



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

TBI Guidelines

The Recommendations

◆ Mo
by

Level I - Standards

*Accepted management strategies with a
high degree of clinical certainty*

◆ Mo

Level II - Guidelines

*Management strategies with a
moderate degree of clinical certainty*

■

■

■

Level III - Options

*Management strategies with
unclear clinical certainty*

TBI Guidelines

Key Concepts

◆ 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 O₂ and CO₂ should we aim for?
Should we hyperventilate TBI patients?*

◆ Oxygen

mmHg / SpO₂ <90%)

Level II

Moderate degree of clinical certainty

...ial cause

...jury

◆ Carbon

PaO₂ >97.5mmHg

P

PaCO₂ 33.7mmHg

...aemia

Level I

High degree of clinical certainty

◆ Hyperventilation

Avoid PaCO₂ <30mmHg

*Hyperventilation used only as temporising
measure in profoundly injured patients
exhibiting signs of herniation
(abnormal posturing, fixed /dilated pupils)*

Circulation

What BP should we aim for?

◆ Hypotension (defined as SBP < 90mmHg)

- May lead to
- Most
- High

Level II

Moderate degree of clinical certainty

Systolic BP > 120mmHg
or MAP > 90mmHg

◆ Hypertension

- May lead to re-bleeding (CPP = MAP – ICP) cerebral oedema,

◆ 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

Circulation

*Which resuscitation fluid should we use
and how much?*

◆ Hypovolaemia not

Level II

Moderate degree of clinical certainty

◆ SAFE

- Hypo
- May be

Prevent hypovolaemia with
liberal use of **crystalloids**
with goal of intravascular
euvolaemia

◆ Avoid glucose-

- Maintain glucose 6-10mmol/l

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**¹⁹⁷⁴ most widely used and most extensively evaluated tool for acute classification and assessment of TBI patients
- ◆ Considered to be the '**Gold Standard**'

Nursing Practice Review Neurology

Keywords: Consciousness/Glasgow Coma Scale/Clinical assessment/Standardisation
• This article has been double-blind peer reviewed

Forty years after its initial implementation, the Glasgow Coma Scale has been updated to address variations in technique that have developed over time

Forty years on: updating the Glasgow Coma Scale

In this article...

- Variations in the use of the Glasgow Coma Scale
- Review of the scale's composition and its application
- Structured approach to assessment

5 key points

- 1 The Glasgow Coma Scale is an integral part of assessing levels of consciousness
- 2 It uses a simple standardised approach
- 3 The scale has been revised to make sure it remains an accurate tool
- 4 The overall coma score should not be used to convey clinical findings
- 5 The scale can be used with children who are over 8 years old

Author Sir Graham Teasdale is emeritus professor of neurosurgery, Institute of Health and Wellbeing, University of Glasgow; Douglas Alan was senior lecturer, School of Health and Life Sciences, Glasgow Caledonian University; Paul Brennan, is clinical lecturer in neurosurgery, Department of Clinical Neurosciences, NHS Lothian, IGMM, University of Edinburgh; Evelyn McElhinney is lecturer, School of Health and Life Sciences, Glasgow Caledonian University; Laura Mackinnon is senior charge nurse, Department of Neurosurgery, Institute of Neurological Sciences, Southern General Hospital, Glasgow.

Abstract Teasdale G (2014) Forty years on: updating the Glasgow Coma Scale. *Nursing Times*; 110: 42, 12-16. Since the Glasgow Coma Scale was developed 40 years ago it has been accepted throughout the world as a method for assessing impaired consciousness. This article addresses the variations in technique that have developed since the scale was published. The details of the composition of the scale and its application are reviewed, and a structured approach to assessment set out. These provide a basis for standardising practice and ensure the scale is useful, in a practical sense, in the future.

The Glasgow Coma Scale (GCS) was developed in 1974 to provide a practical method for the assessment of impaired consciousness (Teasdale and Jennett, 1974). Nursing, medical and other staff welcomed its straightforward approach and use of simple terms to record and communicate their findings; the scale became an integral

part of the care of patients with acute brain injury from head trauma, intracranial haemorrhage and many other causes.

The GCS reflects the initial severity of brain dysfunction, while serial assessments demonstrate the evolution of the injury. Each is crucial for prognosis and an essential tool for research studies.

