# Spontaneous effort causes occult Pendelluft during mechanical ventilation

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**Abstract:** In the injured lung, local negative pleural pressure generated by diaphragmatic contraction is not uniformly transmitted, but is concentrated in dependent lung. This causes Pendelluft (using EIT), with shift of air from non-dependent to dependent lung regions. Thus, during lung-protective ventilation with strictly limited tidal volume, the presence of strong inspiratory effort can result in a hidden, local over-stretch of the dependent lung.

## 1 Introduction

In normal lungs, local changes in pleural pressure ($P_{pl}$) are generalized over the whole pleural surface. However, in a patient with injured lungs, we observed (using EIT) a Pendelluft phenomenon, i.e. movement of air within the lung from non-dependent to dependent regions without change in tidal volume that was caused by spontaneous breathing during mechanical ventilation. We hypothesized that in injured lungs: negative $P_{pl}$ generated by diaphragm contraction has localized effects (in dependent regions) that are not uniformly transmitted; and such localized changes in $P_{pl}$ cause Pendelluft.

## 2 Methods

In seven lavages-injured pigs, EIT data were recorded during spontaneous breathing and muscle paralysis, comparing the temporal pattern and distribution of regional ventilation with the sub-division of the thorax into four zones (ventro-dorsal). For the validation of regional distribution of ventilation, dynamic CT scans were performed to evaluate the absolute movement of air within thick slice. We also measured negative $P_{pl}$ in dependent lung directly using intra-pleural catheters. Further, we estimated the additional airway pressure required to achieve comparable dependent lung inflation in the presence of muscular paralysis by titrating inspiratory airway pressures during paralysis till observing the same delta-Z observed during spontaneous breathing in dependent zones.

## 3 Results

In all lung-injured animals, spontaneous breathing caused Pendelluft, which was associated with more negative local $P_{pl}$ in dependent regions vs. esophageal pressure (-14.3±3.3 vs. –7.1±2.1 $cmH_2O$, P<0.01). During paralysis, there was heterogeneous —but simultaneous— inflation in the dependent as well as in the non-dependent lung regions (Figure 1). Dynamic CT analysis also confirmed the Pendelluft. Continuous recording of EIT was made during the transition from spontaneous effort to complete muscle paralysis (following a bolus of succinylcholine chloride; Figure 2); this demonstrated that the extent of the Pendelluft was proportional to the intensity of the respiratory effort. Comparable over-inflation of dependent lung during paralysis required almost 3-fold greater driving pressure (and tidal volume) vs. spontaneous breathing (28.0±0.5 vs. 10.3±0.6 $cmH_2O$, P<0.01; 14.8±4.6 vs. 5.8±1.6 mL/kg, P<0.05).

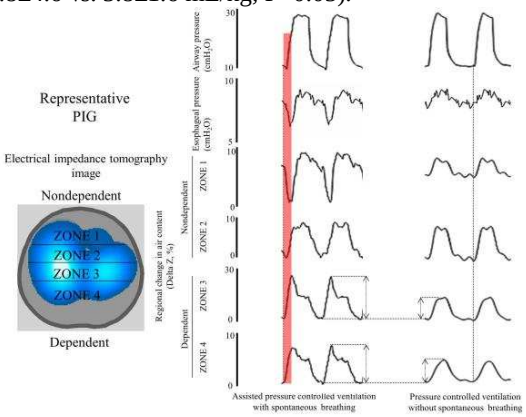


**Figure 1:** EIT Waveforms in Experimental Lung Injury - Spontaneous vs. Ventilator Breaths

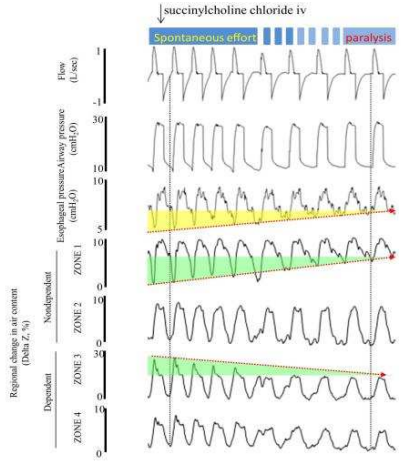


**Figure 2:** EIT Waveforms in transition from spontaneous breathing to muscle paralysis

## 4 Conclusions

Pendelluft may constitute a novel mechanism of ventilator–induced lung injury. The observed overstretch of the dependent lung could cause occult local injury, which cannot be detected (and therefore avoided) using conventional monitoring.