

TITLE : COMPONENTS OF PEEP-INDUCED LUNG-THORAX VOLUME INCREASE

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Introduction. PEEP may improve oxygen exchange by increasing FRC. If unstable lung is recruited at end-inspiration, PEEP may prevent its collapse at end-expiration, and this volume increase would be complete within the first tidal breath after a step change in PEEP. This increase would be proportional to the total respiratory system compliance (C_{LT}). PEEP may also result in an additional increase in lung volume by overcoming forces which require a longer inflation time than the usual end-inspiratory pause. Thus, the full increase in FRC with PEEP would take several breaths and would be larger than that indicated by the C_{LT} . Our objective was to investigate the relative contributions of these two mechanisms.

Methods. 13 adult patients requiring mechanical ventilation for APF were studied. Each was ventilated at a constant rate ($F_{I}O_2$ 0.5 and V_T 13-15% predicted TLC). Airway and esophageal pressure, expired tidal volume (V_E), FRC, $P_{i}O_2$, $P_{a}O_2$, and $P_{a}CO_2$ were measured at increments of 5 cmH₂O of PEEP. Breath-by-breath expired tidal volume was measured after the application of 10 cmH₂O PEEP in nine patients, to determine the time dependency of the lung volume change. This was also examined when PEEP was applied after two hyperinflations. **Calculations.** 1) Total respiratory system compliance (C_{LT}), $V_E/\Delta P_{aw}$, derived from the difference between a period of zero end-inspiratory flow of 1.2 seconds and end-expiration 2) FRC compliance (C_{FRC}) from $\Delta FRC/\Delta PEEP$ 3) $P(A-a)O_2$. The study was approved by the Human Research Committee and informed consent was obtained for each patient.

Results. The mean initial FRC (55% of predicted supine; range 26-101%) correlated with the initial C_{LT} ($r=0.82$; $P<0.001$). Figure 1, is a pressure-volume diagram of the mean LT data. The C_{FRC} increased with increments of PEEP. At each PEEP level the corresponding C_{FRC} is larger than the C_{LT} . These differences are significant at PEEP levels of 8 and 13 cmH₂O. Thus, at the same lung volume, there is a pressure difference between the end-inspiratory and end-expiratory data. Conversely, a volume difference exists between end-inspiration and end-expiration at the same pressure. The volume difference between the end-inspiratory and end-expiratory data maintained by PEEP at 18 cmH₂O, was computed for each patient. This correlated inversely with the associated improvement in $P(A-a)O_2$ ($r=-0.69$; $P<0.05$). The additional volume was $38\pm 9\%$ (SD) of the total FRC change. The relative contributions of the lung and chest wall to the observed LT changes were measured in ten patients. At iso-lung volume the

pressure difference between the end-inspiratory and end-expiratory data was distributed across both the lung and chest wall with the lung component accounting for 0.63 ± 0.25 (SD) of the total LT changes. After a step change in PEEP of 10 cmH₂O, $68\pm 17\%$ (SD) of the total FRC change was complete within the first breath and the volume retained by the patient on the first breath correlated directly with C_{LT} ($r=0.91$; $P<0.001$). Breath-by-breath V_E showed a 98% return to steady state in 4.4 ± 3 (SD) breaths, (27 ± 19 sec). Prior hyperinflation of the lung did not alter the findings.

Discussion. Three time-dependent factors may explain the observed volume changes 1) normal pressure-volume hysteresis 2) stress relaxation 3) recruitment of non-ventilated lung. These data do not permit quantitative discrimination between these factors. We conclude that both the prevention of collapse of unstable alveoli and time-dependent lung and chest wall factors are involved in the mechanism by which PEEP increases FRC and improves oxygen exchange. 98% of the time course of the induced volume change occurs in 2-60 sec. C_{LT} incompletely describes the elastic properties of the lung and its responses to PEEP.

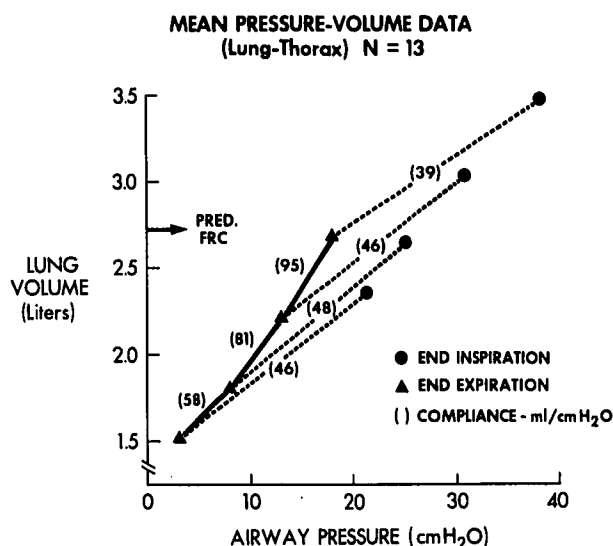


Figure 1

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