Guidelines for the Management of Traumatic Brain Injury

AIM: To provide guidance on the management of patients with traumatic brain injury
SCOPE: Royal Sussex County Hospital & Princess Royal Hospital Intensive Care Units

INDEX

INTRODUCTION

The outcome and potential for successful rehabilitation after brain injury depend both on the primary brain damage, and on the prevention of **secondary brain injury**. Complete standardisation of treatment is impractical, but a common "core approach" is desirable. Little can be done about the primary brain injury, but the duration and severity of secondary insults influence outcome [1]. The trend in reduced mortality and improved outcomes from traumatic brain injury (TBI) has been the result of the use of evidence-based protocols that emphasise the optimisation of physiological parameters, to minimise secondary brain injury.

Much of the supporting evidence for the guidelines comes from:

EBIC: European Brain Injury Consortium 1997 guidelines based on consensus and expert opinion [1]
BTF: Brain Foundation Guidelines 2016 evidence-based guidelines [2]
SIBICC: Seattle International Severe Traumatic Brain Injury Consensus Conference [44, 45]

PATHOPHYSIOLOGY

Damage to the brain following trauma results from the immediate (primary) injury, caused by the impact itself, and secondary brain injury which develops in the hours or days after the initial impact.

Primary Brain Injury

- Disruption of brain vessels
- Haemorrhagic contusion
- Diffuse axonal injury
- Haematoma

Secondary Brain Injury

Systemic secondary insults		Intracranial secondary insults	
Events	Main causes	**Events**	Main causes
Hypoxaemia	• Hypoventilation • Thoracic injury • Aspiration pneumonia • Anaemia	**Raised intracranial pressure and/or brain shift**	• Mass lesion • Vasodilation • Impaired cerebral venous drainage (position, ET tube ties, coughing etc.) • Oedema • Hydrocephalus
Hypotension	• Hypovolaemia • Cardiac failure • Sepsis • Spinal cord injury	**Vasospasm Stroke/infarction**	• Traumatic subarachnoid haemorrhage?
Hypercarbia	• Respiratory depression	**Seizures**	• Cortical brain injury
Hypocarbia	• Hyperventilation, spontaneous or induced	**Infection**	• Skull base fracture • Compound depressed skull fracture
Hyperthermia	• Hypermetabolism • Stress response • Infection		
Hyperglycaemia	• Hypothermia • IV dextrose infusion • Stress response		
Hypoglycaemia	• Inadequate nutrition		
Hyponatraemia	• SIADH • CSW		

ADMISSION AND INITIAL MANAGEMENT

The SIBICC conference [44] developed a list of "Tier Zero" interventions, viewed as fundamental to the care of patients with severe TBI, defined as Glasgow Coma Scale [GCS] Score of 3 to 8:

Tier Zero (Basic Severe TBI Care - Not ICP Dependent)

Expected Interventions:

- Admission to ICU
- Endotracheal intubation and mechanical ventilation
- Serial evaluations of neurological status and pupillary reactivity
- Elevate HOB 30-45°
- Analgesia to manage signs of pain (not ICP directed)
- Sedation to prevent agitation, ventilator asynchrony, etc. (not ICP directed)
- Temperature management to prevent fever
  - *Measure core temperature*
  - *Treat core temperature above 38°C*
- Consider anti-seizure medications for 1w only (in the absence of an indication to continue)
- Maintain CPP initially ≥ 60 mmHg
- Maintain Hb > 7g/dL
- Avoid hyponatremia
- Optimize venous return from head (eg. keeping head midline, ensure cervical collars are no too tight)
- Arterial line continuous blood pressure monitoring
- Maintain SpO2 ≥ 94%

Recommended Interventions:

- Insertion of a central line
- End-tidal CO_2 monitoring

The majority of these should be in place prior to ICU admission. The list is not exhaustive and different targets will be needed in many cases. This is discussed in more detail below.

Monitoring

Recommendation (Action)	Justification (Rationale)
Essential monitoring and access	Standard ICU monitoring plus: • Wide bore oro-gastric tube (or naso-gastric tube if no known or suspected base of skull fracture). • Temperature monitoring • ICP +/- brain tissue oxygen monitoring if appropriate • BIS monitoring to ensure adequate sedation
ICP / CPP monitoring	ICP should be monitored in all salvageable patients with a severe TBI (GCS 3-8 after resuscitation) and an abnormal CT scan. An abnormal CT scan of the head is one that reveals haematomas, contusions, swelling, herniation or compressed cisterns.

	ICP monitoring is indicated in severe TBI with a normal CT scan if two or more of the following features are noted at admission: age over 40 years, unilateral or bilateral motor posturing (a motor score of 2 or 3), or systolic blood pressure (BP) <90 mmHg
	When calculating CPP in TBI, the MAP (used in the equation CPP=MAP−ICP) should be the mean cerebral arterial pressure, estimated to exist at the level of the middle cranial fossa, which can be approximated by positioning (levelling) the arterial transducer at the tragus of the ear. The arterial transducer must be repositioned to remain levelled with the tragus following changes in body elevation or position.
Brain tissue oxygen	If brain tissue oxygen monitoring ($P_{bt}O_2$ - Licox) in use, follow separate $P_{bt}O_2$ optimisation strategy as below
Additional monitoring	Cardiac output monitoring to be considered: • if no response to crystalloid and no response to 0.1microgram/kg/min of noradrenaline • If acute or pre-existing cardiac disease • In systemic sepsis • If cardiac contusion known or suspected • If pulmonary oedema develops Peripheral nerve stimulator if administering muscle relaxants Consider EEG if suspecting seizures or during barbiturate coma
Specific investigations above baseline	• Troponin if chest trauma or > 50 years old • Triglyceride level as baseline • Creatine kinase (CK) • Consider urine and serum osmolality and urine electrolytes

Cervical Spine

Recommendation	Justification
Clear the cervical spine if possible	The cervical spine should be cleared according to local guidelines (see Microguide)
If a hard collar is required change to an **Aspen**	This is less likely to cause ulceration
If injury detected	Management of a detected injury must involve a consultant neurosurgeon
It is strongly recommended that the thoracolumbar spine of an unconscious trauma patient be cleared within 48-72 hours	Beyond this time the morbidity and mortality of spinal precautions (secondary to skin ulceration, pneumonia, thromboembolism, and venous access and airway complications) probably exceed the risk of missed spinal injury [4]

Initial Goals

Recommendation	Justification
Sedation	Full sedation and analgesia, ± paralysis if required
Ventilation	• Aim lung protective ventilation • Target PaO_2 > 13 kPa • Target $PaCO_2$ 4.7-5.3 kPa • Avoid routinely decreasing $PaCO_2$ < 4.0 kPa except in critical situations when hyperventilation may be needed to "buy time" (e.g. prior to theatre or CT scan) • The decision to reduce $PaCO_2$ to < 4.0kPa must be made by a consultant intensivist and neurosurgeon, ideally with $PbtO_2$ monitoring in situ
Surgery Optimise Cerebral Perfusion	Early evacuation of space-occupying lesions CPP > 60 mmHg ICP < 22 mmHg
Brain oxygenation Consider EVD Manage temperature Optimise CVS	$P_{bt}O_2$ > 20 mmHg Consider CSF drainage to manage intracranial hypertension Measure core temperature Avoid temperature > 38 °C Optimal haemodynamic and volume status
Seizure control Feed	Prophylactic AEDs for 1 week (see details below) Establish early enteral feeding - within 24 hours if possible
Control glucose	Blood sugar 4.5-10.0 mmol/L (minimum of daily blood sugars, 2-6hrly if high)
Treat infection	Treat established or clinically significant infection
Coagulation	If mild to moderate TBI (GCS 9-15 after resuscitation) and within 3 hours of injury, give tranexamic acid 1g IV bolus + 1g IV infusion over 8 hours (not applicable to most ICU patients) [50]

MANAGEMENT OF SEVERE TBI GUIDED BY INTRACRANIAL PRESSURE MEASUREMENTS

The most recent versions of the Brain Trauma Foundation TBI guidelines do not contain treatment protocols, owing to a lack of evidence regarding the relative efficacy of interventions [2]. In response, the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC) generated a consensus-based protocol. This three-tier algorithm focusses on treating elevated ICP, wherein higher tiers involve therapies with higher risk [44].

