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Chest physiotherapy prolongs duration of ventilation in the critically ill ventilated for more than 48 hours

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Abstract *Objective:* This study aimed to determine the impact of providing chest physiotherapy after routine clinical assessment on the duration of mechanical ventilation, outcome and intensive care length of stay. *Design and setting:* Single-centre, single-blind, prospective, randomised, controlled trial in a university hospital general intensive care unit. *Patients and participants:* 180 patients requiring mechanical ventilation for more than 48 h. *Interventions:* Patients randomly allocated, one group receiving physiotherapy as deemed appropriate by physiotherapists after routine daily assessments and another group acting as controls were limited to receiving decubitus care and tracheal suctioning. *Measurements and results:* Primary endpoints were initial time to become ventilator-free, secondary endpoints included intensive care unit (ICU) and hospital mortality and ICU length of stay. Kaplan-Meier analysis censored for death revealed a significant prolongation of median time to become ventilator-free among patients receiving physiotherapy ($p = 0.047$). The time taken for 50% of patients (median time) to become ventilator-free was 15 and 11 days, respectively, for physiotherapy and control groups. There were no differences between groups in ICU or hospital mortality rates, or length of ICU stay. The number of patients needing re-ventilation for respiratory reasons was similar in both groups.

Keywords Mechanical ventilation · Physical therapy · Chest physiotherapy · Ventilator weaning · Outcome · Intensive care length of stay

Introduction

Chest physiotherapy (CPT) is an accepted method for increasing pulmonary volumes, clearing secretions and re-inflating atelectatic lungs; however, data from a prospective randomised controlled trial (PRCT) of patients with primary pneumonias suggests that CPT does not help in the acute phase of illness, and indeed these data suggest some disadvantage [1]. There has been some concern that CPT is sometimes inappropriate [2] and not without hazard [3, 4]. Notwithstanding these observations, evidence suggests that CPT is followed by improvements in oxygenation [5–7], compliance, air entry and carbon dioxide clearance [6, 8–15]. Furthermore, it has been suggested that CPT reduces the incidence of ventilator-associated pneumonia (VAP) [16].

The CPT aims in ventilated patients are enhancement of pulmonary volumes to facilitate alveolar recruitment and clearance of bronchial secretions to reduce the risk of sudden or progressive occlusion. These benefits would be expected to accelerate weaning; however, there is no controlled data regarding the impact of CPT. We therefore undertook a single-blind PRCT in patients ventilated for more than 48 h to determine whether CPT shortened weaning and reduced ICU stay or mortality.

Materials and methods

The study was conducted in a university hospital general ICU. The hospital ethics committee wavered consent since patients would be sedated at time of enrolment. Assent was obtained from next of kin.

Elective and emergency patients with varied diagnoses, intubated and ventilated (Servo 300, Siemens Medical Solutions, Sweden) for 48 h and likely to remain ventilated were prospectively randomised to a physiotherapy group (P) or control group (C). Patients previously ventilated during their current ICU stay were excluded. Patient demographics and diagnostic groupings [17] are shown in Tables 1 and 2.

Patients with ventilatory failure due to neuromuscular dysfunction likely to impair ability to wean were excluded.

In our institution CPT is provided by physiotherapists at set times twice daily following routine visits and clinical assessment. The treatment is either a preventive measure or directed towards observed pathology. In general, treatment is thoracic and pulmonary expansion, respiratory muscle exercise and bronchial secretion removal as considered appropriate. In the U.K. it is normal practice for a physiotherapist to assess chest pathology and decide on the need, if any, for CPT. For the trial period physiotherapists continued their normal ICU practice and assessed all patients, whether in the study or not, twice daily.

Assessment was based on auscultation, chest X-ray, CT reports, laboratory results and information from medical and nursing staff. Patients with conditions which might deteriorate following physiotherapy, such as bronchospasm, intracranial pressure greater than 20 mmHg or haemodynamic instability, received physiotherapy once stable. Patients with pleural effusions who do not normally benefit from physiotherapy were not treated. In this study physiotherapists provided thoracic and pulmonary expansion by positioning, manual pulmonary hyperinflation with a Waters bag circuit, rib springing and general mobilisation, including sitting out of bed, when possible. Secretion removal techniques included manual pulmonary hyperinflation with vibration, positioning and drainage with tracheal suctioning.

