Ventilator-Associated Pneumonia: Issues Related to the Artificial Airway

Emili Diaz MD PhD, Alejandro H Rodríguez MD, and Jordi Rello MD PhD

Pooling of contaminated secretions above the cuff of the endotracheal tube predisposes patients to ventilator-associated pneumonia (VAP). Subglottic secretion drainage requires a special endotracheal tube that has a separate lumen that opens in the subglottic region above the tracheal tube. A recent meta-analysis of the 5 randomized clinical trials that evaluated the efficacy of removing these secretions found that this technique significantly reduces the incidence of VAP. One cost-effectiveness analysis showed savings of $4,900 per episode of VAP prevented. Greatest benefit is derived by patients requiring fewer than 10 days of mechanical ventilation and not exposed to antibiotic therapy. Maintaining the intracuff pressure between 25 and 30 cm H_2O is mandatory to guarantee effective drainage and safety. While silver-coated endotracheal tubes reduce pseudomonas pneumonia in intubated dogs and delay airway colonization in intubated patients, evaluation of studies with a variety of case mixes is warranted to identify subsets likely to benefit from the technique before it is implemented on a large scale. A patient who has a colonized airway and who undergoes percutaneous tracheotomy has an increased risk of VAP, particularly due to *Pseudomonas aeruginosa*, in the week following the procedure. As many studies suggest that incidence of VAP is highly dependent on the strategies of airway management, health care workers should be alerted to issues related to the artificial airway. *Key words: ventilator-associated pneumonia, VAP, artificial airway, subglottic secretion drainage, silver-coated endotracheal tube, tracheotomy*. [Respir Care 2005;50(7):900–906. © 2005 Daedalus Enterprises]

Introduction

Implementing mechanical ventilation (MV) implies several changes in the patient's airways. The most important change when a patient is intubated is that the airway loses sterility and becomes colonized within a few hours of starting MV.[1] Many complications can occur in this situation. Ventilator-associated pneumonia (VAP) is the leading infectious complication in patients under MV, affecting from 8% to 28% of patients admitted in the intensive

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care unit (ICU).[2] The risk of VAP is present throughout the MV period, though it is greatest during the first days. Cook et al showed that the risk for VAP is 3% per day in the first week of MV, 2% per day in the second week, and 1% per day later.[3] Indeed, in this period airway care is critical in preventing VAP. From the ventilator to the lungs, all parts and pieces need to be considered when caring for patients on artificial ventilation.

Infection is due to a disequilibrium between host defenses, inoculum size, and microorganism virulence. The presence of a mechanical device inside the airways breaches the respiratory tract's first natural barrier against infection. In addition, co-existing acute or chronic conditions can affect the response to an infection (eg, impaired nutrition status, chronic obstructive pulmonary disease). Neither microorganism virulence nor prior host diseases are modifiable factors; however, the risk of VAP may be decreased by reducing the inoculum size.

There are several effective measures for VAP prevention. Overall rate of nosocomial infection can be reduced by hand-washing and removal of gloves between patients.[4] Specific measures for VAP reduction include oral endotracheal intubation, nonroutine changing of ventilator circuits, use of heat-and-moisture exchangers, and semirecumbent positioning.[4,5] Other measures recommended are subglottic secretion drainage (SSD) and strict control of intracuff pressure, though compliance is poor.[5–7] Other techniques, such as the use of silver-coated endotracheal tubes (ETTs), need to be analyzed. This paper reviews the role of tracheotomy in VAP development, the rationale for silver-coated ETTs, and the importance of intracuff pressure monitoring and randomized controlled trials for evaluating SSD.

