

Physiotherapy intervention in intensive care is safe: an observational study

Litsa Zeppos¹, Shane Patman², Susan Berney³, Julie A Adsett⁴, Julie M Bridson⁵
and Jennifer D Paratz^{4,6}

¹La Trobe University ²University of Notre Dame ³Austin Hospital ⁴Royal Brisbane and Women's Hospital ⁵Royal Hobart Hospital
⁶University of Queensland
Australia

Question: How often do adverse events (including adverse physiological changes) occur during physiotherapy intervention in intensive care? **Design:** A multi-centre prospective observational study. **Participants:** Five tertiary level university-affiliated intensive care units. **Outcome measures:** All physiotherapy intervention in five intensive care units over a three month period. When certain specified changes occurred during physiotherapy intervention, details were noted including diagnosis of patient, intervention, vital signs, radiological changes, co-morbidities, chemical pathology, and fluid balance. **Results:** 12 281 physiotherapy interventions were completed with 27 interventions resulting in adverse physiological changes (0.2%). This incidence was significantly lower than a previous study of adverse physiological changes (663 events in 247 patients over a 24-hour period); the incidence during physiotherapy intervention was lower than during general intensive care. Common factors in the patients who had an adverse physiological change were a deterioration in cardiovascular status (ie, decrease in blood pressure or arrhythmia) in patients on medium to high doses of inotropes/vasopressors, unstable baseline hemodynamic values, previous cardiac co-morbidities and intervention consisting of positive pressure or right side lying. **Conclusion:** The incidence of adverse events during physiotherapy intervention in these five tertiary hospitals was low, demonstrating that physiotherapy intervention in intensive care is safe. [Zeppos L, Patman S, Berney S, Adsett J, Bridson J, Paratz JD (2007) *Physiotherapy intervention in intensive care is safe: an observational study. Australian Journal of Physiotherapy* 53: 279–283]

Key words: Intensive Care, Physiotherapy, Audit, Task Performance Analysis, Critical Incident

Introduction

Physiotherapy intervention is regarded as an important component in the management of patients in intensive care (Risley and Jones 2003) and has been demonstrated to provide both short- and medium-term benefits (Berney and Denehy 2002, Hodgson et al 2000, Ntoumenopoulos et al 2002, Paratz et al 2002). However, there have been some claims that, in intensive care, physiotherapy intervention results in adverse physiological changes (Hammon et al 1992, Singer et al 1994, Weissman et al 1994), ie, clinically-significant alterations in haemodynamic, respiratory, or intracranial parameters necessitating remedial intervention. Since intensive care patients are critically ill, they do have the potential to become unstable during all aspects of management, such as with basic nursing care, position changes, suction, or physiotherapy intervention. Adverse physiological changes can also occur spontaneously in intensive care patients, as shown by Shoemaker et al (1989) who recorded 637 such events in 247 patients over a 24 hour period.

Many of the studies reporting adverse physiological changes during physiotherapy intervention in intensive care have been methodologically flawed. These studies have included patients who were haemodynamically unstable and not representative of patients to whom the intervention would be applied (Hammon et al 1992, Singer et al 1994, Weissman et al 1994). In contrast, other studies have found beneficial or minimal adverse effects during physiotherapy intervention (Berney and Denehy 2002, Berney and Denehy 2003, Hodgson et al 2000, Ntoumenopoulos et al 2002, Paratz et al 2002, Paratz et al 2006, Patman et al 1998). These studies

included patients for whom the questionnaire respondents (Hodgson et al 1999, King and Morrell 1992) had a physiological rationale for physiotherapy intervention.

