Risk Factors for Ventilator-Associated Pneumonia: From Epidemiology to Patient Management

Marc J. M. Bonten, Marin H. Kollef, and Jesse B. Hall

Risk factors for the development of ventilator-associated pneumonia (VAP), as identified in epidemiological studies, have provided a basis for testable interventions in randomized trials. We describe how these results have influenced patient treatment. Single interventions in patients undergoing intubation have focused on either reducing aspiration of oropharyngeal secretions, modulation of colonization (in either the oropharynx, the stomach, or the whole digestive tract), use of systemic antimicrobial prophylaxis, or ventilator circuit changes. More recently, multiple simultaneously implemented interventions have been used. In general, routine measures to decrease oropharyngeal aspiration and antibiotic-containing prevention strategies appear to be the most effective, and the latter were associated with improved rates of patient survival in recent trials. These benefits must be balanced against the widespread fear of emergence of antibiotic resistance. In hospital settings with low baseline levels of antibiotic resistance, however, the benefits to patient outcome may outweigh this fear of resistance. In settings with high levels of antibiotic resistance, combined approaches of non–antibiotic using strategies and education programs might be most beneficial.

Ventilator-associated pneumonia (VAP) is the most common lethal infection observed in patients who require treatment in intensive care units (ICUs). VAP is defined as pneumonia occurring ≥48 h after intubation and the start of mechanical ventilation. This time window is important, so that any infection that is incubating at the time of admission can be excluded. Incidences are highly influenced by the characteristics of the patient population studied and the criteria and techniques applied for diagnosis. When compared with combinations of clinical and radiographic criteria and results of semiquantitative culture of endotracheal aspirates, incidences based on the same criteria with the addition of results of quantitative culture samples obtained by bronchoscopic techniques decrease by 30%–40% [1]. However, the denominator used to calculate rates in these studies consisted of patients undergoing mechanical ventilation for ≥48 h, and these incidence rates may, therefore, be higher than the rates among general medical and surgical ICU patients [1]. If these high incidence rates were to pertain to the broad population of patients undergoing mechanical ventilation, VAP would be one of the most common nosocomial infections in the United States. In point prevalence studies, VAP consistently has the highest mortality and morbidity, and it typically prolongs the duration of hospitalization for an average of 7–9 days per patient [1].

The crude mortality rate for VAP has been cited to be as high as 70%, but there is wide recognition that not all deaths among affected patients are the direct result of infection, but, rather, that the infection is a marker for severity of illness. The mortality attributable to VAP has been defined as the percentage of deaths that would not have occurred in the absence of infection. Some case-matching studies have estimated that one-third to one-half of all VAP-related deaths are the direct result of infection, with higher attributable mortality in cases characterized by bacteremia or in which the etiologic agent is Pseudomonas aeruginosa or Acinetobacter species [2, 3]. However, using similar methodology, others failed to identify attributable
Despite the importance of preventing nosocomial infections, available information suggests that the number of such infections is on the increase, resulting in warnings from professional and national agencies to refocus efforts on their prevention [11, 12]. In this review, we describe how determination of risk factors for the development of VAP in epidemiological studies has led to interventions tested in randomized trials and how the results of these trials have influenced patient management.

**PATHOGENESIS**

For nosocomial respiratory tract infections to occur, the delicate balance between host defenses and microbial propensity for invasion must shift in favor of the capability of pathogens to establish pneumonia. Risk for these infections is determined in part by the duration of exposure to the health care environment and in part by a number of host factors and treatment-related factors that have been identified in the literature (table 1). To consider methods to prevent these infections, it is useful to separate host- and treatment-related factors into modifiable and nonmodifiable groups. Most importantly, pathogens must reach the lower airways. This usually occurs after aspiration of oropharyngeal fluids containing potential pathogens, and, therefore, colonization at this site is almost a prerequisite for the development of VAP. In addition, microorganisms may be introduced directly by inhalation into the lower airways as a result of contamination of medical equipment, and they may reach the lungs after hematogenous spread, although these routes of infection are probably much less common. Host defenses may be impaired because of multiple disease-related alterations or even genetic predisposition, although genetic factors have yet to be explicitly defined.