Four decades after its introduction, the GCS has gained worldwide acceptance (Teasdale et al, 2014). It is now employed in more than 80 countries, has been translated into more than 60 languages and there are more than 16,000 references to its use (Middleton, 2012).

Unfortunately, this widespread use has

TABLE 1. GCS TERMS OF 1974 AND 2014

Indicator of level of consciousness	Term used	
	1974	2014
Eye opening	Spontaneous	Spontaneous
	To speech	To sound
	To pain	To pressure
	None	None
Verbal response	Orientation	Orientated
	Confused conversation	Confused
	Inappropriate speech	Words
	Incomprehensible speech	Sounds
	None	None
Motor response	Obedient commands	Obeys commands
	Localising	Localising
	Flexor	Normal flexion
	Extensor posturing	Abnormal flexion
	None	None

Exposure (Secondary survey)

Does the patient have associated spinal injury?

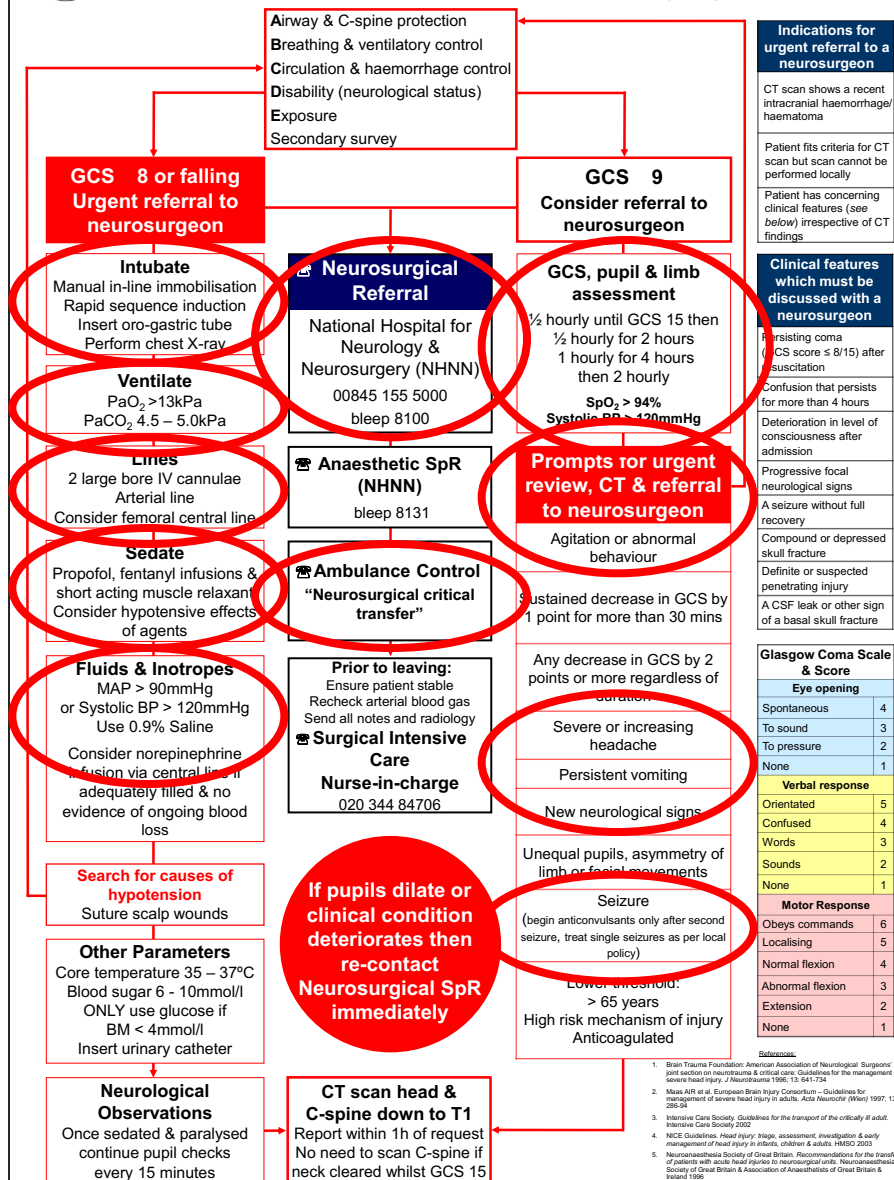
- ◆ 5% incidence of associated cervical spine injury in moderate and severe TBI
- ◆ Assume unstable cervical spine
- ◆ Measures taken to 'clear the cervical spine'
 - *impacts on ability to manage raised intracranial pressure*

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





MANAGEMENT OF ACUTE SPINAL INJURY

Management algorithm for patients with suspected acute spinal injury

University College
London Hospitals
NHS Foundation Trust

- 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

DOES THE PATIENT REQUIRE RADIOLOGICAL INVESTIGATION?

