Mechanical effects of heat–moisture exchangers in ventilated patients

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Although they represent a valuable alternative to heated humidifiers, artificial noses have unfavourable mechanical effects. Most important of these is the increase in dead space, with consequent increase in the ventilation requirement. Also, artificial noses increase the inspiratory and expiratory resistance of the apparatus, and may mildly increase intrinsic positive end-expiratory pressure. The significance of these effects depends on the design and function of the artificial nose. The pure humidifying function results in just a moderate increase in dead space and resistance of the apparatus, whereas the combination of a filtering function with the humidifying function may critically increase the volume and the resistance of the artificial nose, especially when a mechanical filter is used. The increase in the inspiratory load of ventilation that is imposed by artificial noses, which is particularly significant for the combined heat–moisture exchanger filters, should be compensated for by an increase either in ventilator output or in patient's work of breathing. Although both approaches can be tolerated by most patients, some exceptions should be considered. The increased pressure and volume that are required to compensate for the artificial nose application increase the risk of barotrauma and volutrauma in those patients who have the most severe alterations in respiratory mechanics. Moreover, those patients who have very limited respiratory reserve may not be able to compensate for the inspiratory work imposed by an artificial nose. When we choose an artificial nose, we should take into account the volume and resistance of the available devices. We should also consider the mechanical effects of the artificial noses when setting mechanical ventilation and when assessing a patient's ability to breathe spontaneously.

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Introduction

In intubated patients the humidification and warming of inspired gases requires the addition to the ventilator circuit of a heated humidifier, or of an artificial nose [1,2]. Artificial noses were introduced as disposable devices more than 10 years ago, and since then they have been used in anaesthesia. In the most recent years the use of artificial noses has been increasingly extended into the intensive care unit (ICU) setting for patients undergoing long-term mechanical ventilation. The market has increased considerably in parallel with this widespread use, and presently offers a wide range of artificial noses. More than 30 models (for adult patients) are now available on the Italian market alone, making the choice of artificial nose difficult for the clinician. Indeed, the choice of artificial nose requires consideration of different parameters, which often have conflicting merits.

Most studies [3–6] have compared artificial noses and heated humidifiers in terms of humidification efficiency and antimicrobial properties. Only in recent years has attention been paid to the mechanical effects of artificial noses. Although few clinical studies on this aspect are available [7–10], their results are nevertheless consistent and in agreement. The present review examines the available data on the mechanical effects of artificial noses, in order to provide the clinician with some help in making a choice of artificial nose.

It is first necessary to consider how artificial noses modify the geometry of the ventilation circuit. These devices are placed distally to the Y-piece in the circuit, in line with the endotracheal tube. Unlike artificial noses, heated humidifiers are inserted in the inspiratory pathway of the ventilation circuit. Hence, artificial noses are not just part of the ventilation circuit, but are also part of the artificial airway. If we remove a heated humidifier and add an artificial nose, we can expect the following mechanical changes in the entire ventilation circuit: an increase in the apparatus dead space; no relevant change in the inspiratory resistance; an increase in the expiratory resistance; and a decrease in the compressible volume.

ICU = intensive care unit; PEEP = positive end-expiratory pressure; HME = heat–moisture exchanger; ARDS = adult respiratory distress syndrome; COPD = chronic obstructive pulmonary disease.

Therefore, on one hand artificial noses are expected to increase the ventilation requirement, through an increase in dead space ventilation. On the other hand, the increase in the expiratory resistance of the apparatus represents a factor that potentially leads to dynamic pulmonary hyperinflation and intrinsic positive end-expiratory pressure (PEEP), which in turn represents an additional elastic load for inspiration.

The importance of these mechanical effects depends on the design and on the functions of the artificial nose. From the standpoint of functional properties, we can schematically distinguish three kinds of artificial noses. The filters are low-efficiency humidifiers and they are used as barriers against the passage of bacteria and viruses, rather than for humidification purposes. Heat–moisture exchangers (HMEs) serve only as humidifiers. Combined HMEs and filters represent the most recent group of artificial noses, and combine humidification and filtration properties. In general, among the artificial noses, the HME have the lowest volume and lowest resistance, whereas the combined HMEs and filters have the highest volume and highest resistance.

The mechanical effects of artificial noses, regardless of the type of artificial nose, have different clinical implications, and hence are discussed separately.