**RISK FACTORS**

Many risk factors for the development of VAP have been identified. They can be differentiated into modifiable and nonmodifiable risk factors and into patient-related and treatment-related risk factors (table 1). Nonmodifiable patient-related risk factors include male sex, preexisting pulmonary disease, coma, AIDS, head trauma, and multiple–organ system failure. Nonmodifiable treatment-related risk factors include the necessity of neurosurgery, monitoring of intracranial pressure, reintubation, or transportation out of ICU. However, observational studies cannot distinguish causation from noncausal association. Ultimate proof of causality—and, thus, of the potential for modulation to prevent infection—should be provided by empirical testing in prospective trials. Some of the modifiable risk factors have been subjected to empirical testing and will be discussed here. To get a rough estimate of the magnitude of preventive efficacy, we have pooled data from specific interventions and calculated relative risk reductions (and 95% CIs) of the pooled data (table 2).

**PATIENT-RELATED RISK FACTORS AND INTERVENTION STRATEGIES**

**Intubation.** Intubation and mechanical ventilation are, by definition, prerequisites for the development of VAP. Unnec-

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**Table 1. Risk factors for ventilator-associated pneumonia (VAP).**

<table>
<thead>
<tr>
<th>Risk factor class, type</th>
<th>OR (95% CI)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonmodifiable risk factors</strong></td>
<td></td>
<td></td>
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<tr>
<td>Patient-related risk factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td>1.9 (1.4–2.6)</td>
<td>[13]</td>
</tr>
<tr>
<td>Organ System Failure Index of &gt;2</td>
<td>10.2 (4.5–23)</td>
<td>[15]</td>
</tr>
<tr>
<td>Age of &gt;60 years</td>
<td>5.1 (1.9–14.1)</td>
<td>[15]</td>
</tr>
<tr>
<td>Coma</td>
<td>40.3 (3.3–423.1)</td>
<td>[16]</td>
</tr>
<tr>
<td>ARDS</td>
<td>9.7 (1.6–59.2)</td>
<td>[17]</td>
</tr>
<tr>
<td>Head trauma</td>
<td>5.2 (0.9–30.3)</td>
<td>[17]</td>
</tr>
<tr>
<td>Male sex</td>
<td>2 (1.5–2.7)</td>
<td>[18]</td>
</tr>
<tr>
<td>Intervention-related risk factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurosurgery</td>
<td>10 (1.6–64.9)</td>
<td>[17]</td>
</tr>
<tr>
<td>Thoracic surgery</td>
<td>2.16</td>
<td>[19]</td>
</tr>
<tr>
<td>ICP monitor</td>
<td>4.2 (1.7–10.5)</td>
<td>[20]</td>
</tr>
<tr>
<td>Transportation out of ICU</td>
<td>3.8 (2.8–5.5)</td>
<td>[18]</td>
</tr>
<tr>
<td>Reintubation</td>
<td>5.94 (1.27–22.71)</td>
<td>[21]</td>
</tr>
<tr>
<td><strong>Modifiable risk factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention-related risk factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Use of H2-antagonist</td>
<td>2.5 (1.2–5)</td>
<td>[20]</td>
</tr>
<tr>
<td>Use of antacids</td>
<td>20</td>
<td>[19]</td>
</tr>
<tr>
<td>Use of sucralfate</td>
<td>3.44</td>
<td>[19]</td>
</tr>
<tr>
<td>24-h circuit changes, compared with 48-h circuit changes</td>
<td>2.3 (1.2–4.7)</td>
<td>[20]</td>
</tr>
<tr>
<td>Use of antibiotics</td>
<td>3.1 (1.4–6.9)</td>
<td>[15]</td>
</tr>
<tr>
<td>Aerosol treatment</td>
<td>1.9 (1.4–2.5)</td>
<td>[20]</td>
</tr>
</tbody>
</table>

**NOTE.** ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease; ICP, intracranial pressure; ICU, intensive care unit.

