

Prolonged Slow Expiration Technique in Infants: Effects on Tidal Volume, Peak Expiratory Flow, and Expiratory Reserve Volume

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BACKGROUND: Prolonged slow expiration (PSE) is a physiotherapy technique often applied in infants to reduce pulmonary obstruction and clear secretions, but there have been few studies of PSE's effects on the respiratory system. **OBJECTIVE:** To describe PSE's effects on respiratory mechanics in infants. **METHODS:** We conducted a cross-sectional study with 18 infants who had histories of recurrent wheezing. The infants were sedated for lung-function testing, which was followed by PSE. The PSE consisted of 3 sequences of prolonged manual thoraco-abdominal compressions during the expiratory phase. We measured peak expiratory flow (PEF), tidal volume (V_T), and the frequency of sighs during and immediately after PSE. We described the exhaled volume during PSE as a fraction of expiratory reserve volume (%ERV). We quantified ERV with the raised-volume rapid-thoracic-compression technique. **RESULTS:** The cohort's mean age was 32.2 weeks, and they had an average of 4.8 previous wheezing episodes. During PSE there was significant V_T reduction (80 ± 17 mL vs 49 ± 11 mL, $P < .001$), no significant change in PEF (149 ± 32 mL/s vs 150 ± 32 mL/s, $P = .54$), and more frequent sighs (40% vs 5%, $P = .03$), compared to immediately after PSE. The exhaled volume increased in each PSE sequence ($32 \pm 18\%$ of ERV, $41 \pm 24\%$ of ERV, and $53 \pm 20\%$ of ERV, $P = .03$). **CONCLUSIONS:** It was possible to confirm and quantify that PSE deflates the lung to ERV. PSE caused no changes in PEF, induced sigh breaths, and decreased V_T , which is probably the main mechanical feature for mucus clearance. *Key words:* prolonged slow expiration technique; physiotherapy; infant; pulmonary function test; expiratory reserve volume. [Respir Care 2011;56(12):1930–1935. © 2011 Daedalus Enterprises]

Introduction

Chest physiotherapy is commonly employed in the treatment of infants with respiratory diseases.¹ Almost all physiotherapy techniques available for infants are derived from adult studies,²⁻⁵ but the infant respiratory system is differ-

ent from the adult respiratory system, and the effects of chest physiotherapy may not be the same.^{6,7} New chest physiotherapy techniques were developed specifically for infants, in accordance with their physiological characteristics.^{8,9} Prolonged slow expiration (PSE) is one of these new techniques, employed in clinical practice in infants with bronchial obstruction and hypersecretion.¹⁰ In PSE, pressure is exerted on the thorax and abdomen to prolong the expiratory phase and thus promote secretion clearance.^{10,11}

In infants with viral bronchiolitis, PSE improves respiratory distress, lowers heart rate, and increases S_{pO_2} .¹⁰⁻¹³ In 2001, some possible benefits of PSE were described, including improved secretion clearance and reduced hyperinflation.¹² In 2006, Postiaux and co-workers found reduced respiratory distress, lower heart rate, and increased S_{pO_2} after PSE in 19 infants with viral bronchiolitis.¹³

Although PSE is used in several countries, mainly in Europe, in the 20 years since the first description of PSE

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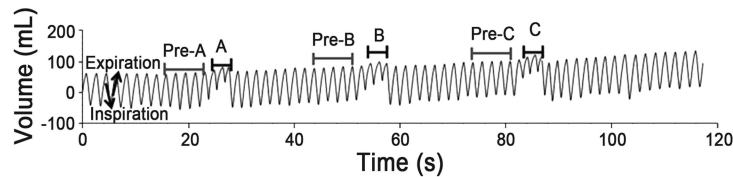


Fig. 1. Example of a volume-time curve during normal breathing and prolonged slow expiration. Pre-A, Pre-B, and Pre-C are the 5 normal breaths before the PSE sequences (A, B, and C).

there has not been a detailed description of PSE in the literature. It is assumed that PSE deflates the lung to expiratory reserve volume (ERV), induces sigh breaths, and does not increase peak expiratory flow (PEF), because it is a slow technique,^{9,12,13} but these assumptions are unproven, and PSE's effects on infant respiratory mechanics are unknown.

Pulmonary function testing in infants allows assessment of pulmonary volumes and capacities, thereby assisting in the determination of disease severity, treatment response, and physiologic changes induced by sigh breaths.¹⁴⁻¹⁶ This assessment may help physical therapists determine changes in pulmonary function during respiratory therapy techniques such as PSE. In infants with histories of wheezing, we studied PSE's effects on tidal volume (V_T), ERV, PEF, and induction of sigh breaths.

