Mechanical Breath
Non-pharmacologic Support for a Failing Heart?

Mechanical ventilation and PEEP are considered relatively contraindicated in patients with failing circulatory function. It is suggested that hypovolemia and cardiac failure should be treated first before mechanical ventilation and PEEP is considered for treatment of hypoxemia in these patients. The application of intermittent mandatory ventilation (IMV) and continuous positive airway pressure (CPAP) to animals, as well as hypoxemia patients with uncompromised hearts, have been shown to exhibit no detrimental effects on cardiac output. It is suggested that since IMV and CPAP permit spontaneous inspiration with fewer controlled mechanical breaths per unit time, a lower intrapleural pressure results which augments venous return and maintains better cardiac output. However, in a group of patients with poor left ventricular reserve following aortocoronary bypass surgery, it was observed that changing from controlled mechanical ventilation (CMV) to IMV had deleterious effects on hemodynamic status. In this issue of Chest (see page 21) Räsänen et al have compared the hemodynamic effects of CMV, IMV and CPAP in patients with acute myocardial infarction. In their study, patients maintained higher cardiac index and lower arteriovenous oxygen content difference during IMV than either CMV or CPAP. Furthermore, myocardial ischemia was observed more often during CPAP alone than during mechanical ventilatory support. This intriguing clinical observation suggests that mechanical breaths may be helpful in the management of patients with acute myocardial infarction.

Mechanical ventilatory support may be beneficial to patients with a failing left ventricle in several ways. Positive pressure ventilation increases intrapleural pressure, thereby diminishing right heart preload. In addition, increased airway pressure may restrict left ventricular filling by mechanical compression of alveolar capillaries (Starling resistor effect). These two conditions may improve the failing heart by optimizing ventricular end-diastolic volume. The increased intrathoracic pressure may also reduce left ventricular afterload due to decreased transmural aortic pressure during mechanical ventilatory support. In patients with underlying cardiopulmonary disease requiring mechanical ventilation, the oxygen cost of breathing may be a significant proportion of the total body oxygen requirement. Thus, in the presence of compromised oxygen delivery state, eg, cardiogenic shock, institution of artificial ventilatory support may decrease the inspiratory oxygen demands and may release substantial quantities of oxygen for use by other body systems. Institution of mechanical ventilation also provides the opportunity for safely sedating the patient, decreasing sympathetic outflow, preventing hypertension and tachycardia and thereby decreasing the strain on the failing left ventricle.

Whether early or prophylactic mechanical ventilation is beneficial in patients with acute myocardial infarction with a failing left ventricle is not clear from the study by Räsänen et al, but it raises a question which requires further detailed study.

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