The Ventilator Circuit and Ventilator-Associated Pneumonia

Richard D Branson MSc RRT FAARC

Historically, the relationship between the ventilator circuit and pulmonary infection was accepted as fact, without any scientific evidence. Hence the term, "ventilator"-associated pneumonia. Recent evidence, however, has demonstrated that the major sources of pneumonia in the ventilated patient are colonization of the gastrointestinal tract, with subsequent aspiration around the endotracheal tube cuff, and contamination by caregivers. In recent years, the relationship of respiratory care equipment to ventilator-associated pneumonia has been studied carefully. A number of clinical trials have demonstrated that routine changing of the ventilator circuit fails to impact the incidence of pneumonia in the ventilated patient. Additional studies evaluating the type of humidification device, type of suctioning device, and frequency of change of the devices have resulted in conflicting evidence. This paper reviews the role of the humidifier, ventilator circuit, and airway suctioning equipment on the pathogenesis and prevention of ventilator-associated pneumonia. *Key words: ventilator-associated pneumonia, mechanical ventilation, humidification, suctioning, ventilator circuit.* [Respir Care 2005;50(6):774–785. © 2005 Daedalus Enterprises]

Introduction

The term ventilator-associated pneumonia (VAP) implies some connection between the presence of the mechanical ventilator and the occurrence of pulmonary infection. This may be a historical connection, as early in the use of positive-pressure ventilation, circuits and humidifiers were nondisposable, cleaned in-house, and reused. The possibility of cross-infection from improperly cleaned equipment was clearly present. Several early reports suggested a link between ventilator circuits, respiratory care equipment, and infection.[1–4] Based on these reports, daily ventilator-circuit changes were the norm, with some institutions changing circuits at each 8-hour shift.

In recent years the etiology of VAP has become better understood and the role of respiratory care equipment has been studied in detail. This paper will discuss the ventilator circuit, humidifier, and related components, along with what role each has in development and or prevention of VAP.

Richard D Branson MSc RRT FAARC is affiliated with the Department of Surgery, University of Cincinnati, Cincinnati, Ohio.

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Correspondence: Richard D Branson MSc RRT FAARC, Department of Surgery, University of Cincinnati, 231 Albert Sabin Way, Cincinnati OH 45267-0558. E-mail: richard.branson@uc.edu.

The Ventilator Circuit

Daily, routine ventilator-circuit changes dominated clinical practice throughout the early use of positive-pressure ventilation. In 1982, Craven et al compared daily ventilator-circuit changes to every-48-hour circuit changes in adult patients.[5] The main end point of this study was not the incidence of VAP, but rather the frequency of positive cultures from inspiratory gas. In effect, these authors were determining the effect of time on ventilator circuit colonization. Positive circuit cultures were seen in 30% of cases in the every-24-hour circuit-change group and in 32% of the every-48-hour circuit-change group. While this study failed to determine the effects on VAP, it did bring to light the importance of these decisions on cost. Craven et al suggested that, if the 20 teaching hospitals in Boston switched from 24-hour to 48-hour circuit changes, a cost savings of $300,000 would be realized.

In 1986, Craven et al conducted a prospective randomized controlled trial of 24-hour versus 48-hour ventilator-circuit changes in 233 adult patients.[6] Using a clinical diagnosis of VAP where physicians making the VAP diagnosis were blinded to the study, they demonstrated a 50% reduction in the incidence of VAP with 48-hour circuit changes. Dreyfuss et al were the first to consider no circuit changes as a possible alternative to some routine frequency.[7] This small study of 63 patients found that there was no significant difference in the incidence of VAP between the 2 groups. Dreyfuss et al also used quantitative cultures to diagnose VAP—an important change from a diagnosis based on clinical criteria. While the results did not reach statistical significance, analysis of the data favors no-circuit-change versus 48-hour changes, with respect to the incidence of VAP.

Kollef et al conducted a prospective randomized trial of once-a-week circuit changes versus no ventilator-circuit changes in 345 adult patients, using blinded investigators to make the VAP diagnosis based on clinical criteria.[8] The incidence of VAP was 29% in the weekly-circuit-change group and 25% in the no-circuit-change group.

Long et al compared circuit changes 3 times a week to circuit changes once a week in a group of adult and neonatal patients.[9] The diagnosis was based on clinical criteria, but investigators making the diagnosis were not blinded. In this diverse group of 447 patients the incidence of VAP was 13% in the 3-times-a-week circuit-change group and 11% in the weekly-circuit-change group.

An analysis of these 4 randomized, prospective trials has been accomplished by Cook at el and by Hess et al, using a meta-analysis.[10,11] The key to success of the meta-analysis is the comparability of the individual study methods. These studies are detailed in Table 1, and the meta-analysis by Hess et al is shown in Figure 1. While there are some differences in the type of circuits, criteria for VAP diagnosis, type of humidifiers used, and variable circuit-change frequencies (48-h changes to no change), the overwhelming evidence appears to support less frequent ventilator-circuit changes. The analysis clearly suggests that, at worst, circuit changes only between patients does not increase the risk of VAP, compared to a routine change frequency.

Hess and others[11,12] also identified a group of studies that evaluate varying circuit-change frequencies in observational studies. These studies do not carry the same weight of evidence as the previously discussed prospective randomized trials, but are worthy of discussion. The first of these studies, conducted in 1978, is mainly of historical interest. Lareau et al studied every-8-hour circuit changes versus 24-hour circuit changes in 484 adult, pediatric, and neonatal patients.[13] The results of this study favored more frequent circuit changes, with the VAP rate in the 24-hour circuit-change group increasing to 11.8%, compared to 7.5% in the every-8-hour circuit-change group. The major limitation of this study and others in this group is the use of historical controls.

