Pre-Hospital Management of Traumatic Brain Injury

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Reference

• Stiver SI, Manley GT
• Prehospital management of traumatic brain injury
• Neurosurg Focus. 2009. 25(4)
Overview

• A bit of background information
• Mechanisms of injury
• Juicy pre-hospital stuff
  – The secondary brain injury
  – Dispatch and triage decisions
Classification

GCS 13-15 (concussion) 80%

GCS 9-12 10%

GCS 3-8 10%

Primary brain injury vs. Secondary brain injury
Statistics

- 1.4 million TBIs in the USA each year
- 10% permanent neurological disability
- One third of all trauma deaths
- Males twice as likely as females
- Children and young adults (about 500,000)

What happens in the pre-hospital setting is critical
Leading mechanisms of injury

RTC, assault, falls – 80% of TBI
And now for the really important bit.....

SECONDARY BRAIN INJURY
Definitions

• Primary brain injury
  – Occurs at time of impact
  – Irreversible
  – Injury prevention strategies (seatbelts, car design)

• Secondary brain injury
  – Chain of events set in motion by primary impact
  – Initiate further injury
  – Exacerbated by:
    • Hypoxia, Hypotension, Abnormal pCO₂
    • Elevated intracranial pressure
Hypoxia (brain oxygenation)

• Airway obstruction and aspiration
  – Low GCS, poor pre-hospital management
Hypoxia (brain oxygenation)

• Brain relies on aerobic metabolism
• *Increased mortality and worse functional outcome* with periods of hypoxia after traumatic brain injury
• Best evidence-based practice
  – Keep saturations as near to 100% as possible
  – Self-ventilating vs. Intubated and ventilated
Is intubation better?

• Two patients require oxygenation:
  – Agitated, won’t keep oxygen mask on, pulls off sats probe, GCS 12
  – Flat, unresponsive, breathing, SpO₂ 91%, GCS 3

• Things to consider:
  – Risks vs. Benefits of pre-hospital anaesthesia
  – Who performs the intubation?
  – The ‘anaesthetic package’
RSI and the ‘anaesthetic package’

• Facilitated pre-oxygenation (adjuncts and sedation)
• Drugs to induce anaesthesia and paralyse
• Intubation and confirmation of placement
• A failed airway drill
• Maintenance of anaesthesia
• Appropriate ventilation strategy
• Neuroprotective manoeuvres
  – Position of patient
  – Collar and tube tie
  – IV fluid administration
  – Hypertonic saline
Flat, unresponsive, GCS 3, SpO₂ 91%

1. Guedel airway, nasopharyngeal airways, jaw thrust, tight fitting oxygen mask
2. Intubation/LMA and ventilation (no drugs)
3. Pre-hospital RSI and ‘anaesthetic package’

*Advantages and disadvantages of each?*
Agitated, removes oxygen and sats probe, GCS 12

1. Gentle restraint, hold on mask
2. Guedel airway, nasopharyngeal airways, tight fitting oxygen mask
3. Pre-hospital RSI and ‘anaesthetic package’
4. Sedation followed by 2. or 3.

Advantages and disadvantages of each?
Oxygenation in TBI - Summary

1. Oxygenation is critical in TBI
2. Must be safe without causing further harm
3. Pre-Hospital intubation requires a full anaesthetic package
   – Advantages and disadvantages
   – Must be done by an expert
Hypotension (cerebral hypoperfusion)

• Trauma Coma Databank (USA)
  – Single episode of SBP <90mmHg
  – At any time pre-hospital or in the ED
  – *Doubled mortality*

• Isolated head injuries don’t cause hypotension
  – Things that we do to patients
  – Other injuries
Cerebral blood flow autoregulation
Importance of CBF regulation

• Oxygen delivery to the brain
  – *Low CBF causes hypoxia and ischaemia*
  – *Loss of up-regulation*
• Intracranial pressure
  – *High CBF causes elevated ICP*
  – *Loss of down-regulation*

In moderate-severe traumatic brain injury:

*Cerebral blood flow is dependent on BP*
Managing hypotension in TBI

• Identify the cause and stop it
  – Bleeding
  – Drugs

• Careful use of intravenous fluid
  – 250ml boluses of 0.9% saline
  – Keep SBP >100mmHg

