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Ability and safety of a heated humidifier to control hypercapnic acidosis in severe ARDS

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Abstract *Objective:* To assess the ability of a heated humidifier to improve CO_2 clearance in ARDS patients submitted to protective ventilation. *Design:* Prospective clinical study. *Setting:* University hospital intensive care unit. *Patients:* During a 12-month period, we studied 11 ARDS patients under protective mechanical ventilation with severe hypercapnia. *Intervention:* When $PaCO_2$ was above 55 mmHg, the heat and moisture exchanger (HME) was removed and patients were ventilated using a heated humidifier (HH) until their recovery or death. The heated humidifier was inserted on the inspiratory limb of the respirator and the inspirated air was saturated to achieve a temperature of 40 °C at the Y connector of ventilator tubing and of 37 °C at the outlet of the endotracheal tube. *Measurements and results:* Mechanical measurements and blood gas analysis were performed just before removal of the HME, and 30 min after mechanical ventilation using HH. Ventilator parameters were kept constant in the two conditions. Using HH instead of HME, $PaCO_2$ was safely decreased by 11±5 mmHg, without any need to change respiratory rate. No significant difference was noted in intrinsic PEEP or airway plateau pressure. Decrease in $PaCO_2$ after HME removal was strongly correlated with the initial value of $PaCO_2$. *Conclusion:* Supposing there is an interest in correcting or limiting hypercapnic acidosis in ARDS patients submitted to protective ventilation, HME removal and use of HH appears to be an efficient and safe way of increasing CO_2 clearance.

Keywords ARDS · Protective ventilation · Permissive hypercapnia · Heat and moisture exchanger · Heated humidifier

Introduction

Retrospective studies have suggested for a long time that the use of a low tidal volume (TV) during mechanical ventilation, to limit lung stretch, was responsible for a dramatic decrease in mortality in ARDS patients [1, 2]. By decreasing minute ventilation, this strategy may induce "permissive hypercapnia" which may be beneficial as previously reported [3]. In fact, the ARDS Network has recently demonstrated, in a randomized control trial, a significant decrease in mortality when using a lower TV than in the traditional approach [4]. However, it has since been suggested that the positive result of this study, at variance with other recent prospective TV reduction studies [5, 6], might be explained in part by a specific effort to correct hypercapnic acidosis [7, 8]. Indeed, the adverse effects of hypercapnic acidosis have been largely documented [9]. The strategy used in the NIH study to improve CO_2 clearance was a high respiratory rate (30 breaths/min) during volume-controlled ventilation. However, we have previously reported that a such strategy during mechanical ventilation in ARDS patients produced dynamic hyperinflation, and impaired right ventricular ejection [10].

Humidification and warming of the inspired gases during mechanical ventilation requires either a heated humidifier (HH) or a heat and moisture exchanger (HME) on the inspiratory limb of the respirator. Some studies in patients with chronic obstructive lung disease (COPD) – during weaning from mechanical ventilation – have demonstrated that using HH instead of HME decreases dead space and respiratory work, and increases CO_2 clearance [11, 12]. Hurni et al. have recently suggested in two cases that HH could efficiently control hypercapnic acidosis in ARDS patients [13]. However, to our knowledge, the potential effect and the safety of a such ventilatory strategy in ARDS patients was never reported in a significant series. The present study was designed to investigate the effects on CO_2 clearance of using HH after removal of HME and its safety.

Materials and methods

Patients

During a 12-month period we studied prospectively 11 successive patients (six men and five women, mean age 50±17 years) who required mechanical ventilation for an episode of acute respiratory failure. All these patients met the North American-European Consensus criteria for ARDS, with an acute onset of respiratory failure, bilateral chest infiltrates, a PaO_2/FiO_2 ratio <200 mmHg, and no evidence of increased left ventricular filling pressure by transthoracic echocardiographic examination. The simplified acute physiologic score (SAPS II), logistic organ dysfunction score (LODS), and lung injury severity score (LISS) were calculated in the first 24 h following admission to the ICU. SAPS II was used to calculate predicted hospital mortality and the "Standard Mortality Ratio" (SMR) was obtained by dividing the observed by the predicted hospital mortality.

