Should We Monitor Pulsus Paradoxus via Pulse Oximetry in Patients with COVID-19 and Acute Respiratory Failure?

To the Editor:

We read with interest the study by Tonelli and colleagues (1), in which they assessed the inspiratory effort of 30 patients with de novo respiratory failure. The inspiratory effort was quantified by measuring the respiratory swings in esophageal pressure (ΔPes). Their findings suggest that the lack of inspiratory effort relief within the first 2 hours of noninvasive ventilation (NIV) is an early and accurate predictor of NIV failure at 24 hours. In practice, patients in whom ΔPes does not decrease by >10 cm H2O after initiating NIV finally require tracheal intubation.

We agree with Tonelli and colleagues (1) that there is a need for an early robust predictor of NIV failure to avoid intubation delay. Such delay may lead to self-inflicted acute lung injury (2). Indeed, persistently strong spontaneous inspiratory efforts simultaneously increase tissue stresses and raise pulmonary transvascular pressures, vascular flows, and fluid leakage (2, 3). This phenomenon has recently been advocated to explain, at least in part, the rapid deterioration of lung function in patients with coronavirus disease (COVID-19) (3).

The study by Tonelli and colleagues (1) suggests that ΔPes may be a robust predictor of NIV failure and may help clinicians in the decision-making process of tracheal intubation. However, we are concerned by the fact that esophageal probes are rarely used and often poorly tolerated in spontaneously breathing patients with acute respiratory failure. Therefore, although Tonelli’s findings make a lot of sense from a physiologic standpoint, we are afraid that the clinical applicability of their esophageal tonometry approach may be limited.

Respiratory swings in pleural pressure induce swings in the arterial pulse, which are known as the pulsus paradoxus. A pulsus paradoxus is classically observed during asthma crisis, and its magnitude is known to reflect the severity of the attack. Cyclic respiratory changes in the arterial pulse are reflected by proportional changes in the pulse oximetry waveform (4). We are well aware that the magnitude of the respiratory swings in the pulse oximetry waveform (also known as the Pleth Variability Index [PVI]) is dependent on volume status and may significantly increase during surgical bleeding (5). However, in patients with acute respiratory failure, PVI depends almost exclusively on the magnitude of changes in pleural pressure (i.e., on the respiratory effort). In this respect, PVI has been proposed to assess the expiratory effort in patients with airway obstruction (6). We believe it may also be used to assess the inspiratory effort during acute respiratory failure related to bacterial or viral pneumonia. All hypoxemic inpatients with COVID-19 are monitored with a pulse oximeter that, in addition to oxygen saturation, could be used to quantify their pulsus paradoxus or PVI (Figure 1). In other words, PVI monitoring may constitute an elegant and practical alternative to the quantification of ΔPes and assist clinicians in the timing of tracheal intubation. Studies are needed to confirm this hypothesis and to clarify which PVI cutoff value would correspond to the best discriminative value of 10 cm H2O reported by Tonelli and colleagues (1) for ΔPes.

Figure 1. Modern pulse oximeters enable the monitoring of the respiratory pleth variability (pulsus paradoxus or Pleth Variability Index). PVI = Pleth Variability Index; SpO2 = oxygen saturation as measured by pulse oximetry.

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CORRESPONDENCE

Reply to Tuffet et al. and to Michael and Shelley

From the Authors:

We received with great interest the letters by Tuffet and colleagues and by Michaud and Shelley commenting on our work (1), which allows us to further explore the main findings of the study.

Tuffet and colleagues correctly noted that the different magnitude of dynamic transpulmonary pressure, as recorded after 2 hours of noninvasive ventilation (NIV) trial and compared with the baseline level of esophageal pressure swing, suggests different ventilator’s adjustments between patients who succeeded or failed NIV. In particular, the median value of pressure support (PS) set at 2 hours was different in the two groups (P < 0.001), whereas positive end-expiratory pressure level was not (Figure 1). With regard to this point, we have to stress that our study was observational and that NIV setting was adjusted by the attending physician who was blind to the study purpose and the physiological observations and that NIV setting was adjusted by the attending physician. Therefore, we agree with Tuffet and colleagues that the esophageal manometry may decrease respiratory drive through the respiratory muscles’ unloading, there may be a subset of patients with acute respiratory distress syndrome in whom PS would not influence the magnitude of effort. Therefore, esophageal pressure monitoring will allow the optimization of ventilatory setting to target the best level of pressure on one hand and the identification of those patients whose effort would not be affected by NIV on the other.

Michaud and Shelley questioned the reproducibility of esophageal manometry because of the supposed scarce patient’s tolerance. We have used a nasogastric tube equipped with an integrated esophageal balloon (NutriVent) that did not modify the size of the probe and was thus less likely to reduce the patient’s tolerance. In addition, the oronasal face mask equipped with a dedicated output for probes (BluestarTM, KOO Medical Equipment) was able to minimize air leaks because of the tube placement. Furthermore, our patients needed prolonged NIV support, so gastric insufflation might occur as a common treatment complication (range, 10–50%) (6), even sonographically detectable at lower inspiratory pressures (e.g., 10–15 cm H2O) in up to 35% of patients (7). In line with these evidences, the insertion of a nasogastric tube during NIV has the theoretical advantage of reducing aerophagia and gastric distension, thus improving the patient to ventilator interaction. These colleagues also discussed the potential application of the pulse oximetry waveform in estimating the magnitude of inspiratory effort and suggested the use of Pletth Variability Index monitoring to assist clinicians in prompting intubation. This would allow a noninvasive, real-time, and easy-to-apply technique to quantify the patient’s respiratory swing. However, in the hypothesis that self-inflicted lung injury might constitute a major component of NIV failure, our study aimed at objectifying the influence of dynamic transpulmonary pressure and esophageal pressure swings on clinical outcome with this setting. Therefore, we relied on manometry as the optimal procedure to obtain reliable and objective measurements of inspiratory effort during spontaneous breathing (8). Although physiological investigations have addressed the rationale for pulse oximetry waveform analysis in spontaneously breathing healthy individuals (9) and in patients undergoing NIV (10), calibration of this measure is rather difficult to obtain, and cutoff values are not available from clinical studies so far. Furthermore, it is likely that inadequate peripheral perfusion, intrathoracic or total blood volume, and arrhythmias would significantly affect Pletth Variability Index monitoring under this condition, as described for all the noninvasive cardiovascular monitoring (11). Reproducible, well-standardized, but still to come, less invasive techniques to assess the patient’s respiratory effort are more than welcomed to enhance their usage in the critical care setting.

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