Prevention of Secondary Brain Injury

Final FRCA Teaching UHCW
November 2021

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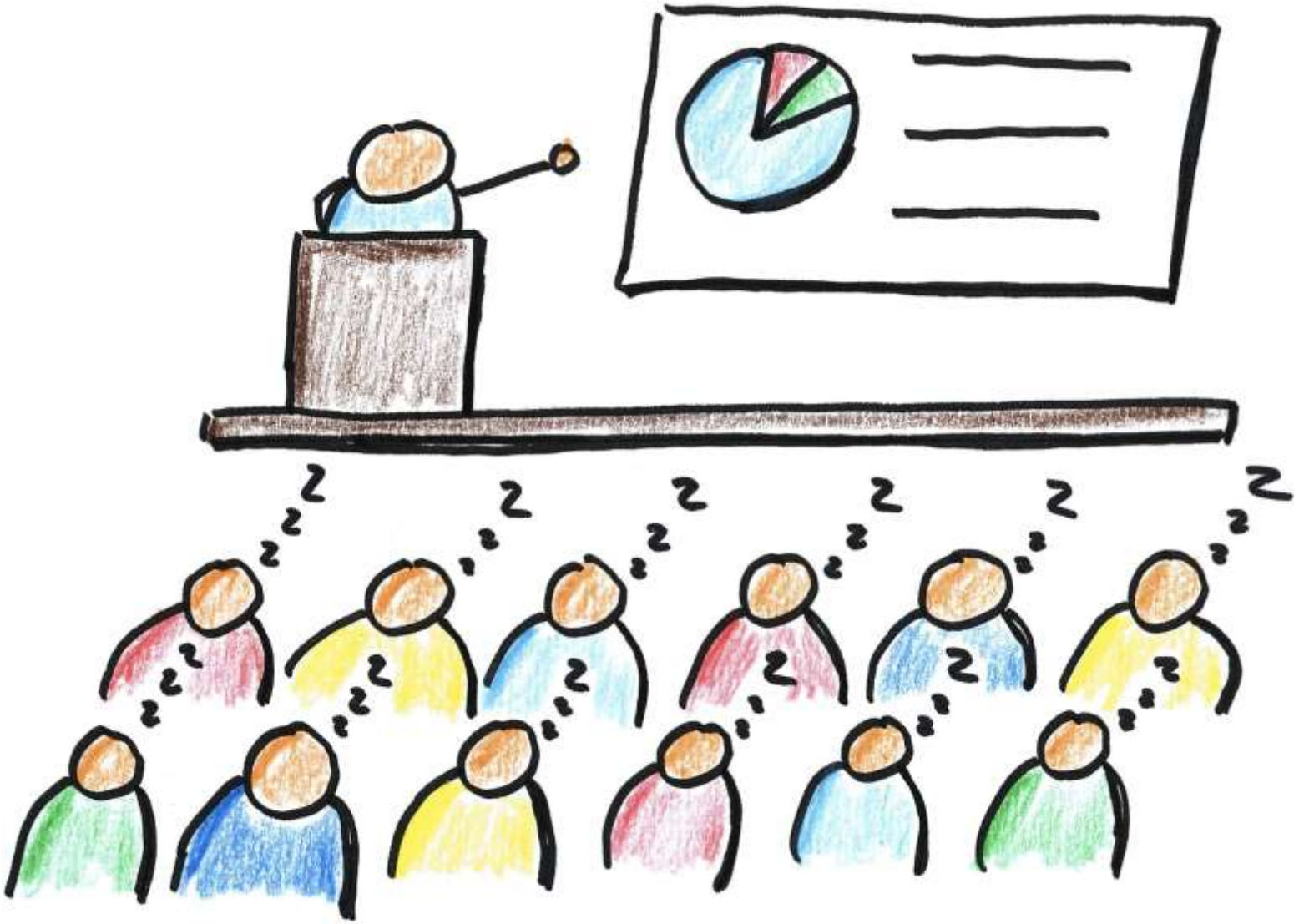
Post-lunch



RCoA Intermediate

- Describes techniques for decreasing the intra-cranial pressure
- Explains the indications for using neurophysiological monitoring [including EEG, evoked potentials and ICP measurement] to benefit patients requiring neurosurgery/neuro-critical care
- Demonstrates understanding of the neurocritical care management of traumatic brain injury [including but not exclusively]:
  - indications for ventilation
  - recognition and management of raised ICP
  - cerebral protection strategies
  - fluid and electrolyte balance in the head injured patient
  - systemic effects of traumatic brain injury
- The principles of management of acute spinal cord injury
- Describes the requirements for safe transfer of patients with brain injury

RCoA Advanced

- Discusses the indications and risks of therapies and monitors available to achieve optimal intracranial pressure and cerebral perfusion in both neuroanaesthesia and neuro-critical care
- Shows awareness of current trends in the management of all aspects of neuroanaesthesia and neuro-critical care

RCoA Stage 2 (2021)

- Key capability F:

  Applies physiological & pharmacological principles to reduce the risk of secondary brain injury in patients presenting with a severe head injury.