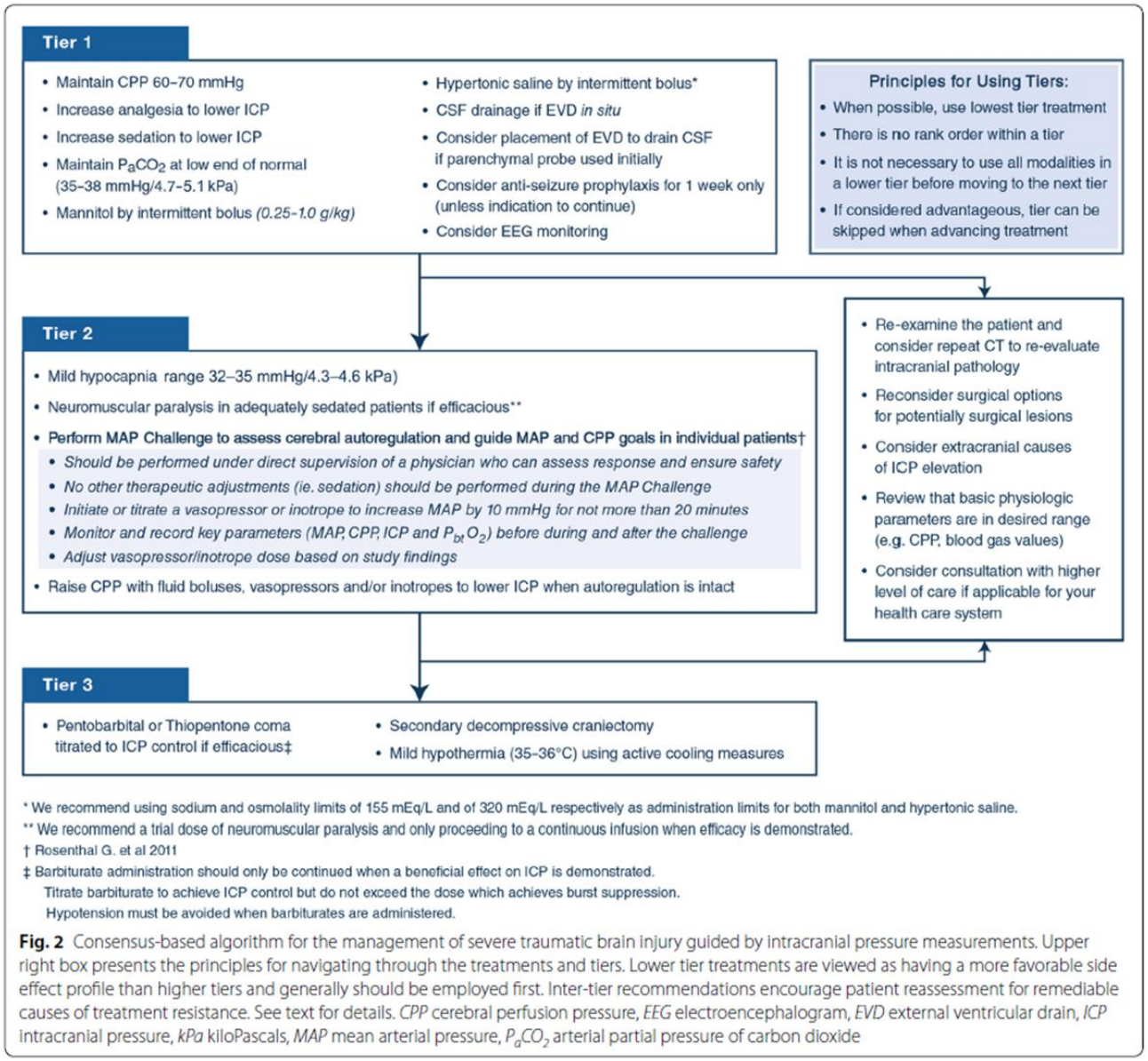
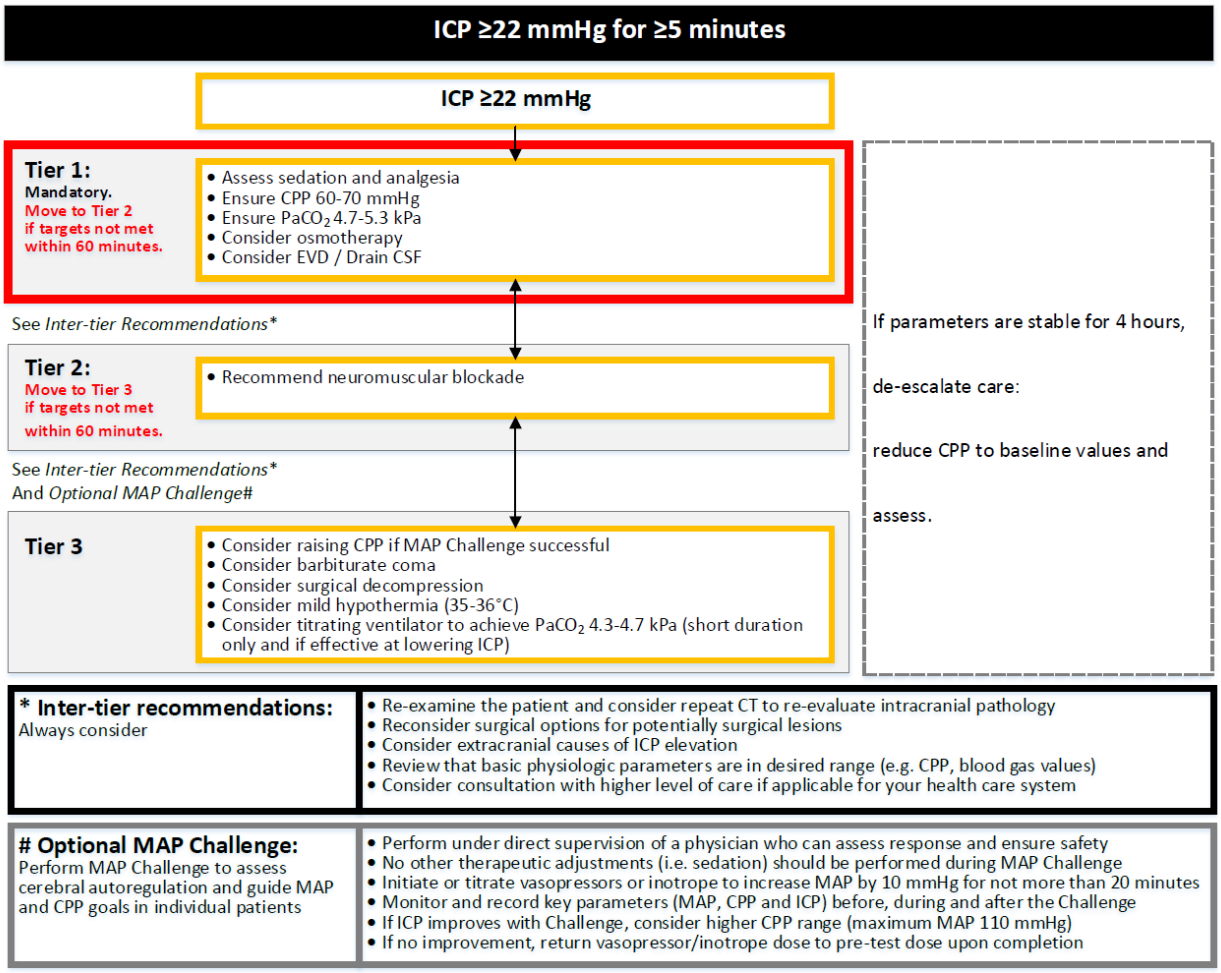


Fig. 2 Consensus-based algorithm for the management of severe traumatic brain injury guided by intracranial pressure measurements. Upper right box presents the principles for navigating through the treatments and tiers. Lower tier treatments are viewed as having a more favorable side effect profile than higher tiers and generally should be employed first. Inter-tier recommendations encourage patient reassessment for remediable causes of treatment resistance. See text for details. *CPP* cerebral perfusion pressure, *EEG* electroencephalogram, *EVD* external ventricular drain, *ICP* intracranial pressure, *kPa* kiloPascals, *MAP* mean arterial pressure, P_aCO_2 arterial partial pressure of carbon dioxide