Audits are an effective way to identify deficiencies in quality of care and are an accepted method of improving patient safety by identifying factors contributing to adverse events. Large audits of adverse events in intensive care have been published (Beckmann et al 1996, Beckmann et al 2003, Buckley et al 1997, Hart et al 1994) which did not record or report any adverse physiological changes associated with physiotherapy intervention. Data concerning the actual incidence of adverse physiological changes during physiotherapy intervention are required in order to determine the safety of physiotherapy intervention in intensive care. Therefore the research question for this prospective observational study was:

How often do adverse events (including adverse physiological changes) occur during physiotherapy intervention in intensive care?

Using information obtained from auditing adverse physiological changes associated with physiotherapy intervention in intensive care, experimental studies can be planned to investigate if these changes occur in particular patients or during particular interventions.

Method

Design

A multicentre prospective observational study was conducted at five tertiary level intensive care units in Australia. Over

a three-month period, data were recorded on any adverse event associated with physiotherapy intervention in any one of these intensive care units. Physiotherapy intervention was defined as intervention completed or directed specifically by a physiotherapist for any of the following: directed positioning, mobilisation, transfer, active or passive exercise, manual hyperinflation, ventilator hyperinflation, recruitment manoeuvres, application of oxygen, suction (endotracheal tube, tracheostomy, oral, or nasal), insertion of airway, manual interventions (eg, percussion or vibration), breathing interventions, and positive pressure interventions including continuous positive airway pressure, bilevel positive airway pressure, intermittent positive airway pressure breathing and positive expiratory pressure with or without oscillation. The senior physiotherapist at each participating intensive care recorded the total number of physiotherapy interventions provided in intensive care during this three-month period and acted as the local co-ordinator assisting staff to complete the data sheet if necessary. Expedited approval was obtained from the relevant institutional ethics committees.

Participants

Median number of beds for each intensive care unit was 22 (IQR 18.5–39).

Outcome measures

Five senior intensive care physiotherapists (median experience in intensive care 15 years, IQR 8.5–21) and one Honours student defined the adverse events (Box 1) by online discussion, achieving consensus by the fourth draft. A data collection sheet was also developed, achieving consensus by the third draft (see Appendix 1 on the eAddenda.) The data sheet and definitions of an adverse event were also reviewed by three senior intensive care physiotherapists independent of this study for content validity.

This data sheet described the adverse change and included a mixture of open and closed responses. If an adverse change occurred, a detailed report was completed by the physiotherapist, comprising details of: patients (including admission diagnosis, conditions arising during hospitalisation, co-morbidities, medications, as well as vital signs, blood gas values, electrolyte and fluid balance immediately prior to episode), adverse event (including change in vital signs, duration, management, and outcome), intervention (including position and equipment), and the demographics of the physiotherapist involved in the intervention (eg, senior or junior).

Data analysis

Data from the completed data collection sheets were collated by the principal investigator (LZ). Results were examined using descriptive and frequency analysis.

Results

A total of 29 completed questionnaires detailing adverse physiological changes associated with physiotherapy intervention in intensive care patients during the three month audit period were returned. On review, two of these returned questionnaires involved episodes that did not conform to the definition of an adverse physiological change and data were discarded. The remaining 27 completed questionnaires related to adverse physiological changes involving 23 patients, ie, some patients experienced an adverse physiological change more than once. The total

Box 1. Definition of adverse events, which include adverse physiological changes.

- Alteration in blood pressure > or < 20% of resting values which necessitates stopping intervention or requires remedial intervention (eg, inotropes)
- Alteration in heart rate > or < 20% of resting values which necessitates stopping intervention or requires remedial intervention
- New arrhythmia (eg, atrial fibrillation, increased number of ectopic beats per minute, ST depression or elevation, increased magnitude of ST depression, bigeminy, trigeminy, ventricular tachycardia, ventricular fibrillation, asystole)
- Desaturation of oxyhaemoglobin >10% of baseline levels or a figure which necessitates stopping intervention or requires remedial intervention
- Pulmonary artery pressure (systolic) over 60 mmHg
- Pneumothorax detected immediately following intervention
- Agitation resulting in detachment of equipment or lines or requiring increased sedation
- Episode related to incorrect procedure (eg, incorrect connection of equipment, level of inspired oxygen too high)
- Fall during mobilisation (eg, transfer to chair, walking, or tilt table)
- Consultative event (ie, asking the nurse to turn a patient to specified side or sit a patient out of bed) resulting in an episode as above within 30 minutes of the request