RCoA Historic SAQs

(2009) A 20 year old is admitted to the Emergency Department having been involved in a road traffic accident. His Glasgow Coma Score (GCS) is 12. He has a closed fracture of his left femur. No other injuries are evident. A CT scan of his head has revealed a right subdural haematoma, and it has been agreed to transfer him to the regional neurosurgical unit 60 miles away.

a) What are your clinical priorities prior to transfer of this patient? (50%)

b) What are the indications for intubation and ventilation prior to transfer following a brain injury of this nature? (30%)

c) Who should accompany this patient for transfer to the neurosurgical unit? (10%)

(2003) A 4 year old child who has been knocked unconscious by a blow from a cricket bat arrives at a paediatric neurosurgical centre. After initial appropriate management a CT scan shows an extradural haematoma. There are no other injuries. Discuss the subsequent management.

(1999) How would you manage the transfer of a patient to a regional neurosurgical unit for evacuation of an extradural haematoma?

(1996) A 40-year old man is admitted with an acute head injury. List the indications for intubation, ventilation and referral to a neurosurgical unit.

RCoA SAQ (2015)

A 54 year-old patient is admitted to the Emergency Department following a traumatic brain injury. A CT scan reveals only cerebral oedema.

a)What is secondary brain injury and when is it likely to occur? (2 marks)

b)Outline the main physiological and cellular changes associated with secondary brain injury. (7 marks)

c)How can secondary brain injury be minimised in this patient? (11 marks)

(8%)

RCoA CRQ (2018 Example)

You are called to the emergency department to assist with the management of a 34-year-old gentleman who has sustained an isolated head injury following a road traffic accident, he requires an urgent CT scan. Upon arrival you find him to be restless, no eye opening to pain, making incomprehensible sounds and extending to pain. His blood pressure is 120/70 mmHg and heart rate 80bpm. He weighs 70kg.

…
b) Why does he need intubation and ventilation? (2 marks)
c) Describe how you would achieve intubation and ventilation. (5 marks)
...
f) Give 6 treatment options available to improve this patient's cerebral perfusion pressure. (6 marks)
g)List 2 intracerebral pathophysiological changes associated with secondary brain injury. (2 marks)

Head Injury

- Commonest cause of death/disability in 1-40 y/o in UK
- UK annually 1.4million attend ED, 200k admitted
- 1/5th Hospital admissions have skull fracture/brain injury
- Classification by GCS:
  - Mild 13-15
  - Moderate 8-12
  - Severe <8-

Primary / Secondary Brain Injury

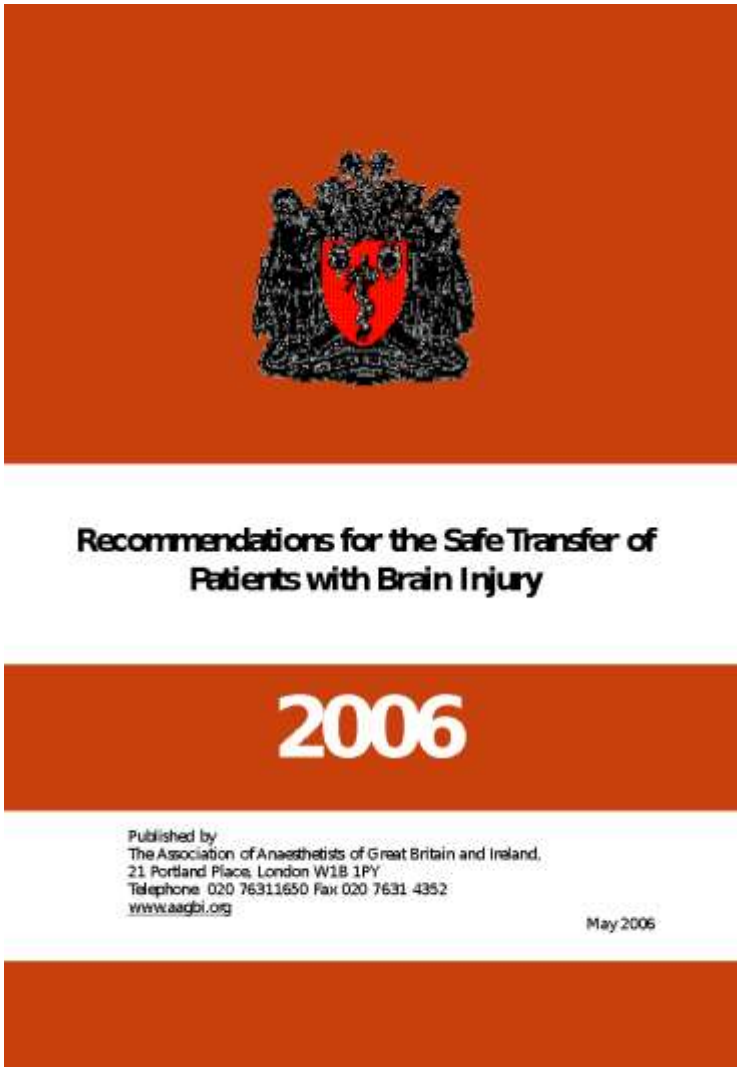
- Primary = Degree of neuronal death/damage at time of impact
- Secondary = CMRO2 dysregulation
  - Reduction in provision of substrates for brain *or* increased requirement for substrates
  - Cell damage from circulating inflammatory mediators/local neuroexcitatory processes
    - Axons
    - Support/BBB

