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Comparison of the effects of heat and moisture exchangers and heated humidifiers on ventilation and gas exchange during non-invasive ventilation

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Abstract *Objective:* To compare the short-term effects of a heat and moisture exchanger (HME) and a heated humidifier (HH) during non-invasive ventilation (NIV). *Design:* Prospective, clinical investigation. *Setting:* Intensive care unit of a university hospital. *Patients:* Twenty-four patients with acute respiratory failure (ARF). *Intervention:* Each patient was studied with a HME and a HH in a random order during two consecutive 20min periods of NIV. *Measurements and results:* Respiratory rate (RR), expiratory tidal volume (VTe) and expiratory minute ventilation (VE) were measured during the last 5 min of each period and blood gases were measured. Mean pressure support and positive end-expiratory pressure levels were, respectively, 15±4 and 6±2 cmH_2O. VE was significantly greater with HME than with HH (14.8±4.8 vs 13.2±4.3 l/min; $p<0.001$). This increase in VE was the result of a greater RR for HME than for HH (26.5±10.6 vs 24.1±9.8 breaths/min; $p=0.002$), whereas the VT for HME was similar to that for HH (674±156 vs 643±148 ml; $p=0.09$). Arterial partial pressure of carbon dioxide ($PaCO_2$) was significantly higher with a HME than with a HH (43.4±8.9 vs 40.8±8.2 mmHg; $p<0.005$), without significantly changing oxygenation. *Conclusion:* During NIV the increased dead space of a HME can negatively affect ventilatory function and gas exchange. The effect of HME dead space may decrease efficiency of NIV in patients with ARF.

Keywords Heat and moisture exchanger · Heated humidifier · Non-invasive ventilation

Introduction

Humidification and warming of the inspired gases performed by artificial devices may be required to prevent the undesirable effects of cool, dry gases on the tracheobronchial epithelium during non-invasive ventilation (NIV), as has been well documented in mechanically ventilated patients [1]. The last International Consensus Conference in Intensive Care that considered the role of NIV [2] reported that inadequate humidification may cause patient distress, especially if pipeline or cylinder gas is used, but no recommendations were made concerning the technique of humidification, probably due to the lack of data concerning the impact of humidification devices on the efficiency of NIV. Two humidifying devices are commonly used: heated humidifiers (HH), which have been commonly used in the past, and heat and moisture exchangers (HME), which are increasingly used due to their simplicity of use and cost-effectiveness [3]. Because the HMEs are placed between the endotracheal tube and the Y-piece in the intubated patient or between the facial mask and the Y-piece in patients managed by NIV, they add a substantial amount of dead space, in contrast to a HH, which is placed in the inspiratory circuit. This reduces alveolar ventilation for a given minute ventilation.

Moreover, HMEs may increase resistance to flow [4, 5]. Some studies [6, 7, 8, 9] performed with intubated patients during weaning trials from mechanical ventila-

tion showed that the additional dead space of the HME induced an increase in minute ventilation to maintain efficient alveolar ventilation. This leads to an increase in work of breathing (WOB) [7] often associated with an increase in arterial partial pressure of carbon dioxide ($PaCO_2$) [6, 9]. Some authors [7, 8] suggested that an additional 5–10 cmH_2O of pressure support is required to counter balance the increase in WOB caused by the HME dead space. However, it is known that the high-pressure levels sometimes required to improve NIV efficiency may induce the risk of face mask intolerance and increased leaks [10, 11, 12]. But the effects of HMEs on the efficiency of NIV remain largely unknown [2, 11].

We thus conducted a physiological study to compare the short-term effects of a HME and a HH on the ventilatory parameters and gas exchange during NIV in 24 patients with hypoxemic ARF.

Material and methods

The experimental protocol was approved by an institutional review board for human subjects (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale; CHU Montpellier). Written informed consent was obtained from each study patient.