Methods

This study was approved by the ethics committee of Federal University of São Paulo, São Paulo, Brazil, and performed in the pediatrics department. All the patients' parents gave written, informed consent.

Patients

We screened all infants referred for pulmonary function testing, and invited full-term babies who had histories of recurrent wheezing (more than 3 wheezing episodes) but no acute respiratory symptoms in the previous 2 weeks. We excluded infants with upper-airway obstruction, symptoms or history of neuropathy, gastroesophageal reflux disease, or thoracic and/or abdominal surgery.

Prior to the pulmonary function testing, the infants had been fasting for a minimum of 3 hours. We administered chloral hydrate (60–80 mg/kg), per our laboratory routine and the recommendations of the American Thoracic Society/European Respiratory Society.¹⁷ During the lung function test and the PSE protocol we continuously monitored S_{pO_2} and heart rate (DX2405, Dixtal, São Paulo, Brazil). The infants were in dorsal decubitus position, with a slight cervical extension created with a small cushion in the scapula region, with no lateralization of the head. We attached a face mask coupled to a pneumotachograph (Hans

Rudolph, Shawnee, Kansas) to the infant's face, and the mask was sealed on the baby's face with a mass to seal the surface and prevent air leakage. The system and face mask's dead space was known and accounted for by the equipment's software. We continuously recorded flow and volume. Each patient had just one set of data collected for analysis. Prior to analyzing the data we checked for the presence of drift in the volume-time curve, from high humidity or temperature.

Prolonged Slow Expiration Protocol

We first recorded 60 seconds of normal breathing, then conducted the PSE protocol. All PSE procedures were performed by the same therapist (FCL). The therapist positioned the hypothenar region of one hand on the thorax, precisely below the suprasternal notch, and the hypothenar region of the other hand on the abdomen, under the umbilical scar. The therapist visually identified the inspiratory and expiratory phases by observing the thorax movement, and at the end of the expiratory phase applied compression with both hands. The hand on the thorax moved in the cranial-caudal direction while the hand on the abdomen move in the caudal-cranial direction. The subsequent 3 inspirations were restricted, and the compression movements were continued into the expiratory phase, per the standard PSE technique.^{13,18} This procedure was repeated 3 times (sequences A, B, and C), with an interval of 30 seconds between each sequence (Fig. 1).

During both normal breathing and PSE we measured PEF, V_T , and expiration time (T_E), and calculated the mean for each sequence (see Fig. 1). We measured the expiratory and inspiratory volume during each PSE manual compression, and we calculated the exhaled V_T during PSE as the difference between them and expressed the value as a percent of ERV (%ERV).

Sigh breaths were identified on the flow-volume and volume-time curves, as a V_T increase of more than 100%. We considered a sigh breath induced by PSE only if it occurred during or just after PSE. We compared the frequency of PSE-induced sigh breath to that during the 60 seconds of normal breathing before PSE.

After the PSE protocol we measured ERV with the raised-volume rapid-thoracic-compression technique, with an in-

fant pulmonary function testing system (Infant Pulmonary Lab, nSpire Health, Longmont, Colorado) as recommended.¹⁵ An inflatable jacket is wrapped around the infant's chest and abdomen, and several sequential lung inflations (inspiratory pressure 30 cm H₂O) are delivered to inhibit respiratory effort prior to the thoracic compression. The forced maneuvers are performed by the automated inflation of the jacket, which compresses the chest and maintains expiratory flow until it approaches zero, or for a maximum of 4 seconds. Then we recorded 40 seconds of normal breathing during which we measured the V_T. We calculated ERV as the mean of the difference between the total exhaled V_T after thoracic compression and the normal V_T, in at least 3 acceptable expiratory curves.¹⁵

Statistical Analysis

The sample size calculation to achieve an alpha error of .05 and 90% power indicated a minimum sample of 14 infants. This was based on a 30% V_T difference between the normal breathing sequence and the PSE sequence observed in a pilot study. All the continuous variables showed normal distribution and are expressed as mean ± SD.

We considered for analysis the mean values from each PSE sequence (A, B, and C) and each normal-breathing sequence (the 5 respirations prior to each PSE sequence), so we used repeated-measures analysis of variance to compare V_T, PEF, and T_E between PSE A, PSE B, and PSE C, and to compare V_T, PEF, and T_E between normal-breathing sequence A, normal-breathing sequence B, and normal-breathing sequence C.

