Traumatic Brain Injury (TBI)
Evidence-based Guidelines in Practice

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Putting Guidelines into Practice

- Incorporating the key recommendations from experts into daily practice can be a challenge.

- Protocols to guide our care at the bedside are essential where patient management is complex and specific.
The Recommendations

**Level I - Standards**
Accepted management strategies with a *high* degree of clinical certainty

**Level II - Guidelines**
Management strategies with a *moderate* degree of clinical certainty

**Level III - Options**
Management strategies with *unclear* clinical certainty

Most treatments in traumatic brain injury are directed by consensus guidance rather than clear evidence.

Most influential attempts to standardise treatment:
- **Brain Trauma Foundation (BTF)**
- **European Brain Injury Consortium (EBIC)**
  1997
- **American Brain Injury Consortium (ABIC)**
  2010
Minimising secondary neurological injury

- **Primary brain injury is unavoidable**
  Other than through prevention e.g. legislation for compulsory wearing of seat belts, crash helmets

- **Secondary brain injury is avoidable**
  Most common and lethal causes of secondary injury
  - Hypoxia
  - Hypotension
TBI Guidelines

Key Concepts

◆ Treating raised intracranial pressure (ICP)
  • Surgery
  • Medical measures

◆ Time critical – ‘The Golden Hour’
  • When what we do will make a difference to outcome

◆ Protocols
  • To streamline acute management and ensure timely transfer to definitive neurosurgery for those who need it
Key message from Guidelines

*Early management can influence outcome*

- Basic principles of resuscitation are vital for good outcome
  - Airway, Breathing, Circulation, Disability, Exposure
  - These principles apply regardless of whatever the clinical area

- Without this, subsequent advanced monitoring in a specialist neurosurgical unit may be of little value in improving ultimate outcome
**Airway and Breathing**

**What $O_2$ and $CO_2$ should we aim for? Should we hyperventilate TBI patients?**

- **Oxygen**
  - Hypoxia (PaO$_2$ <60mmHg / SpO$_2$ <90%)
  - Second most influential cause of secondary brain injury

- **Carbon Dioxide**
  - $	ext{PaCO}_2$ increases ICP
  - $	ext{PaCO}_2$ exacerbates ischaemia

- **Hyperventilation**
  - Level I: High degree of clinical certainty
    - Avoid $\text{PaCO}_2 <30\text{mmHg}$
    - Hyperventilation used only as temporising measure in profoundly injured patients exhibiting signs of herniation (abnormal posturing, fixed/dilated pupils)

- Level II: Moderate degree of clinical certainty
  - $\text{PaO}_2 >97.5\text{mmHg}$
  - $\text{PaCO}_2 33.7\text{mmHg}$
Circulation
What BP should we aim for?

◆ Hypotension (defined as SBP < 90mmHg)
  - May lead to ischaemia
  - Most predominant factor in secondary brain injury
  - Highest correlation with morbidity and mortality

◆ Hypertension
  - May lead to expansion of a haematoma, cerebral oedema, re-bleeding

◆ Permissive hypotension in trauma resuscitation
  - Several injuries requiring contrasting physiological management
  - SBP ≤ 80mmHg or MAP 40-50mmHg until bleeding controlled
  - Reduce BP as little as possible and for as short a time as possible

Level II
Moderate degree of clinical certainty
Systolic BP > 120mmHg or MAP > 90mmHg
(CPP = MAP – ICP)
Which resuscitation fluid should we use and how much?

- Hypovolaemia potentially harmful
  - Circulation

- Avoid glucose-containing solutions
  - Maintain glucose 6-10 mmol/l

SAFE TBI
  - Hypovolaemia
  - May be prudent to avoid other hypo-osmolar solutions such as gelofusine and Hartmann's

Level II

Moderate degree of clinical certainty

Prevent hypovolaemia with liberal use of crystalloids with goal of intravascular euvolaemia
Disability (Neurological status)

What is the best way to assess and classify head injured patients?

- Emergency departments will see many patients with head injury
- 1 in 500 will go on to develop a significant brain injury
- Vital to identify who will need urgent intervention
Disability (Neurological status)

What is the best way to assess and classify head injured patients?

