Non-invasive Ventilation in Blunt Chest Trauma

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What is the role of NIV in Adult blunt chest trauma?

INTRODUCTION

Anaesthetists have an important contribution to make in the multidisciplinary care of patients with blunt chest trauma (BCT). It is necessary for us to have an understanding of current concepts of, and evidence for, modalities of care that are administered in the pre-hospital, emergency room, operating theatre and ICU settings.

BCT can result in injury to the thoracic wall, pleura, lung parenchyma, airway structures, major vessels, heart and pericardium, diaphragm and other structures of the mediastinum (e.g. oesophagus, lymphatics). A review of the anaesthetic management of most of the aforementioned injuries is given by Moloney [1], where the need for surgical intervention of the relevant injury under general anaesthesia with invasive mechanical ventilation is obvious.

Current guidelines suggest that the management of flail chest and lung contusion is predominately supportive with select patients being considered for chest wall fixation [2]. Non-invasive ventilation (NIV) as a modality may be applicable, in principle, to a patient that is conscious, maintaining an airway, haemodynamically stable, cooperative and not needing immediate surgery. Thus, as a supportive ventilator modality, NIV is finding its place in the management of lung contusion and flail chest. This is in keeping with a trend towards a more selective approach to ventilator support for patients with gas exchange abnormalities [2, 3, and 4].
EPIDEMIOLOGY

Flail chest occurs when three or more adjacent ribs are each fractured in at least two places. Pulmonary contusion is the associated injury to the underlying lung tissue [2]. In flail chest, the underlying **lunge contusion appears to be the main determinant of acute and long term respiratory dysfunction and a predictor of morbidity and mortality**. This is outlined well by Cohn [5]. He references some of the following citations.

In 1975, Trinkle and coworkers showed conclusively that respiratory insufficiency associated with flail chest was due to the underlying pulmonary contusion rather than paradoxical respiration [4]. They were able to change the prevailing thinking that all patients with flail chest needed tracheostomy and prolonged mechanical ventilation. Their conclusion, that the focus should be on treating the underlying lung contusion, was progressive.

Pulmonary contusion, together with blood loss, crystalloids or steroid therapy decreased the clearance of bacteria from the lung in a canine model [6]. In a large study by Richardson (n=427), patients were treated for 1) flail chest (n=95), 2) pulmonary contusion without flail chest (n=135), 3) haemothorax, 4) pneumothorax or 5) multiple rib fractures [3]. Most were not intubated (n=328). 50% of the flail chest group required intubation and mechanical ventilation compared to 20% of the isolated lung contusion group. The incidence of pneumonia was high (58%).

Pulmonary contusion has been shown to be an important risk factor for Adult Respiratory Distress Syndrome (ARDS). A 2002 study found that ARDS developed in 200 (5%) of 4397 patients with blunt trauma. The strongest predictors of the development of ARDS were an Injury Severity Score (ISS) higher than 25 (area under ROC curve, 0.72) and pulmonary contusion (area under ROC curve, 0.68) [7].

Between 1998 and 2003 the Israel National Trauma Registry reported 11 966 chest injuries (262 flail chest injuries) out of a total of 118 211 trauma hospitalizations [8]. Concerning the flail chest injuries (n=262), 76% were from road crashes and the mortality was 20.6% (n=54). Flail chest mortality was increased with increasing age and severity (ISS>24). Flail chest, traumatic brain injury (TBI) and other major injuries increased the mortality to 61.1%. In each of the diagnosis groups, mortality percentage was higher with flail chest.

The incidence of pulmonary contusion in the Crash Injury Research and Engineering Network (CIREN) data was 21.7% (566 of 2,604) [9]. Crashes resulting in pulmonary contusion had a mortality of 23.9% and an ISS of 33.1 ± 15.7.
An overall mortality of 18.7% was found in patients with blunt chest trauma in a review of prospectively collected data in a level 1 trauma unit for the years 1998–2003 in the UK [10].

No papers reporting the experience of chest trauma in SA units could be found by this author at the time of writing. Chest injuries (n=150) were studied at a hospital in Tanzania [11]. 72.7% had sustained blunt injuries. Assault was identified in 28% of the cases as the cause of injury. The authors did not report on pulmonary contusion as a type of injury. One case of flail chest was noted. Pneumonia, empyema and ARDS occurred in 3.3, 2.7 and 0.7% of cases respectively. No CT chest imaging seemed to have occurred, likely due to resourcing.