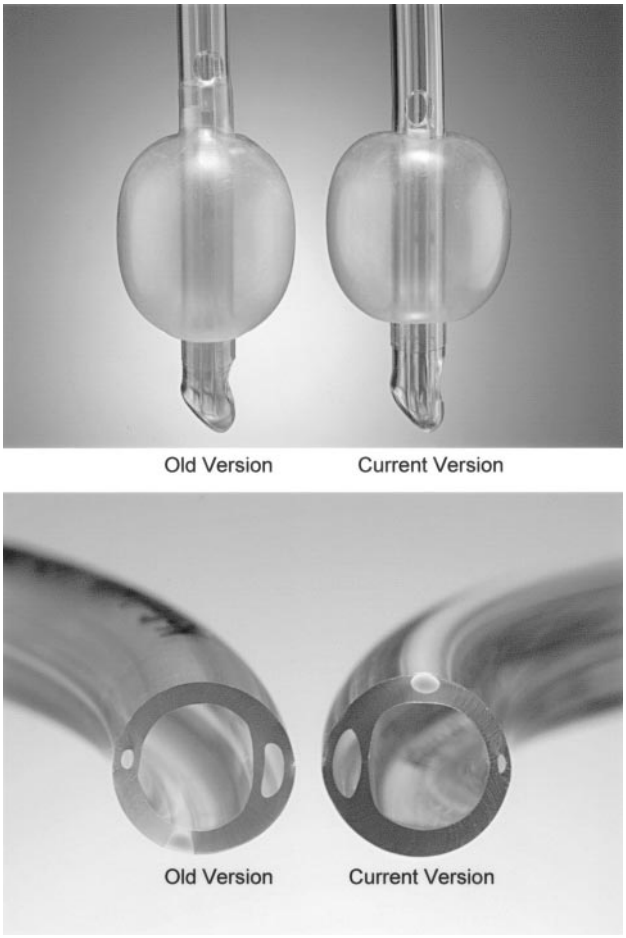

Fig. 1. Endotracheal tubes with dorsal lumen for subglottic secretion drainage. The dorsal lumen opens above the endotracheal cuff. In the current version this hole is closer to the cuff and the lumen is bigger.

Subglottic Secretion Drainage

The main pathogenic mechanism of VAP, especially during the first week of MV, is the aspiration of contaminated secretions from the oropharynx into the lower airway. In patients with endotracheal intubation these secretions are pooled above the cuff of the ETT and may leak around the ETT, entering the lower respiratory tract. SSD is designed to reduce this process.[4] This procedure is performed through a special ETT with a separate lumen that opens in its dorsal side above the cuff (Fig. 1). The dorsal lumen is connected to an evacuation system. Secretions can be drained either intermittently or continuously. With the continuous procedure, secretions accumulate in a mucus collector and are then voided regularly or before if full (Fig. 2). Soft aspiration (20–30 cm H_2O) is used, and the system is checked every 4 hours to assure that the suction lumen is patent. Recommendations for managing SSD are summarized in Table 1.

At least 5 randomized trials have assessed the role of SSD for preventing VAP.[8–12] Four of these studies were performed in patients expected to require more than 72 hours of MV.[8,9,11,12] Three studies[8,9,12] were done in a medical-surgical ICU, and another in a surgical ICU.[11] The remaining study[10] was performed in a cardiothoracic ICU, with MV periods of 1.5 and 1.9 days for SSD and control groups, respectively.

Vallés et al[8] prospectively analyzed 190 patients enrolled in the study. One hundred fifty-three fulfilled inclusion criteria (76 in the SSD group and 77 in the control group). Intracuff pressure was monitored every 4 hours by means of an aneroid manometer. The study protocol included checking the system permeability and intracuff pressure every 4 hours. VAP developed in 39 of the 153 patients, and the rate of VAP was twice as high for patients with standard ETTs (19.9 VAP/1,000 patient days for SSD, 39.6 VAP/1,000 patient days for standard ETTs, relative risk 1.98, 95% confidence interval 1.03 to 3.82). This

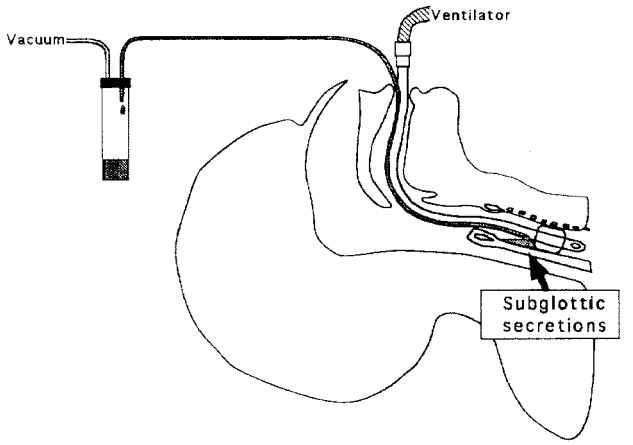

Fig. 2. Diagram of continuous subglottic secretion drainage. (From Reference 8, with permission.)

Table 1. Recommendations for Managing Subglottic Secretion Drainage System

1. Continuous soft aspiration with suction of 20–30 cm H_2O
2. Monitor intracuff pressure every 4 hours and maintain at 25–30 cm H_2O
3. Check system every 4 hours with 2 mL of air to assure that the suction lumen is patent
4. Check the system if no secretions are recovered in the mucus collector

decrease in VAP incidence was due to a significant reduction in episodes caused by Gram-positive cocci or *Haemophilus influenzae*. Episodes of VAP by *P. aeruginosa* and *Enterobacteriaceae* were not affected by SSD. In addition, time to VAP onset was twice as long in the SSD group: 12.0 ± 7.1 days in SSD patients versus 5.9 ± 2.1 days in control patients ($p = 0.003$).

Studies by Mahul et al,[9] Bo et al,[11] and Smulders et al[12] showed similar data, with risk reductions for VAP of odds ratio 0.46 (95% confidence interval 0.23 to 0.93), 0.35 (95% confidence interval 0.125 to 1.01), and 0.22 (95% confidence interval 0.06 to 0.81), respectively. In the study by Kollef et al,[10] performed in a cardiothoracic ICU, the incidence of VAP was 5% in patients with SSD and 8.2% in control-group patients, but the difference was not statistically significant (odds ratio 0.61, 95% confidence interval 0.27 to 1.40).

