Intensive Care Med (1997) 23: 399–405

ORIGINAL

G. A. Iotti
M. C. Olivei
A. Palo
C. Galbusera
R. Veronesi
A. Comelli
J. X. Brunner
A. Braschi

Unfavorable mechanical effects of heat and moisture exchangers in ventilated patients

Received: 14 June 1996
Accepted: 6 December 1996

G. A. Iotti (✉) · A. Palo · C. Galbusera · R. Veronesi · A. Comelli · A. Braschi
Servizio di Anestesia e Rianimazione 1, I. R. C. C. S. Policlinico S. Matteo, I-27100 Pavia, Italy,
FAX: + 39 (382) 503008

M. C. Olivei
Laboratorio di Biotecnologie e Tecnologie Biomediche, I. R. C. C. S. Policlinico S. Matteo, Pavia, Italy

J. X. Brunner
Hamilton Bonaduz AG, Bonaduz, Switzerland

Supported by a grant from I. R. C. C. S. Policlinico S. Matteo
Technical support from Hamilton Bonaduz AG

Abstract *Objective:* To investigate the mechanical effects of artificial noses.
Setting: A general intensive care unit of a university hospital.
Patients: 10 patients in pressure support ventilation for acute respiratory failure.
Interventions: The following three conditions were randomly tested on each patient: the use of a heated humidifier (control condition), the use of a heat and moisture exchanger without filtering function (HME), and the use of a combined heat and moisture exchanger and mechanical filter (HMEF). The pressure support level was automatically adapted by means of a closed-loop control in order to obtain constancy, throughout the study, of patient inspiratory effort as evaluated from airway occlusion pressure at 0.1 s ($P_{0.1}$). Patient's ventilatory pattern, $P_{0.1}$, work of breathing, and blood gases were recorded.
Measurements and main results: The artificial noses increased different components of the inspiratory load: inspiratory resistance, ventilation requirements (due to increased dead space ventilation), and dynamic intrinsic positive end-expiratory pressure (PEEP). The additional load imposed by the artificial noses was entirely undertaken by the ventilator, being the closed-loop control of $P_{0.1}$ effective to maintain constancy of patient inspiratory work by means of adequate increases in pressure support level.
Conclusions: The artificial noses cause unfavorable mechanical effects by increasing inspiratory resistance, ventilation requirements, and dynamic intrinsic PEEP. Clinicians should consider these effects when setting mechanical ventilation and when assessing patients' ability to breathe spontaneously.

Key words Respiration, artificial instrumentation · Heat and moisture exchanger · Respiratory mechanics · Pressure support ventilation · $P_{0.1}$ · Feedback control

Introduction

For the past few years, the literature has consistently underlined the importance of inspiratory circuitry on the inspiratory workload [1, 2]. Humidification and warming of the inspired gases during mechanical ventilation requires the addition either of a heated humidifier or of an artificial nose to the external circuit of the ventilator. The use of artificial noses, initially limited to anesthesia, is now increasingly being extended to the intensive care unit (ICU) for patients undergoing long-term mechanical ventilation. Artificial noses may be an interesting alternative to heated humidifiers, since they offer the advantages of lower cost, simpler maintenance of the respiratory circuitry, and no need for electrical power. Moreover, many artificial noses combine an antimicrobial filtering function.

The humidification efficiency of artificial noses compared to that of heated humidifiers has been extensively investigated both in vitro and in vivo [3–6]. However, data on the mechanical effects of artificial noses are few and controversial. Artificial noses have been reported to increase considerably the resistive load of the ventilator circuit in vitro [7]. However, an in vivo study suggests that artificial noses play a minor role in increasing airway resistance, but only in rare cases in which the filter membrane is exposed to particularly abundant secretions [8]. Lack of relevant mechanical interference was also confirmed by a study on patients with chronic obstructive pulmonary disease (COPD), in which the application of artificial noses was associated with no increase in dynamic pulmonary hyperinflation [9]. In contrast, a more recent study indicates that artificial noses can remarkably increase the mechanical load of respiration in patients ventilated in pressure support mode for acute respiratory failure [10].

In the present study, we analyzed the short-term mechanical effects of two different artificial noses compared to those of a conventional heated humidifier in patients mechanically ventilated for acute respiratory failure. The response to the addition of each humidifier to the ventilator circuit was assessed at steady state in each patient and was evaluated in terms of respiratory pattern, drive, work of breathing, and gas exchange. The study was performed on patients assisted with pressure support ventilation (PSV). For easier comparison of the three trials, the pressure support level was adapted in order to achieve constancy throughout the study of patient inspiratory effort as evaluated from airway occlusion pressure at 0.1 s ($P_{0.1}$).

