Anaesthesia, 2007, **62**, pages 27–33 doi:10.1111/j.1365-2044.2006.04898.x

Pattern of ventilation during manual hyperinflation performed by physiotherapists

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Summary

The aim of this prospective observational study was to document patterns of ventilation during manual hyperinflation by physiotherapists. Manual hyperinflation with a Mapleson-F system was performed on the same patients on two consecutive days. Patterns of ventilation were recorded using a heated pneumotachometer, pressure transducer and custom designed data acquisition and analysis systems. The mean (SE) results were: inspiratory time 1.45 (0.10) s; volume delivered 1.23 (0.07) l; peak inspiratory and expiratory flow rate 1.51 (0.06) $l.s^{-1}$ and 3.26 (0.30) $l.s^{-1}$, respectively and I : E flow rate ratio 0.63 (0.05). All the physiotherapists achieved an increase in volume which was delivered within a safe and effective pressure range and without cardiovascular compromise. Most (26 out of 34 sessions) performed the technique in the way recommended for enhancing secretion clearance. This is the first study to document comprehensively the pattern of ventilation during manual hyperinflation and provides the basis for further clinical trials evaluating its effectiveness for secretion clearance and volume restoration.

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Accepted: 2 September 2006

Manual hyperinflation is used by physiotherapists in many countries to prevent volume loss, re-inflate areas of atelectasis or collapse, and assist secretion removal [1, 2]. The technique was first described in 1968 [3]; however, relatively little has been published documenting the pattern of ventilation in intubated adult patients during manual hyperinflation. The parameters that have been reported are peak inspiratory pressure (PIP) [4–7], volume delivered [5, 6, 8], mean inspiratory flow rate (MIFR) [9] and peak expiratory flow rate (PEFR) [4, 8, 10]. None of these studies included all these measures of the pattern of ventilation.

A variety of breathing systems have been used for manual hyperinflation. Differences in the pattern of ventilation between circuits have been demonstrated in a test lung model [11]. The circuits used in these clinical studies include the Ambu [7], Laerdal [5, 8, 10] and Mapleson-C (valve not described) [6] and with the Heidbrink valve [4, 12]. The Mapleson-F system (sometimes referred to as Ayres or Jackson-Rees) is also used for manual hyperinflation [1, 11] but to date the pattern of ventilation with this system in the clinical setting has not been reported.

The use of a set PIP to standardise performance during manual hyperinflation has been applied in studies evaluating the effectiveness of the technique to enhance secretion clearance [4, 13] and prevent nosocomial pneumonia [14]. The validity of using PIP to standardise performance has not been evaluated.

The aim of this study was to describe the components of the pattern of ventilation in adult intubated patients defined in terms of inspiratory time (T_I), volume delivered (actual and as a percent of spontaneous tidal volume), peak inspiratory flow rate (PIFR), peak expiratory flow rate (PEFR) and inspiratory to expiratory (I : E) flow rate ratio when physiotherapists performed manual hyperinflation with a Mapleson-F system to a PIP of 40 cm H_2O. These findings document current clinical practice and should provide the basis for further research into the effectiveness of manual hyperinflation in preventing or reversing volume loss and enhancing secretion clearance.

Methods

Ethical approval for the study was obtained from the hospital and university human ethics committees. Data were collected on the performance of manual hyperinflation on the same patient once a day on two consecutive days by the junior rotating physiotherapist working in the unit at the time patients were recruited.

A convenience sample of patients who had been intubated for at least 2 days was assessed by an independent (senior) physiotherapist. If physiotherapy intervention requiring manual hyperinflation was indicated, the patient was approached by the researcher and included if consent was obtained. If subjects were unable to give informed consent, the 'person responsible' was approached.

Exclusion criteria were: inability of the person approached for consent to understand English (patient or person responsible); head injury with a baseline intracranial pressure of ≥ 15 cm H_2O or being managed by hyperventilation; unstable cardiovascular status defined as systolic blood pressure < 100 or > 180 mmHg, or mean arterial pressure < 70 or > 110 mmHg; heart rate < 70 or > 120 beats.min^{-1}; on inotropic support; the presence of a pneumothorax without an intercostal catheter; subcutaneous emphysema or an intercostal catheter present showing an air leak; positive end expiratory pressure during synchronised intermittent mandatory ventilation ≥ 10 cm H_2O; requiring continuous positive airway pressure > 15 cm H_2O with a fraction of inspired oxygen (F_{IO_2}) > 0.50; diagnosis of adult respiratory distress syndrome; acute pulmonary oedema; acute bronchospasm; a baseline PIP on positive pressure mechanical ventilation > 40 cm H_2O.