Primary Injury

- Direct axonal loading/shearing/compression
  - Intensity
  - Duration
  - Nature
- Cell wall damage (axons, vessels)

- Oedema
- Axonal injury (“contusion”)
- Haematoma

Stabilisation & Transfer



Recommendations for the Safe Transfer of Patients with Brain Injury

2006

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The Association of Anaesthetists of Great Britain and Ireland,
21 Portland Place, London W1B 1PY
Telephone 020 76311650 Fax 020 7631 4352
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Guidelines

Guidelines for safe transfer of the brain-injured patient: trauma and stroke, 2019

Guidelines from the Association of Anaesthetists and the Neuro Anaesthesia and Critical Care Society

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Summary

The location of care for many brain-injured patients has changed since 2012 following the development of major trauma centres. Advances in management of ischaemic stroke have led to the urgent transfer of many more patients. The basis of care has remained largely unchanged, however, with emphasis on maintaining adequate cerebral perfusion as the key to preventing secondary injury. Organisational aspects and training for transfers are highlighted, and we have included an expanded section on paediatric transfers. We have also provided a table with suggested blood pressure parameters for the common types of brain injury but acknowledge that there is little evidence for many of our recommendations. These guidelines remain a mix of evidence-based and consensus-based statements. We have received assistance from many organisations representing clinicians who care for these patients, and we believe our views represent the best of current thinking and opinion. We encourage departments to review their own practice using our suggestions for audit and quality improvement.

TBI indications for intubation

- GCS </= 8 (inc seizure)
  - Requires transfer to Neuroscience unit, *even if not for surgery*
- Drop GCS by >/= 2 (Motor >/=1)
- Loss laryngeal reflexes/vomiting
- Aggressive/unmanageable
- Ventilatory insufficiency
  - PaO2 <13 with O2, PaCO2 >6 or <4, irregular respiration
- Anticipated airway loss (burns, mandible fracture)

Consider intubation in unstable facial fractures, bleeding in airway, seizures

(note C-spine immobilisation)

Continuous monitoring

- Pupil size/light reaction
- ECG
- Pulse-ox
- IABP
- UOP
- Capnography
- Temperature

Initial Management

- Avoid:
  - Raised ICP (*n.b. treat pain)*
  - Hypotension
  - Hypoxia
  - Hypercapnoea
  - Cardiovascular instability
  - Hyperpyrexia

Pre-transfer

- ABG(s)
- X-rays (ideally trauma series)
- Catheter/UOP
- Blood sugar management
- FBC, U&E
- ?X-match

(*Documentation, equipment/drug preparation, etc)*

Transfer

- Accompanied by suitable anaesthetist/intensivist (trained/competent airway & managing ICP) and assistant
- Other injuries (e.g. bleed, ptx) secured/managed
- Parameters (TBI)
  - MAP >90 mmHg

  (*intravascularly resuscitated; do not squeeze a dry patient)*
  - PaCO2 4.5 – 5.0 kPa

