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Physiotherapy does not prevent, or hasten recovery from, ventilator-associated pneumonia in patients with acquired brain injury

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Abstract *Objective:* To investigate the effect of respiratory physiotherapy on the prevention and treatment of ventilator-associated pneumonia (VAP) for adults in an intensive care unit (ICU) with an acquired brain injury (ABI). *Design and setting:* Two-part, prospective, randomised controlled trial. *Patients:* A total of 144 subjects with ABI admitted with a Glasgow Coma Scale ≤9, requiring intracranial pressure monitoring, and invasive mechanical ventilation (MV) for >24 h; 33 subjects were subsequently diagnosed with VAP. *Intervention:* Respiratory physiotherapy comprised six treatments (positioning, manual hyperinflation and suctioning) in each 24-h period whilst on MV. The Control Group received standard medical/nursing care but no respiratory physiotherapy. *Measurements and results:* There were no significant differences between groups for incidence of VAP, duration of MV, length of ICU stay or clinical variables such as requirement for re-ventilation. *Conclusions:* In adults with ABI, regular respiratory physiotherapy in addition to routine medical/nursing care does not appear to prevent VAP, reduce length of MV or ICU stay. Due to small numbers, it is not possible to draw any conclusions as to whether or not respiratory physiotherapy hastens recovery from VAP.

Keywords Ventilator-associated pneumonia · Acquired brain injury · Physiotherapy

Introduction

Ventilated-associated pneumonia (VAP) is associated with increased duration of mechanical ventilation (MV), intensive care unit (ICU) and hospital stay, and increased in-patient mortality rates [1]. Patients with acquired brain injury (ABI) are commonly admitted to the ICU, frequently aspirate at the time of injury or during intubation, and are therefore particularly at risk for VAP [2]. The incidence of VAP for neurological patients ranges from 23 to 82% [3–9] with a higher incidence of VAP reported only for patients admitted to a trauma ICU [10].

The main goals of respiratory physiotherapy include promotion of effective alveolar ventilation and adequate oxygenation, clearance of airway secretions, maintenance of chest-wall mobility, and enhancement of exercise tolerance and mobility [11]. Respiratory physiotherapy and

early mobilisation are suggested strategies to both prevent and treat VAP [1, 10, 12–17]. In a survey of ICU specialists 83% used respiratory physiotherapy to prevent VAP [15] and patients with VAP were shown to receive significantly more respiratory physiotherapy than those without VAP [18]. However, respiratory physiotherapy may cause profound transient changes in intracranial and haemodynamic measures in patients with ABI [19–24], although the clinical importance of these changes is unclear.

Three studies have investigated whether respiratory physiotherapy has a role in preventing VAP [25–27]. In 46 trauma patients receiving MV for >24 h, Ntoumenopoulos et al. [25] found no significant reduction in the incidence of VAP, duration of MV, length of ICU stay or mortality associated with the addition of twice-daily respiratory physiotherapy to routine nursing care. However, the authors acknowledged that an accurate diagnosis of VAP was not made, and advocated further studies using standard clinical criteria for diagnosing VAP and implementing a standardised respiratory physiotherapy regimen throughout the duration of ICU stay [25]. In a further study of 60 adults receiving MV for >48 h admitted to a combined medical, surgical and trauma ICU, these authors found the addition of twice-daily respiratory physiotherapy to routine nursing care was independently associated with a reduction in VAP, diagnosed by clinical criteria and a modified clinical pulmonary infection score (CPIS) [26]. However, there were no significant differences in duration of MV, length of ICU stay or mortality [26]. In a study of 180 patients receiving MV for greater than 48 h, Templeton and Palazzo [27] found no significant difference in VAP incidence, length of ICU stay or mortality between those receiving non-standardised physiotherapy and a Control Group. To date, no studies were identified that have evaluated the effectiveness of respiratory physiotherapy at hastening the resolution of VAP.

