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**Original Research Article**

# Plateau Pressure and Peak Airway Pressure as Predictors of Mortality in Mechanically Ventilated Patients – A Retrospective Observatioal Study

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**Abstract:** Plateau pressure and peak airway pressure are directly related to the poor outcome of ventilator supported patients. Monitoring of these parameters helps clinicians in early identification of impending mortality and initiation of measures to prevent the same. This is a retrospective study in which 100 patients who were on ventilator support for >24 hours and died there after were studied. Here patient's ventilatory parameters like plateau pressure and peak airway pressure were collected at base line, 1 hour, 30 min, 15 min, 5 min prior to cardiac arrest. Other ventilatory parameters like tidal volume and positive end expiratory pressure (PEEP) were also collected. In all the studied patients plateau pressure and peak airway pressure at 1hour, 30min, 15min, and 5min prior to cardiac arrest were high compared to base line values. These values were statistically significant as 'P' value was < 0.05. We conclude that a continuous, sustained rise in plateau and peak airway pressure above the baseline is useful in predicting mortality in mechanically ventilated patients.

**Keywords:** Plateau airway pressure, Peak airway pressure, Mechanical Ventilator, Mortality.



## INTRODUCTION

Mechanical ventilators are used to increase survival rates in critically ill patients (Slutsky, A. S. *et al.,* 2013) (Dreyfuss D *et al.,* 1998) Advances in mechanical ventilators have made it possible to adjust ventilator settings and at the same time monitor and store various parameters. Appropriate ventilator strategies have helped to improve patient outcome (Dreyfuss, D. *et al.,* 2014) (Burns, K. E. A. *et al.,* 2011) (Luhr, O. R. *et al.,* 2000).

Ventilator settings are useful in predicting mortality and morbidity in mechanically ventilated patients. (Brun- Buisson, C. *et al.,* 2004) (Gajic, O. *et al.,* 2007) Parameters like high tidal volumes and airway pressures have been associated with poor outcomes in mechanically ventilated patients with acute respiratory distress syndrome (ARDS) (Esteban, A. *et al.,* 2002) (Esteban, A. *et al.,* 20011) High peak inspiratory pressure ($P_{max}$), high plateau pressure ($P_{plat}$), driving pressure and increased respiratory rate are potentially modifiable factors predicting poor outcome in ventilator patients (Brochard, L. *et al.,* 1998) (Laffey, J. G. *et al.,* 2016) (Amato, M. B. P. *et al.,* 2015). Thus ventilator parameters can be monitored and modified to improve outcome and predict mortality of mechanically ventilated patients. (Slutsky, A. S. 2005).

This retrospective observational study was conducted to evaluate plateau pressure and peak airway pressure for prediction of mortality in mechanically ventilated patients.

## MATERIAL AND METHODS

Institutional ethical committee approval was taken for the study and as it is a retrospective study, need for consent was waived. Initially ventilatory data of 140 patients who were on mechanical ventilator in an 18 bedded ICU located in this tertiary care hospital was collected for study. Out of these 40 patients were excluded from study due to incomplete or missing data. In the final analysis, 100 death cases were included. These patients had been on invasive mechanical ventilator for more than 24 hours and expired after that.

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**Inclusion Criteria**

Adult patients of either gender who were on mechanical ventilatory support for more than 24 hours and were admitted to intensive care unit (ICU).

**Exclusion Criteria**

- Patients with known cardio-pulmonary disease where baseline peak / plateau pressure were already high. eg- Interstitial lung disease, Pulmonary edema.
- In patients where Endotracheal /Tracheal tube cuff was not inflated.
- Patients with chest tubes in situ or pulmonary fistulas.
- Accidental extubations, disconnections from the ventilator.
- Patients with ventilator positive end expiratory pressure (PEEP) more than 10cm $H_2O$.
- Any technical issue like failure to collect or record/store data from the ventilator.

