

# The Effect Of Varying Inspiratory Flow Waveforms On Alveolar Peak Airway Pressure And Intrinsic Positive End Expiratory Pressure In Mechanically Ventilated Patients

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## Abstract

**Purpose:** To prospectively evaluate peak airway pressure (PaW) intrinsic PEEP (PEEPi), extrinsic PEEP (PEEPe) and peak expiratory flow rate (PEFR) with changing inspiratory flow waveforms during mechanical ventilation in patients.

**Material And Methods:** Ten critically ill mechanically ventilated patients on controlled mechanical ventilation (CMV) with acute respiratory failure admitted to the intensive care unit were evaluated to assess the effects of decelerating, square and sine waveforms on). PaW, PEEPi, PEEPe, PEFR

**Results:** PaW was lower with the decelerating waveform compared to square and sine There was no statistical difference in PEEPi, PEEPe or PEFR with varying inspiratory flow waveforms..

**Conclusions:** Adjustment of inspiratory flow waveforms does not affect PEEPi. A lower PaW coupled with a stable PEEPi makes the decelerating waveform preferred for mechanically ventilated critically ill patients on CMV.

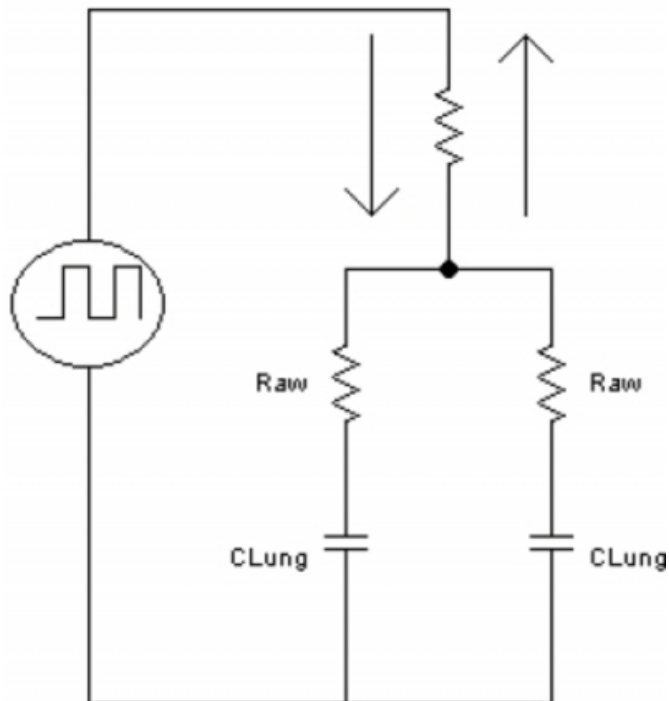
## INTRODUCTION

Clinicians have become increasingly aware of the importance of delivering effective mechanical ventilation while trying to avoid injury associated with different modes. Research has examined untoward effects of certain variables of mechanical ventilation including tidal volume (Vt), intrinsic and extrinsic positive end expiratory pressure (PEEPi and PEEPe) and respiratory rate (RR) (1,2,3,4,5,6). Air trapping and breath stacking often cause development of an increased PEEPi, adverse effects include: altered mechanical ventilation measured variables, hemodynamic compromise, misreading of central venous and pulmonary artery catheter pressure measurements. Other effects are erroneous calculations of static respiratory compliance and increases in work of breathing, which could delay weaning from the ventilator. (7,8,9,10,11,12,13,14) Less obviously, the effect of pressure variation of the inspiratory waveform has been implicated in shear stress damage at the alveolar and lung parenchymal level possibly resulting in a cytokine cascade capable of damaging other organ systems.

Modeling of the lung as a simple electrical capacitance to simulate lung compliance and an electrical resistance as an analog for airway resistance (15) (Fig1) indicates that, at lower respiratory frequencies, levels of PEEPi could be independent of the applied inspiratory waveform. The integrating properties of the compliance and resistance should, theoretically, achieve the same peak inspiratory pressure and PEEPi as long as Vt is the same, and expiratory time is sufficient to prevent breath trapping. This might suggest that the inspiratory waveform should confer no therapeutic advantage with respect to PaW and PEEPi.