Recently, Dezfulian et al[13] published a meta-analysis based on these 5 studies, with a total of 896 patients. Table 2 shows a comparison between randomized trials and meta-analysis. SSD reduced the incidence of VAP by half (risk ratio 0.51, 95% confidence interval 0.37 to 0.71). In addition, patients under SSD treatment remained on MV 2 days less (95% confidence interval 1.7 to 2.3), shortened their ICU stay by 3 days (95% confidence interval 2.1 to 3.9), and delayed VAP onset by 6.8 days (95% confidence interval 5.5 to 8.1). As expected with a procedure that works mostly by reducing episodes of early-onset pneumonia, mortality was not affected by the use of SSD. Risk ratios of meta-analysis for VAP and mortality are shown in Figure 3.

The cost-effectiveness of ETTs equipped for SSD needs a special analysis. ETTs with a dorsal lumen for SSD may be 15 times more expensive than standard ETTs. Mortality is not reduced with SSD, but other outcome measures are modified: for instance, shorter period under MV, and shorter ICU stay. In 2001, Shorr and O'Malley[14] published a study of a hypothetical cohort of ICU patients requiring more than 72 hours of MV. Their study, mainly based on data from Vallés et al[8] for a VAP rate reduction, was a decision-model analysis. The cost was based on a 14-day course of antibiotics and a 5-day increase in length of ICU stay. The study showed that SSD use would result in a savings of $4,992 per case of VAP prevented or a saving of $1,872 per patient on MV. Using their meta-analysis, Dezfulian et al[13] found a saving of $3,535 per case of pneumonia prevented with SSD.

In addition to the efficacy of SSD in preventing VAP, Kollef et al[10] reported that patients treated with SSD ETTs presented no complications.

In summary, SSD is a useful method for preventing VAP, especially early-onset episodes. It can shorten the period on MV and appears to be cost-effective.

Control of Intracuff Pressure

Effective airway care with SSD requires careful checking. SSD needs to combine system permeability with adequate intracuff pressure level. If the system has lost permeability or if the ETT cuff has persistently low pressures, secretions can reach the lower respiratory tract around the cuff, increasing the risk of VAP.[4] This hypothesis was tested in a follow-up study.[15] SSD malfunction was suspected if no secretions were recovered from the mucus collector during a period of 24 hours. The permeability of the system can be checked by injecting sterile saline solution or air into the dorsal lumen. Intracuff pressure was monitored and kept between 25 and 30 cm H_2O. In multivariate analysis, failure of SSD (risk ratio 7.52, 95% confidence interval 1.48 to 38) and persistent intracuff pressure below 20 cm H_2O were associated with the development of pneumonia within the first 8 days of MV. However, endogenous microorganisms are eradicated by antibiotic exposure.[16] In patients under antibiotic therapy, failure of SSD did not significantly prevent the development of VAP (Table 3).

Currently, large-volume, low-pressure ETT cuffs are widely used. Intracuff pressure should be set to balance the risk of mucosal damage and the risk of VAP. In patients on MV, the use of low-pressure cuffs may increase the risk of VAP, whereas high-pressure cuffs may increase the risk of

Table 2. Studies With Subglottic Secretion Drainage

First Author, Year	*n*	Time to VAP (days)		VAP RR (95% CI)	Mortality RR (95% CI)
		SSD	Control		
Valles 1995[8]	190	12	5.9	0.47 (0.21–1.06)	1.09 (0.72–1.63)
Mahul 1992[9]	145	16.2	8.3	0.46 (0.23–0.93)	1.14 (0.62–2.07)
Kollef 1999[10]	343	5.6	2.9	0.61 (0.27–1.40)	0.86 (0.30–2.42)
Bo 2000[11]	68	14	6	0.35 (0.12–1.01)	NR
Smulders 2002[12]	150	NR		0.22 (0.06–0.81)	1,24 (0.49–3.07)
Dezfulian 2005[13]	896	SSD 3.1 days later		0.51 (0.37–0.71)	1.19 (0.82–1.71)

VAP = ventilator-associated pneumonia
RR = risk ratio
CI = confidence interval
NR = not reported
SSD = subglottic secretion drainage

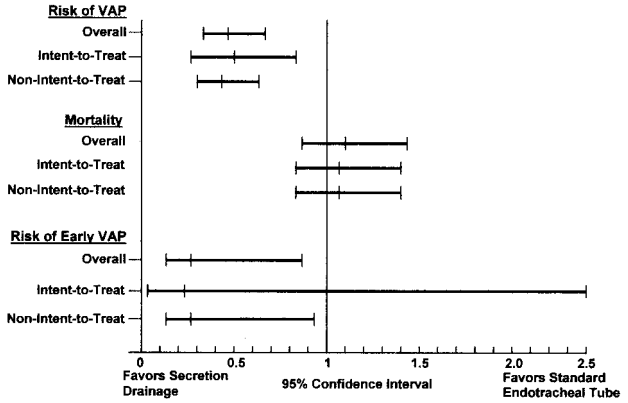

Fig. 3. Risk ratios of meta-analysis for ventilator-associated pneumonia (VAP) overall, early-onset VAP, and mortality. (Adapted from Reference 13.)

