Randomized Comparison of Two Physiotherapy Regimens for Correcting Atelectasis in Ventilated Pre-term Neonates

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Abstract: Chest therapy using manual physiotherapy techniques is an integral part of treating atelectasis in pre-term ventilated neonates. The lung squeezing technique (LST) is used to restore homogeneous inflation of the lungs by means of small amplitude oscillatory chest wall compressions. We compared the effectiveness and safety of using LST with the conventional percussion and vibration (PDPV) protocol for correcting atelectasis in ventilated neonates. Fifty-six pre-term neonates were randomized into an experimental group (n = 26) treated with the lung squeezing protocol and a control group (n = 30) treated with the conventional percussion and vibration protocol. The groups were pre-stratified according to the mode of ventilation: high frequency oscillatory ventilation (HFOV) or conventional mechanical ventilation (CMV). Results showed that LST was more effective for correcting lung atelectasis in pre-term neonates than PDPV. After the first therapy session, full lung re-expansion occurred in 81% of the LST group and in only 23% of the PDPV group ($p < 0.001$). Subgroup analysis showed the superiority of LST in both the CMV ($p = 0.006$) and HFOV subgroups ($p = 0.006$). There was no significant difference in haemodynamic disturbances when the LST group was compared to the PDPV group. LST was more effective than conventional PDPV for re-expansion of lung atelectases among the ventilated pre-term neonates in our study.

Key words: physiotherapy, lung squeezing technique, neonate, ventilation, atelectasis

Introduction

Chest physiotherapy (CPT) utilizing postural drainage, percussion and vibration (PDPV) is conventionally used as a physical means to enhance removal of airway secretions in neonatal intensive care settings [1]. Despite the widespread clinical impression that CPT helps to correct atelectasis, two clinical trials have shown that CPT was ineffective in preventing post-extubation atelectasis [2, 3]. The incidence of atelectasis was higher in the CPT groups in both of the trials. A study in children receiving mechanical ventilation after cardiac surgery suggested that lung compression from percussion may cause more lung atelectasis by decreasing functional residual capacity [4]. A subsequent animal study also showed that percussion in mechanically ventilated dogs induced lung atelectasis [5]. The suggested mechanism of regional atelectasis was forced oscillation generated by percussion, which caused emptying of the air spaces and apposition of the alveolar walls. A recent infant lung study [6] showed that smaller subjects emptied their lung volumes proportionately faster during rapid thoracic compression. The findings suggest that neonates might have different responses to chest wall compressions due to their small size.

Lung squeezing technique

Although atelectasis might be related to mucostasis in

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the airways, other factors such as surfactant deficiency, closure of the smaller and more collapsible airways, insufficient inspiratory effort and chest cage frailty [7–10] might also contribute to the pathology in neonates. There is evidence that differences in regional resistance and regional lung compliance are related to regional inhomogeneities in ventilation [11, 12]. In ventilated infants presenting with atelectasis, regional overinflation in other ventilated lung units has been reported in both respiratory distress syndrome and bronchopulmonary dysplasia conditions [13]. Based on ^{133}xenon lung scans, Moylan and Shannon also deduced that regional overinflation is a causative, rather than a compensatory, mechanism for lobe collapse [13].

The "lung squeezing" technique (LST) is a form of manual chest wall compression performed on the whole hemithorax. The technique differs from conventional PDPV in the following aspects: each set of "lung squeezes" consists of three or four sustained chest compressions lasting for about 5 seconds, followed by a gentle slow "release phase", with the chest wall being completely released; these compressions are given without vibration and not in a gravity-assisted position; delivery of the chest compressions is not intended to be in synchrony with the infant's breathing pattern.

