Traumatic Brain & Spinal Cord Injury Update

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@STHJournalClub
http://sthjournalclub.wordpress.com/
### Objectives

#### Level 2 CPD topics should reflect the whole of the individual’s clinical practice including on-call responsibilities in non-specialist centres. The CPD evidence for this level may be provided, in part, by updates from local experts but it will also include the need for non-external CPD activity through attendance at courses and meetings.

<table>
<thead>
<tr>
<th>Objective</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial management of brain injury (traumatic or spontaneous intracranial haemorrhage)</td>
<td>(2F01)</td>
</tr>
<tr>
<td>Initial management of spinal injured patients</td>
<td>(2F02)</td>
</tr>
</tbody>
</table>

#### The Royal College of Anaesthetists CPD Matrix

Matrix of topics for Continuing Professional Development for Anaesthetists (November 2019)

<table>
<thead>
<tr>
<th>Objective</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial management of brain injury (traumatic or spontaneous intracranial haemorrhage)</td>
<td>(2F01)</td>
</tr>
<tr>
<td>Initial management of spinal injured patients</td>
<td>(2F02)</td>
</tr>
</tbody>
</table>
Objectives

1. Traumatic Brain Injury
   - Update on epidemiology
   - Therapeutic targets & Resuscitation principles
   - Pharmacological therapy
   - Management locations and ICP monitoring

2. Spinal Cord Injury
   - Resuscitation principles
   - Therapeutic targets
   - Pharmacological therapy
Epidemiology of TBI

- Incidence 400/100,000 per year
- Death 6-10/100,000 per year
- Leading cause of death 1-45 years
- 80% occur in males; 50% in children
- Significant financial cost
  - By 2020, RTC will be the cause of 1/3 of the global health burden

<table>
<thead>
<tr>
<th>Initial severity</th>
<th>Initial GCS</th>
<th>Outcome</th>
<th>Good recovery (%)</th>
<th>Moderate disability (%)</th>
<th>Severe disability (%)</th>
<th>Dead or vegetative (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>13–15</td>
<td></td>
<td>45</td>
<td>28</td>
<td>20</td>
<td>8</td>
</tr>
<tr>
<td>Moderate</td>
<td>9–12</td>
<td></td>
<td>38</td>
<td>24</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td>Severe</td>
<td>3–8</td>
<td></td>
<td>14</td>
<td>19</td>
<td>29</td>
<td>38</td>
</tr>
</tbody>
</table>
## Epidemiology

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Number</th>
<th>Median age</th>
<th>% aged &gt; 50 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic Coma Data Bank</td>
<td>1984-1987</td>
<td>746</td>
<td>25</td>
<td>15</td>
</tr>
<tr>
<td>UK Four Centre Study</td>
<td>1986-1988</td>
<td>988</td>
<td>29</td>
<td>27</td>
</tr>
<tr>
<td>EBIC Core Data Survey</td>
<td>1995</td>
<td>1005</td>
<td>38</td>
<td>33</td>
</tr>
<tr>
<td>Rotterdam Cohort Study</td>
<td>1999-2003</td>
<td>774</td>
<td>42</td>
<td>39</td>
</tr>
<tr>
<td>Austrian Severe TBI Study</td>
<td>1999-2004</td>
<td>492</td>
<td>48 (mean)</td>
<td>45</td>
</tr>
<tr>
<td>TARN Review</td>
<td>2003-2009</td>
<td>15173</td>
<td>39 (mean)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Italian TBI Study</td>
<td>2012</td>
<td>1366</td>
<td>45</td>
<td>44</td>
</tr>
<tr>
<td>RAIN Study (UK)</td>
<td>2008-2009</td>
<td>2975</td>
<td>44</td>
<td>Not reported</td>
</tr>
</tbody>
</table>
### Table 3. Six-Month Outcome by the Glasgow Outcome Scale Score