Level II
Moderate degree of clinical certainty

Patient’s acute clinical condition must be documented using a recognised assessment tool

- The Glasgow Coma Scale\textsuperscript{1974} most widely used and most extensively evaluated tool for acute classification and assessment of TBI patients
- Considered to be the ‘Gold Standard’
Exposure (Secondary survey)

Does the patient have associated spinal injury?

- 5% incidence of associated cervical spine injury in moderate and severe TBI
- Assume unstable cervical spine
- Measures taken to ‘clear the cervical spine’
  - impacts on ability to manage raised intracranial pressure
The Emergency Department

How do we implement guidelines and standardise the care of TBI patients?
Acute Management and Transfer of Adults with Traumatic Brain Injury

Indications for urgent referral to a neurosurgeon
- CT scan shows a recent intracranial haemorrhage or haematoma
- Patient fails to follow criteria for CT scan but scan cannot be performed locally
- Patient has concerning clinical features (see below) irrespective of CT findings

Neurosurgical Referral
National Hospital for Neurology & Neurosurgery (NHNN)
0845 155 5000
bleep 8100

Clinical features which must be discussed with a neurosurgeon
- Persistent hypotension
- Persistent agitation
- Persistent abnormal neurological signs
- Sustained decrease in GCS by 1 point for more than 30 minutes
- Any decrease in GCS by 2 points or more regardless of duration
- Progressive focal neurological signs
- Persistent vomiting
- New neurological signs
- Definite or suspected subarachnoid haemorrhage
- A CSF leak or other sign of a bony skull fracture

Fluids & Inotropes
- MAP > 90mmHg
- or Systolic BP > 120mmHg
- Use 0.9% Saline
- Consider norepinephrine infusion via central line if adequately filled & no evidence of ongoing blood loss

Other Parameters
- Core temperature 35 – 37°C
- Blood sugar 6 – 10mmol/l
- ONLY use glucose if BM < 4mmol/l
- Insert urinary catheter

Search for causes of hypotension
- Suture scalp wounds

Neurological Observations
- Once sedated & paralysed continue pupil checks every 15 minutes
- If pupils dilate or clinical condition deteriorates then re-contact Neurosurgical SP on immediately

CT scan head & C-spine down to T1
- Report within 1h of request
- No need to scan C-spine if neck cleared whilst GCS 15

Other Parameters
- Core temperature 35 – 37°C
- Blood sugar 6 – 10mmol/l
- ONLY use glucose if BM < 4mmol/l
- Insert urinary catheter

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Glasgow Coma Scale & Score
- Eye opening
- Spontaneous 4
- To sound 3
- To pressure 2
- None 1
- Verbal response
- Oriented 5
- Confused 4
- Words 3
- Sounds 2
- None 1
- Motor Response
- Dives commands 6
- Localising 5
- Normal flexion 4
- Abnormal flexion 3
- Extension 2
- None 1

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References:
MANAGEMENT OF ACUTE SPINAL INJURY
Management algorithm for patients with suspected acute spinal injury

- TREAT AS UNSTABLE – spinal immobilisation is a priority
- Immobilise with hard collar for suspected cervical injury
- 5 person spinal turn with head hold for all patients
- Spinal board and strapping as appropriate for transporting patient
- Consider mechanism of injury and clinically examine spine
- Moving and handling as per spinal protocol

**APPROPRIATE IMAGING WITHIN 2 HOURS**
Patients with severe TBI should have spine CT (occiput – T2) at time of head CT

**TREAT AS UNSTABLE** – spinal immobilisation is a priority

- Spinal immobilisation is a priority
- Immobilise with hard collar for suspected cervical injury
- 5 person spinal turn with head hold for all patients
- Spinal board and strapping as appropriate for transporting patient
- Consider mechanism of injury and clinically examine spine
- Moving and handling as per spinal protocol

**RADIOLOGY SpR / CONSULTANT TO REVIEW FILMS AND ISSUE REPORT ASAP**

- IS THERE A FRACTURE OR DISLOCATION?
  - A fracture anywhere on spine mandates MRI
  - but this will not be appropriate in acute stage in some patients (e.g. patients with severe TBI)

