Damages from mechanical ventilation have been attributed to barotrauma or volutrauma, to atelectrauma (shear stresses occurring at the interface of open and close lung regions) and to biotrauma (inflammatory response). The force-bearing structure is the lung skeleton, composed of a fibrous network of elastin and collagen, embedded in the extracellular matrix. The elastin fibres are the determinant of the elastic recoil, while the inextensible collagen fibres act as a stop-length when completely unfolded at total lung capacity. Lung cells, anchored to the extracellular matrix may activate the inflammatory cascade if subjected to excessive shape changes, up to the upper limit at rupture. Before this limit, however, the un-physiological distension of the lung cells may result in generalized lung inflammation.

Stress and strain are mechanical phenomena properly referred to microstructures or to small areas of a body. “Stress” is defined as the internal distribution of the counterforce per unit of area that balances and reacts to an external load. The associated deformation of the structure is called “strain”, which is defined as the change in size or shape referred to the initial status. Stress and strain are linked by the following formula:

\[ \text{stress} = k \times \text{strain} \quad \text{(Equation 1)} \]

The clinical equivalents of stress and strain are transpulmonary pressure (airway pressure minus pleural pressure) the ratio of volume change (DV) to the functional residual capacity (FRC, the resting lung volume). At FRC the fibres of the lung skeleton are in their natural resting position and the respiratory muscles are inactive and relaxed. Accordingly, within the range of pressures and volumes for which the stress and strain relationship is linear:

\[ \Delta P_L \text{ (stress)} = E_{L,\text{spec}} \times \frac{\Delta V}{\text{FRC}} \times \text{strain} \quad \text{(Equation 2)} \]

This equation shows that the proportionality constant between stress and strain, called specific lung elastance, is the transpulmonary pressure at which FRC doubles. This parameter reflects the intrinsic elasticity of the lung parenchyma open to gases. As stress and strain, are not measured in clinical practice, we sought to determine the extent to which they can be described by their clinical surrogates, the plateau airway pressure and the tidal volume (V T) referenced to ideal body weight (IBW). Within ALI/ARDS patients and normal subjects a given applied airway pressure produced largely variable stress due to the variability of the lung elastance to respiratory system elastance ratio Analogously for the same applied tidal volume the strain variability within subgroups was remarkable, due to the functional residual capacity variability. Plateau pressure and tidal volume are inadequate surrogates for lung stress and strain and their measurement would ideally allow to tailor a safer mechanical ventilation in the individual patients.