Selections of patients

The study involved 24 patients who, due to various pathologies mainly in the postoperative period, received NIV for ARF. Patients were enrolled if they met at least two of the following criteria: respiratory rate (RR) of 25 breaths/min or more, arterial oxygen partial pressure (PaO_2) below 60 mmHg breathing room air, $PaCO_2$ of 45 mmHg or more and an arterial pH of 7.38 or less. Exclusion criteria were as follows: need for immediate endotracheal intubation or enrollment in another investigative protocol.

Study design

The protocol consisted of comparing two consecutive 20min periods of NIV with pressure support ventilation (PSV), one with a HME (Hygrobac, DAR, Mallinckrodt, St Louis, USA) and the other with a conventional HH (Fisher & Paykel, MR 730, Panmure, New Zealand). The Hygrobac HME is a hygroscopic-hydrophobic device with a dead space of 84 ml. The MR 730 HH is composed of a disposable humidification chamber and heated wire set to maintain proximal airway temperature (Y-piece) at 34°C and chamber temperature at 31°C. The HH was filled with water and placed in the inspiratory circuit The sequence of periods was randomized. The same Evita IV ventilator (Dräger, Lübeck, Germany) was used to deliver PSV by a face mask (Péters, Airvie, Bobigny, France) and connected to an acquisition system software (Evitaview 2.01, Drägerwerk). The PSV level was adjusted for each patient to obtain an expired tidal volume (VTe) of 7–10 ml/kg as measured by the ventilator. The fraction of inspired oxygen (FIO_2) was adjusted to obtain a percutaneous oxygen saturation (SpO_2) above 95% and we used a positive end-expiratory pressure level (PEEP) of 4–8 cmH_2O.

Physiological measurements

In each condition, data were recorded during a 5min period after a 15min breathing pattern stabilization period. The inspired (Vi) and expired (Ve) minute ventilation, the inspired tidal volume (VTi), VTe and the RR displayed on the ventilator during the last 5 min of each period were averaged, based on 10–15 reproducible cycles to the recorded data. We also measured the ratio of expired over inspired minute volume to estimate the leaks around the mask [13]. We used the P0.1 measurement technique developed as an integrated function of the Evita IV ventilator, which was used in NIV in the Hilbert trial [14]. Before each data acquisition, the face mask was carefully positioned in order to minimize gas leaks around the mask. Heart rate (HR), mean arterial blood pressures (MAP) and SpO_2 were continuously monitored. Blood gases were measured using an analyzer. The ventilatory conditions were identical in both sets of measurements.

Statistical analysis

Data are reported as means ± SD. The measurements obtained in each condition studied were compared, with each patient serving as his or her own control. The data were analyzed using Wilcoxon test to detect differences between HME and HH. Regression analysis (Spearman rho) was used when required. Probability values smaller than 0.05 were considered significant.

Results

Patient characteristics are shown in Table 1. At the time of the study, the patients were ventilated with a pressure support (PS) level of 15±4 cmH_2O, a PEEP level of 6±2 cmH_2O, a FIO_2 of 53±12% and the inspiratory trigger was set at 1.4±0.7 l/min. All the patients remained hemodynamically stable during the study period and there was no statistical difference between HH and HME for HR (89±20 vs 90±19 beats/min; ns) and for MAP (94±12 vs 95±12 mmHg; ns).

Results are presented for all the patients, and for the hypercapnic ($PaCO_2$>45 mmHg at inclusion) and non-hypercapnic patients. The mean values of the main ventilatory parameters and P0.1 are presented in Table 2. Blood gas parameter values are reported in Table 3. Individual values for VE, $PaCO_2$ and P0.1 are shown in Figs. 1, 2 and 3, respectively.

The modifications induced by the HME were observed in both the non-hypercapnic and hypercapnic patients, with a marked increase of the RR, $PaCO_2$ and P0.1 for the hypercapnic patients (Tables 2, 3). There was also a nearly statistically significant negative correlation between VTe measured during breathing with HME and change in $PaCO_2$ measured during breathing with HME, as compared with HH obtained in the ten hypercapnic patients (r=–0.71, p=0.02) (Fig.4).The modifications induced by the HME were obtained for the two groups of patients, with a markedly pronounced alteration for RR and $PaCO_2$ for the patients with a $PaCO_2$ higher than 45 mmHg (Tables 2, 3).