NO

COMPLETE ASIA SCORE

COMPLETE CHECKLIST
File both in patient notes

YES

APPROPRIATE IMAGING
WITHIN 2 HOURS

Patients with severe TBI should have spine CT (occiput – T2) at time of head CT

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

DISCUSS NEED FOR
FURTHER IMAGING
CT SCAN / MRI

COMPLETE ASIA SCORE
For awake patients

COMPLETE CHECKLIST

REFER TO NHNN ASAP via
NEUROSURGICAL SpR on call

BLEEP 8100

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

CONSCIOUS SYMPTOMATIC PATIENTS

Radiological evaluation indicated for
all patients who do not meet above
'Preconditions for Clinical Clearance'

Imaging should be technically adequate
and interpreted by experienced clinicians

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

Must include base of occiput to T1

If lower c-spine not visualised CT of
region is indicated

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

Head injury may be accompanied by spinal
injury and measures should always be taken
to 'clear the cervical spine'

RADIOLOGICAL STUDIES FOR UNCONSCIOUS INTUBATED PATIENTS

Standard radiological examination:
lateral, antero-posterior films and
CT scan from occiput to C3

Odontoid view not possible

Plain film radiology cannot exclude
ligamentous injury

Head injury may be accompanied by spinal
injury and measures should always be taken
to 'clear the cervical spine'
Severe TBI patients should have lateral
c-spine x-ray and CT imaging occiput-T2
at time of head CT

American Spinal Injury Association (ASIA) Score

Motor	Sensory	Anal	Bladder
2	2	2	2
3	3	3	3
4	4	4	4
5	5	5	5
6	6	6	6
7	7	7	7
8	8	8	8
9	9	9	9
10	10	10	10

STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

ISC @S

Motor

Sensory

Anal

Bladder

Key

Spinal Cord Injury

UNSTABLE CERVICAL

Management dictated by precise nature and stability
of injury - await instructions from Spinal team

Full spinal precautions: collar / 5 person spinal turn

Nil by mouth – may require immediate surgery
Consider NG tube (to prevent vomiting and aspiration)

Urinary catheter

If cervical traction required contact Spinal nurses
via NHNN Clinical Site Manager – Bleep 8240

STABLE CERVICAL

Maintain full spinal precautions until
Spinal Clearance Checklist completed

May require collar for pain or ligament injury

In sedated patients – apply collar for turning
and when 'waking' from sedation

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

STABLE THORACIC / LUMBAR

Maintain full spinal precautions until
Spinal Clearance Checklist completed

Algorithm from evidence based guidelines and expert opinion
ATLS
Consortium for Spinal Cord Medicine, Early Acute Management in
Adults with Spinal Cord Injury: A clinical practice guideline for
health care professionals, 2008
EAST 2000
Scottish Intercollegiate Guidelines Network (SIGN 2009)
NICE TBI Guidelines 2007
NHNN MDT: Neurocritical care, Spinal Team, Radiologists

Intracranial Hypertension

What is the best algorithm for treating?