Lung contusion does have long term outcomes. Lung volumes on computed tomography (CT) and spirometry were found to be lower in patients who had lung contusion in the previous 1 to 6 years [12]. 58% of patients with lung contusion had fibrous changes on CT. This may account for long term dyspnoea, decreased functional residual capacity (FRC) and lower oxygenation in patients who have suffered lung contusion.
PATHOPHYSIOLOGY

Mechanical force

In the general civilian trauma populations, falls and rapid deceleration after vehicular crashes are the predominant mechanisms of BCT [5, 8, 13]. Interesting work is being done to characterise the thoracic loading that causes pulmonary contusion by matching cases from CIREN with vehicle crash tests [9, 14]. The cases were sorted into one of three groups: occupants who had sustained a pulmonary contusion (PC) and any other chest injury (PC+ and chest+), occupants who did not present with PC but experienced some form of chest trauma (PC- and chest+), and a control group who did not present with chest injury whatsoever (PC- and chest-). 48% of patients in the PC+ and chest + group had experienced a lateral impact compared to 27% in the control group making this type of impact a relevant focus for further research and identifying crash populations at risk of pulmonary contusion. Other reports from CIREN demonstrate two more significant predictors of pulmonary contusion, namely an instantaneous change in velocity (delta V) of more than 45 mph (odds ratio [OR] = 1.9) and a frontal crash into a fixed object (OR = 1.8) [14].

Result of the mechanical force

The transfer of mechanical force to the lung tissue occurs through physical processes such as compression, tearing, laceration, shearing (due to inertia differences between low density alveolar tissue and higher density hilar tissue), and in blast injuries, a spalling effect (shearing/bursting at an interface between a gas and liquid) and implosion effect (rebound overexpansion of gas as a pressure wave passes) [15].

The intrathoracic maximum pressure impulse via intraoesophageal pressure sensor is a good predictor of pulmonary contusion volume [16].

This transfer of energy results in tissue injury with inflammation and bleeding in the lung, with a complex cascade of local and systemic effects, resulting in physiological derangements.

Hoth recently described a model of the pathogenesis of pulmonary contusion in rats [17]. Of note, systemic levels of certain chemokines (IL-ra, MCP-1, MIP-2α and CINC-1) were significantly elevated at 3 hours. All chemokines were found to be significantly elevated at 24 hours. This was found to be consistent with the early polymorphonuclear and subsequent mononuclear infiltration observed in the contused lung. Pulmonary expression of TNF-α, IL-1β, CINC-1, MIP-2α, ICAM-1, and elastase were increased and activated systemic neutrophils showed increased CD-
11b. The study concluded that innate inflammation is activated \textit{locally and systemically}.

Hoth also found that the Toll-Like Receptor 2 and 4 (TLR-2, TLR-4) participate in the lung’s response to pulmonary contusion, characterized by hypoxia, pulmonary oedema, neutrophil transepithelial migration, and increased expression of the innate immunity pro-inflammatory cytokines [18, 19]. TLR’s are potentially at the centre of the lung’s inflammatory response and the body’s systemic response to lung injury.

Chemokine changes and receptor involvement in ALI/ARDS have triggered studies into genetic epidemiology that may impact future prevention and treatment [20].

Animal models show that alveolar type 2 epithelial cell apoptosis [21] and surfactant dysfunction [22] are also important in the pathophysiology of lung contusion.

Finally, the role of atelectasis in pulmonary dysfunction after trauma has been highlighted by Groeneveld [23].

Cohn [5] has outlined, using animal models, how the disease progresses from initial interstitial haemorrhage, followed over the next couple of hours by interstitial oedema and infiltration of neutrophils and monocytes. At 24 hours, protein, fibrin, red blood cells, and prolific accumulations of inflammatory cells are found in the air spaces, with loss of normal architecture as oedema increases. At 48 hours, there are large amounts of fibrin, cell debri, granulocytes, neutrophils, macrophages and fibrin engorgement of lymphatics. By 7-10 days healing is almost complete. The long term issue of fibrosis, as seen on CT, has been mentioned.