Patients and methods

The study involved 10 patients who, due to various pathologies, received PSV for acute respiratory failure. None of the patients was affected by COPD, which was defined following the American Thoracic Society standard [11]. Seven patients were tracheotomized and three orally intubated. The patients were four females and six males, mean age 58 ± 14 years (range 30–83 years). Their average height and weight were 170 ± 6 cm and 63 ± 9 kg, respectively. All patients were awake and free from pathological and/or pharmacological central neural depression. Respiratory center function was normal as judged by clinical examination and blood gas analysis.

Three conditions were tested on each patient: (1) the use of a heated humidifier (control condition); (2) the use of a hygroscopic heat and moisture exchanger without a filtering function (HME); (3) the use of a combined hygroscopic heat and moisture exchanger and mechanical filter (HMEF). The heated humidifier was a blow-by apparatus (Fisher & Paykel, Panmure, New Zealand), composed of a disposable humidification chamber (MR 310) and an electric heater (MR 450), and set to deliver gas at between 32 and 34 °C. HME was a Umid-Vent 2S Flex (Gibeck Respiration, Upplands Väsby, Sweden). HMEF was a Hygroster (DAR, Mirandola, Italy). The heated humidifier was filled with water and placed into the inspiratory limb. The artificial noses (by this term we refer to both HME and to HMEF) were placed distally to the Y piece in the circuit, after removal of the heated humidifier. For the connection to the endotracheal tube, both HMEF and the heated humidifier required an additional flexible tube with an angular joint (Mod. 331/5389, DAR), while HME, which included a flexible tube as an integral part, was supplemented only with an angular joint (Mod. 606/5171, DAR). Therefore, the following assemblies were used: (1) flexible tube plus angular joint, with the heated humidifier in the inspiratory limb of the circuit: (2) HME with flexible tube, plus angular joint; (3) HMEF, plus flexible tube including angular joint. The additional internal volume and the additional resistance of each assembly are listed in Table 1, together with the resistance of the heated humidifier. Volumes and resistances were measured in vitro for one sample of each assembly. Resistance data were obtained at a constant flow of 1 l/s of water vapour-saturated air, after 20 min of flow, an interval that enabled a good saturation of the artificial noses.

Table 1 Internal volume and resistance of one sample of each assembly used for the study

	Volume (ml)	Resistance (cmH_2O/l per s)
Heated humidifier (control)	–	0.50
Flexible tube with angular joint (control)	23	1.36
HME with flexible tube plus angular joint	60	1.57
HMEF plus flexible tube with angular joint	100	2.86

We wanted to stabilize the respiratory activity of each patient throughout the study. Accordingly, we adapted the PSV level in order to achieve a constant $P_{0.1}$, an index of respiratory drive. Given the good relationship between $P_{0.1}$ and patient inspiratory work of breathing during PSV [12–15], constancy of $P_{0.1}$ should correspond to constancy of patient work. A target for $P_{0.1}$ was defined for each patient, and the PSV level was increased whenever actual $P_{0.1}$ rose above the target and decreased whenever actual $P_{0.1}$ fell below the target. In other words, external mechanical support was increased in response to an increase in respiratory drive (possibly due to a rise in the mechanical load of ventilation) and was decreased in response to a decrease in drive (possibly due to a reduction in mechanical load).

The task of adapting PSV was fulfilled by an automatic system, which worked as a $P_{0.1}$ controller [16]. The basis for this system is breath-by-breath monitoring of $P_{0.1}$ [17, 18]. The controller operates closed-loop regulation of the PSV level on the basis of a comparison between actual $P_{0.1}$ and a user-set target for $P_{0.1}$. The PSV level is increased when actual $P_{0.1}$ is above the target and decreased when actual $P_{0.1}$ is below the target.

The $P_{0.1}$ controller had been previously tested and had proved its ability both to bring $P_{0.1}$ to a given target and to maintain $P_{0.1}$ around that target [16]. In the present study, the $P_{0.1}$ target was individually set for each patient and averaged 2.1 ± 0.6 cmH_2O (range 1.2–3.2 cmH_2O). The $P_{0.1}$ target corresponded to the basal $P_{0.1}$ value that the patients exhibited during conventional PSV. To determine this basal $P_{0.1}$ value, each patient was measured for trend value in $P_{0.1}$ over the 20 min immediately before the beginning of the study, while ventilation was still performed in conven-

tional PSV, with the pressure support level chosen by the physician in charge in accordance with clinical evaluation. A heated humidifier (Fisher & Paykel) was used during the evaluation of the basal $P_{0.1}$ value.