The ICP-guided management of severe TBI at RSCH is closely based on this tiered approach, and is detailed in the flowchart below:



MANAGEMENT OF SEVERE TBI GUIDED BY INTRACRANIAL PRESSURE AND BRAIN TISSUE OXYGEN

SIBICC also strongly supported brain tissue oxygen ($P_{bt}O_2$) monitoring as the second parameter to be used, where brain multimodality monitoring was considered. A combination of the two parameters creates a matrix of four potential clinical conditions, as illustrated below [45]:

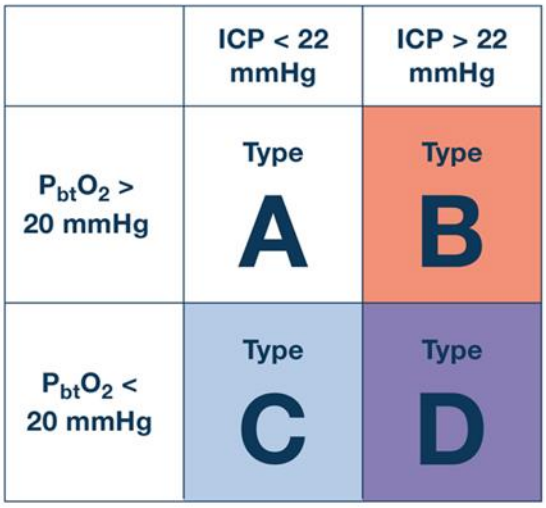


Fig. 2 This matrix provides the schema for the 4 clinical conditions encountered in patients with both ICP and brain oxygen monitors in situ. Type A reflects normal values for both monitors and does not require treatment. Type B involves ICP elevation but normal brain oxygen values; we propose a distinct treatment algorithm for such patients than in those with ICP elevation and unknown $P_{bt}O_2$ values. Type C patients have hypoxic brains but normal ICP and Type D patients have both brain hypoxia and ICP elevation. An ICP of 22 mmHg discriminates normal (lower) and abnormal (higher) values while $P_{bt}O_2$ values of 20 mmHg discriminates normal (higher) and abnormal (lower) values. *ICP* intracranial pressure, $P_{bt}O_2$ partial pressure of brain oxygen

Although there is no level I evidence that supports the use of brain tissue oxygen monitoring, the phase II BOOST2 trial demonstrated that a protocol guided by ICP and $P_{bt}O_2$ could reduce brain hypoxia [46]. These results informed the BOOST3 trial, currently underway in the US, a phase III randomised controlled trial. In parallel, the international BONANZA trial is studying the same question, and RSCH will shortly begin recruiting participants to this investigation. All adult (> 18 years) severe TBI patients should be screened for inclusion. The ICP and $P_{bt}O_2$ -guided protocol is detailed below:

PbtO2 <20 mmHg and / or ICP ≥22 mmHg for ≥5 minutes
PbtO2 ≥20 mmHg, ICP ≥22 mmHg
PbtO2 <20 mmHg, ICP <22 mmHg
PbtO2 <20 mmHg, ICP ≥ 22 mmHg
Tier 1:
Mandatory.
Move to Tier 2 if targets not met within 60 minutes.
• Assess sedation and analgesia
• Ensure CPP 60-70 mmHg
• Ensure PaCO2 4.7-5.3 kPa
• Consider osmotherapy
• Consider EVD / Drain CSF
• Assess sedation and Analgesia
• Ensure CPP 60-70mmHg
• Ensure PaCO2 4.7-6 kPa
• Ensure PaO2 13.3-16 kPa
• Assess sedation and analgesia
• Ensure CPP 60-70 mmHg
• Ensure PaCO2 4.7-6 kPa
• Ensure PaO2 13.3-16 kPa
• Consider osmotherapy
• Consider EVD / Drain CSF
See Inter-tier Recommendations*
Tier 2:
At least one.
Move to Tier 3 if targets not met within 60 minutes.
• Recommend neuromuscular blockade
• Consider titrating ventilator to achieve PaCO2 4.3-4.7 kPa
• Increase CPP to 70-80 mmHg
• Increase PaO2 to 16.0-20 kPa
• Recommend neuromuscular blockade
• Reduce ICP to <22 mmHg
• Transfusion of RBCs to Hb ≥90 g/L
• Consider titrating ventilator to PaCO2 6-6.7 kPa (stop if ICP ≥22 mmHg)
• Increase CPP to 70-80 mmHg
• Increase PaO2 to 16.0-20 kPa
• Recommend neuromuscular blockade
• Transfusion of RBCs to Hb ≥90 g/L
See Inter-tier Recommendations*
And Optional MAP Challenge#
Tier 3
• Consider raising CPP if MAP Challenge successful
• Consider barbiturate coma
• Consider surgical decompression
• Consider mild hypothermia (35-36°C)
• Consider raising CPP if MAP Challenge successful
• Consider titrating ventilator to PaCO2 6-6.7 kPa (stop if ICP ≥25 mmHg)
• Consider PaO2 >20 kPa
• Consider raising CPP if MAP Challenge successful
• Consider barbiturate coma
• Consider surgical decompression
• Consider PaO2 >20 kPa
If parameters are stable for 4 hours, de-escalate care:
First reduce FiO2 and CPP to baseline values and assess.
If ICP remains elevated, accept ICP <25 mmHg if PbtO2 ≥20 mmHg
* Inter-tier recommendations:
Always consider
• Re-examine the patient and consider repeat CT to re-evaluate intracranial pathology
• Reconsider surgical options for potentially surgical lesions
• Consider extracranial causes of ICP elevation
• Review that basic physiologic parameters are in desired range (e.g. CPP, blood gas values)
• Consider consultation with higher level of care if applicable for your health care system
Optional MAP Challenge:
Perform MAP Challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients
• Perform under direct supervision of a physician who can assess response and ensure safety
• No other therapeutic adjustments (i.e. sedation) should be performed during MAP Challenge
• Initiate or titrate vasopressors or inotrope to increase MAP by 10 mmHg for not more than 20 minutes
• Monitor and record key parameters (MAP, CPP, ICP and PbtO2) before, during and after the challenge
• If ICP improves with Challenge, consider higher CPP range (maximum MAP 110 mmHg)
• If no improvement, return vasopressor/inotrope dose to pre-test dose upon completion

FURTHER INFORMATION

Ventilation

Recommendation	Justification
Initial ventilation	Ventilate using a volume-controlled mode (e.g. SIMV) initially. Use ARDS net ventilation guidelines IBW Vt 6ml/kg. Consider a pressure-controlled mode once the risk of secondary brain injury from fluctuating $PaCO_2$ is reduced
Ensure that Vt and minute volume are constant	Variations in ventilation will result in $PaCO_2$ fluctuations and episodic rises in ICP. The fluctuations can have a significant detrimental effect on cerebral blood flow. Set alarm limits for minimum volume close to desired target (± 0.5 L), to detect varying minute volume due to changes in respiratory compliance. When adjusting ventilation only small changes should be made, and time allowed for these to take effect. Measure blood gases 15-20mins after all changes in ventilator settings and record response to change
Monitor blood gases	Target $PaO_2 \geq 13$ kPa (A lower $PaO_2 \geq 11$kPa may be acceptable in patients who have a previous history of lung disease)
	Initially target $PaCO_2$ 4.7 – 5.3 kPa. Mild hyperventilation to $PaCO_2$ of 4.3-4.6 kPa is a tier 2/3 treatment, depending on whether PbtO2 monitoring is or is not present. Avoid reducing $PaCO_2 < 4.3$ kPa if $PbtO_2$ is not measured, except in critical situations. Avoid routinely $PaCO_2 < 4.0$ kPa; this may be needed in an emergency to "buy time" (e.g. prior to theatre or CT scan), but in these instances the patient must be on 100% O_2. **The decision to reduce $PaCO_2$ to < 4.0 kPa must be discussed with a consultant**
	On transfer to CT scan use $ETCO_2$ and ICP blocks from patient's bedside monitor in transport monitor. Stabilise $PaCO_2$ and check correlation of $PaCO_2$ with $ETCO_2$ prior to moving patient. Respiratory failure is common after traumatic brain injury and occurs secondary to direct pulmonary injury, aspiration, or neurogenic pulmonary oedema, but may also occur as a complication of induced arterial hypertension used to maintain CPP.
	Neurogenic pulmonary oedema occurs in the absence of underlying heart and lung dysfunction and may occur within minutes to hours of the injury or it may have a delayed onset. It is believed to be caused by a massive sympathetic discharge, with resultant systemic and pulmonary hypertension. This leads to increased pulmonary capillary pressure and oedema. A centrally-induced increase in capillary permeability may also be present. Symptoms usually resolve in 24 – 72 hours. Treatment is supportive and should focus on maintaining pulmonary function while preventing increases in ICP. Therefore activities associated with patients with diagnosed pulmonary oedema, such as frequent suctioning and turning, should be done with caution.

