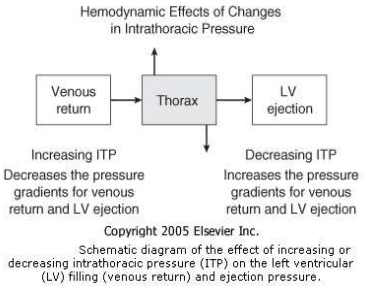
haemodynamic effects of ventilation

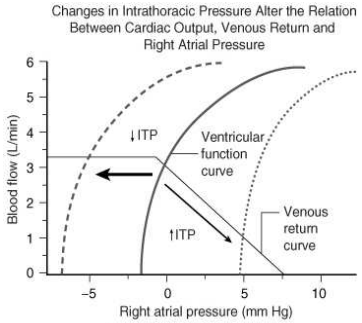
general

- Heart-lung interactions can be broadly grouped, based on three concepts that usually coexist in the clinical setting.
- First, spontaneous ventilatory efforts are exercise; they require oxygen (O2) and blood flow, thus placing demands on cardiac output and producing carbon dioxide (CO2), adding ventilatory stress on CO2 excretion.
- Second, inspiration increases lung volume above resting end-expiratory volume. Thus, some of the hemodynamic effects of ventilation may be due to changes in lung volume and chest wall expansion.
- Third, spontaneous inspiration decreases ITP, whereas positive-pressure ventilation increases ITP; thus, the differences between spontaneous ventilation and positive-pressure ventilation reflect primarily the differences in ITP swings and the energy necessary to produce them.

haemodynamic effects of increased intrathoracic pressure

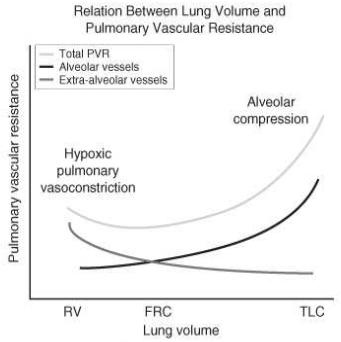


Schematic diagram of the effect of increasing or decreasing intrathoracic pressure (ITP) on the left ventricular (LV) filling (venous return) and ejection pressure.



Schematic representation of the effects of increasing or decreasing intrathoracic pressure (ITP) on steady-state venous return. Note that decreases in ITP that decrease right atrial pressure to below 0 relative to atmospheric pressure increase venous return by only a limited amount, whereas increases in ITP progressively decrease venous return to a complete circulatory standstill.

relationship between lung volume & PVR



Schematic diagram of the relation between changes in lung volume and pulmonary vascular resistance (PVR), with the extra-alveolar and alveolar vascular components separated. Note that pulmonary vascular resistance is minimal at resting lung volume or functional residual capacity (FRC). As lung volume increases toward total lung capacity (TLC) or decreases toward residual volume (RV), pulmonary vascular resistance also increases. However, the increase in resistance with hyperinflation is due to increased alveolar vascular resistance, whereas the increase in resistance with lung collapse is due to increased extra-alveolar vessel tone.

ventricular interdependence

- Changes in RV output must invariably alter LV filling, because the two ventricles are serially linked through the pulmonary vasculature.
- LV preload can also be directly altered by changes in RV end-diastolic volume. If RV volume increases, LV diastolic compliance will decrease by the mechanism of ventricular interdependence.