Impact of Invasive and Noninvasive Quantitative Culture Sampling on Outcome of Ventilator-Associated Pneumonia
A Pilot Study


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We performed an open, prospective, randomized clinical trial in 51 patients receiving mechanical ventilation for more than 72 h, in order to evaluate the impact of using either invasive (protected specimen brush [PSB] and bronchoalveolar lavage [BAL] via fiberoptic bronchoscopy) or noninvasive (quantitative endotracheal aspirates [QEA]) diagnostic methods on the morbidity and mortality of ventilator-associated pneumonia (VAP). Patients were randomly assigned to two groups: Group A patients (n = 24) underwent QEA, PSB, and BAL; Group B patients (n = 27) underwent only QEA cultures. Empiric antibiotic treatment was given according to the attending physician and was modified according to the results of cultures and sensitivity in Group A using PSB and BAL results and in Group B based upon QEA cultures. Bacteriologic cultures were done quantitatively for EA, PSB, and BAL. Thresholds of $\geq 10^5$, $\geq 10^3$, and $\geq 10^4$ CFU/ml were used for QEA, PSB, and BAL, respectively. Microbial cultures from Group A patients were positive in 16 (67%) BAL samples, 14 (58%) PSB samples, and 16 (67%) QEA samples. In Group B patients, QEA microbial cultures yielded positive results in 20 of 27 (74%) samples. In Group A, there was total agreement between culture results of the three techniques on 17 (71%) occasions. In five (21%) cases, QEA coincided with either BAL or PBS. In only two (8%) cases, QEA cultures did not coincide with either PSB or BAL. No cases of positive BAL or PSB cultures had negative QEA cultures. Initial antibiotic treatment was modified in 10 (42%) patients from Group A and in four (16%) patients from Group B (p < 0.05). The observed crude mortality rate was 11 of 24 (46%) in Group A, and 7 of 27 (26%) in Group B, whereas the adjusted mortality rates (observed crude minus predicted at admission) for Groups A and B were 29 and 10%, respectively. There were no statistically significant differences when comparing crude and adjusted mortality rates of Groups A and B. There were no differences in mortality between both groups when comparing pneumonia, considering together Pseudomonas aeruginosa and Acinetobacter spp. (Group A, 33% versus Group B, 27%). There were no differences between Groups A and B with regard to ICU stay duration and total duration of mechanical ventilation. In this pilot study, the impact of bronchoscopy was to lead to more frequent antibiotic changes with no change in mortality. Further studies with larger population samples are warranted to confirm these findings. Sanchez-Nieto JM, Torres A, Garcia-Cordoba F, El-Ebiary M, Carrillo A, Ruiz J, Nuñez ML, Niederman M. Impact of invasive and noninvasive quantitative culture sampling on outcome of ventilator-associated pneumonia: a pilot study.


The accurate diagnosis of ventilator-associated pneumonia (VAP) remains controversial (1-3). Despite more than 10 yr of clinical and animal investigation, no clear agreement has been reached in the medical community about which technique is to be routinely used to diagnose VAP. The knowledge of the histopathologic and microbiologic aspects of human VAP has accentuated the controversy about methods that were previously considered as unequivocal reference techniques (4-8). Recently, several studies (9-12) have suggested that the use of quantitative cultures of endotracheal as-
pirates (QEA) may have a similar diagnostic value compared with invasive techniques such as protected specimen brush (PSB) and bronchoalveolar lavage (BAL). The advantage of QEA is its reliance on the simplicity and cost-effectiveness of the method, as well as the lack of side effects. In fact, it has been suggested that using lavage in mechanically ventilated patients with pneumonia can lead to systemic and sepsis-like effects (13). Furthermore, deterioration in blood gas exchange has been described (14, 15).

For all of these reasons, we decided to carry out a prospective, randomized study to compare the impact on mortality and morbidity of using either invasive (PSB plus BAL) or noninvasive (QEA) techniques to diagnose VAP and to guide antibiotic treatment. In the group of patients undergoing invasive techniques, we also assessed the agreement between microbial cultures of invasive and QEA cultures.

METHODS

Study Population

Between March 1993 and December 1994, we prospectively studied 51 consecutive patients needing mechanical ventilation who had been admitted to the Intensive Care Unit (ICU) of a 1,000-bed teaching hospital that cares for patients from multiple disciplines including surgery and medicine but excluding pediatrics. These patients were suspected of developing nosocomial pneumonia. They were randomly assigned to one of the two groups, each with a different method for etiologic diagnosis of VAP. The causes of ICU admission are shown in Table 1.

