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# Chest physiotherapy for the prevention of ventilator-associated pneumonia

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**Abstract** *Objective:* Pneumonia is an important complication in patients who are intubated and mechanically ventilated, when it is commonly referred to as ventilator-associated pneumonia (VAP). Since VAP may be contributed to by impaired sputum clearance, we studied whether chest physiotherapy designed to enhance sputum clearance decreases the occurrence of VAP. *Design:* Prospective controlled systematic allocation trial. *Setting:* Tertiary teaching hospital ICU. *Patients and participants:* Sixty adult patients intubated and mechanically ventilated for at least 48 h. *Interventions:* Chest physiotherapy (intervention group) or sham physiotherapy (control group). *Measurements and results:* Control and intervention groups were well matched for age, sex, and admission $PaO_2/FiO_2$ ratio, APACHE II score, and Glasgow Coma Score. There were no differences in the duration of mechanical ventilation, length of stay in ICU or mortality. VAP was assessed daily by combined clinical assessment and the clinical pulmonary infection score (CPIS). VAP occurred in 39% (14/36) of the control group and 8% (2/24) of the intervention group (OR=0.14, 95% CI 0.03 to 0.56, *P*=0.02). After adjustment was made by logistic regression for other important variables (APACHE II score, duration of mechanical ventilation, presence of tracheostomy, and GCS score), chest physiotherapy was independently associated with a reduced occurrence of VAP (adjusted OR=0.16, 95% CI 0.03 to 0.94, *P*=0.02). *Conclusions:* In this small trial, chest physiotherapy in ventilated patients was independently associated with a reduction in VAP. This suggested benefit of physiotherapy in prevention of VAP requires confirmation with a larger randomised controlled trial.

**Keywords** Artificial respiration · Pneumonia · Physical therapy · Respiratory tract infections

## Introduction

Ventilator-associated pneumonia (VAP) is common [1, 2], usually bacterial in nature, and develops at least 48 h after the initiation of intubation and mechanical ventilation [3]. A recent review [4] reported that the prevalence of VAP ranged from 9% to 68%, with associated mortality rates ranging from 33% to 71%. In part this variation may be explained by the existence of widely differing diagnostic criteria for VAP [3, 5], confounded further by the non-specific nature of radiographic pulmonary infiltrates, which may be caused by pneumonia (≈30%), pulmonary oedema (≈30%), acute lung injury (≈15%), and atelectasis (≈15%), with up to another 10% remaining of unknown cause [2].

Clinical criteria can provide adequate sensitivity for VAP when compared to other methods, but invasive testing such as bronchoscopy may be required to improve test specificity [5, 6, 7]. A potential problem with the use of invasive diagnostic testing for VAP is that the associ-

ated increased accuracy and cost may not deliver results sufficiently early to influence survival [8].

Many risk factors have been associated with VAP, including the presence of chronic obstructive airway disease, airway intubation, reduced conscious state, intracranial pressure monitoring, airway re-intubation, mechanical ventilation greater than 7 days, use of positive end-expiratory pressure, and supine patient positioning [1, 9, 10]. In particular, airway intubation and mechanical ventilation reduce the normal clearance of airway secretions, increasing the risk of developing VAP [1]. There is some evidence that aggressive preventative measures may reduce the high rates of morbidity associated with VAP in the critically ill [11]. Chest physiotherapy including gravity-assisted drainage, chest wall percussion, chest wall vibrations, and manual lung hyperinflation (bagging) are commonly used intensive care procedures [12]. There is supportive evidence that various combinations of chest physiotherapy assist in the re-expansion of atelectatic lung [13, 14, 15, 16, 17] and confer short-term improvement in total lung-thorax compliance [13, 18] and expiratory flow rates [19]. However, there is no clear evidence that chest physiotherapy aimed at enhancing secretion clearance assists in the prevention or treatment of VAP [20].

The present study prospectively investigated the effect of chest physiotherapy (aimed at enhancing airway secretion clearance) in intubated and mechanically ventilated patients, compared to control patients not receiving this form of therapy, on the prevalence of VAP. Secondary outcomes of interest were the effect of physiotherapy on the duration of mechanical ventilation and admission to the intensive care unit (ICU), and on 28-day mortality.

