Traumatic Brain Injury Part IV: General Intensive Care for Patients with Traumatic Brain Injury

Table of Contents

Traumatic Brain Injury Part IV: General Intensive Care for Patients with Traumatic Brain Injury

First Edition Update **2022**

Reviewers

Carmen Lopez Soto MD, MSc, EDIC, *Consultant in Critical Care and Anaesthesia. King's College Hospital NHS Foundation Trust, London, United Kingdom*

Co-Ordinating Editor

Carmen Lopez Soto MD, MSc, EDIC, *Consultant in Critical Care and Anaesthesia. King's College Hospital NHS Foundation Trust, London, United Kingdom*

Executive Editor

Nathan D. Nielsen MD, MSc, *Associate Professor, Division of Pulmonary, Critical Care and Sleep Medicine, University of New Mexico School of Medicine, Albuquerque, United States; Editorial Board and Sepsis Section Editor, ESICM Academy*

First Edition **2022**

Authors

Virginia Newcombe MD, *Neurosciences and Trauma Critical Care Unit (NCCU), Addenbrooke's Hospital, Cambridge; Perioperative, Acute, Critical Care and Emergency Medicine (PACE) Section, Department of Medicine, University of Cambridge, United Kingdom*

Marco Carbonara MD, *Attending physician Neurointensive Care Unit Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico Milan (Italy)*

Francisco Chacon Lozsan MD, M.Ed, FESC, FACC, FEWM, *Critical Care Medicine/Neurocritical care medicine. Anesthesia and Intensive Care Unit Péterfy Sándor Hospital (Budapest).*

Reviewers

Jessie Rosalind Welbourne MBChB FRCA FFICM, *Consultant in Intensive Care Medicine and Neuroanaesthesia Medical lead for Neuro Intensive Care Plymouth Hospitals NHS Trust*

Editors

Carmen Lopez Soto MD, MSc, EDIC, *Consultant in Critical Care and Anaesthesia. King's College Hospital NHS Foundation Trust, London, United Kingdom*

Assessments Editors

Ashraf Roshdy MD, MBBch, MSc, PhD, FRCP (Edin.), *Consultant, General Intensive Care Unit, St George's University NHS foundation trust, London, UK.; Lecturer and Consultant of Critical Care Medicine, Alexandria University, Egypt*

Filippo Sanfilippo MD, PhD, *Anesthesia and Intensive Care Medicine Institute: Azienda Ospedaliera Universitaria Policlinico Vittorio Emanuele*

Mo Al-Haddad MBChB, FRCA, FFICM, EDIC, MSc (Clinical Education), *Consultant in Critical Care and Anaesthesia, Queen Elizabeth University Hospital Honorary Professor at the University of Glasgow*

CoBaTrICE Mapping Contributors

Cristina Santonocito MD, *Dept. of Anesthesia and Intensive Care, IRCSS-ISMETT-UPMC, Palermo, Italy*

Victoria Anne Bennett MD, MBBS, FFICM, FRCA, *Anaesthetic and Intensive care registrar, University Hospital Lewisham, London, United Kingdom*

Co-Ordinating Editor

Mo Al-Haddad MBChB, FRCA, FFICM, EDIC, MSc (Clinical Education), *Consultant in Critical Care and Anaesthesia, Queen Elizabeth University Hospital Honorary Professor at the University of Glasgow*

Executive Editor

Nathan D. Nielsen MD, MSc, *Associate Professor, Division of Pulmonary, Critical Care and Sleep Medicine, University of New Mexico School of Medicine, Albuquerque, United States; Editorial Board and Sepsis Section Editor, ESICM Academy*

Intended Learning Outcomes

TBI Part IV: General Intensive Care for Patients with TBI

1. Discuss the importance of optimising general intensive care for patients with TBI
2. Summarise the respiratory system management in patients with TBI
3. Describe the gastrointestinal system, nutrition and stress ulceration aspects in patients with TBI
4. Discuss the metabolic disturbances and management in patients with TBI
5. Describe DVT prophylaxsis in patients with TBI
6. Discuss the prevention and control of infections in patients with TBI
7. Describe overall management of extracranial injuries in patients with TBI

eModule Information

Relevant Competencies from CoBaTrICE

TBI Part IV: General Intensive Care for Patients with TBI

- **2.9** Monitors and responds to trends in physiological variables
- **2.10** Integrates clinical findings with laboratory investigations to form a differential diagnosis
- **3.6** Recognises and manages the patient with neurological impairment
- **3.7** Recognises and manages the patient with acute gastrointestinal failure
- **4.1** Prescribes drugs and therapies safely
- **4.2** Manages antimicrobial drug therapy
- **4.4** Uses fluids and vasoactive / inotropic drugs to support the circulation
- **4.6** Initiates, manages, and weans patients from invasive and non-invasive ventilatory support
- **4.7** Initiates, manages and weans patients from renal replacement therapy
- **4.8** Recognises and manages electrolyte, glucose and acid-base disturbances
- **4.9** Co-ordinates and provides nutritional assessment and support
- **6.3** Manages the care of the patient following craniotomy under supervision
- **6.5** Manages the pre- and post-operative care of the trauma patient under supervision
- **7.1** Identifies and attempts to minimise the physical and psychosocial consequences of critical illness for patients and families
- **7.2** Manages the assessment, prevention and treatment of pain and delirium
- **7.3** Manages sedation and neuromuscular blockade
- **7.5** Manages the safe and timely discharge of patients from the ICU

1. General Intensive Care for Patients with TBI

It is impossible to care for the brain without caring for the whole organism. Aggressive treatments, indicated in most severe cases, carry injurious complications to the brain and other organs, as decompressive craniectomy and barbiturates for example. High quality intensive care is needed to complement complex interventions, to mitigate iatrogenic complications and to prevent avoidable morbidity. Many ESICM modules deal with aspects of the overall critical care of the patient and extensive links to these modules are used below. However, certain physiological systems are exposed specifically to TBI-related conditions and complications and these are summarised below.