Cerebral Perfusion

Recommendation	Justification
Autoregulation	An increased CPP can lower ICP if autoregulation is intact. This is due to vasoconstriction reducing cerebral blood volume. If autoregulation is disrupted, an increased CPP may worsen ICP by increasing cerebral blood volume. A MAP challenge (details in flowchart above) to assess autoregulation should therefore be performed only by a practitioner capable of interpreting the results. If MAP challenge successful, benefits should be weighed against risks of pharmacological MAP augmentation [44]

Sedation and Analgesia

Recommendation	Justification
Initial choice Propofol and Fentanyl	• Propofol 2-4mg/kg/hour - maximum 400 mg/hr (20mL/hr of propofol 2%) • Monitor for signs of Propofol Infusion Syndrome – see below • Propofol is contraindicated for the sedation of ventilated children (aged 16 years and below) receiving intensive care. Propofol should only be given in the short term and for no longer than 24 hours in this age group. Caution should be used when administering propofol to patients who are 17-18 years of age • Fentanyl 1-6 micrograms/kg/hr -maximum 600 micrograms/ hour (12ml/hour of fentanyl 50mcg/ml) • Prior to increasing fentanyl dose consider using a test bolus of 25mcg (with caution) to see the impact on ICP. If test bolus has no impact then consider appropriateness of increasing dose
Bolus	A propofol bolus of 10-20mg (0.5 – 1mL of propofol 2%) can be considered to allow nursing, medical or physiotherapy intervention if required, **but be aware of the potential hypotensive effect**. Total doses of propofol should be reviewed each shift, as frequent bolus doses can increase risk of propofol infusion syndrome
Beware opiate withdrawal	Ensure adequate analgesia and if necessary, increase midazolam (within prescribed dosage range) rather than opiates to manage intracranial pressure. Tachyphylaxis is extremely common with morphine infusion, leading to a possible need for dose escalation and a prolonged period of "withdrawal" when therapy is discontinued. Tachyphylaxis and withdrawal symptoms may also occur after prolonged use of fentanyl [2].
Use scoring systems	Use sedation scoring, and in the acute phase aim for no eye opening, no limb movements, no spontaneous cough or on suctioning and no respiratory effort (i.e. RASS -5) Maintain BIS score between 20-30 initially, depending on ICP
Consider NMBs	Neuromuscular blockade is a Tier 2 intervention. If a trial dose is effective in lowering ICP an infusion can be considered but this should be reviewed on a regular basis and discontinued at the earliest opportunity. Further information below

Propofol Infusion Syndrome

EARLY MARKERS	COMMON CLINICAL FEATURES	PREDISPOSING FACTORS
Unexplained metabolic acidosis	Metabolic acidosis	Young age
Elevated serum lactate, creatinine kinase and myoglobin or hyperlipidaemia / triglycerides	Cardiac arrhythmias, particularly bradycardia	Severe head injury
	Early sign is RBBB with convex-curved ST elevation in the right precordial leads. Brugada-like changes. Cases of LBBB have also been seen	Severe critical illness of central nervous system or respiratory origin
	Hyperkalaemia	High dose exogenous catecholamine or steroid administration
	Rhabdomyolysis	Inadequate carbohydrate intake
	Hyperlipideamia	Subclinical mitochondrial disease
	Hepatomegaly	Sepsis
	Renal Failure	
	Rapidly progressive cardiac failure which may be unresponsive to inotropes	

Send serum for daily triglyceride levels and CK if patient on high doses of propofol or on infusion for longer than 48 hours. Lactate should also be monitored on a regular basis.
CK values associated with rhabdomyolysis are very high

Neuromuscular Blockade

Recommendation	Justification
Should be considered if ICP remains high despite use of Tier 1 measures	• A BIS monitor must be in place to ensure patients have adequate analgesia and sedation prior to administration of neuromuscular blockade • Neuromuscular blockade masks neurological changes and potential seizure activity. It is also associated with a longer intensive care unit stay due to an increased incidence of pneumonia. Therefore the appropriateness of its use should be reviewed on a daily basis • Using cisatracurium 150mg/30mL (5mg/mL or 5000 micrograms/mL) as per National Injectable Medicines Guide Monograph. Administer an initial bolus dose of cisatracurium 150 micrograms/kg (calculate using patient's ideal body weight if they are obese) followed by a continuous infusion of 180 micrograms/kg/hr, titrated to achieve 1-2 twitches using the train of four test • Usual dose range 30 – 600 micrograms/kg/hour. A peripheral nerve stimulator must be used when administering neuromuscular blockade
Peripheral nerve stimulator (PNS)	• The use of a PNS minimises the complications of prolonged paralysis by monitoring the degree of neuromuscular blockade • • The PNS delivers 4 pulses over 2 seconds (train of four) • After starting the continuous infusion allow time for drug to reach a steady state, at least 0.5 – 1 hour then the train of four test should be performed hourly until the goal of 1-2 twitches is achieved [17] • For details on nerve stimulator use see separate ICU guideline

Table 2 Management of cisatracurium infusion using PNS

0 twitches	Reduce infusion by 20% increments until 1-2 twitches achieved
1 – 2 twitches	Maintain present infusion rate
3 twitches	Reload with 75micrograms/kg and Increase infusion rate by 50%
4 twitches	Reload with 150micrograms/kg and Increase infusion rate by 100%

Other Drugs

Recommendation	Justification
Barbiturates	• High dose barbiturate administration (see separate Thiopental Microguide guidance for full details) may control elevated ICP refractory to maximum standard medical and surgical treatment (level IIb evidence) [2] • It is associated with significant harm and is a Tier 3 intervention. It must only be given following a discussion with a consultant intensivist and consultant neurosurgeon • Haemodynamic stability is essential before and during barbiturate therapy [2]. • Monitor the BIS during barbiturate coma therapy. The bispectral index value and the suppression ratio values have shown to correlate well with the standard EEG based method to titrate barbiturate therapy. • Aim for BIS values of 10-20 and SR values of 60-80%. This has been shown to correspond to 3-5 bursts per minute on EEG • Hypokalaemia has been reported commonly in patients receiving thiopental infusions[15]. The fall in serum K^+ is not due to an increase in urinary excretion of potassium, it is thought to be due to metabolic changes within the brain. Supplement low plasma K^+ cautiously, if ECG changes indicate need
Steroids	• The use of steroids is not recommended for improving outcome or reducing ICP in TBI. In patients with severe TBI, high dose methylprednisolone was associated with increased mortality and is contraindicated (CRASH trial) • Approximately 15%–20% of TBI patients may develop chronic hypopituitarism, which clearly suggests that TBI-induced hypopituitarism is a frequent problem, in contrast with previous assumptions. However, there are no epidemiological studies showing the burden of the disease in the population. Post-traumatic hypopituitarism is generally characterized by isolated anterior pituitary hormone deficiency rather than multiple hormone deficiencies, and growth hormone deficiency seems to be the most common disorder • Current evidence implies that insufficiency in the hypothalamic-pituitary-adrenal axis during the acute phase after head trauma is associated with a worse neurological outcome, increased need for vasoactive drug therapy due to hemodynamic instability, relative or absolute hypoglycaemia, hyponatraemia, and rapidly progressive hypotension, all of which may increase the risk of morbidity and mortality • Interpretation of endocrine tests in acutely ill patients is challenging and may lead to overdiagnosis. Routine testing of pituitary function or measurement of serum/plasma cortisol levels in acute phase after TBI is **not** recommended [47] • If there is clinical suspicion of cortisol insufficiency (e.g. refractory hypotension, hypoglycaemia, hyponatraemia), empirical treatment with hydrocortisone should begin immediately (e.g. 50mg iv every 6 hours). A random cortisol level should be sampled prior to the first dose [47] • If there is subsequent difficulty in withdrawing glucocorticoid therapy, the patient should be discussed with endocrinology [47] • An overview of pituitary dysfunction in TBI is contained in **Appendix 2** below