## Materials and methods

During a 6-month period (September 1997–February 1998), consecutive general medical, surgical and/or trauma patients admitted to a University teaching hospital Intensive Care Unit were eligible for study enrolment if they required oral intubation and mechanical ventilation for at least 48 h. Exclusion criteria were open heart surgery within 24 h prior to ICU admission, aspiration or community-acquired pneumonia, provision of mechanical ventilation for <48 h, leukopenia (<$4\times10^9$/l), or if the patient had sustained any injury or developed any complication that did not allow chest physiotherapy to be provided. This study was conducted according to the principles established in Helsinki and was approved by The Royal Melbourne Hospital Clinical Research and Ethics Committee which waived the need for informed consent.

Intubated and mechanically ventilated patients were assessed by hospital physiotherapists for inclusion/exclusion criteria and systematically allocated to one of two groups dependent on the date of ICU admission. Blinding of Intensive Care Unit Medical staff and Nursing staff to study group allocation was achieved by the use of a sham physiotherapy intervention in control patients, and all staff including physiotherapists were blinded to the main outcome variable VAP, as defined in this study. Study patients were provided with the same treatment regimen for their entire ICU stay. Patients allocated to the intervention group received chest physiotherapy twice daily commencing on the day of admission to the ICU. This comprised gravity-assisted drainage [21] or positioning in side-lying with the head of the bed horizontal for at least 20 min (with the most affected lung on chest radiograph positioned uppermost), four sets of six cycles of expiratory chest wall vibrations [22], and airway suctioning (at least three times) via the endotracheal or tracheostomy tube interspersed through the treatment. Manual lung hyperinflation was not part of the routine physiotherapy treatment. Once the patient was extubated or removed from mechanical ventilation, chest physiotherapy comprised the same routine of patient positioning and expiratory chest wall vibrations, but was combined with coughing (if patient capable) or airway suctioning (tracheostomy). The control group did not receive chest physiotherapy treatment whilst in the ICU, however sham treatment was provided by physiotherapists. This consisted predominantly of cardiopulmonary assessment and occasional musculoskeletal physiotherapy. In addition, patient re-positioning side-to-side and airway suctioning could be provided by ICU nursing staff without limit as required by the patient's condition.

**Table 1** Clinical pulmonary infection score (CPIS)

| Parameter | CPIS Range | CPIS Score[a] |
|---|---|---|
| Temperature (°C) | 36.5–38.4 | 0 |
| | 38.5–39.0 | 1 |
| | <36.0 or >39.0 | 2 |
| White blood cells (×$10^9$/l) | 4.0–11 | 0 |
| | 11–17 | 1 |
| | >17 | 2 |
| Secretions | ± | 0 |
| | + | 1 |
| | ++ | 2 |
| $PaO_2/FiO_2$ (mmHg) | >240 or ARDS | 0 |
| | ≤240 no ARDS | 2 |
| Chest radiograph infiltrates | Clear | 0 |
| | Patchy | 1 |
| | Localised | 2 |

[a] CPIS (score 0–10) calculated daily for all patients and pneumonia confirmed retrospectively by either: (i) CPIS >8 and one of (B-D) or (ii) CPIS >6 and two of (B-D). [(A) the total CPIS score calculated as above, (B) clinical course on/off antibiotics consistent with pneumonia, (C) lack of evidence for alternative source of sepsis, (D) lung biopsy or post-mortem histology demonstrating pneumonia within relevant time-span]. Pneumonia cannot be diagnosed in the absence of chest radiograph infiltrates

The data to determine the clinical pulmonary infection score (CPIS) (Pugin et al. [23], as modified by A'Court et al. [24], see Table 1) was prospectively obtained daily by the chief investigator (GN) from five commonly used clinical parameters, namely body temperature, leukocyte count, volume and character of tracheal secretions, lowest $PaO_2/FiO_2$ ratio, and chest radiographic changes. All physiotherapy, medical, and nursing staff involved in the study did not have knowledge of the daily CPIS score, as it was not calculated until completion of trial patient recruitment. As part of daily routine ICU care, all patients were clinically evaluated daily for signs of pneumonia, including assessment of the chest radiograph and routine clinical parameters (body temperature, leucocyte count, tracheal secretions). Each patient was then rated by the responsible ICU physician as either having or not having a clinically suspected pneumonia. These subjective judgements by experienced intensive care clinicians were not subject to standardization. For the purposes of this study, VAP was defined as being present when, on the same calendar day, both the clinical diagnosis (yes) and total CPIS score both indicated the presence of pneu-

monia (see Table 1). VAP was defined as early onset when it occurred ≦5 days and late onset when >5 days after ICU admission.