Patients were connected to a modified, computer controllable AMADEUS ventilator (Hamilton Medical AG, Rhäzüns, Switzerland) and studied in a semirecumbent position. The closed-loop control algorithm for $P_{0.1}$ was implemented on a Macintosh SE that was connected to the AMADEUS ventilator and to a PC-based lung function analyzer. Airflow, airway pressure, instantaneous CO_2 concentration, and esophageal pressure signals were measured according to standard techniques. We have described elsewhere the measurement set-up [19, 20] as well as the principles for breath-by-breath calculation of lung function indices [21].

Inspiratory oxygen concentration ($50 \pm 8\%$) and positive end-expiratory pressure (PEEP) level (6 ± 1 cmH_2O) were left unchanged throughout the study.

In order to minimize possible order effects and carryover effects, the three conditions (control, HME, and HMEF) were tested in random order and all measurements were performed in steady state, as assessed from observation of trend values in lung function indices. A steady state was always achieved within 25 min in each condition. At this time, a blood sample for gas analysis was obtained, and data recording of lung function indices was started on the subsequent 160 consecutive cycles. Furthermore, in five patients a simultaneous recording of airflow, airway pressure, and esophageal pressure was obtained for off-line computation of work of breathing. The study protocol was approved by our Institutional Review Board, and informed consent was obtained from each patient or next of kin.

All values were expressed as means ± SD. Comparisons between groups were undertaken with two-way analysis of variance (ANOVA). A $p < 0.05$ level was considered significant.

Calculations

Dead space

From the flow and CO_2 signals, a CO_2 versus volume curve was constructed to determine the series dead space [21, 22]. The physiological dead space, comprising series dead space and alveolar dead space, was calculated as follows [22]:

$$\text{Physiological dead space} = \text{Exhaled tidal volume} \times \left(1 - \frac{PECO_2}{PaCO_2}\right)$$

where $PECO_2$ was the mean expired partial CO_2 pressure in exhaled volume. Dead space ventilation was calculated as the product of physiological dead space and respiratory rate. Alveolar volume was calculated as exhaled tidal volume minus physiological dead space. Alveolar ventilation was obtained as the product of alveolar volume and respiratory rate [22].

Respiratory mechanics measurements

Airway resistance and lung compliance were calculated by the least squares fitting method applied to the transpulmonary pressure [19, 23]. In our measurements, airway resistance was referred to the whole airway distal to the sensor head, i.e., it included the resistance of the endotracheal tube, the angular joint, the flexible tube, and, when used, the artificial nose. The dynamic intrinsic PEEP (PEEPi) was measured as the amount of negative deflection in esophageal pressure preceding the start of the inspiratory flow [24, 25]. Each value for airway resistance, lung compliance, and dynamic PEEPi was an average of five measurements.

Work of breathing. Patient inspiratory work per breath (i.e., the work performed by the patient to inflate both the lung and the chest wall) was determined according to the standard procedure, as previously described [20]. Chest wall compliance was estimated equal to 4% of theoretical vital capacity [26]. Ventilator inspiratory work per breath was calculated from the area enclosed between the inspiratory part of the airway pressure versus tidal volume loop, and the PEEP level. Total inspiratory work per breath was calculated as the sum of patient and ventilator components. Each value was the average of the measurements performed on five consecutive respiratory cycles. Patient, ventilator, and total work were indexed as work per liter and work per minute.

$P_{0.1}$. $P_{0.1}$ was calculated by extrapolation to 0.1 s of the airway pressure drop observed during the short no-flow inspiratory occlusion phase imposed by the pressure-triggered demand valve of the ventilator [17, 18].

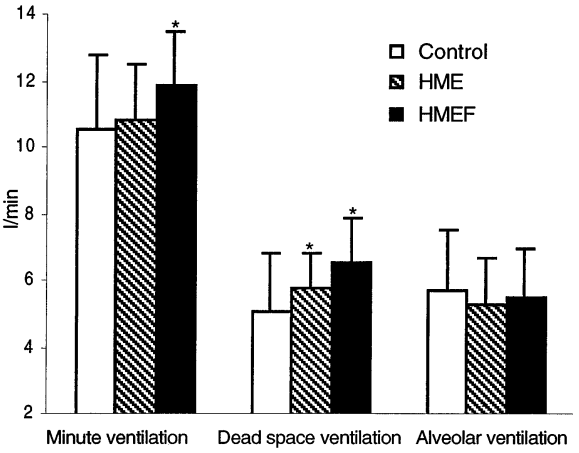

Fig. 1 Minute ventilation and its division between dead space ventilation and alveolar ventilation at steady state in three conditions: control, HME, and HMEF application. Values are mean ± SD. ANOVA: $p < 0.01$ for minute ventilation, $p < 0.005$ for dead space ventilation, NS for alveolar ventilation. * $p < 0.05$ vs control. *HME* heat and moisture exchanger, *HMEF* combined HME and filter