- YES
  - UNSTABLE THORACIC / LUMBAR #
  - Management dictated by precise nature and stability of injury
  - await instructions from Spinal team
  - Full spinal precautions: 5 person spinal turn
  - Nil by mouth – may require immediate surgery
  - Urinary catheter
  - May require collar for pain or ligament injury
  - In seated patients – apply collar for turning and when ‘waking’ from sedation
  - COMPLETE ASIA SCORE
  - COMPLETE CHECKLIST
  - Refer to NHNN ASAP via neurosurgical SpR on call (bleep 8100)

- NO
  - STABLE THORACIC / LUMBAR #
  - Management dictated by precise nature and stability of injury
  - Head injury may be accompanied by spinal injury

- STABLE CERVICAL #
  - Management dictated by precise nature and stability of injury
  - Head injury may be accompanied by spinal injury
  - Management as per Spinal protocol

- COMPLETE ASIA SCORE
- COMPLETE CHECKLIST
- File both in patient notes

**PRECONDITIONS FOR CLINICAL CLEARANCE**
- Fully alert and orientated
- No head injury
- No neck pain
- No abnormal neurology
- No significant other ‘distracting’ injury

- Provided preconditions met proceed to examine neck
- If no bruising, deformity or tenderness and patient has ‘pain free’ range of active movements, radiographic studies are not indicated

**RADIOLOGICAL STUDIES FOR CONSCIOUS SYMPTOMATIC PATIENTS**
- Radiological evaluation indicated for all patients who do not meet above Preconditions for Clinical Clearance

- Plain film radiography
  - 3 view plain film series: lateral, antero-posterior and open-mouth views

- CT should cover any areas of concern or uncertainty on plain film or clinical grounds

**RADIOLOGICAL STUDIES FOR UNCONSCIOUS INTUBATED PATIENTS**
- Standard radiological evaluation:
  - lateral, antero-posterior films and
  - CT scan from occiput to C3

- Oval view not possible
- Plain film radiography cannot exclude ligamentous injury

- Head injury may be accompanied by spinal injury
- Severe TBI patients should have lateral e-spine x-ray and CT imaging occiput-T2 at time of head CT
Intracranial Hypertension

**What is the best algorithm for treating?**

- Basic measures
- Extended measures

**Level II and Level III**
*Moderate or unclear clinical certainty*
Managing intracranial hypertension

**Level III**
*Unclear clinical certainty*
Improving outcome
Treating intracranial hypertension

1. Sedation and analgesia
2. Artificial ventilation
3. Head up 30° and neck straight

**Basic**

4. Mannitol
5. ICP monitoring
6. CSF drainage
7. Decompressive craniectomy

**Extended**

8. Barbiturate coma
Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

Head up 30° and neck straight

Basic

Extended

Level III

Unclear clinical certainty

ICP can be reduced with sedation and artificial ventilation

- Ideal agent rapid onset and recovery
  - Easily titrated
  - Given as infusion rather than bolus

Decompressive craniectomy

Barbiturate coma
Treating intracranial hypertension

- Sedation and analgesia
  - Artificial ventilation
  - Head up 30° and neck straight

**Level II**
*Moderate degree of clinical certainty*

Neuromuscular blocking agent used to aid ventilation rather than to treat ICP

- No direct effect on ICP
- Reduces ability to detect seizures
- Increases pneumonia and sepsis
- Prolonged ITU stay
- Critical illness polyneuropathy

Barbiturate coma
Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

Head up 30° and neck straight

Mannitol

Decompressive craniectomy

Barbiturate coma

ICP monitoring

Level II

Moderate degree of clinical certainty

Basic

Extended

Keeping the head in a neutral position at 30 to 45 degrees of elevation optimal for most brain injured patients

Once hypotension corrected and spinal injury excluded
Treating intracranial hypertension

- Sedation and analgesia
- Artificial ventilation
- CSF drainage
- Head up 30° and neck straight

**Basic**

**Extended**

**Mannitol**

**Level I**

**High degree of clinical certainty**

Large doses 1.8g/kg – 2.1g/kg effective in comatose patients with operative haematoma

**Level II**

**Moderate degree of clinical certainty**

Bolus doses 0.25g/kg - 1g/kg 20% mannitol infused over at least 15 minutes effective for ICP treatment

- Decompressive craniectomy
- Barbiturate coma
Treating intracranial hypertension

