



The “normal” ventilated airspaces suffer the most damaging effects of mechanical ventilation

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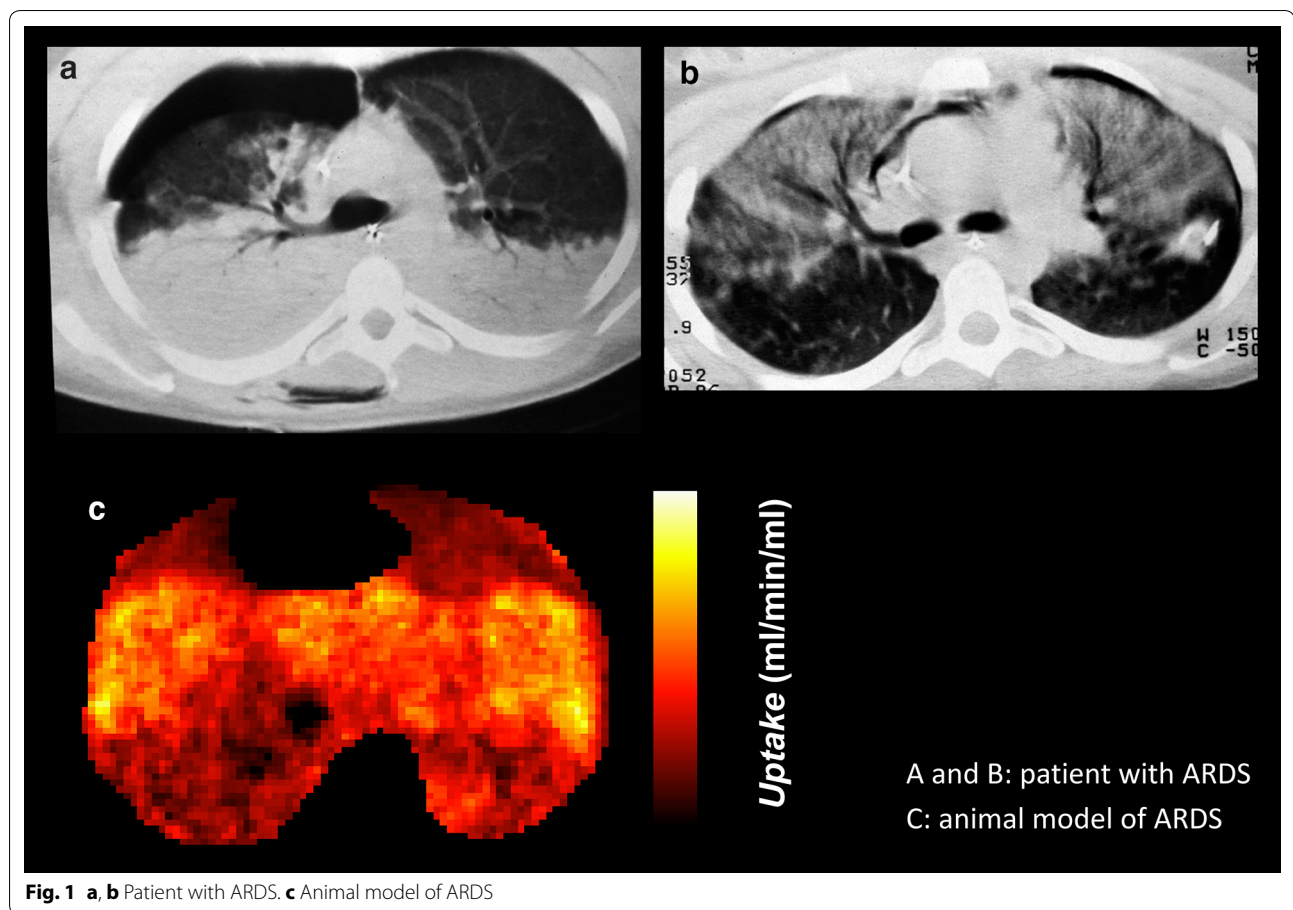
A patient was treated because of acute respiratory distress syndrome (ARDS) with multiple fractures, lung contusion and bilateral pneumothorax. Figure 1a shows the first CT scan when the patient was ventilated in supine position in control mode with at that time (1996) standard high tidal volume of 12 ml/PBW, positive end-expiratory pressure of 5–10 cmH₂O and end-inspiratory pressures of 35–40 cmH₂O. It is from her early phase of ARDS and displays dependent collapse and “normal-appearing” parenchyma ventrally. A follow-up CT after 13 days, now during spontaneous breathing without any mechanical support, demonstrates the opposite pattern with opacities in the ventral portion and clearance of the lower half of the lung (Fig. 1b). This shift of densities

may be explained by the ventilated, “apparently-spared” units suffering more than collapsed, dependent units and hyperinflated non-ventilated units. This resulted in ventilator-induced lung injury (VILI). More recently we got support for this assumption. We studied the distribution of early pulmonary inflammation in a porcine VILI model by measuring regional pulmonary uptake of [¹⁸F]fluoro-2-deoxy-D-glucose with positron emission tomography combined with CT. Ventilated, normally and poorly aerated units in the mid zone of the lung were the primary targets of the inflammation and not hyperinflated or collapsed units (Fig. 1c). Decreased tidal volume/distending pressure and increased ventilated lung volume should thus minimize VILI.

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