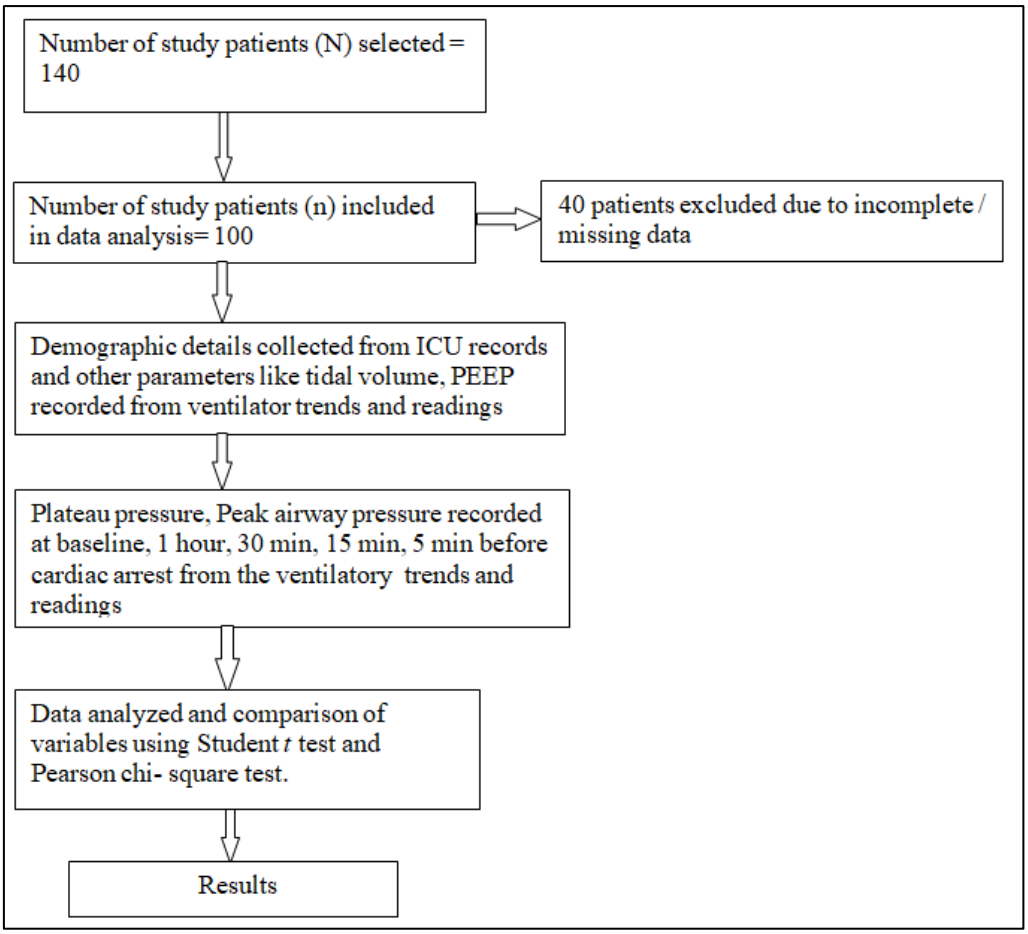


**Figure 1: Data collection of patients based on inclusion and exclusion criteria**

We collected the ventilator data of 100 patients with respect to end point of cardiac arrest retrospectively from the mechanical ventilator and ICU record books. Ventilator parameters were collected after 1hour of initiation of ventilator support so that patient was adequately titrated with respect to disease, ventilator mode, synchrony and tolerance of the ventilator settings. This was monitored based on the stable haemodynamics, oxygen saturation ($SpO_2$), and acceptable blood gas analysis and considered as the baseline values. Ventilator parameters at 1hour, 30 min, 15 min and 5 min prior to cardiac arrest were collected. The data was collected with respect to cardiac arrest, as after this a series of measures like chest compression were taken which interfere with airway pressures. The parameters observed and collected were tidal volume, peak air way pressure, plateau pressure, and PEEP. To maintain uniformity four mechanical ventilators of same company were used to collect the data. Stored data from the mechanical ventilator was collected after death of the patient before the ventilator was used for a new patient. Confirmation of ventilator alarms before death of patient by staff nurse handling the patient was also sought. Demographic details and other relevant data were collected from ICU records.