At present firm recommendations cannot be made regarding the use of respiratory physiotherapy for the prevention or treatment of VAP [28, 29]. The aim of this two-part study was to investigate if the provision of standardised regular respiratory physiotherapy interventions, in addition to routine medical/nursing care, (1) decreased the incidence of VAP (Part A) or (2) hastened the recovery from VAP (Part B) in subjects with ABI.

Materials and methods

This two-part randomised controlled trial was conducted at an inner city quaternary teaching hospital, Royal Perth Hospital (RPH). The study was approved by the Human Research Ethics Committees of RPH and Curtin University of Technology.

Subjects: Part A

Patients aged ≥16 years, having an admission Glasgow Coma Scale (GCS) of ≤9, an intracranial pressure (ICP) monitor or drain in-situ, and requiring invasive MV for >24 h were eligible for inclusion. For this study ABI could have resulted from trauma, hypoxia, infection, tumour, substance abuse, degenerative neurological disease or stroke [30]. Exclusion criteria comprised: patients not for active therapy, those requiring excessive respiratory support [nitric oxide ventilation, fraction of inspired oxygen (FiO_2) > 0.8, and/or positive end expiratory pressure (PEEP) > 10 cm H_2O], unstable haemodynamic status (mean arterial pressure >120 or <60 mmHg, and/or heart rate >120 or <60 bpm, labile mean arterial pressure or heart rate, new cardiac arrhythmias requiring definitive intervention, excessive inotropic requirements, i.e. noradrenaline/adrenaline infusion >30 mg per hour) or with an unstable neurological status [labile ICP or cerebral perfusion pressure (CPP), sustained ICP >25 mmHg, sustained CPP <70 mmHg]. Included subjects who subsequently developed any of the exclusion criteria for a sustained period of ≥12 h (e.g. neurological instability) were withdrawn. Transient changes in medical status or subject unavailability due to procedures may have resulted in a physiotherapy treatment session being missed, but did not trigger subject withdrawal until three consecutive treatments were missed.

Subjects: Part B

Subjects from Part A who fulfiled a diagnosis of VAP were eligible for inclusion in Part B. A diagnostic algorithm, based on Grossman and Fein [31], was used to diagnose VAP. Initially the suspicion of VAP was identified through the daily use of the CPIS [32], based on a threshold score of ≥7. The Principal Investigator, who was not blinded to group allocation, scored the CPIS. In suspected VAP cases, quantitative testing using non-bronchoscopic lavage was then performed by the physiotherapist using standard techniques [33, 34]. The lavage specimen was analysed by staff blinded to the subject's group allocation; any microorganisms were quantitated as either 'few', 'moderate' or 'many' growths, with microbiological confirmation from the non-bronchoscopic alveolar lavage (NBL) culture and microscopy considered the threshold for confirming the diagnosis and aetiology of the VAP [see also electronic supplementary material (ESM)]. Exclusion and withdrawal criteria were as stated for Part A.

Sample size

Part A: Due to lack of published data regarding the effectiveness of respiratory physiotherapy for preventing

VAP in the ABI population, it was assumed the incidence of VAP for the Control Group would be 30%, and an absolute difference between groups of ≥20% as clinically important. With 80% power and an alpha level (two-tailed) of 0.05, a sample size of 65 per group was determined (PowerAndPrecision™ Release 2.0, 2000). Allowing for a 10% withdrawal rate, sample size was increased to 72 subjects per group.

Part B: It was not possible to directly estimate effect size for this arm of the study due to the lack of published data. Further, subject numbers were constrained by VAP incidence and recruitment rates from Part A. Using a large effect size ($d = 1.00$) [35], 80% power and an alpha level (two-tailed) of 0.05, a sample size of 17 per group was determined [35].

Outcome measures

The primary outcome measure in Part A was the incidence of VAP (diagnostic method as noted earlier), defined as the number of subjects in whom a positive NBL diagnosis for infection confirming VAP was obtained. Secondary outcome measures were duration of MV; length of ICU/hospital stay; withdrawal rates; incidence of lobar collapse, bronchoscopy, re-ventilation/re-intubation; re-admission to ICU; mortality, daily CPIS scores and the best/worst daily arterial to inspired oxygen ratio (PaO_2/FiO_2). The primary outcome measures in Part B were duration of MV and length of ICU stay. Secondary measures were as noted for Part A. Outcome measures were measured by appropriately trained RPH staff who were blinded to group allocation.