Hess et al compared every-48-hour circuit changes to once-a-week circuit changes in 3,423 adult patients and found no difference in the incidence of VAP (5.6 to 4.6%).[14] Analysis of their data, however, did demonstrate that while the difference was not statistically significant, the once-a-week circuit changes were associated with a reduced odds ratio (0.82, $p = 0.22$) of developing VAP, compared to 48-hour circuit changes. Additionally, Hess et al demonstrated that changing circuits at 7-day intervals resulted in a 76.6% ($111,530) reduction in the annual cost for materials and salaries. An important point in the analysis of these studies is comparing the circuit-change interval to the duration of ventilation. If most patients are ventilated for < 72 hours, a once-a-week circuit-change interval will not produce the cost savings seen when the mean duration of ventilation is greater.

In a small study, Thompson compared once-a-week to once-every-2-weeks circuit changes in a subacute care unit characterized by long-term ventilatory support.[15] In this trial, a 6-month period of weekly changes was evaluated, followed by a 6-month period of biweekly circuit changes. VAP was determined using clinical criteria. They found no difference in the incidence of VAP between groups (9.7% vs 11.1%). Kotilainen and Keroack compared every-3 day circuit changes to once-a-week circuit changes in an adult population of 234 patients. They found a reduction in VAP rate, from 9.1% to 6.2%.[16] Fink et al compared 48-hour circuit changes to once-a-month circuit changes over a 4-year period in nearly 500 patients, and demonstrated reduced VAP rates (3.5% vs 9.2%) with greater duration of use.[17] The duration of this study included the change from

Table 1. Summary of Randomized Controlled Trials Investigating the Relationship Between Ventilator Circuit Change Frequency and the Risk of Ventilator-Associated Pneumonia

Citation	Study Population	Blinding	VAP Diagnosis	Control Group	Treatment Group	Control Group		Treatment Group		Level	Relative Risk (95% CI)
						n	Pneumonia (%)	*n*	Pneumonia (%)		
Craven 1986[6]	Adult patients requiring mechanical ventilation > 48 h	VAP assessors	Clinical	Circuit changes every 24 h	Circuit changes every 48 h	106	29.2	127	14.2	1	0.48 (0.29, 0.82)
Dreyfuss 1991[7]	Adult patients requiring mechanical ventilation > 48 h	VAP assessors	Quantitative cultures	Circuit changes every 48 h	No circuit changes	35	31.4	28	28.5	1	0.91 (0.42, 1.95)
Kollef 1995[8]	Adult patients requiring mechanical ventilation > 5 d	VAP assessors	Clinical	Circuit changes every 7 d	No circuit changes	153	28.8	147	24.5	1	0.85 (0.58, 1.24)
Long 1996[9]	Neonatal and adult mechanically ventilated patients	None	Clinical	Circuit changes 3 times/wk	Circuit change 1/wk	213	12.7	234	11.1	2	0.88 (0.53, 1.45)
TOTAL						507	22.3	536	16.4		0.76 (0.57, 1.00)

VAP = ventilator-associated pneumonia
CI = confidence interval
(Adapted from Reference 11.)

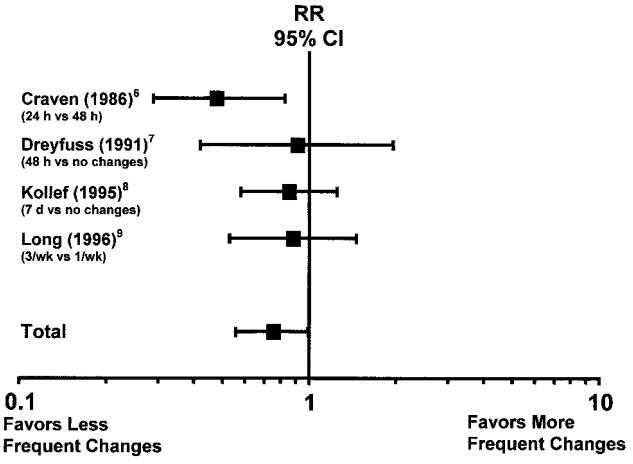

Fig. 1. Meta-analysis of randomized controlled trials investigating the relationship between ventilator-circuit-change frequency and the risk of ventilator-associated pneumonia. RR = relative risk. CI = confidence interval. (Adapted from Reference 11.)

traditional circuits to heated-wire circuits, with no apparent effect on VAP rates. This trial also evaluated costs and demonstrated substantial material and labor savings with the once-a-month circuit changes. Additionally, this paper was the first to demonstrate the additional savings provided by a heated-wire circuit, through virtue of water savings and elimination of condensate.

More recently Han et al[18] and Lien et al[19] compared 48-hour circuit changes with once-a-week circuit changes in 2001. Han et al demonstrated a reduction in VAP, from 9.2% to 3.5%, with once-a-week circuit changes. Lien et al studied VAP rates during a 2-year retrospective review with every-48-hour circuit changes and then prospectively evaluated VAP rates with weekly circuit changes. The diagnosis of VAP was based on clinical criteria. This study showed no difference in the VAP rate (2.9% vs 3.2%) between the 2 circuit-change frequencies. This paper includes more subjects (13,281) than all the other studies combined, and as such substantially influences the results of any meta-analysis performed. These studies were recently reviewed and subjected to meta-analysis by Hess et al.[11] Table 2 list these observational studies of ventilator-circuit changes, and Figure 2 depicts the meta-analysis.

Both Cook et al and Hess et al have recently reviewed this topic from an evidence-based approach.[10,11] Both groups conclude that while the study methods are quite variable, and the types of equipment, types of humidifiers, circuits, suction equipment, and diagnostic methods were

Table 2. Summary of Observational Studies Investigating the Relationship Between Ventilator Circuit Change Frequency and the Risk of Ventilator-Associated Pneumonia