Psychiotherapists were all aware of patient randomisation. Patients were routinely assessed by one of a team of experienced physiotherapists twice a day and their treatments and decisions were documented, facilitating continuity of care. Physiotherapists were at liberty to provide group P with the intensity and frequency of therapy they felt appropriate based on their assessment of the likely treatment benefit. Treatments ranged from none to any of those outlined above directed towards secretion removal and increasing pulmonary volumes. Group-C patients were also routinely assessed twice daily by physiotherapists but were only allowed to receive suctioning, decubitus care and general mobilisation. Nursing and medical staff were blind to whether a patient had been recruited to the study at all. This was achieved because all ICU patients received routine physiotherapists' visits regardless of whether they had been recruited for the study, and the treatment for group P varied from none to full physiotherapy, so it was impossible to identify patient allocation. For ethical reasons all patients were allowed rescue physiotherapy (need for manual hyperinflation and suctioning) for sudden sustained de-saturation due to mucus plugging, outside the times of routine visits. Rescue therapy was provided by bedside nurses or physiotherapist (if immediately available). Rescue events were recorded for the duration of patient ventilation. Transient de-saturations following postural changes not requiring intervention were excluded. No recruitment manoeuvres were undertaken in addition to routine or rescue physiotherapy.

All patients were assessed on ICU rounds at least twice daily by a consultant. Patients received nutrition, investigations and medical treatment according to requirements.

Table 1 Patient demographics

Characteristics	Physiotherapy ($n = 87$)	Controls ($n = 85$)	Probability ($p =$)
Mean age (years; SD)	57.7 (16.5; range: 18–92)	58.2 (18.0; range: 18–85)	0.86
Gender (F/M)	34/53	27/58	0.31
Smokers (%)	55.2	48.2	0.62
Patients receiving tracheostomy (%)	36.7	25.8	0.12
Emergency admissions (%)	89.7	87.1	0.60
Medical/surgical (%)[a]	58.6/41.4	57.6/42.4	0.89
Emergency medical (%)[a]	41.4	42.4	
Elective surgical (%)[a]	10.3	12.9	
Emergency surgical (%)[a]	48.3	44.7	
APACHE II score: risk of death on admission to unit (median; range)	49 (8–96)%	41 (4–97)%	0.10
SOFA score on admission to study (median; range)	10 (3–18)	9 (2–17)	0.22

[a] Medical/surgical definitions based on definitions in APACHE II

Table 2 Reasons for admission based on APACHE II diagnostic categories. *GI*, gastrointestinal; *COPD*, chronic obstructive pulmonary disease

Reason for admission [41]	Physiotherapy ($n = 87$)	Controls ($n = 85$)
Respiratory insufficiency due to COPD	2	1
Respiratory insufficiency due to non-cardiac oedema	0	1
Respiratory insufficiency due to arrest	4	1
Respiratory insufficiency due to infection	6	4
Respiratory insufficiency due to asthma	1	1
Cardiac insufficiency due to cardiac arrest	9	8
Head trauma (non-operative)	1	3
Head trauma (post-operative)	1	0
Intra-cerebral haemorrhage (ICH; non-operative)	9	5
ICH (post-operative)	13	8
Multiple trauma (non-operative)	0	1
Multiple trauma (post-operative)	2	4
Sepsis (post-operative)	10	9
Sepsis (non-operative)	6	7
Peripheral vascular surgery	2	4
Haemorrhagic shock (post-operative aortic aneurysm)	11	14
GI bleed (post-operative)	1	1
GI bleeding (non-operative)	0	1
GI perforation (post-operative)	1	3
GI surgery for ischaemic bowel	0	1
GI obstruction (post-operative)	2	0
Gastrointestinal (acute pancreatitis)	0	1
GI surgery for neoplasm	4	4
Neurological (viral encephalitis)	1	1
Seizures (non-operative)	1	2

