

# Application of Maximum Tolerable Wall Tension Index as an Indicator of Lung Injury following Acid Aspiration

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## Citation

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## Abstract

Alveolar wall tension is the most important single parameter contributing to development of additional lung injury related to positive pressure ventilation in the patients with acute respiratory distress syndrome. In order to evaluate the clinical impact of changes in microvascular damage, we have compared protein leakage to corresponding abnormalities in lung compliance and lung wall tension index. Groups of rats were injured by intrapulmonary deposition of normal saline, acid (pH=1.25), with or without gastric particulate matter. Protein permeability was assessed using 125I-albumin labeled bovine serum albumin. The lungs were then immersed in saline solution and inflated to the degree of air leakage from the surface. The maximum tolerable wall tension index (MTWI) was calculated. The MTWI was significantly lower in acid-injured lungs compared to uninjured lungs ( $179.37 \pm 12.88$  vs  $301.50 \pm 20.06$ ,  $p < 0.01$ ). This value was even lower in rats injured with acid and gastric particulate. The aerodynamic data closely corresponded to the increase in PI,  $0.3 \pm 0.02$  in uninjured lungs vs  $0.81 \pm 0.10$  in acid-injured and  $4.26 \pm 0.51$ , in lungs injured with both acid and particles ( $R^2 = 0.79$ ,  $p < 0.01$ ).

These findings indicate that MTWI is a sensitive technique to assess the extent of lung injury in experimental settings. Wall tension index of the lungs, the product of  $(VT)^{1/3}$  and PAP, can potentially serve as a guide to the safety of positive pressure ventilation.

## INTRODUCTION

Positive pressure ventilation is still the mainstay of therapy in managing the patients with acute respiratory distress syndrome (ARDS).<sup>1</sup> Despite significant improvement in ventilatory management of these patients, positive pressure respiration is associated with development of barotrauma and volutrauma to the lung that further deteriorates the underlying lung injury.<sup>2</sup> Most of the studies involving acute lung injury in small animals, use protein permeability and wet and dry ratio (W/D) weight as indices of lung injury.<sup>3,4,5</sup> Even though, there is a correlation between protein leakage and neutrophil infiltration into the lungs, it is difficult to apply these parameters of lung injury to patients. To examine the clinical impact of changes in microvascular damage, in this study, we have compared protein leakage to

corresponding abnormalities in lung compliance. A new parameter, alveolar wall tension index, is also defined that enables us to assess the extent of lung injury with more accuracy, and is more applicable to the clinical settings.

## METHODS

Use of animals in this study was approved by the Institutional Committee for Laboratory Animals Use and Care (IACUC). Male, Long-Evans rats (250-300g) were anesthetized with halothane. Groups of rats ( $n=9$  per group) were injured by intrapulmonary deposition of normal saline (pH=5.3), acid (pH=1.25), 1.2 ml/kg with or without 40 mg/ml gastric particulate matter (10 m). One ml of 125I-albumin 2% (0.05 Ci) was given intravenously. The animal was awakened and resumed spontaneous ventilation. Following 5 hours, the rat was sacrificed and the lungs were

perfused with saline to clear residual blood from pulmonary vasculature. The lungs were then immersed in saline solution and inflated to the degree of air leakage from the surface. The maximum volume (MV) and mean pulmonary airway pressure (PAP) prior to the leakage were recorded. The maximum tolerable wall tension index (MTWI) was calculated by following formula for each pair of lungs:

$$\text{MTWI} = \text{MV}^{1/3} \times \text{PAP}$$

Airway pressure changes were measured per incremental changes of volume in a continuous fashion. Data were acquired by Labview software and  $dV/dP$  was calculated and used as an index of static lung compliance. MTWI is calculated by utilizing the cubic root of volume which is length and is expressed as a fraction of cm per kg of body weight. The product of length and pressure (cmH<sub>2</sub>O) results in the unit of cm.kg-1.cmH<sub>2</sub>O.

The radioactivity of the lungs and one ml of blood was counted by a gamma counter and the ratio is expressed as permeability index (PI). This parameter has been extensively used in our laboratory as well as other and is closely associated with the extent of lung injury in rats.<sup>4</sup>

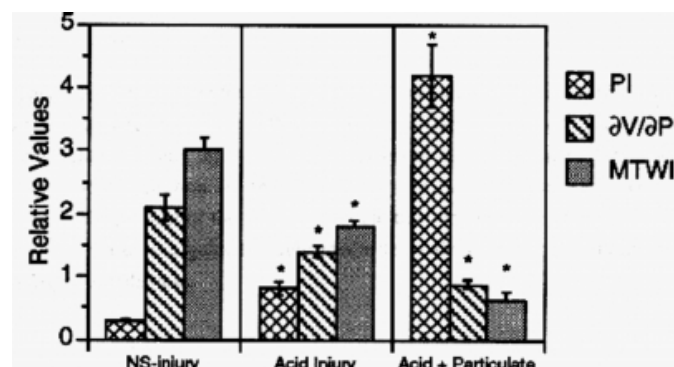
## RESULTS

Animals injured with combination of acid and gastric particulate were less tolerant to increases in pulmonary airway pressure and higher volumes of lung insufflation, when compared to the lungs injured with acid alone and NS-injured lungs. The MTWI was 301.50 +/- 20.06 cm.Kg-1.cmH<sub>2</sub>O for uninjured lungs. It significantly decreased in the lungs injured with acid, 179.37 +/- 12.88 cm.Kg-1.cmH<sub>2</sub>O compared to NS-injured lungs ( $P < 0.05$ ). Addition of gastric particle to the acidic aspirate caused further decrease in the ability of the lungs to tolerate increases in the alveolar wall tensions, 63.50 +/- 13.33 cm.Kg-1.cmH<sub>2</sub>O,  $p < 0.01$ . (See Figure 1) Concomitant measurement of protein permeability revealed a parallel response following similar types of lung injury. Intrapulmonary instillation of acid increased protein permeability from 0.30 +/- 0.02 in NS-injured lungs to 0.81 +/- 0.10 in acid-injured lungs. Intratracheal deposition of acid in conjunction with gastric particulate synergistically increased PI to 4.26 +/- 0.51 ( $P < 0.01$ ). The aerodynamic data closely correlated to the increase in PI ( $R^2 = 0.79$ ,  $p < 0.01$ ). The  $dV/dP$  of the lungs significantly decreased following intratracheal instillation of acid + gastric particulate, 0.71 +/- 0.06 ml.Kg-1.cmH<sub>2</sub>O-1, when

compared to  $dV/dP$  of acid-injured, 1.31 +/- 0.16 ml.Kg-1.cmH<sub>2</sub>O-1, and NS-injured lungs 2.4 +/- 0.09 ml.Kg-1.cmH<sub>2</sub>O-1.

**Figure 1**

Figure 1



Permeability Index (PI) was significantly higher in the rats which were injured with acid or with combined acid and particulate than in NS-injured rats. Lungs were less compliant and tolerated smaller tension (MTWI, values are divided by 100 to fit in the chart for comparison) compared to NS injured lungs (\* $P < 0.05$ )

## CONCLUSION

These findings indicate that MTWI is a sensitive technique to assess the extent of lung injury in experimental settings. The values for this parameter closely correlated with the other indicators of lung injury (capillary leakage of protein and static lung compliance) following acid aspiration. Furthermore, this value can easily be calculated in clinical settings as the product of the cubic root of the tidal volume (VT) and pulmonary airway pressure, and used as a guide to safely administer positive pressure ventilation in patients with respiratory distress.

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