Increase in ventilation requirement

Artificial noses increase dead space by an amount equal to their internal volume. Internal volumes range rather widely, and are mainly dependent on the design of the artificial nose. In this regard, mechanical filters are usually made of a pleated membrane, which results in a remarkable increase in the volume of the device, in order to limit the increase in resistance. In general, HMEs without filtering function are the artificial noses that have the lowest internal volume. The artificial nose volume does not affect the humidification performance of the device, except in those that have very low values [11]. In the latter case the humidification performance is lower, but remains acceptable if low tidal volumes (not higher than 0.5 l) are used [11].

It should be recognized that all the pieces that are used for the connection of the artificial nose to the endotracheal tube contribute toward an increase in the dead space of the apparatus. For example, if we add unnecessary connecting pieces to a HME without filtering function (which is a low-volume artificial nose), we can easily reach the volume of a combined HME and filter (which is a high-volume artificial nose). Artificial noses that include a flexible tube as an integral part, serving both as a connector and as a humidifier, represent a possible way to avoid unnecessary increase in apparatus dead space.

An increase in dead space results in an increase in dead space ventilation. For a given level of minute ventilation, an increase in dead space ventilation implies a decrease in alveolar ventilation, and hence an increase in arterial carbon dioxide tension ($PaCO_2$). To prevent this latter effect, the increase in dead space ventilation due to the artificial nose must be compensated for by an increase in minute ventilation. Different studies [7–10] have indicated that patients ventilated in pressure support ventilation respond to the application of an artificial nose with a significant increase in minute ventilation, which can be higher than 1 l/min in the case of use of a combined HME and filter.

From a clinical standpoint, when we consider how to compensate for the additional ventilation requirement imposed by artificial noses we must distinguish the case of the paralysed patient from the case of the patient who maintains spontaneous respiratory activity and is assisted with a partial ventilatory support mode. In the paralysed patient, the compensation can only be performed by manual adjustments of the ventilator settings. Although not yet described in the literature, this approach can be simply deduced from the operation principles of mechanical ventilation. During volume-controlled ventilation we should increase the setting of tidal volume by a value equal to the nominal volume of the given artificial nose, or, alternatively, we should combine a smaller increase in tidal volume with an increase in the setting of respiratory rate. During pressure-controlled ventilation we should increase just the level of inspiratory pressure, or, alternatively, we should combine a smaller increase in inspiratory pressure with an increase in respiratory rate.

Whatever the ventilator adjustment used, the compensation for the additional dead space of the artificial noses results necessarily in higher tidal volume, higher peak airway pressure and higher mean airway pressure. These effects are clinically negligible in patients with normal or slightly injured lungs. They represent an additional risk of barotrauma and volutrauma in those patients with the most severe alterations of respiratory mechanics, however, such as those observed in severe asthma, adult respiratory distress syndrome (ARDS) and lung fibrosis. On the other hand, the alveolar hypoventilation that results from lack of compensation for the artificial nose dead space may be significant, especially in patients with the most severe alterations in respiratory mechanics, in whom the ventilatory treatment relies on low levels of minute ventilation. It has been observed [12] that the removal of a combined HME and filter, and the consequent reduction in dead space, allowed for a remarkable decrease in $PaCO_2$ in ARDS patients subjected to controlled hypoventilation.

During partial ventilatory support the additional ventilatory requirement due to the artificial nose can be compensated for either by the patient, or by an increase in the

mechanical support provided by the ventilator. In the former, the increase in minute ventilation depends on an increase in the respiratory work performed by the patient [7,9]. The contribution of respiratory rate and tidal volume to this compensatory increase in minute ventilation is variable [7–9], probably because of differences in patients' ventilatory reserves. The patients who have a wider ventilatory reserve are those who increase minute ventilation by raising either the tidal volume alone [7,9], or the tidal volume and the respiratory rate. On the contrary, patients who have a very limited ventilatory reserve are those who increase minute ventilation only by raising the respiratory rate [8]. This second group of patients may not be able to respond to the application of an artificial nose with an increase in minute ventilation sufficient to prevent an increase in $PaCO_2$. This situation is particularly likely in the presence of low tidal volumes, which in turn reflect a low ventilatory reserve [8].

In order to avoid an increase in patient respiratory work, we should compensate for the additional ventilatory requirement imposed by the artificial nose with an increase in the mechanical support. This compensation can be achieved in different ways, depending on the mode of partial ventilatory support in use, although generally it is sufficient to increase the inspiratory pressure support level [9,10]. Regardless of the ventilator settings that are adjusted, the final result is the exposure of the lungs to higher pressures and volumes. Some patients ventilated in partial support modes do not present severe alterations in lung mechanics, and can well tolerate the increased pressure and volume that are required to compensate for the application of an artificial nose. Therefore, in these patients, the need for higher pressures and volumes imposed by artificial noses does not represent an additional risk of barotrauma or volutrauma.