Weaning criteria, determined a priori, were identical to the unit protocol. The weaning protocol started when blood magnesium, potassium and phosphate were in the normal range, PaO_2/FiO_2 ratio > 30 kPa, and $PaCO_2$ was normal for the patient, usually 4.5–6 kPa. Ventilatory support was reduced using pressure support mode, if respiratory rate remained below 25/min. Extubation was undertaken in awake patients who had reached pressure support 10 cm water, PEEP < 10 cm water, respiratory rate < 25/m and secretions could be expectorated by coughing. Unconscious patients with a tracheostomy were judged ventilator-free once breathing on continuous positive airway pressure (CPAP) from free-standing circuits. Ventilator-free status was reached at extubation or CPAP if unconscious. The primary endpoint was time to become ventilator-free, calculated as the time between initiation of ventilation and when ventilator-free. Patients after achieving initial ventilator-free status who developed insufficient CO_2 clearance to avoid drowsiness or sustained hypoxaemia (saturations below 90%) with tachypnoea (respiratory rate > 30/min) were re-ventilated. Patients who at any stage after ventilator-free status needed further ventilatory support were included in the initial ventilator-free time analysis and in a subgroup analysis of re-ventilation.

Secondary endpoints included mortality and length of ICU stay. The incidence of VAP was also recorded. The VAP was diagnosed if at any stage after 48 h a patient simultaneously had all of the following: new chest X-ray infiltrates, positive microbiology culture from tracheal aspirates, a rise in white cell count and temperature (> 38°C). These signs and the micro-organisms isolated were documented on a daily basis.

The end of the study for any individual patient was discharge from ICU or death.

Statistics

Estimation of patient numbers required for this study was based on an interim analysis. The ventilator-free endpoint was anticipated to be complicated by prior death. To minimise risk of type-II errors an arbitrary interim univariate analysis was based on the survivors after recruitment of the first 100 patients.

Primary endpoint analysis was planned to be by Kaplan–Meier survival curves censored for deaths prior to reaching ventilator-free status. Cumulative hazard plots for the groups were planned to determine the most appropriate non-parametric analysis of the Kaplan–Meier curves. If hazard plots were parallel, indicating proportional hazard functions, curves would be analysed by log rank (Mantel–Haenszel) test. If the hazard plots were not parallel, i.e. non-proportional hazards, Kaplan–Meier curves were analysed by Breslow–Gehan–Wilcoxon test. (This test becomes less sensitive as patient numbers progressively reduce with death.)

Demographic data were analysed with Mann–Whitney U and unpaired t-tests as appropriate, where $p<0.05$ was considered to reject the null hypothesis. Secondary

Table 3 Outcome measures

	Physiotherapy ($n = 87$)	Controls ($n = 85$)	Probability ($p =$)
Median time (50% patients within each group) to become ventilator-free based on Kaplan–Meier curve (days; range)	15 (3–82)	11 (3–76)	0.045
Distribution of patients in the first 86 of 172 patients (50%) to become ventilator-free (n; unit survivors)	35 (17)	51 (28)	
Median time days (range in parentheses) for patients among the first 50% of the cohort to become ventilator-free. This includes patients who died on a ventilator	6 (3–9)	5 (3–9)	
Median length of ICU stay among the unit survivors in first 50% of the cohort to become ventilator-free (days; range)	7 (5–18)	9 (5–29)	
Median length of ICU stay among unit non-survivors among the first 50% of cohort to become ventilator-free (days; range)	6 (3–38)	7 (4–61)	
Median length of ICU stay for all patients (days; range)	13 (3–82)	12 (4–76)	0.78
Median length of ICU stay of all ICU survivors (days; range)	14 (5–51)	12 (5–47)	0.35
Median length of ICU stay for all ICU non-survivors (days; range)	11 (3–82)	13 (4–76)	0.6
Patients requiring re-ventilation for respiratory insufficiency at any time after initially becoming ventilator-free (%; number in parentheses)	12.6 (11)	14.1 (12)	0.99
Median time to become re-ventilated (days; range)	1 (1–9)	3.5 (1–6)	0.13
ICU mortality (%; number in parentheses)	46.0 (40)	49.4 (42)	0.76
Median time to ICU death (days; range)	11 (3–82)	13 (4–76)	0.59
Hospital mortality (%; number in parentheses)	52.9 (46)	54.1 (46)	0.88
Median time to hospital death (days; n; range)	12 (46, 3–197)	13 (46, 4–76)	0.76
Ventilator-associated pneumonia (%; number in parentheses)	35 (40.2)	25 (29.4)	0.13
Patients needing additional (rescue) therapy over and above routine at any time while ventilated (n; percentage within group)	45 (51.7)	37 (43.5)	0.28
Total events requiring additional (rescue) therapy during period of ventilation (total routine physiotherapy assessments/treatments during same period); rescue therapy as percentage of routine physiotherapy	68 (2384); 2.8%	51 (2227); 2.3%	

endpoints were analysed by Mann–Whitney U or contingency tables with chi-square analysis for independence. Study endpoints were analysed on an intention-to-treat basis.

