

THE LANGUAGE OF LUNG INJURY: A BRIEF PRIMER

by David Wheeler RRT, NPS



The concept of lung injury is hardly a new one. The injured lung finds applicable reference in texts as wide ranging and ancient as the Bible, The Tibetan Book of the Dead and the Bhagavad-Gita. The well-versed clinician should be able to demonstrate a working knowledge of the spectrum of lung injury in their daily practice thus, it is the intention of this column to inform the bedside clinician by articulating the current "language" and nomenclature of lung injury.

Acute Respiratory Distress Syndrome, (ARDS) - A syndrome characterized by bilateral infiltrates on radiological exam, a PaO₂ / FiO₂ ratio of 200 or less, (300 or less ALI.), and no clinical evidence of elevated left atrial pressure. ARDS is rapid in presentation developing in 4-36 hours. ARDS occurs in roughly 7-20% of mechanically ventilated patients. ARDS is the syndrome at the extreme end of the spectrum of Acute Lung Injury, (ALI).

Acute Lung Injury, (ALI) - A syndrome characterized by bilateral infiltrates on radiological exam, a PaO₂ / FiO₂ ratio of 300 or less, and no clinical evidence of elevated left atrial pressure.

Ventilator Induced Lung Injury – VILI; a spectrum of lung injury caused by the mechanical ventilator. VILI is a complex collection of lung deconstructive elements that manifests itself in a number of forms including **Volutrauma, Barotrauma, Biotrauma, RACE** and **Atelectrauma**. Lung distention and alveolar stretch are the principal elements in the establishment of VILI.

Atelectrauma – This form of lung injury may be caused by low volume ventilation without FRC support where the repetitive

opening and closing of lung units creates and enhances shearing forces that tear individual lung units. These affected alveolar units tend to collapse and become fluid filled. The resulting unexpanded lung regions are then surrounded by expanded lung areas which experience a local amplification of distending pressures and greater injury. The critical variable is volume and overdistension of adjacent lung units that produces the increased "lung water". This may be thought of as a form of "Stress Failure". With circumferential, surface and longitudinal pressure gradients having a role in the destruction of lung units. PEEP above the inflection point may have a role in prevention.

Barotrauma – A gross form of lung injury caused by excessive airway pressure. The mechanically produced pressure gradient between alveoli and bronchovesicular sheaths may result in changes in airway structure and/or function. This fluctuating pressure gradient need only be transiently increased. This injury is characterized by air in interstitial tissues. *Transpulmonary pressure, not PAP, causes this trauma.*

Biotrauma – A cell and inflammatory mediator-based form of Ventilator Induced Lung injury, the emphasis is on biological mechanisms of the universal inflammatory response. Neutrophil released mediators are thought to be catalysts for lung destruction. It may have a synergistic effect with high end inspiratory stretch injury.

Increase in systemic cytokines - Strong evidence suggests that it may be a result of large tidal volumes with no PEEP; Cytokines from the lung epithelium may be responsible for the inflammatory cascade. Alveolar stretch may be the cause for the introduction of bacteria into systemic circulation and bacterial sepsis.

Dynamic hyperinflation - the augmentation of lung volume above the normal hysteresis pattern. Frequently due to trapped volume from *prolonged expiratory time constants*. The retained volume that prevents the lung from returning to normal resting FRC and increases both end expiratory pressures and volumes.

The PaO₂/FiO₂ index (P/F ratio) - quantifies the ratio of arterial oxygen tension to available oxygen concentration. It is a very useful formula in evaluating the degree of intrapulmonary shunt and subsequent compromise of cardiopulmonary function. The PaO₂/FiO₂ index is also valuable indicie of diffusion capability and a primary tool in assessing the degree of injury to the lung. Low diffusion states will have a low ratio of arterial oxygen in relation to a given FiO₂. The PaO₂/FiO₂ index acts to identify the severity of lung injury,

If the PaO₂/FiO₂ index is < **300** one should strongly suspect Acute Lung Injury. (ALI)

If the PaO₂/FiO₂ index is < **200** one should strongly suspect Acute Respiratory Distress Syndrome. (ARDS)

The PaO₂/FiO₂ Ratio quantifies the ratio of oxygen available vs. arterial oxygen required. It is an excellent predictor of mortality and is an essential tool in assessing lung injury.

