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# Neurocritical Care

## A Guide to Practical Management

Springer

*Editors*
John P. Adams
The General Infirmary at Leeds
Great George Street
Leeds LS1 3EX
United Kingdom
John.Adams@leedsth.nhs.uk

Justin McKinlay
The General Infirmary at Leeds
Great George Street
Leeds LS1 3EX
United Kingdom
justin.mckinlay@leedsth.nhs.uk

Dominic Bell
The General Infirmary at Leeds
Great George Street
Leeds LS1 3EX
United Kingdom
dominic.bell@leedsth.nhs.uk

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*John Adams dedicates this book to his wife Kate to compensate for neglect of his responsibilities as husband and father. The families of his fellow editors did not specifically notice or comment and for this we are grateful.*

# Preface

Brain injury is a worldwide leading cause of mortality and morbidity and requires early and appropriate management to minimize these adverse sequelae. Despite such needs, access to specialist centers is limited, forcing both immediate and secondary care of these patients onto generalist staff. These responsibilities are made more problematical by differences in patient management between and even within specialist centers, due in part to an insufficient evidence-base for many interventions directed at brain injury.

This book is borne out of the above observations and is targeted at emergency and acute medicine, anesthetic and general intensive care staff caring for brain injury of diverse etiology, or surgical teams responsible for the inpatient care of minor to moderate head trauma.

Although explaining the various facets of specialist care, the book is not intended to compete with texts directed at neurosciences staff, but aims to advise on optimal care in general hospitals, including criteria for transfer, by a combination of narrative on pathophysiology, principles of care, templates for documentation, and highly specific algorithms for particular problems. It is intended that the content and structure can form the basis of guidelines and protocols that reflect the needs of individual units and that can be constantly refined. Our ultimate goal is to promote informed, consistent, auditable, multidisciplinary care for this cohort of patients and we hope that this text contributes to that process.

# Acknowledgments

We are indebted to our fellow authors who have not only made this book possible, but have approached the task with enthusiasm. All understand and endorse the importance of clear, comprehensive, evidence-based, and consistent advice in the support of colleagues caring for these patients outside the regional center.

We are also grateful for the observations of colleagues responsible for the eventual rehabilitation of these patients, mainly that even minor reductions in neurological deficit by early and appropriate care, can have a significant impact on quality of life, with proportional benefit not only for the patient, but family, health and social care institutions, and society. These observations justify the book and warrant implementation of the contained principles.

Finally, we thank Melissa Morton in the UK and Robin Lyon in New York for all their help and support in bringing this book to publication.

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# Contributors

*John P. Adams*
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Louise Barnes*
Hull Royal Infirmary
Hull and East Yorkshire Hospitals NHS Trust
Hull HU3 2JZ
UK

*Dominic Bell*
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Matthew Clark*
Department of Anesthetics and Intensive Care
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Nicola Cooper*
Leeds Teaching Hospitals NHS Trust
Leeds General Infirmary
Leeds
West Yorkshire LS1 3EX
UK

*Simon Davies*
Department of Anaesthetics
York Hospital NHS Trust
York Hospital
York
North Yorkshire YO31 8HE
UK

*Miles Denton*
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Morgan Feely*
Department of Neurology
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds General Infirmary
Leeds
West Yorkshire LS1 3EX
UK

*Tony Goddard*
Department of Neuroradiology
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Ahamad Hassan*
Department of Neurology
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Simon Holbrook*
Academic Unit of Anesthesia
St. James's University Hospital
Leeds
West Yorkshire LS9 7TF
UK

*Andrew Lindley*
Leeds Teaching Hospitals NHS Trust
Leeds General Infirmary
Leeds
West Yorkshire LS1 3EX
UK

*Kshitij Mankad*
Department of Neuroradiology
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Justin McKinlay*
Department of Anaesthetics and Neurocritical Care
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Paul G. Murphy*
Department of Anaesthesia
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Audrey C. Quinn*
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Jake Timothy*
Department of Neurosurgery
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Michael Vucevic*
Department of Anesthetics
Leeds General Infirmary
Leeds Teaching Hospitals NHS Trust
Leeds
West Yorkshire LS1 3EX
UK