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>19–29</th>
<th>30–39</th>
<th>40–49</th>
<th>50–59</th>
<th>60–69</th>
<th>70–79</th>
<th>≥80</th>
<th>All cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died</td>
<td>63 (19%)</td>
<td>44 (20%)</td>
<td>38 (24%)</td>
<td>47 (31%)</td>
<td>57 (37%)</td>
<td>100 (59%)</td>
<td>67 (79%)</td>
<td>416 (33%)</td>
</tr>
<tr>
<td>Died in the ICU</td>
<td>61 (18%)</td>
<td>40 (18%)</td>
<td>33 (21%)</td>
<td>30 (19%)</td>
<td>41 (26%)</td>
<td>61 (36%)</td>
<td>36 (42%)</td>
<td>302 (24%)</td>
</tr>
<tr>
<td>Vegetative</td>
<td>11 (3%)</td>
<td>10 (4%)</td>
<td>6 (4%)</td>
<td>5 (3%)</td>
<td>5 (3%)</td>
<td>2 (1%)</td>
<td>2 (2%)</td>
<td>41 (3%)</td>
</tr>
<tr>
<td>Severe disability</td>
<td>34 (10%)</td>
<td>28 (13%)</td>
<td>29 (18%)</td>
<td>16 (10%)</td>
<td>32 (21%)</td>
<td>35 (21%)</td>
<td>5 (6%)</td>
<td>179 (14%)</td>
</tr>
<tr>
<td>Moderate disability</td>
<td>66 (20%)</td>
<td>47 (22%)</td>
<td>31 (20%)</td>
<td>37 (24%)</td>
<td>16 (10%)</td>
<td>12 (7%)</td>
<td>4 (5%)</td>
<td>213 (17%)</td>
</tr>
<tr>
<td>Good recovery</td>
<td>162 (48%)</td>
<td>89 (41%)</td>
<td>53 (34%)</td>
<td>49 (32%)</td>
<td>44 (29%)</td>
<td>20 (12%)</td>
<td>7 (8%)</td>
<td>424 (33%)</td>
</tr>
<tr>
<td>Unfavorable</td>
<td>108 (32%)</td>
<td>82 (37%)</td>
<td>73 (46%)</td>
<td>68 (44%)</td>
<td>94 (61%)</td>
<td>137 (81%)</td>
<td>74 (87%)</td>
<td>636 (50%)</td>
</tr>
<tr>
<td>Favorable</td>
<td>228 (68%)</td>
<td>136 (63%)</td>
<td>84 (54%)</td>
<td>86 (56%)</td>
<td>60 (39%)</td>
<td>32 (19%)</td>
<td>11 (13%)</td>
<td>637 (50%)</td>
</tr>
<tr>
<td>Total</td>
<td>336</td>
<td>218</td>
<td>157</td>
<td>154</td>
<td>154</td>
<td>169</td>
<td>85</td>
<td>1273</td>
</tr>
</tbody>
</table>
Tracheal Intubation

- The stress response to laryngoscopy must be attenuated

#Increased Cerebrospinal Fluid Pressure During Laryngoscopy and Intubation for Induction of Anesthesia#

**ROBERT G. BURNEY, M.D.*

**RICHARD WINN, M.D.†

Charlottesville, Virginia‡

**TABLE**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Starting Pressure, cm/H_{2}O</th>
<th>Peak Pressure, cm/H_{2}O</th>
<th>Time to Peak ICP, sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tumor</td>
<td>10</td>
<td>18</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>Tumor</td>
<td>14</td>
<td>32</td>
<td>30</td>
</tr>
<tr>
<td>3</td>
<td>Tumor</td>
<td>18</td>
<td>&gt;40</td>
<td>~25</td>
</tr>
<tr>
<td>4</td>
<td>Aneurysm</td>
<td>20</td>
<td>&gt;60</td>
<td>~25</td>
</tr>
<tr>
<td>5</td>
<td>Tumor</td>
<td>7</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>Tumor</td>
<td>10</td>
<td>18</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>Aneurysm</td>
<td>14</td>
<td>38</td>
<td>20</td>
</tr>
<tr>
<td>8</td>
<td>Tumor</td>
<td>10</td>
<td>22</td>
<td>?</td>
</tr>
<tr>
<td>9</td>
<td>Tumor</td>
<td>20</td>
<td>35</td>
<td>47</td>
</tr>
<tr>
<td>10</td>
<td>Aneurysm</td>
<td>8</td>
<td>17</td>
<td>?</td>
</tr>
<tr>
<td>11</td>
<td>Tumor</td>
<td>20</td>
<td>32</td>
<td>63</td>
</tr>
<tr>
<td>12</td>
<td>Tumor</td>
<td>21</td>
<td>32</td>
<td>?</td>
</tr>
</tbody>
</table>