*a* Only associated with increased risk for VAP caused by *Acinetobacter baumannii.*
necessary intubation, therefore, should be avoided at all times. Noninvasive positive-pressure ventilation (NIPPV) using a face mask could be used as an alternative ventilation mode in ICU patients. The beneficial effects of NIPPV on the development of VAP and on patient survival have been determined in randomized trials involving patients with acute exacerbations of chronic obstructive pulmonary disease [37], acute respiratory failure [38], and, in immunosuppressed patients with pulmonary infiltrates, fever and respiratory failure [39]. Relative risk reductions from these individual studies range from 0.67 to 0.87. Nasotracheal intubation clearly is a risk for the development of nosocomial sinusitis and should, therefore, be avoided as well. Moreover, sinusitis was associated with an increased OR for VAP of 3.8 in one study [40].

Duration of mechanical ventilation. Several studies have identified the duration of ventilation as an important determinant for the development of VAP. The risk of VAP does not appear to be constant over the time of ventilation. In a large cohort study, the risk was estimated to be 3% per day in the first week, 2% per day in the second week, and 1% per day in the third week and beyond [41]. As a result, strategies to reduce the duration of ventilation may decrease the risk for development of VAP, especially when they reduce time on the ventilator in the first week or two of support. Examples of such strategies are protocols to improve methods of sedation administration [42, 43] and to accelerate weaning, either by protocolized methods [44] or by using noninvasive ventilation [45]. Furthermore, staffing levels may influence the length of stay of patients in the ICU, with an inverse relationship between adequacy of staffing levels and the duration of stay and subsequent development of VAP [46, 47].

Aspiration and nutrition. Aspiration of gastric or oropharyngeal contents that are contaminated with colonizing flora is important in the pathogenesis of VAP. The oropharynx appears to be the most important source for microorganisms. Continuous aspiration of subglottic secretions has been associated with significant reductions in the incidence of VAP in 2 randomized trials [33, 34] and with a strong tendency for such a preventive effect in 2 other randomized studies [35, 36]. In a fifth study, subglottic aspiration combined with topical oropharyngeal antimicrobial prophylaxis significantly reduced the occurrence of VAP [48]. The pooled relative risk reduction for VAP of subglottic aspiration (without concomitant use of topical antimicrobial prophylaxis) is 0.45 (95% CI, 0.07–0.95) (table 2).

Supine patient positioning may also facilitate aspiration, which may be decreased by semirecumbent positioning. Using radioactive-labeled enteral feeding, cumulative numbers of entodACHEal counts were higher when patients were placed in a completely supine position (0°), compared with a semirecumbent position (45°) [49, 50]. One randomized trial demonstrated a 3-fold reduction in the incidence of VAP in patients who were treated while in a semirecumbent position, compared with patients who were treated while completely supine [31]. Of note, the occurrence of infection in patients in supine position was strongly associated with the simultaneous administration of enteral nutrition. In a second randomized trial (which has only been published as an abstract to date), the supine position was compared to the standard of care, which appeared to be a head-up position of ∼12° [51]. In that multicenter study, a 45° patient position did not appear to be achievable, and only a mean position of ∼28° was achieved, which was not associated with a reduced incidence of VAP [32].

### Table 2. Relative risk (RR) reductions of pooled data of different intervention strategies for ventilator-associated pneumonia (VAP).