Fluid Management

Recommendation	Justification
Intravenous fluids	• Fluid input: 2.5-3.0 litres / 24 hours initial target • Maintain hydration with early enteral feeding • Supplement with IV Sodium Chloride 0.9% or Hartmanns if enteral feeding not established / appropriate • Avoid dextrose/saline as routine maintenance fluid • Avoid hyponatraemia • Treat hypovolaemia with crystalloid / RBC as appropriate • Hb > 80 • Normal or near normal clotting, especially postoperatively • Platelet count > 100 • INR ≤ 1.3 • APPTR ≤ 1.3 •
Renal	• Urine output ~ 0.5-1 ml/kg/hr • Urine specific gravity should be measured if urine output is in excess of 250mls/hr • Diabetes insipidus (DI) should be suspected if urine output > 250mls/hr for more than 3 hrs and specific gravity < 1005. Confirm by measuring plasma and urinary osmolalities and electrolytes • In DI plasma osmolality rises with a marked rise in Na^+ > 150 mmol/l and urine osmolality is very low with low electrolyte concentrations. In later stages diuresis may be appropriate • If confirmed on laboratory results, urine output continuing to rise and plasma Na^+ >155 mmol/l, consider cautious use of DDAVP. **This should be discussed with consultant intensivist and neurosurgeon - rapid reductions in sodium can be fatal in this cohort** • If Na^+ < 135mmols/l consider hypovolaemic hyponatraemia (Cerebral Salt Wasting) or Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

Surgical Interventions

Recommendation	Justification
Decompressive craniectomy	• Decompressive craniectomy, the surgical removal of a portion of the skull (unilateral or bifrontal), may be performed to relieve elevated intracranial pressure. The role of this therapy remains controversial • In the first large randomised trial, in 2011 the DECRA trial showed no benefit from bifrontal surgical decompressive craniectomy to reduce intracranial pressure, although the definition of refractory intracranial pressure elevation of >20 mmHg for 15 minutes within a one hour period was called into question • The subsequent RESCUEicp trial demonstrated better intracranial pressure control in the surgical group and lower mortality, but higher rates of vegetative state and severe disability in survivors • In view of these results, the decision to perform this procedure most be a consultant decision, which has involved the patient's surrogates, focusing on the patient's previously stated wishes and personal values • Emphasis that lifesaving procedures may not lead to a return to normal function is important, given the larger proportion of survivors with poor outcome in the surgical group
Cerebrospinal fluid drainage	• An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use (Level III) • Use of CSF drainage to lower ICP in patients with an initial Glasgow Coma Scale (GCS) <6 during the first 12 hours after injury may be considered (Level III) [2]

Brain Oxygen Monitoring

Recommendation	Justification
$P_{bt}O_2$ (Licox)	The Licox brain tissue oxygen monitoring system provides direct, real-time measurement of the partial pressure of oxygen in brain tissue ($P_{bt}O_2$). It is unknown whether the use of $P_{bt}O_2$ improves outcomes, and this is being assessed by BONANZA (AUS/EUROPE/UK) and BOOST3 (US) trials. Patients recruited to BONANZA may be randomised to ICP and $P_{bt}O_2$-guided management. Its use may also be considered in any severe TBI patients not recruited to BONANZA. A similar device is available from Raumedic (not currently used at RSCH).
Oxygen Challenge Test	FiO_2 is increased to 100% for up to 2 minutes, and $P_{bt}O_2$ recordings monitored (for the next 10 minutes). This should result in an increase in $P_{bt}O_2$ by at least 5 mmHg during this time period. If the first "challenge" fails, the procedure should be repeated. If the $P_{bt}O_2$ does not increase as expected, the following should occur: 1. An arterial blood gas should be performed using a FiO_2 of 100% to ensure adequate systemic oxygenation. If this identifies a significant problem with systemic oxygenation, this should be managed as appropriate by the treating clinician 2. Obtain a CT scan to determine if there is evidence of haemorrhage, contusion expansion or infarction around the tip of the catheter 3. The $P_{bt}O_2$ catheter can be replaced if the second challenge fails or if the CT scan indicates the probe is malpositioned. Ideally, probes should be replaced within 2 hours 4. At completion of the oxygen challenge the inspired FiO_2 should be weaned back to previous baseline For BONANZA control group participants, research staff will perform the oxygen challenge to monitor reliability, but results will not be shared with clinical staff, to maintain blinding. Probes will not be replaced in control patients should the FiO_2 challenge fail. For BONANZA participants, If the monitor readings are accurate and >20 mmHg, a routine post-op CT scan should be obtained within 24-hours of device placement

Temperature Management

Recommendation	Justification
Avoid hyperthermia	• Hyperthermia following TBI may be due to posttraumatic cerebral inflammation (often seen in the first 24 hours), direct hypothalamic damage (known as neurogenic fever or central fever, and not usually seen in the hyper acute phase) or secondary infection • If the patient has an unexplained fever and they have sustained a DAI or frontal injury there should be an increased suspicion of neurogenic fever. Increased temperature in the post injury period is associated with poorer outcomes • For every 1°C rise in body temperature there is a 13% increase in the metabolic rate[25]. It is important to establish the cause of the hyperthermia as they need to be treated differently. The diagnosis of neurogenic fever is currently a diagnosis of exclusion [24] • Hypothermia was thought to reduce intracranial pressure; however it bears risks, including coagulopathy, immunosuppression and arrhythmia • The EUROtherm 3235 trial demonstrated that hypothermia to 32-35 °C worsened clinical outcome and increased mortality compared to standard care alone • The POLAR RCT 49 did not show that prophylactic hypothermia improved outcome but did increase the risk of complications • Initial target is core temperature in normal range. If no PiCCO in situ, core temperature should be measured with an oropharyngeal or nasopharyngeal probe (a nasopharyngeal probe should not be used in patients with a known or suspected base of skull fracture) • Active cooling measures are frequently required to achieve normothermia (see below) • Mild hypothermia (35-36 °C) is a Tier 3 therapy
Infection	Screen for infection and treat if indicated.
Active cooling	• Discuss appropriate method of cooling with medical team • To maintain normothermia an antipyretic e.g. paracetamol ± patient exposure may be sufficient • Pyrexia should be aggressively managed – if passive measures ineffective then active cooling should be instigated • Surface cooling pads (Arctic Sun) are first-line at RSCH • Shivering should be avoided as it increases oxygen consumption through aerobic muscle activity [25] • A tiered approach to shivering management is appropriate: passive counter-warming (mittens/hat/socks), active counter-warming (warming blanket), increased sedation and finally neuromuscular blockade • If inducing hypothermia, insulin resistance may develop. Monitor Blood glucose 1-2hourly and commence variable rate insulin infusion if indicated.