For the purpose of the study, criteria for inclusion in the suspected pneumonia groups included the presence of new and/or progressive chest radiographic infiltrates appearing after 48 h of mechanical ventilation, and at least two of the following: fever > 38.3°C or hypothermia < 35°C, leukocytosis > 12,000/mm³ or leukopenia < 4,000/mm³, or purulent respiratory secretions. The study excluded patients with immunosuppression (AIDS, transplantation, cancer chemotherapy, blood malignancies).

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Patients were randomly (ratio 1:1) assigned using a computer-generated randomization table into one of the two groups, each with a different method for establishing an etiologic diagnosis of VAP. Group A patients (n = 24) underwent the following diagnostic procedures: quantitative endotracheal aspiration (QEA), then fiberoptic bronchoscopy with protected specimen brush (PSB) sampling and bronchoalveolar lavage (BAL). Group B patients (n = 27) underwent only QEA. Patients from Groups A and B were consecutively included in the study upon clinical suspicion of pneumonia.

All patients from Groups A and B received antibiotic treatment for pneumonia when clinically suspected and after protocol sampling was done. The antibiotic regimen was chosen according to the decision of the attending physician. Antibiotic modifications were performed on the basis of culture and sensitivity results of PSB and/or BAL in Group A, and QEA in Group B. A nitrofuran based modification was also done if patients did not present adequate clinical response evaluated 72 h after beginning of treatment.

In Group A, four patients (17%) did not receive antibiotics before sampling, whereas eight patients (30%) from Group B did not receive antibiotics before QEA sampling. In any case, prior (before pneumonia episode suspicion) antibiotic treatment was specifically not given for pneumonia. In all cases of Groups A and B receiving prior antibiotic treatment, this was withheld at least 12 h before the sampling techniques. For the purpose of the study, initial empiric antibiotic treatment was considered inadequate if the isolated microorganisms were not susceptible (10). Inadequate clinical response at 72 h was considered if there was persistence of fever, leukocytosis, or worsening of pulmonary infiltrates on chest radiographs. Family members in each case gave informed consent for the study to be performed, and permission from the Ethical Committee of our center was granted.

Data Collection

The following variables were recorded from patients in the two groups: age, sex, and APACHE II score on admission. Mean arterial blood pressure and body temperature were measured before, 2 h after, and at 6 and 10 h after the performance of invasive diagnostic techniques. Multiple organ failure (MOF) score, and PaO₂/FiO₂ ratio were measured before and 24 h after bronchoscopy in Group A patients, and at the same intervals for Group B patients. Other variables measured were: serum albumin on admission, hours without antibiotics before sampling, length of stay in the ICU, mechanical ventilation period, and antibiotic cost/patient/day.

Study Protocol

First, QEA were obtained by sterile means from Groups A and B patients using a 22-inch, No. 14 Fr suction catheter and collected in a mucus collector (Mocstrap®, Products Clinics S.A., La Llagosta, Barcelona). A length of approximately 24 cm of the catheter was passed through the endotracheal tube, and secretions were aspirated without instilling saline. These samples were retrieved for quantitative microbial cultures. Then without interrupting mechanical ventilation, through the endotracheal tube and using a special adaptor, the fiberoptic bronchoscope (BF30; Olympus, New Hyde Park, NY) was introduced 10 min later without bronchial suctioning after adequate sedation and curarization for Group A patients. The setting of the ventilator was adapted appropriately during the procedure to ensure proper ventilation and oxygenation. Local anesthetics were not used. Finally, a PSB (Microbiology brush no. 7310; Mill-Rose Laboratory Inc.) sample was retrieved from the area of maximal inflammation corresponding to the area of chest radiograph abnormality in Group A. After PSB sampling, the fiberoptic bronchoscope was wedged into a subsegmental bronchus from the same area where PSB was performed. Seven aliquots of sterile saline (20 ml each) were instilled and aspirated. The first two aliquots were discarded. The mean BAL fluid obtained for processing was 45 ± 20 ml. PSB procedure was always performed before BAL sampling to avoid bacterial contamination from the bronchoscope channel. The following threshold of bacterial cultures were used to distinguish colonization from true infection: QEA, 10³ CFU/ml; PSB, 10⁴ CFU/ml; BAL, 10⁶ CFU/ml (8, 10). Before the protocol procedures, three blood samples were taken for culture from each patient.

