Applying Transpulmonary Pressure in the ICU

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Mechanical ventilation in the intensive care unit (ICU) is usually guided by arterial blood gases, and the parameters used to maintain these blood gases are limited by standards for lung protective ventilation (The Acute Respiratory Distress Syndrome Network, 2000). Airway pressures and tidal volume are minimised for lung protection despite evidence that they may be inadequate surrogates for lung stress and strain (Chiumello et al. 2008). Transpulmonary pressure represents true lung pressure, and physiologically is ≥ 0 cmH2O at end exhalation. Transpulmonary pressure < 0 cmH2O results in a lower functional residual capacity (FRC), lower compliance, and airways are prone to collapse on exhalation (Behazin et al. 2010). Our respiratory therapists hypothesised that a patient admitted to St. Joseph’s Healthcare ICU from an external facility was being ventilated with insufficient positive end-expiratory pressure (PEEP), causing a negative transpulmonary pressure. Here we describe the case, and the outcome.

Patient Case Overview

A 65-year old morbidly obese male (BMI 55.5 kg/m2) with obesity hypoventilation and severe COPD was intubated for respiratory failure, secondary to pneumonia. He received a tracheostomy after 10 days of ventilation and was transferred on the 22nd day of invasive mechanical ventilation to St. Joseph’s Healthcare facility to utilise our bariatric CT scanner to rule out abdominal sepsis.

The patient was sedated and apneic on arrival with ventilation settings as follows:
• Positive end-expiratory pressure (PEEP) of 10 cmH2O;
• Pressure assist control of 20 cm H2O above PEEP;
• Respiratory rate of 20 bpm;
• Inspiratory time of 1.0 seconds;
• FiO2 of 1.0;
• Initial blood gas analysis was pH 7.16 PaCO2 50 mmHg PaO2 215 mmHg HCO3 17 mmHg SaO2 0.99, and
• Haemodynamically, the patient was hypotensive and required norepinephrine to maintain an acceptable blood pressure.

Initial Patient Management

The first 12 hours of ventilation at our facility were challenging. The patient’s ventilation requirements had increased to the following:
• Pressure assist control of 32 cmH2O above PEEP;
• Respiratory rate was 30 bpm;
• Inspiratory time of 1.0 seconds;
• PEEP of 12 cmH2O and FiO2 of 0.5, and
• Blood gas analysis on these settings was pH 7.17 PaCO2 47 mmHg PaO2 63 mmHg HCO3 16 mmHg SaO2 0.9.
An oesophageal balloon was inserted into the patient to determine transpulmonary pressure (Ptp). It was suspected that the patient might not have had the PEEP level needed to achieve a normal end-expiratory transpulmonary pressure (PtpPEEP ≥ 0 cmH2O). The oesophageal balloon catheter was inserted to a depth of 60 cm and gentle compression of the abdomen was done to confirm placement. The catheter was then pulled back 40 cm after which cardiac oscillations were observed to be present, and the waveform clearly different than previously. With the patient sedated and paralysed using a neuromuscular blockade, an expiratory hold was done to obtain a stable transpulmonary reading. The resulting Ptp value was −12 cmH2O. To achieve a transpulmonary pressure close to what would be physiologically normal (Ptp ≥ 0 cmH2O), the PEEP was increased from 12 cmH2O to 24 cmH2O.

**Patient Response**

The 48-hour trend of PaO2/FiO2, respiratory system compliance and peak airway pressure are shown. The blood pH improved as a result of HCO3 increasing to a normal level. The respiratory rate and peak airway decreased significantly despite the CO 2 level remaining 45 – 47 mmHg over 48 hours. The PaO2/FiO2 ratio increased significantly from 126 to 370 as PEEP was titrated, according to Ptp, from an initial increase to 24 cmH2O, and then to 18 cmH2O 48 hours later.

**Improved Outcome**

The use of oesophageal pressure manometry to determine Ptp and set PEEP in this patient resulted in an individualised lung protective strategy. The end result was improved oxygenation, improved ventilation (lower minute ventilation required), improved respiratory system compliance and peak airway pressure below the limitations recommended by literature. The patient was returned to the sending facility two days later with a PEEP of 18 cmH2O, FiO2 of 0.30, PC of 12 cmH2O and a respiratory rate set at 22 bpm.

**Supporting Research**

A study by Behazin et al. found that obese patients have higher pleural pressures than nonobese patients when sedated and paralysed for surgery (Behazin et al. 2010). This causes tidal breathing to occur at a lower FRC, lungs are less compliant and airways are prone to collapse during exhalation. It was also concluded that the pleural pressures were variable, and not predictable by BMI, making the measurement of Pes and Ptp a valuable clinical tool. The level of PEEP required to maintain a Ptp > 0 in this sedated patient was slightly higher than the normal range for surgical patients with BMI levels > 38. The cause of this patient’s elevated pleural pressure may have been due to his fluid requirements secondary to hypotension caused by his sepsis. In my experience, I have seen clinicians be less concerned with elevated PIP in obese patients assuming that the size of the patient implies that they don’t “feel” the pressure. This study helps demonstrate that when PEEP is set optimally, high PIP may not be necessary.

**Benefits To Measuring Transpulmonary Pressure**

The benefit to measuring Ptp is having the possibility to understand the patient’s true lung mechanics. Airway plateau pressure has become a standard in lung protective ventilation, but it is only one part of the Ptp equation (airway pressure – pleural pressure = Ptp). This concern over airway pressure is that pleural pressures are variable and unpredictable. These differences are not distinguished by airway pressure alone. Therefore, the level of PEEP applied in a patient may be insufficient, or excessive. PEEP that is insufficient leads to a negative Ptp and would lead to atelectrauma; PEEP that is excessive can cause a positive Ptp that may lead to overdistension during tidal volume delivery. The benefit of knowing true lung mechanics is extremely valuable in ARDS patients.

Patients with primary (pulmonary) or secondary (extra-pulmonary) causes of ARDS can be easily distinguishable. Once optimal PEEP is set (Ptp of 0 cm H2O) patients with very stiff or non-recruitable lungs (pulmonary ARDS) may have high Ptp Plateau pressures. Monitoring and making attempts to minimise pressures in these patients becomes very important (keeping PtpPlateau < 20 cm H2O) (Chiumello et al. 2008). When patients with optimal PEEP (regardless of the level) ventilate with acceptable PtpPlateau pressures (< 20 cm H2O), they most likely have recruitable lungs and are being ventilated within safe limits. Airway plateau pressures in these patients are not a concern. Therefore, by measuring Ptp the clinician is able to individually target the needs of the patient with ARDS.

There has been some hesitation to use Ptp measurements due to the potential inaccuracy of the measurement (Pes is only an estimate of pleural pressure in one area of the lungs). However, pleural pressures are so variable among patients it seems that the potential risk of an inaccurate Ptp measurement for a patient is much less than the risk of using a generalised approach for setting PEEP and limiting plateau pressure with multiple patients presenting with a variety of illnesses. The measurement of Ptp is much more representative of individual respiratory mechanics than airway pressure alone.

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