◆ Basic measures

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

Level III
Unclear clinical certainty
Improving outcome

◆ Extended measures

Treating intracranial hypertension

Sedation and analgesia



Artificial ventilation



Head up 30° and neck straight



Basic

Extended

Mannitol



ICP monitoring



CSF drainage



Decompressive craniectomy



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

Basic

Ext

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



Basic

Extended

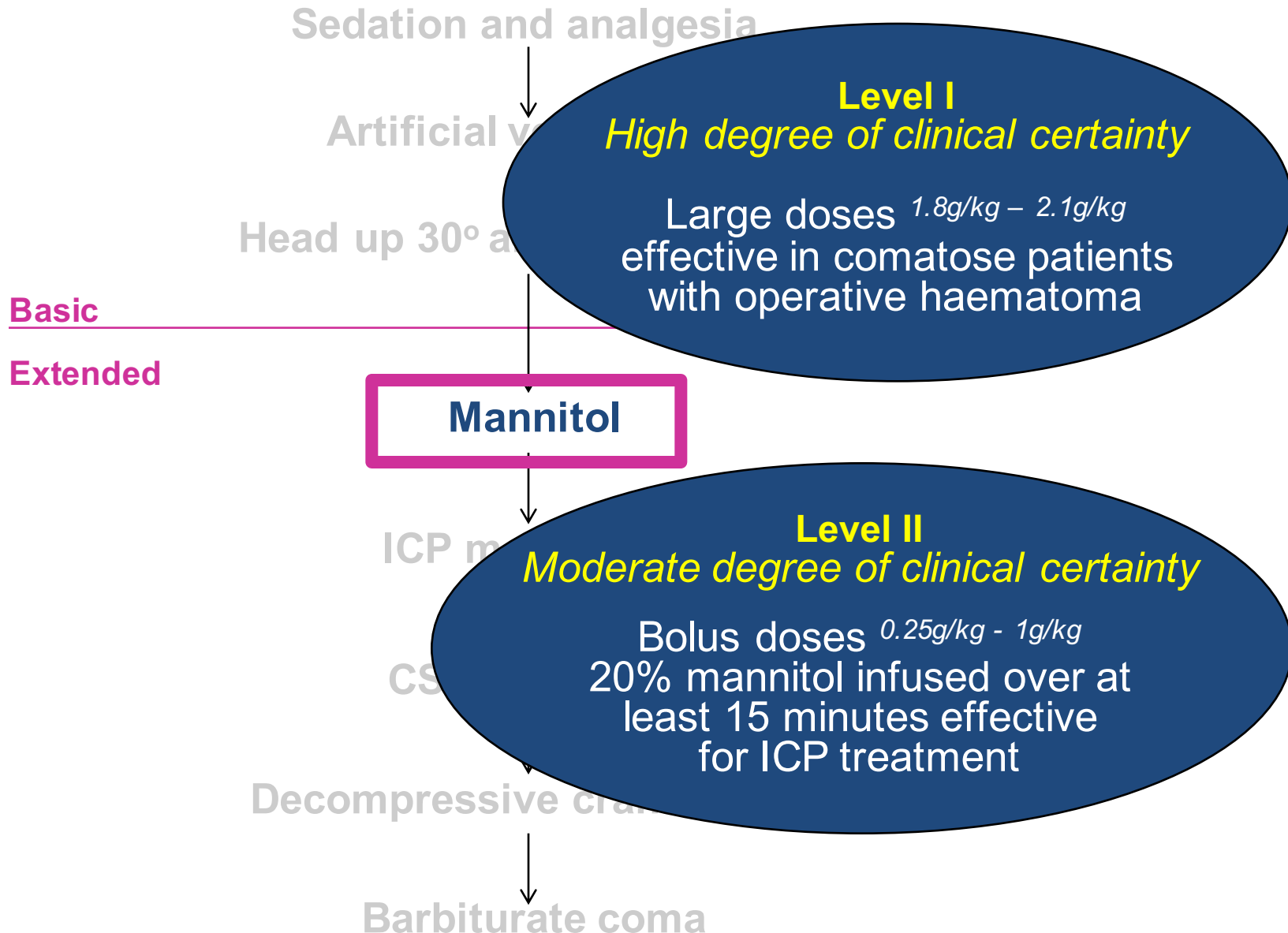
Level II

Moderate degree of clinical certainty

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



Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

Head up 30° and

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

Basic

Extended

Mannitol

ICP monitoring

Aim for serum osmolality
290 - 320mOsm/kg

CSF

Higher levels associated with

- dehydration
- hypokalaemia
- renal failure
- rebound effect (BBB)

Decompressive craniectomy

Barbiturate coma

Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

Head up 30° and

Hypertonic saline 3%, 7.5%, 20%

*Also shown to be effective
for ICP treatment*

Target sodium concentration
145 – 150mmol/l

Basic

Extended

Mannitol

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

Sedation and analgesia

Without ICP monitoring

Keep everything 'normal'

- *Normotension*
- *Normocapnoea*
- *Normovolaemia*
- *Normothermia*
- *Normoglycaemia*

