Electrical impedance tomography and trans-pulmonary pressure measurements in a patient with extreme respiratory drive

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A B S T R A C T
Preserving spontaneous breathing during mechanical ventilation prevents muscle atrophy of the diaphragm, but may lead to ventilator induced lung injury (VILI). We present a case in which monitoring of trans-pulmonary pressure and ventilation distribution using Electrical Impedance Tomography (EIT) provided essential information for preventing VILI.

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1. Introduction

Mechanical ventilation is a critical part of intensive care medicine, but is also known to induce injury to both the lungs and respiratory muscles. In order to minimize injury, protective ventilation strategies are employed based on low tidal volume and relative low levels of PEEP.

During controlled mechanical ventilation, when spontaneous breathing (SB) is absent, atrophy of diaphragm muscle cells has been found already after 12–72 hours after onset of mechanical ventilation [1,2]. Therefore, it has been recommended to maintain diaphragmatic activity and keep respiratory effort intact [3]. In addition, SB has been shown to improve dorsal ventilation, alveolar recruitment and oxygenation by reducing atelectasis, histological damage and cytokine production [4–10].

However, studies have demonstrated that a strong inspiratory effort can cause high trans-pulmonary pressures (P_{TP}), leading to increase in damage to the already injured lung. Yoshiida et al. [11] showed in a rabbit model that SB was beneficial during mild lung injury, but harmful during severe lung injury. This was confirmed in patients with severe ARDS in which the early use of neuromuscular blocking agents (NMBA’s) reduced the risk of barotrauma and improved survival [12].

Extra-corpooreal membrane oxygenation (ECMO) negates the need for mechanical ventilation, allowing for more protective ventilation strategies.

To accomplish a persistent protective ventilation strategy in patients with severe hypoxemia, the monitoring of respiratory parameters is essential, such as P_{TP} [14–16] or electrical impedance tomography (EIT) [17].

We report the case of a patient with interstitial (post-)infectious lung injury who required, as a protective lung strategy, both NMBA’s and ECMO during his stay on the Intensive Care Unit (ICU).

2. Case report

The patient was a 24 years old male with no known medical history. Almost three weeks before admission, the patient went swimming in natural water and consequently developed a cough, pain while coughing and bloody sputum. He was prescribed antibiotics (amoxicillin) by his general practitioner. One week later he was admitted to the ICU department due to progressive respiratory insufficiency and increased work of breathing. He received high flow nasal cannula oxygen therapy (OptiFlow, 35 L/min, F_{IO2} 0.6), but had to be intubated the next day due to fatigue and was ventilated using pressure support mode. High peak pressures (34 cmH_{2}O) with a PEEP of 14 cmH_{2}O and F_{IO2} of 0.7 had to be applied, resulting in a PaO_{2} of 70 mmHg, SaO_{2} of 96%, PaCO_{2} of 59 mmHg and pH of 7.37. X-ray and CT imaging were assessed as bilateral tree-in-bud sign (Fig. 1). After two days of mechanical ventilation, patient was transferred to our hospital (reference center for ECMO) with the question if patient was a candidate for vv-ECMO. Since no diagnosis could be made, broad-spectrum antibiotics were given (cefotaxime, ciprofloxacin, doxycycline) and steroid therapy (methylprednisolone, 160 mg/day) was started for suspected eosinophilic pneumonia.

The patient was ventilated using pressure support ventilation with a PEEP of 18 cmH_{2}O and a peak airway pressure of 28 cmH_{2}O respectively. PaO_{2} and PaCO_{2} were both of 70 mmHg with 50% oxygen at that time. The tidal volume reached values up to 12 mL/...
kg ideal body weight due to strong inspiratory effort. Propofol and remifentanil were increased from 240 to 400 mg/h and from 0.15 to 0.26 μg/kg/h respectively in an attempt to reduce tidal volume. Also, the ventilator mode was switched to volume-controlled mode with a tidal volume of 480 mL (6.4 mL/kg ideal body weight). The patient had persisting spontaneous efforts in a 1:2 ratio: one spontaneous effort during every second mandatory breath (Fig. 2A). An esophageal balloon catheter (NutriVent, Sidam S.R.L., Mirandola, Italy) was inserted and $P_{TP}$ was calculated. $P_{TP}$ was 34 cmH$_2$O during inspiration and $-9$ cmH$_2$O at end expiration (Fig. 2A). Simultaneously electrical impedance tomography (EIT) measurements were performed at the 5th intercostal space (Fig. 3).

On day 4 after intubation, a bolus of rocuronium (80 mg) was administered intravenously followed by a continuous infusion (30 mg/h) due to the high $P_{TP}$. The ventilator mode was switched to pressure-controlled ventilation with a PEEP of 18 cmH$_2$O and peak airway pressure of 28 cmH$_2$O. $P_{TP}$ decreased from 34 to 19 cmH$_2$O during inspiration and $P_{TP}$ increased from $-9$ to $-2$ cmH$_2$O at end expiration (Fig. 2B and C). TIV decreased by 65% and was more homogeneously distributed (Fig. 4). The predominant distribution of inspired gas to the dependent part during an almost entire inspiratory phase dramatically changed to even distribution to both parts after administration of rocuronium.