*Fig 2. Typical responses of ICP to laryngoscopy (L), intubation (T), and mechanical hyperventilation (V) in patient #3 (table).*
Tracheal Intubation

- The stress response to laryngoscopy **must** be attenuated
- Alfentanil 10 µg kg\(^{-1}\) is as effective as remifentanil

### Intracranial Pressure and Hemodynamic Effects of Remifentanil Versus Alfentanil in Patients Undergoing Spinal Cord Injury

<table>
<thead>
<tr>
<th></th>
<th>Placebo (n = 6)</th>
<th>Remifentanil (n = 5) 0.5 µg/kg</th>
<th>Remifentanil (n = 5) 1.0 µg/kg</th>
<th>Alfentanil (n = 5) 10 µg/kg</th>
<th>Alfentanil (n = 5) 20 µg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preinfusion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paco(_2) (mm Hg)</td>
<td>27 ± 4</td>
<td>29 ± 4</td>
<td>28 ± 1</td>
<td>29 ± 2</td>
<td>27 ± 2</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>88 ± 20</td>
<td>80 ± 7</td>
<td>80 ± 11</td>
<td>79 ± 13</td>
<td>84 ± 4</td>
</tr>
<tr>
<td><strong>ICP (mm Hg)</strong></td>
<td>22 ± 12</td>
<td>13 ± 7</td>
<td>14 ± 4</td>
<td>17 ± 5</td>
<td>21 ± 15</td>
</tr>
<tr>
<td>CPP (mm Hg)</td>
<td>66 ± 22</td>
<td>67 ± 11</td>
<td>66 ± 8</td>
<td>62 ± 9</td>
<td>63 ± 18</td>
</tr>
</tbody>
</table>
Tracheal Intubation

- The stress response to laryngoscopy **must** be attenuated
- Alfentanil 10 µg kg\(^{-1}\) is as effective as remifentanil
- Ketamine is my first choice in trauma
  - Propofol and thiopentone are alternatives
  - Etomidate is contraindicated in head injury
- Most patients will require a RSI: suxamethonium and rocuronium are both safe in brain injury
Ketamine