The purpose of LST is to facilitate emptying of the hyperinflated lung units. This decompression effect might be due to a cephalad bias in airflow and greater peak expiratory airflow when compared to inspiratory airflow [14], and was demonstrated to promote re-expansion of atelectatic lung regions [15]. Our pilot study with 11 pre-term, ventilated neonates showed that LST for 10 minutes improved mean respiratory system compliance by 21% [16]. The improvement might be related to the recruitment of more lung units. The objective of this study was to test the efficacy of LST by comparing its effect with that of conventional PDPV for correcting atelectasis in pre-term neonates requiring mechanical ventilation.

The primary outcome measure was re-expansion of the atelectatic lung as demonstrated on chest radiography. Other outcomes documented were recurrence of atelectasis, duration of ventilation (days) and oxygen dependency (days). The effects of ventilation mode on the efficacy of LST and PDPV were also studied. Disturbances in physiological parameters during CPT, including the pulse rate, blood pressure and oxygen saturation, were also studied. Progression of intra-ventricular haemorrhage (IVH) and incidence of cystic brain lesions were also investigated.

Methods

From August 1995 to January 1998, all neonates with gestational ages of less than 37 weeks who required mechanical ventilation were enrolled into the randomized study if they fulfilled the following criteria: presence of a segmental or lobar collapse confirmed on chest X-ray, and granting of parental consent. All eligible neonates were checked by a neonatologist for the level of the endotracheal tube to ensure that the atelectasis was not related to the position of the endotracheal tube. Neonates with the following disease conditions were excluded from the study: persistent pulmonary hypertension, meconium aspiration syndrome, congenital heart defects, pneumonia presenting with generalized patchy consolidation, post-cardiothoracic surgery, pleural effusion and pneumothorax. During the study period, 64 neonates presented with atelectasis, of whom eight were excluded for the above conditions.

The study was approved by the Ethics Committee for Clinical Research of the Chinese University of Hong Kong. Randomization was by computerized random number allocation in sealed opaque envelopes, which were drawn by an independent person. The eligible neonates were randomly assigned to receive either LST or PDPV. Neonates were stratified by their mode of ventilation: high frequency oscillatory ventilation (HFOV) or conventional mechanical ventilation (CMV).

According to our neonatal intensive care unit (NICU) protocol, chest radiography was performed at least daily on all mechanically ventilated neonates for the first 7 days, and subsequently, whenever clinically indicated, as determined by the attending neonatologists or residents. Upon detection of lung atelectasis on chest radiography, either LST or PDPV was performed by one of the investigators (IW). In his absence, chest therapy was performed by another physiotherapist who had been working in the same neonatal unit for more than 2 years and who had been trained to deliver both LST and PDPV.

Therapy sessions were performed twice daily at specified times. The head of the neonate was stabilized during either chest protocols to minimize the occurrence of encephaloclastic porencephaly. Nurses and other medical staff were unaware of the protocol allocation.

After the first therapy session, chest radiograph (anteroposterior view) was repeated after 2 to 3 hours to evaluate the initial response to therapy. If atelectasis did not resolve, physiotherapy using the same method was repeated after 6 hours, followed by a chest radiograph taken 2 to 3 hours later. Second and third chest radiographs were obtained after subsequent treatments only if resolution did not occur. After resolution of atelectasis, chest radiography was performed whenever clinically indicated, e.g. on deterioration of the respiratory status. Clinical indications included significant increase in fractional inspired oxygen (FIO_2) or ventilator settings, significant increase in frequency or severity of bradycardia and desaturation. Post-therapy chest radiography was routinely performed 3 days after lung re-expansion to

detect any recurrence of atelectasis. The number of therapy sessions to attain full re-expansion of the collapsed area and any incidence of recurrence were noted. Chest therapy protocols for all neonates were unchanged during the 3-day period.

In this study, lung atelectasis and the site involved were confirmed by either of the two neonatologists who were blinded to the study protocol and grouping. The interpretation of atelectasis was based on a combination of signs including volume loss, fissure line displacement and pattern of opacification. Full expansion of atelectasis was defined as the absence of opacification, and homogeneous distribution of ventilation in the affected lobe when compared to other lung regions. Intra- and inter-observer reliabilities were assessed using 15 chest radiographs with evidence of complete or resolved lung atelectasis. The same process was repeated 8 weeks later using the same set of chest radiographs. The coefficient of agreement for neonatologists 1 and 2 were 0.93 and 0.87, respectively. Intra-observer reliability (Kappa statistics) was 83.33% and 59.38%, respectively. Inter-observer reliability was 82.93%.