Microbiologic Processing

Endotracheal samples were mechanically homogenized using glass beads and were vortexed for 1 min. PSB samples were aseptically cut into a sterile tube containing 1 ml of Ringer’s lactate and vortexed for 1 min. Serial dilutions (10⁻¹, 10⁻², 10⁻³) from each PSB and BAL sample were prepared in sterile normal saline. One hundred microliters of each dilution were inoculated into the following agar media: 5% sheep blood, chocolate, Centers for Disease Control (CDC) blood, M CConkey, blood coalcol yeast extract (BCYE-a), and Sabouraud dextrose. A II cultures were aerobically incubated at 37°C under aerobic and anaerobic conditions and in a CO₂-enriched atmosphere. Cultures were evaluated for growth 24 and 48 h later and discarded, if negative, 5 d after, except for CDC and Wilkins-Chalgren media, which were evaluated at 7 d and for Sabouraud medium which was evaluated at 4 wk. A II microorganisms isolated were identified by standard laboratory methods (17).
Statistical Analysis

The study was initially designed to detect a 50% reduction (from 40% in Group B to 20% in Group A) in crude mortality rates. Accordingly, the calculated sample size was 182 patients distributed randomly into the two study groups (1:1). Because we observed that the rate of inclusion was lower than expected, and because this type of study could not be performed as a part of a multicenter study because of intercenter variability in epidemiologic factors and methodologic procedures, we decided to discontinue the study and present the data of 51 patients as a pilot study. Besides, at the time of interruption, the crude mortality rate in Group B was higher, although not significantly, compared with that in Group B, which is clearly inconsistent with the initial hypothesis. Results are expressed as mean ± SD. The test for quantitatively different variables and the chi-square test (Fisher’s exact test when needed) to compare proportions were used. All p values are two-sided, and the level of significance was set at 5%. Predicted hospital mortality rates were calculated from APACHE II scores and the diagnostic category scores according to Knaus and colleagues (18). Adjusted mortality rates were compared by using the chi-square test (Fisher’s exact test when needed) to compare proportions. All p values are two-sided, and the level of significance was set at 5%. Predicted hospital mortality rates were calculated from APACHE II scores and the diagnostic category scores according to Knaus and colleagues (18). Adjusted mortality rates were compared by using the chi-square test (Fisher’s exact test when needed) to compare proportions. All p values are two-sided, and the level of significance was set at 5%. Predicted hospital mortality rates were calculated from APACHE II scores and the diagnostic category scores according to Knaus and colleagues (18). Adjusted mortality rates were compared by using the chi-square test (Fisher’s exact test when needed) to compare proportions. All p values are two-sided, and the level of significance was set at 5%.

RESULTS

A total of 51 patients divided in two groups were studied: Group A (n = 24) and Group B (n = 27). General characteristics of the study population are shown in Table 2. There were no significant differences between Group A and Group B with regard to sex, P < 0.05. Predicted mortality on admission was defined as the difference between the number of predicted hospital deaths.

Microbiological Results

Microbial cultures from Group A patients were positive (above the mentioned threshold for each technique) in 16 (67%) of BAL samples, 14 (58%) PSB samples, and 16 (67%) QEA samples. In Group B patients, QEA microbial cultures yielded positive results in 20 of 27 (74%).

The most frequently isolated microorganisms in Group A patients were Pseudomonas aeruginosa (n = 13) and A cinetobacter calcoaceticus (n = 5). In the other hand, in Group B patients, the most frequently isolated microorganisms were A cinetobacter calcoaceticus (n = 8), Streptococcus pneumoniae (n = 5), and Hemophilus influenzae (n = 5). The isolated microorganisms from the different techniques in both groups are shown in Table 3.

Agreement among Microbiologic Techniques (Group A)

In Group A, there was total agreement between culture results of the three techniques on 17 (71%) occasions (11 patients with positive cultures and six with sterile cultures). In five (21%) patients, QEA coincided with either BAL or PSB (partial agreement). In only two (8%) patients, one positive and one negative, QEA cultures did not coincide with either PSB or BAL. No cases of positive BAL or PSB cultures had negative QEA cultures.

