Clinical Application of Lung and Diaphragm Protective Ventilation

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Educational background

2002-2006	Ph.D., Physiology and Physiopathology
2000-2002	Research Fellowship, McGill University, Meakins and Christie Laboratories, Montreal, Canada
1994-2000	Residency, Pulmonology and Critical Care, Paris, Included a M.A. in Physiology
1988-1994	M.D., Université René Descartes, Paris

Professional experience

2016-Present	Medical Director of the Intensive Care Unit and the Weaning Centre, La Pitié-Salpêtrière University Hospital, Sorbonne University Medical Center
2016-Present	PI, Neurorespiratory Interaction in Acute Respiratory Failure, INSERM Research Unit UMR_s 1158 "Clinical and Experimental Respiratory Neurophysiology"
2014-Present	Chair of the Scientific Committee of the REVA Research Network
2013-Present	Full Professor on Intensive Care Medicine, Sorbonne University
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Patients who require invasive mechanical ventilation for acute respiratory failure receive initially controlled mechanical ventilation and sedation. During this initial phase, respiratory muscles are generally inactive since sedation depresses respiratory drive and respiratory muscles efforts. Hence clinicians can mainly focus on lung protective ventilation without having to cope with respiratory muscles effort. As respiratory system function improves, sedation is decreased and, subsequently, spontaneous breathing activity resumes. As soon as spontaneous breathing resumes, clinicians must cope with respiratory muscles effort, until the patient is extubated.

There is accumulating evidence that spontaneous inspiratory efforts may injury the lungs, which termed patient self-inflicted lung injury (P-SILI). First, these efforts can generate high level of transpulmonary pressure that may cause lung injury despite low airway pressure. Second, the pendelluft phenomenon, which is a shift of air to dependent lung regions from other lung regions. Third, increased pulmonary perfusion, which in turn may generate pulmonary edema. Diaphragm injury may also occur. On the one hand, mechanical ventilation unloads the diaphragm and hence may cause disuse diaphragm injury and atrophy. On the other hand, there is growing evidence that vigorous inspiratory effort could injury the diaphragm through excessive diaphragm load and eccentric contraction.

This monitoring and the quantification of respiratory drive and effort helps tailoring treatments such as ventilator settings and sedation during the transition phase between controlled ventilation and weaning. Although esophageal pressure measurement is the reference standard for respiratory muscle effort and lung stress quantification, various non-invasive screening tools have been proposed. Expiratory occlusion pressure (Pocc) detects low and high effort. Ultrasound may also help estimating diaphragm effort. Airway occlusion pressure (P0.1), is a reliable surrogate of respiratory drive and is displayed on most modern ventilators. Additional techniques include electrical activity of inspiratory muscles. Finally, dynamic transpulmonary driving pressure ($\Delta PL,dyn$) is easy to measure with every ventilator and may help quantifying lung stress resulting from high inspiratory effort and high inspiratory pressure support.