



ORIGINAL ARTICLE

Decrease of the occurrence of pneumothorax in newborns with respiratory distress syndrome through reduction of ventilatory parameters

Marisa A.A. Brunherotti, Jacqueline R. Freitas Vianna, Carmem S.T. Silveira

Abstract

Objective: to verify if the strategy of reduction of the ventilatory parameters decreases the occurrence of pneumothorax in preterm newborns with respiratory distress syndrome.

Methods: a longitudinal prospective study of preterm newborns with respiratory distress syndrome was carried out at the Pediatric Intensive Care Center of Hospital Santa Casa de Misericórdia of Franca, from July 1999 to June 2000. One hundred and twenty-seven female and male newborns between the 25th and the 37th week of gestation, with birthweight varying from 625 g to 2,500 g were studied. The ventilatory parameters were analyzed during the assisted mechanical ventilation in two groups of patients: the controlled group and the other group, in which the parameters were reduced, by investigating and comparing the occurrence of pneumothorax. The association of data was verified through chi-square test; level of significance $\alpha = 0.05$ and 0.01, and the difference among the studied parameters was obtained through the Student's t test.

Results: the results show lower parameters of inhalation flow and shorter period of inhalation, generating minor tidal volume, decrease of the occurrence of pneumothorax (8.1% compared to 24.5% of the controlled group), with statistical significance ($\chi^2 = 6.545$; $p < 0.05$), and without significance related to mortality and hospital discharge ($\chi^2 = 0.736$; $p = 0.391$).

Conclusions: this study verified that lower inhalation flow and shorter period of inhalation in preterm newborns with respiratory distress syndrome were associated with the decrease of the occurrence of pneumothorax.

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1. Physical therapist, specialist in Lung Functionality and Psychosomatics. Professor, Universidade de Franca - São Paulo.
 2. Physical therapist, specialist in Lung Functionality. Professor, Universidade de Franca - São Paulo.
 3. Physician, specialist in Pediatrics, Neonatology and Pediatric Intensive Care.

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Introduction

The respiratory system of newborns (NBs) presents a few peculiarities that make them more susceptible to the need of ventilatory support.¹ A few anatomical and physiological factors, such as small airways, cylindrical thorax, horizontal ribs, a large amount of type IIa muscle

fibers, predominance of REM sleep and central nervous system (CNS) immaturity, provide a certain inefficiency in respiratory mechanics, favoring thoracic distortion with a greater incidence of respiratory muscle fatigue and pulmonary collapse, making NBs more susceptible to the need of mechanical ventilation.²⁻⁶

Given the great incidence of complications with mechanical ventilation, we decided to study the therapeutic strategy to minimize potential risks such as pneumothorax (PTX).

The main purpose of mechanical ventilation in NBs with respiratory distress syndrome (RDS) was to maintain blood gases within normal levels by using high tidal volumes with consequent high peak inspiration pressures, which increases the risk of pneumothorax.⁷ These high peak pressures lead to mechanical ventilation-induced lung injury, which is characterized by damage to the lung parenchyma, thus worsening the tissue injury caused by RDS, and predisposing to pneumothorax.⁸

This preexisting injury to the alveolocapillary membrane caused by RDS, which also occurs in mechanically-ventilated patients, is best defined as mechanical ventilation-associated lung injury.⁹

Clinical studies have verified different lung protective strategies through the reduction of ventilatory parameters such as the decrease of tidal volume and of peak inspiratory pressure (PIP). A few of these studies suggest beneficial effects such as the reduction of parenchymatous lesion and mortality.^{10,11}

Due to the difficulty in determining alveolar pressures in clinical models, airway pressures have been used as risk indicators for the occurrence of extra-alveolar air leak.¹²

Nevertheless, there are a few limitations to the use of proximal airway pressures to determine the risk for barotrauma; these proximal pressures may not reflect the pressures reached at the alveolar level, which are affected by changes in resistance and flow.¹³ Other factors, such as excessive variations in pulmonary volumes, with consequent alveolar overexpansion, could also be a primary determinant of iatrogenic lung injury denominated volutrauma.¹⁴

Considering this difficulty in establishing ideal parameters during mechanical ventilatory support, the purpose of this study is to develop a strategy for the reduction of ventilatory parameters in order to decrease the incidence of PTX in NBs with RDS at the Intensive Care Unit of the Santa Casa de Misericórdia Hospital in Franca.