Sympathetic Storm

Recommendation	Justification
Sympathetic Storm	• Dysautonomia, sometimes referred to as sympathetic storming, is an exaggerated stress response that occurs in 15-33% of patients with severe traumatic brain injury. It can occur within the first 24 hours or up to 2 weeks after injury • Signs and symptoms include posturing, dystonia, hypertension, tachycardia (> 130), pupillary dilatation, diaphoresis, hyperthermia (> 38.5°C), and tachypneoa (>20 bpm) • The precise mechanism for the increase in activity of the sympathetic nervous system is unknown but is believed to be a stage of recovery. It is more common in patients with diffuse axonal injury. • In sympathetic storming the feedback mechanism, when the parasympathetic nervous system dampens down the effect of increased activity, does not occur • Episodes can be unprovoked with the signs and symptoms occurring within seconds and can vary from episode to episode. Triggers that may precede an episode include suctioning, repositioning, environmental sensory stimulation (alarms etc.) or fever • It is important to identify the triggers so measures can be taken to reduce the length of the episode, lessen its intensity or even abort the episode • Careful assessment is needed as the differential diagnoses include: an expanding lesion or oedema, seizures, deep vein thrombosis, pulmonary embolus, malignant hyperthermia, central fever, and drug or alcohol withdrawal • Medical management focuses on treating the signs and symptoms in order to reduce the potential adverse effects of prolonged activity of the sympathetic nervous system
Medications	• I.V. agents e.g. fentanyl, morphine, midazolam or clonidine can be used for rapid control • Consider enteral propranolol for maintenance therapy • Propranolol may improve neurological outcomes [48] • Avoid beta blockers if hypotensive or bradycardic

Seizure Prophylaxis and Management

Recommendation	Justification
Avoid seizures	Seizure activity in the early post traumatic period following head injury may cause secondary brain damage as a result of increased metabolic demands, raised intracranial pressure and excess neurotransmitter release.
Drugs	• There is evidence that prophylactic anti-epileptic drugs reduce early seizures, but this is not supported by a reduction in late seizures • Consensus guidelines suggest the prophylactic use of AEDS for 1 week is appropriate [44] • The MaST-PROPHYLAXIS trial (currently recruiting) may demonstrate if levetiracetam or phenytoin is more effective for prophylaxis • Levetiracetam (500mg IV twice daily) is current first-line for prophylaxis at RSCH. It should be given enterally once patient is absorbing • Phenytoin second-line for prophylaxis if levetiracetam contra-indicated • Alternatives: carbamazepine, sodium valproate (caution as associated with higher death rates) • Consult separate AED guidelines (see Microguide) for dosing

Weaning Treatment

Recommendation	Justification
Sedation holidays	• Obtaining accurate neurological examination must be balanced against potential harm of pausing sedation • The risk of secondary brain injury must have reduced to an acceptable level before a reduction in sedation in considered • Consensus on timing of sedation holidays is difficult to achieve • SIBICC guidelines have generated “heat maps” to assist decision-making **(Appendix 3)** [44]
ICP monitor removal	• As with sedation holidays, the key factor guiding removal of advanced neuromonitoring is the risk window for secondary injury • Consensus on timings of ICP monitor removal is also difficult to achieve [44] • Heat maps from SIBICC guidelines are available to guide decision-making **(Appendix 4)** • 72 hours of controlled ICP is almost universally accepted as a suitable time-point for consideration of monitor removal • However, the therapeutic intensity prior to ICP control and GCS motor scores are strong influencers on decisions over removal timings • Brain oxygen monitors, where used, would usually be removed at the same time as ICP probes

General ICU Care

Recommendation	Justification
Positioning	• Avoid venous congestion • Nurse 30° head up unless contraindicated (caution: ensure this does not compromise CPP). If spine not cleared tilt bed 30° head up. • Head and neck in neutral alignment with no neck flexion. • Avoid excessive hip flexion > 90 degrees. • Turn and reposition patient with caution. Use a minimum of 3 staff to maintain head and neck in neutral alignment when turning or repositioning. • Jugular vein compression can be seen as an increase in mean ICP and an increase in ICP waveform amplitude, mainly P2 and P3 • If patient unconscious and cervical spine needs clearance follow unit protocol • If cervical spine fracture confirmed, management is dictated by the precise nature of the injury and its stability – position according to instructions from neurosurgical team or orthopaedic surgeons.
VTE Prophylaxis	• Commence mechanical thromboprophylaxis (compression stockings and intermittent pneumatic compression) in all TBI patients, unless otherwise contraindicated • Consider chemical thromboprophylaxis in patients at low risk of bleeding from 24 hours after admission, if CT appearances stable [51] • Consider chemical thromboprophylaxis from 72 hours in patients at moderate risk of bleeding, if CT appearances stable [51] • Use caution in starting chemical thromboprophylaxis in patients at high risk of bleeding, including those where neurosurgical intervention performed within 72 hours [53] • Consider IVC filter if bleeding risk precludes chemical prophylaxis for a prolonged period; however early prophylactic use of IVC filters is not associated with a reduced risk of symptomatic PE or death [51] • LMWH may be superior to UFH and its use should be guided by anti-Xa assays. UFH should be used as an alternative where indicated by Trust guidelines [51,55]
Transfer	Minimum monitoring when transferring patient (e.g. to CT scan) • ECG • Oxygen saturation • $ETCO_2$ • Arterial BP • ICP The blocks that are in the patient's bedside monitor should be used for the transfer. As soon as the patient is on the portable ventilator and attached to transport monitor you must stabilise $ETCO_2$ prior to transfer Check ABG
Consider Ophthalmology review	If a patient has facial injuries and there is a suspicion that there may be eye damage or there is obvious eye damage refer urgently to the consultant ophthalmologist.

Nutrition	Severe head injury patients are in a hypermetabolic and hypercatabolic state. Prescribe high dose IV vitamins (Pabrinex ®) on admission if any suspicion of chronic alcohol abuse or chronic malnutrition, followed by regular enteral vitamins. Dose as per UHSx alcohol withdrawal prescribing or refeeding syndrome guidelines. Early enteral feeding - aim to start within 24 hours of admission. Follow unit protocol for establishing enteral nutrition. Patients should be fed to attain full caloric replacement by day 7 post injury[2]. Monitor for refeeding syndrome in at risk patients as per UHSx refeeding syndrome guidelines for adults. Follow guidelines for confirming correct positioning of orogastric or nasogastric tubes - see trust intranet / MicroGuide
Control blood glucose	Monitor blood glucose with 2-4 hourly measurements on admission. If blood glucose stable within 4.5-10 mmol/l then reduce frequency. Monitor blood glucose at least once a day. Commence variable rate insulin infusion if indicated. It is recommended that patients receiving intravenous insulin receive a glucose calorie source and that blood glucose values are monitored every 1-2 hours until glucose values and insulin infusion rates are stable and then every 4 hours thereafter. Low glucose levels obtained with point of care testing of capillary blood should be interpreted with caution; as such measurements may overestimate arterial blood or plasma glucose values [20].

GLOSSARY

ABG	Arterial blood gas
AED	Anti-epilepsy drug
ALI	Acute lung injury
APTT	Activated partial thromboplastin time
ARDS	Acute respiratory distress syndrome
BIPAP	Bi-phasic inspiratory pressure
BIS	Bispectral Index
BNF	British national formulary
CK	Creatine kinase
CPP	Cerebral perfusion pressure
CSF	Cerebrospinal fluid
CSW	Cerebral salt wasting
DI	Diabetes insipidus
ECG	Electrocardiogram
EEG	Electroencephalogram
$ETCO_2$	End-tidal CO_2
ET tube	Endo-tracheal tube
EVD	External ventricular drain
FiO_2	Fraction of inspired oxygen
GCS	Glasgow coma scale
Hb	Haemoglobin
HOB	Head of the bed
ICP	Intracranial pressure
IVC	Inferior vena cava
LMWH	Low molecular weight heparin
MAP	Mean arterial blood pressure
NMB	Neuromuscular junction blocking muscle relaxants
PaO_2	Partial pressure of arterial oxygen
$PaCO_2$	Partial pressure of arterial carbon dioxide
PbtO2	Partial pressure of brain oxygen
PBW	Predicted ideal body weight
PNS	Peripheral nerve stimulator
PT	Prothrombin time
RASS	Richmond agitation and sedation score
RSCH	Royal Sussex County Hospital
SAH	Sub-arachnoid haemorrhage
SIADH	Syndrome of inappropriate antidiuretic hormone
SIMV	Synchronised intermittent mandatory ventilation
SMS	Supramaximal stimulation
SpO2	Peripheral oxygen saturation
TBI	Traumatic Brain Injury
UFH	Unfractionated heparin
UHSx	University Hospitals Sussex NHS Foundation Trust
VTE	Venous thromboembolism