Basic

Extended

ICP monitoring

ClinicalTrials.gov

A service of the U.S. National Institutes of Health

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

Barbiturate coma

Treating intracranial hypertension

Sedation and analgesia

Level II

Moderate degree of clinical certainty

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

Basic

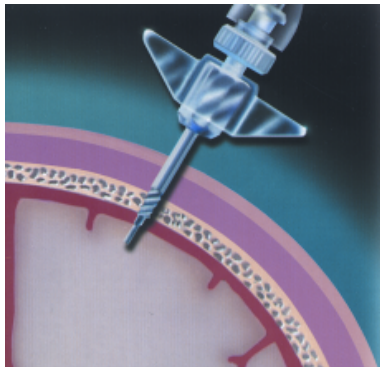
Extended

ICP monitoring

CSF drainage

Decompressive craniectomy

Barbiturate coma



intraparenchymal



Intraventricular

Treating intracranial hypertension

Sedation and analgesia

Level II

Moderate degree of clinical certainty

ICP <20-25mmHg

CPP = 60mmHg

Basic

Extended

ICP monitoring

*Treatment for raised ICP
should be implemented only when
ICP >20mmHg*

Treating intracranial hypertension

Sedation and analgesia

Artificial ventilation

Level II

Moderate degree of clinical certainty

Basic

Extensive

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

Continuous vs ICP directed

CSF drainage

Decompressive craniectomy

Barbiturate coma

Treating intracranial hypertension

Sedation and analgesia



Artificial ventilation

Early decompressive
craniectomy

Level II

Moderate degree of clinical certainty

Effective in lowering ICP

Reducing length of ICU stay



CSF drainage



Decompressive craniectomy



Barbiturate coma

Basic

Extensive

Treating intracranial hypertension

Sedation and analgesia



Artificial ventilation

Early decompressive
craniectomy

Level III

Unclear clinical certainty

Improving outcome

Basic

Extensive



CSF drainage



Decompressive craniectomy

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



Basic

Extended

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



Traumatic Brain Injury (TBI)

ICP Directed Therapy

University College
London Hospitals
NHS

Basic Measures

- ◆ Sedation and analgesia - propofol and fentanyl infusions
- ◆ Artificial ventilation - $\text{PaO}_2 > 13\text{kPa}$, $\text{PaCO}_2 4.5 - 5.0\text{kPa}$, PEEP $5\text{cmH}_2\text{O}$
- ◆ $\text{MAP} \geq 90\text{mmHg}$ or $\text{SBP} \geq 120\text{mmHg}$
- ◆ Blood glucose $6-10\text{mmol/l}$
- ◆ Temperature $35.5 - 37^\circ\text{C}$
- ◆ Head up 30° - provided not hypotensive and thoracic / lumbar spine cleared
- ◆ Commence spine clearance - straight bed tilt until thoracic/lumbar spine clear

Reserved for acute rises in ICP

PEEP
Use of PEEP and effect on ICP should be individualised to achieve PaCO_2 target

Therapeutic goals once ICP monitoring commenced

ICP $< 20 - 25\text{mmHg}$

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 $> 0.2\text{mcg/kg/min}$ or when requirements increasing

ICP $< 20\text{mmHg}$

Continue current therapy

Neurosurgeon may consider 'waking' patient to assess

Consider C-spine clearance and need for further imaging prior to 'wake up'

Neuromuscular blocking agents
NMBAs have no direct effect on ICP but prevent rises produced by coughing on ET tube

Atracurium
- Give bolus and observe effect on ICP
- If effective commence infusion
- If history of asthma use **vecuronium**
- Propofol and NMBAs **not** compatible
- Seizures difficult to detect if paralysed (may be signalled by bilateral pupillary dilatation, small \uparrow ABP + \uparrow ICP)

Therapeutic pathways \rightarrow
Additional considerations \dashrightarrow

ICP $20-25\text{mmHg}$

Check

Pupils - equal and reacting
ET tapes
not tight / impeding venous drainage
Head & neck in neutral alignment
return to supine position
ICP waveform
 PaCO_2 within parameters / adequate PaO_2
Sedation infusions intact

Ensure

Adequate sedation - give bolus and observe effect

Consider

Increasing rate of sedation
Bolus of muscle relaxant - if effective start infusion

Propofol infusion syndrome (PRIS)

- Rare but lethal 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 $> 75\text{mcg/kg/min}$ or when usage at any dose exceeds 48h
- **Daily screen** for \uparrow CK, unexplained acidosis, ECG changes

ICP $> 25\text{mmHg}$

Repeat checks & consider

Reducing PaCO_2 to $4.0-4.5\text{kPa}$
Active cooling to 35°C
Thiopentone (see Protocol)
Insertion of SjvO_2 to allow further manipulation of PCO_2

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

Cerebro-protectors

Have they demonstrated any benefits in TBI?