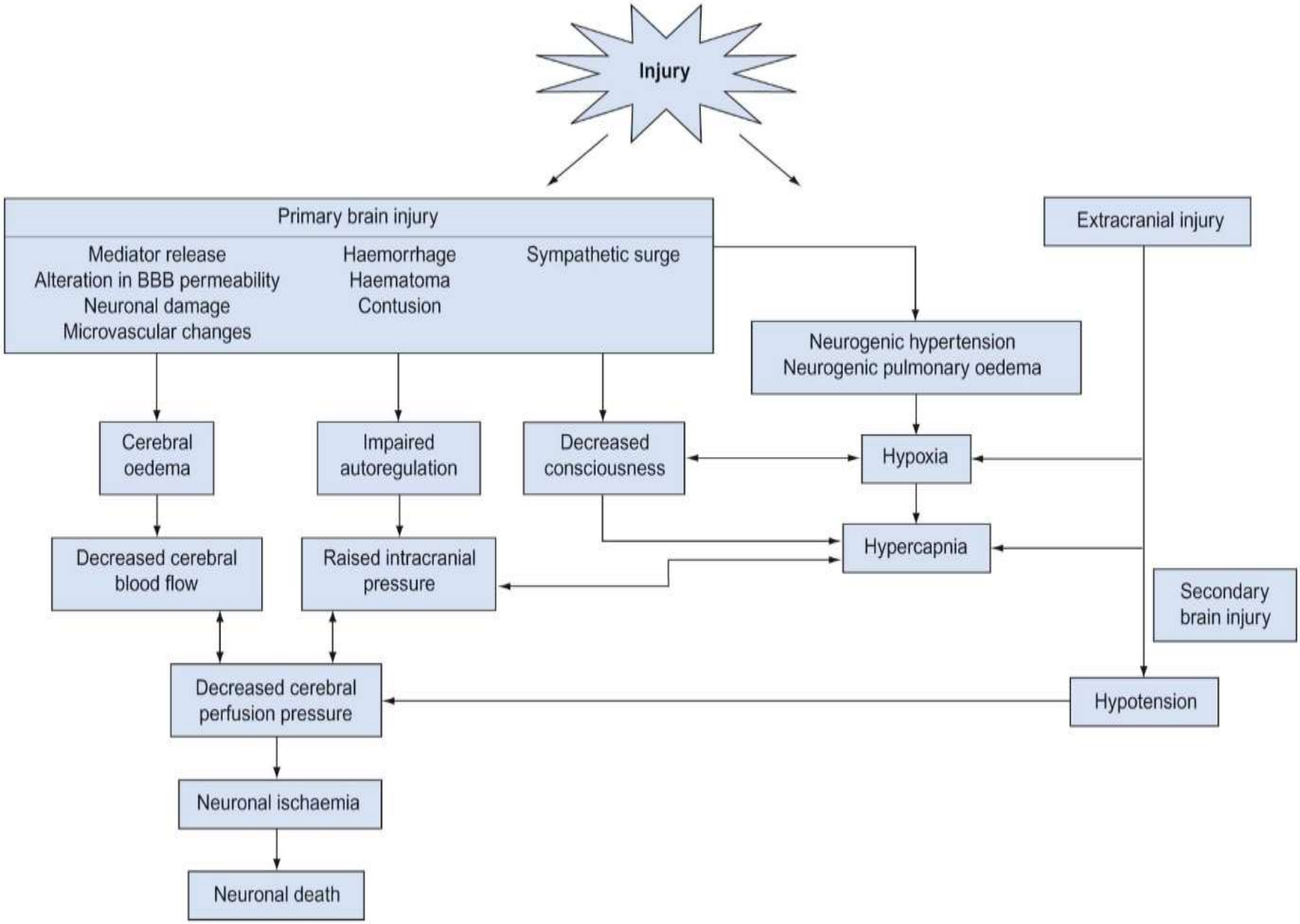
  (*acute change may necessitate 4.0-4.5)*
  - PaO2 >13 kPa

  (*for transfer/initial stability, otherwise aim 10-13)*

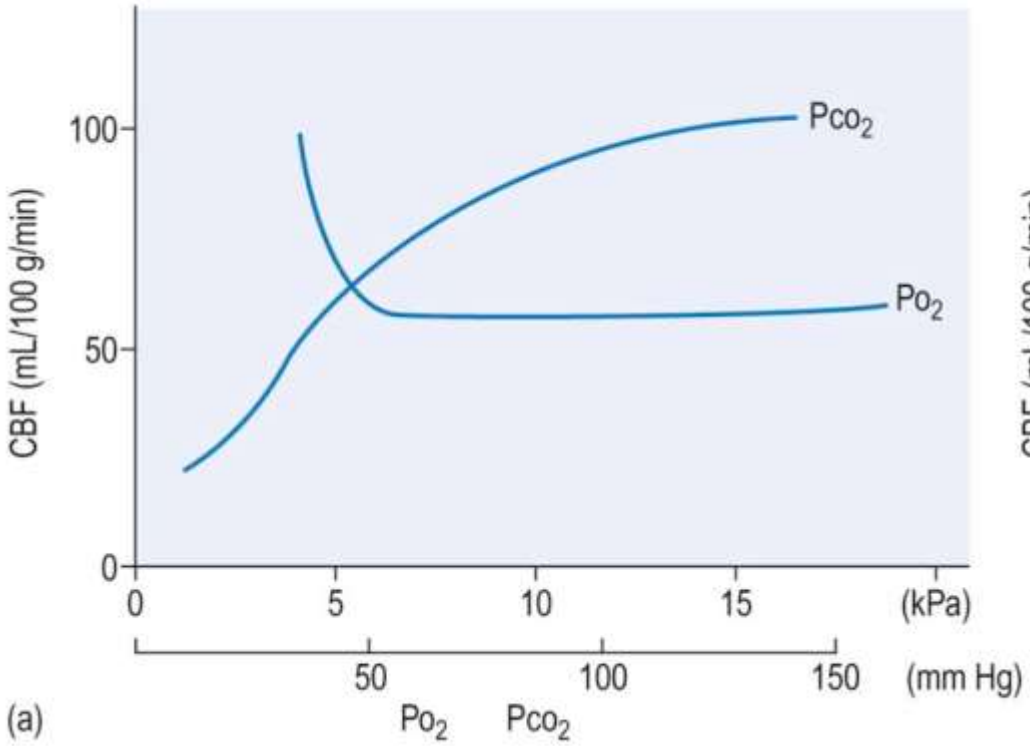
ABG Parameters

Table 3 Physiological variables and fluids for transfer of brain-injured patients. Of note, there is little high-quality evidence to support particular values and this table is the product of discussion and consensus between members of the Working Party. Where possible, the BP targets reflect the recommendations of the European Trauma Course.