Table 3. Risk Factors for Pneumonia in Patients Treated With Subglottic Secretion Drainage Endotracheal Tubes: Relationship With Antibiotic Exposure

Variable	Full Cohort RR (95% CI)	Patients Without Antibiotic RR (95% CI)
Failure of continuous aspiration of subglottic secretions	5.29 (1.24–22.64)	7.52 (1.48–38.07)
Low intracuff pressure	2.51 (0.78–8.03)	4.23 (1.12–15.92)
Coma	1.71 (0.51–5.74)	NR
Continuous sedation	0.42 (0.12–5.74)	NR
Antibiotic use	0.10 (0.01–0.71)	NR
Previous cardiopathy	NR	2.17 (0.58–8.09)

(Adapted from Reference 15.)
RR = risk ratio
CI = confidence interval
NR = not reported

tracheal damage. Maintaining a pressure that isolates the respiratory tract without causing tracheal damage is the goal in any intubated patient. To prevent tracheal injuries the intracuff pressure should be maintained below 30 cm H_2O.[4] Seegobin and Van Hasselt[17] studied the effect of different intracuff pressure in patients who underwent surgery. Using an endoscopic photographic technique, they showed an impairment in tracheal mucosal blood flow with high intracuff pressure and recommended that mean intracuff pressure should not exceed 30 cm H_2O.

Therefore, SSD permeability and intracuff pressure should be checked regularly in order to avoid tracheal mucosal damage without increasing the risk of VAP. Newer designs with a wider, elliptic hole can facilitate drainage and reduce malfunction.

Silver-Coated Endotracheal Tubes

In recent years, several attempts have been made to decrease device-related infections by reducing bacterial biofilm formation. Antimicrobial-coated central venous catheters and urinary silver-coated catheters have been used in some hospitals with this objective. Indwelling devices are affected within a few hours of insertion by biofilm formation on both inner and outer surfaces. Biofilm formation in ETTs has been implicated in the pathogenesis of lower-respiratory-tract infections.[18] These biofilms can harbor a high level of bacteria concentration, and in addition these bacteria cannot be reached by antibiotics.

Silver has interesting medical properties. It prevents biofilm formation, has bactericidal activity, reduces bacterial burden, and reduces inflammation. Silver is approved for use in other preventive strategies, such as urinary catheters.[19] To test the potential bacteria-burden reduction in the respiratory tract with silver-coated ETTs, Olson et al[20] performed an experimental study in ventilated dogs. Eleven animals were included in the final study: 6 controls and 5 with silver-coated ETT. Intubated dogs with silver-coated ETT showed a delay in the appearance of aerobic bacteria

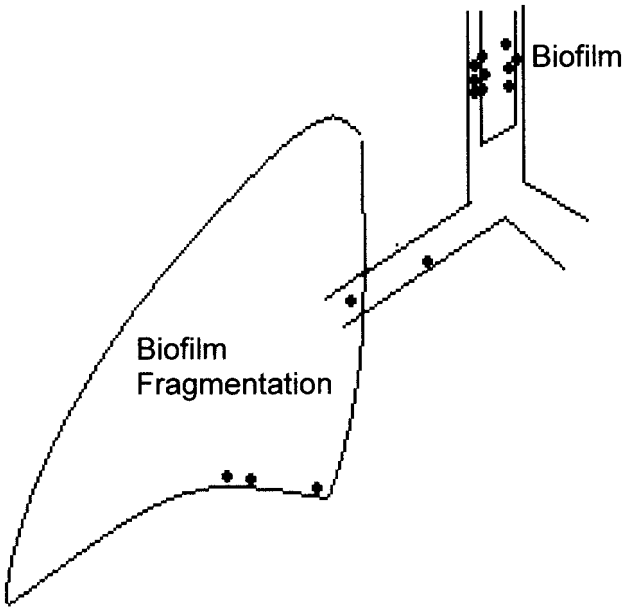

Fig. 4. Biofilm fragmentation with suction system. Biofilm is formed around the endotracheal tube. When biofilm fragmentation occurs, pieces with a high bacterial concentration can reach the lower respiratory tract.

on the ETT surface, especially *P. aeruginosa*,[20] and a lower bacterial burden in lung parenchyma (4.8 ± 0.8 vs 5.4 ± 0.9 log colony-forming units [CFU]/g lung tissue, $p = 0.01$, for silver ETT and control, respectively). Lung inflammation was analyzed by 2 veterinary pathologists. Necropsy studies showed the major lung findings to be congestion and hyperemia, with a higher degree for dogs intubated with standard ETT, for both pathologies.