Patients and methods

A longitudinal comparative study was carried out with 127 preterm NBs (<37 weeks, WHO,1974); 52 patients were female (41%) and 75, male (59%). The birthweight of these preterm newborns varied between 625g and 2,500g,

and their gestational age varied between 25 weeks and 37 weeks, according to Capurro's method.¹⁵ The study was approved by the Ethics Committee of Santa Casa de Misericórdia Hospital in Franca.

The preterm newborns of our study were diagnosed with RDS according to clinical, radiological and gasometric criteria. The clinical criteria were increase in respiratory frequency, persistent cyanosis, Silverman-Anderson score equal to or greater than 7, X-ray with a diffuse reticulogranular pattern in both lungs, with air bronchograms of variable intensity, and gasometric values with $\text{PaO}_2 < 50\text{mmHg}$, $\text{PaCO}_2 > 60\text{mmHg}$ and $\text{pH} < 7.25$.¹

The inclusion criteria were newborn with less than 37 weeks gestational age and weight lower than 2,500g. We excluded newborns with more than 37 weeks of gestation who weighed more than 2,500g and had anomalies.

We used Intermed and Schrist continuous flow, time-cycled, pressure-limited, microprocessor-controlled ventilators with independent electronic circuits for alarm and pressure monitoring. The parameters were changed based on newborns' clinical alterations. These alterations happened at random and on a personal basis on each shift.

The NBs were studied in two controlled cohorts from their first days of life. Group A (historical group) included 53 patients analyzed from June to December 1999, while group B (strategy for the reduction of ventilatory parameters) consisted of 74 patients analyzed from January to June 2000. The analyses were carried out by means of data collection; the following items were registered on a control chart: name, date of birth, hospital admission, gender, type of birth, weight, ventilator parameters, complementary exams, use of surfactant, and incidence of pneumothorax. These data were collected every day, always at the same time, on the morning shift, excluding Sundays, during the period of mechanical ventilatory support.

The difference between the two groups lies in the mechanical ventilation parameters that were changed in an attempt to develop a strategy for the reduction of pneumothorax in the first group. The modified parameters were flow (IV) and inspiratory time (IT), in an attempt to obtain a lower tidal volume (TV). The mean of the parameters was obtained after one to five days of mechanical ventilation.

Inspired volume and inspiratory time are adjusted in time-cycled ventilators, and the product of these parameters determines the tidal volume. Therefore $\text{TV} = \text{IT} \times \text{IV}$ (TV = ml; IT = sec and IV = ml/sec).^{16,17}

Inspiratory time depends on the time constant and will vary according to lung compliance. The generally used IV is three times higher than the minute volume (MV). The TV depends on lung compliance, on the pressure gradient (inspiratory pressure - PEEP); in general a TV of 6 to 8 ml/kg is used. The use of high TVs (10 to 12 ml/kg) may result in alveolar overexpansion and in the risk of air leak syndrome (ALS).^{18,19}

The mean airway pressures were obtained through the equation expressed by

$$\text{MAP} = k (\text{PIP} - \text{PEEP}) \cdot \text{IT} / (\text{IT} + \text{ET}) + \text{PEEP}^1.$$

PTX was diagnosed through clinical manifestations and were confirmed radiologically. NBs may be asymptomatic or show variable degrees of respiratory distress, such as moaning, tachypnea and retractions. There is sudden worsening of respiratory insufficiency, agitation, cyanosis, and decrease in oxygen saturation. On examination of the chest, the affected side might be bulgy; on auscultation, the vesicular murmur is low. The heart sounds may be muffled, and there may be cardiac tamponade, hypotension, and shock. The liver and the spleen might be palpable due to the lowering of the diaphragm. If early diagnosis is not established, death may occur.^{20,21}

The results obtained were submitted to statistical analysis using chi-square tests, with a significance level of $\alpha = 0.05$ and Student's t-test with a significance level of $\alpha = 0.05$ and 0.01 (Sokal & Rolph, 1981).²²

Results

The study included 127 preterm newborns. Fifty-three patients in group A (control) presented an average weight of 1,395g and a standard deviation of 493.6g, and 74 patients in group B (with reduction of ventilatory parameters) presented an average weight of 1,547g and the standard deviation of 510g. The distribution of the patients into groups, according to gender, gestational age and birthweight, was homogeneous.

