EDITORIAL

High-frequency Nasal Cannula Oxygenation in Thoracic Trauma: Unrealistic Expectations?

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The Global Disease Burden study 2019 assessed the trends of severe thoracic trauma over 29 years and found that the number of severe chest injury cases reached 7.95 million in 2019.[1] While we do not have national epidemiological data from India, Narayanan et al., published a 3-year analysis of traumatic chest injury cases which presented to a Level I Trauma Center.[2] Chest injuries were present in 30.9%, with blunt chest trauma (BCT) being the mechanism of injury in over 83% and the mortality rate was 11%. Commonly chest injuries, (more often blunt, but also penetrating) present with rib fractures (with or without flail chest), pneumothorax, hemothorax, and hemopneumothorax, lung contusion, cardiac, and major vascular injuries. Less commonly sternal fractures and esophageal rupture are also seen. Thoracic trauma patients with acutely non-fatal injuries are susceptible to hypoxia due to a variety of mechanisms. Chest wall disruption due to rib fractures (flail) leads to paradoxical respiration, decreased lung compliance, aggravated swings in pleural pressure, impaired ventilation and perfusion matching oxygenation, and flooding of alveoli due to underlying contusion. A 75-year-old review, which used the term "crush injuries" of the chest, described these mechanisms in great detail.[3]

Cullen et al., published retrospective data about patients with flail chests comparing controlled mandatory ventilation, intermittent mandatory ventilation (IMV), and IMV with PEEP.[4] The duration of mechanical ventilation and ICU LOS was lower in patients given IMV and IMV with PEEP. They suggested that the problem being treated in these patients was not structural (i.e., flail), but rather due to an underlying ARDS-like condition (i.e., pulmonary contusion). Maintaining spontaneous breathing during IMV and PEEP helped by restoring the lung volume, improving VQ mismatch, restoring functional residual capacity, and decreasing the shunt. They also suggested that shortening the duration of ventilation helped the patients by reducing the infectious complications.

Tanaka et al. started using CPAP when they deemed epidural analgesia alone was not enough for managing patients with flail chests.[5] They suggested that the improvement in outcomes in their patients was due to what they termed as "internal pneumatic stabilization."

A randomized controlled trial compared CPAP delivered with mask and regional analgesia to invasive ventilation with PEEP.[6] Mask CPAP led to decreased duration of ventilation (4.5 ± 2.3 vs 7.3 ± 3.7, $p = 0.0003$) d, ICU LOS (5.3 ± 2.9 vs 9.5 ± 4.4, $p \leq 0.0001$) d, and Hospital LOS (5.3 ± 2.9 vs 9.5 ± 4.4, $p \leq 0.0001$) d. Most importantly, non-invasive CPAP also reduced the number of patients with pneumonia as well as overall infectious complications. The physiological mechanism of utility of CPAP with and without multiple rib fractures and acute lung injury was elegantly demonstrated by Schweiger et al.[7] Lung injury was induced with oleic acid in a porcine model. They created the model to evaluate the effect of CPAP (4–22 cmH_2O) on the shunt fraction, alveolar mechanics, V/Q matching, and CVs The lung compliance increased when PEEP was titrated upwards and reached pre-fracture or baseline status 60 mins of PEEP titration (to maintain $SpO_2 >$ 90%). With the application of CPAP, shunt and venous admixture decreased, and functional dead space returned to near normal. There was minimal effect on the stroke volume and mean aortic pressure. The pathophysiological and clinical evidence confirms that ensuring adequate delivery of PEEP or CPAP (i.e., PEEP where the patient breaths spontaneously) is essential for supporting these patients. With improvement in oxygenation, there should be a decreased need for tracheal intubation and invasive mechanical ventilation. Historically, increasing flows while delivering high-flow oxygen by masks or cannulas has been problematic due to the technical inability to humidify and heat the gases. Waugh and Granger evaluated two high-flow oxygen devices for their ability to humidify nasal cannula devices to reach body temperature and pressure saturated (BTPS i.e., 37°C and 100% relative humidity or absolute humidity of 43.9 mg H_2O/L) in a bench study.[8] They found that while the Vapotherm device achieved this target, the Salter Labs unit did not, reaching only 72–78% relative humidity, (flows between 5 and 15 L/min), and at ambient but, not at body temperature. This study opened the doors for the use of fully heated and humidified oxygen at high flows.