- Sedation and analgesia
- Artificial ventilation
- Head up 30° and neck straight

**Basic**

**Extended**

**Mannitol**

Repeated regular administration of mannitol over several days *not recommended*

**Aim for serum osmolality 290 - 320 mOsm/kg**

*Higher levels associated with*
  - dehydration
  - hypokalaemia
  - renal failure
  - rebound effect (BBB)*

**ICP monitoring**

**CSF drainage**

**Decompressive craniectomy**

**Barbiturate coma**
Treating intracranial hypertension

- Sedation and analgesia
- Artificial ventilation
- CSF drainage
- Head up 30° and neck straight

Basic

Extended

Mannitol

Hypertonic saline 3%, 7.5%, 20%
Also shown to be effective for ICP treatment
Target sodium concentration 145 – 150 mmol/l

Mannitol may have a detrimental effect on mortality when compared to hypertonic saline
Cochrane Database Syst Rev. 2007 Jan 24(1)

No strong evidence on
- concentration
- method of administration (bolus or infusion)

Must be given through a central venous line

Decompressive craniectomy
Barbiturate coma
Treating intracranial hypertension

Without ICP monitoring

Keep everything ‘normal’
- Normotension
- Normocapnoea
- Normovolaemia
- Normothermia
- Normoglycaemia

ICP monitoring

ClinicalTrials.gov

Guidelines for Managing Severe Traumatic Brain Injury (TBI)
Without Intracranial Pressure (ICP) Monitoring

Barbiturate coma
Treating intracranial hypertension

Level II
Moderate degree of clinical certainty

ICP–Targeted Therapy remains the ‘gold standard’ in the management of severe TBI patients

ICP monitoring

CSF drainage

Decompressive craniectomy

Barbiturate coma

Sedation and analgesia

Artificial ventilation

CSF drainage

intraparenchymal

Intraventricular

Basic

Extended

intraparenchymal

intraparenchymal
Treating intracranial hypertension

Level II

Moderate degree of clinical certainty

ICP < 20-25 mmHg
CPP = 60 mmHg

ICP monitoring

Treatment for raised ICP should be implemented only when ICP > 20 mmHg
Sedation and analgesia

Artificial ventilation

CSF drainage

Level II
Moderate degree of clinical certainty

CSF drainage (via external ventricular drain) helps in the management of raised ICP

Continuous vs ICP directed

CSF drainage

Decompressive craniectomy

Barbiturate coma
Sedation and analgesia

Artificial ventilation

CSF drainage

Head up 30° and neck straight

Basic

Extended

Mannitol

Decompressive craniectomy

Early decompressive craniectomy

Level II

Moderate degree of clinical certainty

Effective in lowering ICP

Reducing length of ICU stay

Early decompressive craniectomy

CSF drainage

Decompressive craniectomy

Barbiturate coma
Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

CSF drainage

Head up 30° and neck straight

Basic

Extended

Mannitol

Decompressive craniectomy

Barbiturate coma

ICP monitoring

Treating intracranial hypertension

Early decompressive craniectomy

**Level III**

*Unclear clinical certainty*

Improving outcome

CSF drainage

BMC Neurol. 2016 Jan 5;16(1):1 Prospective randomized evaluation of therapeutic decompressive craniectomy in severe traumatic brain injury with mass lesions (PRECIS): study protocol for a controlled trial
Treating intracranial hypertension

- Sedation and analgesia
- Artificial ventilation
- Head up 30° and neck straight

**Level I**
*High degree of clinical certainty*
Barbiturates **not** indicated for
prophylactic treatment or prevention
of intracranial hypertension

**Barbiturate coma**
Treating intracranial hypertension

- Sedation and analgesia
- Artificial ventilation
- Head up 30° and neck straight

- Basic
- Extended

Mannitol

**Level II**

*Moderate degree of clinical certainty*

High dose barbiturates effective for lowering ICP when other methods are ineffective