Procedure

Written informed consent was obtained from next of kin. In Part A, subjects were randomly assigned to groups via a random numbers table [35] and then received interventions as per their allocated group. Subjects in Part B were re-randomised to groups using the same procedure.

All subjects

All patients were intubated (orally) prior to inclusion. The time lapse between intubation and commencement of the study protocol was <24 h in all cases. The standard medical/nursing care provided to all subjects included: invasive MV titrated to achieve targeted arterial blood gases, haemodynamic support including vasopressor and other intravenous infusions to maintain mean arterial pressure and desired CPP levels, sedation and active cooling for ICP and temperature control, an oro/nasogastric tube, early enteral nutrition; infection surveillance and antibiotic therapy as clinically indicated. Selective digestive decontamination and antibiotic treatment were not routinely initiated, and continuous subglottic suctioning was not performed.

Subjects remained in the 'intervention' phase of the study whilst receiving MV. All subjects, irrespective of group allocation, received routine nursing care involving pressure area care and position changes every 3–4 h. In the absence of contra-indications, this included maintenance of a 30° head-up position, in supine or side-lying. Nursing staff performed airway suctioning as required utilising the same technique as the physiotherapists, but pre-oxygenation was not standard practice and manual hyperinflation (MH) was not performed. All subjects received assessment of passive limb range of movement once daily by a physiotherapist. Neurological rehabilitation (e.g. postural/balance exercises) was commenced for all subjects once ICP monitoring ceased, weaning of MV/sedation had commenced, a tracheostomy was in-situ or the subject extubated and cardiovascular and respiratory function were stable.

Control Group

Subjects in the Control Group in both parts of the study did not receive respiratory interventions by a physiotherapist nor did physiotherapists give any input to assist nursing staff with direction, rationale or choice of positioning.

Treatment Group

Subjects in the Treatment Group in both parts of the study received six respiratory physiotherapy treatments in each 24-h period, comprised of positioning, MH and airway suctioning. If unilateral changes on chest X-ray and/or auscultation were apparent, the subject was positioned, where possible, with the affected side uppermost (head elevated 30°). If bilateral changes were evident on assessment, positioning in left or right side-lying was alternated between each intervention. If no abnormal chest X-ray/auscultation findings were detected, positioning was determined as per standard pressure area care routines. The subject's position was maintained for at least 30 min. In conjunction with positioning, MH was performed by the physiotherapist using a standard technique [36, 37]. Upon completion of MH subjects were reconnected to the ventilator at pre-treatment settings and airway suctioning was performed by the physiotherapist. Pre-oxygenation (using the inbuilt ventilator feature) prior to the suction procedure was used. To minimise stimulation and limit potential increases in ICP, no more than two suction passes were performed after MH. During a single respiratory physiotherapy treatment two to four cycles of MH and suctioning

were performed. On average each respiratory physiotherapy treatment lasted 30 min. Respiratory physiotherapy continued until the subject was weaned off MV.

Statistical analyses

Analyses were performed using the SPSS® Graduate Pack 11.5 for Windows™ statistical package. Data were analysed using both an intention to treat philosophy and analysis by treatment principle. Success of the randomisation process in achieving comparable groups was assessed using Chi-square tests (nominal data) and *t* tests for independent samples (continuously distributed variables). Chi-square tests and independent *t* tests were used to test for differences in outcome measures between groups. Kaplan Meier survival analysis was undertaken. A P value of less than 0.05 was considered significant.