We also used repeated-measures analysis of variance to compare %ERV between PSE A, PSE B, and PSE C, and to compare %ERV within the 3 inspirations in the sequences. For differences with P ≤ .05 we used a post hoc Bonferroni test.

We compared the number of PSE-induced sigh breaths with the McNemar chi-square test. We calculated the Pearson coefficient to analyze the correlation between %ERV and age, between %ERV and the number of wheezing episodes, between age and sigh breaths, and between the number of wheezing episodes and sigh breaths. We used the paired t test to compare the V_T, PEF, and T_E values from the normal breathing sequences and the PSE sequences. The level of rejection of the null hypothesis was set at 5%. All analyses were made with statistics software (SPSS 13.0, SPSS, Chicago, Illinois).

Results

Twenty-two infants were included. Four did not complete the study: one due to cough during the examination, and 3 due to technical difficulties (premature awakening

Table 1. Subjects*

Female/male	9/9
Age (weeks)	32.2 ± 11.4
Length (cm)	68 ± 4
Weight (kg)	8.3 ± 1.0
Wheezing episodes, no.	4.8 ± 1.9
Tidal volume (mL/kg)	9.3 ± 1.2
Tidal volume (% of predicted)	116 ± 19
Respiratory rate (breaths/min)	40 ± 7

* All values except female/male are mean ± SD.

Table 2. Lung Function Variables During Prolonged Slow Expiration Technique*

	Prolonged Slow Expiration Sequence			P
	A	B	C	
Peak expiratory flow (mL/s)	164 ± 45	147 ± 52	147 ± 47	.08
Tidal volume (mL)	49 ± 14	43 ± 18	47 ± 16	.10
Expiratory time (s)	0.5 ± 0.2	0.5 ± 0.1	0.5 ± 0.1	.75

* Values are mean ± SD.

Table 3. Lung Function Variables During Normal Breathing*

	Normal Breathing Sequence			P
	A	B	C	
Peak expiratory flow (mL/s)	149 ± 32	150 ± 32	154 ± 33	.54
Tidal volume (mL)	80 ± 18	83 ± 17	83 ± 17	.58
Expiratory time (s)	0.8 ± 0.1	0.8 ± 0.2	0.8 ± 0.3	.60

* Values are mean ± SD.

and insufficient sedation). Thus, we analyzed the data from 18 infants (Table 1).

The duration of the PSE protocol ranged from 8 to 10 min. During the protocol, none of the infants exhibited signs of respiratory distress, expiratory grunting, or wheezing. During the protocol, in all the infants, S_{pO₂} remained above 93%, and the heart-rate range was 110–150 beats/min.

The mean ± SD V_T was 116 ± 19% of predicted.¹⁵ PSE flattened the inspiratory and expiratory flow-volume curves. There were no statistically significant changes in PEF, V_T, or T_E between PSE sequences A, B, and C (PEF P = .08, V_T P = .10, T_E P = .75, Table 2) or between the normal breathing sequences (PEF P = .54, V_T P = .58, T_E P = .60, Table 3). Comparing the normal breathing sequences and PSE sequences, there was a mean V_T reduction of more than 40% (P < .001, Fig. 2) and a consequent T_E reduction (P < .001).

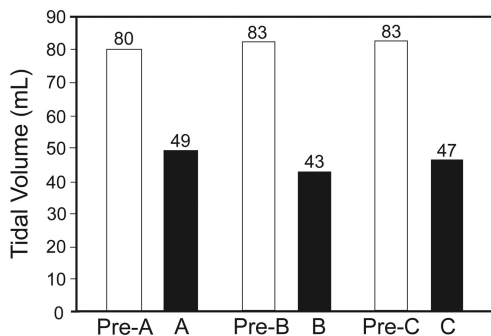


Fig. 2. Tidal volume before (Pre-A, Pre-B, and Pre-C) and during 3 prolonged slow expiration sequences (A, B, and C). $P < .001$ for all 3 differences.

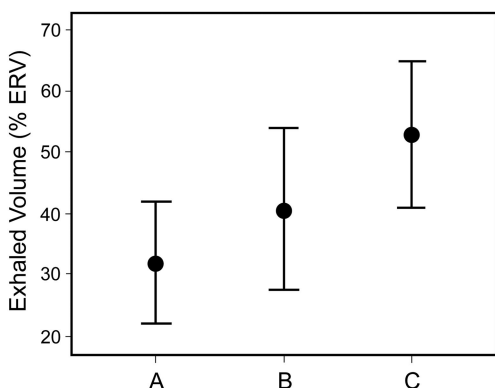


Fig. 3. Exhaled volume in 3 consecutive sequences (A, B, and C) of prolonged slow expiration technique, as a percentage of expiratory reserve volume (%ERV). $P = .04$ for A versus B. $P < .001$ for B versus C. $P < .001$ for A versus C.