**Barbiturate coma**
Intracranial Hypertension

*Putting the recommendations into practice*
ICP Directed Therapy

Basic Measures

- Sedation and analgesia - propofol and fentanyl infusions
- Artificial ventilation - PaO₂ > 13kPa, PaCO₂ 4.5 – 5.0kPa, PEEP ≥ 5cmH₂O
- MAP ≥ 90mmHg or SBP ≥ 120mmHg
- Blood glucose 6-10mmol/l
- Temperature 35.5 – 37°C
- Head up 30° - provided not hypotensive and thoracic/lumbar spine cleared
- Commence spine clearance – straight bed tilt until thoracic/lumbar spine clear

Therapeutic goals once ICP monitoring commenced

ICP < 20 – 25mmHg
CPP = 60mmHg

To attain CPP ensure adequate fluid resuscitation before starting vasopressors
Insert oesophageal Doppler if indicated to guide fluid management
Doppler mandatory when vasopressors > 0.2mcg/kg/min or when requirements increasing

ICP > 25mmHg

- Check
  - Pupils - equal and reacting
  - ET tapes not tight / impeding venous drainage
  - Head & neck in neutral alignment
  - return to supine position
  - ICP waveform
  - PaCO₂ within parameters / adequate PaO₂
  - Sedation infusions intact
- Ensure
  - Adequate sedation - give bolus and observe effect
- Consider
  - Increasing rate of sedation
  - Bolus of muscle relaxant - if effective start infusion

Repeat checks & consider

- Reducing PaCO₂ to 4.0-4.5kPa
- Active cooling to 35°C
- Thiopentone (see Protocol)
- Insertion of SjvO₂ to allow further manipulation of PCO₂

Neurosurgeon

Obtain management plan
- CSF drainage
- Decompressive craniectomy

Dosing for analgesics and sedatives

- Propofol 0.5mg/kg test bolus 20-75mcg/kg/min infusion
- Fentanyl 2mcg/kg test dose 2-5mcg/kg/h infusion
- Midazolam 2mg test dose 2-4mg/h infusion

Propofol infusions complicated (PRIS)

- Rare but fatal complication of propofol infusion (particularly in association with use of vasopressors)
- Common clinical features include: hyperkalemia, hepatomegaly, lipemia, metabolic acidosis, myocardial failure, rhabdomyolysis, renal failure
- Always consider the diagnosis in patients receiving propofol, but particularly when using doses >75mcg/kg/min or when usage at any dose exceeds 48h
- Daily screen for CK, unexplained acidosis, ECG changes
No drug has shown statistically significant improvement in outcome.

Cerebro-protectors

*Have they demonstrated any benefits in TBI?*

- Dexanabinol
- Tirilazad
- Magnesium
- Erythropoietin
- Progesterone

NMDA antagonists

- Tranexamic acid
- Amantadine
- Zolpedim

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<tr>
<th>CEREBRO-PROTECTOR</th>
<th>STUDY OUTCOME</th>
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<tr>
<td>DEXANABINOL</td>
<td>Safe but not effective in TBI</td>
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<tr>
<td>TIRILIZAD</td>
<td>No evidence to support use</td>
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<td><em>Cochrane Database Syst Rev.</em> 2000; (4)</td>
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<td>MAGNESIUM</td>
<td>Continuous infusions for 5 days given within 8 h of moderate or severe TBI were not neuro-protective and might even have a negative effect</td>
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<td>ERYTHROPOIETIN</td>
<td>No benefit – more adverse events</td>
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<tr>
<td>PROGESTERONE</td>
<td>No clinical evidence to support usage</td>
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<td></td>
<td>NEJM.org. 2014 December 10</td>
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<tr>
<td>NMDA ANTAGONISTS</td>
<td>No neuro-protection and may worsen outcome</td>
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<td>CNS drugs 2001, 15:533-81</td>
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<tr>
<td>TRANEXAMIC ACID</td>
<td>Neither moderate benefits nor harmful effects can be excluded.</td>
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<td><em>BMJ</em> 2011; 343</td>
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<tr>
<td>AMANTADINE</td>
<td>No overall improvement</td>
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<td>nejm.820 org March 1, 2012</td>
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<tr>
<td>ZOLPEDIM</td>
<td>Results variable and effects short-acting</td>
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Seizure management
Should patients be given prophylactic anticonvulsants?

Steroids
Are steroids indicated in TBI?

Hypothermia
Does induced hypothermia improve outcome?

DVT prophylaxis
What is the safest way to prevent DVT?