Other patient outcome measures included the median duration of mechanical ventilation, the median length of stay in the ICU (ICU survivors only), and the mortality in ICU and at 28 days. Other data recorded included demographic information, details of admission diagnosis, and medical or surgical procedures, comorbid factors including as appropriate a pre-operative American Society of Anaesthesiologists Score (ASA) [25] as appropriate, drug therapy and smoking history. The APACHE II score [26] was calculated in the first 24 h after admission to the ICU. Sub-group analyses were undertaken as suggested by of the main outcome variable VAP and severity of disease indices.

Standard medical and nursing care was provided for both groups according to The Royal Melbourne Hospital ICU guidelines. This included haemodynamic support (intravenous infusions and vasopressor infusions to maintain mean arterial pressure according to clinician preference) and infection care with surveillance based on Gram stain and cultures of blood, urine, oropharyngeal, and tracheal secretions. Early enteral nutrition was encouraged. Other medical care provided as clinically appropriate during the ICU stay included antibiotic therapy and therapeutic fibreoptic bronchoscopy (sputum removal for atelectasis). No patient received selective decontamination of the digestive tract. A tracheostomy was performed in those patients who had been intubated for one week or more and who were medically judged to require mechanical ventilation for at least one more week, or for airway protection and sputum clearance as per ICU policy.

Weaning from mechanical ventilation was commenced when a patient's condition was medically stabilised and when a $PaO_2$ >60 mmHg on $FiO_2$ <0.5 (or equivalent $PaO_2/FiO_2$ ratio) could be maintained, with a respiratory rate <25 $min^{-1}$, temperature <38 °C, and heart rate <130 $min^{-1}$. Extubation was performed when the patient had been weaned successfully to a low level of mechanical ventilatory support (continuous positive airway pressure of 5 $cmH_2O$ and pressure support of 10 $cmH_2O$) combined with the presence of adequate cough and gag reflexes with minimal airway secretions.

### Statistical analysis

VAP and other categorical variables were compared between treatment groups using Fisher exact test. Ordinal data were compared with Mann Whitney U-tests, and continuous or near-continuous data with unpaired *t*-tests. Daily CPIS measurements were summarised as an arithmetic mean before statistical analysis, as recently recommended [27]. Initial univariate analyses were followed by step-wise logistic regression (validated by a backward step-wise logistic regression) including all variables with univariate $P$ <0.1 across the main outcome VAP (SYSTAT Version 10, SPSS, Chicago, Ill., USA). Assuming a VAP proportion of 40% in control patients, this study was planned to include 80 patients in each of two groups so as to have an 80% power ($\alpha$=0.05) to detect a 20% absolute reduction (from 40% to 20%) in the proportion of patients with VAP [28].

## Results

During the 6-month study period, a total of 253 intubated and mechanically ventilated patients were admitted to the ICU, of whom a total of 177 patients were subsequently excluded as ineligible. Major reasons for exclusion were intubation less than 48 h (51 intervention group, 50 control group), community-acquired pneumonia (21 intervention group, 25 control group), and unstable cardiovascular or neurological function or injury preventing positioning for chest physiotherapy (six intervention group, nine control group). Other reasons for exclusion were recent open heart surgery, admission with a tracheostomy, an immunocompromised status, pneumonectomy or lost data (six intervention group, nine control group). A further 16 patients were removed subsequently due to death less than 48 h after admission to the ICU (two intervention group, five control group) or a diagnosis of terminal illness on ICU admission (four intervention group, five control group). Overall, 60 patients fulfilled all the entry criteria, with 24 patients allocated to the intervention group and 36 patients allocated to the control group based systematically upon the date of ICU admission.

The two trial groups were well matched for age, sex, admission APACHE II, and Glasgow Coma Scores and $PaO_2/FiO_2$; however, in the surgical patient subgroup the ASA score was significantly higher in those who received control treatment (Table 2). There were no significant differences in several patient characteristics known to be risk factors for VAP, including COAD, smoking, cardiomyopathy, and use of steroids pre-morbidly. During the ICU stay there were no differences in the frequency of cardiac arrest or in the use of intravenous narcotics and sedatives, paralytic agents or intracranial pressure monitoring and/or drainage (Table 2). Almost all patients developed radiological signs of acute lung collapse and/or consolidation (approximately 95%,), however the median duration of atelectasis was 3 days, and there were no differences between the groups (Table 3). Likewise a trend towards a lower use of tracheostomy in the intervention group was present but this difference also was not statistically significant (Table 3). In contrast, the mean CPIS was significantly higher in the control group as was the rate of VAP (Table 3). The control group received mechanical ventilation for a median of 0.8 days longer than the intervention group, but this difference was not statistically significant and there was no difference in the median length of stay in ICU survivors (Table 3). Although there was no significant difference in 28-day mortality, an adverse trend in ICU mortality in the intervention group was noted (Table 3). Most of these deaths in ICU were as a result of withdrawal of active medical care (5/6, 84%) as compared to the control group (1/3, 33%). Overall, the development of VAP was associated with significant increases in median duration of mechanical ventilation (6.9 days versus 3.8 days), frequency of tracheostomy placement, and unexpectedly, somewhat lower admission APACHE II scores (Table 4).

