Thoracic electrical impedance tomography to minimize right heart strain following cardiac arrest

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ABSTRACT

Titrating ventilator settings to minimize pulmonary arterial pressures and optimize both ventilation and oxygen delivery can be challenging following cardiac arrest. Erroneous ventilator adjustments can lead to unnecessary strain on the right ventricle that may be particularly vulnerable during the acute recovery. We report a child with fulminant myocarditis who was mechanically ventilated using thoracic electrical impedance tomography to optimize regional lung inflation and possibly curtail right ventricular afterload following cardiac arrest.

Keywords: Cardiac arrest, children, electrical impedance, mechanical ventilator, myocarditis

INTRODUCTION

Although the clinical manifestations of myocarditis are relatively uncommon,[1] it is one of the most common causes of sudden cardiac death without recognized heart disease.[2] Its fulminant form is associated with high requirements of conventional mechanical ventilation (CMV) with inotropic and/or extracorporeal membrane oxygenator support[3] and mortality rates ranging from 9% to nearly 50%.[4] Despite the substantial cardiovascular effects of CMV,[5] the current management guidelines for pediatric myocarditis[4,6,7] do not include an approach for positive pressure ventilation amidst right ventricular insufficiency.

Thoracic electric impedance tomography (EIT) is a noninvasive imaging modality that can assess real-time regional lung volumes without harmful radiation.[8] In adults, it has been utilized in postoperative cardiac surgery patients to target positive end-expiratory pressures (PEEPs) that were associated with optimal regional ventilation distributions.[9] Improving these distributions should impede high pulmonary vascular resistance by mitigating alveolar hypo- or hyperinflation. We report a child who lost spontaneous circulation secondary to fulminant myocarditis and was mechanically ventilated using electrical impedance tomography to possibly curtail right ventricular afterload.

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CASE REPORT

An otherwise well 18-month-old girl who had a recent history of bocavirus bronchiolitis presented to the emergency department with a 1-day history of increased work of breathing, tachypnea, and a productive cough. Her physical examination was documented positive for coarse crackles and a mild wheeze. Her chest roentgenogram demonstrated a small right pleural effusion. She was admitted to the pediatric ward with a diagnosis of a viral lower respiration infection and the following vital signs: blood pressure, 110/68 (81) mmHg; heart rate, 148; respiratory rate, 55; and oxygen saturations, 99% on room air and a temperature of 36.5°C.

Twelve hours after admission, she experienced a pulseless electrical activity cardiac arrest. Her resuscitation was 26 min long, consisting of chest compressions, five intravenous doses of epinephrine, bag-mask

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ventilation, and intubation. Her liver was noted to be 5 cm below the costal margin after receiving 20 ml/kg of intravenous isotonic crystalloid. Pertinent laboratory parameters following the arrest included significant acidemia (arterial blood gas pH, 6.99; lactate 12.1, mmol/L), central venous saturation of 47%, elevated international normalized ratio (4.4), mild ischemic transaminitis, C-reactive protein of 3.9 mg/L, and evidence of significant myocardial injury (troponin high sensitivity, 80.3 ng/L; N-terminal prohormone of brain natriuretic peptide, >70,000 pg/mL).

She was promptly transferred to the pediatric intensive care unit where epinephrine and milrinone infusions were initiated and adjusted to support the mean arterial pressures >45 mmHg. A 12-lead electrocardiogram showed sinus tachycardia with nonspecific T-wave and ST-segment changes in the precordial leads. Her echocardiogram demonstrated significant biventricular dysfunction, mild-to-moderate tricuspid regurgitation, moderate mitral regurgitation, and an enlarged left ventricle with an ejection fraction of 23%.