KETAMINE

Just Say NEIGH
### TABLE I
**CARDIOVASCULAR EFFECTS OF KETAMINE IN NON-PARALYzed GOATS**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>5 min†</th>
<th>10 min†</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral blood flow (ml/100g·min⁻¹)</td>
<td>77 ± 7</td>
<td>109 ± 12*</td>
<td>96 ± 9*</td>
<td>88 ± 9</td>
</tr>
<tr>
<td>Cerebral metabolic rate (O₂M)/100 g⁻¹·min⁻¹</td>
<td>4.31 ± 0.36</td>
<td>3.82 ± 0.27*</td>
<td>4.0 ± 0.32*</td>
<td>4.12 ± 25</td>
</tr>
<tr>
<td>Mean Arterial blood pressure kPa (torr)</td>
<td>13.6 ± 0.5</td>
<td>16.3 ± 0.7*</td>
<td>15.9 ± 0.8*</td>
<td>14.2 ± 0.3</td>
</tr>
<tr>
<td>(102 ± 4)</td>
<td>(123 ± 5*)</td>
<td>(120 ± 6*)</td>
<td>(107 ± 2)</td>
<td></td>
</tr>
<tr>
<td>Paco₂ kPa (torr)</td>
<td>4.5 ± 0.5</td>
<td>5.4 ± 0.9*</td>
<td>5.2 ± 1.2*</td>
<td>4.5 ± 1.1</td>
</tr>
<tr>
<td>(34 ± 4)</td>
<td>(41 ± 7*)</td>
<td>(39 ± 9*)</td>
<td>(34 ± 8)</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE II
**CARDIOVASCULAR EFFECTS OF KETAMINE IN PARALYzed GOATS**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>5 min†</th>
<th>10 min†</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral blood flow (ml/100g·min⁻¹)</td>
<td>68 ± 4</td>
<td>62 ± 5</td>
<td>65 ± 5</td>
<td>61 ± 6</td>
</tr>
<tr>
<td>Cerebral metabolic rate (O₂M)/100 g⁻¹·min⁻¹</td>
<td>4.34 ± 0.39</td>
<td>3.71 ± 0.33*</td>
<td>4.05 ± 0.36</td>
<td>4.32 ± 0.45</td>
</tr>
<tr>
<td>Mean Arterial blood pressure kPa (torr)</td>
<td>13.8 ± 4</td>
<td>15.2 ± 4</td>
<td>15.6 ± 0.5*</td>
<td>15.4 ± 0.5*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Ketamine

- Ketamine increases ICP/CBF in spontaneously breathing volunteers (III)
- No effect on ICP with controlled ventilation and sedation (III)
- Greater CPP maintained with ketamine and lower vasopressor requirements (II)
- No effect on cerebral autoregulation (III)
Breathing

- Hypoxia associated with worse outcome
- Marked hypo- and hypercapnia similarly bad
  - Must calibrate PaCO$_2$ with ETCO$_2$
Mean CO₂ (PaCO₂ + ETCO₂/2) vs. PaCO₂-ETCO₂
Breathing

• Hypoxia associated with worse outcome
• Marked hypo- and hypercapnia similarly bad
• What about PEEP?

Positive End-Expiratory Pressure Alters Intracranial and Cerebral Perfusion Pressure in Severe Traumatic Brain Injury

Toan Huynh, MD, Marcia Messer, RN, Ronald F. Sing, DO, William Miles, MD, David G. Jacobs, MD, and Michael H. Thomason, MD

<table>
<thead>
<tr>
<th>PEEP (cm H₂O)</th>
<th>ICP (mm Hg)</th>
<th>CPP (mm Hg)</th>
<th>CI (L/min/m²)</th>
<th>Do₂i (mL O₂/min/m²)</th>
<th>Do₂s (mL O₂/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–5</td>
<td>14.7 ± 0.2</td>
<td>77.5 ± 0.3*</td>
<td>3.8 ± 0.1</td>
<td>593 ± 9</td>
<td>167 ± 6</td>
</tr>
<tr>
<td>6–10</td>
<td>13.6 ± 0.2</td>
<td>80.1 ± 0.5</td>
<td>4.0 ± 0.1</td>
<td>629 ± 21</td>
<td>167 ± 6</td>
</tr>
<tr>
<td>11–15</td>
<td>13.1 ± 0.3†</td>
<td>78.9 ± 0.7†</td>
<td>3.8 ± 0.1</td>
<td>560 ± 21</td>
<td>153 ± 6</td>
</tr>
</tbody>
</table>

* p < 0.001 vs. PEEP 6–10 cm H₂O; † p < 0.001 vs. PEEP 0–5 cm H₂O.
• A single episode of hypotension doubles mortality

<table>
<thead>
<tr>
<th>Secondary Insults</th>
<th>Number of Patients</th>
<th>% Total Patients</th>
<th>Outcome Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Good or Moderate</td>
</tr>
<tr>
<td>Total cases</td>
<td>699</td>
<td>100.0</td>
<td>42.9</td>
</tr>
<tr>
<td>Neither</td>
<td>456</td>
<td>65.2</td>
<td>51.1</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>78</td>
<td>11.2</td>
<td>44.9</td>
</tr>
<tr>
<td>Hypotension</td>
<td>113</td>
<td>16.2</td>
<td>25.7</td>
</tr>
<tr>
<td>Both</td>
<td>52</td>
<td>7.4</td>
<td>5.8</td>
</tr>
</tbody>
</table>