Subgroup analyses performed by dichotomising the study patients based upon the overall median APACHE II score of 19.5, showed that in those patients with higher APACHE II scores (control $n$=17, intervention $n$=13) there was a lower frequency of VAP in the intervention

**Table 2** Patient demographics and management. (*SD* standard deviation, *APACHE II* acute physiology and chronic health evaluation score, *ASA* American Society of Anaesthetists preoperative score for surgical subgroup, *GCS* Glascow Coma Score, *COAD* chronic obstructive airways disease, *Sedatives/Narcotics/Paralysis (pharmacological)* continuous intravenous administration of relevant drugs at any time during ICU stay, *ICP/VD* number and percentage of patients with intracranial pressure monitoring and/or extraventricular drainage during ICU stay)

| Data | Control (*n*=36) | Intervention (*n*=24) | *P* |
|---|---|---|---|
| Age, mean (SD), years | 65.1 (14.6) | 65.0 (14.3) | 0.99 |
| Male/female | 24/12 | 10/14 | 0.07 |
| APACHE II score, mean (SD) | 18.8 (5.4) | 20.7 (6.9) | 0.26 |
| ASA, median (range) | 4 (3–5) (*n*=21) | 4 (3–4) (*n*=11) | 0.04[a] |
| Admission GCS, mean (SD) | 9.2 (5.2) | 8.6 (4.9) | 0.74 |
| Admission $PaO_2/FiO_2$, mean (SD) mmHg | 325 (122) | 359 (146) | 0.35 |
| COAD, *n* (%) | 11 (30.6) | 6 (25.6) | 0.77 |
| Smoking, *n* (%) | 11 (31.4) | 4 (19) | 0.37 |
| Cardiomyopathy, *n* (%) | 3 (8.3%) | 1 (4.2%) | 0.64 |
| Cardiac arrest, *n* (%) | 5 (13.9%) | 3 (12.5%) | 1.00 |
| Steroids, *n* (%) | 5 (13.9%) | 2 (8.3%) | 0.69 |
| Sedatives & narcotics, *n* (%) | 34 (94.4%) | 20 (83.3%) | 0.21 |
| Paralysis (pharmacological), *n* (%) | 4 (11.1%) | 3 (12.5%) | 1.00 |
| ICP/VD, *n* (%) | 11 (30.6%) | 5 (20.8%) | 0.55 |

[a] Mann Whitney U-test

**Table 3** Outcome data

| Data | Control (*n*=36) | Intervention (*n*=24) | *P* |
|---|---|---|---|
| Tracheostomy, *n* (%) | 17 (47) | 7 (29) | 0.19 |
| Bronchoscopy, *n* (%) | 3 (8) | 1 (4) | 0.64 |
| ICU Antibiotic use, *n* (%) | 35 (97) | 21 (87) | 0.29 |
| Lung collapse/consolidation, *n* (%) | 34 (94) | 23 (96) | 1.00 |
| Duration lung collapse, median (range), days | 3.0 (0–20) | 3.0 (0–19) | 0.46 |
| CPIS, mean (SD)[a] | 4.4 (1.5) | 3.6 (0.8) | 0.01 |
| VAP, *n* (%)[b] | 14 (39) | 2 (8) | 0.01 |
| VAP≦5 days (*n*) | 9 | 2 | |
| >5 days (*n*) | 5 | 0 | |
| Ventilation duration, median (range), days | 5.2 (2.2–20) | 4.4 (2–31) | 0.39 |
| ICU duration, median (range), days[c] | 5.8 (2.6–25) | 5.6 (2–35) | 0.89 |
| ICU mortality, *n*/*n* (%) | 3/36 (8) | 6/24 (25) | 0.14 |
| 28-day mortality, *n*/*n* (%) | 8/33 (24) | 5/18 (28) | 0.28 |