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Several mechanisms contribute to the increase in alveolar PaO_2 following the application of HFNCO: Heating and humidification of delivered gas, replacement of dead space gases with oxygen-enriched gases, improving gas mixing in large airways, and generation of positive pressure in the airways.[9] Parke et al. studied the effect of flow during HFNCO on the PEEP generated in patients after cardiac surgery, with the mouth open and closed, and found that the amount of PEEP generated was 0.35 cmH_2O/10L flow with mouth open, whereas it was 0.70 cmH_2O/10L flow when the mouth was closed.[10] It thus appears that the application of HFNCO cannot generate PEEP > 4 cmH_2O. While healthy volunteers can keep their mouths closed during physiological studies, one would not expect sick patients to be able to do this, while struggling to breathe. While increasing the flow rate does decrease the work of breathing, it does not do this consistently across all patients by generating PEEP to counter the intrinsic PEEP.[11]

In this issue of the journal, Elsayed et al., present the results of a randomized controlled trial comparing HFNCO with oxygen supplantation using a Ventury mask in patients with pulmonary contusion following thoracic trauma.[12] Most patients (90%) had rib fractures in both groups and underlying unilateral unilobar pulmonary contusion (83%) and the P/F ratio was < 200. The median Thorax Trauma Severity Score (TTSS) and Wagner and Jamieson lung contusion were similar at baseline and APACHE II scores were quite low (4 in both groups). As expected, there was a significant improvement in P/F ratio within 1 hour in the HFNCO group, it was maintained throughout the study period. HFNCO did not reduce the rate of intubation, invasive ventilation, or mortality.

HFNCO has also been compared to conventional oxygen therapy (COT) or NIV, in earlier studies in patients with BCT. The common findings were an immediate improvement in oxygenation, and reduced ICU and hospital LOS, with no difference in other endpoints such as the need for intubation or mortality. In the OptiTHO trial, early prophylactic NIV was given for 4 hours, followed by HFNCO for at least 48 hour, while the other group received COT and late NIV, in case the PF ratio was reduced to < 200.[13] The trial was stopped after recruiting 141 patients, due to futility, as there was no difference in the primary endpoint, i.e., need for intubation [7% (5/71) vs 8.6% [6/70]), adjusted OR = 0.72 (95% CI: 0.20–2.43), p = 0.60]. The rate of other complications (pulmonary infection, delayed hemothorax, or delayed ARDS) was similar in both groups. At the time of randomization, study patients had high-risk BCT (TTSS ≥ 8), but did not have ARF, but the PF ratio was <300. Zhu et al. compared HFNCO with NIV in patients with mild-moderate ARF in patients with BCT, for treatment failure.[14] While the treatment failure rates were similar [11.1% HFNC vs 16.5% NIV group, risk difference of 5.36% (95% CI: −5.94–16.10%; p = 0.366)] the reasons for failure were different. HFNCO failed when the patients developed respiratory distress, while NIV failed due to NIV intolerance. While one of the most important criteria for the usefulness of new treatment is whether the patient can tolerate the treatment, we must understand that with the NIV, it is the interface that decides whether a patient will tolerate NIV or not. In one study in COVID-19 patients, helmet NIV reduced the need for intubation significantly (10% vs 43.3%, p = 0.004) compared to facemask NIV.[15] The PF ratio was much higher with Helmet NIV (263.57 ± 31.5 vs 209.33 ± 20.5, p = 0.00). The patients were more comfortable with helmet NIV on assessment using a Visual Analogue Score (VAS: 7.20 ± 0.5 vs 4.53 ± 0.629, p = 0.00). The duration of NIV and ICU-LOS were also lower with the helmet interface. In a physiological study in patients with AHRF, Grieco et al. found that helmet NIV improved oxygenation to a greater extent (PF ratio 255, IQR 140–299 vs 138, IQR 101–172; p = 0.001), reduced respiratory rate, inspiratory effort, and dyspnea as compared to HFNCO, while the patient comfort levels were similar.[16] Helmet NIV significantly reduced the rate of intubation in ARDS patients compared to facemask NIV (18.2% vs 61.5%; absolute difference, −43.3%; 95% CI: −62.4% to −24.3%; p < 0.00), and the trial was stopped early due to efficacy.[17]

It therefore appears that the PEEP/CPAP as a part of non-invasive support can prevent the need for intubation; however, NIV rather than HFNCO is more likely to deliver the desired PEEP. Helmet NIV improves oxygenation to a greater extent due to this guaranteed delivery of PEEP and is much better tolerated. While HFNCO may be a promising tool in situations when the patient is mildly hypoxic, using NIV may be more helpful in moderate hypoxia, making the selection of appropriate non-invasive support as per the patient's need.

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