Manual hyperinflation was performed using a Mapleson-F circuit consisting of an elbow connector with gas inlet port and a 2-litre antistatic rebreathing bag (Rüsch) with the end loop removed (standard bedside resuscitation and manual hyperinflation circuit in this ICU). When using this circuit for manual hyperinflation during inspiration the open end of the bag is pinched closed, the bag is squeezed to deliver the breath and then released to allow expiration. Thus during inspiration it is a closed circuit which allows pressure monitoring. The circuit was connected to wall oxygen at 15 l.min^{-1} and F_{IO_2} 1.0 (as per the ICU protocol). Before the manual hyperinflation breaths commenced, the measurement devices were connected to the circuit (Fig. 1). Custom designed data acquisition and analysis systems [15] were used to record inspiratory and expiratory flow rates and calculate volume delivered. The pneumotachometer was calibrated prior to each treatment session with a 3-litre calibration syringe (Vacumed, Ventura, CA). The acquisition system was modified to include a second recording channel and graphical user interface to record and display airway pressure from a Validyne pressure transducer. Inspiratory time was calculated manually by subtracting the time when flow changed from inspiratory to expiratory, from the time inspiration commenced as recorded by the analysis system.

The junior rotating physiotherapists received no special instruction as to how to perform manual hyperinflation

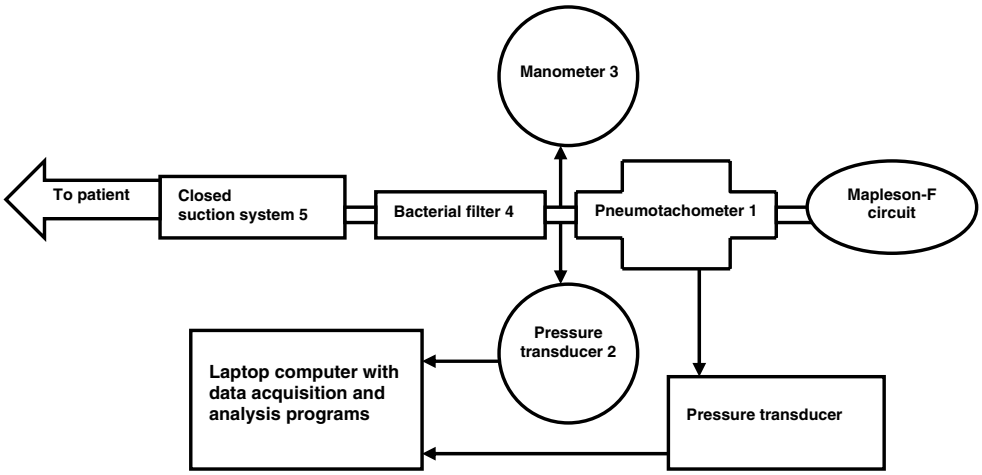

Figure 1 Experimental set up. 1) heated pneumotachometer model 3813, Hans Rudolph Inc., Kansas City, MO, USA. 2) Validyne pressure transducer DP45, Validyne Engineering Corp., Northridge, CA, USA. 3) Astra Meditec, Wika. 4) Pall Medical, Portsmouth, UK. 5) Portex Steri-Cath, FR 12 ref no. 6110-12, Smiths Medical, Kent, UK.

other than the aim was to empty the bag during inspiration, and/or cease the inspiration when a peak inspiratory pressure of 40 cm H_2O was reached by watching the manometer included in the circuit. The manometer reading was calibrated against a water manometer. Six sets of six breaths were performed. No end inspiratory hold was used. If the patient was receiving bronchodilators the treatment was performed between 1 and 3 h after their administration.

The experimental procedure was as follows:

- The therapist decided how they wanted the patient positioned and suction was performed until secretions were clear. The sputum was discarded.
- The patient was left undisturbed for 10 min (rest).

Table 1 Patients' demographic data.