Results

Total minute ventilation tended to increase with the use of artificial noses. Compared to the control condition value of 10.6 ± 2.3 l/min, minute ventilation averaged 10.9 ± 1.6 l/min with HME and 11.9 ± 1.6 l/min with HMEF. The increase in minute ventilation was significant with HMEF. Figure 1 shows that there were no differences in alveolar ventilation between the three different conditions; the increase in minute ventilation observed with the artificial noses was due to increased dead space ventilation. This latter result is explained by the measurements for dead space and ventilatory pattern

Table 2 Respiratory variables at steady state in three conditions: control, HME and HMEF. Values are mean ± SD (*PEEP* = positive end-expiratory pressure, *PEEPi* dynamic intrinsic PEEP, P_{aw} airway pressure, PaO_2 partial pressure of oxygen in arterial blood, $PaCO_2$ partial pressure of CO_2 in arterial blood)

	Control	HME	HMEF	p [a]
Series dead space (ml)	108 ± 29	146 ± 26*	188 ± 24*	< 0.0001
Physiological dead space (ml)	195 ± 99	227 ± 78*	256 ± 72*	< 0.0001
Alveolar volume (ml)	203 ± 62	196 ± 53	207 ± 67	0.83
Tidal volume (ml)	398 ± 111	423 ± 96	463 ± 98*	< 0.025
Respiratory rate (cycles/min)	27 ± 6	27 ± 6	26 ± 4	0.57
$P_{0.1}$ (cmH_2O)	2.2 ± 0.6	2.1 ± 0.6	2.2 ± 0.6	0.62
Pressure support ventilation level above PEEP (cmH_2O)	12.8 ± 6.4	14.8 ± 5.4*	17.6 ± 5.6*	< 0.001
P_{aw}, peak (cmH_2O)	21.4 ± 5.4	23.3 ± 4.5	26.3 ± 4.7*	< 0.001
P_{aw}, mean (cmH_2O)	12.5 ± 1.7	13.4 ± 1.1	14.6 ± 1.5*	< 0.001
pH	7.47 ± 0.03	7.46 ± 0.03	7.48 ± 0.03	0.3
PaO_2 (mm Hg) (kPa)	118 ± 36 (16.1 ± 4.9)	132 ± 34* (17.9 ± 4.6)	139 ± 38* (18.9 ± 5.2)	< 0.01
$PaCO_2$ (mm Hg) (kPa)	40 ± 11 (5.4 ± 1.5)	41 ± 11 (5.5 ± 1.4)	40 ± 10 (5.4 ± 1.3)	0.43
Airway resistance (cmH_2O/l per s)	10.4 ± 4.1	12 ± 4.6*	13.6 ± 5.4*	< 0.001
Lung compliance (ml/cmH_2O)	34 ± 8	35 ± 11	33 ± 11	0.73
Dynamic PEEPi (cmH_2O)	0.9 ± 0.5	1.3 ± 0.4	1.6 ± 0.9*	< 0.05

[a] ANOVA
* $p < 0.05$ compared with control values

which are summarized in Table 2. The addition of the artificial noses to the artificial airway resulted in increases in series dead space and physiological dead space. Alveolar volume did not change, while tidal volume tended to increase, especially with HMEF. We found no difference in respiratory rate between the three conditions.

As shown in Table 2, the use of artificial noses was associated with a significant increase in airway resistance. A mild increase in dynamic PEEPi was found with HMEF. Over the study, there were no changes in lung compliance.

Figure 2 shows total inspiratory work per minute and its division between patient and ventilator for each of the three conditions studied. The inspiratory work tended to increase with the use of artificial noses. Compared to the control condition value of 13.6 ± 8.6 joule/min, total inspiratory work per minute averaged 19.2 ± 9.1 joule/min with HME and 22.3 ± 9.9 joule/min with HMEF. The increase in total inspiratory work per minute was significant with HMEF. The higher work associated with the artificial noses was provided entirely by the ventilator: the use of the artificial noses was associated with increases in ventilator inspiratory work, while the work performed by the patient was not affected. Likewise, $P_{0.1}$ remained constant in the three conditions, as shown in Table 2. The higher respiratory load associated with the artificial noses and expressed by our values for total inspiratory work was compensated for by the $P_{0.1}$ controller. Compared to the control condition, the PSV level applied by the $P_{0.1}$ controller was higher with HME and further increased with HMEF. As a result, the use of HMEF was associated