Data were analysed with Statview (Statview, SAS Institute, Cary, N.C.).

Results

One hundred eighty patients entered the study between November 1996 and January 2000, 91 randomised to chest physiotherapy (group P) and 89 to controls (group C). Eight patients were withdrawn from the study, two became

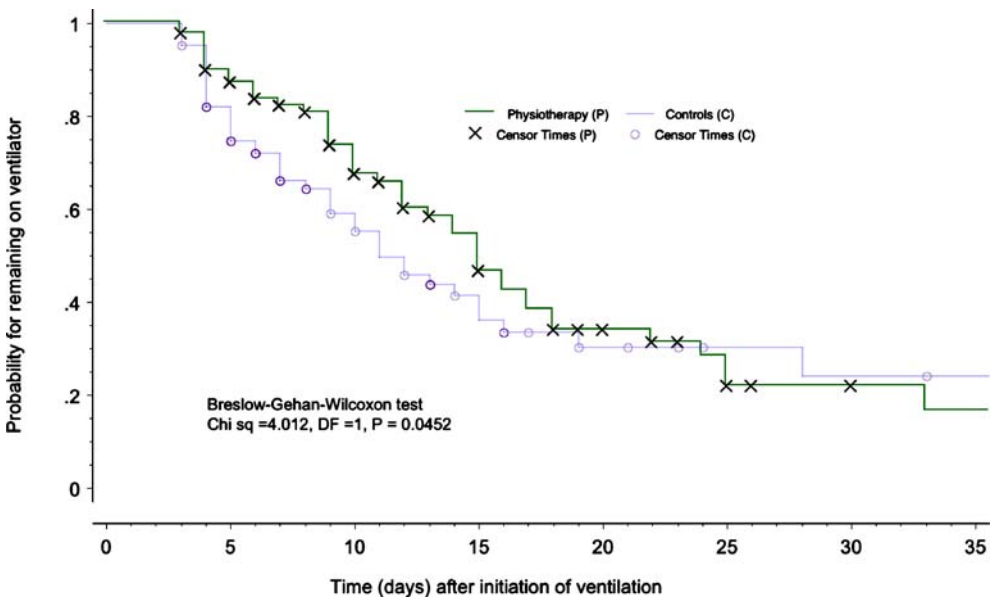

Fig. 1 Kaplan–Meier probability of remaining on a ventilator for control patients and those receiving chest physiotherapy. Data has been censored for patients who died prior to becoming ventilator-free

brain-stem dead after randomisation and before treatment started, 2 had therapy withdrawn due to overwhelming illness, 2 had their randomisation inadvertently revealed within 24 h and 2 patients were transferred to another ICU while ventilated.

Interim analysis of the first 100 patients showed that time to become initially ventilator-free for 23 ICU survivors in group P ($n = 49$) was 14.04 ± 10.7 days (mean $\pm$ SD) vs. 8.56 ± 4.7 days for 23 ICU survivors in group C ($n = 51$). Power calculations based on survivors, with type-1 error < 0.05 and 80% power, suggested a need for at least 76 patients in each group. The study was continued aiming for 90 patients in each group.

The study resulted in over 4,611 physiotherapy assessments among 172 patients. There were no differences between groups in demographics or case-mix based on APACHE II diagnostic categories (Tables 1, 2) [17]. Median APACHE II estimated risk of death based on the first 24 h of ICU admission was slightly higher in the physiotherapy group; this failed to reach significance (49 and 41%, groups P and C, respectively, $p = 0.1$). Median Sequential Organ Failure Assessment (SOFA) scores on the day of admission to the study were similar: 10 and 9 for groups P and C, respectively ($p = 0.22$) [18].

There were no differences in ICU mortality nor the time to death. Median time to death was 11 and 13 days in the physiotherapy and control groups, respectively. Table 3 summarises the outcome measures.

More patients had a tracheostomy in group P (36.7%) than group C (28.8%); this was not significant ($P = 0.12$).