Ventilatory strategy

All patients were sedated with midazolam and sufentanil, paralyzed with cisatracurium during the time required for respiratory measurements, and ventilated in volume-controlled mode, using a 7200 Puritan Bennet (Puritan Bennet, Carlsbad, Cakif., USA), equipped with an HME (Pall Biomedical, East Hills, N.Y., USA), whose measured instrumental dead space was 100 ml. The airway plateau pressure (Pplateau), measured with an end-inspiratory pause of 0.5 s, was kept below 30 cmH_2O (27±4 cmH_2O). In the event of severe hypercapnia during ventilation, defined as a $PaCO_2$ above 55 mmHg, HME was removed and patients were ventilated using a heated wire humidifier (MR 850, Fisher & Paykel, Auckland, New Zealand), until their recovery or death. The HH was inserted on the inspiratory limb of the respirator and the inspirated air was saturated to achieve a temperature of 40 °C at the Y connector of ventilator tubing, and of 37 °C at the outlet of the endotracheal tube. No circuit changes were performed throughout ventilatory support. This protocol was included in a routine strategy and informed consent was not judged to be necessary.

Respiratory measurements and data collection

Airway pressure (P) and volumes (V) were measured with the pressure transducers and pneumotachographs incorporated into the ventilator. Static compliance of the respiratory system (Crs) was calculated as TV/(Pplateau–PEEP). Blood gas analysis was performed just before removal of HME, and 30 min after mechanical ventilation using HH. Ventilator parameters were kept constant in the two conditions, included a constant inspiratory flow of 50 l/min, a TV of 7.6±0.6 ml/kg, an RR of 16±2 breaths/min, and an I:E ratio of 1:2. The PEEP selected was that producing oxygenation improvement without requiring additional hemodynamic support, resulting in an average PEEP of 6±2 cmH_2O.

Intrinsic PEEP (PEEPi) was measured in ZEEP by occluding the airway during a prolonged end-expiratory pause of 4 s by use of the end-expiratory hold button of the ventilator [14]. The decrease in the dead space ratio (Vd/Vt) with HH was calculated – and not measured – as the ratio between instrumental dead space of HME (100 ml) and the tidal volume. The mean duration of mechanical ventilation, as well as the duration of HH use, was noted.

Statistical analysis

Statistical calculations were performed using the StatView 5 (SAS Institute, Cary, N.C., USA). Data are expressed as mean±SD. Respiratory parameters with the HME and HH devices were compared by means of a Wilcoxon signed-rank test. A P value less than 0.05 was considered significant.

Table 1 Individual initial data, etiologic features, and outcome of ARDS. (*SAPS II* new simplified acute physiology score, *LISS* lung injury severity score, *P/F* PaO_2/FiO_2, *Crs* static compliance of the respiratory system)

	Sex	Age (years)	SAPS II	LISS	P/F	Crs (ml/cmH_2O)	Etiology	Recovery
1	F	30	53	3.3	63	22	Aspiration	Yes
2	M	51	30	3.3	141	28	Pneumonia	Yes
3	M	75	54	3	49	27	Pneumonia	No
4	M	69	33	4	65	25	Pneumonia	No
5	M	46	46	3	56	22	Pneumonia	Yes
6	M	20	37	2.3	111	34	Aspiration	Yes
7	F	53	73	3.3	95	20	Pneumonia	Yes
8	M	81	78	3	58	30	Pneumonia	No
9	F	44	63	3	57	25	Aspiration	No
10	F	45	34	3	66	18	Pneumonia	Yes
11	F	27	34	3.3	45	14	Pneumonia	Yes
M±SD		49±19	49±17	3.1±0.4	73±30	24±6		

Table 2 Respiratory measurements before and after removal of the heat and moisture exchanger. (*HME* heat and moisture exchanger, *HH* heated humidifier, *Pplateau* plateau pressure, *MV* minute ventilation, *PEEPi* intrinsic PEEP)