Patient	Age	Gender	Admission diagnosis (past respiratory history) CXR report on the day of treatment	Therapist (no. of sessions)
1	29	M	burns 62% BSA 1 minor patchy airspace change R base 2 minor L basal atelectasis	RP1 (2)
2	80	M	arrest, chest infection/R sided pneumonia (COPD, intensive care admission for respiratory failure previously) 2 bilateral pleural effusions	RP1 (1)
3	76	F	acute pulmonary oedema (smoker 50 pack/year, probable COPD) 1 hazy R base	RP1 (1)
4	75	M	seizures, cause unknown 1 airspace changes both bases, ? aspiration or pulmonary oedema 2 Some clearing of basal effusions 1 airspace changes both bases, some clearing basal effusions 2 persistent R basal opacity, new minor L basal opacity, atelectasis most likely	RP2 (2) RP3 (2)
5	62	F	Oral morphine sulphate overdose (smoker 30 pack/years, COPD) 1 & 2 L lung and R upper zone consolidation	RP4 (2)
6	80	M	Myasthenia gravis (ex-smoker 15 pack/years) 1 L basal atelectasis, R lower zone opacity possibly pleural fluid	RP4 (1)
7	59	M	generalised weakness, cause unknown (tuberculosis as a child, ex-smoker) 1 R basal atelectasis, possibly R pleural effusion 2 no change, some collapse R base	RP4 (2)
8	35	F	burns 10% BSA (smoker) 1 clear	RP5 (1)
9	37	M	burns 70% BSA (smoker) 1 & 2 L lung and R upper zone consolidation	RP5 (2)
10	72	F	Pancreatitis 1 Bilateral pleural effusions with some collapse 2 L and R lower lobe consolidation and volume loss right lower lobe pneumonia, bowel resection	RP5 (2)
11	83	F	1 Lung fields clear 2 Small bilateral effusions, minor atelectasis both bases	RP5 (2)
12	58	M	intra-operative hypertension – excision floor of mouth, anterior glossectomy, bilateral neck dissection (smoker, ETOH) 1 changes of COPD present, band shadowing L lower zone 2 mild prominence upper lobe vessels	RP6 (2)
13	50	M	R basal ganglia haemorrhage (ex-smoker, COPD, recurrent aspiration pneumonia, sleep apnoea) 1 still band shadowing at each lung base 2 persistent L basal collapse/consolidation and R basal atelectasis	RP7 (2)
14	77	M	increasing shortness of breath (ex-smoker, emphysema) 1 & 2 clear	RP8 (2)
15	86	M	occipitocervical fusion (COPD) 1 clear 2 minor bilateral basal opacity	RP9 (2)
16	73	F	? B-cell lymphoma (smoker, COPD) 1 lungs appear clear 2 suggestive of interstitial oedema	RP10 (2)
17	83	F	respiratory failure post emergency infrarenal abdominal aortic aneurysm repair (ex smoker, recurrent chest infections) 1 & 2 persisting bibasal opacity	RP11 (2)
18	31	M	burns 70% BSA (smoker) 1 & 2 lungs and pleural spaces clear	RP11 (2)

M, male; F, female; BSA, body surface area; R, right; L, left; COPD, chronic obstructive pulmonary disease; RP, rotating physiotherapist.

- Treatment consisted of six sets of six manual hyperinflation breaths (based on the protocol of Hodgson et al. [13]). Suction was used as necessary during the treatment and at the end until secretions were clear as determined by the therapist.
- The patient was left undisturbed for 10 min (rest).

The treating physiotherapists and patients were blinded with respect to the values of the measurements recorded (volume delivered, T_I, PIFR, PEFR, I : E flow rate ratio).

Spontaneous tidal volume was recorded from the ventilator prior to the patient being positioned. Heart rate, mean arterial blood pressure and oxygen saturation were manually recorded from the over-bed monitor (Datex Ohmeda S/5 Critical Care Monitor, Madison, WI, USA) every 5 min for 10 min before, during and after treatment.

The pattern of ventilation for all 34 treatment sessions are reported. Data were analysed using SPSS version 14, and the results are presented as descriptive statistics (mean (SE) and range) and correlations (Pearson's *r*). The difference between measures for the same therapist treating the same patient on two occasions (intratherapist variability), are described in two ways. First, the mean value on day 1 was divided by the mean value on day 2 for each measure (percentage difference) and second, the mean (SE) and range for the absolute difference in the measures for the two sessions by the same therapist were calculated (SPSS version 14).