In group A (54 patients), there was an incidence of 13 cases of PTX (24.5%) and in group B (74 patients) there was an incidence of six cases of PTX (8.1%), yielding significance ($p < 0.05$) to the comparison between the groups (Table 1).

The comparison between the groups did not present any statistical significance. Group A presented a 50.9% death rate and a 49.1% rate of hospital discharge, while group B presented a 43.2% death rate, and a 56.8% of hospital discharge ($p = 0.391$). (Table 1)

The weight of the patients in group A who presented pneumothorax averaged 1,203g, and they received an average PEEP of 6.26 cmH₂O, IP of 21.87 cmH₂O, RF of 30.30 cycles/minute, IT of 0.64 sec and inspiratory flow of 8.26 l/m and MAP of 11.57. The weight of the patients in group B averaged 1,192g and they received an average PEEP of 6.11 cmH₂O, an average IP of 21.8 cmH₂O, RF of 28.12 cycles/minute, an average IT of 0.637 sec, inspiratory flow of 6.38 l/m, and MAP of 10.38. The comparison between the two groups showed that IT and IV presented a significant difference ($p < 0.01$) and a MAP ($p < 0.05$). The other parameters did not present a statistically significant difference (Table 2).

Discussion and conclusion

In the neonatal period there is a great risk of ALS development, because there is a greater frequency of respiratory failure at this age; consequently, the use of ventilatory support and neonatal resuscitation procedures with the application of positive pressure is deemed necessary.

Table 1 - Comparison of the incidence of pneumothorax and the result of patients from Group A and Group B

Result	Pneumothorax incidence				Deaths			
	Group A		Group B		Group A		Group B	
	n	%	n	%	n	%	n	%
Positive	13	24.5	6*	8.1	27	50.9	32†	43.2
Negative	40	75.5	68	91.9	26	49.1	42	56.8

* Less significant than those who were exposed, according to chi-square ($\chi^2 = 6.545$; $p < 0.05$)

† Nonsignificant difference ($\chi^2 = 0.736$; $p = 0.391$)

Table 2 - Comparison between the means (standard deviation) of Group A and Group B for the following parameters

Parameter	Group A		Group B		Student's <i>t</i> -test	
	Mean	SD	Mean	SD	<i>t</i>	p
IT	0.64	0.04	0.59 †	0.06	5.204	0.000
Flow	8.26	1.21	6.38 †	1.11	9.102	0.000
PIP	21.87	3.75	21.08 ‡	3.16	1.280	0.203
PEEP	6.26	0.71	6.11 ‡	0.85	1.088	0.279
RF	30.30	7.97	28.12 ‡	7.59	1.564	0.120
ET	1.49	0.57	1.71 ‡	0.77	-1.834	0.069
MAP	11.57	2.71	10.38 *	2.37	2.621	0.010

* ($p < 0.05$) and † ($p < 0.01$) according to the Student's *t* test

‡ nonsignificant difference

Ogata et al. have reported a variable incidence regarding respiratory failure and the ventilation method used in newborns with RDS. In this study, the incidence of PTX was 3.5% in newborns that did not receive ventilatory support, 11% in the ones using continuous positive airway pressure (CPAP), and 33% in the ones that needed mechanical ventilation with the use of positive end-expiratory pressure (PEEP).²¹

In retrospective studies involving 119 newborns with RDS, Madansky et al. found some form of air leak in 27% of the newborns. Of these, 24% developed air leak syndrome during inhaled oxygen treatment, 16% during the use of CPAP, and 34% during intermittent mandatory ventilation. Among newborns diagnosed with meconium aspiration syndrome, 41 presented some form of air leak, while patients with transient tachypnea presented a 10% incidence of air leak.²³

Goldberg and Abdenour mentioned the incidence of 24% of pneumothorax in mechanically-ventilated preterm newborns with RDS who did not receive surfactant, compared to 5.9% in the group that received surfactant as treatment for RDS. Other papers show a greater incidence of PTX in newborns who, when mechanically ventilated, received a greater peak inspiratory pressure (PIP), longer inspiratory time (IT), and greater mean airway pressure.^{24,25}