**Statistical Analysis**

For our analysis continuous variables are presented as mean +/- standard deviation if normally distributed and median if non-normally distributed. Categorical variables are presented as counts and percentages. Comparisons between variables are conducted using the Student t test for continuous variables and the Pearson chi- square or Fisher exact

test for categorical variables. Statistical analysis was performed by using STATA Version 14. The parameters observed at baseline, 1hour, 30 min, 15 min and 5 min prior to baseline were compared and a P value > o.o5 was considered statistically significant.

## RESULTS

In our study, out of 100 patients 62 were male and 38 were female (Table 1).

**Table 1: Characteristics of patients**

| **Patient characteristics** | | **Mean ( Std Deviation)** |
|---|---|---|
| Age (Years) | | 49.51 ( ± 16.3741) |
| Sex | Male | 62 |
| | Female | 38 |

The mean age of the study group was 49.51years (SD ±16.3741). The collected data included ARDS as well as non ARDS patients. Out of 100 patients, most of them (19) had an admitting diagnosis involving respiratory system (Table 2). Poisoning, endocrine, cardiovascular, renal, neurological and others like postoperative cases were the remaining patients.

**Table 2: Admitting diagnosis and Initial ventilatory parameters**

| ***Admitting Diagnosis** | | **#Initial Ventilatory Settings** | | | | | **Mean Ventilatory Parameters** | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Tidal Volume(ml/ Kg) | Respiratory Rate (per min) | PEEP Cm of $H_2O$ | $PaO_2$ | $PaCO_2$ | Tidal Volume | Respiratory Rate | PEEP |
| Respiratory (19) | Aspiration Pneumonia – 10 | 6 – 8 | 12-15 | 5-8 | 101.4 ± 1.35 | 37± 3.74 | 6.5 ± 0.707 | 13.5 ± 1.35 | 6.6± 1.34 |
| | Pulmonary Tuberculosis – 4 | 7 – 8 | 12-13 | 5-8 | 112.25 ± 16.33 | 45 ± 1 | 7.25 ± 0.5 | 13 ± 0.81 | 6.5± 1.29 |
| | Traumatic Contusion – 3 | 6 – 8 | 14-15 | 5-7 | 93 ± 5.19 | 45.33 ± 1.15 | 6.66 ± 0.57 | 14.66 ± 0.57 | 6.33 ± 1.5 |
| | COPD exacerbation – 1 | 8 | 15 | 5 | 90 | 46 | 8 | 15 | 5 |
| | Bronchopneumonia – 1 | 7 | 12 | 5 | 90 | 48 | 7 | 12 | 5 |
| Poisoning (15) | Organophosphorous Compound – 13 | 5 – 8 | 12-14 | 5-10 | 92.92 ± 17.12 | 42.07 ± 3.79 | 6.76 ± 0.72 | 13.07 ± 1.18 | 6.84 ± 1.9 |
| | Tricyclic Antidepressants – 2 | 8 | 12-13 | 5 | 76 | 45.5 ± 0.707 | 8 | 12.5 ± 0.707 | 5.51 ± 0.707 |
| Endocrine (12) | Diabetic Keto Acidosis – 3 | 5 – 8 | 12-14 | 5-8 | 77.66 ± 1.52 | 45 | 6.66 ± 1.52 | 12.66± 1.15 | 6 ± 1.73 |
