Weaning and Respiratory Muscle Dysfunction

The Egg-Chicken Dilemma

Dependence on mechanical ventilation following the resolution of an acute respiratory illness is a major health-care problem. About 80% of patients who receive mechanical ventilation in the ICU resume spontaneous breathing in a few days. The remaining 20% experience difficult weaning due to a combination of the unresolved primary illness or a preexisting cardiorespiratory or neuromuscular disease. Forty-one percent of the overall time spent in the ICU was reported to be devoted to weaning with large differences between patients with different etiologies necessitating mechanical ventilation. The process of weaning accounted for more than half of the length of ICU stay in patients with COPD, cardiac failure, or neurologic problems.1

There is a need to shorten the time to weaning. Endotracheal intubation may cause complications that increase morbidity and mortality.2 Long-term sequelae may also occur directly related to intubation. Furthermore, heavy sedation or paralysis, particularly in the initial days of a critical illness, may lead to a generalized myopathy. Critical illness neuromyopathy, which is a combination of myopathy and neuropathy, may develop in critically ill patients with sepsis and multiple organ failure.3 Other animal experiments have suggested that diaphragm atrophy does not occur if the paralysis is partial or intermittent. Impairment in skeletal muscle strength in the ICU also could be a consequence of electrolyte disturbances, or a direct effect of hypercapnia, hypoxia, malnutrition, treatment with corticosteroids or other agents, and hemodynamic instability. The use of continuous IV sedation as well as the improper use of controlled mechanical ventilation may be associated with prolonged mechanical ventilation and with the development of selective diaphragmatic atrophy after only 48 h.7

Respiratory muscle weakness following all of the above-mentioned conditions is one of the major determinants of weaning failure in patients receiving

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mechanical ventilation. Are patients who fail a trial of weaning from mechanical ventilation at considerable risk for developing respiratory muscle fatigue? Or rather does respiratory muscle fatigue induce an inability to wean? This is a question of major clinical importance. Patients who fail a trial of weaning are at a disadvantage when compared with those who successfully wean from mechanical ventilation because they have greater abnormalities in their lung mechanics. In this issue of CHEST (see page 553), Chang et al faced another side of respiratory muscle function, namely, diaphragm endurance. In a small number of patients who were admitted to the ICU for a number of different reasons and were successfully weaned from mechanical ventilation, these authors evaluated respiratory muscle endurance after ICU discharge. They found that endurance was negatively correlated with the duration of mechanical ventilation but not with ICU length of stay or weaning duration. These authors concluded that patients who receive mechanical ventilation for > 48 h and are successfully weaned have reduced inspiratory muscle endurance that worsens with a longer duration of mechanical ventilation. On the basis of their results, they suggest some potential benefits of inspiratory muscle training (IMT).

This study leaves us with some speculative problems. Indeed, on the basis of this small study, we do not know whether reduced respiratory muscle endurance is the cause or the consequence of prolonged mechanical ventilation. Furthermore, the suggestion of utility of IMT is purely speculative and rather questionable. The rationale for using IMT in the ICU is controversial. Respiratory muscle function per se is not the only factor involved in a delay in weaning. As a matter of fact, respiratory pump efficiency is a result of the balance between the load that the respiratory system has to face and its capacity. In patients with some diseases, such as COPD, ARDS, interstitial lung disease, and chronic heart failure, the elastic and resistive loads are elevated as much as three to four times compared to levels in healthy individuals. In these populations, particularly in COPD patients, IMT was suggested as a possible intervention strategy for increasing the inotropic or endurance properties of the diaphragm.

Most of the information on the role of respiratory muscles in weaning success or failure has been obtained by studies in COPD patients, who are not representative of all of the patients evaluated in this study. COPD patients are not likely to benefit much from this specific treatment. It has been shown that the impaired contractile effect of the diaphragm in these patients is due to the altered geometric shape of the diaphragm dome rather than to muscle atrophy. The diaphragm of patients with COPD is as good as that of healthy subjects in generating pressure at comparable lung volumes, showing an adaptive change toward the slow-twitch characteristics of the muscle fibers and thus increasing resistance to fatigue. Furthermore, it has been reported that weaning failure was not associated with low-frequency fatigue of the diaphragm. Factors protecting the patient from respiratory muscle fatigue include rib cage and expiratory muscle recruitment, down-regulation of respiratory motor output, and early reinstitution of mechanical ventilation.

A recent metaanalysis conducted by Lotters et al concluded that IMT is an important component of pulmonary rehabilitation in severely impaired but stable COPD patients in the community, leading to improvement in respiratory muscle strength and endurance. Nevertheless, the improvement has not been shown to lead to better clinical well-being and outcome. The lack of benefit is not surprising because in stable severely impaired COPD patients, the maximum inspiratory pressure is not reduced to such a level as to compromise spontaneous breathing. In patients experiencing weaning failure, even a small increase in inspiratory muscle strength or endurance could have a significant effect on clinical outcome. An interesting potential role of IMT may be in preventing steroid-induced myopathy. In a randomized-controlled trial, Weiner et al showed that the inotropic and endurance capacity of inspiratory muscles had been spared from damage due to 2 weeks of therapy with corticosteroids only in the group of patients undergoing specific training.

Case studies have been published describing resistive endurance or eucapnic hyperventilation IMT programs in difficult-to-wean patients. These endurance training methods resulted in modest increases in inspiratory muscle strength. Furthermore, it has been shown that although endurance training increases muscle redox potential in healthy subjects, patients with COPD show a reduced ability to adapt to endurance training, which is reflected in a lower capacity to synthesize reduced glutathione. More dangerously, it has been shown also that inspiratory loading is associated with diaphragm injury.

Little attention is usually directed to these problems in the ICU. This fact has been underlined by a European survey focusing on the role of physiotherapists in European ICUs. In a sample of 102 European ICUs, only 75% had at the least one physiotherapist working exclusively in the ICU with an enormous difference in the number of physiotherapists therein employed. Furthermore, this survey showed that physiotherapists usually gave enough attention to respiratory therapy, mobilization, and positioning, whereas they neglected early training sessions.
In conclusion, a randomized controlled study on the effect of IMT on weaning is lacking. Although designing and performing such a study may be challenging to researchers, the motivation might be even greater in the light of the study by Chang et al.

Nicolino Ambrosino, MD, FCCP
Pisa, Italy

References


Acute and Chronic Respiratory Failure in Patients With Obesity-Hypoventilation Syndrome

A New Challenge for Noninvasive Ventilation

The increased prevalence of adult obesity has been one of the most striking epidemiologic phenomenon in most countries around the world during these last 100 years. It is also a matter of great concern in children 5 to 12 years old in whom the prevalence of obesity has been multiplied by three in the United States and by four in France between 1960 and 2000.¹

Because adult obesity is a risk factor for obstructive sleep apnea syndrome (OSAS), obesity-hypoventilation syndrome (OHS), acute hypercapnic respiratory failure (AHRF), and higher incidence of respiratory postsurgical complications, it is not surprising to see that obesity is now considered as an emerging cause of chronic respiratory failure (CRF) requiring domiciliary ventilatory assistance.² CRF in obese patients is a frequent issue in clinical practice and is usually diagnosed either in the context of an AHRF in the emergency department or when investigating a potential diagnosis of OSAS or even during a preoperative evaluation. Approximately 10% of patients with OSAS have daytime hypercapnia, often associated with pulmonary hypertension.³ Obstructive airways disease may be associated in some of these patients and constitutes the so-called overlap syndrome.⁴–⁶ Another clinical picture of CRF in