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Interdependence between elevated intra-abdominal, pleural, and airway opening pressure in severe acute respiratory distress syndrome with extracorporeal membrane oxygenation

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Editor—Detection of airway opening pressure (AOP) above atmospheric pressure in patients with acute respiratory distress syndrome (ARDS) is a simple bedside measure with relevant physiological and clinical consequences.[1] Airway opening pressure is the threshold level for start of alveolar inflation, and when it is higher than externally set PEEP, undetected AOP can lead to overestimation of driving pressure[2] and to underestimation of the potential for lung recruitment.[3] Several mechanisms contribute to the development of elevated AOP during ARDS, such as impaired surfactant.[4,5] The recent coronavirus disease 2019 (COVID-19) pandemic led to a dramatic surge in intubated patients with ARDS admitted to intensive care.[6] The number of patients, the stress on healthcare workers, and the need for careful isolation limited the ability to perform extensive clinical and physiological testing in these patients.[7] We present here unique physiological measures on the interdependence between AOP, pleural pressure (P_{pl}, estimated from oesophageal pressure), and intra-abdominal pressure (IAP) obtained by standard bedside monitoring in a patient affected by COVID-19 ARDS fully supported by extracorporeal membrane oxygenation (ECMO). The institutional ethics board of Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Italy approved this study. The data used or analysed are available from the corresponding author on reasonable request.

A 46-yr-old intubated patient was admitted to the ICU of the Ospedale Maggiore Policlinico with a diagnosis of severe ARDS from severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection confirmed by reverse transcription–polymerase chain reaction. He had no comorbidities except for mild depressive state and obesity (BMI=32). After 8 days, oxygenation had not improved despite multiple sessions of prone positioning and administration of nitric oxide, and decreased compliance hindered application of protective mechanical ventilation. Thus, veno-venous ECMO was started through a femoral–femoral approach. Tidal volume (V_t) was reduced to keep driving pressure <14 cm H_2O and ventilatory frequency (VF) to 10 bpm with PEEP of 15 cm H_2O. Ten days later, respiratory conditions deteriorated further as a result of superinfection and alveolar bleeding, and a second ECMO system was added through a double-lumen jugular approach to produce peripheral oxygen saturation >80%. On ECMO Day 18, the patient developed abdominal compartment syndrome and oliguria. Possible causes included intestinal obstruction from opioid-induced constipation, hypoperfusion, or SARS-CoV-2 infection itself.[8,9] The patient was ventilated on pressure-controlled mode, and as V_t became minimal (0.7 ml kg^{-1} predicted body weight; Supplementary Table S1) in association with development of abdominal hypertension, we performed comprehensive bedside physiological measures to monitor the clinical evolution and confirm

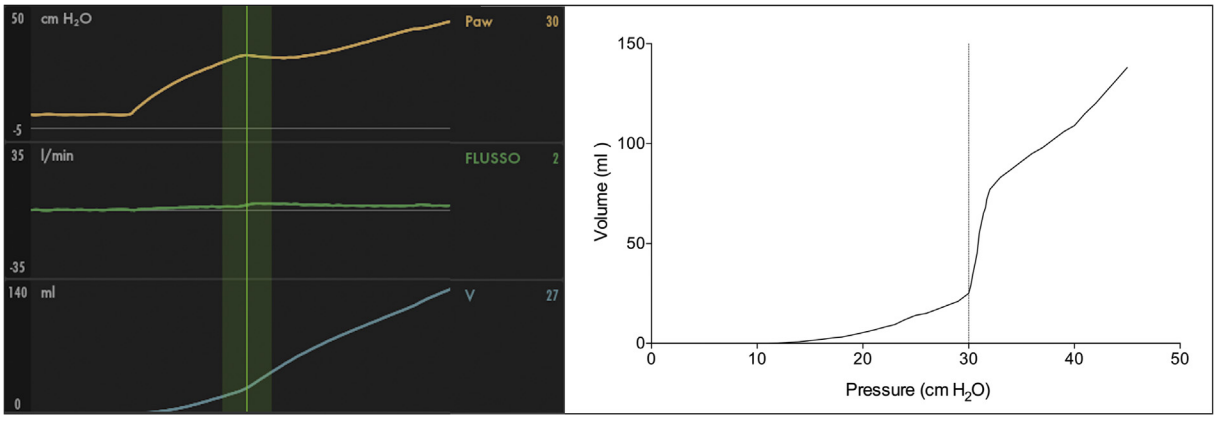

Fig. 1. Airway opening pressure (AOP). On the left, the airway pressure, flow, and volume *vs* time waveforms from the ventilator. The cursor indicates the AOP at 30 cm H_2O. On the right, the *P*–*V* curve obtained by manual plotting of values obtained from the ventilator (see text for details). The dashed line shows the steep increase in the slope of the curve at the AOP (30 cm H_2O).

physiological mechanisms. After 51 days, the patient died as a result of septic shock.