PSE-induced sighs were observed in 7 infants significantly more frequently than those observed during normal breathing (1 infant) ($P = .03$). Each patient sighed only once. In patients who sighed during PSE, the mean \pm SD V_T increased from 73 ± 12 mL to 204 ± 69 mL, compared to V_T without versus with sigh breath ($P < .001$) or 9 ± 1 mL/kg to 26 ± 8 mL/kg ($P < .001$). The only patient who sighed during normal breathing increased V_T from 93 mL (11 mL/kg) to 200 mL (23 mL/kg) during the sigh breath. There was no significant correlation between age and the presence of sigh breaths ($r = -0.27$, $P = .30$) or between the number of wheezing episodes and induction of sigh breaths ($r = -0.32$, $P = .20$).

Three of the 18 patients awoke before raised-volume rapid-thoracic-compression, so ERV could be determined in only 15 infants. There was an increase in exhaled volume during the PSE period, in comparison to normal tidal breathing. Progressive exhaled volume quantified as a fraction of ERV was observed in each sequence: the mean

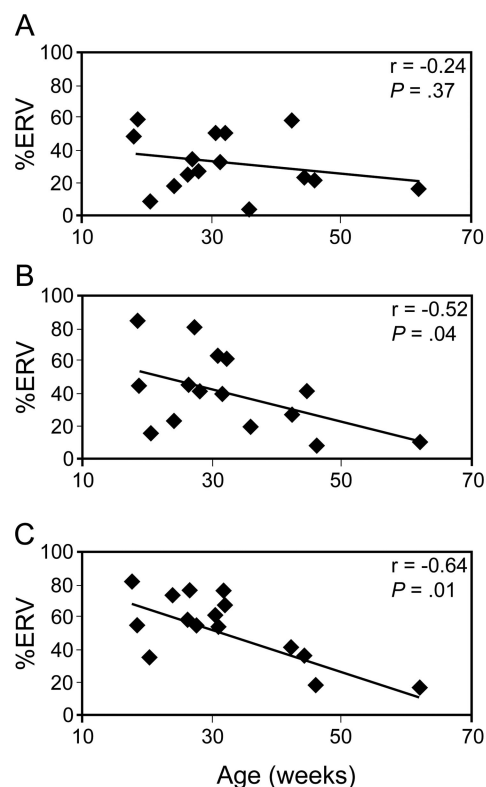


Fig. 4. Correlation between age and exhaled volume (expressed as a percentage of expiratory reserve volume [%ERV]) in 3 consecutive sequences (A, B, and C) of prolonged slow expiration.

%ERV was $32 \pm 18\%$ in sequence A, $41 \pm 24\%$ in sequence B, and $53 \pm 20\%$ in sequence C (Fig. 3).

There was a negative correlation between age and %ERV in sequences B and C ($r = -0.52$ and $r = -0.64$, respectively, Fig. 4). There was no significant correlation between the number of wheezing episodes and %ERV ($r = -0.09$, $P = .60$) in any sequences.

There were no significant differences in the variability of exhaled volumes in the PSE sequences. Sequence A varied 17 ± 10 mL, sequence B varied 21 ± 12 mL, and sequence C varied 20 ± 13 mL (all $P > .05$).

Discussion

The benefit of conventional chest physiotherapy in infants remains controversial.¹⁹⁻²² Disagreements stem from the use of techniques developed for adults, which do not take into consideration the particularities of the infant respiratory system. New chest physiotherapy techniques especially developed for infants have been employed in recent years.²³ The existing studies of PSE suggest benefits in infants with hypersecretion or hyperinflation.^{12,13} But a detailed description of PSE's effect on lung volumes and mechanics has been lacking, and is needed as a reference for further PSE studies and improvements.

The present study demonstrated previously unproven V_T , PEF, and %ERV effects of PSE in infants with histories of wheezing. PSE deflated the lung to ERV and induced sigh breaths. Our first observation about the flow-volume curve during PSE was the expressive variation of volume. The V_T reduction during PSE was approximately 40% of the baseline volume. This can be explained by the thoracic restriction during the inspiratory phase, which limits the inspired volume in order to prolong the expiratory phase. We did not evaluate mucus clearance, but previous studies found that PSE improved mucus clearance.^{18,19,24} Our supposition is that mucus clearance may be improved by the large variations in V_T , but this theory needs study.