	Traumatic brain injury (incl. traumatic subarachnoid haemorrhage)	**Intracerebral haematoma/ haemorrhagic stroke**	**Acute ischaemic stroke**	**Spontaneous subarachnoid haemorrhage**
Systolic blood pressure (mmHg)	> 110 (and MAP > 90 mmHg) < 150	< 150 if within 6 h of onset of symptoms and immediate surgery not planned	> 140 < 185 (if candidate for/has received i.v. thrombolysis); or, < 220 (if thrombolysis is contraindicated and being transferred for thrombectomy)	> 110 < 160
$PaCO_2$ (kPa) If impending uncal herniation a brief period of $PaCO_2$ 4.0–4.5 may be used)	4.5–5.0	4.5–5.0	4.5–5.0	4.5–5.0
Oxygenation (kPa) or saturation (%) (avoid hyperoxia)	$PaO_2 \geq 13$	$PaO_2 \geq 13$	Aim ≥ 95% (add oxygen only if < 95%)	$PaO_2 \geq 13$



Soni, Bernsten 2014 Oh's Intensive Care Manual, 7th Edition



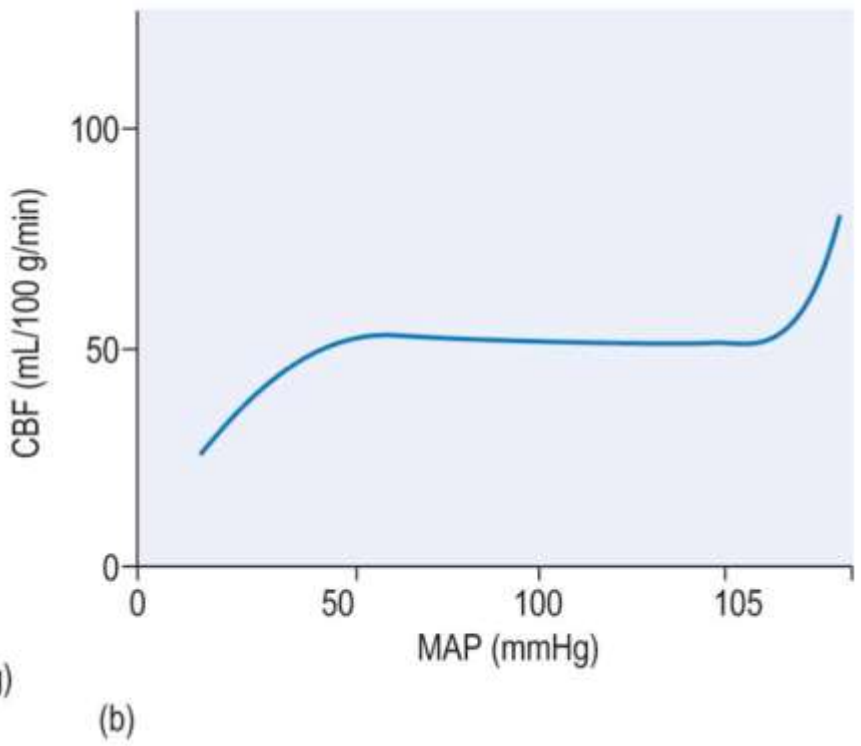


Figure 52.2 (a) The relationship between the partial pressure of oxygen (P_{O_2}) and carbon dioxide (P_{CO_2}) and cerebral blood flow (CBF). (b) The relationship between mean arterial pressure (MAP) and cerebral blood flow under normal circumstances, illustrating the range of autoregulation.

TBI Cerebral Blood Flow

- Hypoperfusion
  - 0-72hrs
  - Loss autoregulation, dependence upon MAP
  - Cytotoxic/ischaemic oedema
- Hyperaemia (25-30% pts)
  - First week
  - Inflammatory mediators
  - Vasogenic (BBB) oedema
- Vasospasm (10-15%)
  - Two weeks
  - Impaired autoregulation (overactive), supranormal MAP requirement
  - May coexist with neurogenic hypertension (usually >day 5)