Thus a current model for lung contusion would include local and systemic inflammatory responses, bleeding into alveoli, pulmonary oedema, atelectasis and surfactant dysfunction. These lead to the known physiological derangements of ventilation/ perfusion (V/Q) mismatch, increase in intrapulmonary shunt, increased lung fluid and loss of lung compliance.
DIAGNOSIS

The pathophysiology of lung contusion may manifest as tachycardia, tachypnoea, haemoptysis, hypotension, confusion, hypoxaemia, hypercarbia and increased work of breathing. Examination of the overlying thoracic cage may reveal contusion and rib fractures or even flail chest. Later signs may include decreased air entry, crackles or wheeze. The clinical diagnosis can be difficult to make in the polytrauma patient.

CT imaging is the current gold standard for diagnosis of pulmonary contusion [5]. In particular, Cohn cites the under-diagnosis of lung contusion by plain chest radiography [24] and the ability of CT to stratify lung contusion according to percentage volume which appears to correlate with physiological dysfunction [25, 26]. The use of magnetic resonance imaging, nuclear imaging and ultrasonography are also being studied.

VENTILATORY MANAGEMENT

Current management of flail chest and pulmonary contusion generally concerns correcting respiratory dysfunction, maintaining pulmonary toilet, pain management, fluid management and, if needed, surgical management of the unstable chest wall [2]. This section will focus on correcting respiratory dysfunction.

Animal experiments are able to demonstrate the benefit of continuous positive airway pressure (CPAP) on the respiratory dysfunction due to lung contusion. Schweiger reported the application of CPAP to decrease shunt, improve matching of V/Q, and reduce the requirement for supplemental oxygen, without any significant impairment in cardiovascular function in a study using pigs [27].

Randomized Control Trials (RCTs) concerning NIV in blunt chest trauma are starting to appear, but there are few in number.
Gunduz et al [28] conducted a prospective, randomised study of CPAP given via a face mask to spontaneously breathing patients (CPAP group, n=25) compared with intermittent positive pressure ventilation (IPPV) with endotracheal intubation (ET group, n=27) in patients with flail chest who required mechanical ventilation. Patients included in the study had all of the following:

- a) five or more rib fractures in a row, or three or more segmental (two fractures in one rib) rib fractures on CXR or CT chest and confirmed by the presence of a flail segment (paradoxical motion of the chest wall);
- b) acute respiratory distress and severe dyspnoea with RR $\geq 25$/min;
- c) $\text{SpO}_2 \leq 90\%$ while breathing 10 l/min oxygen in the emergency room;
- d) $\text{PaO}_2/\text{FiO}_2 \leq 300$ while receiving $\text{FiO}_2 >0.5$ in the ICU.

The intubation and ventilation protocol and CPAP protocol were reasonable, and similar to local practice. Patients were monitored in ICU. The ET group received propofol and fentanyl infusions, and the CPAP group had morphine patient controlled analgesia (PCA) with visual analog scale (VAS) monitoring for analgesia. The two groups appeared similar. In the first two days, $\text{PaO}_2$ was significantly greater in the ET group (p<0.05), but this reduced after 2 days to similar levels to the CPAP group.

There were no differences in $\text{PaCO}_2$ between the two groups. Outcomes are shown in table 1 below.

![Table 4: Complications related to death](image)

<table>
<thead>
<tr>
<th>Complication</th>
<th>ET group (n=21) (n (%))</th>
<th>CPAP group (n=22) (n (%))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>10 (47.6%)</td>
<td>2 (9.1%)</td>
</tr>
<tr>
<td>Urinary infection</td>
<td>0 (0%)</td>
<td>2 (9.1%)</td>
</tr>
<tr>
<td>Sepsis</td>
<td>5 (23.8%)</td>
<td>2 (9.1%)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>4 (19.1%)</td>
<td>1 (4.5%)</td>
</tr>
<tr>
<td>MOF</td>
<td>1 (4.8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>ARDS</td>
<td>1 (4.8%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

MOF, multiple organ failure; ARDS, acute respiratory distress syndrome.

**Table 1. Complications related to death, Gunduz et al [28].**

Key outcome differences were lower nosocomial infection (p=0.001) and lower mortality (p<0.01) in the CPAP group, but similar ICU length of stay and $\text{PaO}_2$ values.