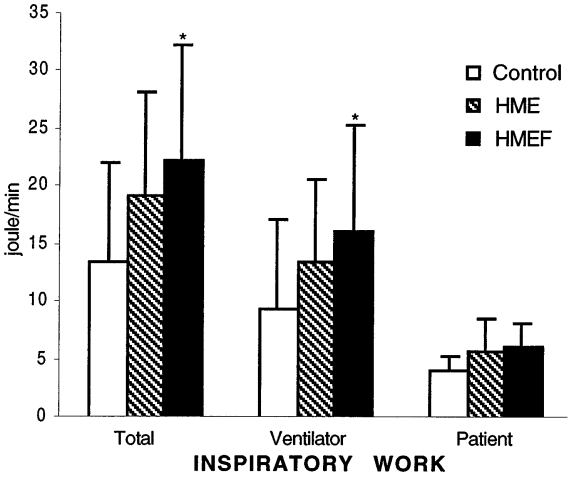

Fig. 2 Total inspiratory work in joule/min and its division between ventilator and patient at steady state in three conditions: control, HME, and HMEF application. Values are mean ± SD. ANOVA: $p < 0.05$ for total work, $p < 0.05$ for ventilator work, NS for patient work. * $p < 0.05$ vs control. *HME* and *HMEF* as in Fig. 1

with the highest values for peak airway pressure and mean airway pressure. Figure 3 provides an example of the $P_{0.1}$ controller response to the application of the three conditions in a representative patient.

Blood gas analysis data in all conditions investigated are listed in Table 2. Constancy of alveolar ventilation between the three given conditions resulted in constancy of the partial pressure of carbon dioxide in arterial blood ($PaCO_2$) and pH. The use of the artificial no-

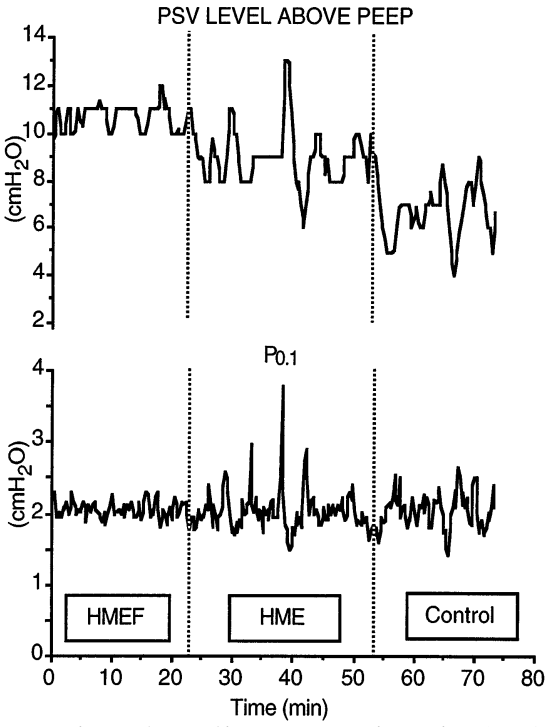

Fig. 3 Experimental record in a representative patient for PSV level above PEEP *top panel* and $P_{0.1}$ *bottom panel,* during sequential application of HMEF, HME, and no artificial nose (control). Constancy of $P_{0.1}$ was maintained by the $P_{0.1}$ controller, which decreased the average PSV level in the conditions of lower inspiratory load, HME, and control. As shown by the continuous changes in PSV level, the $P_{0.1}$ controller was continuously working in order to maintain the $P_{0.1}$ target, which was set at 2 cmH_2O. The positive spike of $P_{0.1}$ in the middle of the HME period provides an example of the efficiency of the $P_{0.1}$ controller. The sharp rise in $P_{0.1}$ was immediately counteracted by a temporary increase in PSV level. *HME* and *HMEF* as in Fig. 1

ses was associated with a slight but significant increase in the partial pressure of oxygen in arterial blood (PaO_2); the change did not differ significantly between HME and HMEF.

Discussion

The main finding of our study is that the use of an artificial nose may lead to a relevant increase in total inspiratory work of breathing. The particular ventilation mode applied in the study stabilized the spontaneous inspiratory activity of our patients, so that all the energy required to compensate for the artificial nose additional inspiratory load was provided by the mechanical ventilator, which increased the PSV level during the artificial nose trials.