Hypoxia = $\text{Pao}_2 < 60$ mm Hg; hypotension = SBP < 90 mm Hg. Reprinted with permission from Chesnut et al. 10
Circulation
Chesnut et al. J Trauma 1993

• A single episode of hypotension doubles mortality
• Head injury alone rarely causes hypotension
• Treatment of cardiovascular instability takes precedence over direct head injury intervention
• No evidence for any one vasopressor
• Trials with permissive hypotension excluded those with TBI
Retrospective analysis

15 733 patients with TBI following blunt trauma

### Table 3

Optimal SBP and mortality in isolated moderate to severe TBI patients.

<table>
<thead>
<tr>
<th>Age group</th>
<th>Optimal SBP</th>
<th>Mortality</th>
<th>AOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>15–49 years (n = 10284)</td>
<td>&lt;110 mmHg</td>
<td>21%</td>
<td>1.98</td>
</tr>
<tr>
<td>50–69 years (n = 3093)</td>
<td>&lt;100 mmHg</td>
<td>29%</td>
<td>2.20</td>
</tr>
<tr>
<td>50–69 years (n = 3093)</td>
<td>≤110 mmHg</td>
<td>20%</td>
<td>1.60</td>
</tr>
<tr>
<td>≥70 years (n = 2356)</td>
<td>&lt;110 mmHg</td>
<td>38%</td>
<td>1.92</td>
</tr>
</tbody>
</table>

Adjusted odds ratio (AOR): Optimal SBP is compared to SBP reference groups, adjusting for age, gender, ISS ≥ 16, and GCS ≤ 8; for age 50–69 (≥100 mmHg), for age ≥ 70 (≥110 mmHg)
Circulation
Hassler et al. Resuscitation 2012
- Prospective work from TARN dataset
- 3444 patients with penetrating trauma
Prospective work from TARN dataset

47,927 patients with blunt trauma

Odds ratio of death and its association with systolic blood pressure

Circulation
Hassler et al. Resuscitation 2011

and multivariate analysis (adjusted for age only and adjusted for age, gender, ISS and GCS) of categories of SBP levels (mean ± 95% confidence interval) with the corresponding odds of death, compared to the baseline at 130–139 mmHg.
Editorial

Blood pressure management in trauma: from feast to famine?

In 2007, the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) published a review of trauma services in England, Wales and Northern Ireland [1]. This demonstrated that almost 60% of major trauma cases received care that was judged to be less than good practice. This led to a review of major trauma care within England by the National Audit Office [2], which subsequently led to the creation of major trauma centres (MTCs). As a result of the advent of MTCs the management of trauma within the UK has changed radically over the past few years, from both organisational and clinical perspectives [3]. This has led to an upsurge in interest amongst medical professionals as to how best to manage trauma cases. The basis of much of UK trauma management over the past 30 years has been derived from teaching from the Advanced Trauma Life Support (ATLS) courses [4]. These guidelines have faced increasing criticism over the past decade, especially regarding their insensitivity to change (with updates typically occurring on a 3–4 year cycle) and applicability to UK practice [5, 6].

Despite these weaknesses, some ATLS concepts have become enshrined into trauma management and teaching, though without much supporting evidence. Examples of this include the trimodal distribution of death following trauma and the ATLS classification of shock, both of which have been shown to be theoretical concepts rather than useful clinical entities [7–10].

Further challenges to ATLS management recommendations came from the implementation of military trauma strategies. The conflicts in Iraq and Afghanistan led to the development of novel management strategies in both trauma resuscitation and surgery (discussed in detail in a recent supplement in this journal [11]), some of which have been translated back to civilian practice. One area of focus is the concept of permissive hypotension as part of a damage control resuscitation (DCR) strategy in the management of the bleeding trauma patient.