LST protocol

Each set of "lung squeezes" consisted of three or four sustained chest compressions lasting for about 5 seconds, followed by a gentle slow "release phase", with the chest wall completely released. LST was performed on each side of the hemithoraces, with the neonate in the supine position and in the horizontal position, without head elevation, for 10 minutes, followed by endotracheal suctioning.

PDPV protocol

Percussion and vibration in modified postural drainage positions were performed with the neonate lying alternately on his/her two sides, in the horizontal position without head elevation, for 10 minutes, followed by endotracheal suctioning. The amount of sputum collected in the suction catheter was extracted and measured using a calibrated 1 mL syringe.

Statistical analysis

All data analyses were performed according to the intention-to-treat principle. Data were first tested using the Kolmogorov-Smirnov normality test and the Levene Median test for equal variance. Normally distributed data were expressed as means ± standard deviations (SDs) and analysed using unpaired Student's t tests. Non-normal data were expressed as medians and 25th and 75th percentiles, and were analysed using the Mann-Whitney rank sum test. For all neonates with unresolved atelectasis, the numbers of therapy sessions were ranked in ascending order in all Mann-Whitney rank sum tests. Categorical data were analysed using Chi-squared or Fisher's exact tests, as appropriate. Progression of IVH was analysed using McNemar's test. For all the tests, a p value of 0.05 or less was considered statistically significant. The alpha level was adjusted in all subgroup analyses based on the number of comparisons.

The required sample size was calculated to ensure detection of clinically important changes in the primary outcome. We considered a reduction of two sessions of CPT to be clinically relevant; based on pilot data, in order for this difference to be detected, 52 neonates (26 in each arm) were required to achieve a two-sided alpha level of 0.05 and 80% power.

Results

From August 1995 to January 1998, there were 56 neonates who met the inclusion criteria and who were randomized for treatment. Two neonates, one from each group, were withdrawn from the study due to critical clinical conditions. One required repeated bronchial lavage and the other required nitric oxide therapy. Both treatment arms were similar in their demographic characteristics (Table 1). Distribution of lung atelectasis among various lobes was similar between the two groups ($\chi^2 = 1.972$, degrees of freedom, df = 5, $p = 0.853$); right upper lobe collapse constituted the majority of lobar collapse in both groups (66% in the LST group, 75% in the PDPV group).

Re-expansion of lung atelectasis

After the first session of physiotherapy, resolution of atelectasis was achieved in 81% (21/26) of the LST group, but in only 23% (7/30) of the PDPV group ($\chi^2 = 16.154$, df = 1, $p < 0.001$). The number of therapy sessions required to attain full re-expansion of the atelectatic lung/lobes was also significantly smaller in the LST group than in the PDPV group ($p < 0.001$) (Figure). Analysis of neonates in the CMV or HFOV subgroups showed that in both subgroups, significantly fewer sessions of LST were required to completely re-expand the atelectatic lobes compared to PDPV (CMV, $p = 0.006$; HFOV, $p = 0.006$) (Table 2).

Recurrence of lung atelectasis

The rate of recurrence of lung atelectasis was calculated as the number of subjects with signs of recurrent atelectasis, expressed as a percentage of the total number of subjects in the same group. The rate of recurrence was similar in both groups ($\chi^2 = 0.774$, df = 1, $p = 0.781$): 54% (14/26) and 47% (14/30) in the LST and PDPV groups, respectively. Subgroup analysis showed that in patients undergoing CMV, significantly fewer sessions of LST were required to correct recurrence compared to patients undergoing PDPV (CMV, $p = 0.012$). Nonetheless, for patients undergoing HFOV, the LST and PDPV data did not differ significantly ($p = 0.948$) (Table 2).