<table>
<thead>
<tr>
<th>Preventive strategy, reference(s)</th>
<th>No. of patients with VAP/total no. of patients</th>
<th>RR reduction (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Selective digestive decontamination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oropharyngeal, intestinal, and systemic prophylaxis vs. control [23]</td>
<td>16 226/1437 493/1446</td>
<td>0.54 (0.45–0.63)</td>
</tr>
<tr>
<td>Oropharyngeal, and intestinal prophylaxis vs. control [23]</td>
<td>10 89/624 165/638</td>
<td>0.45 (0.28–0.62)</td>
</tr>
<tr>
<td>Oropharyngeal, intestinal, and systemic prophylaxis vs. systemic prophylaxis [23]</td>
<td>6 107/475 195/640</td>
<td>0.26 (0.09–0.43)</td>
</tr>
<tr>
<td>Oropharyngeal prophylaxis vs. control [24–26]</td>
<td>3 13/125 70/181</td>
<td>0.73 (0.5–0.96)</td>
</tr>
<tr>
<td>Oropharyngeal and systemic prophylaxis vs. control [27]</td>
<td>1 13/58 23/30</td>
<td>0.71 (0.51–0.83)</td>
</tr>
<tr>
<td>Systemic prophylaxis vs. control [28]</td>
<td>1 12/50 25/50</td>
<td>0.52 (0.15–0.73)</td>
</tr>
<tr>
<td>Stress ulcer prophylaxis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sucralfate vs. ranitidine [29]</td>
<td>8 160/914 202/911</td>
<td>0.21 (0.05–0.38)</td>
</tr>
<tr>
<td>Postpyloric vs. gastric feeding [30]</td>
<td>7 60/221 81/227</td>
<td>0.24 (0.01–0.41)</td>
</tr>
<tr>
<td>Semirecumbent vs. supine patient position [31, 32]</td>
<td>2 15/151 19/156</td>
<td>0.24 (0.01–0.41)</td>
</tr>
<tr>
<td>Subglottic aspiration [33–36]</td>
<td>4 45/425 81/421</td>
<td>0.45 (0.23–0.61)</td>
</tr>
</tbody>
</table>
The summarized results of both trials lead to a relative risk reduction of 0.18 (95% CI, −0.39 to 0.76) (table 2).

**Stress ulcer prophylaxis.** Both H₂-antagonists and antacids have been identified as independent risk factors for VAP. By decreasing intragastric acidity and increasing intragastric volume (in the case of antacids), gastric colonization and aspiration can be stimulated, favoring the development of VAP.

Sucralfate has been postulated as an alternative agent for stress ulcer prophylaxis, because it does not decrease intragastric acidity (as do H₂-antagonists), nor does it increase gastric volume significantly (as do antacids). However, double-blind, randomized trials have failed to confirm the preventive effects of using sucralfate [52, 53]. In meta-analysis, ranitidine was, when compared with sucralfate, associated with a 4% higher incidence of VAP [29]. However, ranitidine provided better prevention against gastric bleeding in high-risk patients undergoing ventilation [53]. Moreover, the presumed association between gastric colonization and increased incidence of VAP has been questioned [52, 54, 55]. The summarized results of 8 trials comparing sucralfate with ranitidine for stress ulcer prophylaxis yielded a relative risk reduction for VAP of 0.21 (95% CI, 0.05–0.38) (table 2). Thus, although the use of sucralfate may offer some means of reducing the risk of VAP, as determined on the basis of current published data, this benefit may be more than offset by increased risk of gastrointestinal hemorrhage.