In 1990, Bolliger and Van Eeden [29] randomly allocated patients with multiple rib fractures to two groups: 1) a CPAP group (n=36) with lumbar epidural buprenorphine
or intercostal nerve block with bupivacaine, and 2) an endotracheal intubation and ventilation group (n=33) with systemic morphine analgesia. Patients included had all of the following:

a) more than 3 rib fractures
b) hospital admission within 24 hours of injury
c) insufficient cough mechanism due to pain or pre-existing lung disease

However, one of the exclusion criteria was severe lung contusion (alveolar infiltrate underlying rib fractures on CXR and PaO₂ < 8 kPa on a FiO₂ of 0.4 via face mask). No CT chest images were taken. It is likely that patients with multiple rib fractures had underlying pulmonary contusion not detected by plain CXR. No details of the ventilation protocol for the intubation group were supplied, rather targets of a SpO₂ > 90% and a PaCO₂ < 6 kPa were allocated. The two groups were similar at the 5% significance level except for ISS score which was higher in the intubated group. The authors discuss this, explaining that this was due to greater number of blunt abdominal injuries in the intubated group, but the abdominal injuries were considered less severe than the chest injuries in both groups. They felt that the difference was not clinically significant.

This complicates the interpretation of outcomes, shown below in tables 2 and 3:

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**Table 2. Outcomes in patients with non-penetrating injuries to the chest (NIC), Bolliger [29].**

<table>
<thead>
<tr>
<th>Data</th>
<th>CPAP Mask</th>
<th>Intubation</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>36</td>
<td>33</td>
<td>. . .</td>
</tr>
<tr>
<td>Days of treatment</td>
<td>4.5 ± 2.3</td>
<td>7.3 ± 3.7</td>
<td>0.0003*</td>
</tr>
<tr>
<td>Days in intensive care</td>
<td>5.3 ± 2.9</td>
<td>9.5 ± 4.4</td>
<td>&lt;0.0001*</td>
</tr>
<tr>
<td>Days in hospital</td>
<td>8.4 ± 7.1</td>
<td>14.6 ± 8.6</td>
<td>0.0019*</td>
</tr>
<tr>
<td>No. of patients with</td>
<td>10</td>
<td>24</td>
<td>0.002‡</td>
</tr>
<tr>
<td>complications†</td>
<td></td>
<td></td>
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</table>

* t-test (mean ± SD).
† Detailed analysis in Table 3.
‡ χ² analysis.
Table 3. Complications in patients with NIC, Bolliger [29].

A signal of significantly lower risk of pneumonia in the CPAP mask group may still be evident in this study.

Hernandez et al in 2010 performed the most recent RCT using NIV in chest trauma-related hypoxaemia [30]. Patients with PaO₂/FIO₂ ≤200 for ≥8 hrs while receiving oxygen by high-flow mask within the first 48 hrs after thoracic trauma were included. Exclusion criteria were reasonable. Patients were randomized to remain on high-flow oxygen mask (control group, n=25) or to receive NIV (NIV group, n=25) using bi-level positive airway pressure (BiPAP). Thoracic neuraxial block was universally supplied unless contraindicated. The primary end point was intubation; secondary end points included length of hospital stay and survival. The BiPAP protocol was reasonable, and the intubation criteria were well outlined.

The Acute Physiology and Chronic Health Evaluation II (APACHE II) score was higher in the NIV group (p=0.02). However, the study was still stopped early due to a
significant difference in the intubation rate, in terms of less frequent intubations ($p=0.02$) and later intubations ($p<0.01$) in the NIV group. The Cox regression multivariate analysis adjusted for age, gender, APACHE II at study entry, and chronic heart failure showed that NIV was the only variable significantly associated with the reduced intubation rate.

The outcomes are shown below in table 4:

<table>
<thead>
<tr>
<th>Table 2—Intubation and Secondary Outcome Variables of Patients</th>
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</thead>
<tbody>
<tr>
<td>NIMV Group</td>
</tr>
<tr>
<td>(n = 25)</td>
</tr>
<tr>
<td>Intubation rate</td>
</tr>
<tr>
<td>Causes of intubation</td>
</tr>
<tr>
<td>Signs of exhaustion</td>
</tr>
<tr>
<td>Refractory hypoxemia</td>
</tr>
<tr>
<td>Inability to clear respiratory secretions:</td>
</tr>
<tr>
<td>Major agitation</td>
</tr>
<tr>
<td>Pneumothorax post randomization</td>
</tr>
<tr>
<td>Ventilator-associated pneumonia</td>
</tr>
<tr>
<td>ARDS</td>
</tr>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Multiorgan failure</td>
</tr>
<tr>
<td>ICU stay, d&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>ICU mortality</td>
</tr>
<tr>
<td>Hospital stay, d&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hospital mortality</td>
</tr>
</tbody>
</table>

See Table 1 for expansion of abbreviations.