Increase in apparatus inspiratory resistance

The importance of the additional respiratory resistance imposed by the ventilator circuit has been well documented. Studies have demonstrated that excessive circuit resistance can prolong patient weaning and cause respiratory muscle fatigue [13,14]. Nunn [15] recommended an upper limit for external resistance to ventilation of about $7\,cmH_2O/l$ per s.

In vitro, the resistance of artificial noses presents a linear increase with increasing flow rate, and a slight increase with the duration of use [16–19]. Artificial noses are primarily low-resistance devices, although their resistance values range widely, from 0.7 to $3.8\,cmH_2O/l$ per s at an airflow of 60 l/min in dry state. *In vivo*, the resistance of artificial noses does not increase significantly after 24 h of clinical use, but does in rare cases in which the filter membrane is exposed to particularly abundant secretions [20–22]. This latter situation can be avoided, however, by correct positioning of the artificial nose, which should be placed in an upward orientation in the ventilator circuit [23].

When we assess the actual change in inspiratory resistance due to the application of an artificial nose, we must take into account the decrease in inspiratory resistance that results from the removal of the heated humidifier from the ventilator circuit. In practice, the net change in the inspiratory resistance corresponds to the difference between the resistance of the artificial nose and the resistance of the heated humidifier. Because the resistance values of artificial noses are not much higher than those of heated humidifiers, which are low-resistance devices, the application of an artificial nose results in no change, or in a just slight increase in the total inspiratory resistance of the apparatus [10,17,24].

The clinical implication is that the mild increase in inspiratory resistance imposed by an artificial nose just moderately decreases the efficiency of mechanical ventilation, and can be easily compensated. Indeed, in patients paralysed and subjected to volume-controlled mechanical ventilation, the ventilator directly guarantees the constancy of the set tidal volume. During volume-controlled ventilation the contribution of the artificial nose to the resistive load can simply be evaluated by comparing the difference between peak and plateau pressures with and without the artificial nose in place [25]. In patients subjected to pressure-controlled ventilation, or to pressure-support ventilation, the mild increase in inspiratory resistance due to an artificial nose can simply be compensated for by an increase in the inspiratory pressure delivered by the ventilator, or, alternatively, by a small increase in the inspiratory work performed the patient.

Of much greater importance is the increase in the inspiratory airway resistance that an artificial nose may indirectly produce in case of insufficient humidity output [26]. This can lead to the accumulation of viscous secretions on the internal wall of the endotracheal tube, especially during prolonged mechanical ventilation. The consequence is a significant increase in inspiratory resistance, and an increased risk of sudden endotracheal tube obstruction [3,6,26,27].

Increase in apparatus expiratory resistance and potential generation of intrinsic PEEP

Artificial noses, being placed on the artificial airway, increase the expiratory resistance. This effect does not affect the expiratory work, except when exhalation is active. However, the increase in expiratory resistance due to the artificial nose invariably results in a decrease in the speed of exhalation, a condition which increases the risk of dynamic pulmonary hyperinflation. This condition, which is commonly measured in terms of intrinsic PEEP, takes place when full exhalation is impeded by the start of

Table 1

Increases in the compressible volume, respiratory impendance components and total work of breathing associated with heated humidifiers and different artificial noses

		HME		
	Heated humidifier	Low volume	High volume	HME–filter
Apparatus compressible volume	+++	+/0	+	++
Apparatus deadspace	0	+	++	+++
Dead space ventilation	0	+	++	+++
Apparatus inspiratory resistance	+	+	+	++
Apparatus expiratory resistance	0	+	+	++
Intrinsic PEEP	0	0	0	+*
Total work of breathing	0	+/0	+	++

All the changes have been scored as follows: 0, negligible; +, mild; ++, moderate; +++, severe. *The increase in intrinsic positive end-expiratory pressure (PEEP) may be absent in chronic obstructive pulmonary disease patients with dynamic bronchial collapse. Note that this table describes the typical effects attributed to each humidifier category. Specific devices of any category may perform much worse or better than indicated. For example, in heat–moisture exchanger (HME)–filters, electrostatic rather than mechanical filtration may reduce all mechanical effects.

the next inhalation. The mechanism for intrinsic PEEP consists in an imbalance between the speed of exhalation (which is too low) and the duration of the expiratory phase of a cycle (which is too short). In particular, an artificial nose may generate an intrinsic PEEP by acting as an external resistor that slows exhalation.