**Enteral nutrition.** Enteral nutrition has been considered a risk factor for the development of VAP, mainly because of an increased risk of aspiration [56]. However, its alternative (parenteral nutrition) is associated with higher risks of developing intravascular device–associated infections, complications of line insertions, higher costs, and loss of intestinal villous architecture, which may facilitate microbial translocation. For the latter, it has been advised to feed critically ill patients enterally as early as possible. However, a strategy of early (i.e., day 1 of ventilation) administration of enteral feeding for patients undergoing mechanical ventilation was, compared with late administration (i.e., day 5 of ventilation), associated with a higher risk for ICU-acquired VAP [57]. In another attempt to reduce aspiration, gastric nutrition has been compared with postpyloric feeding. Two studies using radioisotopes to compare the incidence of aspiration were not conclusive [58, 59]. Seven studies evaluated the risks for VAP in patients randomized to receive either gastric or postpyloric feeding [30]. Although significant differences were not demonstrated in any individual study, postpyloric feeding was associated with a significant reduction in VAP in meta-analysis (relative risk reduction, 0.24; 95% CI, 0.01–0.41) [30]. Few studies have evaluated the approaches of intermittent or acidified enteral feeding, and reductions in the incidence of VAP have not been demonstrated in any randomized trial [60, 61].

**Modulation of colonization.** Colonization of the upper respiratory tract is a prerequisite for the development of VAP. Oropharyngeal colonization, either present on admission or acquired during the ICU stay, has been identified as an independent risk factor for the development of VAP caused by enteric gram-negative bacteria and *P. aeruginosa* [54]. Modulation of oropharyngeal colonization, either by antibiotics [24–27] or chlorhexidine therapy [62], appears to be an effective measure to prevent VAP. Combined results of 3 studies of topical oropharyngeal antibiotics showed a relative risk reduction for VAP of 0.73 (95% CI, 0.50–0.96).

Oropharyngeal decontamination is, together with gastric and intestinal decontamination, one of the components of selective decontamination of the digestive tract (SDD). SDD has been associated with significant reductions in the incidence of VAP in most studies [23], although the methodological study quality appeared to be inversely related to the magnitude of the preventive effects [63]. Of note, the preventive effects of SDD for VAP are considerably lower in ICUs with high endemic levels of antibiotic resistance [64–67], and, in such settings, SDD will increase the selective antibiotic pressure for drug-resistant microorganisms [68–71]. The pooled data from 15 studies comparing patients who received the full SDD regimen with control patients yielded a relative risk reduction for VAP of 0.54 (95% CI, 0.45–0.63) [63] (table 2). Pooled data from 5 studies comparing patients who received the full SDD regimen with a control group of patients receiving systemic prophylaxis yielded a smaller relative risk reduction for VAP (0.26; 95% CI, 0.09–0.43), whereas data from 10 studies comparing only patients who received topical prophylaxis with control patients yielded a relative risk reduction for VAP of 0.45 (95% CI, 0.28–0.62) [23].

In addition to prevention of VAP, significant reductions of ICU-mortality in patients receiving SDD have been reported recently. After stratification based on APACHE II scores, Krueger et al. [72] randomized 265 patients in a double-blind design to receive a regimen containing intravenous ciprofloxacin for 4 days, as well as topical colistin and gentamicin applied to the nostrils, mouth, and stomach. Control patients received intravenous and topical placebo. The overall relative risk for ICU mortality was 0.76 (95% CI, 0.53–1.09), but, in the subgroup of patients with APACHE II scores of 20–29, the relative risk was 0.51 (95% CI, 0.3–0.88) [72]. In a second study, an impressive relative risk reduction in both ICU (36%) and hospital mortality (23%) for patients receiving SDD was demonstrated [73]. With almost 1000 patients enrolled, this was the largest trial of SDD performed thus far. The reduction in the mortality rate reported was the highest in any individual trial and even exceeded the most positive predictions for mixed populations in meta-analyses. Moreover, patients receiving SDD had a
shorter length of ICU stay, and fewer patients acquired colonization with antibiotic-resistant, gram-negative bacteria. Of interest, patients were not individually randomized to receive SDD or not, but 2 identical wards were randomized in an open design, and all admitted patients received the standard of care in their unit. If possible, patients were randomized to 1 of the 2 wards. However, because no cross-over was performed, it cannot be completely excluded that structural differences between the 2 wards might have influenced patient care. Therefore, these exciting findings should be confirmed. It is important to note that rate of colonization with drug-resistant, gram-negative bacteria and vancomycin-resistant enterococci at the start of study and at the time of patient admission were very low, and none of the patients were colonized with methicillin-resistant Staphylococcus aureus [73].