What if the patient with a head injury has non-compressible haemorrhage?
Hypotension - Summary

• One episode of hypotension *at any time* after TBI *doubles* the patient’s chance of dying

• Best evidence-based practice:
  – Keep SBP >100mmHg for isolated head injury
Abnormal pCO₂

- Arterial CO₂ concentration (pCO₂)
- End tidal CO₂ concentration (ETCO₂)

- pCO₂ is directly linked to cerebral blood flow
  - Low pCO₂ causes decreased cerebral blood flow
  - High pCO₂ causes increased cerebral blood flow

- For isolated TBI, death less likely if ventilated in target range (4 to 4.5 kPa) (Odds Ratio 0.3)
Why $pCO_2$ is crucial (too low)
Why \( pCO_2 \) is crucial (too high)
Head injury and ventilation

• ETCO$_2$ is a direct reflection of ventilation
• High ETCO$_2$ suggests hypoventilation
  – Direct central suppressant effect
  – Airway obstruction
• Low ETCO$_2$ suggests hyperventilation
  • Over-enthusiastic ventilator settings
What can we do to optimise pCO$_2$?

1. Ensure open airway
2. Take control of ventilation
3. Measure ETCO$_2$ and react to high or low
Abnormal pCO₂- Summary

• Low ETCO₂ is bad
  – Decreased cerebral blood flow (and oxygenation)

• High ETCO₂ is bad
  – Increased cerebral blood flow and ICP

The only way to definitively control ETCO₂ is to take control of ventilation and continuously monitor the actual value
Elevated intracranial pressure

• Patients with moderate-severe TBI die of:
  1. Hypoxia and aspiration
  2. Raised ICP
  3. Complications of long hospitalisation
How do we recognise raised ICP?

• Usually severe head injury (GCS 3)
• Significant drop in GCS (2 points or more)
• Pupil signs
  1. Irregular
  2. Both unreactive
  3. Unequal (>1mm difference)
• Cushing response
  – Hypertension
  – Bradycardia
How can we fix raised ICP

• Temporary
  – Keep ETCO$_2$ in appropriate range (*not* low)
  – Control BP
  – Hypertonic saline
  – Mannitol

• Surgical intervention
  – Craniectomy
  – Evacuation of clot
  – ICP bolt (direct measurement of pressure)
Raised ICP - Summary

- Usually a pre-terminal event
- Can be temporarily reduced pre-hospital
  - Prompt recognition
  - Neuroprotective package of care
  - Additional drugs
DISPATCH AND TRIAGE
Triage facts (1)

1. In severe TBI, better survival rates achieved if patient has neuroprotective treatment on scene and during transport to hospital

2. Mortality rate >90% if sub-dural haematomas are not evacuated **within 4 hours** of injury (30% if evacuated within 4 hours)
Triage facts (2)

3. Only one hospital in Essex has neurosurgery

4. Secondary transfers cause significant delay and independently increase mortality

5. Royal London and Addenbrooke’s take patients from Essex if doctor decides to bypass local (will change soon)
Dispatch

• Some patients with head injury will benefit from:
  1. Additional drugs
  2. Anaesthetic package
  3. Neuroprotective care
  4. Local hospital bypass to neurosurgical centre

• Who can provide this additional care?
  – BASICS and HEMS?
  – Critical care paramedics?
Getting HEMS to help

• Two types of activation
  – Based on 999 call
  – Crew requests

• Difficult to pick up moderate-severe head injuries over phone from lay-person!

• Crew request:
  – Via HEMS desk in Chelmsford
  – Guarantee to launch within 3 minutes if available
  – Doctor may call on-scene crew to offer initial advice
Summary

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• Mechanisms of injury
• Juicy pre-hospital stuff
  – The secondary brain injury
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Take home messages

• Secondary brain injury is preventable
• The following are bad:
  – Hypoxia
  – Hypotension
  – Abnormal pCO$_2$
  – Raised ICP
• For moderate and severe TBI:
  – A whole package of pre-hospital care can be provided
  – The triage decision is critical
QUESTIONS?

Scene

A&E

Prevention

Theatre

ITU

Rehab

Home

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