	HME	HH	*P*
MV (l/min)	7.7±1.2	7.7±1.2	
PaO_2 (mmHg)	69±15	65±10	NS
SaO_2 (%)	88±7	89±5	NS
$PaCO_2$ (mmHg)	67±9	56±6	0.003
pH	7.20±0.11	7.26±0.06	0.005
Pplateau (cmH_2O)	27±4	27±4	NS
PEEPi (cmH_2O)	2±1.5	2±1.5	NS

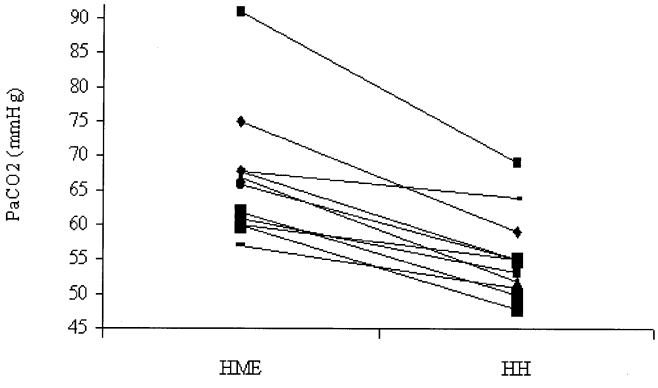

Fig. 1 Individual patient values for $PaCO_2$ with HME and HH. $PaCO_2$ was significantly lower with the HH than with the HME

Results

The clinical characteristics of the 11 patients are given in Table 1. ARDS appeared severe with a PaO_2 / FiO_2 ratio below 100 (73±30), a SAPS II of 49±17. The hospital mortality predicted from SAPS II was 41%. Four patients died, and seven recovered, resulting in a overall mortality rate of 36%. The SMR was 0.88. The mean duration of mechanical ventilation was 16.8±11.4 days, with 12±9 days using HH.

After HME removal, mechanical ventilation using HH was responsible for a significant decrease in $PaCO_2$ (–11±5 mmHg, P=0.003), associated with a significant increase in pH (+0.06±0.04, P=0.005), whereas minute ventilation was kept constant, as shown in Table 2. Individual values of $PaCO_2$ are indicated in Fig. 1. No significant effect was reported on PaO_2 and SaO_2. HME removal induced a calculated reduction in Vd/Vt of 0.19±0.04. Decrease in $PaCO_2$ after HME removal was strongly correlated with the initial value of $PaCO_2$, as shown in Fig. 2 (r=0.78, P=0.004). No significant difference was noted in PEEPi or Pplateau after HME removal.

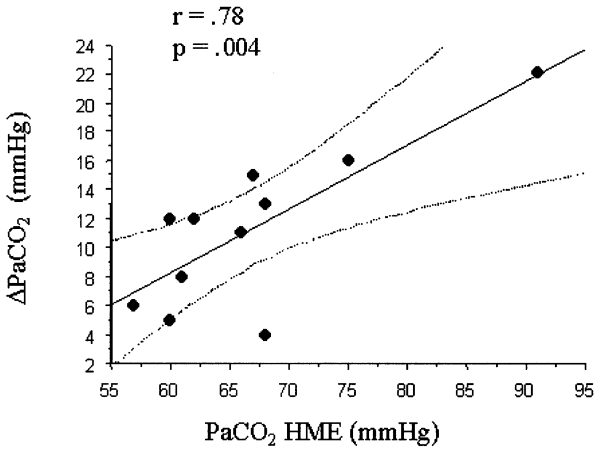

Fig. 2 Relation between $PaCO_2$ at baseline with HME and the amount of decrease in $PaCO_2$ with HH ($\Delta PaCO_2$). The $\Delta PaCO_2$ was significantly correlated with the baseline value of $PaCO_2$