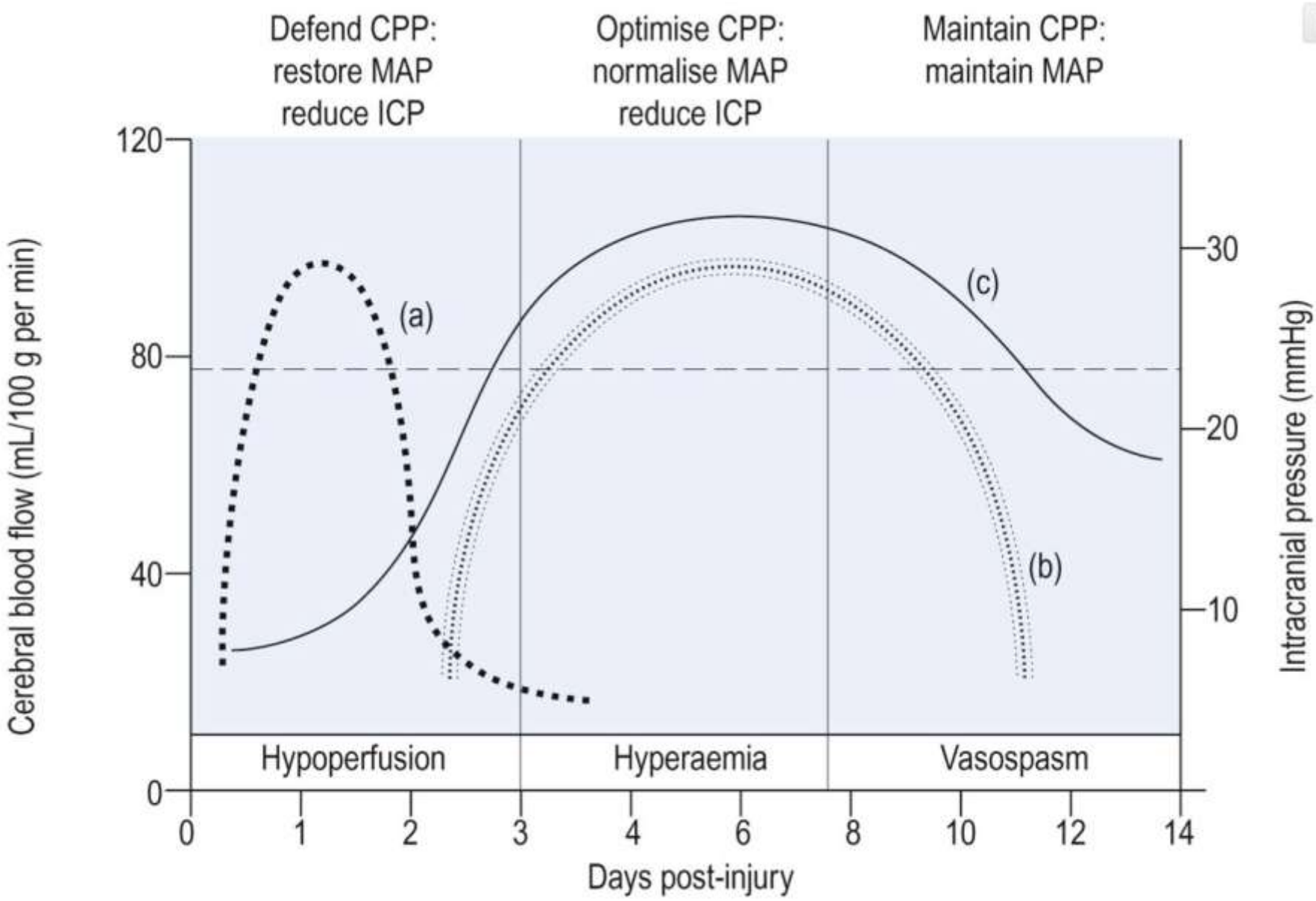


Figure 75.2 Conceptual changes in cerebral blood flow and intracranial pressure (ICP) over time following traumatic brain injury: (a) cytotoxic oedema; (b) vasogenic oedema; (c) cerebral blood flow. CPP = cerebral perfusion pressure, MAP = mean arterial pressure.