Antibiotic Treatment

Thirty-nine (76%) patients from Groups A and B received prior antibiotic treatment (either monotherapy or combination therapy) before inclusion in the protocol for fever or suspicion of sepsis. In no case was prior antibiotic treatment specifically given for pneumonia. Twenty (83%) patients belonged to Group A, and 19 (70%) to Group B, p = NS. The mean period of antibiotic treatment was 6 ± 3 d for both groups (Table 1). In Group A the prior antibiotics administered were the following: third-generation cephalosporins, 11; vancomycin, 9; imipenem, 4; ciprofloxacin, 3; piperacillin, 2; amikacin 2; erythromycin, 1; aztreonam, 1; amoxicillin/clavulanate, 1; metronidazole, 1. In Group B, the prior antibiotics given were: second- and third-generation cephalosporins, 10; aminoglycosides, 5; vancomycin, 3; imipenem 3; ciprofloxacin, 2; piperacillin, 2; co-trimoxazole, 2; erythromycin, 1; metronidazole, 1.

In patients with positive bacterial cultures, initial antibiotic treatment was modified in 10 (42%) from Group A, and in four (16%) from Group B (p < 0.05) because of etiologic findings. The following isolated microorganisms were responsible for the antibiotic modification in Group A: L legionella spp. in one, Pseudomonas aeruginosa in seven, A cinetobacter calcoaceticus in one, and polymicrobial (Pseudomonas aeruginosa, Hemophilus influenzae, Streptococcus pneumoniae) in one. On the other hand, organisms implicated in the modification of initial antimicrobial therapy in Group B were: A cinetobacter calcoaceticus in one, Pseudomonas aeruginosa in one, Legionella spp. in one, and polymicrobial (Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus) in one. In 13 patients (six from Group A and seven from Group B) the prior antibiotic treatment was modified (Table 1). The following isolated microorganisms were responsible for the antibiotic modification in Group A: L legionella spp. in one, Pseudomonas aeruginosa in seven, A cinetobacter calcoaceticus in one, and polymicrobial (Pseudomonas aeruginosa, Hemophilus influenzae, Streptococcus pneumoniae) in one. On the other hand, organisms implicated in the modification of initial antimicrobial therapy in Group B were: A cinetobacter calcoaceticus in one, Pseudomonas aeruginosa in one, Legionella spp. in one, and polymicrobial (Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus) in one. In 13 patients (six from Group A and seven from Group B) the prior antibiotic treatment was modified (Table 1). The following isolated microorganisms were responsible for the antibiotic modification in Group A: L legionella spp. in one, Pseudomonas aeruginosa in seven, A cinetobacter calcoaceticus in one, and polymicrobial (Pseudomonas aeruginosa, Hemophilus influenzae, Streptococcus pneumoniae) in one. On the other hand, organisms implicated in the modification of initial antimicrobial therapy in Group B were: A cinetobacter calcoaceticus in one, Pseudomonas aeruginosa in one, Legionella spp. in one, and polymicrobial (Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus) in one.
Group B), antibiotics were not withheld despite the absence of significant bacterial growth.

A nitric modifications were also evaluated for cases of late-onset (≥7 d) pneumonia. Late-onset pneumonia was considered in 14 patients in Group A and 11 in Group B, with eight and three antibiotic modifications, respectively (p = 0.22).

**Morbidity and Mortality**

Patients from Group A had an ICU stay duration of 28 ± 17 d compared with that of Group B patients (26 ± 18 d), p = NS. With regard to the total mechanical ventilation period, patients in Group A were artificially ventilated for 23 ± 12 d versus 20 ± 17 d for patients in Group B, p = NS.

The crude observed mortality rate was 46% (11 of 24) in Group A and 26% (seven of 27) in Group B. The crude mortality considering both Groups A and B together was 35% (18 patients). There were no statistically significant differences when comparing crude mortality rates of Groups A and B (p = NS). In five patients from Group A (five of 11, 45%), the cause of death was directly attributable to pneumonia. This occurred in three patients (three of seven, 43%) from Group B; p = NS. The predicted mortality rates for Groups A and B on admission were 12 ± 7 and 15 ± 7%, respectively (p = NS).

There were no differences when comparing adjusted mortality rates for Group A (29%) and Group B (10%) (p = NS). There were no differences with regard to mortality among patients from Groups A and B in relation to the positivity or negativity of the diagnostic technique used: Group A, seven of 18 (39%) versus four of six (67%); Group B, five of 20 (25%) versus two of seven (28%), p = NS.

Mortality directly from VAP associated with the isolation of *Pseudomonas aeruginosa* and *Acinetobacter* calcoaceticus in both Groups A and B was 33% (five of 15), and 27% (three of 11), respectively, p = NS. In each group there were no differences when comparing the mortality of the latter etiologies with that caused by other isolates or without etiologic diagnoses (Group A: five of 15, 33% versus six of nine, 67%; Group B: three of 11, 27% versus four of 16, 25%; p = NS).