Permissive hypotension (or hypotensive resuscitation) was conceived because of the theoretical risk of excess fluid administration’s interfering with the endogenous coagulation process, by inducing a dilutional coagulopathy, by clot disruption from an increase in arterial pressure, or through the abolition of reflex physiological vasoconstriction. This led to the recommendation that fluid administration should be delayed until haemorrhage has been controlled, even though this often would result in a period of suboptimal end-organ perfusion. Typical systolic blood pressure (SBP) targets in permissive hypotension are 70–90 mmHg although a recent article [12] has suggested that DCR targets should be a SBP and mean arterial pressure (MAP) of 80 and 50 mmHg, respectively. Over the past three years, guidelines from professional bodies (including the ambulance service) and review articles have been published, recommending the use of permissive hypotension in trauma management [13–16], but it is unclear whether there is robust evidence to support this practice, especially for the UK trauma population.

The concept of permissive hypotension was first described by Cannon et al. [17], a group of Captains in the Army Medical Corps, from their experience in the management of injuries received during the First World War. They noted that "Injection of a fluid that will increase blood pressure has dangers in itself. If the pressure is raised before the surgeon is ready to check the bleeding that may take place, blood that is surely needed may be lost." It is of note, however, that in the same series of articles, one of Cannon's co-authors, Cowell [18], recognised the problems associated with periods of prolonged hypoperfusion, stating "...the treatment of
<table>
<thead>
<tr>
<th>Parameter</th>
<th>BTF</th>
<th>EBIC</th>
<th>AAGBI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>AVOID SpO₂ &lt;90% PaO₂ &lt;8 kPa PaCO₂ &lt;3.3 kPa</td>
<td>TARGET SpO₂ &gt;95% PaO₂ &gt;10 kPa PaCO₂ 4.0-4.5 kPa</td>
<td>TARGET PaO₂ &gt;13 kPa PaCO₂ 4.5-5.0 kPa</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>AVOID SBP &lt;90 mmHg</td>
<td>TARGET MAP &gt;90 mmHg SBP &gt;120 mmHg</td>
<td>TARGET MAP &gt;80 mmHg</td>
</tr>
<tr>
<td>Neurological</td>
<td>ICP &lt;20 CPP 50-70 (probably 60)</td>
<td>ICP &lt;20-25 CPP 60-70</td>
<td>ICP &lt;20-25 CPP 60-70</td>
</tr>
</tbody>
</table>
Osmotherapy
Cochrane Collaboration 2008

• Mannitol - no effect on mortality
• Both hypertonic saline (HTS) and mannitol reduce ICP in the short term to similar degrees
• Both associated with morbidity if given in excess
• HTS may have a role in low-volume resuscitation in trauma
  – Highly irritant; best delivered centrally
  – Potential for error is huge
Where should TBI be managed?

Harrison et al. Health Technol Assess 2013

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No or Late Transfer (n=263)</th>
<th>Early Transfer (n=584)</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/12 Mortality</td>
<td>107 (41%)</td>
<td>109 (19%)</td>
<td>0.52 (0.34-0.80)</td>
</tr>
<tr>
<td>6/12 Unfavourable Outcome</td>
<td>169 (65%)</td>
<td>307 (53%)</td>
<td>0.88 (0.28-2.79)</td>
</tr>
<tr>
<td>≤ 70 years</td>
<td>-</td>
<td>-</td>
<td>0.47 (0.29-0.77)</td>
</tr>
<tr>
<td>&gt; 70 years</td>
<td>-</td>
<td>-</td>
<td>1.53 (0.44-5.30)</td>
</tr>
<tr>
<td>Isolated TBI</td>
<td>-</td>
<td>-</td>
<td>0.80 (0.42-1.42)</td>
</tr>
<tr>
<td>Major extracranial injury</td>
<td>-</td>
<td>-</td>
<td>0.22 (0.10-0.47)</td>
</tr>
<tr>
<td>Mild/Moderate TBI</td>
<td>-</td>
<td>-</td>
<td>0.73 (0.36-1.50)</td>
</tr>
<tr>
<td>Severe TBI</td>
<td>-</td>
<td>-</td>
<td>0.43 (0.24-0.78)</td>
</tr>
</tbody>
</table>