Results

Eighteen patients and 11 junior rotating therapists were recruited for the study. Demographic data for the patients recruited are shown in Table 1.

Patient 3 self-extubated overnight between day 1 and 2 and thus data are only available for one treatment session. Due to technical difficulties, data were not obtained for one of the treatment sessions for patients 2, 6 and 8, and from the pressure transducer for patient 2 during the second treatment. Patient 4 was seen by two different therapists over 4 days, patient 10 went to theatre on day 2 for a laparotomy and was seen on day 3. Data sets were therefore obtained for 34 treatment sessions. Of these, 15 paired data sets were available to compare intratherapist variability.

The length of time intubated before commencing the study ranged from 2 to 28 days (mean 10 days). The mode of respiratory support prior to the treatment sessions was oxygen therapy only (3 via trache mask), continuous positive airway pressure (12) and pressure support ventilation (19). The F_{IO_2} ranged from 0.25 to 0.40 (mean 0.33). No treatments were stopped because of cardiovascular instability.

The mean and standard error (SE) values and range for all measures of the pattern of ventilation are shown in Table 2. The majority of therapists used an inspiratory time of between 1 and 2 s. For two treatment sessions, PIP was well outside the mean (values of 19 and 57 cm H_2O). Of the remaining PIP data, 69% were in the range 35–42 cm H_2O and 31% in the range 30–34 cm H_2O. Spontaneous tidal volume ranged from 0.33 to 0.86 l and the therapists delivered a volume between 133% and 510% of tidal volume. For 26 treatment sessions the average I : E flow rate was ≤ 0.90.

Inspiratory time was highly correlated with volume delivered ($r = 0.88$, $p < 0.001$; Fig. 2) and there was a trend for T_I to be correlated with PIFR ($r = -0.31$, $p = 0.07$; Fig. 3).

With the exception of PEFR, the majority of therapists demonstrated $\leq 20\%$ variability for each of the measures of pattern of ventilation (Table 3). The mean absolute difference for each measure, again with the exception of PEFR, was small (Table 3).

Table 2 Values for T_I, PIP, volume (delivered, spontaneous and $V_{MHI}/V_T\%$), PIFR, PEFR and the I : E flow rate ratio.

	T_I (s)	PIP* (cm H_2O)	Spontaneous tidal volume (l)	Volume delivered (l)	$V_{MHI}/V_T\%$	PIFR ($l.s^{-1}$)	PEFR ($l.s^{-1}$)	I : E ratio flow rate
Mean (SE)	1.45 (0.10)	36 (1)	0.47 (0.03)	1.23 (0.07)	274 (23)	1.51 (0.06)	3.26 (0.30)	0.63 (0.05)
Range	0.51–2.66	19–57	0.33–0.86	0.64–2.16	133–510	0.81–2.31	1.06–7.18	0.24–1.48
No. of sessions	< 1 s 9	< 20 1			< 150% 3			≤ 0.90 26
	1 < 2 s 18	20 < 30 0			150 < 250% 13			> 0.90 8
	2 < 3 s 7	30 ≤ 40 30			250 < 350% 10			
		> 40 2			350 < 450% 6			
					≥ 450% 2			

T_I, inspiratory time; PIP, peak inspiratory pressure; $V_{MHI}/V_T\%$, volume delivered during manual hyperinflation as a percent of spontaneous tidal volume; PIFR, peak inspiratory flow rate; PEFR, peak expiratory flow rate; I : E, inspiratory to expiratory.
*$n = 33$.

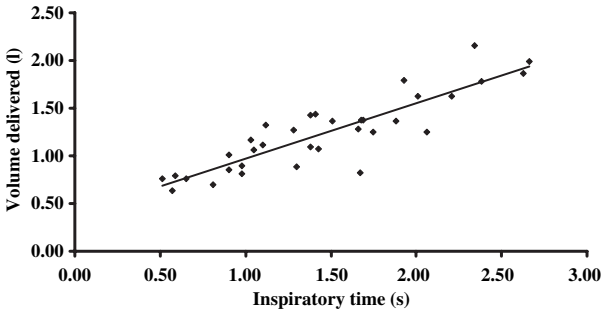

Figure 2 Scatter plot of volume delivered against inspiratory time.