The leaks around the mask did not differ significantly between the study conditions: mean values were 18±14% with the HME and 16±12% with the HH. The temperature measured at the Y-piece was significantly higher when a HH was used than when a HME was used (31.2±2.0 vs 27.8±2.8°C; p<0.001).

Table 1 Characteristics of the patients (*SAPS II* Simplified Acute Physiology Score II, PaO_2 arterial partial pressure of oxygen, $PaCO_2$ arterial partial pressure of carbon dioxide, *RA* reserve alcalin: bicarbonate)

Patient	Diagnosis	Gender	Age (years)	Weight (kg)	Height (m)	SAPS II	PaO_2 (mmHg)	$PaCO_2$ (mmHg)	RA (mmol/l)	Ph
1	Postoperative failure	M	70	50	1.63	27	43	36	33	7.57
2	Postoperative failure	F	71	109	1.65	22	46	38	23	7.39
3	Postoperative failure	M	53	75	1.81	40	72	33	21	7.39
4	Postoperative failure	F	49	73	1.62	25	52	35	26	7.48
5	Postoperative failure	F	69	72	1.57	43	53	44	34	7.49
6	Pneumonia	F	46	46	1.64	32	52	32	21	7.43
7	Postoperative failure	M	42	100	1.74	27	53	41	25	7.40
8	Congestive heart failure	F	79	65	1.58	35	42	33	21	7.40
9	Postoperative failure	F	72	120	1.55	28	63	39	23	7.37
10	Septic chock	F	59	49	1.59	55	78	85	30	7.16
11	Acute pancreatitis	M	57	95	1.86	43	65	51	34	7.42
12	Septic pulmonary infarct	M	48	69	1.68	42	57	43	31	7.46
13	Postoperative failure	M	67	94	1.75	35	66	43	29	7.43
14	Pneumonia	F	85	52	1.58	48	43	61	37	7.39
15	Pneumonia	M	81	68	1.71	70	47	43	32	7.45
16	Postoperative failure	F	70	74	1.59	40	59	40	26	7.46
17	COPD exacerbation	M	40	75	1.73	50	70	65	36	7.34
18	Pneumonia	M	50	45	1.67	80	70	54	28	7.31
19	COPD exacerbation	M	58	65	1.60	19	73	69	30	7.29
20	Acute pancreatitis	M	31	70	1.77	21	73	36	30	7.53
21	Postoperative failure	M	52	49	1.76	37	54	34	26	7.48
22	Congestive heart failure	M	70	59	1.69	55	52	45	35	7.50
23	Postoperative failure	M	46	82	1.77	39	53	45	24	7.43
24	Acute pancreatitis	M	45	77	1.78	25	65	48	29	7.39
	Mean	15M/9F	59	72	1.68	35	58	46	29	7.41
	SD		14	20	0.09	13	11	13	5	0.09

Values for arterial blood gas measurements were obtained at the enrolment in room air, except for patients 3, 9, 13 and 20, who were breathing with flow of oxygen between 6 and 10 l/min

Table 2 Respiratory parameters and P0.1 values during the two conditions (*RR* respiratory rate, *VTe* expired tidal volume, *VE* minute ventilation)

		HH	HME	*p* value Wilcoxon-test
RR (breaths/min)	All (*n*=24)	24.1±9.8	26.5±10.6	0.002
	Hypercapnic (*n*=10)	25.6±10.9	28.9±12.4	0.011
	Non-hypercapnic (*n*=14)	23.0±9.2	24.8±9.2	0.044
VTe (ml)	All (*n*=24)	643±148	674±156	0.088
	Hypercapnic (*n*=10)	638±183	679±191	0.213
	Non-hypercapnic (n=14)	642±132	663±140	0.306
VE (l/min)	All (*n*=24)	13.2±4.3	14.8±4.8	<0.001
	Hypercapnic (*n*=10)	14.8±5.2	16.6±5.3	0.007
	Non-hypercapnic (*n*=14)	12.0±2.7	13.9±3.8	0.004
P0.1 (cmH_2O)	All (*n*=19)	1.8±0.9	2.5±1.6	0.001
	Hypercapnic (*n*=8)	2.0±0.8	2.7±1.3	0.017
	Non-hypercapnic (*n*=11)	1.6±1.1	2.2±1.9	0.035