| | Diabetes – 7 | 7 – 8 | 12-14 | 5-8 | 111.71 ± 1.38 | 47.57± 4.39 | 07.14±0.8 9 | 13.14 ± 1.06 | 6± 1.29 |
| | Hypertension – 2 | 7 | 12-14 | 5 | 100 | 49.5 ± 6.36 | 7 | 13 ± 1.41 | 5 |
| Infection (11) | Septic Shock – 6 | 6 – 8 | 12-14 | 5 | 102.5 ± 23.56 | 31.5 ± 1.64 | 7.16 ± 0.75 | 12.5± 0.83 | 5 |
| | MODS – 1 | 6 | 12 | 5 | 92 | 33 | 6 | 12 | 5 |
| | Malaria – 1 | 6 | 12 | 5 | 91 | 33 | 6 | 12 | 5 |
| | Dengue – 3 | 6 – 8 | 12-15 | 5 | 92.33 ± 1.15 | 38.66 ± 1.15 | 7 ± 1 | 14± 1.73 | 5 |
| Cardio-vascular (10) | MI – 7 | 6 – 8 | 13-14 | 5-10 | 92 ± 6.02 | 37.71 ± 3.72 | 6.85 ± 0.69 | 14.14 ± 1.57 | 7.57 ± 2.07 |
| | CCF – 1 | 7 | 14 | 8 | 93 | 47 | 7 | 14 | 8 |
| | IHD – 2 | 7 – 8 | 12-15 | 6-8 | 95.5 ± 4.94 | 44 ± 5.65 | 7.5 ± 0.707 | 13.5 ± 2.12 | 7 ± 1.41 |
| Neurological (10) | CVA – 6 | 6 – 7 | 12-14 | 5-7 | 82 ± 9.69 | 43.16 ± 10.55 | 6.33 ± 0.5 | 12.33 ± 0.81 | 5.33± 0.81 |
| | Head Injury – 1 | 7 | 13 | 5 | 180 | 43 | 7 | 13 | 5 |
| | Hypoxic Encephalopathy – 1 | 6 | 12 | 5 | 69 | 45 | 6 | 12 | 5 |
| | Metabolic Encephalopathy – 2 | 7 – 8 | 14 | 5 | 63.5 ± 4.94 | 45.5 ± 0.707 | 7.5 ± 0.707 | 14 | 5 |
| Renal (7) | ESRD – 5 | 7 – 8 | 12-16 | 5-8 | 84.4 ± 12.93 | 42.8 ± 3.11 | 7.4 ± 0.54 | 14 ± 1.87 | 6.4 ± 1.51 |
| | ATN – 2 | 7 – 8 | 14-16 | 5 | 82 ± 1041 | 41.5± 2.12 | 7.5± 0.707 | 15 ± 1.41 | 5 |
| GIT (5) | GI Bleed – 2 | 7 – 8 | 15 | 5 | 73 ± 7.07 | 45.5 ± 3.53 | 7.5± 0.707 | 15 | 5 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Hepatic Failure – 1 | 8 | 16 | 8 | 65 | 46 | 8 | 16 | 8 |
| | Alcoholic liver disease -1 | 7 | 14 | 5 | 90 | 49 | 7 | 14 | 5 |
| | Pancreatitis -1 | 7 | 14 | 6 | 68 | 40 | 7 | 14 | 6 |
| Others (11) | Post-Operative – 7 | 6 – 8 | 12-14 | 5-7 | 93.28 ± 3.86 | 37.28 ± 5.7 | 6.71 ± 0.755 | 12.57 ± 0.97 | 5.42 ± 0.786 |
| | Snake Bite – 3 | 6 – 7 | 12-15 | 5-6 | 98± 3.46 | 41.66 ± 3.57 | 6.66 ± 0.57 | 14± 1.73 | 5.66 ± 0.57 |
| | Disseminated Tuberculosis – 1 | 6 | 15 | 7 | 101 | 50 | 6 | 15 | 7 |

*****Admitting Diagnosis are divided based on the primary system/organ involved, however patients were having multiple organs involved and have more than one diagnosis.

**#** Initial ventilatory settings were recorded titrated based on the patient requirements and confirmed by haemodynamic stability, ventilatory tolerance and arterial blood gas analysis after 1 hour of initiation of the ventilatory support.

Admitting diagnosis is the documented cause of cardio-respiratory arrest leading to death of the patient.

