PEEP, resulting in impaired gas exchange with respiratory acidosis. It is not compensated by the increase of V˙E. These physiological effects of HME cause respiratory distress in patients. Our study also shows that an 8 cm H2O increase in PSV is required to compensate for the increase in inspiratory effort produced by the HME. HME has been increasingly used in intensive care units for almost 10 years to provide humidification through heating and to prevent ventilation-related pneumonia. However, only a few studies have looked at the possible effects of ventilation on mechanical efficacy. Both the HME and HH devices slightly increase the inspiratory resistance which remains stable over time after prolonged use of the HME. In addition, the internal volume of the HME, ranging from 50 to 90 ml in adults, can significantly increase the instrumental dead space in the patient's ventilator circuit. This could potentially have an adverse effect on the mechanics of the ventilation, especially in SB modes and MV weaning. The selection of the two PSV levels tested was directed to consider extubation and those that are commonly used during MV weaning. HME-induced Wi overload was observed not only at low PSV levels (7 cm H2O) but also at relatively high PSV levels (15 cm H2O). These results are more important because PSV is not only a traditional MV weaning or extubation technique, but should increasingly be used in the early stages of treatment of acute respiratory failure. Our comparative data between HH-7 and HME-15 confirm that PSV levels should be increased by 8 cm H2O when using HME in the study. To our knowledge, no clinical studies compare the effects of HME and HH on the outcome of the patients' withdrawal process. However, the clinician must be aware of the HME-induced Wi overload in SB modes, in addition to the endotracheal tube and ventilator circuits, to avoid the erroneous assumption that patient ventilation may be difficult, "unavailable", or even. It cannot be extubated, especially in patients with difficult to wean CRF. We only assessed one type of HME and our results could not be extrapolated to another HME based on instrumental dead space and inspiratory resistance. Unless HMEs with lower internal volumes are assessed in such situations, clinicians may be offered two options for patients with weaning potential: either increase PSV levels sufficiently or use the tee technique more drastically, avoiding any connection to the ventilator.

CONCLUSION:

The use of HME with PSV in patients with difficult or potentially difficult to separate CKD can result in inspiratory strain, increased ventilation requirements, impaired gas exchange with ventilatory acidosis, and respiratory failure. To counteract these deleterious effects, it seems necessary to increase the PSV level by 8 cm H2O. Accordingly, the type of AHD used may adversely affect the mechanical efficiency of ventilation, especially in SB modes, and significantly alter the interpretation and outcome of the weaning process in these patients. Unless PSV levels are significantly elevated, this type of HME should not be recommended for use in patients with CKD in whom weaning is difficult or potentially difficult.

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