Citation	Study Population	VAP Diagnosis	Control group	Treatment group	Control Group		Treatment Group		Level	Relative Risk (95% CI)
					n	Pneumonia (%)	*n*	Pneumonia (%)		
Lareau 1978[13]	Adult, pediatric, and neonatal mechanically ventilated patients	Clinical	Circuit changes at 8-h intervals	Circuit changes at 24-h intervals	213	7.5	271	11.8	4	1.57 (0.89, 2.79)
Hess 1995[14]	Adult mechanically ventilated patients	Clinical	Circuit changes at 2-d intervals	Circuit changes at 7-d intervals	1,708	5.6	1,715	4.6	4	0.83 (0.62, 1.11)
Thompson 1996[15]	Adult mechanically ventilated patients in a subacute care facility	Clinical	Circuit changes at 7-d intervals	Circuit changes at 14-d intervals	31	9.7	18	11.1	4	1.15 (0.21, 6,24)
Kotilainen 1997[16]	Adult mechanically ventilated patients	Clinical	Circuit changes at 3-d intervals	Circuit changes at 7-d intervals	88	9.1	146	6.2	4	0.68 (0.27, 1.69)
Fink 1998[17]	Adult mechanically ventilated patients	Clinical	Circuit changes at 2-d intervals	Circuit changes at 30-d intervals	336	10.7	157	6.4	4	0.59 (0.30, 1.17)
Han 2001[18]	Adult mechanically ventilated patients	Clinical	Circuit changes at 2-d intervals	Circuit changes at 7-d intervals	413	9.2	231	3.5	4	0.38 (0.18, 0.79)
Lien 2001[19]	Adult mechanically ventilated patients	Clinical	Circuit changes at 2-d intervals	Circuit changes at 7-d intervals	6,213	2.9	7,068	3.2	4	1.14 (0.94, 1.38)
TOTAL					9,002	4.1	9,606	3.8		0.87 (0.63, 1.18)

VAP = ventilator-associated pneumonia
CI = confidence interval
(Adapted from Reference 11.)

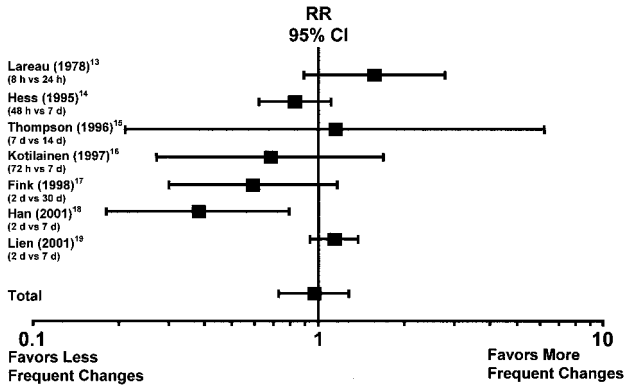

Fig. 2. Meta-analysis of observational studies investigating the relationship between ventilator-circuit-change frequency and the risk of ventilator-associated pneumonia. RR = relative risk. CI = confidence interval. (Adapted from Reference 11.)

not always clearly stated, there appears to be, at worst, no difference in VAP rates with prolonged use of the circuit. According to the meta-analysis by Hess et al, the 4 prospective randomized controlled trials favor less frequent ventilator-circuit changes, with a relative risk of 0.76 and 95% confidence interval of 0.57 to 1.00, which is a statistically significant ($p > 0.05$) difference. Data from the observational trials also favor less frequent ventilator-circuit changes, with a relative risk of 0.87 and a 95% confidence interval of 0.62 to 1.18. This difference is not statistically significant.

While there appears to be overwhelming evidence that lengthening circuit changes does not alter the incidence of VAP, there is no explanation why. Clearly, circuit colonization occurs rapidly with the source emanating from the patient.[1,2,5] A frequent explanation has been that less frequent circuit changes prevent introduction of nosocomial organisms. The "don't break the circuit" school of thought seems to make common sense, but this element of circuit maintenance has not been specifically studied. Pneumonia continues to be caused by organisms found predominantly in the gastrointestinal tract. The use of selective digestive decontamination alters the organisms seen in VAP. While,

clearly, poor hand hygiene probably results in nosocomial infection, the idea that VAP is prevented or lessened by maintaining circuit integrity remains unproven.

The duration a circuit can be used safely in a single patient is not known. It appears as though one circuit per patient seems to be the de facto standard. One criticism of the multitude of studies evaluating circuit change frequencies is the absence of data on duration of ventilation in the prolonged-circuit-change group. As an example, Fink et al compared 48-hour circuit changes to 30-day circuit changes, with the longer circuit use being associated with lower VAP rates.[17] Yet, review of this study does not tell us how long the patients in the 30-day-circuit-change group were ventilated. If mean ventilation time was $<$ 1 week, the conclusions might be altered. Cost savings would also be changed based on a mean duration of ventilation $<$ 30 days.

Cost savings are major drivers in lengthening the duration of circuit use. Craven et al[5] were the first to demonstrate significant cost savings associated with an additional day of circuit use. Several of the studies described here have evaluated costs related to circuit-change intervals.[5,7,8,14,15–17,19] Comparisons of these studies using different circuit-change intervals and varying equipment across 2 decades and 3 continents is all but impossible. Cost savings, however, are real and can be divided between reductions in use of disposable equipment, reduced circuit maintenance, and at least theoretically by reductions in VAP. A note of caution regarding reduced practitioner time to change circuits translating to reduced costs: reduced costs are realized only if practitioner time is reallocated to other duties or the number of practitioners is reduced.

The American Association for Respiratory Care's evidence-based guideline on care of the ventilator circuit makes the following recommendation:

> Ventilator circuits should not be changed routinely for infection control purposes. The available evidence suggests no patient harm and considerable cost savings associated with extended ventilator-circuit change intervals. The maximum duration of time that circuits can be used safely is unknown.[11]

Type of Circuit

Ventilator circuits may be reuseable or disposable and may also contain a heated wire to reduce condensate formation. The proposed advantages of the heated-wire circuit include reduced condensate, decreased need for breaking the circuit to empty condensate, and reduced water usage. The latter is associated with a cost savings.[20] Few studies have directly compared heated-wire circuits to non-heated-wire circuits with respect to the incidence of VAP.

