CRITICAL CARE

COMMENTARY

Diaphragm weakness and mechanical ventilation - what's the critical issue?

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See related research by Hermans *et al.*, http://ccforum.com/content/14/4/R127

Abstract

While animal studies indicate that controlled mechanical ventilation (MV) induces diaphragm weakness and myofiber atrophy, there are no data in humans that confirm MV *per se* produces diaphragm weakness. Whether or not diaphragm weakness results from MV, sepsis, corticosteroids, hyperglycemia, or a combination of these factors, however, is not the most important issue raised by the recent study from Hermans and colleagues. This study makes an important contribution by providing additional evidence that many critically ill patients have profound diaphragm weakness. If diaphragm weakness of this magnitude is present in most mechanically ventilated patients, a strong argument can be made that respiratory muscle weakness is a major contributor to respiratory failure.

Numerous animal studies indicate that controlled mechanical ventilation (MV) induces diaphragm weakness and myofiber atrophy, but no data in humans confirm MV *per se* produces diaphragm weakness. In their recent publication, Hermans and colleagues [1] used an objective nonvolitional technique of bilateral anterolateral magnetic stimulation of the phrenic nerves to measure twitch transdiaphragmatic pressure (TwPdi) and assessed the degree of diaphragm weakness in ten critically ill mechanically ventilated patients. Importantly, they found an average TwPdi value of 11.5 ± 3.9 cm water, which represents a 70% reduction in diaphragm strength when compared to normal individuals. In their analysis, the authors suggest that duration of MV is associated with decreased diaphragm force generation. This interpretation, however, may be misleading. Importantly, nine of the ten patients in their study were septic and eight out of ten received corticosteroids. Moreover, no information regarding glucose control is included. Since sepsis, corticosteroid use and hyperglycemia are major risk factors for acquired weakness [2,3], it seems plausible that these conditions also contributed to the diaphragm weakness observed.

Whether or not diaphragm weakness results from sepsis, respiratory muscle unloading from MV, corticosteroids, hyperglycemia, or a combination of these factors, however, is not the most important issue raised by this study. Hermans and colleagues [1] should be congratulated because their study makes an important contribution by providing additional evidence that many critically ill patients have profound diaphragm weakness [4,5]. If diaphragm weakness of this magnitude is present in most mechanically ventilated patients, a strong argument can be made that respiratory muscle weakness is a major contributor to respiratory failure.

Consider this - mechanical ventilators are not artificial lungs but simply machines that substitute for the respiratory pump. The fact is that the respiratory pump does not have an unlimited capacity; if it did, theoretically, some patients would require augmented oxygen delivery and/or end expiratory pressure but none would require MV. For patients with normal respiratory muscle function, respiratory failure usually occurs when the respiratory workload becomes too high for the normal pump to maintain ventilation. In principal, any reduction in pump function below normal should increase the propensity for respiratory failure to develop, with the level of respiratory workload required to induce respiratory failure directly related to the level of pump function. Specifically, the lower the pump function, the lower the respiratory workload required to induce respiratory failure. If this concept is correct, the level of respiratory muscle dysfunction reported by Hermans and colleagues should be a major contributor to respiratory failure.

Unless the patient has a known neuromuscular disorder, critical care physicians often overlook diaphragm weakness as an important factor contributing to respiratory failure and weaning difficulties in a significant

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number of patients. We focus on improving lung function, perhaps because conceptually this is easier to understand, easier to assess using chest radiographs, and, for the most part, the treatment options are relatively straightforward. On the other hand, if we recognize that diaphragm weakness is present, what can we do? Regrettably, the current approach to diaphragm weakness in critically ill patients is similar to the approach to pulmonary hypertension 30 years ago. Physicians once believed pulmonary hypertension was extremely rare, and there were no treatments. Today, we recognize that pulmonary hypertension is more prevalent, we have better tools to diagnose this problem, and we have a growing ensemble of pharmacological agents to treat patients with this disorder. To make such progress in dealing with the problem of respiratory muscle dysfunction in critically ill patients, we need better diagnostic tools, a better understanding of the pathophysiology of this disorder and, most importantly, we need to develop rational, specific and effective treatments. Once these goals are met, we may be able to substantially shorten the duration of MV in ICU patients and improve long-term outcomes in this growing population of patients.

Abbreviations

MV = mechanical ventilation; TwPdi = twitch transdiaphragmatic pressure.

Competing interests

The authors declare that they have no competing interests.

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