The natural logarithm cumulative hazard plot for the initial time to become ventilator-free demonstrated non-proportional hazards. The Kaplan–Meier curve was right censored for patients who died while on the ventilator and analysed by the Breslow–Gehan–Wilcoxon test. Median time (time for 50% of patients within a group) to become ventilator-free was 15 and 11 days for groups P and C, respectively ($p = 0.047$; Fig. 1). The time difference between groups fell to 3 days when 60% of patients within each group became ventilator-free, but by the time 75% of patients were ventilator-free within each group there were no differences. Patients needing re-ventilation for respiratory reasons were similar: 14.1 and 12.6% in groups C and P, respectively. The median time before patients required re-ventilation was 1 and 3.5 days for groups P and C respectively, which was not significant. Ventilator-associated pneumonia was observed in 60 of the 172 patients, 35 and 25 in groups P and C, respectively ($P = 0.13$). There were more patients needing rescue therapy in the physiotherapy group than controls 51.7% (68 events) and 43.5% (51 events), respectively; this was not significant. Rescue physiotherapy represented less than 3% of the total physiotherapy received by the patients.

Discussion

As far as we are aware, this is the first PRCT to explore the impact of CPT on the time to initially become ventilator-free among patients ventilated for more than 48 h. Physiotherapy was provided after twice-daily routine assessments and based on techniques which improve lung volumes and secretion removal. About 50% of patients in each group additionally needed at least one episode of rescue physiotherapy for desaturation due to mucus plugging while ventilated. These treatments represented less than 3% of the total physiotherapy received and are unlikely to have influenced study outcome.

Surprisingly, the median time to become ventilator-free (the time for 50% of patients in a group to become ventilator-free) was 4 days longer in those who received physiotherapy than a control group. It is notable from the Kaplan–Meier curve (Fig. 1) that the groups separated after day 5. At this time 10% of physiotherapy patients had become ventilator-free compared with 25% of controls. This difference gradually reduced so that by day 19 the proportion of ventilator-free patients was 75% in both groups. The reason for this pattern is not clear. An a posteriori comparison of the 147 patients who became ventilator-free by day 19 with those 24 patients who still remained ventilated revealed a difference in the incidence of smoking: 48% among those weaned in the first 19 days compared with 66% among those still ventilated. One might speculate that increased prevalence of this factor overwhelmed the advantage held by the control group. Physiotherapy had no effect on length of ICU stay, mortality or the time to death. The incidence of VAP, risk of death based on APACHE II and SAPS II, and distribution of neurological patients were not different between groups.

Inevitably, some patients who initially became ventilator-free later required repeat ventilator support. Twenty-three patients required re-ventilation for respiratory reasons. There were no significant differences in the number of patients requiring re-ventilation nor the time between being initially ventilator-free and re-ventilation. Interestingly, time to ICU discharge was not shorter for control patients despite less time to become ventilator-free. It is probable that this endpoint was confounded by the effect of re-ventilation, which delayed discharge in both groups. Additionally, at the time of the study our institution had no step-down facilities which potentially delayed discharge for both groups.

The observed longer median ventilation period for those receiving CPT might have been due to study bias or a consequence of CPT as practiced in our unit.

Potential sources of bias include severity of illness and case-mix; however, reasons for admission to ICU, APACHE II risk of death and SOFA were not significantly different [17, 18].

There were more neurosurgical patients in the physiotherapy group (24 vs. 16 controls); however, the protocol for the first 72 h for these patients includes minimal physiotherapy until intracranial cerebral pressure (ICP) becomes more stable. These patients can suffer temporary perturbations in ICP and cerebral perfusion pressure (CPP) during suctioning [19–22].

Bias related to administration of treatment by medical and nursing staff was minimised by study design, which although not only concealed the patients' randomisation but also whether a patient was in the study at all. It is unlikely that there was bias due to medications which influence weaning, there had been no sedation (fentanyl and midazolam infusions), or fluid protocol changes over the study period. The study lasted over 3 years and might have introduced bias from personnel change, this would have affected both groups.

Weaning guided by protocol and clinical judgement might have introduced bias; however, premature decisions would have become manifest in re-ventilation rates. The re-ventilation rates for respiratory insufficiency were not significantly different between groups.