number of physiotherapy interventions recorded during this audit period was 12 281, and the incidence of adverse physiological changes associated with physiotherapy intervention was therefore 0.2%. The total number of actual changes in physiological parameters was 41, as often multiple parameters (heart rate and blood pressure) altered during intervention.

The 23 patients in whom an adverse physiological change was recorded had a mean age of 57.7 years (range 16 to 89) and included 15 males. Median length of intensive care stay was 3.0 days (IQR 2.0 to 12.5) and time on ventilation 3 days (IQR 2.0 to 10.0). Ventilation modes included synchronised intermittent mandatory ventilation (n = 10), spontaneous with CPAP/pressure support (n = 11) and pressure controlled ventilation (n = 1). The median level of positive end expiratory pressure was 7 cmH₂O (IQR 5.0 to 8.75), median FiO₂ 0.40 (IQR 0.35 to 0.55) and median PaO₂/FiO₂ ratio (a measure of oxygenation) 192.7 (IQR 162.2 to 290.0) In this sample of patients with adverse physiological changes associated with physiotherapy intervention, there were heterogeneous reasons for admission to intensive care (Table 1). Pre-existing cardiac co-morbidities were present in 96%, and a high percentage of patients (78%) demonstrated abnormal vital signs prior to intervention. Central venous pressure was within normal range for the majority of the patients. Of patients who experienced an adverse physiological change during physiotherapy intervention, 86% were on vasopressor or inotropic support (Table 1).

Table 1. Admission diagnosis, inotropic and/or vasopressor support, intervention, and adverse events for each episode.