Skull fractures
Do patients with skull fracture need prophylactic antibiotics?

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic anticonvulsants?

Steroids

Are steroids indicated in TBI?

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Does induced hypothermia improve outcome?

DVT prophylaxis

What is the safest way to prevent DVT?

Approximately 20-25% of patients with severe TBI can be expected to have at least one post-traumatic seizure (PTS)

Skull fractures

Do patients with skull fracture need prophylactic antibiotics?
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Do patients with skull fracture need prophylactic antibiotics?

Level I

High degree of clinical certainty

Treating patients at high risk with prophylactic AEDs for 1 week prevents early (<7 days) PTS

Prophylactic AEDs never been shown to reduce mortality or morbidity
Almost everything else!
Other frequently asked questions

Seizure management

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Almost everything else!

Other frequently asked questions

Skull fractures

Do patients with skull fracture need prophylactic antibiotics?

Level I

High degree of clinical certainty

Continuation of prophylactic AEDs beyond 1 week not recommended

Treatment does not prevent late (>7days) post-traumatic epilepsy
Seizure management

Should patients be given prophylactic anticonvulsants?

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Hypothermia

Does induced hypothermia improve outcome?

DVT prophylaxis

What is the safest way to prevent DVT?

Almost everything else!

Other frequently asked questions

Skull fractures

Do patients with skull fracture need prophylactic antibiotics?

Our practice to treat with AEDs only after 2nd witnessed seizure
Seizure management
Should patients be given prophylactic anticonvulsants?

**Steroids**

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**Almost everything else!**

Other frequently asked questions

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Does induced hypothermia improve outcome?

**DVT prophylaxis**

What is the safest way to prevent DVT?

**Level I**

*High degree of clinical certainty*

Steroids **not** indicated in the treatment of TBI

May cause complications that worsen outcome
Seizure management
Should patients be given prophylactic anticonvulsants?

Steroids
Are steroids indicated in TBI?

Hypothermia
Does induced hypothermia improve outcome?

DVT prophylaxis
What is the safest way to prevent DVT?

Skull fractures
Do patients with skull fracture need prophylactic antibiotics?

Hyperthermia worsens outcome
CMRO$_2$ decreases by 5-7% for each degree celsius

Hypothermia has an unequivocal effect in reducing ICP

Almost everything else!
Other frequently asked questions
Almost everything else!

Other frequently asked questions

Seizure management
Should patients be given prophylactic anticonvulsants?

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What is the safest way to prevent DVT?

Skull fractures
Do patients with skull fracture need prophylactic antibiotics?

Level III
Unclear clinical certainty

Induced hypothermia does not reduce mortality but may improve neurological outcome in survivors

Induced hypothermia remains controversial but widely used in practice

Eurotherm (2015) stopped early due to higher mortality in hypothermia group
Almost everything else!
Other frequently asked questions

Level I
High degree of clinical certainty

- Intermittent pneumatic compression is initial method of choice + TEDs

Level III
Unclear clinical certainty

- Prophylactic doses of anticoagulation probably carry only small risk of bleeding by 2 or 3 days after injury
  - Always discuss with neurosurgeon

DVT prophylaxis
What is the safest way to prevent DVT?

Skull fractures
Do patients with skull fracture need prophylactic antibiotics?
**Seizure management**

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What is the safest way to prevent DVT?

---

Almost everything else!

Other frequently asked questions

---

**Skull fractures**

Do patients with skull fracture need prophylactic antibiotics?

---

**Level II**

Moderate degree of clinical certainty

Use of prophylactic antibiotics **not** recommended even if CSF leak
In the Intensive Care Unit

How do we implement guidelines and standardise the care of TBI patients?
**Traumatic Brain Injury (TBI)**