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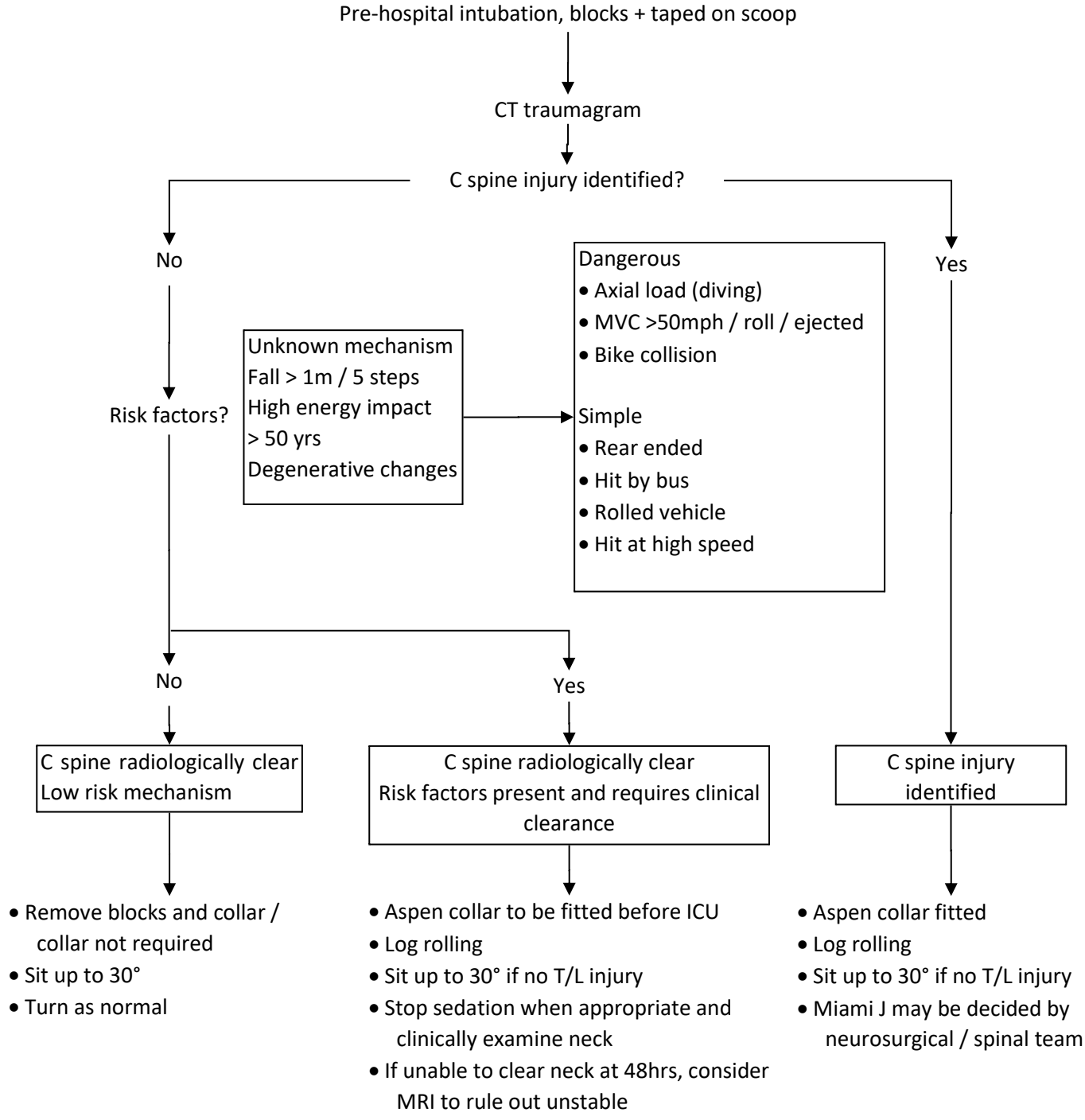
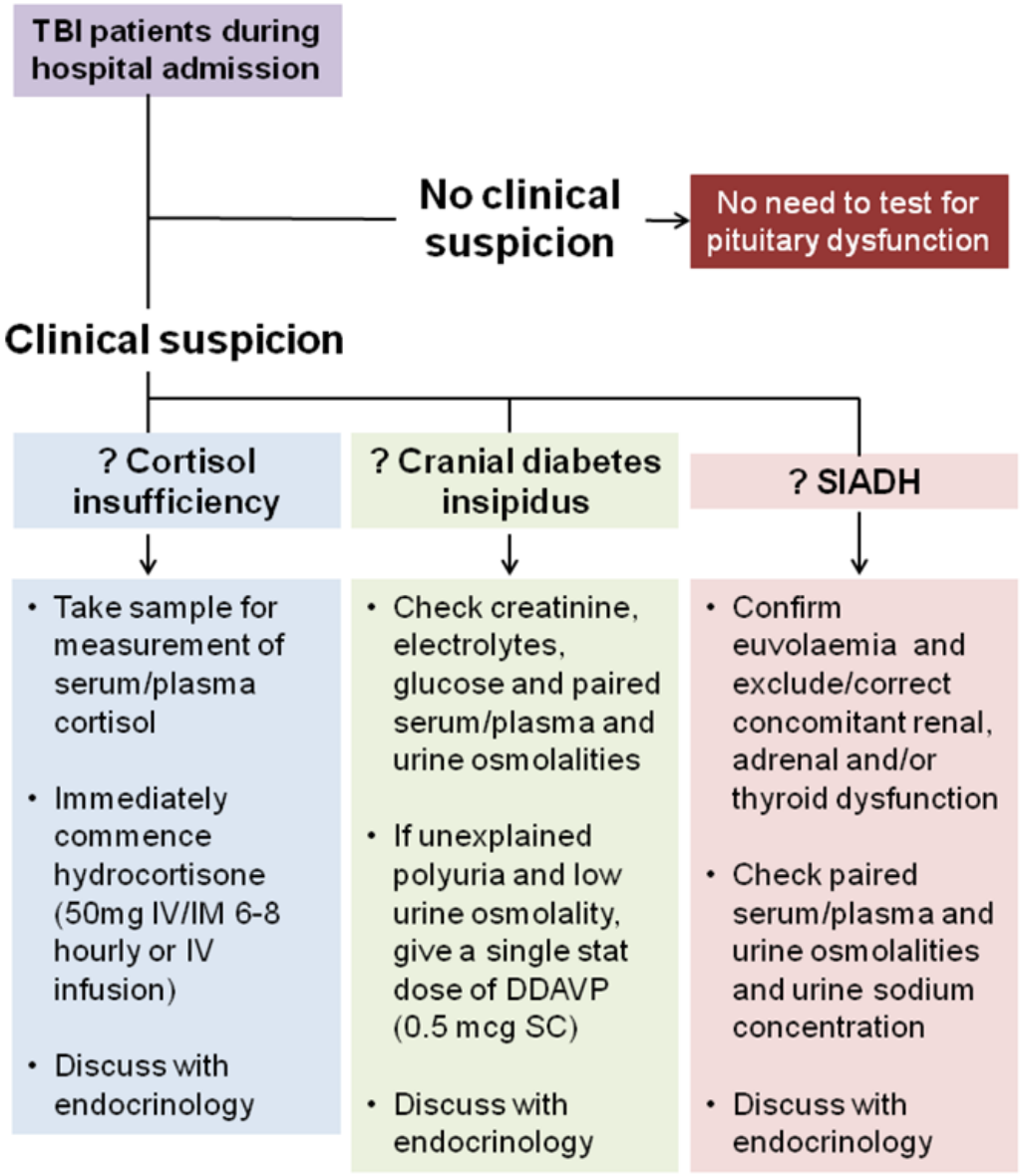
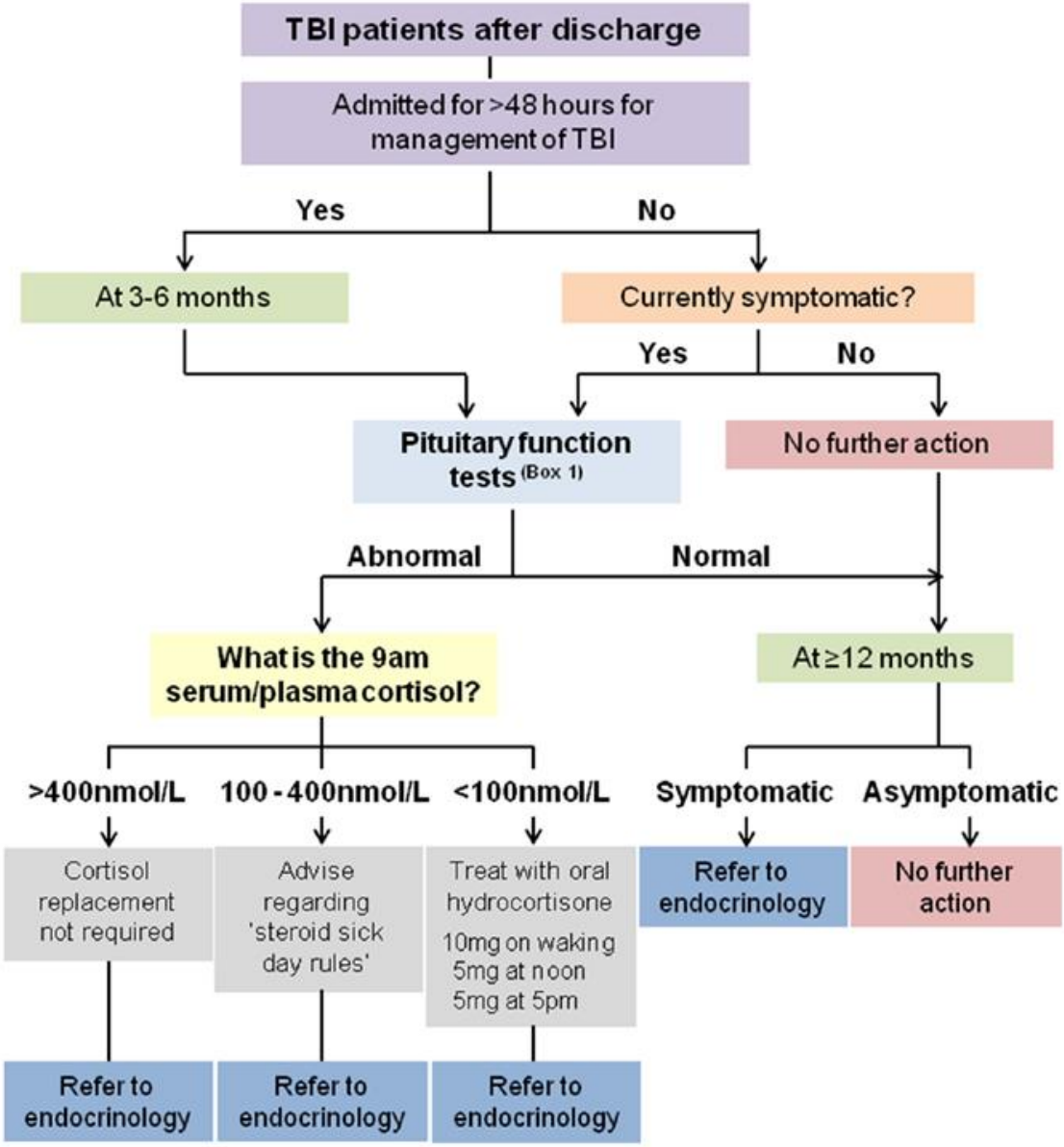
APPENDIX 1 CERVICAL SPINE IMMOBILISATION FLOWCHART