She was supported on a Hamilton-G5 ventilator (Hamilton Germany GmbH), with the following settings on Adaptive Pressure Ventilation (APV-CMV): PEEPs of 7 cmH_2O; fraction of inspired oxygen (FiO2) of 60%; respiratory rate of 35; and set tidal volume of 5.0 ml/kg based on ideal bodyweight providing peak pressures of 28 cmH_2O. Her dynamic lung compliance (C_{dyn}) was measured at 3.7 ml/cmH_2O. An hour postadmission, her ventilation was assessed with EIT (Dräger Pulmovista 500, Drägerwerk AG and Co. KGaA, Lübeck, Germany).

Initial EIT measurements showed that the majority of ventilation (79%) was distributed to the ventral regions [Figure 1]. A recruitment maneuver was then performed using incremental PEEPs and constant driving pressure to minimize hemodynamic effects.

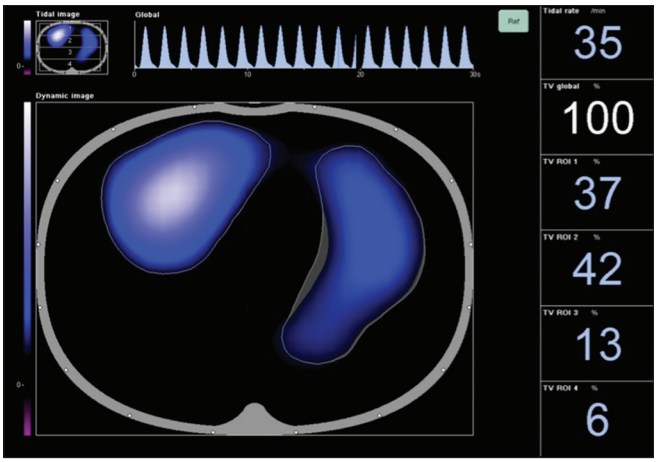

Figure 1: Initial PEEP at 7 cmH_2O. Distribution of ventilation primarily to ventral regions (ROI 1 + ROI 2 = 79%). PEEP: Positive end-expiratory pressure

The PEEP was increased until dorsal ventilation on EIT no longer improved. A decremental PEEP titration was performed until a decreasing change in end-expiratory lung impedance was identified, signifying continuing loss of end-expiratory lung volume at the set PEEP level. Using EIT, the PEEP trial estimates the regions of overdistension and collapse and identifies the level that mitigates both [Figure 2]. Our trial revealed an optimal PEEP of 12 cmH_2O, which resulted in an increase in dorsal ventilation to 30% [Figure 3], and in C_{dyn} to 4.9 ml/cm H_2O, and a decrease of FiO_2 – 40%, heart rate (179–151 bpm) and mean arterial blood pressure (78–74 mm Hg).

After 12 h, her ventilator settings remained stable. Her epinephrine was weaned and corresponded with improvements of central venous saturations (75%) and lactemia (<2 mmol/L), and there were no dysrhythmias. A day later, her laboratory results revealed that she was parvovirus IgM positive. She steadily convalesced, was not treated with immunoglobulins or steroids, and was discharged home 2 weeks later, neurologically intact, with cardiology follow-up.

DISCUSSION

Following cardiac arrest in children with fulminant myocarditis, the right ventricle is compromised through a variety of mechanisms. At a cellular level, the degree of inflammation coupled with ischemic reperfusion injury causing cytokine cascades and free radical reactions may dramatically influence recovery. Furthermore, afterload may be markedly elevated because of pulmonary venous congestion, contusions and atelectasis following chest compressions, and the possibility of regional hyperinflation with bag-mask ventilation and/or conventional mechanical ventilation (CMV).