No drug has shown statistically significant improvement in outcome

Dexanabinol

NMDA antagonists

Tirilazad

Tranexamic acid

Magnesium










Amantadine

Erythropoietin

Zolpedim

Progesterone

Stocchetti et al. Neuroprotection in acute brain injury: an up-to-date review Critical Care (2015) 19:186

CEREBRO-PROTECTOR	STUDY OUTCOME
 DEXANABINOL	<i>Safe but not effective in TBI</i> Lancet Neurol. 2006 Jan;5(1):38-45
 TIRILIZAD	<i>No evidence to support use</i> Cochrane Database Syst Rev. 2000;(4)
 MAGNESIUM	<i>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</i> Lancet Neurol. 2007 Jan;6(1):29-38
 ERYTHROPOIETIN	<i>No benefit – more adverse events</i> Stocchetti et al. Critical Care (2015) 19:186
 PROGESTERONE	<i>No clinical evidence to support usage</i> NEJM.org. 2014 December 10
 NMDA ANTAGONISTS	<i>No neuro-protection and may worsen outcome</i> CNS drugs 2001, 15:533-81
 TRANEXAMIC ACID	<i>Neither moderate benefits nor harmful effects can be excluded.</i> BMJ 2011;343
 AMANTADINE	<i>No overall improvement</i> nejm.820 org march 1, 2012
 ZOLPEDIM	<i>Results variable and effects short-acting</i> Am J Phys Med Rehab 2014, 93:101-13

Almost everything else!

Other frequently asked questions

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?

Valadka, A.B. & Andrews B.T. 2005 Neurotrauma: Evidence-Based
Answers to Common Questions Thieme Medical Publishers

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic anticonvulsants?

Steroids

Are steroids indicated?

Does it

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

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?

Level I

High degree of clinical certainty

Does

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*

Do patients benefit from antibiotics?

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic anticonvulsants?

Steroids

Are steroids indicated?

Level I

High degree of clinical certainty

Does

Continuation of prophylactic AEDs
beyond 1 week **not** recommended

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

Do patients with skull fractures need prophylactic antibiotics?

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic anticonvulsants?

Steroids

Are steroids indicated?

Does incidence

Our practice to treat with
AEDs only after
2nd witnessed seizure

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?

Level I

High degree of clinical certainty

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

*May cause complications that
worsen outcome*

Do patients benefit from antibiotics?

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic antiepileptics?

Steroids

Are steroids indicated in TBI?

Hypothermia

Does induced hypothermia improve outcome?

DVT prophylaxis

What is the safest way to manage DVT?

Skull fracture

Do patients with skull fracture benefit from craniectomy?

Hyperthermia worsens outcome
CMRO₂ decreases by 5-7%
for each degree celsius

Hypothermia has an
unequivocal effect in
reducing ICP

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic antiepileptics?

Steroids

Are steroids indicated in TBI?

Hypothermia

Does induced hypothermia improve outcome?

DVT prophylaxis

What is the safest option?

Skull fracture

Do patients with skull fractures benefit from craniotomy?

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?

Almost everything else!

Other frequently asked questions

Seizure management

Should patients be given prophylactic anticonvulsants?

Steroid

Are steroids in

Level II

Moderate degree of clinical certainty

Does induce

*Use of prophylactic antibiotics **not** recommended even if CSF leak*

What is the safe ... DVT?

Skull fractures

Do patients with skull fracture need prophylactic antibiotics?

In the Intensive Care Unit

How do we implement guidelines and standardise the care of TBI patients?