Plateau Pressure - (PPLAT) PPLAT is the pressure measured at the end of inspiration during an inflation hold. This inflation hold allows inspired gas to equilibrate in regions of the lung with incongruous time constants. PPLAT is the pressure required to

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Reabsorption along Venule End of Systemic Capillary

The extent of the reabsorption that occurs along the venule end of the capillary can also be calculated. What needs to be taken in consideration at the venule end is that fluid has filtered out of the vasculature at the arteriole end, thereby altering the CHP and COP. The CHP decreases to about 10 torr because fluid left at the arteriole end. The loss of fluid at the arteriole end makes the proteins in the plasma more concentrated at the venule end. The COP increases slightly to around 28 torr. The IHP and IOP remain the same because this fluid compartment is so vast that the filtered fluid essentially has no impact on these two pressures.

The hydrostatic pressure gradient along the venule end is calculated by subtracting the IHP from the CHP.

$$\text{CHP} - \text{IHP} = \text{hydrostatic pressure gradient at venule end} \\ 10 \text{ torr} - (-3 \text{ torr}) = 13 \text{ torr}$$

The osmotic pressure gradient along the venule end is determined again by subtracting the IOP from the COP.

$$\text{COP} - \text{IOP} = \text{osmotic pressure gradient at the venule end} \\ 28 \text{ torr} - 4 \text{ torr} = 24 \text{ torr}$$

Subtracting the osmotic pressure gradient from the hydrostatic pressure gradient provides the reabsorption pressure at the venule end of the systemic capillary.

$$\text{hydrostatic pressure gradient} - \text{osmotic pressure gradient} = \text{reabsorption pressure} \\ 13 \text{ torr} - 24 \text{ torr} = -11 \text{ torr}$$

The negative sign does not indicate subatmospheric pressure. Instead, it represents the direction of the fluid movement at the

venule end of the capillary. This direction is opposite that at the arteriole end, and indicates that reabsorption predominates here.

Filtration-Reabsorption Pressure Difference

The net magnitude of the fluid movement along the systemic capillary can be obtained by subtracting the reabsorption pressure of 11 torr from the filtration pressure of 12 torr

$$12 \text{ torr} - 11 \text{ torr} = 1 \text{ torr}$$

A net movement of 1 torr has left the vasculature during the blood's passage through the systemic capillary. Fluid does not accumulate within the interstitium. If so, edema would develop.

What happens to this filtered fluid? Does it merely continue to accumulate in the interstitium? No, lymph vessels, beginning as blind microscopic end-pouches in the outreaches of the interstitium, prevent the accumulation of this transudated fluid. The lymphatic system continuously siphons fluid away from the interstitium and deposits it ultimately into general circulation.

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counterbalance end inspiratory forces 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 inevitable resistive forces present during active inspiration. PPLAT faithfully approximates alveolar pressure and as such is a very useful clinical assessment tool.

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. Indeed, a PPLAT of 35 cmH₂O represents the normal peak alveolar pressure necessary to reach TLC. It has been suggested that PPLAT equal to, or in excess of that needed to reach TLC would facilitate lung injury or impede efforts to ventilate the already hyper-inflated lung. Plateau pressure is needed to calculate total lung compliance as the relationship between PPLAT and delivered volume. This lung and chest wall compliance is derived in the following manner.

$$CI = Vt / (\text{PPLAT} - \text{PEEP}_{\text{tot}})$$

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

The difference between Peak Airway Pressure and PPLAT is a function of resistive forces in the patient ventilator system. Raw is calculated by looking at the pressure gradient between the peak airway pressure and the plateau divided by the flow.

$$\text{Raw} = \text{PAP} - \text{PPLAT} / \text{Flow (L/sec.)}$$

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.

Repetitive Alveolar Collapse & Expansion – RACE; This form of VILI is described as a product of alveolar instability that is non-homogenous. The inflation deflation pattern in the acutely injured lung causes this form of VILI. Healthy alveoli with stable

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properties adjacent to very unstable alveoli collapse and or are injured as a result of sheer stress and incongruent opening and closing times. Non-homogenous time constants result in a rocking or tearing stress motion. Altered alveolar mechanics render inflection point measurements suspect and perhaps even useless.