These patients were intubated for various reasons like decrease in lung mechanics, airway protection, low GCS, compromised haemodynamics with multiple ionotropic and vasopressor support secondary to the admitting illness.

The mean PEEP in our study was 6.06 cm of $H_2O$ (SD ± 1.43) and the mean tidal volume was 6.9 ml/kg (SD ± 0.75 ml/kg) (Table 3).

**Table 3: Ventilator Parameters**

| | | | P value |
|---|---|---|---|
| Plateau pressure (cm of $H_2O$) | Base line | 25.46 ( SD ±4.125) | |
| | 1 hour prior to cardiac arrest | 29.11 (SD ±4.373) | < 0.00001 |
| | 30 min prior to cardiac arrest | 34.13 (SD ±3.839) | < 0.00001 |
| | 15 min prior to cardiac arrest | 40.58 (SD ±3.58) | < 0.00001 |
| | 5 min prior to cardiac arrest | 43.39 (SD ±3.572) | < 0.00001 |
| Peak airway pressure (cm of $H_2O$) | Base line | 31.97 (SD ± 4.86) | |
| | 1 hour prior to cardiac arrest | 36.98(SD ±5.01) | 0.001338 |
| | 30 min prior to cardiac arrest | 42.23 (SD ±5.04) | 0.000236 |
| | 15 min prior to cardiac arrest | 45.57 (SD ± 4.381) | < 0.00001 |
| | 5 min prior to cardiac arrest | 49.53 (SD ± 4.416) | < 0.00001 |
| Positive end expiratory pressure ( PEEP) (cm of $H_2O$) | | 6.06 (SD ± 1.43) | |
| Tidal volume (ml /kg) | | 6.9 (SD ± 0.75) | |

The average of baseline plateau pressure was 25.46(SD ± 4.125).The mean plateau pressure at 1hour prior to cardiac arrest was 29.11(SD ± 4.373) and 30min prior to arrest was 34.13(SD ± 3.839).The mean plateau pressures observed at 15min and 5 min prior to cardiac arrest were 40.58(SD ± 3.58) and 43.39(SD ± 3.572) respectively. The 'P' value as compared to baseline plateau pressure was < 0.00001 for all of them and hence statistically significant (Figure 2).

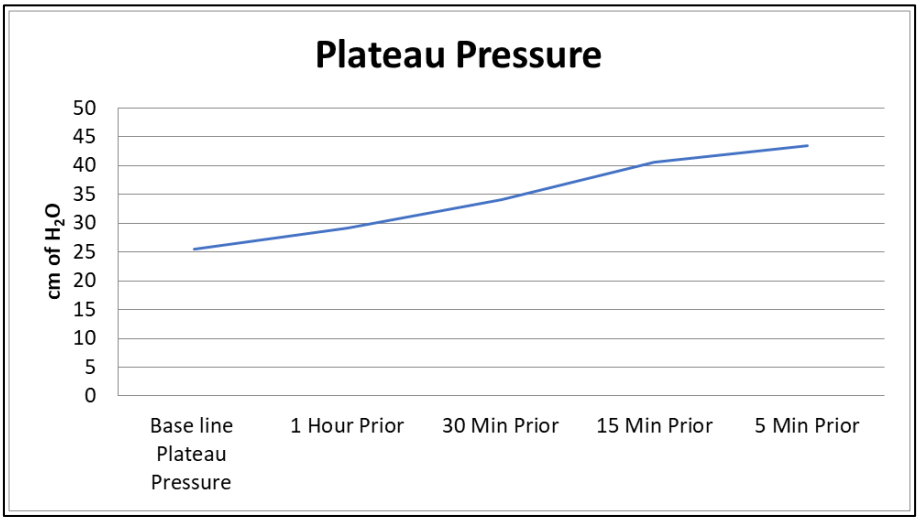


**Figure 2: Change in Plateau Pressure with respect to baseline before cardiac arrest**

The mean of baseline peak airway pressure was 31.97(SD ± 4.860). The mean peak pressure at 1hour prior to cardiac arrest was 36.98 (SD ± 5.01) and 30min prior to arrest was 42.23(SD ± 5.040). The mean peak pressures observed at 15min and 5 min prior to cardiac arrest were 45.57(SD ± 4.381) and 49.53(SD ± 4.416) respectively. The 'P' values were statistically significant for all (Figure 3).