[a] Clinical pulmonary infection score. The mean serial CPIS score was the average daily CPIS for each patient across their entire ICU stay
[b] Odds ratio (OR)=0.14, 95% confidence interval (CI)=0.03–0.56
[c] Survivors of ICU only (control group *n*=33, intervention group *n*=18)

**Table 4** Patients with and without VAP. (*APACHE II* acute physiology and chronic health evaluation score, *GCS Glasgow* coma score, *Antibiotic use* antibiotic use at any time during ICU stay)

| Data | No VAP (*n*=44) | VAP (*n*=16) | *P* |
|---|---|---|---|
| Age mean (SD), years | 65.6 (15.2) | 63.6 (12.0) | 0.61 |
| APACHE II, mean (SD) | 20.5 (6.2) | 16.9 (4.8) | 0.02 |
| GCS, mean (SD) | 8.2 (4.9) | 10.9 (5.3) | 0.09 |
| Ventilation duration, median (range), days | 3.8 (2–31) | 6.9 (2–20) | 0.02 |
| Tracheostomy, *n* (%) | 14 (32) | 10 (62) | 0.04 |
| Antibiotic use, *n* (%) | 40 (91) | 16 (100) | 0.57 |

**Table 5** Predictors of VAP. Overall logistic regression model Hosmer-Lemeshow goodness-of-fit statistic=5.53, *df*=7, *P*=0.60. (*OR* odds ratio, *CI* confidence interval, adjusted by logistic regression for all other variables in this table)

| Parameter | *P* | | OR (95% CI) Adjusted |
|---|---|---|---|
| | Univariate | Adjusted | |
| Group (intervention vs control)[a] | 0.02 | 0.02 | 0.16 (0.03–0.94) |
| Tracheostomy (no vs yes) | 0.04 | 0.13 | 0.22 (0.03–1.58) |
| APACHE II[b] | 0.02 | 0.16 | 0.90 (0.77–1.05) |
| Ventilation duration[c] | 0.02 | 0.76 | 1.03 (0.86–1.23) |
| GCS[d] | 0.09 | 0.28 | 1.10 (0.92–1.32) |

[a] Univariate OR=0.14, 95% CI 0.030.56
[b] APACHE II with OR reflecting a 1 unit increment in the score
[c] Mean number of days receiving mechanical ventilation
[d] Glasgow coma score with OR reflecting a 1 unit increment in the score

group ($n$=0) compared with the control group ($n$=5) ($P$=0.05). In patients ventilated for longer than the overall median of 4.65 days (control $n$=20, intervention $n$=10), there was a statistically significant ($P$=0.02) reduction in the frequency of VAP in the intervention group ($n$=1) compared with the control group ($n$=11).

Variables with univariate $P$ values <0.1 (Table 4) were included in a stepwise logistic regression with backward elimination to determine those parameters independently associated with development of VAP (Table 5). This multivariate analysis demonstrated that the provision of chest physiotherapy remained independently associated with a reduced frequency of VAP following adjustment for several factors including the presence of a tracheostomy, the severity of illness (APACHE II score), the GCS, and the duration of mechanical ventilation.

## Discussion

The critically ill patients in this study were intubated and mechanically ventilated for at least 48 h and received chest physiotherapy which included gravity-assisted drainage, chest wall vibrations, and airway suctioning twice per day or sham physiotherapy treatment which consisted of cardiopulmonary assessment and musculoskeletal movement. In a multivariate analysis, the provision of chest physiotherapy aimed at enhanced airway secretion clearance was independently associated with reduced occurrence of VAP.

The pathogenesis of VAP has been assumed to include chiefly micro-aspiration of abnormally colonised oropharyngeal or gastric contents to the lower airways [9]. Recent well-controlled trials have suggested that the semirecumbent position [9] or continuous sub-glottic aspiration [29] may significantly reduce the risk of VAP. Other non-pharmacological attempts to reduce VAP have included the use of heat and moisture exchangers and reduced changes of ventilator circuits [3, 4, 11, 30].