A prospective, randomized, phase-II pilot study has recently been completed,[21] testing silver-coated ETTs in ICU patients. The main objective was to determine whether silver-coated ETTs reduce the incidence and/or delay the time of onset of colonization, when compared to non-coated ETTs in mechanically ventilated patients. In this study the proportion of patient days with quantitative endotracheal aspirates $> 10^5$ CFU and $> 10^6$ CFU was greater among patients with uncoated ETTs (26.7% vs 11.2%, $p = 0.01$, and 18.7% vs 5.2%, $p < 0.01$, respectively). Upon removal, microbial burden was found to be 55.9×10^6 CFU/mL and 38.8×10^6 CFU/mL in control and silver-coated ETTs, respectively. This high level of bacterial concentration in the inner surface of the ETT can play a role in the development of late-onset VAP when biofilm fragmentation occurs. This fragmentation can be facilitated while suctioning endotracheal secretions (Fig. 4). In this pilot study, blood silver analysis was performed at day 1 of MV and when the patient was extubated. No increase in blood silver or loss in the ETT was found.

These data confirm that silver-coated ETTs are safe, reduce bacterial biofilm, and can delay airway colonization. More information, in the form of randomized clinical trials, is warranted to determine their efficacy in preventing VAP.

Table 4. Main Differences Between 2 Recent Studies Evaluating Incidence of Pneumonia in Intensive Care Unit Patients Who Underwent Tracheotomy

Variable	Georges et al[27]	Rello et al[28]
Tracheotomy technique	Surgical	Percutaneous
Antibiotic prophylaxis	No	Yes
Pneumonia incidence (%)	25.9	18.1
Days before pneumonia onset (mean)	17.8	13
Days pneumonia onset after tracheotomy (mean)	8.7	7.0
Mortality pneumonia/ no pneumonia (%)	54.3/33.3	34.3/33.3

Tracheotomy

Prolongation of weaning and decrease of consciousness are the most frequent indications for tracheotomy in the ICU. The potential advantages of early tracheotomy over translaryngeal intubation have been debated in critically ill patients for more than 20 years. Four prospective, randomized clinical trials have evaluated the effects and timing of tracheotomy in short-term-ventilated critically ill patients.[22–25] The studies divided the population into early versus late tracheotomy, though the timing differed in all four. Patients were randomized to receive early or late tracheotomy, with different cut-offs: at days 3–4 versus 14 in one study,[22] before or after 7 days,[23] at days 3–5 versus days 10–14,[24] and finally within the first 48 hours of MV versus days 14–16.[25] The first 3 studies had 289 patients,[22–24] and the relative risk for VAP was 0.88 (95% confidence interval 0.70 to 1.10), suggesting that early tracheotomy does not prevent VAP development. In 2004 Rumbak et al[25] published a prospective, randomized trial to test the timing of tracheotomy in 120 patients expected to require more than 2 weeks of MV. Patients in the tracheotomy group had lower incidence of pneumonia (5% vs 25%) and lower mortality (31.7% vs 61.7%). However, though both groups had similar baseline characteristics, early tracheotomy patients spent less time on MV than the prolonged-intubation group (7.6 d vs 17.4 d) and less time in the ICU (4.8 d vs 16.2 d).

Kollef et al[26] reported the presence of tracheotomy as a risk factor for VAP. Five hundred twenty-one patients were included in the study: 77 were diagnosed with VAP, 24 (31.2%) of whom had had a previous tracheotomy. Having a tracheotomy was selected in the multivariate analysis as an independent risk factor for VAP (odds ratio 3.14, 95% confidence interval 2.18 to 4.50).

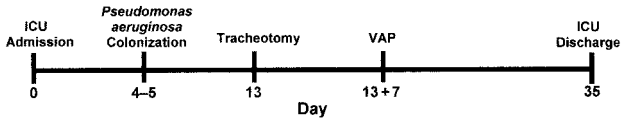

Fig. 5. Scheme of ventilator-associated pneumonia (VAP) onset in patients who underwent percutaneous tracheotomy. ICU = intensive care unit. (Data estimated from References 28 and 29.)

Recently, 2 observational studies have evaluated the role of tracheotomy in the development of VAP in patients on MV.[27,28] Differences between the studies are summarized in Table 4. Notably, Georges et al[27] used surgical tracheotomy, and the study by Rello et al[28] was performed in patients with percutaneous dilational tracheotomy. Georges et al[27] aimed to assess the incidence of nosocomial pneumonia and risk factors for pneumonia in patients having surgical tracheotomy. One hundred thirty-five patients were studied, with a mean of 17.8 days on MV before surgical tracheotomy. Thirty-seven episodes of VAP were diagnosed in 35 (25.9%) patients 8.7 days after tracheotomy. Episodes were divided into early (within 5 d after surgical tracheotomy) or late (more than 5 d after surgery). The presence of hyperthermia was a risk factor for both early and late VAP, whereas the presence of positive endotracheal aspirates with more than 10^5 CFU/mL of pathogens and the maintenance of sedation more than 24 hours after surgery were associated with early VAP. Late VAP was also associated with sedation before tracheotomy.