Results

Part A

Of the 5,297 patients admitted to the RPH ICU between 1st November 2000–30th June 2004, 193 patients fulfiled the inclusion criteria. Consent was obtained for 144 subjects (74.6%), with 72 randomised to the Treatment and Control Groups, respectively (Fig. 1). Groups were comparable for all demographic data except gender and body mass index (BMI) (Table 1). Thirty-three of the 144 subjects (22.9%) were diagnosed with VAP: 14 Treatment Group (19.4%) and 19 Control Group subjects (26.4%) ($P = 0.32$). Kaplan Meier survival analysis suggests that death did not influence the incidence of VAP in this study ($P = 0.241$). No significant differences between groups were identified for this or any of the remaining outcome measures (Tables 2, 3). Additional clinical data are provided in Table 4 and Figs 3, 4, 5 (see ESM). Table 7 (ESM) summarises the primary bacteria isolated in the diagnosis of VAP.

Sixteen subjects (11.1% of total sample) did not complete the study as per their allocated group and were withdrawn, five (6.9%) from the Treatment Group and 11 (15.3%) from the Control Group ($P = 0.11$). Reasons for subjects not completing study interventions included: cessation of active management (8), development of withdrawal criteria (5), self-extubation and subsequent non-invasive ventilation (1) and physiotherapy staffing issues (2). Data collection for the 16 subjects who did not complete their allocated intervention continued and their data were analysed using an intention to treat philosophy. Subjects who did not complete their allocated intervention were older (54.1 ± 21.9 vs. 42.2 ± 18.9 years, $P = 0.02$), admitted with a lower GCS (4.2 ± 1.9 vs. 5.3 ± 2.0, $P = 0.04$), and had a higher acute physiological and chronic health evaluation (APACHE) II score (24.1 ± 6.6 vs. 19.9 ± 5.3, $P < 0.01$) than the remaining subjects. Eleven of these 16 subjects (68.8%) subsequently died (six in ICU, five on the neurosurgical ward), compared with 23 of the 128 subjects (18.0%) who completed their allocated intervention ($P < 0.01$).

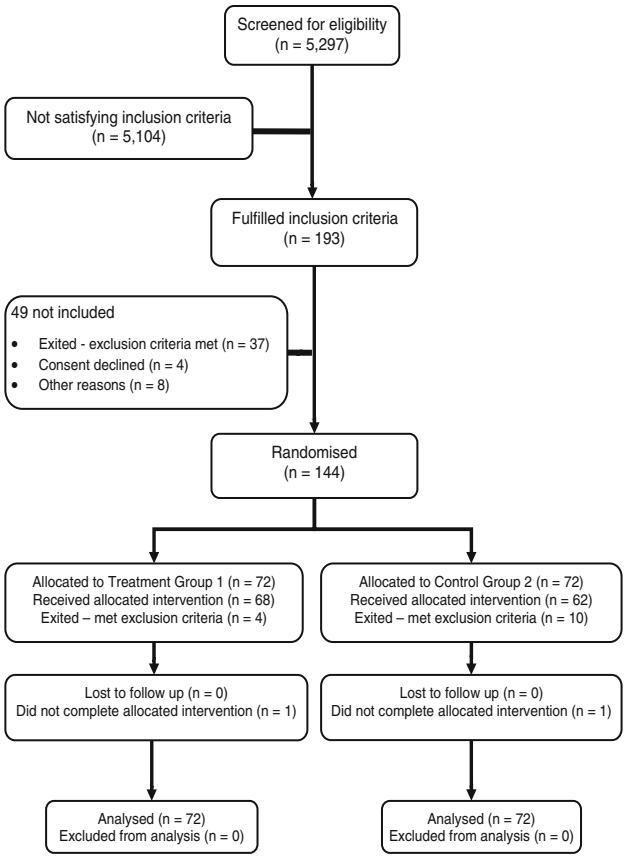

Fig. 1 Flow diagram of the process of randomisation for Part A of the study

Part B

Results for Part B are detailed within the ESM. Respiratory physiotherapy did not appear to significantly hasten the recovery of VAP in terms of duration of MV, length of ICU/hospital stay or clinical variables such as the daily CPIS score.