Despite the more protective ventilation strategy, gas exchange deteriorated ($PaCO$_2$ of 120 mmHg, PaO$_2$ of 68 mmHg with an FiO$_2$ of 0.7$). In an effort to improve gas exchange, the patient was put in prone position on day five of mechanical ventilation, without success. Immediately thereafter, the patient was put on veno-venous ECMO via the femoral vein and internal jugular vein. Blood flow was 5 L/min with a sweep gas flow of 2.5 L/min. Blood gases improved quickly (SaO$_2$ 91%–94%, PaO$_2$ kept stable around 60 mmHg with an FiO$_2$ of 0.3, pH 7.17 to 7.53, PaCO$_2$ 120 to 42 mmHg). During ECMO, ventilation was continued on pressure-controlled mode, with PEEP decreased from 18 to 14 cmH$_2$O and peak pressure from 28 to 24 cmH$_2$O, resulting in tidal volumes of 2.4 mL/kg. During the next five days, PEEP was reduced to 10 cmH$_2$O and peak pressure to 20 cmH$_2$O. The sedation was lowered in order to allow spontaneous breathing resulting in tidal volumes of 4.6–9.3 mL/kg. In the meanwhile, blood culture became positive for the presence of leptospirosis, for which the patient was already being treated with cefotaxime and doxycycline.

The patient slowly improved and was weaned from ECMO after 12 days (day 15 after intubation). He was weaned from the ventilator within the next five days. Another ten days later he was discharged to the ward and finally discharged from the hospital 56 days after initial admission. A definitive diagnosis was missing, but an organizing pneumonia was mentioned as the most probable cause.

3. Discussion

In this case report we describe a patient who showed great inspiratory effort during assisted mechanical ventilation. The inspiratory effort resulted in large trans-pulmonary pressure. This was dramatically decreased after the administration of muscle relaxation, also resulting in a more even distribution between the dependent and non-dependent lung as shown by EIT.

On the one hand one likes to encourage persistent diaphragmatic activity in order to prevent VIDD. On the other hand, it is known that an increased respiratory drive — resulting in high $P_{TP}$ — causes mechanical stress in the lung, which may contribute to ventilated-induced lung injury (VILI). In an experimental sheep study, induced spontaneous hyperventilation induced reduced compliance and severe lung edema, finally leading to death, whereas none of the sheep who received muscle relaxant after induced hyperventilation and controlled mechanical ventilation with a fixed low tidal volume developed lung edema and all animals survived [22]. Another experimental study showed that the same level of inflation as during spontaneous breathing (6 mL/kg), during muscle paralysis 2–3 times higher driving pressures (28 vs 10 cmH$_2$O) and 2–3 times larger tidal volumes (15 vs 6 mL/kg) were required, indicating overstretching of the dependent lung regions due to spontaneous effort [23]. Similarly, in this case the TIV in the dependent lung regions before administration of an NMBA was 3.8 times higher as compared to the TIV after NMBA administration. Additionally, the peak $P_{TP}$ was 10 cmH$_2$O higher than the physiological limit of 25 cmH$_2$O [16], but was reduced to below this physiological limit after NMBA administration.

These findings suggest that muscle paralysis plays an important role as part of the ventilation strategy to decrease the risk of VILI in patients with significant spontaneous effort. It is apparent that the protective effect of muscle paralysis is case-specific and requires extensive monitoring [24]. Monitoring of ventilation distribution may be important in all patients with high respiratory effort for clinical decision-making regarding the ventilation strategy, such as
prostate positioning and application of lung-replacing therapy like ECMO.

Although the spatial resolution is limited, EIT can accurately visualize changes in aeration and regional ventilation [19,25]. Most importantly, it is non-invasive and more readily available compared to the golden standard, computed tomography imaging. In the case we presented, EIT imaging clearly showed inhomogeneous ventilation indicating overstretch in the dorsal parts of the lung. Measurement of the PTP confirmed that the inhomogeneous ventilation distribution was associated with increased pressure surpassing physiological limits. PTP measurements are regarded as an important value to respiratory monitoring [14,16,24,26–28]. The esophageal pressure used to calculate PTP is generally overestimated in supine patients, requiring a correction of approximately 5 cmH2O [14]. The PTP that is presented in this report are not corrected for that underestimation. However, a difference of approximately 5 cmH2O does not change the interpretation of the values of the PTP or the chosen course of treatment.

In summary, administration of NMBA’s increased the homogeneity of ventilation distribution and reduced peak PTP in a patient with strong spontaneous effort. Inhomogeneous ventilation can be detected at the bedside using EIT imaging. Monitoring of the regional ventilation and trans-pulmonary pressure could provide useful information in determining personalized ventilation strategies.

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