Our group performed a prospective study to determine the epidemiology in patients with tracheotomy receiving short-term MV.[28] Ninety-nine patients with percutaneous dilational tracheotomy were studied. There were 18 VAP episodes, diagnosed a median of 7 days after the tracheotomy (Fig. 5). This distribution contrasts with the lack of pneumonia episodes in the week prior to the procedure. Gram-negative bacilli were the etiological agents in 88.8% of VAP. *P. aeruginosa* was the most frequent single etiological agent in VAP in patients who underwent tracheotomy (8/18). The other microorganisms were *Acinetobacter baumannii* (2), *Klebsiella pneumoniae* (2), *Proteus mirabilis* (2), *Escherichia coli* (1), *H. influenzae* (1), methicillin-resistant *Staphylococcus aureus* (1), and methicillin-sensitive *S. aureus* (1). In patients with tracheotomy, no clinical variables were related to development of VAP, Nor was mortality affected by VAP in this group of patients.

Tracheotomy in the ICU setting has been used to facilitate weaning from the ventilator. Early tracheotomy in a medical ICU has been reported to reduce the period of ventilation and may thus reduce the risk of pneumonia. However, most episodes of pneumonia occur in the week after the tracheotomy, often caused by *P. aeruginosa*. Although the development of surgical site infection with the percutaneous dilational procedure is unlikely, antibiotic prophylaxis should be considered in order to reduce the consequences of bacterial aspiration during the procedure and subsequent development of VAP, because the risk of VAP is increased. Empirical therapy for VAP after tracheotomy should be based on agents active against *P. aeruginosa*.

Summary

Incidence of VAP is highly dependent on the quality of airway management by health care workers. Artificial-airway care for preventing VAP needs a multidisciplinary approach involving physicians, nurses, and respiratory therapists. Within the first week of MV, several preventive measures aimed to reduce the inoculum into the airway may reduce the development of VAP. The role of previous antibiotic administration is critical, and the potential role of biofilm and interventions aimed at reducing it need further consideration. Health care workers should receive active education on issues related to the artificial airway, because strategies of management have a key role in development of VAP.

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Discussion

MacIntyre: I remember when the subglottic drainage system came out. I thought that was a pretty cool idea, and we tried it for awhile. The problem is that it's just very hard to use. It would get clogged up fairly easily. There was also some concern about tracheal trauma from it. And it is a more expensive system, so it was difficult to get our staff to either use it initially or to change endotracheal tubes. Is there anything on the horizon that can do subglottic suctioning that's going to be easier to use, more reliable, and perhaps a little bit cheaper?

Rello: I agree that the cost is higher, but I think that the cost estimation analysis is more important than the individual cost in a specific patient. Second, I strongly discourage the replacement of a patient intubated with a standard endotracheal tube by an SSD tube, because the benefit probably will be lost. I think it is compensated by risk of pneumonia due to reintubation. We *never* replace tubes, and I think that what is important is that professionals who should perform an intubation in a patient who is expected to be under mechanical ventilation longer than 72 hours should have the tube available from the beginning.

The third point is safety. I think that safety and efficacy are highly related with the control of intracuff pressure. Indeed, an article, which dealt with sheep, that was reported in *Critical Care Medicine* in November[1] will be answered by a letter by Vallés et al, who have more than 10 years of experience with the SSD tube.[2] It is not equivalent to compare a patient in supine position with a sheep ventilated in prone position; in addition, larynx anatomy is different in sheep. Moreover, authors do not report how often intracuff pressure was monitored to maintain it at 25 cm of water. Finally, the model of pneumonia is completely different, because the animals developed *Pasteurella multocida* pneumonia. That flora is not present in ventilator-associated pneumonia.

In necropsies of patients who died, tracheal injury was not recognized. Endogenous pathogens such as *H. influenzae* were uncommon, but these organisms correlated closely with the patient being transferred from other hospitals with conventional tubes and failure to check the intracuff pressure. In all patients, either with the standard tube or with the SSD tube, a pressure above 30 cm H_2O should be avoided. Our practice was to check the intracuff pressure every 4 hours, and it was our objective to maintain it between 25 and 30 cm H_2O. An experimental study reported that no injury in the mucosa is associated with pressures under 30 cm H_2O.

The other observation is that over 5 years in which more than 2,000 patients have received SSD tubes in my former hospital, only 1 patient required surgery for tracheal stenosis. I worked in another hospital 5 years ago; only 1 patient has required surgery for tracheal stenosis. That patient had Down syndrome, he was ventilated in the prone position, he had serious problems of communication, and he was agitated. Probably all those conditions contributed to the injury.