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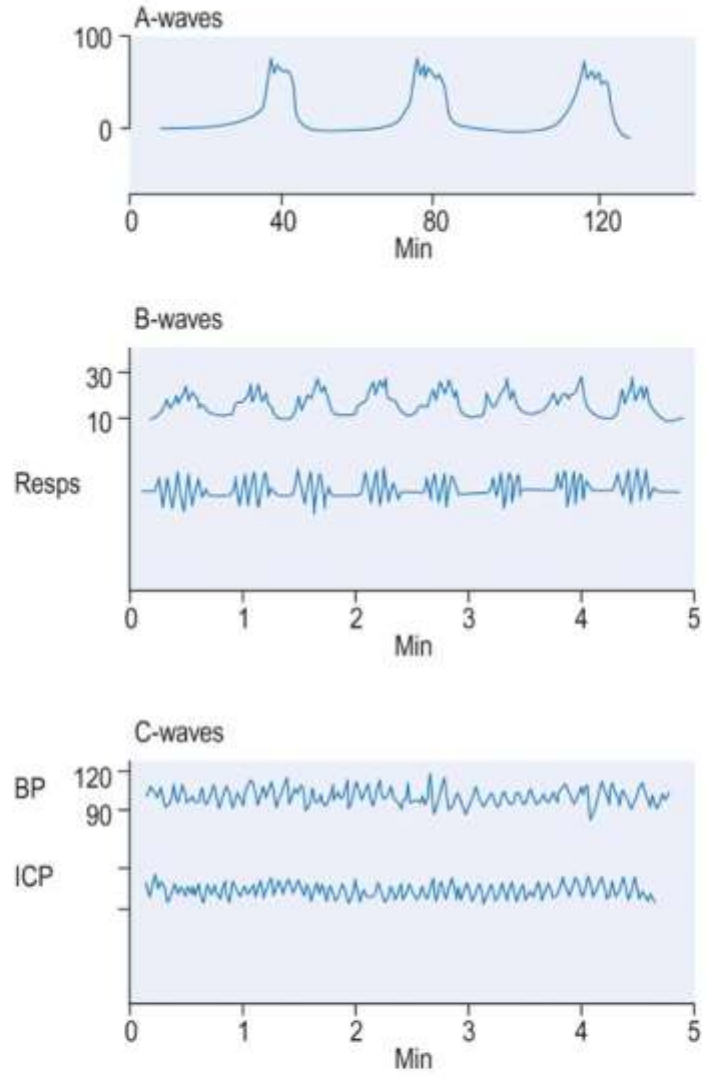

Figure 52.3 Intracranial pressure (ICP) waveforms. A-waves are plateau waves of 50–100 mmHg, sustained for 5–15 minutes, associated with raised ICP and compromised cerebral blood flow. B-waves are small changes in pressure every 0.5–2 minutes, often associated with breathing patterns and possibly due to local variations in the partial pressure of oxygen and carbon dioxide. C-waves are low-amplitude oscillations with a frequency of about 5 per minute, associated with variation in vasomotor tone. Resps = respirations; BP = blood pressure.

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Secondary brain insults following traumatic brain injury that are associated with increased morbidity and mortality

Systemic	Intracranial
Hypoxia	Seizure
Hypotension	Delayed haematoma
Hypocapnia	Subarachnoid haemorrhage
Hypercapnia	Vasospasm
Hyperthermia	Hydrocephalus
Hypoglycaemia	Neuroinfection
Hyperglycaemia	
Hyponatraemia	
Hypernatraemia	
Hyperosmolality	
Infection	

General *Guideline* Parameters

- A/B
  - PaO2 >8/10/13 (BTF/EBIC/AAGBI)
  - PaCO2 4.5-5 (not <4)
- C
  - MAP >90 (higher if CPP dictates)
  - Intravascularly euvolaemic (avoid hypervolaemia)
  - Correct anaemia/coagulopathy
  - Optimise venous drainage (head up 20-30 degree, loose ties)
- D
  - ICP <20; CPP >60
- Everything else…
  - Slight hypernatraemia (Na 145-150); avoid >160/hyperosmolality >320.
  - Treat seizures
  - Glucose 6-10

Every patient is different (e.g. vasospasm vs ICP, CO2 vs waking, etc…)

Risk vs benefit (LMWH, C-spine immobilisation)

Craniectomy improve outcome?

- RescueICP
- Prospective RCT, ICP >25mmHg
  - Despite position/sedation/paralysis, then diuresis, osmotic manipulation
- Craniectomy vs Medical (barbiturates)
- Significant Mortality/Morbidity difference, "Good" outcome similar

- Hutchinson, P. J., Kolias, A. G., Timofeev, I. S., Corteen, E. A., Czosnyka, M., Timothy, J., ... & Wadley, J. (2016). Trial of decompressive craniectomy for traumatic intracranial hypertension. New England Journal of Medicine, 375(12), 1119-1130.

Cooling improve outcome?

