The most common cause of unsuccessful weaning from mechanical ventilation is thought to be the failure of the respiratory muscle pump. The alterations in lung volume and intrathoracic pressure that occur during weaning can have detrimental effects on cardiovascular performance and lead to a decreased supply of oxygen to the overworked respiratory muscles. Thus, the complex interactions between the heart and the lung during spontaneous breathing can determine whether the patient can be weaned successfully.

The most characteristic finding in patients who fail a weaning trial is that they develop rapid shallow breathing immediately upon discontinuation of mechanical ventilation [1]. Compared with successes, patients who fail weaning have a higher respiratory rate and lower tidal volume throughout the weaning trial. The increase in frequency is commonly associated with dynamic hyperinflation, as reflected by the presence of intrinsic positive end-expiratory pressure (PEEPi). During a trial of spontaneous breathing, dynamic PEEPi was higher in the failure patients than in the success patients. From the start to the end of the trial, dynamic PEEPi increased progressively in weaning-failure patients, suggesting the development of dynamic hyperinflation; however, expiratory muscle contraction may have also contributed [2].

Hyperinflation has numerous adverse effects on respiratory muscle function. The increase in lung volume causes the inspiratory muscles to shorten, with a consequent decrease in their force of contraction. Flattening of the diaphragm is associated with an increase in its radius of curvature, which, according to the law of Laplace, causes a decrease in the efficiency of transdiaphragmatic pressure generation. The medial rather than axial orientation of the muscle fibers may cause diaphragmatic contraction to produce rib-cage deflation rather than expansion. The zone of apposition between the diaphragm and the rib cage is decreased by hyperinflation, which also decreases the efficiency of chest wall expansion. Likewise, the horizontal (rather than the normal oblique bucket-handle) orientation of the ribs makes it more difficult for the respiratory muscles to expand the rib cage [3]. In addition, hyperinflation forces the patient to breathe on the upper, less-compliant portion of the pressure-volume curve, so that the inspiratory pressure requirement for a given tidal volume is increased. Because tidal volume is incompletely exhaled, alveolar pressure remains positive at the end of expiration. Thus, when the patient begins to inspire, he has to first generate a negative inspiratory pressure equal in magnitude to the level of PEEPi, which results in a marked increase in the work of inspiration.
An increase in lung volume can also have detrimental effect on pulmonary vascular resistance. Indeed, lung volume is one of the major determinants of pulmonary blood flow. The pulmonary vasculature can be divided into alveolar and extra-alveolar vessels. Alveolar vessels are located within the alveolar walls and are surrounded by alveolar pressure. As lung volume increases from residual volume to total lung capacity, resistance of the alveolar vessels increases secondary to compression and the associated increase in transpulmonary pressure. The extra-alveolar vessels are surrounded by lung interstitial pressure, which is similar to intrathoracic pressure. As lung volume increases, interstitial pressure decreases as a result of the increased elastic recoil of the lung. These opposing forces induce a complex relation between lung volume, pulmonary vascular resistance, and pulmonary vascular capacitance. However, the net effect of increasing lung volume is to increase pulmonary vascular resistance, which, in turn, will cause a decrease in cardiac index [4].

The second major alteration in breathing that can affect cardiovascular function during weaning is changes in intrathoracic pressure that occur upon switching from mechanical ventilation to spontaneous breathing (Fig. 12.1).

![Fig. 12.1. Recordings of right ventricular stroke volume ($SV_{RV}$), left ventricular stroke volume ($SV_{LV}$), transmural right atrial pressure ($P_{ra}$), airway pressure ($P_{aw}$) and pleural pressure ($P_{pl}$) during spontaneous ventilation (left) and positive-pressure ventilation (right) in an intact, closed-chested canine model. The vertical dotted lines are reference points for the start and end of a breath for each condition. See text for details. (Modified from [4], with permission)