Ventilator Associated Pneumonia, (VAP) - Manifests itself more that 48-72 hours post intubation. The intubated patient has an increased risk of VAP 6-21x greater than the non-intubated patient. The chances for VAP increase 3% per day for the first 5 days of ventilation, 2% per day at 5 to 10 days and 1% for every day after day ten. It is estimated that half of all VAP occurs within the first four days of mechanical ventilation. It has also been stated that VAP may be attributed to an increased mortality in the range of 33-50%. Indeed, it is again emphasized that the best way to avoid VAP is to aggressively avoid intubation and mechanical ventilation. If the patient *must* be intubated every possible strategy must be employed to craft a rapid extubation stratagem. Certain key behaviors connected spatially and chronologically at the patient's bedside have a significant effect on the prevention of VAP. Elevating the head of the bed greater than 30 degrees, DVT prophylaxis, PUD prophylaxis, daily sedation vacations, daily spontaneous breathing trials, aggressive mouth care and uncompromising weaning plans have a dramatic effect in preventing VAP.

Volutrauma – This is a form of VILI where large delivered tidal volumes overdistend alveoli with regionally differing time constants. These intimate lung units experience volume stress to alveolar capillary walls and significant intratidal sheer forces. This augments increased alveolar-capillary permeability, alveolar destabilization and loss of surfactant. The overdistension leads to inflammation, increase vascular permeability and loss of surfactant production. This excess stress is more prevalent in instances of high tidal volume ventilation without PEEP.

I believe it was Victor Hugo who said, "The human intellect is always on the march and with it; language is in constant movement." This is especially true in the discipline of critical care. The definitions in this column are changing as you read them. As our understanding of the mechanisms of lung injury develops, the language of VILI will evolve as well.

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"Mr. Anderson, I've always had trouble dealing with authority figures... Would you mind if I started calling you Skip?"

ity. To minimize possible confusion of clinicians by the new computerized PFT interpretation, the new computerized interpretation was withheld from the reports transmitted to the clinicians during this PI project. After one month of data collection, the percent agreements in each of the categories were tallied for all the patients. An agreement percentage above 90% in each category was deemed acceptable agreement. A Table was constructed with the categories and the agreement percentage. A statistical p value less than 0.05, derived using a non-parametric test for data that can only be "Disagreement" or "Agreement" meant there was only one chance in 20 that the percentage agreement was due only to chance.

Results are a summary and analyses of data. For this project, seventy-eight patients were studied in the month of interest. The agreement in each category was: restriction = 96%, obstruction = 100%, combined defect = 92%, bronchodilator responsiveness = 100%, flow volume loop defect = 100%, air trapping = 95% and diffusion capacity = 92%. This meant there was acceptable agreement, with all categories above 90%, with a p value statistically significant at 0.037.

The Conclusion is the computerized PFT interpretation using the new equipment agrees with the physician interpretation. Therefore, for this project, the Hypothesis was supported, corresponding to a "yes" answer to the Question. (Note: When writing the Conclusion, the Hypothesis must be addressed whether it was supported or not.)

Reflections offer an opportunity to critique the project by suggesting possible modifications that could improve the quality of future research. For example, comparison of the computerized interpretation performed by both the prior software and the new equipment software could have provided a better comparison than using the physician's interpretation. More categories for "Disagreement" versus "Agreement" could be added. Lastly, more than one physician could provide an independent interpretation so that an interobserver variability could be calculated. Reflections can also include a comparison with other similar research projects.

Clinical Implications explain the relationship between the research project and the clinical world. For this project, the new computerized PFT interpretation was validated and the preliminary PFT interpretation could be trusted.

Future Research follows the theme that completed research should lead to new research. For example, this project led to validation of the computerized interpretations using a method that could be applied to any new PFT equipment purchase, such as exercise or sleep equipment.

Bibliography lists references from similar research in journals or books and should also include the reference for the statistical methods.

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Conflicts of Interest (COI) list any COI for each person involved in the project. COI includes being a member of a speaker's bureau, being a consultant, owning stock, receiving services or receiving gifts from companies related to the project.

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