The reduction in lung volume is physiologically associated with the protective reflex of the airways, which restores lung volume by sigh breathing (Hering-Breuer deflation reflex).²⁵ In the present study, 7 infants sighed during PSE. We concluded that the prolonged expiratory phase during PSE exhales a substantial portion of the lung volume and thus evokes the Hering-Breuer reflex, indicated by sigh breaths. The Hering-Breuer reflex is more easily induced in younger infants because of the immaturity of their pulmonary receptors, whereas infants with chronic respiratory diseases are less prone to the Hering-Breuer reflex, due to pulmonary impairment.^{26,27} In the present study, however, the induction of sigh breaths was not correlated with age or severity of respiratory disease, expressed as number of wheezing episodes. The small age range and the clinical stability of the studied infants, however, do not allow definite conclusions.

It has been presumed that PSE can evoke sigh breaths, but it has not been objectively determined until the present study. Sighing may benefit these patients by improving alveolar ventilation and lung volume. We previously found that sighs can improve forced vital capacity.¹⁶

After 3 PSE sequences, an average of 53% of the ERV was exhaled, and there was a 20–25% increase in exhaled volume after each sequence. This effect was successively greater if more consecutive PSE sequences were applied. An excessive increase in exhaled volume could reduce S_{pO_2} and lead to respiratory distress caused by bronchial collapse, which may occur when residual volume is reached. Thus, the experience and training of the physiotherapist are essential, so that PSE is applied without placing the infant at risk. After 3 PSE sequences we observed a mean exhalation of 52% of ERV, which is certainly a safe percentage of the lung volume.

We know of no previous studies that calculated %ERV in spontaneously breathing infants during PSE. Studies in adults with chronic pulmonary disease have described a reduction in final expiratory volume after physiotherapy techniques that actively promote a prolonged expiratory phase, as does PSE.^{28,29}

We observed a statistically significant inverse correlation between age and %ERV. There was a greater exhaled volume among the younger infants, and this may be explained by the greater compliance of the thoracic cage in these infants,³⁰ which suggests that PSE may be more effective in younger infants. Fewer sequences of manual compression may be needed in these younger infants. The physiotherapist must be alert for the risk of respiratory distress if many PSE sequences are applied consecutively.

The fact that there was no significant correlation between %ERV and the number of wheezing episodes indicates that the effectiveness of the exhalation volume was not associated with the severity of the illness in this group. However, further studies are needed to corroborate this finding in different diseases and in different severities.

PSE was described as a slow technique that does not increase airway flow.¹⁸ We confirmed this hypothesis in our study. Postiaux and collaborators assessed PSE in infants with respiratory illness, by monitoring flow curves, and also found no change in expiratory flow.²⁴ Rapid-compression physiotherapy techniques are not indicated in infants because of thorax instability.^{1,31}

Manual physiotherapy techniques are always dependent on the ability and practice of the physical therapist, and are susceptible to practice differences and variation.^{32,33} In the present study all the PSE sequences were applied by the same physiotherapist, and we found no important practice variation in the PSE sequences. Research is needed on PSE practice variation between compressions and between physiotherapists. A study of other physiotherapy techniques in adults found minimal variability when manual chest physiotherapy was performed by a trained physiotherapist.³⁴

We did not observe any wheezing exacerbations during the PSE protocol. We chose infants with histories of wheezing so as to study patients similar to those on whom PSE is usually performed. Unfortunately, safety reasons limit the feasibility of sedation and lung function test in infants with acute wheezing or respiratory distress. Chloral hydrate at doses of 60–80 mg/kg is described as safe and with minimal alterations in gasometrical and pulmonary variables such as respiratory-system compliance, respiratory drive, and lung volumes.^{17,35-37} In a sedated infant, PSE could minimally interfere with respiratory variables, and during clinical practice it is not rare to treat sedated patients. We believe that the chloral hydrate did not affect these variables during our study.

Conclusions

Our results confirm previous assumptions about PSE in infants. V_T was reduced during PSE. PEF was maintained as expected in slow techniques. PSE promoted sigh breaths, thereby demonstrating that the change in volume caused by PSE stimulates the Hering-Breuer deflation reflex. An-

other important finding was that PSE deflates the lung to ERV. Now we know that it is possible to reach the fraction of ERV with PSE. Thus, %ERV exhaled is greater, increasing the number of successive PSE sequences (cumulative effect). Age was negatively correlated with the %ERV changes, so we must be careful with younger infants, whereas the severity of the illness did not affect %ERV. We found no important PSE technique variability in the one physiotherapist who performed all the PSE sequences.

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