Our group compared heated-wire circuits, non-heated-wire circuits, and heat-and-moisture exchangers (HMEs) in a small study of 200 patients.[21] This study has been criticized for the randomization method, which was intended to prevent use of HMEs in patients felt to be at risk for HME or endotracheal-tube occlusion.[22] In one arm of our study, 49 patients used a heated-wire circuit and 48 patients used a non-heated-wire circuit. Circuit changes were every 7 days, open-circuit suctioning was used, and cultures of respiratory secretions, ventilator circuits, and humidification devices were done. VAP was diagnosed based on clinical criteria. The VAP rate was 8% in the heated-wire circuit group and 5% in the non-heated-wire circuit group. This difference was not statistically significant, in part owing to the small number of patients. These data were evaluated by Hess et al,[11] demonstrating a relative risk of VAP of 1.57 in favor of the non-heated-wire circuits. However, the wide 95% confidence interval of 0.55 to 4.45 suggests that no difference is the best conclusion.

Fink et al[17] studied ventilator-circuit changes over a 4-year period. In the final year, heated-wire circuits were introduced and circuit-change interval was lengthened to every 30 days. They found no difference in VAP rate. This paper utilizes a detailed cost analysis, demonstrating reduced costs associated with heated-wire circuits and monthly circuit changes. However, mean duration of ventilation in the monthly circuit-change group was around 6 days. While cost savings may be associated with the use of heated-wire circuits, these savings are often overestimated. Our group showed that in 1995 the setup cost of a non-heated-wire system was $12.97, while the heated-wire system cost $24.95. The reduced use of water and elimination of condensate with the heated-wire circuit results in reduced costs with prolonged use.[21]

Other aspects of humidifier systems have been evaluated, including the use of disposable versus reusable water reservoirs,[23] with no apparent effect of VAP. As practices have changed with respect to circuit-change intervals, use of closed-circuit suctioning, and introduction of metered-dose inhalers, future studies of heated-wire versus non-heated-wire circuits in prolonged ventilation ($>$ 7 d) seem warranted.

Type of Humidifier

Heating and humidifying dry medical gases delivered to the instrumented airway is a standard of care. Heat and humidity can be provided by heated-water humidifiers or by passive devices commonly known as artificial noses. A more proper term is HME. An HME may contain a hygroscopic salt to improve moisture retention, resulting in a hygroscopic HME, and/or a filter, resulting in a heat-and-moisture-exchanging filter (HMEF). The passive operation, elimination of condensate, and low cost of HMEs have popularized this device over the last decade. The

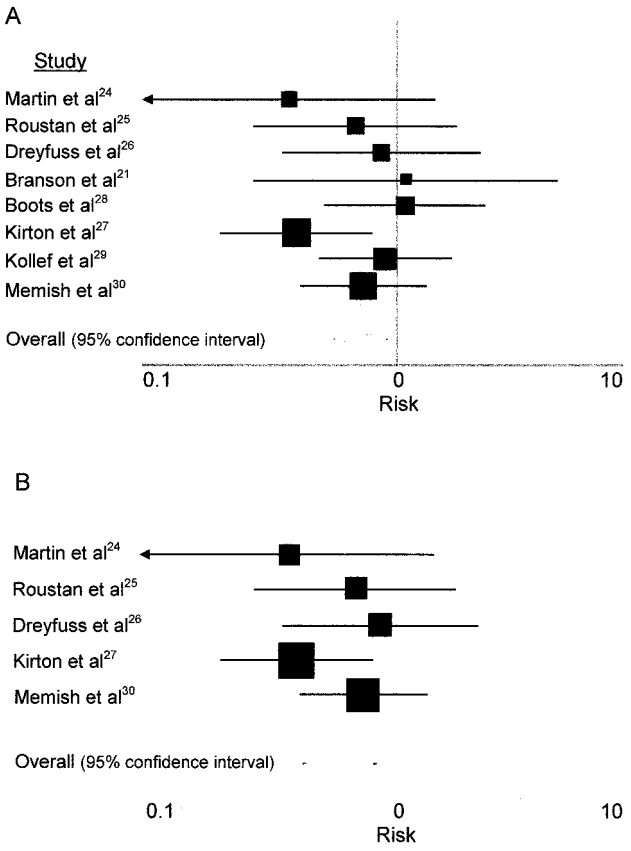

Fig. 3. A: Analysis of ventilator-associated pneumonia in all randomized controlled trials comparing heated humidifiers with heat-and-moisture exchangers. B: Analysis of ventilator-associated pneumonia in randomized controlled trials comparing heated humidifiers with heat-and-moisture exchangers over 7 days of mechanical ventilation. The diamond shape indicates the summary relative risk and 95% confidence interval. Size of squares is inversely proportional to the variance of the studies. (Adapted from Reference 33, with permission.)

elimination of condensate and need to break the circuit to handle condensate suggests that the HME may result in a reduced risk of VAP.

A number of studies have been published comparing HME use to heated humidifiers, with respect to the incidence of VAP.[24–32] These studies vary widely in the type of HMEF and heated humidifier (heated or non-heated-wire circuit) as well as in the circuit-change interval, type of suctioning (open vs closed) and in the duration of ventilation. Diagnostic criteria for the presence of VAP was also different in these studies.

Cook et al,[10] Hess et al,[11] and Kola et al[33] have performed meta-analyses on the studies, comparing HMEs to heated humidifiers for the incidence of nosocomial pneumonia. The most recent of these, by Kola et al, demonstrates a reduction in the relative risk of developing VAP in the HME group (relative risk 0.7, 95% confidence interval of 0.5–0.94). Perhaps more striking is the analysis of studies with a mean duration of ventilation > 7 days, where the relative risk for VAP falls to 0.57 in the HME group, with a 95% confidence interval of 0.38–0.83.[33] These data are shown in Figure 3.

As with any meta-analysis, the results can be heavily influenced by a single trial. In this case, the study by Kirton et al demonstrating a relative risk of 0.41 and a 95% confidence interval of 0.2–0.86 significantly influences the outcome.[27] In the other 7 studies, involving 733 patients, there was a trend toward a reduced VAP rate favoring HME use, but these differences were not statistically significant.