Traumatic Brain Injury (TBI)

ITU Management Protocol

University College
London Hospitals
NHS Foundation Trust

NEUROLOGY

Neurological Assessment
If patient not sedated

GCS, pupils and limbs as condition requires
Minimum 1 hourly until established as stable

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

Patient sedated / ICP monitored

Maintain ICP <20-25mmHg
1 hourly pupil check

Follow protocol for ICP Directed Therapy

ICP therapy is only definitively indicated if raised ICP demonstrated by monitoring, if there is CT evidence of increased ICP or clinical signs of developing intracranial herniation

ICP monitoring mandatory once patient sedated and ventilated

20% Mannitol
To control acutely elevated ICP
Bolus 0.25-1.0g/kg infused over at least 15 minutes

VITAL SIGNS

Heart rate

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 prolonged do daily to monitor progression
- Avoid drugs which exacerbate QT prolongation
Maintain K⁺ >4.5mmol

ECG changes after TBI
- ST segment depression
- Prolonged QT interval
- Bundle branch block
- Sinus arrhythmias
MI must be excluded
Troponin levels should be taken at time of change and 12 hours later
In acute phase pharmacological management of ECG abnormalities should be implemented with care

Blood Pressure

Non-sedated patient

Record BP 1 hourly
SBP >120mmHg
or
MAP >90mmHg

Patient sedated / ICP monitored
Arterial line

Transducer measured at level of heart

Record BP 1 hourly
BP to maintain CPP = 60mmHg

If unable to maintain CPP with fluid replacement start noradrenaline
Avoid hyperaemia - if CPP >70mmHg reduce vasopressors

Femoral CVP line

Head down tilt for jugular line contraindicated

IV crystalloids

Maintain / restore normovolaemia and normal blood chemistry
Consider oesophageal Doppler - mandatory when vasopressors >0.2mcg/kg/min
Aim for serum osmolality 290-300mosmol/l

Respiratory

SaO₂ monitoring

Non-ventilated patient

Record respiratory rate
Oxygen therapy only if SaO₂ <95%

Ventilated patient

Admission chest x-ray

Record ET tube length

In acute phase aim for:

PaO₂ >13kPa

PaCO₂ 4.5-5kPa

HYPERVENTILATION reserved for acute rises in ICP
Use of PEEP and its effect on ICP should be individualised to achieve PaO₂ target

Suctioning / chest physiotherapy
Prevent hypoxia and hypercarbia and Excessive / prolonged increases in ICP
Pre-oxygenate with 100% O₂
Consider sedation bolus
Maximum 3 catheters in 1 session
Closed suction circuit in all patients

Temperature

Maintain temperature 35.5-37°C

Paracetamol 1g qds if temperature >37.5°C

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

Persistent hyperpyrexia may be result of damage to hypothalamus but microbiological causes must be investigated

If temperature shows peaks and troughs ? Infection
If temperature sustained despite above intervention ? hypothalamic damage

HYDRATION AND NUTRITION

Monitor fluid balance

Aim for intake of 2-3l / 24h
Consider individual patient requirements

1st choice: IV crystalloids

Avoid hypotonic fluids and dextrose containing fluids
Can worsen cerebral oedema and ischaemia and plasma glucose

NG tube / enteral feeding
Commence feeding as soon as possible unless contraindicated - follow protocol

Blood glucose 6-10mmol

Daily bloods

Monitor Na⁺

If hyponatraemia (<135mEq/L) or hypernatraemia (>150mEq/L)

Follow protocol for Sodium and Water Balance

Dosing for analgesics & 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 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

Atracurium

- Give bolus and observe effect on ICP
- If effective commence infusion (0.5mg/kg/h)
- If history of asthma use vecuronium
- Propofol and NMBAs not compatible (infuse as per UCLH guidelines)
- Seizures difficult to detect if paralysed (may be signalled by bilateral pupillary dilatation, small ↑ ABP + ↑ ICP)

GI / RENAL

Routine urinalysis on admission
Monitor urine output

Observe for Diabetes Insipidus (DI)
Follow protocol for Sodium and Water Balance

Chart bowel movements

Prescribe regular aperients

Senna once daily 10-20ml
Movicol BD 25ml

ANALGESIA / SEDATION

Non-ventilated patient
Paracetamol

morphine

Ventilated patient
Propofol infusion 20-75mcg/kg/min
Fentanyl infusion 2-5mcg/kg/min
Midazolam infusion 2-4mg/h
Infusion rates > the above must be

Follow protocol for ICP Directed Therapy

PREVENTION of DVT

Mechanical prophylaxis of VTE

Anti-embolic stockings

Intermittent pneumatic compression (IPC) device

LMWH

Consider after 5-7 days
Discuss with Neurosurgeon

If VTE develops during this time an IVC Filter should be inserted

SEIZURE CONTROL

Routine prophylaxis with antiepileptic drugs (AEDs) 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⁻¹ which equates to ~35-70µmol l⁻¹

N.B. Acute toxicity is uncommon

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

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'