According to Amato et al., the application of a protective strategy for pulmonary ventilation using PEEP below the inflection point of the pressure/volume curve, with a tidal volume of 6ml/kg and inspiratory pressure lower than 20 cmH₂O, decreased the incidence of barotrauma and

increased the frequency of weaning from mechanical ventilation, when compared to conventional mechanical ventilation.¹⁰

In a newborn, the duration of IT is much shorter, as well as the inspired volume, which reflects a low specific compliance. Relating TV to IT, the IP is much higher in newborns than in adults.¹ The use of very low IPs may cause hypoventilation with hypoxemia and hypercapnia; on the other hand, the use of high IPs is associated with the development of ALS, increase of long-term pulmonary vascular resistance (PVR) and chronic pulmonary disease.²⁶

Inspiratory time depends on a time constant, which is directly related to lung compliance. When lung compliance is low, the time constant also diminishes. Therefore, the IT used in ventilation may be as low as 0.3 to 0.5 sec. When lung compliance is nearly normal, the time constant is higher and, consequently, higher ITs (close to 0.5 to 0.7 sec) will be necessary.²⁷

Inspired volume has a direct relationship with IT and IP. A high flow through the ventilator (> 6 l/m) easily reaches the predetermined pressure, but it exposes the alveoli to a peak pressure for a prolonged time, providing high tidal volumes.²⁶ The high flow also causes a turbulent flow that elevates airway resistance, which increases the respiratory effort. The energy expenditure necessary to overcome the resistance at expiration can be so high that an undesirable PEEP may occur at the end of expiration.²⁸ This kind of ventilation can be effective to correct hypoxemia, but it is related to a high incidence of lung injury, such as PTX.¹⁸

The incidence of PTX is high in RDS when high pressures or high volumes during mechanical ventilation are used. Amato *et al.* have suggested that the value of the application of a protective strategy for mechanical ventilation with the use of low tidal volume and inspiratory pressure to minimize lung injury may not only reduce complications but also mortality in RDS patients.²⁹

The results show a greater incidence of PTX (24.5%) in the group that received greater IV and IT, when compared to the group in which these parameters were reduced (8.1%).

The most recent studies focus on ventilatory strategies with pulmonary protection, i.e., those that recruit appropriate lung volume without overexpansion, therefore reducing the development of PTX.^{11,30}

Through this study we verified that an IT lower than 6 secs and an IV lower than 7 l/min at the initial phase of RDS are related to a lower PTX frequency, but this protective strategy attempt with a lower flow and inspiratory time is not associated with mortality reduction.