*Abigail Walker*
Department of Anesthesia
Christchurch Hospital
Christchurch
Canterbury
NZ

# Glossary of Terms and Abbreviations

| | |
|---|---|
| A/B/C | Airway, breathing, circulation |
| ALI | Acute lung injury |
| APTT | Activated partial thromboplastin time |
| BAL | Bronchoalveolar lavage |
| BIS | Bispectral index |
| BP | Blood pressure |
| CMV | Cytomegalovirus |
| CNS | Central nervous system |
| COAG | Coagulation screen |
| CPP | Cerebral perfusion pressure (MAP-ICP) |
| CRP | C-reactive protein |
| CSF | Cerebrospinal fluid |
| CT | Computed tomography |
| CVP | Central venous pressure |
| CXR | Chest X-ray |
| ECG | Electrocardiogram |
| EEG | Electroencephalogram |
| ESR | Erythrocyte sedimentation rate |
| $EtCO_2$ | End-tidal carbondioxide concentration |
| FBC | Full blood count |
| $F_iO_2$ | Fraction of inspired oxygen |
| GCS | Glasgow coma scale |
| Gluc | Glucose |
| HAS | Human albumin solution |
| Hb | Hemoglobin |
| HIV | Human immunodeficiency virus |
| HR | Heart rate |
| HSE | Herpes simplex encephalitis |
| IABP | Invasive arterial blood pressure |
| ICP | Intracranial pressure |
| ICU | Intensive care unit |
| INR | International normalized ratio |
| IV | Intravenous |
| LFTs | Liver function tests |
| LP | Lumbar puncture |
| MAP | Mean arterial pressure |

| | |
|---|---|
| MI | Myocardial infarction |
| MRI | Magnetic resonance imaging |
| MRSA | Methicillin-resistant Staphylococcus aureus |
| NaCl | Sodium chloride |
| NEAD | Non-epileptic Attack Disorder |
| NGT | Nasogastric tube |
| NICE | National Institute for health and Clinical Excellence |
| NJT | Nasojejunal tube |
| NPE | Neurogenic Pulmonary Edema |
| NSAID | Non-steroidal anti-inflammatory drug |
| ODM | Oesophageal doppler monitor |
| OGT | Orogastric tube |
| $P_aCO_2$ | Partial pressure of carbondioxide (arterial blood) |
| $P_aO_2$ | Partial pressure of oxygen (arterial blood) |
| PCR | Polymerase chain reaction |
| PCWP | Pulmonary capillary wedge pressure |
| PE | Pulmonary embolism |
| PEEP | Positive end-expiratory pressure |
| $PbtO_2$ | Partial pressure of brain tissue oxygen |
| PPI | Proton pump inhibitor |
| PVS | Persistent vegetative state |
| $S_aO_2$ | Arterial oxygen saturation |
| Spp | Species |
| $S_{jv}O_2$ | Jugular venous oxygen saturation |
| TB | Tuberculosis |
| U&Es | Urea and electrolytes |
| UK | United Kingdom |
| Vt | Tidal volume |
| VTE | Venous thromboembolism |
| WCC | White cell count |
| WFNS | World Federation of Neurosurgical Socities |

# 1
# Brain Injury and Dysfunction: The Critical Role of Primary Management

M.D. Dominic Bell

## Key Points

1. In traumatic brain injury, maintain mean arterial (MAP) blood pressure >80 mmHg.
2. Avoid hypoxia at all costs; keep $P_aO_2$ >13 kPa, using PEEP if necessary.
3. Keep $P_aCO_2$ 4.5–5.0 kPa; hyperventilate only if there are signs of impending brainstem herniation.
4. Keep the neck in neutral position; always consider the possibility of cervical spine injury.
5. Maintain 15° head up position (as long as MAP adequate).
6. Do not give mannitol if patient is hypotensive. Speak to a Regional Neurosurgical Center before giving additional doses.