More recently, Kranabetter et al compared VAP rates in patients using HMEs with a historical control using heated humidification without a heated-wire circuit.[32] These authors took advantage of an institutional switch from active to passive humidification in the intensive care unit to evaluate differences in the devices. They compared 1,887 patients using heated humidification to 1,698 patients using HMEFs. All patients were in a surgical intensive care unit, and VAP was diagnosed using clinical criteria. During a 42-month period, they identified 99 cases of VAP. The incidence for VAP was found to be 13.5 (heated humidification) and 9.6 (HMEF) per 1,000 ventilator days: a VAP rate of 32.3% and 22.4% per 1,000 patients, respectively. The rate of VAP among the groups ($p = 0.068$) and the incidence of VAP per 1,000 ventilator days ($p = 0.089$) were not statistically significant. When Kranabetter et al evaluated the rates of VAP in patients requiring mechanical ventilation for more than 2 days ($n = 540$), the difference was statistically significant ($p = 0.012$).

Heavily influenced by the data of Kirton et al and Kranabetter et al, the evidence appears to suggest that use of an HME reduces the risk of VAP. What remains unclear is the mechanistic effect. One possibility is simply the elimination of condensate from the circuit. While circuits are typically colonized by bacteria from the patient, it is the presence of condensate that allows the bacteria to thrive. The second possibility is the reduced need to break the circuit to remove the condensate, thus reducing contamination from caregivers and the environment. Third, the elimination of condensate prevents inadvertent lavage of the airway from contaminated condensate. Finally, in several studies the use of a filter in the HME has been suggested as a possibility.[24,25,27,32] However, the effect has been seen with filtration and without filtration. Additionally, the types of filters used vary widely in efficiency. The anesthesia literature has reviewed these possibilities and found that both types of filters commonly used, electret and hydrophobic, eliminate circuit contamination.[34–39]

Only one study has compared HME versus HMEF in mechanically ventilated patients with respect to the incidence of VAP.[40] Thomachot et al compared a hygroscopic HME filter (electret filter) to a hygroscopic HME filter

Table 3. Contraindications for Heat and Moisture Exchanger Use in Mechanically Ventilated Patients

HME Exclusion	Rationale
Hypothermia	HMEs return a portion of the exhaled temperature and moisture. If exhaled temperature and humidity are reduced, inspired humidity will be reduced.
Bloody secretions, pulmonary edema fluid	Occlusion of the HME and subsequent increased work of breathing and/or barotraumas
Air leak (bronchopleural fistula, incompetent airway cuff)	If exhaled volume does not enter HME, heat and moisture will be lost.
Noninvasive ventilation	Leaks contribute to inefficiency, dead space increases work of breathing, rebreathing of CO_2, and either minute ventilation or P_{aCO_2} will increase.
Thick tenacious secretions	Normal HME operation results in a net loss of moisture from the respiratory tract, predominantly from the major airways—increases risk of mucus plugging and airway occlusion.
Lung-protective ventilation (low tidal volumes)	Increased dead space increases required minute ventilation or leads to hypercapnia.
Difficult-to-wean patient with respiratory-muscle fatigue	Dead space and resistance increases work of breathing and complicates use of spontaneous breathing trials.

HME = heat-and-moisture exchanger

(hydrophobic filter) in a group of 136 postoperative patients. There was no difference in the duration of ventilation, number of tracheal aspirations, incidence of atelectasis, or the incidence of VAP (hygroscopic HME filter 27/1,000 ventilator days vs HMEF 30/1,000 ventilator days). In a second trial, Thomachot et al compared 2 types of HMEF and again found no difference in the rate of circuit colonization or rate of VAP.[41]

Like the circuit-change interval, the mechanism by which the HME may reduce the risk of VAP is not entirely clear. HME should be used in all patients in whom there is no contraindication.[20,42–44] However, there are clear contraindications for HME use: the risk of airway occlusion from insufficient humidity, increased dead space in patients receiving lung-protective ventilation and small tidal volumes, occlusion of the HME by blood or secretions, elevated dead space and resistance confounding spontaneous breathing trials, and hypothermia. Each of these has been detailed by a number of authors.[45–52] Clearly, while HMEs carry the advantages of low cost, passive operation, mobility, and possibly reduced risk of VAP, we must use these devices only in patients who will benefit, without increasing the risk of other untoward outcomes. Hess noted quite succinctly that, while HME use may be associated with a decreased relative risk of VAP of 0.8, the risk of airway occlusion when using an HME increases by 3.84, with a 95% confidence interval of 1.92 to 7.69.[11] HME use should be avoided in select patients; the reasons and rationale are shown in Table 3.

Duration of HME Use

Following the success of prolonged usage of ventilator circuits with no impact on function or risk of VAP, investigators logically questioned the extended use of a single HME.[53–61] Our group compared daily HME changes to every-5-day changes in a group of surgical patients.[53] Ventilator-circuit changes were performed weekly, and patients with contraindications for HME use were excluded. We found no difference in the incidence of VAP between groups. While the study was designed to evaluate once-every-5 day changes, the mean duration of ventilation in the study group was only 3 days. Our conclusion, then, suggested that a single HME could be used for 72 hours safely. Thomachot et al compared every-day HME changes to every-7-day HME changes and found no difference in the incidence of VAP (17% vs 24%).[54] In this trial, 49 of 71 patients who were randomized to the 7-day HME-change group were ventilated for $\geq$ 7 days. There were no instances of airway occlusion in either group during this study.

Dejdaini et al[55] and Daumal et al[56] each compared 24-hour HME changes to 48-hour HME changes and found no difference in the incidence of VAP. Hess et al included these 4 studies[53–56] in a meta-analysis of extended HME use and concluded that there was no effect on VAP (relative risk 0.58, 95% confidence interval 0.24 to 1.41, $p = 0.14$).[11] These data are shown in Figure 4 and Table 4.