Values are means ± SD

Table 3 Gas exchanges during the two conditions ($PaCO_2$ arterial partial pressure of carbon dioxide, PaO_2/FIO_2 arterial partial pressure of oxygen/fraction of inspired oxygen ratio)

		HH	HME	*p* value Wilcoxon-test
pH	All (*n*=24)	7.44±0.06	7.42±0.06	<0.001
	Hypercapnic (*n*=10)	7.43±0.07	7.40±0.07	0.024
	Non-hypercapnic (*n*=14)	7.45±0.05	7.44±0.05	0.013
$PaCO_2$ (mmHg)	All (*n*=24)	40.8±8.2	43.4±8.8	<0.001
	Hypercapnic (*n*=10)	46.0±10.2	49.4±10.5	0.007
	Non-hypercapnic (*n*=14)	37.1±3.4	39.1±3.9	0.006
PaO_2/FIO_2 (mmHg)	All (*n*=24)	221±80	229±79	0.325
	Hypercapnic (*n*=10)	214±79	212±66	0.779
	Non-hypercapnic (*n*=14)	225±83	236±86	0.051

Values are means ± SD

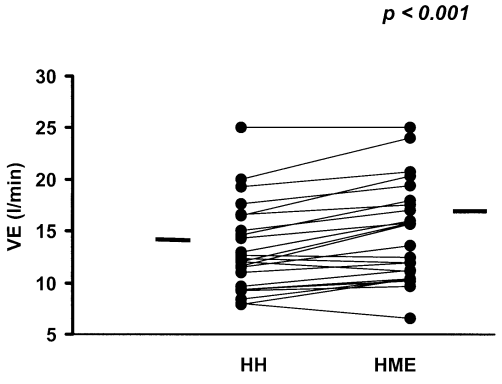

Fig. 1 Individual variations of minute ventilation (VE: l/min) during the two conditions studied. VE was significantly higher with the heat and moisture exchanger (HME) than with the heated humidifier (HH)

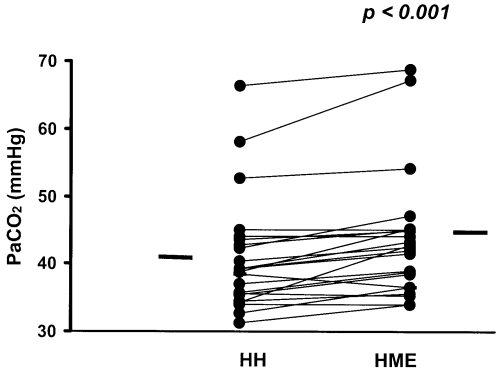

Fig. 2 Individual variations in arterial partial pressure of carbon dioxide ($PaCO_2$) between the heat and moisture exchanger (HME) and the heated humidifier (HH). $PaCO_2$ was significantly higher with the HME than with the HH

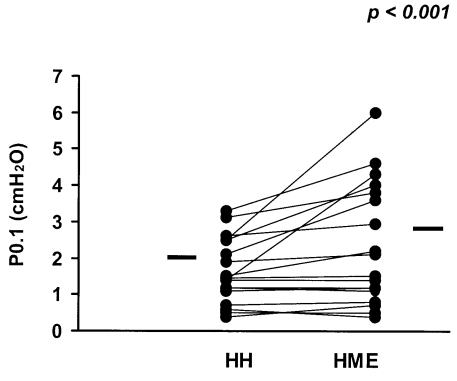

Fig. 3 Individual variations of occlusion pressure (P0.1) during the two conditions studied. P0.1 was significantly higher with the heat and moisture exchanger (HME) than with the heated humidifier (HH)