## Introduction

The human brain, in structure and function, represents the pinnacle of biological evolution. Even the most rudimentary non-volitional role of matching ventilation to demand or maintaining homeostasis is phenomenally complex for an organism vulnerable to disease or dysfunction of the component tissues and organs, and more particularly when exposed to mechanical, chemical, and thermal hazard as every environmental extreme is challenged. The coordination of physical movement, played out at the highest level in sport and the performing arts, rightly warrants recognition as a marker of complex neuronal activity, but conventionally, as a form of intelligence, bows to the cognitive capacity of the human brain. Numerical and literary skills, communication, memory, and knowledge are entry-level cognitive skills, with man's advances through understanding of both science and nature representing a higher plane. Reasoning and judgment, coupled with awareness of the needs of others and social skills arguably constitute the highest form of human intelligence. Interlinked with this function are those characteristics of personality and emotional status which generate individual uniqueness. These may be reflected in our achievements, as in career choice, or functional and artistic creativity, or our behavior relating to those achievements, as in innovation, ambition, and leadership. These higher functions also have an emotional dimension covering conscience, charity and self-sacrifice, enthusiasm, and the ability to love, rejoice and grieve.

This refinement and complexity of normal cerebral function is, however, associated with certain inherent vulnerabilities carrying significant implications for the management of either primary or secondary brain pathology or dysfunction. Tissues such as bone are able to regain normal architecture after injury, complex organs such as the liver and kidney are able to regenerate with restoration of original levels of function, and heart, lung, and pancreas are able to withstand devascularization and subsequent transplantation. The specialization of cellular structure and function within the central nervous system, however, appears to exclude a capacity for repair and renewal after anything other than the most trivial insult. Brain

tissue has a high requirement for oxygen and energy substrates to maintain both structure and function, leaving little reserve in the face of impaired delivery. Even with normal arterial oxygen content, circulatory arrest will result in loss of consciousness within 15 seconds, and given the high oxygen requirements simply to maintain cellular integrity, more than 5 minutes of circulatory arrest at normothermia will result in neuronal death and a significant multifaceted neurological deficit. These aspects demonstrate the exquisite vulnerability of the brain to the so-called secondary cerebral insults, with cellular hypoxia being the commonest final pathway.

There is a gradient of sensitivity of the different neural tissues to a global insult such as hypoxia, whereby the loss of higher function precedes loss of motor activity, with ventilatory effort maintained until immediately prior to death. This pattern parallels the picture of recovery from such an insult, the extreme end of the spectrum being the persistent vegetative state, where the patient is self-ventilating, but has no awareness of environment or self. This demonstrates that survival alone cannot be considered a satisfactory outcome from brain injury, and that all effort must be directed toward preventing, where possible, even the most subtle changes to personality and cognitive function at the other end of the spectrum, that would require the skills of a clinical psychologist to objectively quantify. Failure to address these aspects results not only in significant disability for patient and family, but phenomenal burden and cost to society.

This edition of the series, devoted to neurocritical care and the prevention or minimization of such avoidable neurological deficit, examines the theory and evidence-base behind the various management strategies expected of a regional unit. The secondary aim is to define and promote principles of care that can be deployed by any discipline, at any level of seniority, at any location, at any time, for any patient, with any pathology, and at any stage. Such principles, both clinical and procedural, are essential, given that most neuropathology arises outside the setting of a specialist center, and many patients will not access that center, either because neurosurgical intervention is not required, other injuries require immediate management, or because of limited bed availability.

Given the vulnerability of the brain as outlined earlier, it is unacceptable if the patient accrues additional avoidable morbidity in these circumstances, or indeed while awaiting or during transfer to the regional unit, through ignorance. Clinical experience also highlights how patient care can be compromised due to a lack of clarity and consistency in the referral process and acceptance by the regional unit, resulting in a hiatus in care with neither party taking full responsibility for these aspects. Such a scenario is arguably more unacceptable than ignorance, and demands explicit policy from the center and audit of process to monitor compliance.

## Role of the Regional Neurosurgical Center (RNC)

Fundamental to optimal patient management and any relationship with the regional center is an understanding of the specific services provided there. The greatest demand will be for care of traumatic brain injury, followed by subarachnoid hemorrhage, but the centers also have an emerging involvement in conventional "strokes." Thrombolysis or interventional radiology for an ischemic infarct are being increasingly adopted as appropriate emergency care, mirroring the approach taken to occlusive coronary events. The implications of managing these patients as medical emergencies cannot be overestimated, but the care and cost implications of the current conservative evaluative approach to strokes are significant, regardless of the impact on the patient and family.