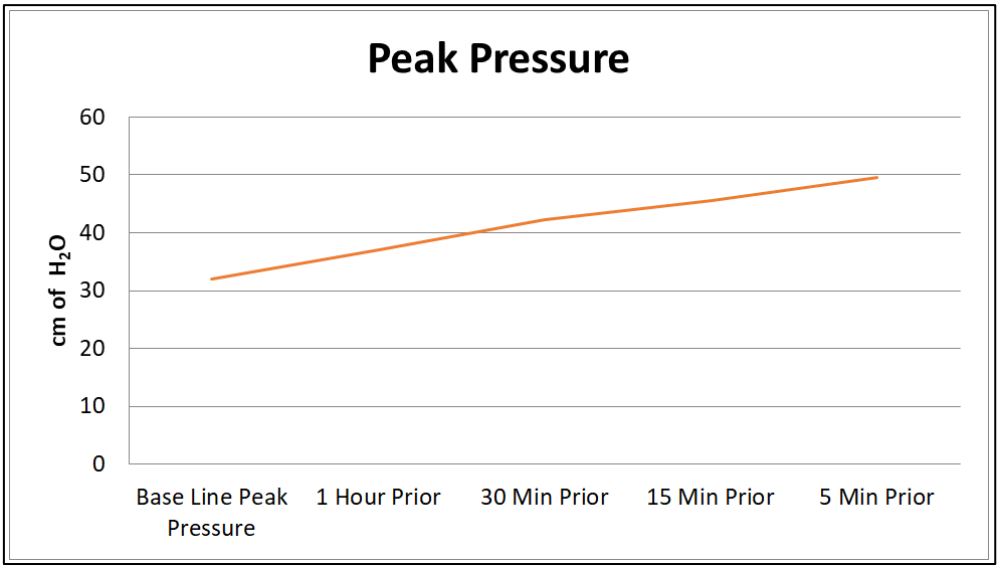


**Figure 3: Change in Peak Pressure with respect to baseline before cardiac arrest**

We also observed that this data correlated with the alarm pattern for plateau and peak airway pressure on the mechanical ventilator; that is the ventilator had given an alarm for the rise in peak and plateau pressure. The attending staff nurse confirmed the presence of ventilator alarm during that period. It was noted that in 54 patients the ventilator alarm for increase in airway pressures was muted without any intervention by attending staff nurse. Resident Doctors were informed about the ventilator alarm in 26 patients. For others we were unable to collect the correct information due to lack of coordination between the staffs.

## DISCUSSION

Plateau pressure is the pressure required to maintain inflation of lungs when there is no flow of air. It is measured during end inspiratory hold on the mechanical ventilator. It is increased when high PEEP, inspiratory flow and tidal volumes are used (Villar, J. *et al.*, 2016).

Peak airway pressure (PAP) is the maximum pressure measured during one respiratory cycle usually at the end of inspiration. It helps to deliver tidal volume by overcoming airway resistance which includes both non-elastic and elastic respiratory components. In a volume limited ventilator, the tidal volume is preset. The ventilator uses variable pressure to deliver this preset volume. High peak pressure is observed in conditions of decreased lung compliance or high airway resistance. Peak airway pressure is inversely related to lung compliance (Tremblay, L. N. *et al.*, 2005) (Dreyfuss, D. *et al.*, 1988) (Rose 2010) (Sarkar *et al.*, 2007).