Episode	Admission diagnosis	Inotropic and/or vasopressor support	Intervention	Adverse event
1	Multitrauma	Noradrenaline (2 µg/min)	Manual hyperinflation	Mean arterial pressure 70 to 60 mmHg
2	Community acquired pneumonia	Noradrenaline (10 µg/min)	Right side lying, manual hyperinflation	Mean arterial pressure 70 to 40 mmHg
3	Closed head injury	Noradrenaline (12 µg/min)	Manual hyperinflation	Intracranial pressure 19 to 32 mmHg
4	Closed head injury + multi-trauma		Percussion and vibration, manual hyperinflation	Intracranial pressure 22 to 50 mmHg
5	Community acquired pneumonia + acute coronary syndrome	Noradrenaline (8 µg/min) Dobutamine (8 µg/min)	Right side lying, manual hyperinflation, endotracheal suction	Saturation of peripheral oxygen 96 to 85% Mean arterial pressure 75 to 59 mmHg
6	Community acquired pneumonia	Noradrenaline (9.5 µg/min) Dobutamine (2 µg/min)	Manual hyperinflation	Mean arterial pressure 76 to 60 mmHg
7	Community acquired pneumonia		Walk	Respiratory rate 33 to 50 breaths/min
8	Septic shock	Noradrenaline (6 µg/min) Dobutamine (6 µg/min)	Right side lying, manual hyperinflation	Heart rate 85 to 38 beats/min
9	Septic shock	Noradrenaline (3 µg/min) Dobutamine (2 µg/min)	Right side lying, manual hyperinflation	Heart rate 80 to 44 beats/min
10	Burns, septic shock, acute respiratory distress syndrome	Noradrenaline (12 µg/min)	Passive movements	Mean arterial pressure 78 to 55 mmHg
11	Cardiac arrest	Noradrenaline (5 µg/min)	Right side lying, manual hyperinflation, endotracheal suction	Heart rate 80 to 165 beats/min Mean arterial pressure 76 to 125 mmHg
12	Pancreatitis Multi-organ system failure, septic shock		Right side lying	Heart rate 108 to 25 beats/min Mean arterial pressure 75 to 48 mmHg
13	Closed head injury		Manual hyperinflation	Mean arterial pressure 120 to 140 mmHg
14	Closed head injury		Manual hyperinflation, endotracheal suction	Mean arterial pressure 132 to 160 mmHg
15	Closed head injury		Manual hyperinflation, endotracheal suction	Heart rate 70 to 48 beats/min, intracranial pressure 8 to 26 mmHg
16	Septic shock	Noradrenaline (8 µg/min) Dobutamine (2 µg/min)	Right side lying, manual hyperinflation, endotracheal suction	Mean arterial pressure 70 to 57 mmHg
17	Septic shock	Noradrenaline (6 µg/min)	Right side lying, manual hyperinflation, endotracheal suction	Mean arterial pressure 91 to 71 mmHg
18	Septic shock	Noradrenaline (8 µg/min)	Manual hyperinflation	Atrial fibrillation – left bundle branch block, multiple ventricular ectopic beats
19	Closed head injury	Noradrenaline (7 µg/min)	Manual hyperinflation – wrong connection	Saturation of peripheral oxygen 96 to 88%
20	Post laparotomy	Noradrenaline (6 µg/min)	Manual hyperinflation	Systolic arterial pressure/diastolic arterial pressure 110/73 to 210/117 mmHg
21	Closed head injury		Manual hyperinflation	Agitated – self extubated
22	Aspiration pneumonia		Right side lying, ventilator hyperinflation, endotracheal suction	Saturation of peripheral oxygen 97 to 78%
23	Community acquired pneumonia, acute pulmonary oedema		Right side lying, ventilator hyperinflation, endotracheal suction	Mean arterial pressure 90 to 62 mmHg
24	Cardiac arrest, acute pulmonary oedema	Noradrenaline (3 µg/min)	Right side lying, head down tilt, ± positive end expiratory pressure	Mean arterial pressure 75 to 58 mmHg
25	Abdominal aortic aneurysm repair	Noradrenaline (6 µg/min)	Right side lying, head down tilt, ventilator hyperinflation	Mean arterial pressure 100 to 150 mmHg
26	Post coronary artery bypass graft, low cardiac output	Adrenaline (4 µg/min) Milirone (15 µg/min)	Supine – right side lying	Systolic arterial pressure/diastolic arterial pressure 115/75 to 80/40 mmHg Saturation of peripheral oxygen 100 to 85% Heart rate 110 to 200 beats/min
27	Post coronary artery bypass graft intra-aortic balloon pump, nitric oxide	Noradrenaline (18 µg/min) Adrenaline (2 µg/min)	Supine – right side lying	Systolic arterial pressure/diastolic arterial pressure 103/67mmHg to unrecordable Saturation of peripheral oxygen 97 to 88 mmHg Pulmonary artery pressure 53 to 90 mmHg

Alterations in the cardiovascular system accounted for 0.3% of the total interventions and were the most commonly-reported adverse physiological changes (78%), with a decrease in pulse pressure (ie, a decrease in the difference between systolic and diastolic blood pressure) the most frequent of all adverse physiological changes (42%). Arrhythmias, (chiefly bradycardias) were responsible for 15% of adverse physiological changes which represented < 0.1% incidence of arrhythmia within the total number of physiotherapy interventions during the three month audit.

The episodes of adverse physiological change in this audit resulted in: spontaneous recovery once the physiotherapy intervention was ceased (30%), recovery after specific intervention (59%) and no details given (11%). Interventions to remediate the adverse event included either an increase in inotropes for decreased blood pressure (n = 5), alteration in position (n = 6), cardiopulmonary resuscitation for bradycardia (n = 1), hyperoxygenation with manual hyperinflation (n = 1), or an increase in inspired level of oxygen (n = 1). Intraventricular drainage to correct increased intracranial pressure was required for two patients.