<sup>a</sup>Expressed as median (25th-75th percentiles).

Table 4. Primary and secondary outcomes, Hernandez [30]

The assertion of a lack of Level 1 evidence regarding the management of flail chest-pulmonary contusion in terms of ventilation, as reported by Bastos [2], probably still stands. However, RCTs are starting to appear with significant differences in outcomes. In particular, lower rates of pneumonia and mortality, likely through the reduced need to intubate, are seen.
SUGGESTED APPROACH

A current approach to the role of NIV in blunt chest trauma and its reasoning, based on the aforementioned evidence, is suggested below.

As shown in figure 1, a function of a global injury score (e.g. APACHE, ISS), CT risk stratification, clinical predictors and the ABG may predict the severity of respiratory dysfunction. This will determine the type of positive pressure therapy used, in the form of NIV for moderate dysfunction or ETI with ventilation and recruitment for severe dysfunction or failed NIV. Non-conventional therapies such as ILV or ECMO may be considered with severe dysfunction.

The transitions points of therapy, points A and B, will need to be determined from further research. However, point B will probably be determined by the inherent limit on pressure support (Psup) and PEEP that is practically possible with NIV. The population of patients that are suitable for NIV management (between points A and B) may be larger than generally perceived.

**Figure 1.** Progressive respiratory dysfunction and categories of ventilator therapy. ABG=arterial blood gas, ETI=endotracheal intubation, ILV=independent lung ventilation, ECMO=extracorporeal membrane oxygenation.
A draft algorithm in figure 2 is suggested based on a) inclusion and exclusion criteria from existing studies, b) the above model of therapy based on progressive respiratory dysfunction, and c) practical experience.

**Figure 2. Draft algorithm of the role of NIV in Adult blunt chest trauma.**
Examples of exclusion criteria [30]:

1) hypercapnia (Pa co 2 . 45 mm Hg)
2) orotracheal intubation indicated for another reason;
3) need for emergency intubation;
4) standard contraindications for NIMV (active gastrointestinal bleeding, low level of consciousness, multiorgan failure, airway patency problems, lack of cooperation, or hemodynamic instability);
5) severe traumatic brain injury;
6) facial trauma with pneumocephalus, skull base fracture, orbit base fracture, or any facial fracture involving a sinus;
7) cervical injury when treatment contraindicated a facial mask;
8) bronchopleural fistula;
9) gastrointestinal trauma

Research will be needed to:

- validate inclusion and exclusion criteria,
- design a model for prediction of the degree of respiratory dysfunction as suggested above, and how this correlates to pressure therapy possible with NIV (in particular the role of measuring lung contusion volume by CT as a form of risk stratification seems promising). This will also help to determine points A and B.
- validate an improvement in short and long term outcomes if the above algorithm is applied.

Limitations will also need to be explored regarding cost, availability and expertise. However, these seem likely to all be less limiting than the current method of ETI and ventilation in ICU settings.

A final important point to highlight is that NIV, as the initial mode of therapy for blunt chest trauma, may have a larger role to play at primary and secondary level hospitals. Patients with isolated, non-surgical chest injuries, who would benefit from NIV, tend to be managed at these hospitals in our country, compared to tertiary level hospitals that would predominantly care for polytrauma patients who in general might meet exclusion criteria for NIV therapy. NIV would though have an important role in the extubation protocol for these intubated and ventilated patients at tertiary level hospitals.
CONCLUSION

Lung contusion appears to be the main determinant of acute and long term respiratory dysfunction and a predictor of morbidity and mortality in non-surgical isolated blunt chest trauma. There is a trend towards a more selective approach to ventilator support for patients with gas exchange abnormalities. Randomised control trials are few but are starting to appear, with a possible signal of reductions in pneumonia and mortality due to less frequent intubations. However, inclusion and exclusion criteria for NIV and research to determine respiratory dysfunction stratification, particularly with CT lung contusion volume, are required. NIV would need to be available at the level of hospital that cares for the defined appropriate population, likely primary and secondary level hospitals.

REFERENCES


29. Bolliger CT, Van Eeden SF. Treatment of Multiple Rib Fractures. Randomized Controlled Trial comparing ventilatory with nonventilatory Management. Chest 1990; 97:943-948