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Solomkin: I want to bring up some experience with yeast, which is in biofilms. If organisms are recovered from the biofilm, for example by sonication, they turn out to have a much more drug-resistant pattern. Do you have any experience with recovering biofilm organisms to look at their susceptibilities in that way? Also, did you look at the MICs [minimum inhibitory concentrations] through time with those organisms?

Rello: The data I presented are preliminary data that are being analyzed currently and will be presented at the American Thoracic Society conference in San Diego.[1] I have no information on sensitivities, but it is probable that bacteria that survive in the biofilm are more resistant.

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Solomkin: I suspect the silver is blocking the development of resistance.

Rello: I have no objective data.

Niederman: In that paper in *Critical Care Medicine*,[1] didn't they demonstrate an interaction between patient position and the use of the SSD tube? And for efficacy? It seemed like it worked better when they were supine, as opposed to upright. Do you have any thoughts about patient positioning in relation to the effectiveness of the subglottic secretion drainage?

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Rello: It was not specifically analyzed.

Niederman: They had 3 animal groups, one that was using the tube upright and one that was using it supine, and, if I remember correctly, it was more effective when they were supine than when they were upright.

Rello: It is expected that the pool of secretions is down and the lumen is opening to the dorsal way of the tube. I hope that it will be effective in patients in supine positions; probably the effect is lost in patients in prone position.

Niederman: But I think they said it was less effective in the semi-recumbent position. In other words, we've emphasized the 30 degrees as being important, but if you're supine, you have less chance of leaking around the cuff, whereas if you're upright there's more chance of leaking around the cuff. We've focused on the semi-recumbent position as an advantage to avoid gastric aspiration, but we haven't really talked about what effect that position has vis-à-vis aspiration around the cuff. You can imagine that it would be the reverse.

Rello: Unpublished data with a follow-up longer than 6 hours in patients in semi-recumbent position suggest that the effect may be lost after 8 hours. Our policy was to maintain patients in the semi-upright position, approximately 30 degrees, rather than 45 degrees. Most of the time it's possible, but obviously, when the patient is transferred to a CT [computed tomography scanner], if he is in shock, if the nurses need to clean him, or in other conditions, semi-upright condition is not maintained. So my interpretation is that the benefit of semi-recumbent position is due to a reduction of bacterial burden aspirated to the lungs. And probably both systems work in a synergistic way, but it is just an opinion.

Kollef: In your study it seemed like tracheostomy was a risk factor for pneumonia, particularly maybe with pseudomonas, but there are some studies that suggest that *early* tracheostomy may reduce the occurrence of VAP. Do you think that has to do with whether those patients are already colonized with an organism like pseudomonas at the time that they get the tracheostomy, and if that's the case and you are recommending prophylaxis, would that be an argument to support earlier tracheostomy?

Rello: This is a difficult question to answer. I was specifically reviewing the literature on this point, and there are 4 multicenter, randomized, prospective trials comparing early tracheostomy with late tracheostomy. One paper, from 1997, did not report any difference.[1] It determined the effect of early (days 3–5) versus late (days 10–14) tracheostomy on ICU length of stay. The other paper, from 1990, concluded that early tracheostomy has an overall risk equivalent to that of tracheal intubation.[2] Group 1 patients

were to receive tracheostomy within 1 to 7 days, and group 2 patients underwent tracheostomy 8 or more days after admission. The third study[3] was reported in 1984, and it compared 34 patients receiving early tracheostomy with 40 who underwent late tracheostomy. That manuscript concluded that patients could undergo translaryngeal intubation for up to 2 weeks without significantly increased complications, including pneumonia relative to translaryngeal intubation.

In 2004 a paper in *Critical Care Medicine*[4] compared tracheostomy on the first day of ICU admission versus 2 weeks after admission, and it was reported that the incidence of pneumonia was lower. But the days of mechanical ventilation were significantly lower in the early-tracheostomy group, so I think that, to the best of my interpretation, the effect is to reduce the period of mechanical ventilation, reducing the number of days exposed to risk of VAP.

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St John:* I just want to add a comment to Neil MacIntyre's about subglottic suctioning, about the lumens being reported to occlude—

MacIntyre: Not just reported; it happens all the time in my unit.

St John: About 6 months ago there was a change introduced in the design of the Hi-Lo Evac tube. The lumen was made about 75% larger than the original design, and the dorsal lumen opening was lowered slightly, a little bit closer to the cuff. Now we've done a fair amount of internal benchtop testing with simulated laboratory secretions at different viscosities, and have shown a pretty big improvement in suctioning efficiency. We're hopeful that these improvements will help.

Hess: Something that comes up in the ICU every so often in tracheostomized patients is whether we should routinely use an inner cannula to decrease the risk of VAP—change those out every so many hours, or every day, and so forth. Whenever this comes up, I do a literature search, and again I can't find anything in the literature. Is there anything out there that I'm missing that shows that if you use an inner cannula in your tracheostomy tubes and change them on a regular basis, it decreases VAP?

