# B1.B.TO OPEN OR NOT TO OPEN THE INJURED LUNG? MAYBE, THIS IS NOT THE RIGHT QUESTION!

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Since 1967, when Ashbaugh and Petty [1] described Acute Respiratory Distress Syndrome (ARDS) in intensive care (ITU) patients who shared the common features of tachypnea and hypoxemia along with opacification on chest x-rays and poor lung compliance secondary to different underlying causes, the syndrome has been extensively studied and defined. The current definition, known as "Berlin definition", was developed by a consensus panel of experts in an attempt to provide a measurable spectrum of severity and improve clinicians' ability to recognize patients with ARDS. [2]

Despite the changes that were implemented, the "Berlin definition" failed to include underlying pathology and ARDS remains an "umbrella" that encompasses a broad range of causative conditions, both intrapulmonary and extrapulmonary, such as direct chest trauma and pancreatitis. The common diagnosis may facilitate research in terms of diagnostics, mechanisms and interventions but at the same time implies that the cause is immaterial; the risk is, once the "diagnosis" is made, we may assume that supportive measures are also therapeutic and focus on how to fine tune them to make them more efficacious but neglect the importance of controlling the underlying condition in order to increase patients' survivability.

This fundamental flaw seems to be particularly true in the case of mechanical ventilation (MV) in ARDS patients. Throughout the years, it has been recognized that MV can worsen lung injury and, as a consequence, an open lung approach (OLA) strategy, proposed initially by Lachmann [3], has been adopted as the mainstay ventilation strategy in this population. From a physiological standpoint, it seems appealing to try to minimize ventilator induced lung injury by opening the deflated regions of the lung and preventing repetitive opening/closing of alveoli. We assumed though, that less detrimental effects should automatically translate to a therapeutic benefit and that this, relatively simple, intervention would work for every patient with ARDS despite their heterogeneous, complex pathophysiology and the relatively consistent failure of randomized controlled trials to demonstrate any mortality benefit. In the past, at least 3 RCT's (ALVEOLI [4], LOVS [5], EXPRESS [6]) were mostly unsuccessful in improving mortality of ARDS patients with the use of higher PEEP and recruitment manoeuvres (RM). In the ART trial [7], which was published last year, mortality was increased in moderate to severe ARDS patients who were ventilated with an OLA strategy when compared to a standard (ARDSnet protocol) "protective" mechanical ventilation strategy. Additionally, OSCILLATE and OSCAR trials that studied high frequency oscillation ventilation (HFOV), a ventilation mode based on the OLA concept, showed either no benefit or even a harmful effect in adult ARDS patients despite better lung mechanics and less hypoxemia.

These results may indicate that the consequences from the opening/closure of the injured lung may not be so important or that the applied PEEP may be inadequate to

prevent repetitive opening and closure in the population under study. Indeed, Gattinoni et al. have identified that recruitability is extremely variable in ARDS patients and that PEEP values proposed by most clinical guidelines fail to keep the lung open [10]. A recent study by Cressoni et al. [11], in patients with early ARDS, found that, at the generally accepted "safe" airway pressure of 30 cmH2O, a 10–30% of the potentially recruitable lung tissue remains always closed in patients with moderate and severe ARDS; with the use of CT scan, this study also confirmed the previous findings of Gattinoni that ventilating a patient with a tidal volume of 6–8 ml/kg and 15 cmH2O of PEEP is largely insufficient to prevent cyclic lung tissue opening and closing; finally, it demonstrated that increasing PEEP decreased inhomogeneity only by 3–4% of the total lung volume in mild and moderate ARDS and failed to modify lung heterogeneity in the patients with severe ARDS.

Where should we stand as clinicians? Should we continue applying an OLA to all ARDS patients who require mechanical ventilation or is it time to start exploring other ventilation strategies like "permissive atelectasis" [12]? Maybe, before anything else, it's more imperative to realize that mechanical ventilation is just a supportive tool which can have opposite effects in different individuals; hence, a universally accepted, 100% safe ventilation strategy doesn't exist and implementation of mechanical ventilation should be tailored to each patient, guided by their pathophysiology, hemodynamics, lung mechanics and recruitability.

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