This study might be criticised for its "black-box" approach, i.e. patients in group P were recipients of any combination of treatments to match their evolving pathology, including no treatment at all. However, the study aim was to measure the impact of the physiotherapy package as practiced in our unit, and therefore a black-box approach was appropriate. It is acknowledged that with such a design it would be impossible to identify the useful elements of physiotherapy for a particular pulmonary pathology, and further would be impossible to determine manoeuvres which disproportionately disadvantaged the physiotherapy group. Intrinsically, a black-box approach increases risk of type-I and type-II errors, particularly in small studies, because many variables remain unidentified. For this reason we undertook an interim analysis taking into account the effect of mortality to estimate study size.

A further bias might be the influence of non-blinded physiotherapists. We made the assumption that physiotherapists would provide treatment which they considered would benefit a patient. Although some therapies might have been considered inappropriate by an independent group of physiotherapists, the absence of evidence-based data makes judgement of appropriateness of therapy a matter of opinion.

The immediate and short-term benefits of CPT for patients with secretions have been well documented [9, 23, 24]; however what has not been clear is whether physiotherapy directed to increase lung volumes and remove secretions leads to earlier weaning in ventilated patients. It is notable that although studies show benefits immediately after physiotherapy, most have failed to show sustained improvements in oxygen saturation, gas exchange or pulmonary compliance [7, 25–27]. A recent non-randomised study hinted at prophylactic benefit from CPT among patients ventilated for more than 48 h. It showed that CPT was associated with an impressive reduction of VAP, although duration of ventilation, length of ICU stay and mortality were unchanged [28]. By contrast, our study showed no difference in the incidence of VAP; indeed, it was non-significantly higher among those who received physiotherapy.

Although this study was not designed to explain our unexpected adverse observations, we might speculate about some processes associated with CPT that might be contributory.

Alveolar atelectasis can follow tracheal suctioning which fails to respond to recruitment manoeuvres with manual hyperinflation [27, 29]. Conceivably, derecruitment and micro-collapse is more likely in infected areas producing secretions. It is likely that de-aerated lung causes local hypoxic vasoconstriction, which reduces antibiotic access and infection resolution.

It is also reasonable to suggest that a suction catheter provides inadvertent inoculation of the lower trachea with infected proteinaceous material from a tracheal tube.

One aim of CPT is to mobilise bronchial secretions by repositioning and cough stimulation. Such manoeuvres in intubated patients might cause tracheal tube cuff deformation, and micro-aspiration of stagnant sub-glottic secretions. This deformation would be in addition to the continuous rhythmic cuff changes with each ventilator cycle. Coughing increases intra-abdominal pressure and potentially increases the risk of regurgitation and micro-aspiration.

Chest physiotherapy is also associated with temporary haemodynamic and metabolic disturbances. Manual hyperinflation has been noted to reduce cardiac output, whereas dysrhythmias, blood pressure perturbations, increases in oxygen consumption and carbon dioxide production have also been reported [3, 30–40]; however, others suggest that these disturbances are not observed [41]. Whether such changes adversely affect weaning is unknown.

In studies examining patient response to intensive care interventions, CPT was found to be the intervention which most altered metabolic rate, varying from 20 to 52% [36, 40, 42]. Aitkenhead showed that, despite sedation, patients receiving CPT increased endogenous norepinephrine and epinephrine concentration by 50 and 150%, respectively. This resulted in rate pressure products which have been associated with complications in patients with ischaemic heart disease [35]. These observations contrast with others which indicate no haemodynamic changes [43, 44]. Haemodynamic disturbances, when they occur, are usually temporary; it is difficult to know whether they influence weaning.

Although CPT altered the median time to become ventilator-free, it had no impact on mortality, perhaps because outcome is overwhelming determined by the

severity of acute pathophysiology, premorbid state and effectiveness of specific therapies.

Conclusion

This PRCT examined the impact of physiotherapy on weaning patients requiring ventilation for more than 48 h. We found that the first 50% of patients to be weaned within a CPT group took 4 days longer to wean than a control group. There was no impact on outcome or ICU length of stay. The suggestion is that simple suctioning and decubitus positioning is at least as effective as CPT in such patients. These results cannot be extended to non-ventilated patients and do not invalidate CPT for sudden events such as atelectasis. The authors suggest that this study should be repeated before embarking on the difficult task of exploring mechanisms accounting for our observations.

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