The present study shows that these devices increase flow resistance, impose an increase in ventilation requirements, and may lead to dynamic pulmonary hyperinflation. An evident effect of the artificial noses on flow resistance is underlined by our data on airway resistance. As defined in Patients and Methods, our measurements for airway resistance included the resistance of the artificial nose. Our finding of a significant increase in airway resistance during both the artificial nose trials confirms that the resistance of these devices increases the inspiratory workload. The increases in airway resistance were even slightly higher than we could have expected from the resistance measured in vitro for each assembly.

However, the increase in inspiratory resistance due to the artificial nose was moderate and insufficient to explain the whole inspiratory workload imposed by these devices. Indeed, the use of HMEF was associated with an increase in total inspiratory work of about 60 % of the value observed in the control condition, while airway resistance increased just by 30 %. An increase in minute ventilation during artificial nose use has been recently reported in patients receiving PSV [10]. Our study confirms this effect as a major mechanism of increased inspiratory workload and provides evidence that the increase in minute ventilation observed during artificial nose use represents the compensation for the additional dead space of the humidifying devices. The increases in series dead space that we observed with the application of artificial noses corresponded to the difference in volume of each given assembly; these differences are deducible from the volumes listed in Table 1. The addition of an artificial nose to the ventilator circuit significantly increases the volume of the artificial airway, and thus the dead space. In our study the increase in dead space ventilation resulting from the use of artificial noses was associated with a corresponding increase in minute ventilation, which enabled constancy of alveolar ventilation, and hence of $PaCO_2$ and pH. It is evident that the need for increased ventilation that was imposed by the artificial noses represented an additional inspiratory workload.

The third mechanism by which artificial noses may increase the inspiratory load of respiration is represented by dynamic pulmonary hyperinflation. This effect is demonstrated in our study by the slight, but significant, increase in dynamic PEEPi that we found when HMEF was used. This finding is in contrast with the result of a previous study [9]. Unlike our study, that study was performed on COPD patients, which provides an explanation for the different result. An artificial nose promotes dynamic pulmonary hyperinflation by working as an external resistor opposed to the expiratory flow. In COPD patients, dynamic pulmonary hyperinflation is mainly dependent on intrinsic flow limitation due to expiratory bronchial collapse. It is likely that in COPD patients the addition of an external resistor opposes bronchial collapse and decreases intrinsic flow

limitation, so that the overall result may be no change in PEEPi. In contrast, in non-COPD patients, with no bronchial collapse, an external resistor necessarily increases the expiratory time constant of the system, and hence increases PEEPi.

In our study, the additional inspiratory workload imposed by the artificial noses was compensated for by the mechanical ventilator, which automatically increased the PSV level. Evidently, because of the above analyzed mechanisms (increased inspiratory resistance, increased ventilation requirements, and increased dynamic PEEPi), the use of an artificial nose potentially had a stimulating effect on the respiratory center. By automatically promoting an increase in PSV level, our closed-loop control of $P_{0.1}$ was able to offset this potential increase in patient inspiratory activity. Consequently, the inspiratory work performed by our patients remained stable. This result confirms the effectiveness of our $P_{0.1}$ control system in stabilizing the respiratory drive at a desired level [16], and underlines that the maintenance of $P_{0.1}$ constancy may result in constancy of patient inspiratory work.

During conventional mechanical ventilation, without feedback regulations, the unfavorable mechanical effects resulting from the addition of an artificial nose to the ventilator circuit can only be compensated for with a manual adjustment of ventilator settings. In other words, we should increase the inspiratory pressure level during pressure control ventilation and PSV, while during volume control ventilation we should increase the minute ventilation setting. In our study the unfavorable mechanical effects of the artificial noses were counterbalanced by increases in PSV level. As a result, tidal volume, peak airway pressure, and mean airway pressure increased, especially with HMEF.

The need for higher pressures and higher volumes which is associated with the use of artificial noses appears to conflict with current trends in mechanical ventilation [27]. There is general agreement that measures aimed at limiting lung exposure to high pressure and volume should be implemented whenever possible. These measures include any strategy that enables a reduction in the volume of the artificial airway. A radical but not broadly applicable solution for this target is represented by the high frequency ventilation and the endotracheal gas insufflation techniques [28, 29]. A simpler approach in current practice is to evaluate carefully the real need in each case for any ventilator circuit component which actually increases the apparatus dead space, like an artificial nose.

Our data concerning gas exchange suggest the hypothesis that use of an artificial nose may be associated with an improvement in arterial oxygenation. We can explain this result by considering that artificial nose use was associated with an increase in tidal volume [30], in airway pressure regimen, and in end-expiratory lung volume, this third effect being due to increased dynamic pulmonary hyperinflation. Whatever the explanation, it seems that control of oxygenation is more sensibly obtained by the adjustment of PEEP level and not by the use of an artificial nose with the consequent need for increases in applied pressure and in ventilation volumes.