Discussion

This study found that a regular respiratory physiotherapy regimen, comprised positioning, MH and suctioning repeated six times per day, when provided in addition to

Table 1 Demographic characteristics of the 144 subjects in Part A and 33 subjects in Part B

Variable	Part A (prevention of VAP)			Part B (treatment of VAP)		
	Treatment Group ($n = 72$)	Control Group ($n = 72$)	*P* value	Treatment Group ($n = 17$)	Control Group ($n = 16$)	*P* value
Age (years)[a]	45.8 ± 19.0 17–85	41.1 ± 20.0 16–81	0.15	34.1 ± 16.3 16–72	37.9 ± 17.8 16–66	0.52
Gender (male/female)	51/21	36/36	0.01	12/5	9/7	0.39
Race[b]						
Caucasian	62 (86.1)	64 (88.9)	0.36	17 (100.0)	14 (87.5)	0.13
Aboriginal	8 (11.1)	4 (5.6)		0 (0.0)	0 (0.0)	
Other	2 (2.8)	4 (5.6)		0 (0.0)	2 (12.5)	
BMI (kg/m^2)[a]	27.6 ± 5.3 18.5–42.1	25.5 ± 6.5 17.1–54.3	0.04	26.4 ± 6.2 19.2–42.1	27.0 ± 7.4 19.5–47.6	0.81
GCS[a]	5.4 ± 2.0 3–9	4.9 ± 2.0 3–9	0.21	4.5 ± 2.0 3–8	4.9 ± 1.9 3–8	0.55
APACHE II score[a]	20.3 ± 5.7 9–39	20.5 ± 5.6 8–38	0.82	21.0 ± 6.2 11–34	18.2 ± 6.4 5–27	0.22
Cause of ABI[b]						
MVA/MBA	29 (40.3)	29 (40.3)	0.53	10 (58.8)	7 (43.8)	0.70
SAH/ICH	26 (36.1)	30 (41.7)		3 (17.6)	5 (31.3)	
Alleged assault	7 (9.7)	2 (2.8)		0 (0.0)	1 (6.3)	
Fall	6 (8.3)	6 (8.3)		3 (17.6)	2 (12.5)	
Other	4 (5.6)	5 (6.9)		1 (5.9)	1 (6.3)	
Chest injuries	20	21	0.85	7	7	0.88
Respiratory history[b]						
Nil	58 (80.6)	58 (80.6)	0.46	11 (64.7)	10 (62.5)	0.64
COPD	4 (5.6)	3 (4.2)		1 (5.9)	3 (18.8)	
Asthma	5 (6.9)	9 (12.5)		4 (23.5)	2 (12.5)	
Other	5 (6.9)	2 (2.8)		1 (5.9)	1 (6.3)	
Smoking history[b]						
Non	42 (58.3)	39 (54.9)	0.38	9 (52.9)	9 (60.0)	0.30
Current	23 (31.9)	26 (36.6)		7 (41.2)	4 (26.7)	
Ex < 6/52	0 (0.0)	2 (2.8)		0 (0.0)	2 (13.3)	
Ex > 6/52	7 (9.7)	4 (5.6)		1 (5.9)	0 (0.0)	
Chronic sputum production	5	5	1.0	1	2	0.51
VAP diagnosis day (from ICU admission)[a]	NA	NA	NA	5.8 ± 4.0 1–12	5.3 ± 3.1 2–14	0.71

n number, *P* probability, *SD* standard deviation, *BMI* body mass index, *kg* kilograms, *m* metres, *GCS* Glasgow coma scale, *APACHE* acute physiological and chronic health evaluation, *ABI* acquired brain injury, *MVA* motor vehicle accident, *MBA* motor bike accident, *SAH* subarachnoid haemorrhage, *ICH* intracranial haemorrhage, *COPD* chronic obstructive pulmonary disease, *Ex < 6/52* ceased smoking within the last 6 weeks, *Ex > 6/52* ceased smoking over 6 weeks ago, *NA* not applicable