Other authors have evaluated prolonged HME use.[57–63] Kollef et al compared once-a-week HME changes to heated humidification in a randomized controlled study of 300 patients.[57] The mean duration of ventilation in the HME group was only 4.6 days, which means only about 15% of patients actually used the same HME for 7 days. We believe these data support the work of others,[53,54] that a period of 3–4 days is the maximum use of a single HME. Similar data were shown by Ricard et al in a study of weekly HME changes.[58] In this study of 33 patients, 40 HMEs were used for the entire 7-day study. The authors

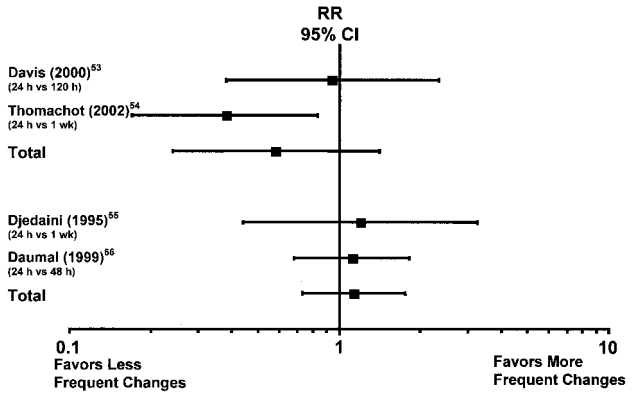

Fig. 4. Meta-analysis of studies investigating the relationship between change frequency for passive humidifiers and the risk of ventilator-associated pneumonia. RR = relative risk. CI = confidence interval. (Adapted from Reference 11.)

noted that the efficiency of HMEs appeared to wane with > 48 hours' use in patients with COPD. They recommended that HME changes in COPD patients be limited to 48 hours. Additional trials support the use of HMEs for 48 hours without any negative impact on performance or rates of VAP.[59–62] An interesting trial by Thomachot et al suggests that the combination of an HME and a small heated humidifier (HME Booster, Medisize, The Netherlands) can be used for 96 hours without adverse effects. The addition of the HME Booster increases the moisture delivered to the patient by 3–5 mg H_2O/L.[63]

While most manufacturers recommend every-24-hour HME changes, the literature seems to support up to 96 hours use of a single HME in non-COPD patients. This prolonged use does not adversely affect the moisture output of the HME or alter the incidence of VAP.

Closed Versus Open Suction

Traditional suctioning of the artificial airway includes hyperoxygenation, disconnection from the ventilator, and insertion of a sterile, disposable, single-use suction catheter.[20] Closed-circuit suction systems were introduced nearly 25 years ago as a method to reduce complications associated with the traditional suctioning procedure. The purported advantages of closed-circuit suctioning include: no need to break the circuit; maintenance of ventilation, oxygenation, and positive end-expiratory pressure; and reduced environmental and caregiver contamination. The maintenance of positive end-expiratory pressure during closed-circuit suctioning has been shown to reduce hypoxemia from lung derecruitment.[64,65] However, when these devices were introduced, they were to be changed every 24 hours, making routine use financially impractical.

The relationship between VAP and closed-circuit suctioning has not been studied with the same rigor applied to ventilator-circuit changes and HME use. In fact, early studies of closed-circuit suction catheters demonstrated higher levels of catheter colonization.[66,67] The concern was that when saline was introduced to cleanse the catheter or to stimulate a cough, the bacteria from the catheter would be lavaged into the airway. Several studies have compared open- and closed-circuit suctioning with respect to costs and incidence of VAP.[68–75] Two prospective, randomized trials of open- versus closed-suction systems found no difference in the incidence of VAP.[68,69] Combes et al randomized 104 patients with negative tracheal aspirates at study entry to open ($n = 50$) or closed suctioning ($n = 54$).[70] They found that the incidence of VAP was 3.5 times greater in the open-circuit suctioning group and that VAP increased the length of stay by 17 days. This report is important in that it demonstrates how closed-circuit suctioning may be more expensive to purchase but that the overall patient costs can be substantially reduced by use of these devices. Interestingly, these authors attributed the reduction in VAP rate to elimination of the need to "open" the circuit for traditional suctioning. More recently, 3 trials have demonstrated no effect of prolonged use of closed-circuit suction catheters on the incidence of VAP.[71–73] Zeitoun et al recently compared open- to closed-circuit suctioning in a randomized, prospective trial of 47 patients.[74] They found that the VAP rate was reduced, although the numbers in this study are quite small.

The evidence or lack thereof regarding closed-circuit suction catheters and the safe duration of their use results in a quandary. While there is no resounding proof that closed suctioning reduces VAP compared to open suctioning, there is also no proof that it increases the risk of VAP. In turn, while observational studies suggest greater colonization of closed-circuit suction catheters, there is no evidence that this increases the risk of VAP. The conventional wisdom that fewer breaks of the circuit result in a lower risk of contamination clearly supports the use of closed-circuit suctioning.[75,76] The duration that a closed-circuit suction system can be safely used is unknown. In concert with the recommendation on ventilator circuits, it appears that a closed-circuit suction system can be used in a single patient for as long as the ventilator circuit is used. The caveats being the same, the closed suction system should be changed if there are signs of dysfunction or when esthetics dictate.

One study reported that the use of closed-circuit suction systems results in significantly less environmental contamination, compared to open techniques.[77] While this has no impact on VAP, it does reduce exposure of caregivers. Additionally, the prolonged use of closed-circuit systems significantly reduces costs.[71–74] The American Association for Respiratory Care's recommendation regarding closed suction systems states:

Table 4. Summary of Studies Investigating the Relationship Between Change Frequency for Passive Humidifiers and the Risk of Ventilator-Associated Pneumonia

Citation	Study Population	VAP Diagnosis	Control Group	Treatment Group	Control Group		Treatment Group		Level	Relative Risk (95% CI)
					n	Pneumonia (%)	*n*	Pneumonia (%)		
Randomized Controlled Trials										
Davis 2000[53]	Adult mechanically ventilated patients	Clinical	HME changed every 24 h	HME changed every 120 h	100	8.0	120	7.5	1	0.94 (0.38, 2.34)
Thomachot 2002[54]	Adult mechanically ventilated patients	Clinical	HME changed every 24 h	HME changed every 7 d	84	26.2	71	9.9	1	0.38 (0.17, 0.83)
Total					184	16.3	191	8.4		0.58 (0.24, 1.41)
Observational Studies										
Djedaini 1995[55]	Adult mechanically ventilated patients	Quantitative cultures	HME changed every 24 h	HME changed every 48 h	61	9.8	68	11.8	4	1.20 (0.44, 3.25)
Daumal 1999[56]	Adult mechanically ventilated patients	Quantitative cultures	HME changed every 24 h	HME changed every 48 h	174	14.4	187	16.0	4	1.12 (0.68, 1.82)
Total					235	13.2	255	14.9		1.13 (0.73, 1.76)

VAP = ventilator-associated pneumonia
CI = confidence interval
HME = heat and moisture exchanger
(Adapted from Reference 11.)