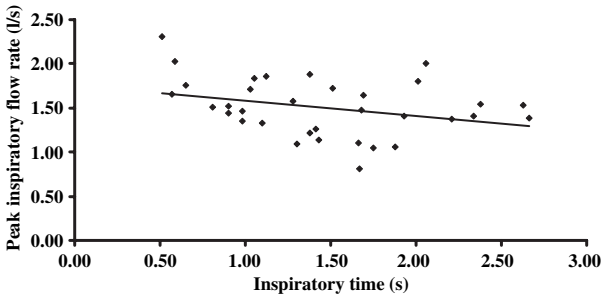

Figure 3 Scatter plot of peak inspiratory flow rate against inspiratory time.

Discussion

Previous descriptions of the performance of manual hyperinflation [3, 16] have recommended a slow inspiration over 3 s. Although there was a range of T_I in this study, intratherapist variability for the same patient was small. The finding of a mean T_I less than the recommended 3 s is in keeping with previous reports of physiotherapist performance in test lung models [11, 17]. The studies by Thomas and Wong [17] and Maxwell and Ellis [11] demonstrated that a longer inspiratory time resulted in a slower inspiratory flow rate. In this study there was a trend for a longer T_I to be associated with a slower PIFR. A slower inspiratory flow rate is thought to assist in a more even distribution of ventilation as the influence of airway resistance on distribution is reduced [18]. Clinically this is important, as over-distension of normal alveoli has been linked to pulmonary damage [19]. The effect of a using a 3-s T_I on inspiratory flow rate and distribution of ventilation during manual hyperinflation requires further investigation in the clinical setting.

The volume delivered by therapists in this study was similar to the ranges previously reported for the Laerdal and Mapleson-C circuits at comparable PIP with the exception of the study by Clarke et al. [6], where PIP was considerably higher (Table 4). Despite standardisng the PIP for this study there was a range of volumes delivered. This was not unexpected as the patients had a variety of pulmonary pathology and therefore it would be anticipated that their pulmonary compliance would also vary. However, as the intratherapist variability for PIP and volume delivered was small, it appears that using PIP to standardise volume delivered in the same patient may be useful. The volume delivered and PIP generated by the majority of therapists in this study should be great enough to assist volume recruitment [20].

The correlation between T_I and volume delivered can be accounted for by the design of the Mapleson-F circuit. During inspiration the volume delivered to the patient consists of not only that squeezed from the reservoir bag, but also the volume being delivered from the oxygen line (in this study 15 $l.min^{-1}$) as it is not isolated from the

Table 3 Description of intratherapist variability in performance for T_I, PIP, volume delivered, PIFR and PEFR (percentage and absolute intratherapist difference, mean (SE) and range).

		Percentage difference day 1 compared to day 2 [no. of therapists (cumulative %)]			
	T_I (s)	PIP (cm H_2O)	Volume delivered (l)	PIFR ($l.s^{-1}$)	PEFR ($l.s^{-1}$)
≤ ±10%†	6 (40%)	12 (80%)	6 (40%)	8 (53%)	4 (27%)
±11 ≤ ±20%‡	6 (80%)	1 (87%)	5 (73%)	4 (80%)	4 (53%)
> ±20%	3	2	4	3	7
		Absolute intratherapist difference			
Mean (SE)	0.20 (0.04)	4 (2)	0.18 (0.03)	0.17 (0.03)	0.65 (0.14)
Range	0.05–0.52	0–22	0.02–0.43	0.01–0.38	0.08–1.90

T_I, inspiratory time; PIP, peak inspiratory pressure; PIFR, peak inspiratory flow rate; PEFR, peak expiratory flow rate.
Cumulative percentage = number of therapists as percent of total (15).
†≤ 10% variability. ‡≤ 20% variability.

Table 4 Comparison of volume delivered, peak inspiratory and expiratory flow rate, and inspiratory pressure during manual hyperinflation in the clinical setting.