[a] For continuous data values reported are mean ± SD and range

[b] Data are numbers of subjects with percentage of group in parentheses

Table 2 Duration of mechanical ventilation and length of stay for the 144 subjects in Part A and 33 subjects in Part B

Variable	Part A (prevention of VAP)			Part B (treatment of VAP)		
	Treatment Group ($n = 72$)	Control Group ($n = 72$)	*P* value	Treatment Group ($n = 17$)	Control Group ($n = 16$)	*P* value
Duration of MV (hours)	172.8 ± 119.8 138.7 16.8–624.2	206.3 ± 157.1 157.1 26.5–737.3	0.18	342.0 ± 185.3 301.0 101.0–737.3	351.0 ± 174.7 299.7 90.2–715.8	0.89
Length of ICU stay (hours)	224.2 ± 122.4 208.0 21.5–668.0	256.4 ± 184.5 206.1 44.1–900.8	0.22	384.7 ± 179.6 342.0 101.0–747.7	397.9 ± 190.7 323.5 90.2–900.8	0.84
Length of hospital stay (days)	36.4 ± 49.9 24.6 2.8–357.1	25.4 ± 20.0 21.4 1.8–82.0	0.09	46.5 ± 41.8 33.8 4.2–178.1	32.4 ± 19.9 27.6 3.8–82.0	0.23

Data are mean ± SD, followed by median, and range

n number, *P* probability, *MV* mechanical ventilation, *ICU* intensive care unit, *SD* standard deviation

Table 3 Clinical information for the 144 subjects in Part A and 33 subjects in Part B

Variable	Part A (prevention of VAP)			Part B (treatment of VAP)		
	Treatment Group ($n = 72$)	Control Group ($n = 72$)	*P* value	Treatment Group ($n = 17$)	Control Group ($n = 16$)	*P* value
Lobar collapse	24 (33.3)	23 (31.9)	0.86	2 (11.8)	7 (43.8)	0.04
Bronchoscopy	2 (2.8)	6 (8.3)	0.15	1 (5.9)	2 (12.5)	0.51
Re-ventilation	8 (11.1)	11 (15.3)	0.46	2 (11.8)	1 (6.3)	0.58
Re-admission to ICU	2 (2.8)	2 (2.8)	1.00	0 (0.0)	0 (0.0)	1.00
Mortality						
Total[a]	13 (18.1)	21 (29.2)	0.12	2 (11.8)	2 (12.5)	0.95
In ICU[b]	7 (53.8)	14 (66.7)	0.46	2 (100.0)	2 (100.0)	0.95

Data are numbers of subjects with percentages in parentheses
n number, *P* probability, *ICU* intensive care unit
[a] Mortality within 90 days of hospital admission
[b] Data are numbers of subjects with percentage of total mortality in parentheses

routine medical/nursing care, did not significantly reduce the incidence of VAP, length of MV or ICU/hospital stay for adults with ABI (Part A). Due to the small numbers diagnosed with VAP it is not possible to draw any conclusions as to whether respiratory physiotherapy hastens the recovery from VAP in terms of duration of MV, length of ICU/hospital stay or clinical variables such as the daily CPIS score (Part B).

The findings of Part A of this study are consistent with those of Ntoumenopoulos et al. [25] and Templeton and Palazzo [27], who also failed to demonstrate a significant decrease in the incidence of VAP with regular respiratory physiotherapy. However, in the Ntoumenopoulos et al. [25] study, patients had lower APACHE II scores than in the present study, placing their subjects at a lower risk of developing VAP [38], and those with ABI were excluded thereby limiting comparison to the present study. Further limitations of their study [25] included the small sample size ($n = 46$), low statistical power of 0.21 [28], non-specific clinical criteria for VAP diagnosis, and early withdrawal of subjects with suspected VAP who went on to receive more intensive respiratory physiotherapy. Whilst the study by Templeton and Palazzo included some patients with ABI, physiotherapy interventions were not standardised, hence limiting comparison with our study [27]. In contrast to our study, it has been reported that twice-daily respiratory physiotherapy significantly reduced the incidence of VAP [26]. In that study of 60 adults, 27% of whom had ABI, an incidence of VAP of 39 and 8% in the control and respiratory physiotherapy groups, respectively, was found [26]. The heterogeneity of their study populations (general medical, surgical and/or trauma patients) [26, 27] may account for the different response to respiratory physiotherapy. Additionally, differences between the samples of Ntoumenopoulos et al. [26] and the present study may have affected outcomes (e.g. age, APACHE scores, number of subjects with chronic obstructive pulmonary disease—all recognised risk factors for VAP) [39–42] and number of subjects with lung collapse (more likely to be responsive to respiratory physiotherapy) [43, 44]. The inability of prophylactic respiratory physiotherapy to significantly decrease duration of MV or length of ICU stay in this study is consistent with previous findings [25, 26].