The most common physiotherapy intervention when these 27 episodes occurred was administration of increased positive pressure (59%), manual or ventilator hyperinflation, or recruitment manoeuvre. During manual hyperinflation the most common circuit was the Mapleson C circuit (80%). A manometer was not used with any circuit during manual hyperinflation but a positive end expiratory pressure valve was used for any patient with a positive end expiratory pressure > 7.5 cmH₂O. Endotracheal or tracheal suctioning featured in 19% of adverse episodes, representing < 0.1% of total interventions. Open rather than closed suctioning was used in the majority of episodes involving suction. The majority of patients who experienced adverse events were positioned in right side lying during intervention (Box 1).

Discussion

In the intensive care environment there is a higher risk of errors in patient management, therefore safety reporting and analyses of adverse events are important to improve patient care (Beckmann et al 1996). Recent recommendations have encouraged reporting that is voluntary, confidential, non-punitive and, importantly, involves feedback and education to staff involved (Osmon et al 2004).

Overall, there was an extremely low incidence of adverse events associated with physiotherapy intervention. Most of the adverse events were physiological changes. This incidence of adverse physiological changes was much lower than the only reported study of spontaneous changes in haemodynamics recorded in intensive care patients over 24 hours (Shoemaker et al 1989). However the patients described by Shoemaker et al were all defined as high-risk surgical patients who were being monitored invasively via a pulmonary artery catheter. The incidence of arrhythmias in the current audit was also lower than that found in a previous survey of spontaneous abnormal rhythms in critically-ill patients (Artucio and Pereira 1990). Although previous empirical studies in intensive care have reported adverse effects during physiotherapy intervention (Hammon et al 1992, Weissman et al 1994) the aim of these studies was to use physiotherapy intervention as a physiological stressor and subsequently investigate the effectiveness of various medications to suppress abnormal cardiovascular and metabolic responses. The physiotherapy intervention was

not aligned with accepted practice (Hodgson et al 1999).

Adverse physiological changes during suction, a potentially dangerous manoeuvre performed by various health professionals, have also been recorded in a previous study. Robles et al (2002) reported adverse physiological changes occurring in 48% of suctioning interventions. In this current audit, suctioning was performed by physiotherapists and adverse physiological changes occurred < 0.1% of the time.

Most patients in this audit received manual hyperinflation using a Mapleson C circuit. It has been shown (McCarren and Chow 1996) that a Mapleson C circuits results in significantly larger inspiratory pressures and tidal volumes than other circuits during manual hyperinflation. The applications of manual hyperinflation in those patients who experienced adverse events did not include a manometer in the resuscitation bag circuit, despite previous recommendations that this would monitor and limit airway pressure adequately (Redfern et al 2001).

A potential cause of respiratory deterioration during manual hyperinflation is disconnection from positive end expiratory pressure and subsequent derecruitment. However, the maximum positive end expiratory pressure was 10 cmH₂O and all patients with a baseline greater than 7.5 cmH₂O had a positive end expiratory pressure valve in situ during manual hyperinflation. There were only two reports of desaturation on disconnection from the ventilator as part of the manual hyperinflation procedure.

An additional finding was that adverse changes in haemodynamics occurred when patients were placed into right side lying. Large hemodynamic changes (decrease in mean arterial pressure and right ventricular diastolic volume) have previously been reported in critically-ill patients dependent on inotropes turned into right side lying (Bein et al 1996). The results of our audit contrast with Berney and Denehy (2003) who investigated haemodynamics in critically-ill patients and found no adverse effects from side lying. Importantly they ensured that haemodynamics were within normal limits before enrolment. The majority of patients in this audit who experienced adverse physiological changes had abnormal haemodynamic values.