		Volume delivered (l)		PIFR ($l.s^{-1}$)		PEFR ($l.s^{-1}$)		PIP (cm H_2O)	
	Circuit	mean (SD)	range	mean (SD)	range	mean (SD)	range	mean	range
Current study	Mapleson-F	1.23	0.64–2.16	1.51	0.81–2.31	3.26	1.06–7.18	36	19–57
Maclean et al. [8]†	Laerdal		0.97–2.03			1.22	0.70–1.56	not reported	
Jones et al. [12]	Laerdal					0.70 (0.17)–1.60 (0.30)		not reported	
	Mapleson-C					0.66 (0.20)–1.67 (0.38)			
McCarren & Chow [5]‡	Laerdal	0.97 (0.21)	0.53–1.64	0.71 (0.36)	0.14–1.84			33	12–64
Clarke et al. [6]	Mapleson-C	1.12 (0.27)	0.71–1.51					51	37–74
Berney et al. [4]	Mapleson-B (modified)					1.91 (0.05)–2.19 (0.06)	40	40	

PIFR, peak inspiratory flow rate; PEFR, peak expiratory flow rate; PIP, peak inspiratory pressure.
†Condition A – manual ventilation without chest compression, corrected for volume delivered. ‡Mean inspiratory flow rate (calculated V_T/T_I).

inspiratory port. A longer inspiratory time thus allows additional flow from the oxygen line.

The PEFR reported exceeded that previously documented for the Laerdal, Mapleson-B and Mapleson-C circuits (Table 4). Other authors have shown that different circuits and exhale valves have varying resistance to expiratory flow [21, 22] and that this can influence PEFR [11]. With the Mapleson-F circuit used in this study, during expiration the reservoir bag fills first and provides little resistance to flow. Once the bag has filled, gas leaves through the open end of the reservoir bag which does not contain an exhale valve. Thus it is possible that the faster PEFR reported is because resistance to expiration is less than for circuits that do include a valve.

As discussed earlier, airway pressure is in part related to respiratory system compliance or, expressed in another way, lung and chest wall recoil. Thus at the same PIP, PEFR should be the same unless there is resistance to expiratory flow. The range in PEFR demonstrated in this study could be due to differences in airway resistance between patients, patient effort during expiration and/or variability in how the therapists manipulated the bag during expiration. The use of 'rapid release' of the bag has been shown to increase PEFR when using the Mapleson-F circuit [11], but the precise performance of this technique has not been described.

The PEFRs documented exceed those shown to promote secretion movement in tube models [23–25] and a sheep model [26]. However, it has been suggested that although there may be a critical PEFR at which secretions start to move, during bidirectional gas flow an I : E flow rate ratio < 0.9 may be also be important [25]. In eight of the 34 sessions in this study, therapists produced an I : E flow rate ratio > 0.9 and thus their technique of manual hyperinflation may not assist secretion removal. Maxwell & Ellis [11] reported that approximately half of the physiotherapists who performed manual hyperinflation as per their current practice using a test lung model failed to achieve an I : E flow rate ratio < 0.9. When these therapists delivered an equivalent volume, but performed inspiration over 3 s, all achieved an I : E flow rate ratio < 0.9. The effect of a 3-s inspiratory time on I : E flow rate ratio during manual hyperinflation in the patient population needs to be documented.

Conclusions

This is the first study to document a comprehensive range of measures of pattern of ventilation including T_I, volume delivered, PIFR, PEFR and the I : E flow rate ratio in intubated patients when physiotherapists perform manual hyperinflation as per current clinical practice using a Mapleson-F circuit. The pattern of ventilation during manual hyperinflation with other circuits needs to be more fully documented.

In this study all physiotherapists achieved an increase in volume which was delivered within a safe and effective pressure range and without cardiovascular compromise. However, almost one quarter did not produce the I : E flow rate ratio suggested for enhancing secretion clearance. The findings have documented a variability in pattern of ventilation during manual hyperinflation between patients at a set PIP, but suggest that using a set PIP may standardise individual therapists' performance in the same patient. Although pattern of ventilation will differ between patients due to varying pathologies, the results of this study suggest that some aspects of operator performance, the T_I and PIFR generated, may also contribute to this variability. Use of PIP alone to standardise the performance of manual hyperinflation between therapists in the same patient requires investigation. In addition, standardisation of

operator performance and documentation of the pattern of ventilation in clinical studies examining the efficacy of manual hyperinflation for preventing or treating volume loss and assisting secretion clearance is required.

Acknowledgements

The authors wish to thank the medical, nursing, physiotherapy staff and patients of Concord Repatriation General Hospital, in particular Mr James Parkin.

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