Table 1. Demographic data of neonates and ventilation parameters

	LST (n = 26)	PDPV (n = 30)	*p*
Gestational age, wk			
Mean ± SD	27.9 ± 2.9	28.3 ± 3.7	0.656
Range	23.9–36.6	22.7–35.9	
Birth weight, g			
Mean ± SD	1,042 ± 472	1,036 ± 469	0.706
Range	585–2,900	540–2,046	
Recruitment weight, g			
Mean ± SD	1,265 ± 544	1,157 ± 557	0.332
Range	650–2,970	540–2,700	
Lung lobes with atelectasis	29	32	
Antenatal steroid administration	61.5% (16/26)	53.3% (16/30)	0.728
Surfactant administration	65.4% (17/26)	70.0% (21/30)	0.935
Male gender	46.2% (12/26)	66.7% (20/30)	0.202
FIO_2*	0.40 (0.25, 0.50)	0.40 (0.28, 0.60)	0.755
Peak inspiratory pressure (cmH_2O)*	14 (9, 19)	15 (11, 19)	0.828
CMV	11	16	
HFOV	15	14	

*For FIO_2 and peak inspiratory pressure, data are expressed as median (25th percentile, 75th percentile). LST = lung squeezing technique; PDPV = percussion and vibration; FIO_2 = fractional inspired oxygen; CMV = conventional mechanical ventilation; HFOV = high frequency oscillatory ventilation.

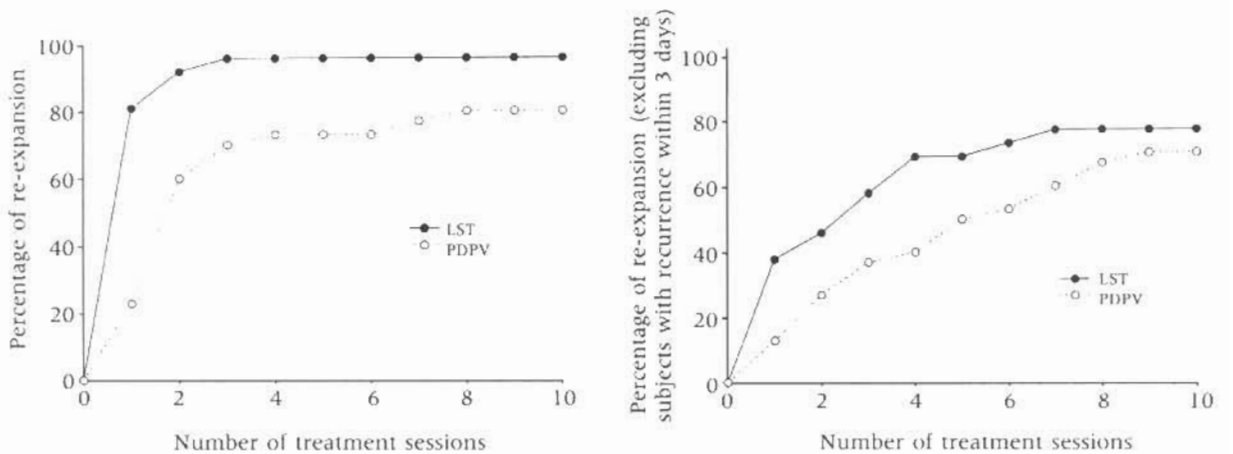

Figure. Resolution of atelectasis. Left panel: cumulative percentage of subjects who attained full re-expansion after chest therapy. Right panel: cumulative percentage of re-expansion excluding subjects with recurrence within 3 days. LST = lung squeezing technique; PDPV = percussion and vibration.

Failure to re-expand atelectasis

The atelectasis failed to resolve despite repeated physiotherapy sessions in two neonates (8%) in the LST group and in four neonates (13%) in the PDPV group ($p = 0.675$). Both neonates in whom LST failed were ventilated using HFOV, while all four patients in whom PDPV failed were ventilated using CMV.