The role of the regional center for this range of pathology can be summarized as intervention, neuro-specific monitoring, and advice for referring units. Given that vascular pathology is addressed in subsequent chapters, the role is only outlined in greater depth, as below, for traumatic brain injury:

1. To expedite removal of a significant intracranial hematoma
2. To monitor for the potential expansion of a less significant hematoma
3. To provide specialized monitoring (e.g., intracranial pressure, jugular venous oximetry) to direct the neuro-intensive care of the diffuse axonal injury
4. To undertake radical surgical maneuvers for refractory intracranial hypertension, e.g., decompressive craniectomy or lobectomy for extensive contusion

Although it could be argued that a patient should be transferred to a specialized unit for

imaging and assessment of the patient to make the above distinctions, CT scanning in the referring hospital has reduced the necessity for this and digital image transfer should improve the quality of discussion and decision-making. Furthermore, it is clearly not in the interest of the majority of patients to be transferred for the sole purpose of diagnosis.

## Indications for Patient Transfer

- *Group 1*: Transfer delayed only for correction of secondary cerebral insults or for life-saving surgery (e.g., expanding extradural hematoma with localizing signs).
- *Group 2*: Requires urgent transfer following optimization and life and limb saving surgery (e.g., subdural hematoma with no mass effect).
- *Group 3(a)*: Patients should only be transferred after absolute stabilization given that the overall principles of care are to avoid secondary cerebral insults, rather than to offer neuro-specific therapies (e.g., contusional injury with no mass effect).
- *Group 3(b):* Some non-neurosurgical intensive care units (ICU) monitor ICP in cases of diffuse axonal injury; transfer may become necessary if the ICP subsequently becomes difficult to control.

## Organizing the Response

*Groups 1-3(a)* above demonstrate the importance of the primary decision-making which involves diagnostic skills, confident liaison with the regional center, and an appropriate level of care in the event of retention of the patient. This responsibility usually falls to the attending anesthetist or intensive care specialist following initial stabilization in the emergency department. This individual has a pivotal role in coordinating this process and therefore assumes both clinical and logistical responsibilities (see Table 1.1).

**Table 1.1.** Roles of the attending specialist during the primary management of patients with traumatic brain injury

1. Primary resuscitation
2. Neurological assessment
3. Deciding on the need for intubation, sedation and ventilatory support
4. Management of problems such as convulsions
5. Interpretation of CT scans adequate for prioritization of treatment options
6. Prioritizing and expediting essential general surgical and orthopedic interventions
7. Deciding on transfer or retention after such interventions
8. Maintaining neurological observations
9. Avoiding secondary cerebral insults or expansion of any intracranial pathology
10. Organizing further CT scans in the event of retaining a patient
11. Maintaining dialog with the neurosurgeons and the neurosurgical intensive care
12. Deciding, in the face of massive injury, that no overall benefit from transfer exists

## Avoidance of Secondary Cerebral Insults

No treatment strategy can reverse neuronal death caused by the primary brain injury, but much can be done to avoid preventable secondary neuronal death and subsequent deficit. These secondary insults share a final common pathway that takes areas of the brain compromised by the primary injury, or indeed the whole brain, toward irreversible ischemia (see Fig. 1.1).

Secondary cerebral insults can be triggered by intracranial or systemic factors, which either reduce cerebral oxygen delivery or increase cerebral oxygen consumption (Table 1.2). In addition, an increase in the volume of brain, blood, or CSF, or an expanding space occupying lesion (e.g., hematoma) may increase the pressure within the rigid skull and trigger global ischemia. Focal damage may be caused by local compression or shearing forces.

### Cerebral Oxygen Delivery

Cerebral oxygen delivery depends upon:

(a) An adequate circulating volume at a perfusion pressure above the lower level of cerebral autoregulation.
(b) An adequate amount of oxygenated hemoglobin that dissociates appropriately at tissue level.