Chest wall being a closed cavity, the dynamics of heart and lung are inter-related. Whenever there is decrease in myocardial contractility leading to decrease in cardiac output, the back pressure is transmitted to pulmonary vasculature. This back pressure leads to increase in pulmonary vascular resistance. This again leads to increase in transpulmonary pressure. This is independent of previous lung injury or pathology (Henderson, W. R. *et al.*, 2012) (Baedorf, K. E. *et al.*, 2016). In a failing heart, the forward pumping capacity of the ventricles is decreased. This leads to increase in preload and subsequent increase in pulmonary circulation, decreased lung compliance and hence increase in plateau and peak airway pressure (Perkins, M. W. *et al.*, 1989) (Pinsky, M. R. 1985).

Hence as per our study a persistent and continuous increase in plateau and peak airway pressure is an indicator of impending cardiac failure.

Results of our study show that monitoring peak and plateau airway pressures is important for all patients on mechanical ventilator. Data in our studies show that there is a continuous and consistent increase in peak airway pressure and plateau pressure above baseline prior to cardiac arrest. After cardiac arrest resuscitation was attempted but these patients could not be revived. We observed that there was an increase in both peak and plateau airway pressure approximately 1 hour prior to cardiac arrest and it continued to increase

as compared to baseline till the patient went into cardiac arrest.

In these patients there was no change in the set ventilator parameters like tidal volume, respiratory rate and PEEP during this time. Our results suggest that high PAP and high plateau reflect potentially injurious stress on the heart due to complex multi-dimensional factors as our patients had varied diagnosis. Fabinne D Simonis *et al.,* found variables like $P_{max}$ or $P_{plat}$, airway pressure, PEEP, tidal volume, $FiO_2$, respiratory rate, dead space fraction, compliance and blood gas analysis parameters such as in arterial blood ($PaO_2$), $PaCO_2$,pH and bicarbonates levels as predictors of mortality. They concluded that higher $P_{max}$ was a potentially modifiable factor associated with increased in in-hospital mortality in critically ill patients without ARDS. They also showed that lower airway pressures were directly linked with higher survival rate (Simonis *et al.,* 2018). In another study conducted by Sarina, K. Sahetya, *et al.,* a greater airway pressure and plateau pressure were associated with increased mortality in patients without ARDS, similar to the findings in patients with ARDS (Sahetya, *et al.,* 2019). In our study we observed that along with plateau pressure, an increase in peak airway pressure is also a useful predictor of mortality. In a study conducted by Nato *et al.,* they have shown that increase in airway pressure is associated with increased post-operative complications (Neto, A. S. *et al.,* 2016). In a study done by Schmidt, M. F. S. *et al.,* with each cm $H_2O$ increase in airway pressure higher mortality was noted (Schmidt, M. F. S. *et al.,* 2018).

All these emphasize that an increase in plateau pressure and peak airway pressure can be used in as an alarm for impending cardiac arrest and appropriate measures can be taken for the same in the best interest of the patient's outcome. We would also like stress the importance of ventilator alarms which help in alerting about the changes in ventilator parameters.

The strengths of our studies are firstly we have collected data from 100 patients who were on same company and model ventilator so that uniformity is maintained and margin of error is minimized. Secondly, we have collected data from both ARDS and Non-ARDS patients. Lastly, we validated association between plateau and peak airway pressure with mortality in our study.

The limitation of our study is that data collected includes patients with a wide range of diagnosis and ventilator strategies differ accordingly. However we found that irrespective of the strategy used the finding that increased peak and plateau pressure are useful predictors of impending cardiac arrest holds good. In our study we have not taken sedation and paralysis of patient into account. In a patient with spontaneous breathing attempts there is a change in the chest wall mechanics which may alter the airway pressures. Due this the airway pressure measurement may be imperfect , but still the continuous and consistent increase in peak and plateau airway pressure monitored at regular time intervals is significant.

## CONCLUSION

Our finding demonstrates that a continuous, consistent increase in plateau pressure and peak airway pressure above the base line values are useful ventilator parameters to predict mortality of patients on mechanical ventilation. This will also help in better management of mechanically ventilated patients with respect to early identification and intervention required to improve their outcome.

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**Conflict of Interest:** No competing interests.

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