As no previous studies were identified that investigated the effect of respiratory physiotherapy on the rate of recovery of VAP, there are no data with which the findings of Part B of the current study can be compared. With the exception of a greater incidence of lobar collapse in the Control Group, no significant differences were found between the Part B groups for duration of MV, length of ICU/hospital stay or clinical variables such as the daily CPIS and PaO_2/FiO_2 ratio, requirement for bronchoscopy and mortality.

Whilst the duration of MV and length of stay in ICU were reduced in favour of the Treatment Group in both Parts A and B of the study, these differences were not statistically significant, mainly due to the large variance in the data. Thus, although respiratory physiotherapy has been previously shown to result in short-term improvements in airway clearance, lung compliance and oxygenation in intubated patients [37, 44–49], any beneficial physiological effects associated with physiotherapy in our study did not translate to changes in clinical endpoints such as the incidence of VAP, duration of MV, or length of ICU stay. The ABI population is well recognised as being at a higher risk for VAP, particularly patients with a severe ABI [3–6, 18, 38, 42, 50–52]. It may be that respiratory physiotherapy aimed at preventing VAP may be ineffective because of the severity of the underlying neurological disorder and thus the overwhelming presence of risk factors for VAP. It may also be that the general ICU management of ABI, with its focus on ICP/CPP control and strategies such as stimuli minimisation, makes it problematic to provide effective respiratory physiotherapy. It is also acknowledged that duration of MV and ICU stay may have been affected by multiple confounding variables. For example, the timing and

pacing of weaning from MV can be influenced by the patient's neurological status and clinical stability, timing of weaning of sedation and tracheostomy insertion, overall ICU workload and bed availability status and the personal approach of the ICU specialist, which in turn can affect duration of MV and ICU stay. Therefore it could be questioned as to whether these outcomes were sufficiently sensitive to detect a beneficial effect associated with respiratory physiotherapy. As the cost of an ICU bed-day is high, future research, including estimates of the sample size, should focus on the ability of respiratory physiotherapy to affect length of ICU stay. Finally, it is becoming apparent that no single intervention or strategy (such as respiratory physiotherapy) is universally effective in preventing VAP, but instead a number of interventions/strategies working synergistically may offer the greatest chance for modifying the incidence of VAP [42, 53].

There are several limitations to our study. Sample size determination was based on the assumption of a VAP rate of 30%. The actual incidence of VAP was lower than anticipated hence the potential for respiratory physiotherapy to affect the incidence of VAP may have been limited. Although significant differences between groups in gender distribution and BMI were noted in Part A, these factors have not been reported as risk factors for VAP [39–42]. It is acknowledged that a further limitation of Part B of the study is that the response of Part B patients may have been influenced by their group allocation in Part A.

In conclusion, in this study, use of a regular respiratory physiotherapy regimen comprising positioning, MH and suctioning, in addition to routine medical/nursing care, did not significantly decrease the incidence of VAP, duration of MV or length of ICU stay in adults with ABI. Due to limited subject numbers it is not possible to conclude whether or not respiratory physiotherapy hastens the recovery of those patients who developed VAP by reducing duration of MV or ICU/hospital stay.

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