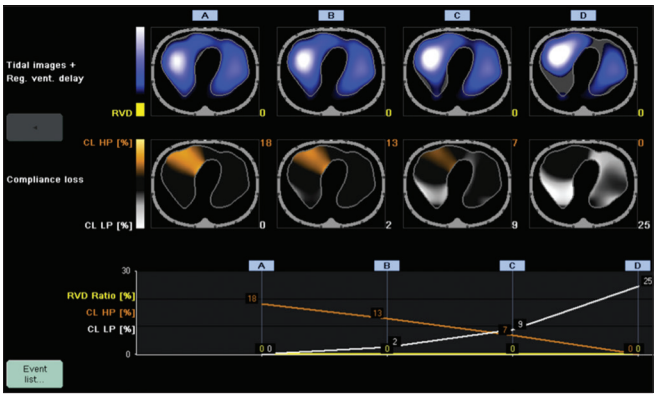

Figure 2: Optimal PEEP determination. Decremental PEEP trial highlighted at 15 (A), 13 (B), 10 (C), and 8 (D) cmH_2O. Orange pixels show decreased compliance toward highest PEEP levels (CL HP%) signifying overdistention, and white pixels show decreased compliance toward lowest PEEP levels (CL LP%) signifying collapse. Intersection of CL HP% and CL LP% is between B and C, suggesting optimal PEEP between 10 and 13 cmH_2O. PEEP: Positive end-expiratory pressure

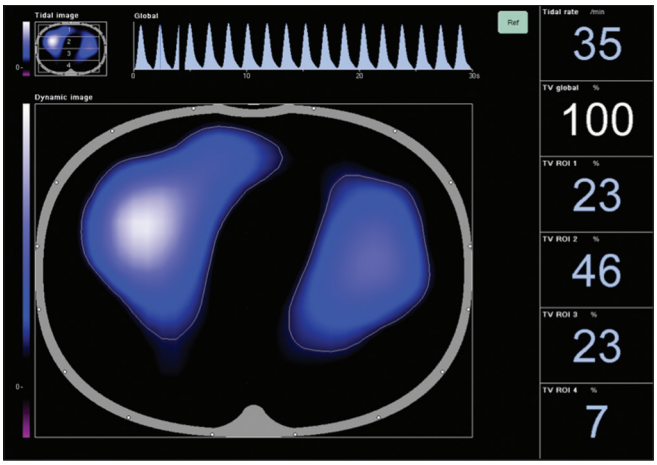

Figure 3: Optimal PEEP at 12 cmH_2O. PEEP of 12 cmH_2O showing an overall improvement in ventilation distribution, with a shift to toward the dorsal regions (ROI 3 + ROI 4 = 30%). PEEP: Positive end-expiratory pressure

The fear related to increasing PEEP following cardiac arrest is warranted, given the traditional view that positive pressure ventilation increases mean airway pressure and subsequently right ventricular afterload. Our experience with EIT challenges this notion on two fronts. First, careful lung recruitment maneuvers may be followed by an optimal PEEP that is higher than the baseline. However, the improved lung compliance usually decreases the driving pressure (ΔP or peak inspiratory pressure – PEEP) while minimizing increases in mean airway pressure. Our patient's C_{dyn} and Vt improved by 40% and 33%, respectively, which supports the premise of optimizing and potentially increasing PEEPs in this scenario. Second, pulmonary vascular resistance is at its lowest when the lungs are at functional residual capacity.[5] This translates to optimal regional ventilation without areas of hypo- or hyperinflation. In our patient, this was achieved by a 63% increase of dorsal ventilation without concomitant ventral hyperinflation.

The idea of titrating CMV in a postresuscitated right ventricle without real-time regional lung data is potentially dangerous, especially given the availability of bedside EIT. Recommendations to optimize cardiopulmonary recovery may include utilizing EIT (together with conventional medical therapy) for initial CMV settings and with all ventilator adjustments throughout the recovery phase. Optimizing PEEP to attenuate pulmonary vascular resistance may also be very applicable with other etiologies of cardiac arrest and in postoperative cardiac patients with significant right ventricular dysfunction or single ventricle lesions with pulmonary over/undercirculation.

CONCLUSION

The deleterious effects of CMV on the right ventricle following cardiac arrest from fulminant myocarditis should not be overlooked. EIT has the potential to mitigate these effects by guiding ventilator adjustments that impede right ventricular deterioration and facilitate right-sided afterload reduction.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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