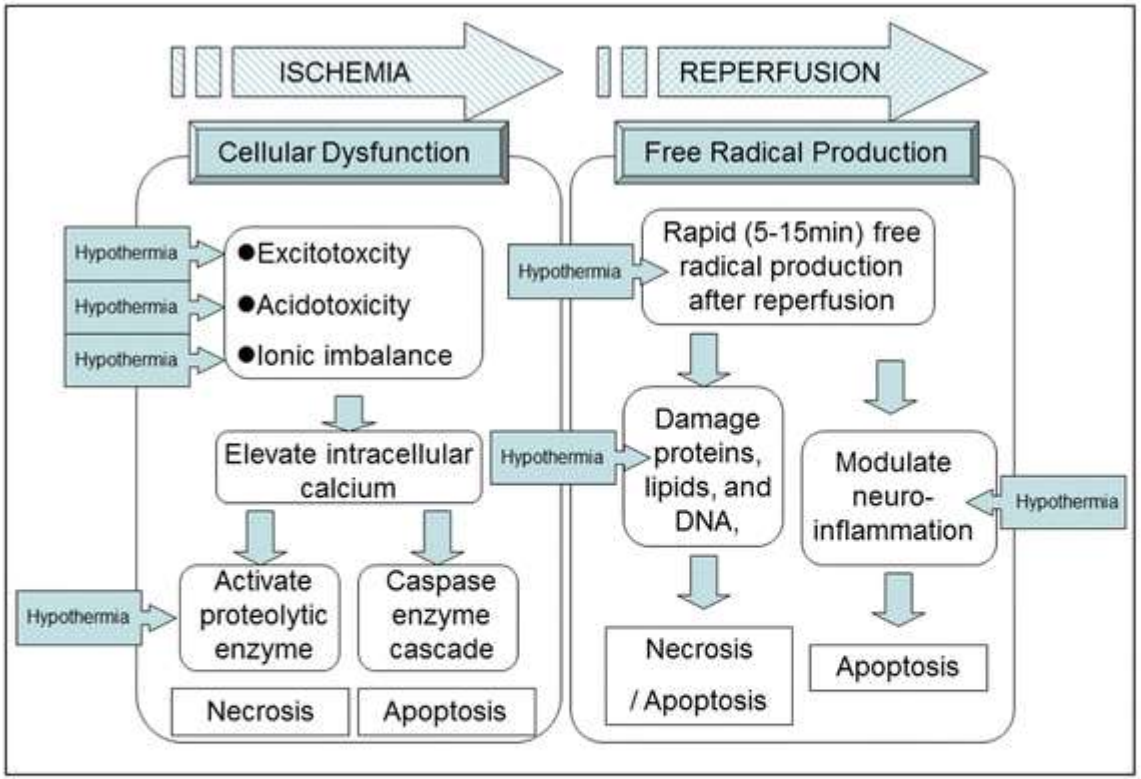


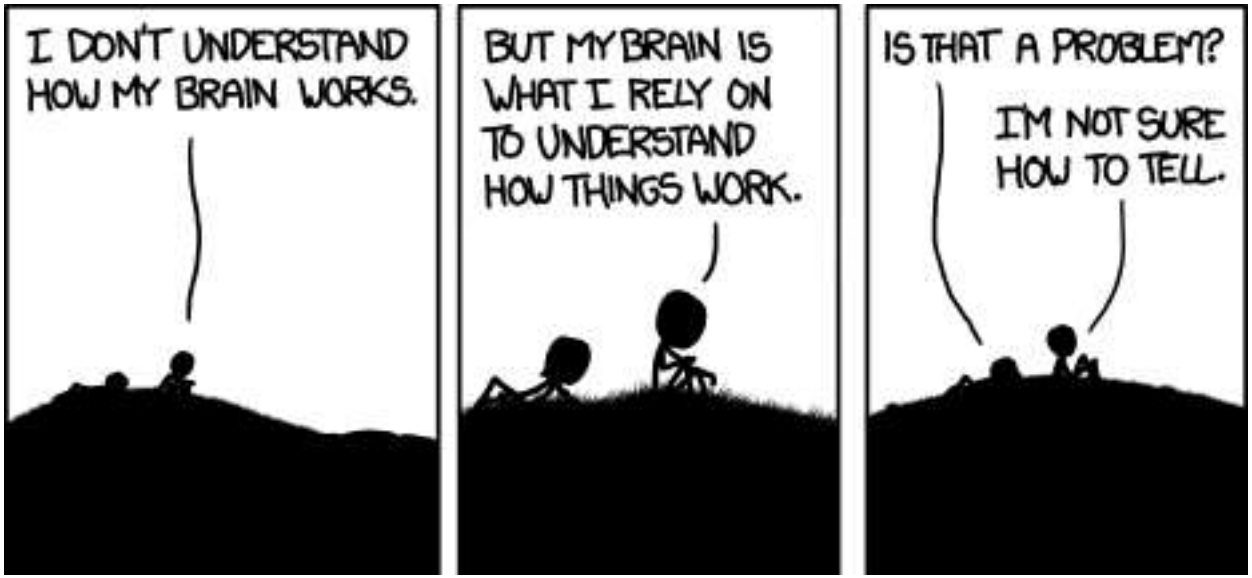
Fig. 1
The schema of mechanisms of ischemic/reperfusional (I/R) brain injury and the effective point of hypothermia treatment. The pathology of I/R injury is approximately separated as two mechanisms, i.e., the cell death following cellular dysfunction in ischemic phase and the free radical production in reperfusion phase. The *boxed arrow* with entered "Hypothermia" means the estimated effective points in I/R cascade

Andrews, P. J., Sinclair, H. L., Rodriguez, A., Harris, B. A., Battison, C. G., Rhodes, J. K., & Murray, G. D. (2015). Hypothermia for intracranial hypertension after traumatic brain injury. New england Journal of Medicine, 373(25), 2403-2412.

Thanks for listening

- *Questions please*

bit.ly/3D2su3x



Critical Care TBI

- Indications for airway security
- Mechanisms of primary injury
- Secondary injury supportive management
  - Consideration CMRO2
  - Changes in cerebral blood flow
  - Supportive treatment goals