Changes in ventilator parameters

Ventilator parameters immediately before and 6 hours after the first session of intervention were documented. In both groups, there was a decrease in FIO_2 and peak inspiratory pressure after intervention, although the differences between the two groups were not statistically significant (Table 3).

Haemodynamic changes

In both groups during delivery of CPT, heart rate, blood pressure (systolic, diastolic, and mean) and arterial oxygen percent saturation (SaO_2) were continuously monitored. The largest deviation of these parameters from the pre-treatment baseline values were recorded, with exclusion of data obtained during and after endotracheal suction. Within both groups, all parameters

Table 2. Number of treatment sessions to attain full re-expansion of lung atelectasis

	LST	n	PDPV	n	*p*
Number of treatment sessions to attain full re-expansion					
All neonates	1 (1, 1)	26	2 (1, 3)	30	< 0.001
CMV	1 (1, 1)	11	2 (1, 2)	16	0.006
HFOV	1 (1, 1.75)	15	2 (2, 4)	14	0.006
Number of treatment sessions to attain full re-expansion including resolution of recurrence					
All neonates	2.5 (1, 11)	26	5 (2, 8)	30	0.059
CMV	1 (1, 3)	11	3 (2, 6)	16	0.012
HFOV	4 (1, 11.25)	15	6.5 (3, 11)	14	0.948

Data are expressed as median (25th percentile, 75th percentile). LST = lung squeezing technique; PDPV = percussion and vibration; CMV = conventional mechanical ventilation; HFOV = high frequency oscillatory ventilation.

except for SaO_2 were significantly different from their respective pre-treatment values. The changes between the two groups were not significantly different (Table 3).

Presence of pulmonary secretions

Secretions extracted immediately after CPT were defined as obvious when the amount of sputum collected was more than 0.2 mL. Presence of secretion did not differ between the two groups: LST, 42% (11/26); PDPV, 40% (12/30), $p = 0.923$. Less than 0.2 mL of secretions was collected from 59% of the subjects.

Other clinical outcomes

Duration of ventilation, duration of oxygen dependency and the occurrence of bronchopulmonary dysplasia (BPD) were compared between the two groups. The mean duration of mechanical ventilation was shorter in the PDPV group (23 ± 15 days) than in the LST group (31 ± 28 days) ($p = 0.241$). Nonetheless, the duration of oxygen dependency was shorter in the LST group (median, 25; 25th percentile, 9; 75th percentile, 63) than in the PDPV group (median, 34; 25th percentile, 11; 75th percentile, 42) ($p = 0.815$). The occurrence of BPD did not differ between the two groups (LST, 50%; PDPV, 43%; df = 1; $p = 0.818$). The mortality rate was similar between the two groups (LST, 31%; PDPV, 43%; $p = 0.489$).

Intraventricular haemorrhage

Progression of IVH was documented by serial cranial ultrasound performed for 3 days after the first CPT session. IVH in five neonates (19%) in the LST group progressed from grade 0–2 to grade 3–4. Seven neonates (23%) in the PDPV group presented with a similar pattern. Two infants, one from each group, progressed from normal to grade 3–4 IVH. After excluding the subjects with pre-therapy high-grade IVH, the observation in the PDPV group was not significantly different from that in the LST group (Fisher's exact test, $p = 0.73$). There was no significant difference in the occurrence of multiple cystic brain lesions between the two groups (LST, 12%; PDPV, 13%; $p = 1.00$). The demographic characteristics of the neonates presenting with cystic brain lesions are presented in Table 4.