Rello: Our practice is to change the inner cannula every day, but this is not an evidence-based decision. Another aspect and a potential question is if there are any benefits of SSD in patients with tracheostomy. In my opinion, if the benefit is focusing on other pathogens than *Pseudomonas aeruginosa*, and in patients with early-onset pneumonia, the benefit is unlikely. Our current policy is to perform tracheostomies mainly in patients with difficulty of weaning.

Branson: We don't do any prophylaxis before we do a tracheostomy. Are you doing it for the surgical procedure itself, and if you are, what are you giving?

Rello: Our consideration was that the airway is contaminated in intubated patients, and so that is a surgical procedure in a contaminated area, and our policy was to give a single shot of an antibiotic that was initially a nonantipseudomonal agent, such as amoxicillin-clavulanate or first-generation cephalosporin. The hypotheses was to find a correlation between the pathogens present in the airway at the moment of the procedure—or the day before—with infection. However, it was not a statistical correlation between the pathogen identified in pneumonia and tracheal aspirate. Full data are shown in the manuscript. I am interested to know Dennis Maki's opinion about the indication of surgical prophylaxis in this process.

Maki: It's probably not unreasonable to give one dose.

Solomkin: With percutaneous technique it's very difficult to imagine how you're going to get a surgical-site infection. We do not give prophylaxis. We are working very hard to reduce antibiotic exposure; and to cover what was there in that kind of a patient we would probably have to go to something like vancomycin and piperacillin/tazobactam, which I just don't think can be really justified.

Rello: What is true is that 15 years ago, when we performed surgical tracheostomies, the incidence of surgical wound infections in tracheostomies in the ICU was high.

Solomkin: That was with an open technique?

Rello: Yeah. And with percutaneous procedures it is very unlikely.

Kallet: Something occurred to me when you mentioned that your staff checks cuff pressures every 4 hours. I remember years ago when we had the manual systems with the long tubing

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connected to the pressure manometer, that the dead space of the tubing sometimes lowered the cuff pressure, and I would actually create a leak. So I'm now concerned about the risk of tracheal aspiration just from frequently monitoring cuff pressures in this way. We now use a spring-loaded device with a visible color indicator that allows you to check the cuff pressure without having to hook up an extra device for that. I was wondering what device you use, and that's actually an open question for other members of the panel.

Rello: I don't remember the exact number, but the information is shown in the manuscript. It is performed every 4 hours, by agreement between staff and according to the prior evidence in some patients; after 4 hours many patients had pressures under 30 cm H_2O. We were even analyzing, in a pilot study of 4 patients, a specific device that automatically maintained the cuff pressure. But we didn't receive economic support to do a randomized clinical trial, and we didn't know what is the effect of continuously maintaining the pressure near 30 cm H_2O.

Kallet: Has anyone brought to your attention that, when trying to check the pressure itself, they had problems with just almost creating a leak or the patient coughing? Has anyone relayed that to you?

Rello: No.

Kallet: I would like Rich Branson's and Dean Hess's opinion on that also.

Hess: A technique that's been used for many years to clear secretions from above the cuff—I don't know that it's ever been studied—is to apply positive pressure and let the cuff down, and then with the Yankauer clear the secretions out of the pharynx. So I guess that's one consideration in relation to what you're saying.

As far as technically what we use, we have a homemade system that is a low-compliance, low-volume system, so it doesn't let very much air out of the cuff when we make the measurements. One of the things that is a paradigm shift for many respiratory therapists is the idea of checking the cuff pressure and keeping it between 20 and 25 mm Hg, which I think relates to the 25–30 cm H_2O that Jordi Rello pointed out. Many respiratory therapists are used to a minimal-leak, minimal-seal technique, which I point out is a minimal-aspiration technique.

MacIntyre: Ted Kolobow, I think, a number of years ago, developed an endotracheal tube that provided jet ventilation and had this very interesting series of little flaps that wrapped around the tube. I think they used it only in animals; I don't think they ever did it in humans. The jet would ventilate the animal, but the exhalation was allowed to go around these little flanges and would actually sort of blow the material out around the tube. They claimed that not only did it provide good ventilation, but it was a good airway toilet technique as well. I'm not sure it ever caught on, or ever got to human stages; does anybody know?

St John: I'm not aware of any human data on it, but I was aware of the animal data, and you're right; it was like gills of a sort.

Branson: Yes. That's what we called them. That was Miroslav Klain.[1] Anyway, the problem was that it depends on constant airway pressure, so if the ventilation was ever interrupted, the patient would aspirate everything that was above the cuff. I have to say that I don't even know if the therapists routinely measure cuff pressures at our place. They inflate the cuff so that it seals and there's no leak. Then at that point the cuff pressure is irrelevant.

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