- Control group had more severe injuries
  - Bilateral unreactive pupils (22% vs. 10%)
  - Extracranial injury (42% vs 36%)
  - Prehospital hypotension (8% vs 5%)
6/12 Mortality 39% (ICP care) vs. 41% (Clinical) [P=0.60]
– < 25% in hospital within 60 min & only 45% came by ambulance
– ICP target similar to DECRA (ICP >20mmHg for 15 min)
– ICP bolts sited on the basis of GCS alone
“The lack of improvement in head injured patients is typified by the apparent overall lack of progress in head injury care, which is suggested by the failure to identify a single therapy to improve outcome despite over 250 randomised controlled trials.

However, several studies have shown that the institution of packages of specialist neurosurgical or neurocritical care is associated with improved outcomes.”
Summary (TBI)

- Hypoxia & hypotension are bad for the brain
  - Permissive hypotension has no place in TBI
  - MAP > 80 mmHg and/or SBP > 110 mmHg
- Anything we can do to avoid these are good (including ketamine, PEEP & calibration ETCO₂)
- ICP monitors are of no value in Bolivia or Ecuador
- TBI should be managed in a neurosurgical centre
  - If GCS 3-8 and aged < 70
Epidemiology of SCI

- Incidence 15-40 per million per annum
  - cf TBI 4000 per million
- 70-80% occur in males
Epidemiology of SCI

- Incidence by location
  - Cervical 75%
  - Thoracic 10%
  - Lumbar 10%

- Incidence of fractures with SCI
  - Cervical 40-50%
  - Thoracic >95%
  - Lumbar >85%
Manual In-line Stabilisation

• Origin uncertain – ATLS guidance 1984
• Data from cadaveric studies, healthy volunteers and case series (n=96)
• Direct laryngoscopy/intubation cause less cervical movement than a jaw thrust
• Several studies suggest MILS has no effect on cervical segment movement

<table>
<thead>
<tr>
<th>Method</th>
<th>Grade 1</th>
<th>Grade II</th>
<th>Grade III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal positioning</td>
<td>129</td>
<td>26</td>
<td>2</td>
</tr>
<tr>
<td>MILS</td>
<td>75</td>
<td>48</td>
<td>34</td>
</tr>
</tbody>
</table>
Cervical Collars
Sundstrøm et al. Journal of Neurotrauma 2014

• Most spinal injuries are stable; those that are unstable have already caused irreversible damage
• Collars do not immobilise the cervical spine
• Exaggerated rate of secondary SCI without collars
• Numerous associated complications

• Authors suggest:
  – Spinal board with head blocks & straps
  – Collars only for difficult extrication
  – Unconscious, nonintubated trauma patients should be transported in modified left lateral
Clearing the Spine

Why bother?

• Avoidance of skin damage secondary to collars (6-67%)
  – Ulceration
  – Sepsis
• 30 degree head-up tilt to reduce pneumonia
• Exacerbation of raised ICP
• Increased demands on nursing care
• Exacerbation of agitation especially in TBI
Clearing the Spine

Comparative effectiveness of using computed tomography alone to exclude cervical spine injuries in obtunded or intubated patients: meta-analysis of 14,327 patients with blunt trauma

A review

David M. Panczykowski, M.D., Nestor D. Tomycz, M.D., and David O. Okonkwo, M.D., Ph.D.