Table 3. Failure rate, changes in ventilation parameters 6 hours after treatment, maximum deviation from pre-treatment haemodynamic parameters during therapy

	LST (n = 26)	PDPV (n = 30)	*p*
Failure to re-expand atelectasis	2	4	0.675
Change in FIO_2	−0.01 (−0.05, 0)	0 (−0.1, 0)	0.980
Change in PIP (cmH_2O)	−0.2 (−1.5, 1)	0 (−0.3, 0)	0.845
Heart rate (beat/min)	9 (5, 15)	14 (6, 22)	0.079
SBP	8 (5, 14)	8 (6, 15)	0.685
DBP	5 (3, 11)	6 (4, 11)	0.596
MBP	7 (4, 12)	6 (5, 14)	0.755
SaO_2	0 (−1, 2)	0 (0, 0)	0.207

Data are expressed as median (25th percentile, 75th percentile). LST = lung squeezing technique; PDPV = percussion and vibration; FIO_2 = fractional inspired oxygen; PIP = peak inspiratory pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; MBP = mean blood pressure; SaO_2 = arterial oxygen percent saturation.

Table 4. Demographic characteristics of neonates presenting with multiple cystic brain lesions

Subject	Gestational age, wk	Body weight, g	Outcome	Nature of lesion	IVH grade	Diagnosed after initial treatment, d
LST						
1	24.4	680	Died	Left subependymal cyst	2	2
2	29.4	1,340	Survived	Left subependymal cyst	3	30
3	25.7	690	Died	Multiple cystic areas	4	9
PDPV						
4	27	1,150	Died	Cystic changes	2	64
5	23.9	550	Died	Periventricular cyst	3	22
6	26.9	910	Survived	Cystic changes	3	2
7	26.4	700	Survived	Left subependymal cyst	4	13

IVH = intraventricular haemorrhage; LST = lung squeezing technique; PDPV = percussion and vibration.

Discussion

Conventional PDPV has been used for the removal of retained airway secretions by augmenting the effect of gravity on bronchial clearance, with the neonate in postural drainage positions. It is assumed that accumulated secretions and airway obstruction is the primary cause of atelectasis. However, recent studies did not demonstrate the short-term benefits of PDPV in post-extubation atelectasis [2, 3]. This result is in contrast to the results of an earlier study that included subjects presenting with mucus retention such as meconium aspiration and bacterial pneumonia [17]. If the relative contribution of retained mucus to the development of atelectasis was lesser in neonates, PDPV may not show its benefit.

In our literature search, many studies on the efficacy of the PDPV protocol for ventilated infants [18–20] were conducted more than 1 decade ago. Because there has been much advancement in the management of neonates throughout the 1990s, resulting in improved survival of 500–800 g infants, as well as in infants born at 26 weeks of gestation or less [21], our study provides new evidence regarding the treatment effects of PDPV and LST for this patient population.

HFOV was introduced as a new mode of neonatal respiratory management; the near-constant distending pressure generated by high-frequency oscillations has been used to optimize alveolar re-expansion and to promote uniform lung inflation [22]. We chose the mode of ventilation as an important prognostic factor. Pre-stratification was performed to investigate how chest therapy interacted with the mode of ventilation.

In neonates with uneven ventilation distribution, hyperinflated lung units may compress adjacent lung units and predispose them to atelectasis. Conversely, manoeuvres that decompress these hyperinflated lung units might facilitate the resolution of atelectasis. Theoretically, chest wall compressions by either percussion or LST would produce a similar decompression effect on hyperinflated lung units; however, this benefit was not observed in previous trials utilizing PDPV. It is possible that the brief and forced oscillations of percussion on the chest wall promoted the emptying of lung units with low time constants (such as small collapsible airways), therefore causing more atelectasis. Apparently, LST decompresses the slowly emptying, hyperinflated lung units, which facilitates lung re-expansion.

In our trial, LST or PDPV was delivered in the horizontal position. One study showed that the prone, head elevated tilt position reduced hypoxaemic and bradycardic events in pre-term infants, which might be attributed to perfusion and ventilation distribution in the lungs [23]. Supine, head elevated tilt positions also improved oxygenation, which was not related to diaphragm activity [24]. Because there were no available data on the effect of head elevated tilt position in neonates lying on their sides, we chose a horizontal position in both protocols to eliminate this effect.