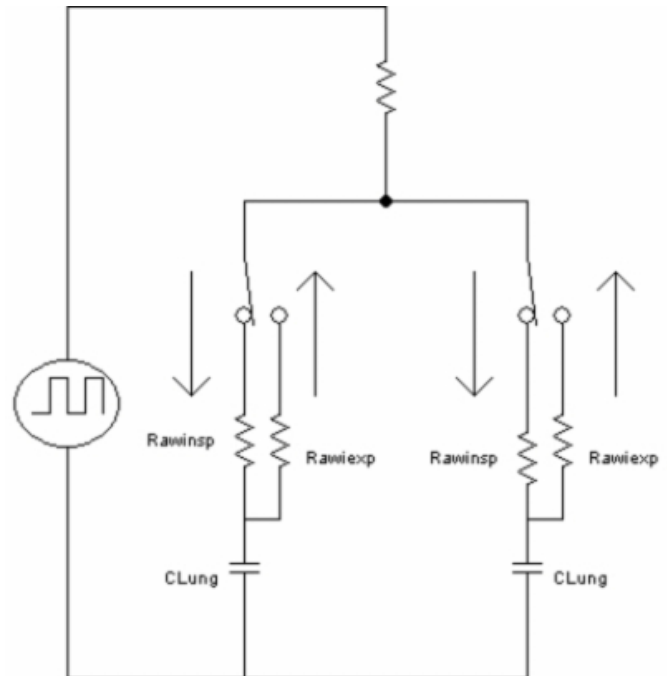
**Figure 1**

Figure 1: A simple electrical analog model of the lung and airways composed of fixed a fixed airway resistance and lung compliance independent of the applied waveform (the mean airway pressure should be the same).



**Figure 2**

Figure 2: Airway resistance may be flow sensitive and exhibit dynamic characteristics that are flow dependent. This could explain why the decelerating flow waveform might generate less harmful effect within the lung.



Studies on varying inspiratory waveform patterns in mechanically ventilated patients and their effect on gas exchange, respiratory mechanics and hemodynamic parameters, however, have been scarce and largely inconclusive. Different waveforms might have deleterious effects on respiratory mechanics by altering other respiratory parameters such as PaW, PEEPi, PEEPe and peak expiratory flow rate (PEFR).

This study seeks to evaluate the effects of three different inspiratory flow waveforms on PaW, PEEPi, PEEPe and PEFR in critically ill mechanically ventilated patients with acute respiratory failure on controlled mechanical ventilation (CMV) and to assess the adequacy of the electrical lung model in this type of application.

## **MATERIALS AND METHODS**

Ten mechanically ventilated patients with acute respiratory failure admitted to the intensive care unit (ICU) of a teaching hospital were evaluated and selected to enter this observational study. The Critical Care and Anesthesia Research Committee conferred approval to evaluate accepted techniques of mechanical ventilation support. This study did not require randomization. All patients were orotracheally intubated with Sheridan low-pressure cuffed

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endotracheal tubes varying in internal diameters from 7 to 9 mm. An esophageal balloon was positioned in the lower third of the patient's esophagus (35 to 40 cm mark from the nose) and connected to a Bicore CP-100 Pulmonary Monitor (Allied Healthcare Products, Irving, CA). Chest radiographs and negative esophageal pressure deflection during inspiration determined adequacy of placement. All patients were afebrile, spontaneously breathing and connected to a Puritan Bennett 7200AE (Pleasanton, CA) with the following settings: volume-cycled ventilator in the controlled mechanical ventilation mode of operation, tidal volume of  $6 \pm 1$  mL/kg, a respiratory rate of 10 breaths/minute, FiO<sub>2</sub> of 0.40 and PEEP of 10 cm H<sub>2</sub>O or less. Pressure sensitivity trigger was reduced to 2 cm H<sub>2</sub>O. All patients were mechanically ventilated for less than one week at initiation of the study. All parameters were recorded by the Bicore Pulmonary Monitor. The parameters measured were: minute ventilation (Ve), respiratory rate RR (breaths/minute), PEEPi, PEEPe, PEFR, mean airway pressure (PaW), change in airway pressure (dpaW).

The patients were randomly placed on each flow pattern for an equilibration time of 5 minutes, then data were collected on each waveform for 2 minutes, downloaded for analysis by the Bicore CP-100 conversion program and loaded to an IBM-PC (CP-100 CVT1.2, Bicore Monitor System). This program converts a binary data file produced by the CP-100 monitor-logging program to an ASCII format parameter file for use with any spreadsheet program. For every patient a mean and standard deviation for every variable was calculated. The initial peak flow was adjusted to attain a constant mean airway pressure (MAP) for the three different waveforms. The inspiratory/expiratory ratio was maintained in a range of 1:2-1:3.

Patients excluded from this study were those with previous esophageal surgery, known abnormal esophageal anatomy and any contraindication to placement of a naso-gastric tube. Data are presented as mean  $\pm$  standard deviation (SD). Statistical analysis was performed using analysis of variance (ANOVA) and a nonparametric Wilcoxon test (Kruskal-Wallis  $\chi^2$ -test) with a general regression linear model. A value of  $p \leq 0.1$  was considered statistically significant.