A reduced tracheobronchial transport velocity (and therefore reduced clearance of airway secretions) resulting from endotracheal intubation and mechanical ventilation has been suggested to be an important precedent in the development of VAP [1]. In this context, enhanced expiratory flow rates with chest wall vibrations [19] and removal of airway secretions with various combinations of chest physiotherapy [19, 31] may serve as an alternative therapeutic option in the prevention of VAP. However, a recent review [32], based upon findings in surgical patients [33] has suggested that chest physiotherapy is of no benefit for the prevention of VAP. Our findings in contrast suggest that the use of combined chest physiotherapy may be useful in selected ICU patients for the prevention of VAP. It remains uncertain which physiotherapy manoeuvre or combination thereof was most responsible for the benefits in this investigation resulting from treatment comprising gravity-assisted drainage, chest wall vibrations, and airway suctioning. A reduction in VAP may have resulted from increased secretion clearance (as a result of increased expiratory flow rates [19] associated with chest wall vibrations and head down positioning) and improved re-expansion of collapsed or atelectatic lung. This point requires further investigation, as an earlier small study in mechanically ventilated trauma patients [20] was unable to demonstrate that chest physiotherapy with manual lung hyperinflation, gravity-assisted drainage, and airway suctioning significantly influenced the rate of VAP.

Unexpectedly in this investigation, increased occurrence of VAP showed a univariate association with lower admission APACHE II scores, although this relationship did not persist after adjustment for other clinically relevant parameters. The non-randomised allocation of therapy and relatively small size of the present study may in part explain this finding. In addition, a recent surveillance investigation [34] failed to identify admission APACHE II score as a predictor of VAP. Most of the cases of VAP in our study (69% of cases) were of early onset (≦5 days) making them likely to be a result of aspiration, impaired airway secretion clearance as a complication of airway intubation, or community-acquired. The investigation by Akça et al. [34] also identified a similar time course, with early onset VAP comprising 67% of cases in a group of medical, surgical, and trauma patients intubated and ventilated for at least 48 h.

The small size of the current study and the non-randomised allocation of therapy also may have contributed to the absence of a survival benefit with chest physiotherapy in this study. However, most of the ICU deaths, especially in the chest physiotherapy group, were due to poor patient prognosis and withdrawal of active medical care. Notably there were no significant differences in the 28-day mortality rate between treatment groups (Table 3).

A number of factors limit the general applicability of our study's findings regarding the efficacy of chest physiotherapy in mechanically ventilated patients. First, the non-randomised systematic allocation of our trial patients on the basis of date of admission to the ICU may have introduced bias. However, study inclusion and outcome criteria were detailed and prospectively specified, with medical and nursing clinicians remaining unaware of the main outcome measures used (CPIS and VAP as defined for this study). In addition, the final study groups were well matched for several major demographic and severity of illness variables, except the ASA score in the surgical sub-group. A second limitation of the current study was the use of clinical instead of invasive bronchoscopic criteria for the diagnosis of VAP, which may have increased the frequency of the diagnosis of VAP above the true rate; however, this effect would have influenced both treat-

ment groups similarly. The gold standard for the diagnosis of VAP remains elusive, as bacterial count thresholds used to define bronchoscopically positive samples have not been standardized [2] and the routine use of invasive diagnostic testing for all pulmonary infiltrates suspected to be pneumonia, although strongly advocated [35], has not been universally accepted. Thirdy, a recent investigation by Fabregas et al. [6], using as reference the presence of both histological pneumonia and positive lung cultures immediately after death, demonstrated that the CPIS as originally devised [23] was not any more accurate (sensitivity 77%, specificity 42%) at diagnosing VAP than conventional clinical criteria. Nevertheless, the CPIS scoring system may remain a useful screening tool to assess the likelihood of pneumonia and as such has been shown to be useful to direct antibiotic therapy [36]. The CPIS scoring system also may prove to be a useful means to direct chest physiotherapy treatment in future prospective investigations. Fourth, the large number of patients excluded from this investigation (mainly due to insufficient duration of ventilation and pre-existing pneumonia) reduced the sample size and limited the generalisability of our conclusion to those patients intubated and ventilated for at least 48 h. Finally, the intensive care physicians in this current investigation were not required to follow any uniform diagnostic pathway for their clinical suspicion of pneumonia, potentially leading to variation in it's diagnosis. Any adverse effect of this variation upon the diagnostic accuracy of VAP may have been reduced by defining VAP in this study as existing only when the semi-objective CPIS concurred with the Intensive Care Physician's clinical assessment.

In conclusion, this study of intubated and mechanically ventilated critically ill patients found that twice daily chest physiotherapy comprising gravity-assisted drainage, patient positioning in side-lying or head down, chest wall vibrations, and airway suctioning via the endotracheal tube was independently associated with a reduction in the occurrence of VAP. Further study in the form of a larger randomised controlled trial of this clinical intervention in patients at risk of VAP is warranted to confirm these findings and to establish which of the combination of physiotherapy techniques holds most therapeutic potential in the prevention of VAP.

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