### Cerebral Oxygen Consumption

To avoid excessive cerebral oxygen consumption in the context of compromised cerebral oxygen

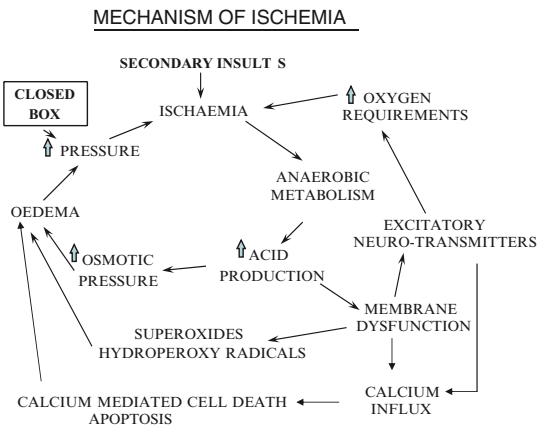


**FIGURE 1.1.** Mechanism of ischemia in brain injury.

**TABLE 1.2.** Intracranial and systemic causes of secondary brain injury

| Intracranial | Systemic |
|---|---|
| Expanding contusion/hematoma | Hypotension |
| Cerebral edema | Hypoxia |
| Vascular injury/carotid dissection | Hypo or Hypercapnia |
| Seizures | Pyrexia |
| Hydrocephalus | Coagulopathy |
| Vasospasm | Hypo or hyperglycemia |
| Pneumocephalus | Anemia |
| Intracranial infection | Sepsis |

delivery, it is essential to recognize and actively treat any seizure activity and to provide adequate analgesia and sedation, once a patient is intubated and ventilated. Pyrexia should be treated with active cooling measures once the patient is stabilized on the ICU. Hyperglycemia, which is believed to increase cerebral oxygen consumption, should be targeted during all epochs of care.

## Expansion of Intracranial Contents

(a) *Space-Occupying Lesions,* for example, hematomata or contusions
The key priority is to determine whether urgent neurosurgery is required. General supportive care includes avoidance of aspects that allow a hematoma to expand through loss or dilution of platelets or coagulation factors. Hypothermia, hypocalcemia, and administration of large volumes of colloid solutions should be avoided. These aspects assume greatest significance in the context of a subdural or intracranial hematoma, where such attention may avoid the need for surgical intervention.

(b) *Brain Edema* – Four Mechanisms:

1. *Hydrostatic edema:* occurs when arterial pressure exceeds the upper limit of autoregulation or when there is *venous congestion* (head-down position, pressure on the jugular veins, high intrathoracic pressure).
2. Osmotic edema: non-ionic crystalloid solutions such as *dextrose* become, in effect, free water once the sugar component is metabolized.
3. *Oncotic edema:* due to low plasma proteins; can become important when the blood–brain barrier (BBB) is damaged.
4. *Inflammatory edema*: the inflammatory response to insults such as trauma or hypoxia can lead to increased capillary permeability and disruption of the BBB. It is critically important to avoid preventable insults such as osmotic edema when this has arisen.

The management of cerebral edema and raised intracranial pressure traditionally involves administration of mannitol. This can only be effective if the BBB is intact, there is mass rapid movement of water from the tissues into the circulating compartment, and finally rapid excretion via the kidneys of both mannitol and water. The main role of mannitol is to *temporarily* reduce the amount of brain water and thereby reduce overall intracranial pressures and relieve pressure on vital structures such as the brainstem. This buys time before definitive neurosurgical intervention. By reducing the size of normal brain, abnormal areas including hematomata can expand, generating a greater shearing effect. If mannitol is used indiscriminately with a deranged BBB, the molecule can diffuse across and ultimately contribute to the development of osmotic edema. This is more likely to occur with hypotension and poor renal perfusion such that the mannitol is not excreted.

### *Increase in Cerebral Blood Volume*

1. *Arterial*: $\uparrow P_aCO_2$ is the commonest avoidable cause of cerebral arterial vasodilatation.
2. *Venous*: discussed earlier, for example, neck positioning, endotracheal tube ties.