The use of closed suction catheters should be considered part of a VAP prevention strategy. When closed suction catheters are used, they do not need to be changed daily for infection control purposes. The maximum duration of time that closed suction catheters can be used safely is unknown.[11]

Role of the Bedside Manual Resuscitator

A number of studies have implicated patient transport from the intensive care unit as a risk factor for VAP and suggested that the manual resuscitator might be a culprit.[78–81] Clearly, the manual resuscitator is colonized with bacteria from the patient's airway. Cleaning and capping of the resuscitator when not in use should be routine. An HME or filter attached to the resuscitator may reduce contamination of the components and protection from large volumes of secretions, but then the filter becomes colonized. Additionally, this may be an expensive option. Perhaps the best way to reduce any risk of VAP due to the manual resuscitator is to assure that it remains attached to a holder and is not lying in the bed or on the floor. While colonization of the manual resuscitator is common, there is no evidence that this affects VAP rate. The role of transport may include changing patient position for testing and increasing the risk of aspirating oropharyngeal secretions around the endotracheal tube cuff.

Role of Nebulizers

The nebulizer was identified as a possible source of aerosol condensate with a link to VAP as early as the 1960s.[1,2] Craven et al identified the nebulizer as a source of VAP in an early study.[82] This risk is associated with bad practice, where nebulizers remain in the circuit after the medication is delivered and condensate is allowed to be aerosolized back to the patient. More recently, outbreaks of VAP have been linked to contaminated nebulizers.[83,84] Kollef et al identified the use of aerosol therapy as an independent variable increasing the risk of VAP in ventilated patients.[78] This discussion prompts the question, if metered-dose inhalers replaced up-draft nebulizers, would the risk of VAP be reduced? This of course falls in line with the "don't break the circuit" school of thought and deserves further study. Guidelines for the care and cleaning of nebulizers used during mechanical ventilation have been provided by CDC.[85] The shortcomings of these recommendations have been detailed by Hess.[86]

Summary

The role of respiratory care equipment in the prevention and etiology of VAP remains a constant concern for the bedside practitioner. While some issues are well defined and the evidence base is clear, others remain unclear. Future studies should include the best practices of a single ventilator circuit per patient and closed suctioning, along with specific interventions (metered-dose inhaler vs nebulizer) to determine the role of these therapies.

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Discussion

Niederman: I have a different hypothesis that I'd like you to comment on. I think there were ways that the ventilator circuit was handled that added to the risk of pneumonia, and we've learned what those mistakes are, and we don't make them anymore. Now we've seen that with good handling of the circuits it doesn't matter how often you change them. My guess is that that isn't necessarily the final answer. The reason is that you wouldn't expect that it could make any difference when you're hooking it up to an ETT [endotracheal tube] that's loaded with biofilm, and the question is, if we could finally improve on ETTs, would it then be worth restudying all of these circuit questions again? My guess is that it might make a difference how often you change circuits, if you can achieve a clean ETT to change the circuit and hook it up to.

Branson: That's a good question. The rationale behind doing these studies of prolonged circuit changes was exactly that. If you're not going to change the ETT, what's the point of changing the circuit, because the circuit becomes contaminated, colonized by the patient—not the other way around.

Niederman: But you could conceive of the idea that if you somehow had a barrier between the patient and the circuit—meaning a tube that couldn't become colonized—that it would make sense to not amplify the patient's bacteria in the circuit. If we ever achieve the point where you really have a sterile barrier, or a barrier that doesn't grow bacteria between the patient and the circuit, then I think it would be important to restudy the circuit.

Maki: The thing that makes my head hurt is that most VAP is caused by aspiration around the ETT cuff.

Niederman: Not always. In the studies of subglottic secretion drainage, late-onset VAP could not be prevented by subglottic secretion drainage, since it doesn't work that well for pseudomonas VAP; and so the idea might be that those organisms get there in a different way. The relevant hypothesis here is that patients become colonized with whatever they've got in the trachea. They aerosolize those bacteria, which get into the tube and then into the circuit. In the tube and in the circuit they grow without antibiotics or host defenses touching them, and they

get reintroduced and re-inoculated and lead to infection. So, even if you had a sterile tube—not an antibacterial but a *sterile* tube—where it didn't support the growth, you could still conceive of a way that the colonized patient could aerosolize their bacteria, some of it backwash into the condensate, sit there for awhile, and then for a variety of reasons get re-inoculated in larger numbers back into the patient. That's why I don't know that it's an irrelevant idea just yet.

Branson: My point is that if we do selective decontamination and we don't change VAP, we just change the bugs, that makes me think the origin in most of these is from the gastrointestinal tract somewhere. Then you have the ventilator circuit. Is the HME reducing VAP because the circuit is dry? There's no condensate, so there's no way for that bacteria to live, and, if so, then why doesn't a heated-wire circuit reduce the incidence of VAP? Or maybe we just haven't studied it long enough to figure it out. You have data, and you say, "Well, it's because of this." But then you do something else that provides you the exact same condition and you don't get the same result, which doesn't allow me to make really good recommendations on what we ought to do.

Kollef: I remember reviewing a couple of those papers looking at HMEs versus heated-wire systems, like the Kirton et al paper in *Chest*[1] and the problem with them was that they didn't look at condensate formation. That was one obvious flaw in those studies. So you don't know if one group had more condensate than the other, and that, in my mind, would have been a very important potential predictor for pneumonia occurring. In fact, the reality is that, even with a heated-wire system in place, substantial condensate can form, and that can be an issue.

The other thing I'm just going to throw out there is that when we talk about not changing HMEs—and this would support Mike Niederman's point—where did the HMEs get occluded? Did they get occluded from the secretions and the material that comes up via the ETT? I think if we can modify the ETTs and potentially even modify more proximal components of the circuit, in terms of their ability to form biofilm and such, that may have a more important role, and it may actually point toward less frequent manipulation of those devices, which we're already doing.