Twelve patients (Table 1) who had abnormal cardiovascular values pre-intervention and were requiring medium to high levels of pressor support, demonstrated a decrease in blood pressure during the application of a positive pressure manoeuvre. Patients requiring this level of pressor support have poor systemic vasoconstriction and are unable to compensate if increased positive pressure is delivered (Jellema et al 2000, Paratz et al 2002, Paratz and Lipman 2006). Further education is required in order to educate junior physiotherapists about the effects of increased positive pressure on patients with unstable haemodynamics.

A previous survey indicated that physiotherapists in Australian intensive care units are proactive in early mobilisation (standing, tilt table, sit out of bed, walking) (Chang et al 2004). Only one adverse physiological change during this audit was associated with mobilisation of the patient by a physiotherapist, suggesting that overall this intervention is applied safely.

Of interest was the finding that 78% of patients who had adverse physiological changes associated with physiotherapy

intervention had cardiovascular or intracranial values outside normal levels prior to intervention. Comments made on the data sheets by the physiotherapists concerned, showed they were aware that these patients had abnormal values, but they were directed to treat the patient by medical staff in the belief that the benefits would outweigh the risks associated with intervention. This occurred with all levels of physiotherapist and is an important professional issue. As physiotherapists are primary practitioners and have prime responsibility for their management strategies, issues of patient safety, clinical governance, and responsibility are factors to be considered.

There were a number of limitations to this audit. Only hospitals with tertiary level intensive care units were studied. Different results may have been obtained in intensive care units in smaller or private hospitals. A further potential limitation was the compliance by physiotherapists in completing details about adverse physiological changes. In two of the participating hospitals, physiotherapists in intensive care tend to work in pairs which would serve as a checkpoint. Although the completion of the audit form was anonymous, most physiotherapists involved in an intervention which led to adverse physiological changes appeared keen to discuss the event and possible causes. Another limitation is that details of *all* physiotherapy interventions (ie those not associated with adverse physiological changes) were not recorded. However such extensive data were beyond the scope of this study.

The overwhelming majority of physiotherapy interventions in this three-month audit period were safe. Associations in this audit cannot imply causality and whether the adverse physiological changes were spontaneous is not known.

eAddendum: Appendix 1 available at www.physiotherapy.asn.au.

Correspondence: Dr Jennifer Paratz, C/- Dept of Intensive Care, Royal Brisbane Hospital, Herston, Queensland 4029, Australia. Email: j.paratz@uq.edu.au