This assessor-blinded randomized trial evaluated the effectiveness of the two chest physiotherapy techniques used in treating atelectasis in mechanically ventilated neonates. With either technique, re-expansion of the atelectatic site was achieved within a short time. After the first therapy session, significantly more neonates responded to LST than to PDPV. The superiority of LST was demonstrated in neonates receiving either CMV or HFOV. By the completion of two therapy sessions, the majority of the neonates (92%) had responded to LST while only 60% had responded to PDPV. Thus, LST was more effective in correcting atelectasis than PDPV in our neonates. Nearly 60% of our subjects produced less than 0.2 mL of secretions during treatment, suggesting that uneven distribution of ventilation, rather than sputum

retention, was the major cause of atelectasis in our neonates. Therefore, resolution of atelectasis was better in neonates receiving LST. The finding that LST is more effective in treating atelectasis is of particular importance because LST may be used to treat signs of uneven distribution ventilation and prevent the development of atelectasis.

The presence of secretion was seen in similar percentages of infants in both groups. LST had a similar effect on bronchial clearance, which may be a secondary effect of the cephalad bias in airflow generated during LST.

With either protocol, about half of the re-expanded lobes had recurrence of alveolar collapse within 3 days. This high recurrence rate may be related to the paucity of collateral ventilation and to the alveolar instability in neonates [25], or the slow reversal of the intrinsic factors leading to ventilation heterogeneity. From subgroup analysis, LST was effective in resolving recurrence of alveolar collapse in neonates on CMV, but this was not seen in neonates on HFOV. We hypothesize that this may be due to confounding factors associated with HFOV, which contribute to the instability of alveolar expansion. This requires further investigation.

Secondary outcomes and adverse effects

The potential adverse effects of CPT were also studied. All the neonates demonstrated haemodynamic changes, including increased heart rates, systolic, diastolic and mean blood pressures, after LST or PDPV. The changes in the two groups were similar. There was no difference in short-term benefits for both groups in terms of duration of mechanical ventilation or oxygen dependency. The occurrence of BPD and mortality were also similar between the two groups.

Although this was not a primary aim of our clinical trial, we are most concerned about the potential association of physiotherapy with intracranial lesions. There was a considerable number of neonates whose IVHs progressed from grade 0–2 to grade 3–4 in both the LST group (19%) and the PDPV group (23%). This observation should be interpreted with caution by, firstly, understanding the known contributing factors to IVH. The pathogenesis of IVH and cystic brain lesions is multifactorial, with intravascular, vascular and extravascular contributions, and the risk of IVH is inversely related to gestational age and birth weight [26]. Early sepsis and failure to give antenatal steroid treatment are also associated with the development of high grade IVH [21]. In our trial, although we observed some infants who progressed to high grade IVH, more importantly, there were only two infants from among the non-IVH infants (2/26, 1 from LST group and 1 from PDPV group) who progressed from normal to high grade IVH after the commencement of CPT. This suggests that IVH progression was possibly due to other underlying pathology instead of being related to CPT. One recent study also showed that CPT was not associated with any abnormal neurological outcomes, and the occurrence of cystic brain injuries was similar in both physiotherapy and non-physiotherapy groups [27]. In our trial, there was no significant difference in the occurrence of cystic brain lesions between the LST and PDPV groups, and no incidence of encephaloclastic porencephaly, as defined by Harding et al [28], was identified.

Conclusions

In this trial, we studied the efficacy of two chest physiotherapy protocols for treating atelectasis in pre-term neonates requiring mechanical ventilation. LST was a useful alternative for correcting atelectasis, without any additional adverse effects when compared to conventional PDPV. To our knowledge, this is the first randomized trial evaluating the effects of different therapy techniques on pre-term neonates requiring mechanical ventilation, including HFOV. Although some studies show that post-extubation physiotherapy does not result in any additional benefit, we demonstrated that physiotherapy is useful in resolving atelectasis in pre-term neonates under mechanical ventilation.

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