The patient's clinical characteristics are an important factor influencing the potential of artificial noses to generate intrinsic PEEP. A mild increase in intrinsic PEEP has been found in two studies [9,10] in which artificial noses were applied to patients without evidence of chronic obstructive pulmonary disease (COPD). On the contrary, another study [28] reported no increase in intrinsic PEEP during use of artificial noses on COPD patients. To explain these different results, it must be observed that in COPD patients intrinsic PEEP 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 the intrinsic flow limitation, so that the overall result may be no change in pulmonary hyperinflation. In contrast, in non-COPD patients, who have no bronchial collapse, an external resistor necessarily increases the expiratory time constant of the system, and hence may increase dynamic pulmonary hyperinflation.

The slight increase in intrinsic PEEP caused by artificial noses may provide an explanation for the slight increase in arterial oxygen tension that has been observed during application of artificial noses [8,10]. This latter effect could better be explained by the increase in tidal volume that results from the increased need for ventilation imposed by the artificial nose, however.

Conclusion

The major unfavourable mechanical effect of artificial noses is the increase in dead space, with consequent increase in the ventilation requirement. Also, artificial noses increase the inspiratory and expiratory resistance of the apparatus, and may mildly increase intrinsic PEEP. All of these effects result in a significant increase in the inspiratory workload. Table 1 summarizes all of the mechanical effects that are typical of different artificial nose categories and of heated humidifiers.

The increase in the inspiratory workload caused by artificial noses should be compensated for by an increase either in ventilator output or in patient's work. Three studies [7,9,10] have evaluated the energy expenditure required to compensate for artificial noses *in vivo*. In one of these [10] the patient's respiratory activity was maintained constant during application of different artificial noses. This required a mean increase in the inspiratory pressure support of 2 cmH_2O during use of a HME without filtering function, and of 5 cmH_2O during use of a combined HME and filter. From the viewpoint of energy expenditure, the HME and combined HME and filter tested in that study required a mean increase in ventilator work of 5.6 and 8.7 J/min, respectively. Similar results have been found in another study [9], in which the workload associated with different models of combined HME and filter was entirely compensated for by the patients. In patients assisted with partial ventilatory support, the additional work required to compensate for the artificial noses can be estimated also by measuring $P_{0.1}$ [7,9], which is an index of respiratory drive, and has been shown to correlate well with the patient's respiratory work. Patients mechanically ventilated have been shown to respond to the application

of a combined HME and filter with a significant increase in $P_{0.1}$ [7,9].

It is likely that most patients are able to compensate for the increase in inspiratory work imposed by artificial noses. This is not the case for patients with very limited respiratory reserve, however. The unfavourable mechanical effects of artificial noses add to the well known unfavourable mechanical effects of the endotracheal tube and ventilator demand valves [29]. 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 of these elements might lead clinicians wrongly to classify some patients as ventilator-dependent who actually could be weaned [8,30].

On the other hand, if we wish to avoid an increase in patient's work when an artificial nose is used, we should set the ventilator to deliver higher pressures and volumes. The consequence is an increased risk of barotrauma and volutrauma in the patients with the most severe alterations of respiratory mechanics, such as those observed in severe asthma, ARDS and lung fibrosis. In these categories of patients the need for higher pressures and volumes appears to conflict with the current trends in mechanical ventilation. There is general agreement that measures aimed at limiting lung exposure to high pressures and volumes should be implemented, especially in patients with the most severe alterations in respiratory mechanics. In these cases, any strategy that enables a reduction in dead space may be helpful. Therefore, in current practice we should carefully evaluate the real need, in each case, for any ventilator circuit component that actually increases the dead space of the apparatus, like an artificial nose.

When clinicians choose the artificial nose to be used in the ICU context, they should take into account the volume and resistance of the available devices. The pure humidifying function is compatible with just a moderate increase in apparatus dead space and resistance. On the contrary, the combination of a filtering function with the humidifying function may critically increase the volume and the resistance of the artificial nose, especially when a mechanical filter is used. A study that compared the mechanical effects of a HME and of a combined HME and filter [10] has shown that lesser unfavourable mechanical effects were caused by the HME, which was a low-volume device, without any antimicrobial filtering function. Presently, there is no clear evidence that 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 [3–5,31–33]. 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 unfavourable mechanical effects.

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