Avoiding Respiratory and Peripheral Muscle Injury During Mechanical Ventilation

Diaphragm-Protective Ventilation and Early Mobilization

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KEYWORDS

• Limb muscle weakness • Respiratory muscle weakness • Ventilator dependence • Intensive care unit • Mechanical ventilation

KEY POINTS

- Both limb muscle weakness and respiratory muscle weakness are exceedingly common in critically ill patients.
- Respiratory muscle weakness prolongs ventilator dependence, predisposing to nosocomial complications (including limb muscle weakness) and death.
- Limb muscle weakness persists for months after discharge from intensive care and results in poor long-term functional status and quality of life.
- Major mechanisms of muscle injury include critical illness polymyoneuropathy, sepsis, pharmacologic exposures, metabolic derangements, and excessive muscle loading and unloading.
- The diaphragm may become weak because of excessive unloading (leading to atrophy) or because of excessive loading (either concentric or eccentric) owing to excessive ventilator assistance.

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Crit Care Clin 34 (2018) 357–381
https://doi.org/10.1016/j.ccc.2018.03.005
criticalcare.theclinics.com

INTRODUCTION

The introduction of positive-pressure ventilation in the mid-20th century led to the advent of the modern intensive care unit with all its previously unimagined possibilities for survival and recovery from severe life-threatening illness. Yet, as survival after critical illness has dramatically improved (general intensive care unit [ICU] mortality rates after acute respiratory failure are now estimated at 25%–30%[1]), attention has increasingly turned to the sequelae of critical illness in survivors. The impact of critical illness persists long after ICU discharge as survivors struggle with functional disability, cognitive impairment, and neuropsychiatric illness in the months and years after critical illness.

Muscle weakness affecting both the respiratory muscles and peripheral muscles of the axial skeleton is thought to be a key mediator of this protracted disability after critical illness. Limb muscle atrophy and dysfunction can persist for months after "recovery" from acute respiratory failure. Respiratory muscle weakness is a major risk factor for prolonged mechanical ventilation, a development that entails a grave risk of long-term morbidity and mortality. Muscle injury during critical illness is, therefore, a subject of vital importance.

This article provides an overview of the epidemiology, mechanisms of respiratory muscle and limb muscle weakness during critical illness, and impact of both forms of muscle weakness on clinical outcomes. We outline strategies to prevent and treat limb and respiratory muscle weakness in critically ill patients.

DEFINITION AND EPIDEMIOLOGY

Muscle weakness is defined as impairment in the force-generating capacity of muscle. Because the force generated by muscle tissue is correlated with the magnitude of the neural contractile stimulus, reliable muscle function assessment requires a standardized stimulus. Standardization is accomplished either by eliciting a maximal stimulus (eg, maximal contractile effort) or by applying a standardized nonvolitional stimulus (eg, twitch magnetic stimulation). Techniques for assessing muscle function in ICU patients are summarized in **Table 1**. These techniques are reviewed in detail elsewhere.[2,3] Clinicians should bear in mind that maximal volitional efforts may be difficult to obtain in critically ill patients owing to impaired consciousness, delirium, sedation, or central fatigue.

Respiratory Muscle Weakness

A substantial proportion of ICU patients—up to 80%—develop respiratory muscle weakness at some point during their ICU stay.[4,5] Diaphragm weakness is present in the majority of patients at ICU admission.[6,7] This early diaphragm weakness has been associated with markers of illness severity and is associated with an increased risk of death but not prolonged ventilation, suggesting that early diaphragm weakness represents a form of reversible organ failure related to the patient's critical illness.

In patients who are ready to commence the weaning phase of ventilation, the prevalence of diaphragm weakness varies between 63% and 80%.[8–10] Estimates of the prevalence of diaphragm weakness are somewhat lower when diaphragm ultrasound examination is used to assess diaphragm function, ranging between 29% in patients submitted to the first spontaneous breathing trial[11] and 36% in prolonged weaning patients.[12,13]

These rather dramatic prevalence estimates are corroborated by histologic studies of diaphragm biopsies obtained from brain dead organ donors and mechanically