The unfavorable mechanical effects of artificial noses add to the well known unfavorable mechanical effects of the endotracheal tube and ventilator demand valves [31]. When the ability of a patient to be weaned from mechanical ventilation is evaluated, it is important to take into account not only the tube and the ventilator, but also the additional workload and the increased need for ventilation that is imposed by an artificial nose. Lack of consideration of the mechanical effects of all these elements might lead clinicians to classify wrongly as ventilator dependent patients those who could actually be weaned [32].

When clinicians choose the artificial nose to be used in the ICU, they should carefully evaluate the available devices from the viewpoint of resistance and volume. Minimizing the volume and the resistance of artificial noses probably conflicts with the need to combine the filtering with the humidifying functions. Comparison of the results for the two artificial noses used in the present study shows that the less unfavorable mechanical effects were caused by HME, which is a low-volume device, without any antimicrobial filtering function. Presently, there is no clear evidence that either simple artificial noses or noses combined with filters decrease the incidence of ventilator-associated pneumonia, especially when we consider that, in the ICU patient, colonization and infection of the airways follow very complex pathways [3, 5, 33]. On the other hand, the possible anti-infective action of artificial noses might simply depend on the fact that these devices considerably reduce condensate accumulation in the ventilator circuit. Therefore, a reasonable compromise could be to forego the filtering function, a choice that enables a reduction in the volume and resistance of the device and hence a reduction in its unfavorable mechanical effects.

On the basis of our results, we draw the following conclusions: (1) Artificial noses may cause a clinically significant loss in the efficiency of ventilatory support. This loss results from increased inspiratory resistance, increased dead space ventilation, and increased dynamic hyperinflation. Therefore, artificial noses should be carefully evaluated from the viewpoint of both resistance and volume. (2) Artificial noses, by offering several practical advantages, remain an attractive alternative to heated humidifiers. In current practice, we recommend that when choosing an artificial nose, clinicians should give preference to low-volume and low-resistance devices. In this circumstance, a combined filtering function is optional, whereas the adequacy of humidification remains an absolute priority.