## RESULTS

Ten critically ill mechanically ventilated patients with acute respiratory failure were evaluated. Their mean age was  $61 \pm 12$  years (range 35 to 77 years). The causes of respiratory insufficiency were complications of thoracic surgery in five

cases, gastric resection in one case, genitourinary surgery in one case and one a complication of breast surgery. All ten patients remained hemodynamically stable during the trial period. Respiratory variables of each patient measured on three different inspiratory flow waveform patterns were compared. Variables are shown in Table 1.

## Figure 3

Table 1: Mean and SD of respiratory variables with varying inspiratory flow waveforms.

Inspiratory Flow Waveform Patterns			
Variables	Decelerating	Square	Sine
Ve (L)	14 $\pm$ 4	13 $\pm$ 4	12 $\pm$ 4
RR (breaths/min)	19 $\pm$ 5	19 $\pm$ 5	18 $\pm$ 4
PEEPi (cmH <sub>2</sub> O)	2.3 $\pm$ 3.2	2.5 $\pm$ 3.1	2.4 $\pm$ 4.1
PEEPe (cmH <sub>2</sub> O)	8.5 $\pm$ 4.3	7.8 $\pm$ 3.2	8.5 $\pm$ 3.4
PEFR (L/min)	84 $\pm$ 13	78 $\pm$ 10	80 $\pm$ 16
PaW (cm H <sub>2</sub> O)	41 $\pm$ 9	45 $\pm$ 7	44 $\pm$ 11
dpaW (cm H <sub>2</sub> O)	33 $\pm$ 8	39 $\pm$ 5	37 $\pm$ 8

PaW and dpaW were lower with decelerating waveform ( $p < 0.05$ ). There was no statistically significant change in PEEPi, PEEPe or PEFR with variation in inspiratory flow waveform. Ve and RR were equal in all the experiments (volume-controlled ventilation). There were no statistically significant differences between the square and sine waveforms in other pulmonary mechanics parameters.

## DISCUSSION

All three inspiratory waveforms applied to assist patients with respiratory failure did not increase PEEPi. No significant differences were noted in PEFR among the three waveforms. Some studies in the past have suggested that there are no significant differences in efficacy of gas exchange (<sub>16,17,18,19,20,21</sub>) or hemodynamics (<sub>18,19,20,21,22,23</sub>) among different waveforms. Other studies have demonstrated improved cardio-respiratory function, including improved gas exchange and respiratory mechanics, with the decelerating inspiratory flow waveform ventilation in normal and diseased lungs compared with the accelerating or constant waveforms (<sub>16,19,21,22,23,24,25,26</sub>). In theory, the sine waveform is the most physiologic in terms of efficiency and energy expenditure in healthy lungs. The decelerating waveform, however, seems to be advantageous with increasing respiratory requirements because the respiratory compliance is improved. A clinical study demonstrated that the use of the decelerating waveform may have beneficial

clinical implications in critically ill mechanically ventilated patients with acute respiratory failure because it improved pulmonary mechanics (<sub>16</sub>). We previously described that the peak airway pressures were significantly lower using the decelerating inspiratory flow waveform and that dynamic compliance improved compared with the other waveforms (<sub>7</sub>).

The difficulty in measuring airway pressure should not be underestimated. Direct alveolar pressure measurement under flow conditions is technically challenging and impractical in the clinical setting. Furthermore the readouts provided by mechanical ventilators can at times be confusing due to interchangeable terms used. These measurements represent pressures measured at the proximal end of the endotracheal tube and can be misleading if interpreted as alveolar pressure. The dynamics of gas flow through the airways manifest non-intuitive pressure effects at the alveolar level. Significant to our study was the method used to measure the various airway pressures encountered. The Bicore device measures airway pressures by the use of an esophageal balloon positioned within the thoracic cavity and better represents intra pleural and airway pressures at the alveolar level. These pressures are, furthermore, measured dynamically as opposed to some methods that require special airway maneuvers to elude these values (<sub>30</sub>). Consequently, PaW, and PEEP<sub>i</sub> can be more accurately observed while varying the inspiratory waveform parameters. A randomized and comparative trial of fifty-four patients with COPD was performed applying constant, decelerating, and sine waveforms in a random order. The decelerating waveform produced statistically significant reductions of peak inspiratory pressure, mean airway resistance, physiologic dead space ventilation (<sub>31</sub>). There was also a significant increase in alveolar-arterial oxygen pressure difference with the decelerating flow waveform, but there were no significant changes in pulmonary variables and other hemodynamic measurements. The most favorable flow pattern for ventilated patients with COPD appeared to be the decelerating waveform. In an additional study on ten chronic obstructive pulmonary disease patients receiving mechanically ventilation for acute respiratory failure ventilated with a constant or a decelerating inflation flow profile; tidal volume and respiratory frequency were similar during the experimental conditions. The authors concluded that the inspiratory waveform profile had no significant cardiorespiratory effect in intubated COPD patients mechanically ventilated for acute respiratory failure. (<sub>32</sub>)