**Systemic antibiotics.** The role of systemic antibiotics in the development of VAP is unclear. In one study, prior administration of antibiotics had an adjusted OR of 3.1 (95% CI, 1.4–6.9) for development of VAP [15]. Moreover, antibiotics clearly predispose patients to subsequent colonization and infection with antibiotic-resistant pathogens [74]. In contrast, prior antibiotic exposure conferred protection (risk ratio, 0.37; 95% CI, 0.27–0.51) for VAP in another study [41]. Preventive effects of intravenous antibiotics were evaluated in only 1 randomized trial: administration of cefuroxime at the time of intubation reduced the incidence of VAP in patients with closed head injury [28]. Moreover, further circumstantial evidence for efficacy of systemic antibiotics also follows from the results of meta-analyses of SDD, which have suggested that the intravenous component of SDD is essential for improved patient outcome [23, 75].

**Ventilator circuit-related factors.** Although the majority of VAP episodes likely arise from aspiration of contaminated secretions around the endotracheal tube, in some circumstances, colonization of the ventilator circuit undoubtedly leads to lower airway contamination and, eventually, pneumonia. A large number of studies have been conducted to determine the magnitude of risk related to a number of ventilator circuit factors and management strategies. A large number of prospective, randomized trials have shown that the frequency of ventilator circuit change does not affect the incidence of VAP [76, 77]. Use of passive humidifiers with or without filtering capacity has been shown to decrease circuit colonization by bacteria, but it has not been shown to reduce the incidence of VAP, and, with regard to infection control, there is no benefit to changing passive humidifiers any more frequently than every 48 h [78–82].

**Combined interventions.** Although the optimal approach to reducing VAP is unclear, recent studies indicate that educating health care workers who care for patients who are receiving mechanical ventilation can decrease VAP rates [83–87]. In times of limited resources, focusing health care workers’ efforts on the prevention of VAP is important, especially given the association between inadequate staffing in the ICU setting and the occurrence of nosocomial infection [47, 88–90]. In addition, there are data supporting the benefit of education-based infection-control interventions targeting regional health care systems [91].

Recently, an education-based program with multiple interventions has been shown to reduce the occurrence of VAP at an academic medical center [84]. The centerpiece of this educational initiative was a 10-page self-study module, including information on the following topics related to VAP: (1) epidemiology and scope of the problem, (2) risk factors, (3) etiology, (4) definition, (5) methods to decrease risk, (6) procedures for collecting suctioned sputum specimens, and (7) clinical and economic outcomes influenced by VAP [84]. (The study module and self-examinations can be obtained as a CD-ROM through the Association for Professionals in Infection Control and Epidemiology.) Risk factors for VAP that were specifically addressed included those promoting aspiration (supine positioning and gastric overdistention) and those associated with bacterial colonization of the upper airway and stomach (prior antibiotic exposure and the use of stress ulcer prophylaxis). A section from the self-study module outlining specific risk-reduction strategies addressed in the infection-control policy is shown in table 3 [84, 92]. This integrated education-based system aimed at reducing VAP was subsequently taken to 2 community hospitals and a pediatric hospital for implementation [92]. Relative reductions in the occurrence of VAP of 38%–61% occurred in the hospitals that implemented this education-based program as part of their mandatory training for patient care providers [92].

Although multiple interventions to reduce VAP are available, studies show that they are not being widely implemented. Cook et al. [93] compared Canadian and French ICUs with regard to the use of 7 strategies to control secretions and care for ventilator circuits to prevent VAP and to reduce overall health care costs. Adherence to specific prevention guidelines for VAP was more common among French ICUs (64% vs. 30%; P = .002), but rates were low in both countries. These investigators also found that published recommendations did not appear to substantially affect whether prevention interventions were used within individual ICUs. Similarly, a European survey found that 37% of ICU practitioners were not following published recommendations for the prevention of VAP [94]. The most common reasons for nonadherence were disagreement with the interpretation of clinical trial findings (35%), lack of resources (31%), and costs associated with the implementation of specific interventions (17%).
How do I reduce the risk of VAP in my patients?