Mortality was analyzed according to different thresholds of APACHE II score on admission. The survivors and nonsurvivors according to these cutoff points are shown in Table 4. This table shows the greatest differences in mortality between Groups A and B for patients with an APACHE II score >18. Group B patients with APACHE II scores >18 had significantly higher mortality (p = 0.032) when compared with those with scores <18. There were no statistically significant differences when comparing mortality in patients from Groups A and B.

When analyzing mortality according to the adequacy of antibiotic treatment (only in patients with positive bacterial cultures), in 24 patients in whom antibiotic treatment was considered correct, six (25%) died (two from Group A and four from Group B, p = NS). On the other hand, in patients with inadequate antibiotic treatment (n = 14), six (43%) died (five from Group A and one from Group B, p = NS). There was no statistical difference when comparing mortality between patients in whom antibiotic treatment was considered adequate and those who did not receive correct antibiotics.

**DISCUSSION**

The main finding in this pilot study was the absence of differences in mortality and morbidity when comparing invasive versus noninvasive diagnostic management of mechanically ventilated patients with nosocomial pneumonia. In addition, quantitative endotracheal aspirate isolates agreed with those obtained by invasive methods (PSB and/or BAL) used for establishing the diagnosis.

The mortality of ventilator-associated pneumonia (VAP) ranges from 20 to 70% (19). However, the attributable mortality has been reported to be 30% (20). Prognostic factors for a poor outcome from nosocomial pneumonia include inappropriate antibiotic treatment (16). Furthermore, other investigators have found that prior antibiotic treatment is a risk factor for the development of infection with high-risk microorganisms such as *Pseudomonas aeruginosa* or *Acinetobacter* spp. (21). For all these reasons, it seems likely that all efforts directed towards the achievement of a microbial diagnosis of VAP are justified. This diagnosis can be achieved by using either invasive or noninvasive techniques. During the last 10 yr, a number of clinical and experimental studies have shown that invasive techniques performed through a fiberoptic bronchoscope using protected specimen brushing or BAL offer good diagnostic information for patients with VAP (2). However, these techniques are expensive, time-consuming, and not free from complications (1, 14, 15). Recent investigations have shown that noninvasive methods such as quantitative cultures of endotracheal aspirates have reasonable overall diagnostic accuracy (9–12).

The controversy over the use of invasive versus noninvasive techniques has been recently discussed in a debate (1, 2). In that debate it was emphasized that nobody has shown a decrease in mortality from VAP when using invasive techniques systematically. Our pilot study has shown no differences in mortality (for both crude and adjusted) or morbidity (duration of mechanical ventilation, length of ICU stay), suggesting that when management was directed by bronchoscopic data, outcome was not improved, and less invasive approaches can also provide accurate data for the appropriate management of patients with VAP. A recent study by Timsit and coworkers (22) supports our findings since they did not find differences in mortality comparing clinically suspected VAP with microbiologically confirmed VAP using invasive techniques. However, there are some imbalances and pitfalls in our study that merit consideration. The most important limitation is the small sample size studied, which can induce a type II statistical error. This warrants studies with larger sample sizes. However, we did not find differences in mortality, and in fact there was a trend for higher mortality in the group managed with invasive techniques. Second, initial antibiotic treatment was modified based upon invasive techniques in 10 patients versus four from those managed by noninvasive techniques. Third, the incidence of *Pseudomonas aeruginosa*, considered an organism with a high associated mortality, was higher in patients.

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<th>APACHE II</th>
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For definition of groups, see Table 1.
managed with invasive techniques. This has to be interpreted as a randomization aberration probably affected by the limited sample size. However, the number of cases of A cinetobacter calcoaceticus (another microorganism with high associated mortality) was greater in patients managed with noninvasive techniques, balancing in part the differences observed when considering only Pseudomonas aeruginosa. In fact when Pseudomonas aeruginosa and A cinetobacter calcoaceticus were considered together the mortality was similar when comparing Groups A and B and also when comparing the mortality associated with the isolation of these organisms with other etiologies in the same group. Fourth, in patients with negative cultures or cultures below the threshold, antibiotics were not withheld, and they were continued and/or started according to criteria of the attending physician. This design was used because it is well known that quantitative cultures have false negative results, particularly when prior antibiotic treatment is given (5). Fifth, most patients received prior antibiotic treatment before sampling. It would be interesting to perform the same type of studies in patients without any antibiotic treatment. Finally, an additional consideration is that we did not administer our antibiotic treatment on the basis of Gram stains and/or the detection of intracellular organisms in BAL. A different study is warranted to access the impact of rapid techniques in guiding the initial empirical antibiotic treatment of VAP. This was not the case in our study.