- 7 missed injuries of which 3 unstable
- Sensitivity/Specificity of CT >99.9% (cf NEXUS 99%)
- -ve LR < 0.001%
- 1 in every 4776 patients have missed injury
(My) Rules for Clearing the Spine

• HRCT CT of C-spine (1-2 mm slices)
  – C0 – T2 (but T4 better)
  – Reported by consultant MSK/neuroradiologist
  – Discussed with spinal/neurosurgical consultant
• [Consider AP/lateral C-spine radiographs]
• CT reconstructions of thoracolumbar spine
• AP/Lateral radiographs thoracolumbar views
• NB. Semi-rigid collar (Aspen/Philadelphia) in interim
Neurological Deterioration after Surgery

• Due to prolonged deformation and/or hypotension
  – Hyperflexion worse than hyperextension
• Both are unlikely during DL
• AFOI may not be safer
  – Several claims in US Closed Claims Database
• 5% patients with SCI will deteriorate
  – Early (24 h)
  – Later (1-7 days)
  – Late (weeks [post-traumatic ascending myelopathy])
High-dose methylprednisolone steroid therapy is the only pharmacologic therapy shown to have efficacy in a phase three randomized trial when administered within eight hours of injury. One trial indicates additional benefit by extending the maintenance dose from 24 to 48 hours, if start of treatment must be delayed to between three and eight hours after injury. There is an urgent need for more randomized trials of pharmacologic therapy for acute spinal cord injury.
NASCIS II

• **Design**
  – Multicentre, prospective, randomised, double-blind trial.

• **Patients**
  – 487 patients with acute spinal cord injury (95% follow up)

• **Exclusions**
  – Injuries below L1, children

• **Randomisation**
  – Treatment 1: Methyprednisolone 30 mg kg$^{-1}$ bolus, then 5.4 mg kg$^{-1}$ h$^{-1}$ for 23 hours
  – Treatment 2: Naloxone 5.4 mg kg$^{-1}$ bolus, then 4.5 mg kg$^{-1}$ h$^{-1}$ for 23 hours
  – Treatment 3: Placebo
NASCIS II

• **Assessment**
  – Motor scale (0-5) in 14 muscle groups (total 70)
  – Sensory (Pin prick & touch) in 29 dermatomes (total 58)

• **(Author’s) Results**
  – Patients receiving steroids within 8 h had a statistically significant improvement of 5 points on the motor score at 6 months and 1 year (P=0.03)

• **Safety**
  – Wound infection & PE doubled in steroid group (NS)
NASCIS II

• All +ve results are from post hoc analyses
• Time cut off (8 h) is arbitrary
• 78 discrete post hoc tests
• 60 t-tests for neurological outcomes
• Correct hypotension (SBP <90mmHg) ASAP (III)
• Target MAP 85-90 mmHg for 7 days post injury (III)
  – Compared to historical controls
  – >50% with cervical injuries will require vasopressors
  – Complications common in first 7 days post injury
    • Hypotension, bradycardia
    • Ventilatory failure on average 4.5 days post injury
    • Intubation rates: ≥C5 100% cf 79% ≤ C6
Timing of Surgery

Early versus Delayed Decompression for Traumatic Cervical Spinal Cord Injury: Results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS)

Michael G. Fehlings¹, Alexander Vaccaro², Jefferson R. Wilson¹, Anoushka Singh¹, David W. Cadotte¹, James S. Harrop², Bizhan Aarabi³, Christopher Shaffrey⁴, Marcel Dvorak⁵, Charles Fisher⁵, Paul Arnold⁶, Eric M. Massicotte¹, Stephen Lewis¹, Raja Rampersaud¹
Summary (SCI)

• Avoid hypotension & hypoxia
• Try to clear the spine ASAP (CT neck with brain)
• Patients with high SCI may be best managed on HDU/ICU for > 7 days
• There is no place for steroid therapy in SCI
• Much of the best care is supportive & “SHO work”
  – LMWH
  – Stress ulcer prophylaxis
  – Aperients
• Surgical timing is still uncertain
References

References


19. Hasler RM et al. Systolic blood pressure below 110mmHg is associated with increased mortality in penetrating major trauma patients: Multicentre cohort study. *Resuscitation* 2011; 83: 476-81.