The primary intervention to prevent any nosocomial infection is hand washing. Careful infection-control practices related to respiratory care are also essential to preventing VAP. Health care workers should use the following recommendations for all patients receiving mechanical ventilation.

To prevent bacterial colonization of the aerodigestive tract:

- Meticulous hand hygiene with the use of soap and water or a waterless hand antiseptic agent is essential before and after ventilator contact or patient suctioning;
- Do not change ventilator circuits and/or in-line suction catheters unless visibly soiled or malfunctioning;
- Do not use HMEs for patients with excessive secretions or hemoptysis (be sure to provide alternative form of humidification);
- Change HMEs every 24 h or when visibly soiled with secretions;
- Drain condensate from ventilator circuits per policy, using appropriate technique to avoid contamination of the circuit.

To prevent aspiration of contaminated secretions:

- Maintain adequate ventilation and cuff pressure;
- Place ventilated patients in semirecumbent position with head of bed elevated to ≥30°, as tolerated, even during transport;
- Drain ventilator circuit condensate before repositioning patient;

To avoid gastric distention monitor gastric residual volumes before initiating gastric feedings via nasogastric, orogastric, or percutaneous gastrostomy tubes:

- Remove nasogastric tubes as soon as possible.

To reduce risk of VAP when suctioning a ventilated patient:

- Use clean gloves for in-line suctioning and sterile gloves for single use catheter suctioning;
- Do not store catheter where it can become contaminated or contaminate clean supplies;
- Oral suction catheters (e.g., Yankauer) should be stored in a nonsealed paper or plastic bag when not in use;
- Do not lay suction catheters on ventilator;
- Suction when necessary. Frequent unnecessary suctioning may introduce organisms into the lower respiratory tract.

Other key points to reduce the risk of VAP include the following:

- Avoid nasal intubation;
- Adequately secure endotracheal tube and take necessary measures to prevent accidental extubation;
- Avoid overuse of multiple antibiotics;
- Limit stress ulcer treatment if possible;
- Use daily chlorhexidine oral rinse (only for patients undergoing cardiothoracic surgery);
- Provide immunizations (e.g., influenza, pneumococcus, and *Haemophilus influenzae* B).

**NOTE.** Presented with permission from [92]. HME, heat moisture exchanger.

**CONCLUSIONS**

As summarized in table 2, along with routine measures to diminish aspiration, antimicrobial prophylaxis appears an effective strategy for preventing VAP. Importantly, in most studies assessing antimicrobial prophylaxis, VAP was diagnosed with bronchoscopic techniques, thereby minimizing the risk that the prophylactic antibiotics induced false-negative results of diagnostic cultures. Unfortunately, antibiotic resistance has become so prevalent in many ICUs that the benefits of prophylactic use of antibiotics for patients could be overcome by the increased selective pressure for antibiotic-resistant pathogens. The latter has been an important argument against the widespread use of such strategies [95, 96]. Moreover, the long-term effects of prophylactic antibiotic use on resistance development are unclear. On the other hand, improved patient survival resulting from antimicrobial prophylaxis, as has been suggested by 2 recent studies [72, 73], would necessitate a reappraisal of this balance. If these findings are confirmed in other settings, their application could well be advised, especially for high-risk patients in settings with low levels of antibiotic resistance or when specific problem antibiotic-resistant pathogens are encountered. It is therefore imaginable that, in the near future, baseline levels of antibiotic resistance will determine which infection-prevention strategies should be used. In settings with high levels of antibiotic resistance, a combined approach of different non–antibiotic using strategies in combination with education programs for health care workers might be most beneficial.

**References**