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Branson: Humidification is one of those issues that is just a small part of the whole mechanical ventilation system, but there are places in Europe where they use nothing but HMEs, on every single patient, no matter what. And in the United Kingdom I observed nurses aerosolizing saline to the patients—a lot of the times unbeknownst to the physicians—to try to improve the moisture getting to the lower respiratory tract. But also in Australia and New Zealand, where everybody gets heated humidification at 40°C at the Y-piece, my overwhelming impression is that all those patients seem to do fine, no matter what you do.

I don't have good answers. You get the argument that if you used "optimum" humidification, maybe you'd reduce VAP because you'd improve mucociliary function. Obviously, what is "optimum" humidification is a question we've never been able to answer. Does it require heated humidification? Or can it be done with an HME?

Hess: I had not thought about this before, so I don't know how it will come out, but if the circuit might be important with a clean ETT, then the circuit might be important without an ETT. I don't think anybody has shown that you need to change the circuit with noninvasive ventilation.

Niederman: It's an interesting idea, but, again, I think that the defense mechanisms between the circuit and the trachea are very different when you don't have a tube than when you do. So it wouldn't be hard to imagine why it could be important with the tube in and not important without a tube.

Hess: Point well made.

Branson: I have thought that an antibiotic-impregnated or coated ventilator circuit is a bad idea. Might it actually be a good idea?

Niederman: Why do you think it's a bad idea?

Branson: I thought it would be expensive, compared to traditional circuits, for no real benefit.

Niederman: Again, right now we don't know if there would be a benefit. I'm not surprised that the studies came out the way that they did. It's hard to imagine they could have come out any other way, because once you got rid of the obvious errors, which were shown by the earlier studies, and you continue to know how to handle this stuff, you hooked up a clean circuit to a dirty tube, and what did you expect? If you somehow find a way not to have a dirty tube, then I don't know what would happen.

What you really need is something that doesn't support the growth of bacteria as well as doesn't support the adhesion of mucus, because mucus will support the growth of bacteria. So it's a complicated system. I don't even know what the answer is for the circuits. All I'm saying is that I'm not convinced we know the answer, but if we get to a point where the tube is not a factor, we can restudy the circuit.

Branson: My other issue is that the HME reduces VAP potentially because of the loss of condensate. I don't think filtering the gas does anything at all. The manufacturers say that the filter reduces VAP, but it's not the filtering, it's the dryness in the circuit. In a study published in one of the Australian intensive care journals,[1] they inoculated anesthesia circuits with bacteria and then ventilated the test lung. They found that if they ventilated it with an HME, where there's no humidity in the circuit, all those bacteria die within about 2 hours because they become desiccated. So I think *that's* the important issue: a dry circuit.

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Kallet: The other thing that struck me is the number of reasons we break into a ventilator circuit: for giving aerosol treatments, for changing capnography adapters that often clog up with secretions. We're constantly breaking into the circuits for numerous reasons. And also the behavior at the bedside. You mentioned the manual resuscitator being draped over the oxygen flow meter; I've seen them draped over the suction control for the nasogastric tube. I think, particularly when the ICU census and acuity are high, the behavior that we have at the bedside can be contributing to this problem. That the manual resuscitator is not placed on a clean field, but slips off from the bedside or onto the sheets that have been contaminated with gastrointestinal bacteria. So it becomes a really complicated issue, because we can't really control what happens at the bedside, particularly when the clinician is under high stress.

Branson: I agree. I think one of the big changes we've made recently is that, aside from doing the more frequent oral care, every patient has a Yankauer suction setup at the bedside, and now there's a holder for it, as opposed to it just hanging on the wall or sitting on top of the ventilator. All those things are being addressed now, and I think we need to look at them more carefully.

I can't tell you how many times I have had trauma patients who want a drink of water, and during visiting hours a family member walks over to the sink, soaks a washcloth with water, and sticks it in the patient's mouth to suck on. Maybe we need to look at such practices, as well as giving ice chips and putting towels on people's foreheads, but the way we handle all the equipment at the bedside needs to be improved. We're not going to handle it sterilely, but we need to handle it more cleanly, and nobody who actually goes and sees patients can say that they don't walk into the ICU and see the manual resuscitators lying on the floor, because when you're in a hurry, and you had to bag somebody and you reach to hang it on the valve that turns the oxygen off, and you miss, it goes on the floor, and I *know* we don't throw those away just because they go on the floor. Maybe we need some mechanism, some cover, to protect those as well.

Hess: An issue I don't think you addressed, or maybe I fell asleep during that part of the talk, is that transporting patients is another risk factor for VAP.

Branson: You *were* asleep! You were having a dream about Boston College winning the NCAA, and along with the Red Sox and the Patriots you had the trifecta! Again, I ask the question, and I'm anxious for the answer. Is transporting the patient a risk for VAP, because they need to be transported because they're sick, they're going to CT [computed tomography] because they have an intra-abdominal infection or whatever, or is it a risk because generally when we transport a patient, we lay the patient flat down in the CT scanner, or is it the manual resuscitator, or is it all 3 things? I don't know.

Kollef: It's probably all of them: aspiration and colonization, and I think anything we can do to affect those, but to me it's pretty simple. I think Mike Niederman said it before: if you aspirate a bolus of contaminated fluid, you're likely to get pneumonia; if you can prevent that aspiration, that's probably beneficial.

Park: In our acinetobacter outbreak, 2 factors associated with acquisition were transport to radiology and transport to the operating room. We hypothesized that the CT table, the anesthesiologists' scrubs, and other contacts might serve to pass pathogens from one patient to another. This may be an additional factor associated with transports.

Branson: We transport patients on transport ventilators, so they get a whole new circuit and HME before they go downstairs, so I'm not worried about them getting a bolus of condensate, but again, since they still seem to have a risk of VAP, I believe it's aspiration around the cuff, because we're changing the patient's position substantially at the CT scanner and in moving the patient off the bed and onto the table.