Atributable mortality of VAP (around 30%) is a feature that has been demonstrated in two studies (19, 20). The existence of this attributable mortality is one of the arguments used to establish the microbiologic diagnosis of pneumonia in order to improve appropriate antibiotic therapies. In addition, the use of initial inappropriate antibiotic treatment was a factor leading to a poor prognosis in a multivariate analysis (16), which probably explains the major part of attributable mortality. This has been confirmed in a recent cohort study in which initial antimicrobial therapy and other confounding factors (initial diagnosis, indication for mechanical ventilation, age, sex, and APACHE II on admission) were controlled and the mortality of VAP was not different from that of matched control subjects (40 versus 39%) (21). We found that in the subgroup of patients with APACHE II score < 18, the crude mortality was much less, although not significant, in patients managed with noninvasive techniques. This finding is difficult to explain. However, the following explanations could be given: (1) populations from Groups A and B differed in the etiology of VAP; (2) severity of illness could be different, but this was not the case in the present study because APACHE II on admission for both groups was comparable; and in addition, there were no differences between the predicted mortality rates calculated for both groups; (3) initial antibiotic therapies could have been more appropriate in Group B than in Group A. The latter is the most plausible explanation since there were more antibiotic changes because of etiologic findings in Group A than in Group B. This again suggests that the attributable mortality of VAP is probably more dependent on the adequacy of the initial antibiotic treatment than on the use of one or another type of diagnostic sampling. This could explain why patients in Group A had a higher mortality rate associated with the isolation of more resistant organisms. However, defining these organisms on bronchoscopy was not associated with an improved outcome. In the present study, however, we did not find statistical differences when comparing mortality of patients with adequate versus inadequate antibiotic treatment, but this could be explained by the sample size.

Our microbiologic findings led to antibiotic changes in 10 patients from Group A versus four from Group B. Pseudomonas aeruginosa was the microorganism that more frequently motivated these changes since antipseudomonal antibiotics were not included in the initial empiric treatments, or there was resistance to the antipseudomonal drugs used. In a recent study of PSB in suspected VAP (23), quantitative cultures led to antimicrobial changes in 38% of the patients, with inadequate initial therapeutic plans being the most frequent reason, particularly in those cases of late onset. A recently published Spanish multicenter study (24) of 430 cases of ICU-acquired pneumonia showed that study patients with inappropriate empirical (34%) treatment had a higher attributable mortality (25 versus 16%), a higher number of complications per patient (2.2 versus 1.7), a higher incidence of shock (28 versus 17%), and higher percentages of gastrointestinal bleeding (21 versus 11%). In another recent study (25), patients with clinical suspicion of VAP had a high mortality rate regardless of whether BAL cultures confirmed the clinical diagnosis of VAP. When adequate antibiotic therapy was initiated very early, mortality rate was reduced when compared with adequate therapy that was delayed until bronchoscopy was performed or BAL results were known. The lessons learned from our study and others are that the important issue in VAP is to start adequate antibiotic treatment as soon as possible. Recent American Thoracic Society guidelines (26) have developed empiric strategies based on the severity, risk factors, and the duration of hospitalization. We believe that the early compliance with these guidelines, and the strict surveillance and knowledge of the resistance patterns in each ICU, can improve our success in choosing the right empiric strategies and perhaps correct part of the attributable mortality and morbidity caused by VAP.

We found a total agreement on 71% of occasions between culture results of all the techniques that were performed in Group A patients (QEA, PSB, and BAL). Importantly, QEA cultures did not miss any microorganisms isolated when using invasie PSB and/or BAL. These results reinforce the current concept that this type of culture is of similar reliability to those obtained by invasive techniques, as has been suggested in at least three recently published reports (9–11).

In summary, we found no differences in mortality and morbidity when comparing invasive versus noninvasive diagnostic management of mechanically ventilated patients with nosocomial pneumonia. Invasive methods may give diagnostic information that leads to antibiotic changes, but these antibiotic changes are not associated with an improved outcome. Probably, initial inadequate antibiotic treatment and delay of starting adequate therapy are the key factors for explaining the attributable mortality of VAP. The sample size of the present investigation warrants larger studies.

References


