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Enhancing Our Understanding of Breathing Mechanics in Nonintubated Patients with Acute Hypoxemic Respiratory Failure

Over the last 50 years, we have seen remarkable advancements in the understanding and management of acute hypoxemic respiratory failure (AHRF) (1). Baro-/volutrauma (2) and pendelluft (3, 4) are part of the standard vocabulary, particularly in reference to lung injury and acute respiratory distress syndrome (5). Advances in noninvasive strategies for managing AHRF bring novel choices that avoid the need for an endotracheal tube. High-flow nasal oxygen (HFNO) (6) and the helmet interface (7, 8) are two of the latest options on the menu for clinicians to consider. The ability to deliver high FiO_2 and high positive end-expiratory pressure (PEEP) without an endotracheal tube is an important advance brought by HFNO and helmet interfaces. Yet, even such noninvasive strategies are not without potential harm. Indeed, ventilator-induced lung injury may occur with the helmet interface. There is even recent discussion of patient self-induced lung injury (9), though its clinical importance remains to be fully elucidated (10).

It is upon this backdrop that a better understanding of lung mechanics and gas exchange physiology would be welcome. In this issue of the *Journal*, Menga and colleagues (pp. 1310–1323) performed sophisticated physiological evaluations of 15 patients with severe AHRF ($\text{PaO}_2/\text{FiO}_2 \leq 200$) (11). Three different noninvasive strategies were used: helmet pressure support, helmet continuous positive airway pressure (CPAP), and HFNO. Each strategy lasted 1 hour, and the order was randomly assigned for each patient. All patients were semirecumbent at 45 degrees. Esophageal manometry and electrical impedance tomography of the thorax were performed. Inspiratory esophageal and transpulmonary pressure swings were surrogates for inspiratory effort (12) and lung distension, whereas V_T and end-expiratory lung volume were detected by electrical

impedance tomography. Primary endpoints of interest included inspiratory effort, lung volumes (V_T and end-expiratory lung volume), and gas exchange.

A surprising finding in this study was the significant reduction in inspiratory effort when pressure support was delivered via the helmet, a finding not seen with either helmet CPAP or HFNO. This interesting finding was particularly noteworthy in those patients who had very high inspiratory efforts measured during helmet CPAP or HFNO. To the extent that high inspiratory effort may put patients at risk for worsening lung injury, it seems optimal to use the helmet with pressure support for those with high inspiratory drive. Unfortunately, esophageal manometry is not widely used, which limits the ability to easily detect such a high drive in many patients.

Oxygenation was significantly improved with the helmet interface, regardless of whether pressure support or CPAP was used. HFNO did not improve oxygenation as effectively as either of the helmet interfaces. It is extremely important to remember that helmet CPAP should never be used via a mechanical ventilator. The fresh gas flow from ventilators set to CPAP only is not adequate to wash out CO_2 exhaled into the helmet, and this strategy will quickly lead to hypercapnia and suffocation. In contrast, helmet CPAP with fresh gas flow at 60 L/min from an air/oxygen blender (as was done in this trial) ensures that no significant CO_2 rebreathing occurs (13).

V_T values were lower with HFNO than with the helmet interface, regardless of whether CPAP or pressure support was used. This was due to PEEP leading to lung recruitment in the dorsal dependent lung regions. Pendelluft was common in all patients, though it was most common with HFNO, significantly less with helmet CPAP, and even more reduced with helmet pressure support. The end-expiratory lung volume measurements were higher with either helmet interface than they were in the HFNO group.

Although the findings in the study are interesting, there are important noteworthy limitations. The use of esophageal pressure as a surrogate for respiratory effort may have limitations, particularly when there was no measurement of transdiaphragmatic pressure performed. Ten of the 15 patients studied had AHRF from coronavirus disease (COVID-19). Obviously, it was not possible to

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analyze this small number of patients to determine if there was a difference between AHRF caused by COVID-19 and AHRF of other causes. This is important, given the discrepancy in outcomes noted with helmet ventilation used because of COVID-19 (8, 14). The helmet interface may not be tolerated by all patients, particularly those with high anxiety and/or claustrophobia. Although there are data suggesting that dexmedetomidine may be helpful to improve helmet tolerance in some (15, 16), such an approach must be used with extreme caution. Because dexmedetomidine does not suppress respiratory drive, it stands to reason that it may be the best option for sedation and analgesia with AHRF. Nevertheless, sedating any patient with hypoxemia and respiratory distress may be risky. This is particularly true because the helmet interface may act as a physical barrier to a cry for help from an unstable patient. Careful and frequent monitoring is mandatory if any sedative/analgesic is being considered.

We can thank Menga and colleagues for providing a thorough advance in our understanding of physiology with noninvasive strategies for the management of AHRF. Clearly, the management of AHRF cannot be approached with a “one size fits all” mentality. Last, with the increasing list of options, it is important to recognize when one approach is not working. There is no substitute for bedside patient assessment and the ability to recognize subjective distress and high energy expenditure with the work of breathing. Recognizing when a noninvasive approach has failed and that endotracheal intubation is necessary is critically important to the success of any noninvasive strategy. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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