Discussion

Retrospective studies have suggested a significantly improved prognosis in ARDS patients by using a low tidal volume to limit lung stretch during mechanical ventilation [1, 2], whereas the first prospective studies have failed to demonstrate such an improvement [5, 6]. By decreasing minute ventilation, this strategy may be responsible for "permissive hypercapnia", and may be in part responsible for increased survival in ARDS, as previously reported by Hickling et al. [3]. Some authors have even suggested a protective role of systemic carbon dioxide tension against tissue injury, and thus proposed the concept of "therapeutic hypercapnia" [15]. Hypercapnia increases oxygen delivery to organs, by increasing cardiac output, and shifting the haemoglobin-oxygen dissociation curve rightwards. In addition, acidosis may reduce oxygen consumption. However, the adverse effects of hypercapnic acidosis have been largely documented [9]. Hypercapnic acidosis may adversely effect right ventricular performance by inducing pulmonary arteriolar vasoconstriction leading to pulmonary hypertension [16, 17]. We have previously reported in ARDS patients that hypercapnia was significantly and independently associated with the presence of an acute cor pulmonale at echocardiography, reflecting an acute right ventricular dysfunction, in response to a dramatic increase in afterload [18].

A recent cooperative study seems to have closed 10 years of controversy, by demonstrating a significant reduction in mortality with tidal volume reduction in a large population of patients meeting the criteria for Acute Lung Injury or Acute Respiratory Distress Syndrome [4]. It has since been suggested that the positive result of this study might be explained not only by limiting lung stretch, but also by a systematic correction of hypercapnic acidosis [7, 8]. In this way, attempting to

limit hypercapnia during protective ventilation actually seems laudable.

The strategy used in the NIH study [4] to improve CO_2 clearance after tidal volume reduction was unusual. The authors utilized a high respiratory rate (30 breaths/min) during volume-controlled ventilation, and nothing is reported about the consequence of a such strategy on dynamic hyperinflation and hemodynamics. We have previously shown that high respiratory rate in ARDS patients produced dynamic hyperinflation and impaired right ventricular ejection, by increasing right ventricular afterload [10]. Supposing that hypercapnia acidosis may be corrected or limited in ARDS patients submitted to protective ventilation, an apparatus dead space reduction would probably have been more efficient and more tolerated than a high respiratory rate. It was previously suggested by Richecoeur et al. that removal of the 15-cm-long tubing connecting the proximal tip of the endotracheal tube and the Y-piece reduced the instrumental dead space by 40 ml [19]. Unfortunately, these authors combined the reduction in apparatus dead space with a high RR, thereby rendering the interpretation of $PaCO_2$ variations difficult. In our study, we demonstrated in 11 ARDS patients submitted to protective ventilation the ability of such an alternative strategy to control hypercapnic acidosis. Using HH, by decreasing apparatus dead space, $PaCO_2$ can be safely decreased without any need to change respiratory rate.

HME has become widespread in the intensive care unit in recent years, because of its many advantages, such as easier management and reduced cost [20]. Recently, Ricard et al. have reported that a such strategy was more often used in France than in Canada (93% versus 35.3% of the intensive care units, respectively) for any duration of mechanical ventilation, and that HME was largely used in ARDS patients [21]. Since HME is placed between the endotracheal tube and the Y piece, it adds a substantial amount of dead space, reducing alveolar ventilation [12]. It has previously been shown that HH efficiently improves CO_2 clearance during weaning trials, notably in COPD patients [11], but to our knowledge, its efficiency compared to HME was never reported in a series of mechanically ventilated ARDS patients. Although not specified, it seems, however, that HH was largely used in the NIH study, possibly explaining in part the ability of a high respiratory rate to normalize $PaCO_2$ in this study [4].

In our study, the baseline value of $PaCO_2$ with HME accurately predicts the effect of HME removal on CO_2 clearance improvement. The higher the baseline hypercapnia, the higher was the observed decrease in $PaCO_2$ after HME removal, suggesting the use of this strategy in the most hypercapnic ARDS patients only. As reported in our results, mechanical parameters, as plateau pressure and PEEPi, did not change with HME or with HH, in contrast to a previous study where PEEPi was significantly lower with HH [12]. However, in this study, the difference in PEEPi, although significant, was extremely low and clinically irrelevant [12].

In conclusion, supposing there is an interest in correcting or limiting hypercapnic acidosis in ARDS patients submitted to protective ventilation, HME removal and use of HH appears to be an efficient and safe way of increasing CO_2 clearance.

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