**ITU Management Protocol**

**Neurological Assessment**

If patient not sedated

GCS, pupils and limbs as condition requires

Minimum 1 hourly until stable

Consider follow-up CT scan next day or earlier if clinically indicated

**ECG monitoring**

Continuous ECG monitoring

Record HR 1 hourly

12 Lead ECG on admission

Further 12 lead ECG

- When a change occurs

- When QT interval prolongs

- Avoid drugs which exacerbate QT prolongation

Maintain K⁺ > 4.5mmol

**Sodium and Water Balance**

Follow protocol for Sodium and Water Balance

**Observe for Diabetes Insipidus (DI)**

Routine urinalysis on admission

**Monitor urine output**

**Consider follow of increased ICP or clinical signs of**

**Maintain ICP <20**

ICP monitoring mandatory once

GI / RENAL

or earlier if clinically indicated

**Movicol BD 25ml**

**Patient sedated / ICP monitored**

Maintain ICP <20-25mmHg

1 hourly pupillary check

Follow protocol for ICP Directed Therapy

**Follow protocol for**

**Cortical Non-contrast CT scan**

**SEIZURE CONTROL**

**Phenytoin**

**Loading dose**: 15mg/kg

**Daily dose**: 300mg nocte

**Check for therapeutic level**

Goal: 10-20μg/ml which equates to ~35-70μmol/l

N.B. Acute toxicity is uncommon

If patient does have a seizure then local practice should determine the duration of anticonvulsant

**Prophylaxis with antiepileptic drugs (AEDs) not recommended**

If consultant preference to prescribe give short course of 7 days and then review

**Phenobarbitone**

**Loading dose**: 30mg/kg

**Daily dose**: 100mg nocte

**Check for therapeutic level**

Goal: 10-50μg/ml which equates to ~35-175μmol/l

**N.B. Acute toxicity is uncommon**

**Propofol infusion syndrome (PRIS)**

- Always consider the diagnosis in patients receiving propofol, but particularly when using doses >75mcg/kg/min or when usage at any dose exceeds 48h

- **Daily screen** for CK, unexplained acidosis, ECG changes

**Dosing for analgesics & sedatives**

**Propofol**

0.5-1.0mcg/kg test bolus

20-75mcg/kg/min infusion

**Fentanyl**

2mcg/kg test dose

2-5mcg/kg/min infusion

**Midazolam**

2mg test dose

2-4mg/h infusion

**Propofol infusion syndrome (PRIS)**

- Always consider the diagnosis in patients receiving propofol, but particularly when using doses >75mcg/kg/min or when usage at any dose exceeds 48h

- **Daily screen** for CK, unexplained acidosis, ECG changes

**Neuromuscular blocking agents**

- NMBAs have no direct effect on ICP but prevent rises produced by coughing on ET tube

**Atarcurium**

- Give bolus and observe effect on ICP

- If effective commence infusion (0.5mcg/kg/min)

- If history of asthma use dextrose containing fluids

**NG tube / enteral feeding**

Commence feeding as soon as possible unless contraindicated — follow protocol

**Blood glucose 6-10mmol**

**GI / RENAL**

Routine urinalysis on admission

Monitor urine output

Observe for Diabetes Insipidus (DI)

Follow protocol for Sodium and Water Balance

**Alkalosis, acidosis, ECG changes**

**Avoid hypotonic fluids and dextrose containing fluids**

**Can worsen cerebral oedema and ischaemia and plasma glucose**

**Temperature**

Maintain temperature

35.5-37°C

Paracetamol 1g qds

if temperature >37.5°C

**Respiratory**

**SaO₂ monitoring**

Non-ventilated patient

Record respiratory rate

Oxygen therapy only if SaO₂ <95%

Ventilated patient

Admission chest x-ray

**Consider use of cooling blanket if ICP persistently high despite adequate sedation**

**If patient cooled but not fully sedated**

This can cause shivering which will increase ICP

**Temperature**

If patient does have a seizure then local practice should determine the duration of anticonvulsant

**Seizure control not recommended**

If consultant preference to prescribe give short course of 7 days and then review

Phenytoin

Loading dose: 15mg/kg

Daily dose: 300mg nocte

**Check for therapeutic level**

Goal: 10-20μg/ml which equates to ~35-70μmol/l

N.B. Acute toxicity is uncommon

If patient does have a seizure then local practice should determine the duration of anticonvulsant

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Putting evidence-based recommendations into a clinical pathway

Does it help?

- Helps streamline initial management
- Helps standardise subsequent management in ICU
- Can significantly improve patient outcome
- Can make patient management more cost effective
  - Higher costs in the acute setting
  - But significant cost reductions later if overall outcome is improved
- Can be confident that we are ‘doing no harm’