Using a standard ICU ventilator (Servo-u®; Getinge, Solna, Sweden), we were able to measure total PEEP, plateau pressure, and V_t under clinical settings through end-inspiratory and end-expiratory occlusions. Keeping in mind that classic respiratory mechanics can be misinterpreted in the presence of AOP higher than PEEP, driving pressure was 12 cm H_2O, plateau pressure was 31 cm H_2O, and respiratory system compliance was 4.6 ml cm H_2O^{-1} (Supplementary Table S1). Measurement of calibrated oesophageal pressure (Esophageal Balloon Catheter Set; Cooper Surgical, Trumbull, CT, USA)[10] using the haemodynamic monitor during occlusions allowed us to measure end-inspiratory and end-expiratory P_{pl}. P_{pl} was extremely elevated, and the end-expiratory value was 30 cm H_2O. The end-expiratory transpulmonary pressure was −11 cm H_2O with a driving transpulmonary pressure of 11 cm H_2O and an elastance-derived inspiratory transpulmonary pressure of 28 cm H_2O (Supplementary Table S1). A switch to volume-controlled mode with long inspiratory time (12 s), low VF (4 bpm), and V_t >300 ml allowed us to perform a low-flow pressure–volume (*P*–*V*) curve between 5 and 45 cm H_2O, and to detect the AOP at the bedside (Fig. 1, left). We also manually collected the *P*–*V* values every 3 cm H_2O and to form the classical *P*–*V* curve by commercial software (Fig. 1, right), which confirmed an AOP value of 30 cm H_2O. The compliance of the respiratory system between 5 and 30 cm H_2O was similar to the airway circuit (1.5–2 ml cm H_2O^{-1}). Recognition of an upper inflection point around 40 cm H_2O may indicate over-distention of the ventilated regions, further suggesting that standard protective ventilation in this patient would have been impossible.

Finally, 50 ml of normal saline was infused through the urinary catheter into the bladder. Then, the catheter was clamped and IAP was recorded at end expiration, showing a value of 30 cm H_2O. Measures of IAP and AOP were repeated in the following days: as the abdominal condition improved, IAP gradually decreased, and after 1 week IAP was 15 cm H_2O and AOP was 19 cm H_2O.

In severe COVID-19 ARDS supported by ECMO, standard bedside physiological monitoring allowed us to show equalisation of extremely elevated IAP, P_{pl}, and AOP in the presence of abdominal compartment syndrome. The interdependence between IAP and AOP values was lost as the abdominal condition improved, with AOP remaining steadily high despite return to normal IAP values.

Airway closure in ARDS might result from elevated pleural pressure with reduced lung size causing compression of distal airways[4] or depletion of surfactant with high surface tension causing airway collapse.[5] Moreover, pleural pressure can be increased by external (e.g. abdominal pressure) or intrinsic (e.g. increased lung weight) forces.[8] Perfect correspondence between IAP, P_{pl}, and AOP has not been reported previously and generates the hypothesis that the presence of extremely elevated IAP applied to a closed system (rigid abdominal and chest wall and collapsed non-ventilated lung) can become the only external force determining elevated pleural pressure and AOP. The persistence of elevated AOP even after the decrease of IAP may suggest that other mechanisms related to intrinsic compressive forces and surfactant dysfunction became predominant. Even though previous studies have not reported correspondence between AOP and pleural pressure,[1] the almost complete lung collapse as a result of profound hypoventilation leading to a virtually closed intrathoracic system may explain the pressure equalisation that we observed. From a clinical point of view, the measurement of AOP may be useful to personalise PEEP or to perform further diagnostic investigations targeted at intestinal function.

The physiological measures in this COVID-19 ARDS patient supported with ECMO suggest a new mechanism for elevated AOP: extremely elevated IAP is transmitted to the pleural space and, in the presence of complete lung collapse, becomes the threshold value for airway opening.

Declarations of interest

TM received personal fees from Fisher & Paykel, Dräger, and Mindray outside of the present work. GG received payment for

lectures from Draeger Medical, Getinge, Fisher & Paykel, Biotest, and Thermo Fisher, and travel/accommodation/congress registration support from Getinge and Biotest, which are all outside of the present work. AP reports personal fees from Maquet, Novalung/Xenios, Baxter, and Boehringer Ingelheim, all outside the submitted work. Other authors have no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bja.2020.06.044.

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Opening Pandora's box: surgical tracheostomy in mechanically ventilated COVID-19 patients

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Keywords: acute respiratory distress syndrome; aerosol-generating procedure; COVID-19; ICU; mechanical ventilation; surgical tracheostomy

Editor—Coronavirus disease 2019 (COVID-19) is the third coronavirus infection witnessed in past two decades.[1] Key lessons derived from the cumulative experience of Asian and European ICUs, which dealt with critically ill patients during the early phase of the COVID-19 pandemic, and lessons from the previous severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) outbreaks allowed hospitals in the UK to prepare preventive measures and adopt specific processes for the COVID-19 crisis.[2] Proactive management strategies implemented during this crisis based on previous knowledge may have limited the number of COVID-19-related hospital admissions, reduced mortality, and limited the initial trajectory of coronavirus spread in the UK.

Our hospital has seen an increase in demand for open surgical tracheostomies in COVID-19 patients admitted to the ICU. In contemporary critical care practice the most common