References

1. Marini JJ (1987) The role of the inspiratory circuit in the work of breathing during mechanical ventilation. Respir Care 32: 419–427
2. Dennison FH, Taft AA, Mishoe SC, Hooker LL, Eatherley SB, Beckham RW (1989) Analysis of resistance to gas flow in nine adult ventilator circuits. Chest 96: 1374–1379
3. Roustan JP, Kienlen J, Aubas P, Aubas S, du Cailar J (1992) Comparison of hydrophobic heat and moisture exchangers with heated humidifier during prolonged mechanical ventilation. Intensive Care Med 18: 97–100
4. Misset B, Escudier B, Rivara D, Leclercq B, Nitemberg G (1991) Heat and moisture exchangers vs heated humidifiers during long-term mechanical ventilation. Chest 100: 160–163
5. Branson RD, Davis K, Campbell RS, Johnson DJ, Porembka DT (1993) Humidification in the intensive care unit. Prospective study of a new protocol utilizing heated humidification and a hygroscopic condenser humidifier. Chest 104: 1800–1805
6. Mebius C (1992) Heat and moisture exchangers with bacterial filters: a laboratory evaluation. Acta Anaesthesiol Scand 36: 572–576
7. Ploysongsang Y, Branson R, Rashkin MC, Hurst JM (1988) Pressure flow characteristics of commonly used heat-moisture exchangers. Am J Respir Crit Care Med 138: 675–678
8. Chiaranda M, Verona L, Pinamonti O, Dominioni L, Minoja G, Conti G (1993) Use of heat and moisture exchanging (HME) filters in mechanically ventilated ICU patients: influence on airway flow resistance. Intensive Care Med 19: 462–466
9. Conti G, De Blasi RA, Rocco M, Pelaia P, Antonelli M, Bufi M, Mattia C, Gasparetto A (1990) Effects of heat-moisture exchangers on dynamic hyperinflation of mechanically ventilated COPD patients. Intensive Care Med 16: 441–443
10. Pelosi P, Solca M, Ravagnan I, Tubiolo D, Ferrario L, Gattinoni L (1996) Effects of heat and moisture exchangers on minute ventilation, ventilatory drive, and work of breathing during pressure support ventilation and acute respiratory failure. Crit Care Med 24: 1184–1188
11. American Thoracic Society (1987) Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD). Am J Respir Crit Care Med 136: 225–243
12. Galbusera C, Iotti G, Palo A, Olivei M, Brunner JX, Emmi V, Sala Gallini G, Braschi A (1993) Relationship between $P_{0.1}$ and inspiratory work of breathing during pressure support ventilation (PSV). Am J Respir Crit Care Med 147: A876
13. Foti G, Cereda M, Banfi G, Pelosi P, D'Andrea L, Pesenti A (1993) Simple estimate of patient inspiratory effort (PE) at different levels of pressure support (PS). Am J Respir Crit Care Med 147: A876
14. Alberti A, Gallo F, Fongaro A, Valenti S, Rossi A (1995) $P_{0.1}$ is a useful parameter in setting the level of pressure support ventilation. Intensive Care Med 21: 547–553
15. Berger K, Sorkin I, Norman R, Rapoport D, Goldring R (1996) Mechanism of relief of tachypnea during pressure support ventilation. Chest 109: 1320–1327
16. Iotti GA, Brunner JX, Braschi A, Laubscher T, Olivei MC, Palo A, Galbusera C, Comelli A (1996) Closed-loop control of $P_{0.1}$ applied to pressure support ventilation: algorithm and application in intubated patients. Crit Care Med 24: 771–779
17. Olivei M, Iotti G, Brunner JX, Palo A, Mencherini S, Galbusera C, Bobbio Pallavicini F, Braschi A (1992) Automatic method for continuous evaluation of $P_{0.1}$ on an occlusion phase shorter than 100 msec. Intensive Care Med 18 [Suppl 2]: A258
18. Palo A, Olivei M, Iotti G, Brunner J, Galbusera C, Braschi A (1992) Continuous measurement of $P_{0.1}$ during mechanical ventilation. Validation and clinical applications. Acta Anaesthesiol Ital 43: 382–392
19. Iotti GA, Braschi A, Brunner JX, Smits T, Olivei M, Palo A, Veronesi R (1995) Respiratory mechanics by least squares fitting in mechanically ventilated patients: applications during paralysis and during pressure support ventilation. Intensive Care Med 21: 406–413
20. Iotti GA, Braschi A, Brunner JX, Olivei M, Palo A (1995) Noninvasive evaluation of instantaneous total mechanical activity of the respiratory muscles during pressure support ventilation. Chest 108: 208–215
21. Brunner JX, Wolff G (1988) Pulmonary function indices in critical care patients. Springer, Berlin Heidelberg, pp 18–58
22. Fletcher R (1985) Dead space, invasive and non-invasive. Br J Anaesth 57: 245–249
23. Uhl RR, Lewis FJ (1974) Digital computer calculation of human pulmonary mechanics using a least square fit technique. Comput Biomed Res 7: 489–495
24. Haluszka J, Chartrand DA, Grassino AE, Milic Emili J (1990) Intrinsic PEEP and arterial PCO_2 in stable patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 140: 1194–1197
25. Begin P, Grassino A (1991) Inspiratory muscle dysfunction and chronic hypercapnia in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 143: 905–912
26. Agostoni E, Mead J (1964) Statics of the respiratory system. In: Fenn WO, Rahn H (eds) Handbook of physiology, sect 3, respiration, vol 1. American Physiological Society, Washington, DC, pp 387–409
27. Slutsky AS (1994) Consensus Conference on Mechanical Ventilation – January 28–30, 1993 at Northbrook, Illinois, USA. Part I. Intensive Care Med 20: 64–79
28. Nakos G, Zakinthinos S, Kotanidou A, Tsagaris H, Roussos C (1994) Tracheal gas insufflation reduces the tidal volume while $PaCO_2$ is maintained constant. Intensive Care Med 20: 407–413
29. Johnson BT, Similowski T, Levy P, Viires N, Pariente R (1990) Expiratory flushing of airways: a method to reduce dead space ventilation. Eur Respir J 3: 1202–1205
30. Blanch L, Fernandez R, Vallés J, Solé J, Roussos C, Artigas A (1994) Effect of two tidal volumes on oxygenation and respiratory system mechanics during the early stage of adult respiratory distress syndrome. J Crit Care 9: 151–158
31. Brochard L, Pluskwa F, Lemaire F (1989) Improved efficacy of spontaneous breathing with inspiratory pressure support. Am J Respir Crit Care Med 136: 411–415
32. Kirton O, DeHaven CB, Morgan JP, Windsor J, Civetta J (1995) Elevated imposed work of breathing masquerading as ventilator weaning intolerance. Chest 108: 1021–1025
33. Martin C, Perrin G, Gevaudan MJ, Saux P, Gouin F (1990) Heat and moisture exchangers and vaporizing humidifiers in the intensive care unit. Chest 97: 144–149