References

- Artucio H, Pereira M (1990) Cardiac arrhythmias in critically ill patients: epidemiologic study. *Critical Care Medicine* 18: 1383–1388.
- Beckmann U, West LF, Groombridge GJ, Baldwin I, Hart GK, Clayton DG, Webb RK, Runciman WB (1996) The Australian Incident Monitoring Study in Intensive Care: AIMS-ICU. The development and evaluation of an incident reporting system in intensive care. *Anaesthesia and Intensive Care* 24: 314–319.
- Beckmann U, Bohringer C, Carless R, Gillies DM, Runciman WB, Wu AW, Pronovost P (2003) Evaluation of two methods for quality improvement in intensive care: facilitated incident monitoring and retrospective medical chart review. *Critical Care Medicine* 31: 1006–1011.
- Bein T, Metz C, Keyl C, Pfeifer M, Taeger K (1996) Effects of extreme lateral posture on hemodynamics and plasma atrial natriuretic peptide levels in critically ill patients. *Intensive Care Medicine* 22: 651–655.
- Berney S, Denehy L (2002) A comparison of the effects of manual and ventilator hyperinflation on static lung compliance and sputum production in intubated and ventilated intensive care patients. *Physiotherapy Research International* 7: 100–108.
- Berney S, Denehy L (2003) The effect of physiotherapy treatment on oxygen consumption and haemodynamics in patients who are critically ill. *Australian Journal of Physiotherapy* 49: 99–105.
- Buckley TA, Short TG, Rowbottom YM, Oh TE (1997) Critical incident reporting in the intensive care unit. *Anaesthesia* 52: 403–409.
- Chang AT, Boots R, Hodges PW, Paratz J (2004) Standing with assistance of a tilt table in intensive care: a survey of Australian physiotherapy practice. *Australian Journal of Physiotherapy* 50: 51–54.
- Hammon WE, Connors AF Jr, McCaffree DR (1992) Cardiac arrhythmias during postural drainage and chest percussion of critically ill patients. *Chest* 102: 1836–1841.
- Hart GK, Baldwin I, Gutteridge G, Ford J (1994) Adverse incident reporting in intensive care. *Anaesthesia and Intensive Care* 22: 556–561.
- Hodgson C, Carroll S, Denehy L (1999) A survey of manual hyperinflation in Australian hospitals. *Australian Journal of Physiotherapy* 45: 185–193.
- Hodgson C, Denehy L, Ntoumenopoulos G (2000) An investigation of the early effects of manual lung hyperinflation in critically ill patients. *Anaesthesia and Intensive Care* 28: 255–261.
- Jellema WT, Groeneveld AB, van Goudoever J, Wesseling KH, Westerhof N, Lubbers MJ, Kesecioglu J, Van Lieshout JJ (2000) Hemodynamic effects of intermittent manual lung hyperinflation in patients with septic shock. *Heart and Lung* 29: 356–366.
- King D, Morrell A (1992) A survey on manual hyperinflation as a physiotherapy technique in intensive care units. *Physiotherapy* 78: 747–750.
- McCarren B, Chow C (1996) Manual hyperinflation: a description of the technique. *Australian Journal of Physiotherapy* 42: 203–208.
- Ntoumenopoulos G, Presneill JJ, McElholm M, Cade JF (2002) Chest physiotherapy for the prevention of ventilator-associated pneumonia. *Intensive Care Medicine* 28: 850–856.
- Osmon S, Harris CB, Dunagan C, Prentice D, Fraser VJ, Kollef MH (2004) Reporting of medical errors: an intensive care unit experience. *Critical Care Medicine* 32: 727–733.
- Paratz J, Lipman J, McAuliffe M (2002) The effect of manual hyperinflation on hemodynamics, gas exchange and respiratory mechanics in ventilated intensive care patients. *Journal of Intensive Care Medicine* 17: 317–324.
- Paratz J, Lipman J (2006) Manual hyperinflation causes nor epinephrine release *Heart and Lung* 35: 262–268.
- Patman S, Jenkins S, Bostock S (1998) Cardiovascular responses to manual hyperinflation in post-operative coronary artery surgery patients. *Physiotherapy Theory and Practice* 14: 5–12.
- Redfern J, Ellis E, Holmes W (2001) The use of a pressure manometer enhances student physiotherapists' performance during manual hyperinflation. *Australian Journal of Physiotherapy* 47: 121–131.
- Risley JG, Jones MO (2003) Physiotherapy in intensive care. In Bersten AD, Soni N, Oh TE (Eds) *Oh's Intensive Care Manual* (5th ed). London: Butterworth Heinemann, pp 33–40.
- Robles RMP, Corcoles GT, Torres LM, Munoz RF, Cantos De La CY, Arias RS, Parra ML, Alia RML (2002) Frequency of the adverse events during the hygiene of the critical care patients. *Enfermería Intensiva* 13: 47–56.
- Shoemaker WC, Appel PL, Kram HB (1989) Incidence, physiologic description, compensatory mechanisms, and therapeutic implications of monitored events. *Critical Care Medicine* 17: 1277–1285.
- Singer M, Vermaat J, Hall G (1994) Hemodynamic effects of manual hyperinflation in critically ill mechanically ventilated patients *Chest* 106: 1182–1187.
- Weissman C, Kemper M, Harding J (1994) Response of critically ill patients to increased oxygen demand: hemodynamic subsets. *Critical Care Medicine* 22: 1809–1816.