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Refractory Hypoxemia? Is Positive End Expiratory Pressure Always the Answer?

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A patient presents with a witnessed aspiration event during an outpatient endoscopy while in the left lateral recumbent position. They are referred to the ED for evaluation. A chest X-ray (Figure 1) showed unilateral left-sided opacities. They have progressive hypoxemia and respiratory distress over the next hour and require intubation. The fraction of inspired oxygen (FiO_2) is set at 100 percent with an initial positive end-expiratory pressure (PEEP) of 10cm H_2O to maintain an oxygen saturation of 89 percent. The emergency physician increases the PEEP to 18cm H_2O based on the PEEP/FiO_2 table established for acute respiratory distress syndrome (ARDS).[1] As the PEEP is titrated beyond 10cm H_2O, the hypoxemia worsens, and the oxygen saturation is now 72 percent.

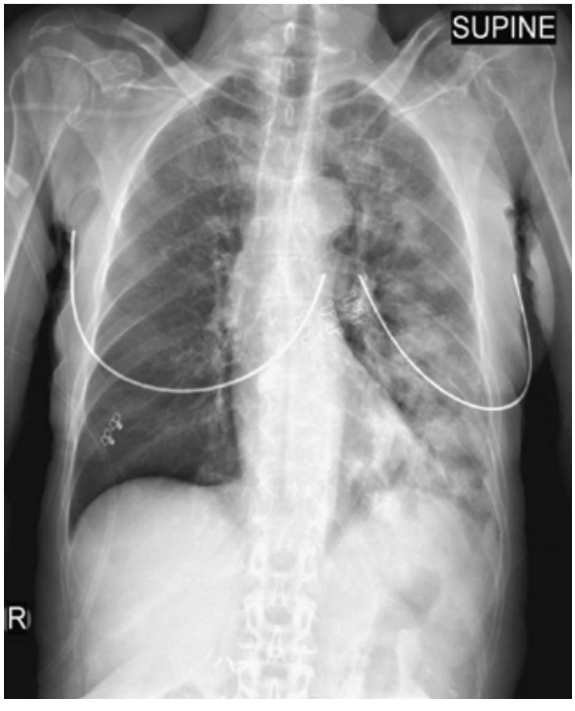

Figure 1. The chest X-ray demonstrates unilateral infiltrates of the left lung. (Images courtesy of Yaïr Glick, Radiopaedia.org, rID: 53647)

How PEEP Improves Oxygenation in Diffuse Processes (ARDS)

ARDS is a diffuse infiltrative process that affects the alveoli by disrupting the surfactant responsible for increasing surface tension and maintaining alveoli in their open state. As the alveoli become increasingly permeable, they develop edema that causes impaired gas exchange and ultimately collapse. These compromised alveoli require greater pressures to recruit and, as a result, can remain closed throughout a normal respiratory cycle. Hypoxemia develops as blood passes through intra-alveolar capillaries of unventilated lung parenchyma, causing a right to left intrapulmonary shunt.[2,3]

PEEP can be a helpful intervention to improve oxygenation by recruiting previously collapsed alveoli and increasing lung volumes. PEEP works to increase the lung's functional residual capacity by applying a constant pressure to the alveoli throughout the entirety of the respiratory cycle, preventing alveolar collapse. Maintaining alveoli in their open state facilitates more efficient gas exchange by allowing each inhalation to start on the steeper, more compliant part of the lung's pressure-volume curve, optimizing both oxygenation and ventilation.[3,4]

Utilizing PEEP, it may be possible to recruit collapsed or atelectatic alveoli to participate in gas exchange, allowing for increased ventilation, improved ventilation and perfusion matching, and decreasing shunt physiology. This intervention works well in diffuse lung processes such as ARDS

that involve larger, more homogenous areas of recruitable lung. However, high PEEP titration may not be beneficial when employed in focal lung processes such as pneumonia.

How PEEP May Worsen Gas Exchange

In focal infiltrative pathologies such as lobar pneumonia or unilateral aspiration pneumonia, an increase in PEEP can compromise the properly functioning alveoli disproportionately more than it recruits the collapsed alveoli, resulting in worsening hypoxemia. Because PEEP cannot be selectively applied to pathologic lung, the whole pulmonary system is subjected to the same end-expiratory pressure. The healthy, highly compliant lung reacts much differently than the poorly compliant diseased lung tissue, and can cause iatrogenic lung injury or worsen gas-exchange.

Poorly compliant, atelectatic lung requires greater pressures to facilitate adequate gas-exchange compared to healthy tissue. In contrast, healthy lung parenchyma is more compliant and responds to a smaller change in pressure with comparatively greater increases in lung volume. As PEEP is titrated in an effort to recruit injured alveoli, overdistention of the healthy, compliant tissue can occur. As these healthy alveoli begin to over distend, the surrounding capillaries are stretched and narrowed, resulting in a mechanical impedance of the capillary circulatory flow through intra-alveolar capillaries. This blood flow (i.e, perfusion) that was previously shunted towards the healthy lung, via compensatory hypoxic vasoconstriction of the poorly ventilated alveoli, is consequently diverted away from these now iatrogenically highly resistant capillary beds. This worsens ventilation and perfusion mismatch (Figure 2).[3] As a result, the poorly oxygenated collapsed alveoli receive a higher percentage of capillary blood flow, paradoxically worsening shunt physiology and hypoxemia.

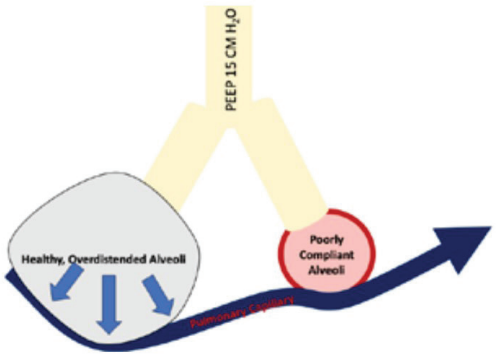

Figure 2. The effect of PEEP on a healthy and injured alveolar capillary unit. The blood is shunted to the injured alveolus if the healthy alveoli is over distended, potentially worsening shunt and gas exchange.

In addition to unintentionally worsening shunt physiology through increasing pulmonary capillary resistance, positive pressure in the thoracic cavity can worsen hypoxemia by decreasing cardiac output. PEEP increases pressures in the thoracic cavity and consequently may impair venous return to the heart. This loss of venous return will decrease the cardiac preload and subsequently the cardiac output, further worsening hypoxemia.[5]

Conclusion

The application of higher PEEP can be either beneficial or detrimental depending on the clinical scenario. Diffuse processes, such as ARDS, typically respond favorably to higher levels of PEEP through alveolar recruitment of diseased lung. Conversely, in focal processes, higher PEEP may worsen gas exchange through iatrogenic right to left intrapulmonary shunt and impaired cardiac preload. The emergency clinician must be ready to apply this physiology and apply PEEP expertly in their next intubated patient.

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References

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