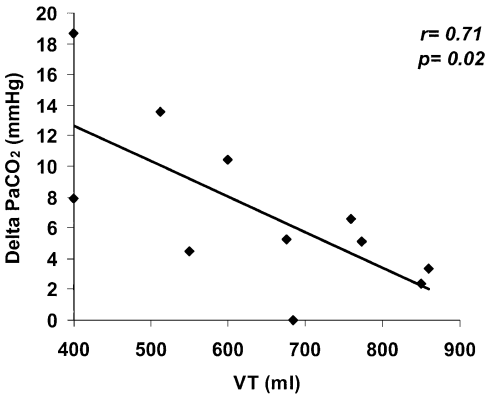

Fig. 4 Negative correlation between tidal volume (VT) value with heat and moisture exchanger (HME) and delta arterial partial pressure of carbon dioxide ($PaCO_2$) (changes in $PaCO_2$ measured during breathing with HME as compared with HH) obtained in the ten hypercapnic patients

Discussion

The main finding of our study is that the use of a heat and moisture exchanger (HME) in patients managed by non-invasive ventilation (NIV) for acute respiratory failure (ARF) can lead to a relevant increase of $PaCO_2$, despite significant increases of minute ventilation.

Non-invasive ventilation delivered via a face mask is being increasingly used in patients with ARF as a means to reduce endotracheal intubation and associated short- and long-term complications [2, 15]. However, the necessity of intubation occurs in about 50% of ARF patients (25–70% according to the literature [2, 11]). Improving the clinical efficiency of NIV is a daily challenge for clinicians. Inadequate humidification can cause patient distress in intubated patients [16, 17] and recently life-threatening complications have been reported with prolonged NIV because of under-humidified gases [18]. During controlled mechanical ventilation, HMEs are effective with little or no effect on respiratory mechanics. However, many recent studies in intubated or tracheotomized patients have shown that the dead space added to the circuit has been implicated as a source of ventilatory impairment during inspiratory PSV [6, 7, 8, 9]. HME dead space appears to have the greatest effect on ventilatory parameters, indeed the low resistance (2.0 cmH_2O/l per s at 1 l/s) of the HME used in our study cannot be ignored.

Our findings indicate that HMEs increase minute ventilation and $PaCO_2$. Minute ventilation was increased by increasing RR when the VT was constant. This was probably due to the constant PS level, making changes in VT more difficult than those of RR. Nevertheless, the size of the VE increase was insufficient effectively to increase alveolar ventilation and prevent CO_2 retention, as shown by the increase of $PaCO_2$ and the decrease in pH

under HME. As might be expected, the adverse effect of adding dead space with the HME was more pronounced in patients with lower VT and/or with a $PaCO_2$ higher than 45 mmHg (Tables 2, 3 and Fig. 4).

These NIV findings are consistent with previous trials, which compared the use of the two humidification devices with intubated or tracheotomized patients [6, 7, 8, 9] and demonstrated an increase of VE [6, 7, 8, 9], RR [6, 9] and $PaCO_2$ [6].

Our study had several limitations. We did not directly calculate patient WOB, but we measured the occlusion pressure, an index for inspiratory effort [19, 20, 21]. P0.1 increases significantly when a HME is added to the ventilatory circuit, suggesting that the HME could alter the efficiency of NIV in some patients, especially in very weak patients. The P0.1 parameter was used in NIV by Hilbert et al. [14]. Indeed, we did not measure the WOB, but the increase in minute ventilation resulting from the added space and the increase in P0.1 could lead to an overload of the respiratory muscles. Another limitation of the study is that we did not compare the impact of the two devices without PEEP. The use of PEEP is known to increase the leaks around the mask, and this may lead to dead space wash-out.

In conclusion, our findings suggest that during NIV, the increased dead space of a HME can negatively affect ventilatory function and gas exchange, especially in hypercapnic and very weak patients. Further studies are needed to better determine the impact of the HME on the outcome of NIV. Pending the results of further studies, when choosing between available humidification devices, physicians should take into account the physical characteristics as well as the temperature and moisture output of each available device.

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