All Positive Airway Pressures Are Not Created Equal!

In 1936, Poulton and Oxon suggested that application of positive airway pressure (PAP) had therapeutic value in the management of patients with cardiogenic pulmonary edema. However, subsequent studies showed that PAP caused elevation of intrathoracic pressure which may decrease venous return and thereby reduce cardiac output. Based on these observations, in patients with left ventricular dysfunction (LVD), mechanical ventilation and the application of PAP have been considered relatively contraindicated. Recent studies suggest that hemodynamic consequences of PAP will depend on the initial intravascular volume status and myocardial reserve of the individual.

During normal inspiratory effort and Müller maneuver (forced inspiration against a closed glottis), transmural left atrial filling pressure and left ventricular volume increases. In a patient with LVD who has normal or high intravascular volume, the negative intrathoracic pressure generated by inspiratory effort may impede left ventricular ejection by an increase in left ventricular volume. Also, generation of negative pericardial surface pressure will increase the needed force that the left ventricle must generate to empty during systole. Thus, in patients with LVD, application of PAP not only improves myocardial function by increasing oxygen availability and decreasing right and left ventricular preload, but it may also reduce left ventricular wall stress, diminish left ventricular afterload, and upgrade left ventricular compliance.

In this issue (see page 158) Räsänen et al report their observation that application of PAP in a group of patients with moderate-to-severe LVD resulted in a significant improvement in oxygenation. But more importantly, they observed that patients with severe LVD tolerated PAP better than those with moderate disease. The exact mechanism for the observed hemodynamic stability during PAP in their patients is not clear. Räsänen and co-workers suggest that raised intrathoracic pressure improved the function of the failing heart by decreasing its afterload. If further studies with more specific assessment of left ventricular function confirm their result, application of PAP will become an attractive adjunctive therapy for the management of patients with pulmonary edema due to acute LVD.

In a subject breathing spontaneously, PAP can be applied by several different systems with varied cardiopulmonary effects; PAP can be applied during expiratory phase of the respiratory cycle (expiratory positive airway pressure or EPAP), or during the entire respiratory cycle (continuous positive airway pressure or CPAP). The CPAP has been shown to increase functional residual capacity more than an equal level of EPAP. However, EPAP is known to cause less reduction in venous return. Furthermore, it has been established that in order to minimize the work of breathing during CPAP therapy, the airway pressure should be maintained at a constant value throughout the respiratory cycle. In patients with LVD, the presumed beneficial hemodynamic effects of PAP are mainly due to the absence of negative pleural pressure during the entire respiratory cycle. Thus, CPAP is the system of choice for management of patients with LVD. When CPAP is used to increase mean pleural pressure, then it is prudent to employ a system that provides enough gas at minimal effort to match the patient's inspiratory flow demand. When spontaneous inspiratory flow rate exceeds that immediately available in the CPAP circuit, then substantial reduction in pleural pressure created during inspiration may negate the full potential hemodynamic benefits. Unfortunately, several commercially available ventilators utilize mechanisms in their CPAP systems that increase resistance to gas flow, thus necessitating significant reduction in airway pressure during the inspiratory phase of spontaneous ventilation. This is graphically illustrated in Figure 1 Räsänen et al. The end-inspiratory airway pressure of the patient is subatmospheric and occasionally atmospheric during what is labeled CPAP of 5 and 10 mm Hg, respectively. Accordingly, this particular patient was receiving EPAP instead of CPAP.

The ideal CPAP system is one that provides minimal inspiratory and expiratory flow resistance, maintains constant PAP, and requires the least work of breathing. This may be best accomplished with a high continuous gas flow circuit rather than the currently available demand systems.

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Replenishing the Starved Patient
When Do Lung Immune Cells Recover?

Starvation and malnutrition remain prevalent problems in clinical practice. The World Health Organization reports that more than one third of the world population suffer from malnutrition. Even in industrialized societies, no hospital ward lacks undernourished patients. Such patients are more susceptible to pneumonia, and in the critically ill malnourished patient, pulmonary sepsis and respiratory failure are among the major causes of death.

Prolonged protein and calorie restrictions produce abnormalities in pulmonary defense mechanisms, pulmonary structure and function, control of breathing and respiratory muscular contractility. Such changes have recently been reviewed in this journal by Rochester and Esau. At present, our understanding of why malnourished patients are more vulnerable to pulmonary infections remains incomplete.

Immunologically, nutritional deficiencies have been shown to be associated with impairments of T-lymphocytes, complement system, polymorphonuclear leukocyte bacterial killing, B-lymphocyte function and antibody production. On the other hand, no change was detected in the chemotactic and bactericidal functions of peritoneal macrophages obtained from rats subjected to protein calorie malnutrition. In another study, peripheral monocytes of children suffering from severe malnutrition were also found to have unaltered bactericidal and phagocytic capabilities.

The latter two studies suggest that the mononuclear phagocytic system (MNP) maintain its normal immune functions and are unaffected by protein calorie malnutrition. However, the pulmonary alveolar macrophage, which plays an important role in lung host defense, is a different breed of MNP since its metabolic and cell surface characteristics are different from those of peritoneal, liver or other resident tissue macrophages. Thus, the findings of normal MNP function in malnourished patients should not be extrapolated directly to the alveolar macrophage without further investigation.

Our recent study in rats in fact shows that starvation can reduce alveolar macrophage phagocytic activity to less than half of the normal, using an in vitro assay technique against radioactively tagged microorganisms. Interestingly, on refeeding the animals, a delay of up to three weeks in the recovery of alveolar macrophage phagocytic activity was noted, even though the clinical appearance and the absolute lymphocyte counts of these animals had returned to normal within a week of refeeding. This is also in contrast to experiments by other investigators which showed that a number of systemic immune functional parameters recovered in less than three weeks on refeeding malnourished animals. In rats, the mean turnover rate of alveolar macrophage is about three weeks, which seems to suggest that the effect of starvation on the resident alveolar macrophages may be irreversible, and that only when the whole macrophage cell population in the lungs has been replaced would the complete recovery of its phagocytic activity become apparent.

Although this hypothesis requires further confirmation, these findings may be of clinical importance if they can be extrapolated to patients. Firstly, the intensity of functional changes due to malnutrition in the peripheral monocytes may not be as striking as in the more mature alveolar macrophages, which are also more efficient scavenger cells. Secondly, while other systemic immunologic functions may be normal or had recovered early after replenishing a malnourished patient, a longer period may be required for the local lung immune cells to recuperate fully. This would