PLATEAU PRESSURE, (PPLAT) : A CLINICAL BRIEF

by David Wheeler RRT, NPS

The intention of this column is to briefly discuss the critically important concept of Plateau Pressure, (Pplat). Pplat is the most consequential of bedside parameters for the mechanically ventilated patient. This discussion must begin with an examination of Pplat in the abstract before we can comprehend fully, the considerable clinical utility of this measurement.

Plateau Pressure is the pressure measured at the end of inspiration during an inflation hold in volume targeted ventilation or the pressure at the end of a fully equilibrated breath when pressure cycling is employed. One must bear in mind that in time cycled pressure limited ventilation, the Pplat is equal to the pressure threshold only when a sufficient number, (4-5) of inspiratory time constants are fulfilled. In all methods of ventilation the inflation hold must be sufficient to allow inspired gas to equilibrate in regions of the lung with incongruous time constants.

Pplat is the pressure required to counterbalance the end inspiratory forces of the lung and is related to the static end inspiratory elastic recoil pressure of the total respiratory system. Airway pressure measured during an end inspiratory occlusion replicates the elastic threshold stress to the pulmonary system sans the comprehensible resistive forces present during active inspiration. Pplat faithfully approximates alveolar pressure and as such is an indispensable assessment tool for the mindful clinician.

Elevated Pplat will alert the clinician to increased alveolar pressure. Incremental changes in Pplat are inversely related to lung compliance. An increase in the plateau pressure signals a fall in the global lung compliance whereas a decrease in Pplat will signal an increase in global lung compliance.

In the context of global lung assessment it is important that we remain cognizant of the reality that variations in Pplat may be present throughout the lung field. These regional variations will be subject to regional variants in resistance, compliance and the pulmonary time constant. Although we must be aware in a conceptual sense of the very regional nature of Pplat, we must treat the lung globally and fashion a ventilation strategy aimed toward producing the lowest possible Pplat.

Indeed, it has been noted elsewhere, that a Pplat of 35 cmH2O represents the normal peak alveolar pressure necessary to reach total lung capacity, (TLC). It has been suggested that a Pplat equal to, or in excess of that needed to reach TLC would exaggerate the potential for lung injury and greatly impede efforts to ventilate the tragically hyper-inflated lung.

Plateau pressure is needed to calculate total lung compliance vis a vie the relationship between Pplat and delivered volume. This measure of what is commonly referred to as static compliance is derived in the following manner; Cl = Vt / (Pplat - PEEPtot).

Common causes of decreased compliance in the mechanically ventilated patient include; main-stem intubation, pneumothorax, ALI, CHF, ARDS, reperfusion injury, pleural effusion, and chest wall deformity.

The difference between Peak Airway Pressure and Pplat is a function of the resistive forces in the patient ventilator system. Raw is calculated by looking at the pressure gradient between the peak airway pressure and Pplat divided by the flow. In this way pressure, time and volume relationships are defined for the bedside clinician. Resistance is calculated at the bedside thusly; Raw = PAP - Pplat / Flow (L/sec.).

Common causes of increased Raw include; bronchospasm, bronchoconstriction, secretions, airway obstruction, narrow endotracheal tube and mucosal edema. Bear in mind that both inspiratory and expiratory Raw may vary widely in different pathologies. Assessments remain highly contextualized.

The concept of a stable Pplat is also context driven in the notion of viscoelastance. Viscoelastance is the character of lung mechanic observed during the pressure changes commensurate with the phase change from the termination of inspiratory flow, (and attendant mechanical bronchodilation), in the airways to the equilibration of volume in lung regions with highly individualized time constants. This equilibration of regional lung units is necessary to create a stable Pplat. A decaying pressure profile is reflective of erratic; non-homogenous regional time constants and tissue viscance. Therefore, increased pressure gradients reveal greater lung-tissue viscoelastance and greater potential for lung injury.

Indeed, one must remain attentive to the total patient. Some authors would put forth that Pplat is an inaccurate surrogate for lung distension and that end-inspiratory transpulmonary pressure provides physiologically more relevant information than approximations gleaned from Pplat. This may be true to a

continued on next page

degree however, I must stress that the only context that would negate the clinical efficacy of Pplat is in cases where extremely high abdominal pressures distort lung elastance and the subsequent assessment of Pplat.

Pplat has a great clinical utility in the application and titration of PEEP. Clearly in pathophysiological states where there is low potential for lung recruitment, as demonstrated by relentless and fixed elevated Pplat, PEEP cannot create alveolar recruitment where there is very little lung with "potential for recruitment". In this patient population the use of PEEP may amplify ventilator induced lung injury by enhancing pulmonary overdistention. Thus, the use of greater PEEP levels in these patients may impair the desired clinical outcome. The lack of potential for alveolar recruitment is demonstrated by the fixed high Pplat. One must know that patients with a greater percentage of potential for lung recruitment will have more severe primary lung injury. The effect of PEEP on lung recruitment is coupled with the percentage of potentially recruitable lung and the percentage of potentially recruitable lung is decidedly linked with the overall severity of lung injury.

Conversely, when one demonstrates a decrease in Pplat with the application of greater levels of PEEP then one must assume a correlating increase in the end-expiratory lung volume and alveolar recruitment without alveolar overdistention. The mindful clinician must titrate PEEP levels in an assessment based evidence driven fashion. The frequent assessment of Pplat is essential for titrating PEEP levels to lung mechanics. The step-changes in compliance that can be demonstrated with applied PEEP indicate greater potential for alveolar recruitment. The notion is to apply PEEP early and create futher opportunities for lung stabilization and recruitment. The existence of PEEPi is a powerful variable in patient respiratory system mechanics as the presence of PEEPi creates both increased peak airway pressure and end-inspiratory Pplat.

Plateau pressure must be maintained at the lowest levels possible whilst maintaining adequate ventilation. Maintaining a minimal pH of 7.30 is a reasonable ventilation goal. Patients ventilated with low tidal volume ventilation with a mean tidal volume 7.6 mL/kg; mean Pplat of 24.6 cm H2O and mean PEEP 14.8 cm H2O demonstrated a relative reduction in inflammatory broncho-alveolar lavage and peripheral blood cytokines. Lower assessments of Pplat, (≤25 cm H2O) have been linked to dramatic reductions in mortality.

The beneficial effect of a ventilation strategy that lowers both tidal volume and Pplat appears to be independent of ARDS etiology. The focus for the informed clinician must remain the prevention of VALI and VALI. It is essential to comprehend that alveolar pressure alone does not provide a measure of alveolar distension. One must be clear that it is the absolute transpulmonary pressure (alveolar pressure minus pleural pressure), that engenders lung injury.

It is now known that mortality is reduced in direct proportion to the decrease in Pplat, with a slope of 0.015. Thus, reducing Pplat from 35 to 25 cm H2O may yields a mortality reduction of 15%. Pplat is an indispensable assessment tool in the judicious development of patient focused, physiologically sound, evidence based ventilation strategies. The informed clinician will understand that mechanical ventilation strategies based on patient-centered assessments gain increased relevance as we fully comprehend both VALI and VILI. Therefore, it is an essential component of compassionate care that clinicians customize ventilator settings to patient specific physiologic changes.

David Wheeler, RRT-NPS is the Educational Coordinator for the Cardio-Thoracic Anesthesia and Respiratory Therapy Departments at the Cleveland Clinic. He can be reached at wheeled@ccf.org.