Patients with obstructive lung disease are particularly prone to developing PEEP<sub>i</sub> and therefore have difficulty triggering the ventilator. At constant PEEP<sub>i</sub>, the decelerating and the two other waveforms did not impair alveolar recruitment. In a study by Nilsestuen, et al, respiratory rate and tidal volume were fairly similar and constant throughout the study. The authors concluded that bedside evaluation for the presence of PEEP<sub>i</sub> should be routinely performed and corrective adjustments made when appropriate (<sub>33</sub>)

A protective ventilatory strategy that uses a low-tidal volume and repetitive alveolar recruitment-derecruitment decreased pulmonary and systemic cytokines and, more importantly, decreases mortality (<sub>2,29</sub>). At the same time, lower tidal volume ventilation is one of the strategies that might increase PEEP<sub>i</sub> in mechanically ventilated patients (<sub>30</sub>). A study conducted by de Durante and coworkers demonstrated that the ventilatory settings employed in the ARDSNet low Vt group may generate an auto-PEEP of  $5.8 \pm 3$  cmH<sub>2</sub>O. These findings suggest that patients with ARDS, ventilated at relatively high respiratory rates develop greater PEEP<sub>i</sub> than when ventilated at lower rates, even for the same minute ventilation. This mechanism may produce decreased lung injury secondary to recruitment–derecruitment, and hence provides a plausible explanation for some of the decreased mortality observed in the ARDSNet trial in the 6 ml/kg group (<sub>34</sub>). Vieillard Baron and coworkers investigated the effects of increasing respiratory rate from 15 to 30 breaths/min, while maintaining a constant plateau pressure. Those authors reported a PEEP<sub>i</sub> of  $6.4 \pm 2.7$  cmH<sub>2</sub>O at 30 breaths/min, which was associated with an increased right ventricular outflow impedance and a decreased cardiac index. The authors concluded that, in acute lung injury ARDS patients, the use of higher respiratory rate at constant plateau pressure in order to increase minute ventilation is unable to improve elimination of carbon dioxide, while it generates auto-PEEP (<sub>35</sub>). It is possible to speculate that the observed difference in survival could be related to the difference in total PEEP. (<sub>36</sub>) The ARDSNet findings pertain to the possible role of PEEP<sub>i</sub> causing the observed difference in mortality between different tidal volume strategies.

In our study, both lower PaW and dpaW were generated by the decelerating waveform compared to sine and square waveforms, while the mean airway pressure was kept constant at all flow patterns. The lower peak pressure during ventilation suggests a flow mediated airway resistance,

which can be accounted for by a high initial peak flow followed by an even distribution of ventilation. A partial narrowing of some portion of the conducting airways during the higher initial flow of the decelerating waveform may be responsible for this. This is comparable in effect to the changes in airway resistance that occurs during expiratory flow and may account for the hysteresis observed in the flow-volume loop across the respiratory cycle. A simple electrical analog model of the lung and airways composed of a fixed airway resistance and lung compliance independent of the applied waveform will result the mean airway pressure to be the same. These results show that any simple electrical model used to describe this requires modification as an explanatory tool for understanding the differences between the various inspiratory waveforms. An alternative model of airway resistance may be flow sensitive and exhibit dynamic characteristics that are flow dependent. This could explain why the decelerating flow waveform might generate less harmful effect within the lung. More interesting, perhaps, are the findings with regard to  $\text{dpaw}$ . This parameter may relate more closely to the physical excursions that alveoli undergo during the respiratory cycle. These measurements suggest that decelerating waveform ventilation may cause less barotrauma and can be used as an added source of protection to the airways in these patients.

One of the limitations of this study is the small number of subjects. Larger randomized clinical studies are needed to provide support of the results of this study. Another limitation is short time of equilibration and short time on each of the waveforms. We do not know if a longer period of time might increase  $\text{PaW}$ ,  $\text{dpaw}$ , and  $\text{PEEPi}$  but it should theoretically not cause major variations in pulmonary mechanics. Further clinical testing of respiratory mechanics with decelerating waveforms in different modes of ventilation is worth serious consideration. All the findings, thus far, support the hypothesis that this waveform may be more effective in the ventilation of critically ill patient with respiratory failure. The importance of delivering effective mechanical ventilation to critically ill patients with respiratory failure while avoiding associated lung injury or significant hemodynamic compromise cannot be overemphasized. It is possible that a small numeric advantage in respiratory parameters with decelerating waveform pattern can lead to a noticeable beneficial effect on clinical outcomes in critically ill patients with respiratory failure.

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