APPENDIX 2 PITUITARY DYSFUNCTION FOLLOWING TBI [47]




TBI patients after discharge
Admitted for >48 hours for management of TBI
Yes
No
At 3-6 months
Currently symptomatic?
Yes
No
Pituitary function tests (Box 1)
No further action
Abnormal
Normal
What is the 9am serum/plasma cortisol?
At ≥12 months
>400nmol/L
100 - 400nmol/L
<100nmol/L
Symptomatic
Asymptomatic
Cortisol replacement not required
Advise regarding 'steroid sick day rules'
Treat with oral hydrocortisone
10mg on waking
5mg at noon
5mg at 5pm
Refer to endocrinology
No further action
Refer to endocrinology
Refer to endocrinology
Refer to endocrinology

Box 1 Screening for pituitary dysfunction post-traumatic brain injury (post-TBI)

The following tests (serum/plasma sample)* should be performed at 09:00:

Men

Urea, creatinine and electrolytes
Free T4 and thyroid-stimulating hormone (TSH)
Cortisol
Luteinising hormone (LH), follicle-stimulating hormone (FSH), testosterone, sex hormone-binding globulin, albumin

Women

Urea, creatinine and electrolytes
Free T4 and TSH
Cortisol† and

In premenopausal women

If menstrual cycle has become abnormal post-TBI, check LH, FSH, oestradiol

In postmenopausal women

FSH

*If in doubt, check local laboratory requirements.
†If the patient is taking an oestrogen-containing oral contraceptive pill or oral hormone replacement therapy, we recommend seeking endocrine advice prior to assessing hypothalamic–pituitary–adrenal axis function.

APPENDIX 3 – HEAT MAP TO GUIDE SEDATION HOLIDAYS [4]

Patients with NO intracranial hypertension since monitor insertion

NO INTRACRANIAL HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Patients with intracranial hypertension requiring Tier 1 treatment - now controlled

MILD INTRACRANIAL HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Patients with intracranial hypertension requiring Tier 2-3 treatment - now controlled

MODERATE-SEVERE HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Fig. 5 Consensus views on the safety of performing a sedation holiday aimed at obtaining an accurate neurological examination in patients whose ICP is controlled under different degrees of active management. The heatmap represents a summary analysis of the likelihood of each panelist to halt sedation to get an optimized neurological exam under differing conditions of stable pupillary status, GCS [20] motor score, modified CT classification (see "Methods"), duration of acceptable ICP with ongoing treatment, and degree of treatment previously required for any intracranial hypertension (none, Tier 1, or Tier 2 or 3). Green, yellow, and red indicate "safe to proceed", "consider proceeding with caution" and "do not proceed", respectively, with transitional shades reflecting intermediate trends. To use, choose the heatmap representing the ICP treatment history, then the appropriate status cell reflecting categorization of the patient in terms of the variables presented. The color in the relevant cell reflects the tendency of the CWG to perform a sedation holiday in that circumstance. It is up to the treating physician to consider the value of that tendency in making the final decision. *AP* abnormal pupils, *CT* computed tomography; *DI* diffuse injury as defined in the Marshall CT Head Score, *GCS* Glasgow Coma Scale, *EML* evacuated mass lesion as defined in the Marshall CT Head Score, *ICP* intracranial pressure, *NP* normal pupils

APPENDIX 4 – HEAT MAP TO GUIDE MONITOR REMOVAL [44]

Patients with NO intracranial hypertension since monitor insertion

NO INTRACRANIAL HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Patients with intracranial hypertension requiring Tier 1 treatment - now controlled

MILD INTRACRANIAL HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Patients with intracranial hypertension requiring Tier 2-3 treatment - now controlled

MODERATE-SEVERE HYPERTENSION		GCS_M 6		GCS_M 5		GCS_M 4		GCS_M 1-3	
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 24 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 48 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								
		NP	AP	NP	AP	NP	AP	NP	AP
ICP "acceptable" for > 72 hours	DI 1-2								
	EML / DI 1-2								
	DI 3								
	EML / DI III								

Fig. 4 Consensus views on the safety of intracranial pressure monitor removal in patients with acceptable ICP (no longer requiring active ICP management). The heatmap represents a summary analysis of the likelihood of each CWG member to remove the ICP monitor under differing conditions of stable pupillary status, GCS [20] motor score, modified CT classification (see "Methods"), duration of acceptable ICP, and degree of treatment previously required for any intracranial hypertension (none, Tier 1, or Tier 2 or 3). Green, yellow, and red indicate "safe to proceed", "consider proceeding with caution" and "do not proceed", respectively, with transitional shades reflecting intermediate trends. To use, choose the heatmap representing the ICP treatment history, then the appropriate status cell reflecting categorization of the patient in terms of the variables presented. The color in the relevant cell reflects the tendency of the CWG to withdraw the ICP monitor in that circumstance. It is up to the treating physician to consider the value of that tendency in making the final decision. *AP* abnormal pupils, *CT* computed tomography, *DI* diffuse injury as defined in the Marshall CT Head Score, *GCS* Glasgow Coma Scale, *EML* evacuated mass lesion as defined in the Marshall CT Head Score, *ICP* intracranial pressure, *NP* normal pupils