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References

1. Goldsmith JP, Karotkin EH. Introduction to assisted ventilation. In: Goldsmith JP, Karotkin EH, editores. Assisted ventilation of the neonate. 3rd ed. Philadelphia: WB Saunders; 1996.p.1-19.
2. Muller NL, Bixan AC. Chest wall mechanics and respiratory muscles in infants. *Pediatr Clin North Am* 1979;26:503-16.
3. Roussos CS, Macklen PT. Diaphragmatic fatigue in man. *J App Physiol Respirat. Environ Exercise Physiol* 1977;43:189-97.
4. Scarpelli EM, Auld PAM, Goldman HS. Pulmonary disease of the fetes, newborn and child. Philadelphia: Lea & Febiger; 1978.
5. Kattan M. Long term sequelae of respiratory illness in infancy and childhood. *Pediatr Chin North Am* 1979;26(3):525-35.
6. Avery ME, Fletcher BD, Willians RG. Hyaline membrane disease. In: Avery ME, Fletcher BD, Willians RG, editores. The cring and its disorder in the newborn infant. Philadelphia: WB Saunders; 1981.
7. Truman TL; Todre ID. Acute respiratory distress syndrome. In: Todres ID, Tugate JH, editores. Critical care of infants and children. Boston: Little Brown; 1996.p.147-159.
8. Slutsky AS. Mechanical ventilation. *Chest* 1993;104:1833-59.
9. International Consensus Conference Committee:International Consensus Conferece in Intensive care medicine: Ventilator – associated lung injury in ARDS. *Am J Respir Crit care med* 1999;160:2118-24.
10. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, *et al.* Effect of a Protective-ventilation Strategy on Mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998;338(6):347-54.
11. Brochard L, Roudot-Thoraval F, Roupie E, Delclaux C, Chastre J, Fernandez-Mondejar E, *et al.* Tidal volume reduction for prevention of ventilator-induced lung injiery in acute respiratory distress syndrome. The multicenter trail croup on tidal volume reduction in ARDS. *Am J Respir Crit Care Med* 1998;158:1831-8.
12. Petersen GW, Baier H. Incidence of pulmonary barotrauma in a medical ICU. *Crit Care Med* 1983;11:67-9.
13. Gammon RB, Shin MS, Groves RH Jr, Hardin JM, Hsu C, Buchalter SE. Clinical Risk factors for Pulmonary Barotrauma: a multivariate analysis. *Am J Respir Crit Care Med* 1995; 152: 1235-40.
14. Dreyfuss D, Saumon G. Role of Tidal Volume, FRC and end – expiratory volume in the development of pulmonary edema following mechanical ventilation. *Am Rev Respir Dis* 1993; 148:1194-203.
15. Capurro H, Konichezky S, Fonseca D, Caldeyrobarcia R. Simplified method for diagnosis of gestational age in newborn - infant. *J Pediatric* 1978;93(1):120-2.
16. Chatburn RL. Principles and practice of neonatal and pediatric mechanical ventilation. *Resp Care*1991;36:569-874.
17. Kaiserman KB, Cunningham MD, Martin G, Stevens J. Optimal tidal volume in the neonate during conventional mechanical ventilation. *Clinical Research* 1992;40(1):A84-A84.
18. Kumar A, Pontoppidan H, Flake KJ, Wilson RS, Laver MB. Pulmonary barotrauma during mechanical ventilation. *Cret Care Med* 1973;4:181-6.
19. Steier M, Ching N, Roberts EB, Neolon TF. Pneumothorax complicating continuais ventilatory support. *J Thorac Cardiac Surg* 1974;67:17-23.
20. Krebs VLJ, Trostes EJ. Complicações da ventilação mecânica. *Pediatria Moderna* 2000;XXXVI:junho (edição especial).
21. Ogata ES, Gregory GA, Kitterman JA, Phibbs RH, Tooley WH. Pneumothorax in the respiratory distress syndrome: incidence and effect on vital signs, blood gases, and pH. *Pediatrics* 1976; 58:177-83.
22. Sokal RR, Rolph FJ. Biometry. 2nd ed. San Francisco: WH Freemann and Co.; 1981.
23. Madansky DL, Lawson EE, Chernick V, Taeusch HW. Pneumothorax and other forms of pulmonary air leak in newborns. *Am Rew Resp Dis* 1979;120:729-37.
24. Goldeberg RN, Abdenour GE. Air leak syndrome. In: Spitzer AR, editor. Intensive care of the fetus and neonate. St. Louis: Mosby-yea; 1996.p.629-640.
25. Glenski JA, Hall RT. Neonatal pneumopericardium: analysis of ventilatory variables. *Crit Care Med* 1984;12:439-42.
26. Spitzer AR, Fox WW. Positive presure ventilation: pressure limited and time - cyded ventilations. In: Goldsmith JP, Karotkin EH, editores. Assisted ventilation of the neonate. 3rd ed. Philadelphia: WB Saunders; 1996.p.176-186.
27. Heicher DA, Kasting DS, Harrod JR. Prospective clinical comparison of two methods for mechanical ventilation of neonates: rapid rate and short inspiratory time versus slow rate and long inspiratory time. *J Pediatr* 1981;98:957-61.
28. Sampietro VI, Azevedo MPO, Resende JG. Medida da resistência do fluxo aéreo em peças nasais de CPAP [site na internet]. Disponível em: <http://www.sbp.com.br/jornal/00-03.04/artarig4.html>>. Acessado 11 de novembro de 2001.

29. Kacmareck RM. Strategies to optimize alveolar recruitment. *Crit Care* 2001;7:15-20.
30. Brower RG, Shanholtz CB, Fessler HE, Shade DM, White P Jr, Wiener CM, et al. Prospective, randomized controlled clinical trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. *Crit Care Med* 1999;27(8):1492-8.

Corresponding author:

Dr. Marisa A.A. Brunherotti
PROENE - Programa de Estudos em Neonatologia
Rua Thomas Gonzaga, 1932 – Centro
CEP 14400-540 – Franca, SP, Brazil
E-mail: brunherotti@uol.com.br