1. 1. Central nervous system

1. 1. 1. Avoid oversedation

The negative effect of oversedation has been elucidated in patients with extracranial disease and includes cardiovascular depression with increased need for inotropes and longer mechanical ventilation duration. However, sedation in severe TBI is a standard, first-tier treatment to decrease metabolic rate, thus improving cerebral tissue tolerance to ischemia and, more importantly, reducing ICP via a reduction of cerebral blood volume. In those patients in whom ICP is stable and normal and CT findings don't show potentially evolutive lesion, sedation should be regarded as in general ICU, stopped or reduced substantially: daily assessment of the need for sedation and stopping sedation unless indicated is part of standard ventilator care.

See ACE Courses on Pain, Agitation and Delirium in Intensive Care and Mechanical Ventilation.

In text References

(Kress et al. 2000; Oddo et al. 2016)

1. 1. 2. Agitation after withdrawal of sedation

Prolonged sedation and analgesia may be associated with tolerance and features of withdrawal when drugs are stopped. These signs and symptoms include central nervous system activation, gastrointestinal disturbances and sympathetic hyperactivity. Agitation and delirium are common in general ICU and can occur also in TBI patients; unfortunately, no delirium scale has been validated for this population and, as in general ICU patients, treatment is mainly multifactorial with limited efficacy of antipsychotic medications.

Other central nervous system manifestations include hyperactive deep tendon reflexes, clonus, frequent yawning, sneezing, and hypertonicity. A reversible instability and elevation of ICP can be observed. Withdrawal syndrome is more severe, appears earlier in younger patients, and is a particular problem in paediatric ICU.

Symptoms due to withdrawal can be confused and overlap with those due to paroxysmal sympathetic hyperactivity (PSH), defined by an international consensus panel as “a syndrome, recognized in a subgroup of survivors of severe acquired brain injury, of simultaneous, paroxysmal transient increases in sympathetic (elevated heart rate, blood pressure, respiratory rate, temperature, sweating) and motor (posturing) activity”. This is a syndrome complicating severe TBI and other severe brain injury caused by loss of cortical inhibitory pathways and subsequent liberation of spinal circuit excitation, characterised by increased heart rate, respiratory rate, and blood pressure, as also diaphoresis and hyperthermia. Actual proportion of affected TBI patients varies according to diagnostic criteria and study design, but it’s been reported from 8 to 32% among most severe cases. Also, among these patients, PSH may mimic withdrawal of sedation or opioids. In the case of withdrawal, elevated sympathetic activity may almost be considered and is dulled in the acute phase by sedation and analgesia applied to control elevated ICP levels. However, during weaning from sedation, paroxysmal sympathetic hyperactivity may appear and last for weeks thus representing a potentially treatable cause of increased secondary morbidity.

1. 1. 3. Management of withdrawal and prevention of “oversedation”

Oversedation increases the risk of tolerance and consequently of withdrawal syndrome. Best treatment for withdrawal syndrome and complications due to sedation is early interruption or minimization of sedatives. Unfortunately, more severely injured patients may have higher ICP levels which need more sedation; the same patients are more at risk of paroxysmal sympathetic hyperactivity.

The effect on outcome of PSH is currently debated, but it is believed to be detrimental; thus, treatment of its effects sound sensible. Level of evidence supporting PSH therapy is generally low, and it relies mainly on expert opinions and local policies. Supportive therapy, such as adequate nutrition and physical therapy, is recommended; main drug classes used in prevention are opioids and intravenous sedatives in ICU, while treatment relies on beta or alpha blockers and benzodiazepines.

See the ACE Course on Pain, Agitation and Delirium in Intensive Care.

In text References

(Meyfroidt, Baguley and Menon. 2017; Williamson et al. 2016)

References

- Kress JP, Pohlman AS, O'Connor MF, Hall JB., Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation., 2000, PMID:10816184

- Oddo M, Crippa IA, Mehta S, Menon D, Payen JF, Taccone FS, Citerio G, Optimizing sedation in patients with acute brain injury., 2016, PMID:27145814
- Meyfroidt G, Baguley IJ, Menon DK., Paroxysmal sympathetic hyperactivity: the storm after acute brain injury., 2017, PMID:28816118
- Williamson DR, Frenette AJ, Burry L, Perreault MM, Charbonney E, Lamontagne F, Potvin MJ, Giguère JF, Mehta S, Bernard F., Pharmacological interventions for agitation in patients with traumatic brain injury: protocol for a systematic review and meta-analysis., 2016, PMID:27855720

1. 2. Respiratory system

1. 2. 1. Patient positioning

Appropriate positioning may reduce the frequency of pulmonary complications and silent (micro-) aspiration. The patients should be head-up at 30-45° and should be nursed alternating their positioning on their back, right and left side. Nursing head-up is also part of the ventilator care bundle (see above under sedation). Moreover, ICP is reduced in head-up position, probably due to better venous drainage, making it even more desirable.

In text References
(Wang et al. 2016)

1. 2. 2. Early and ultra-early tracheostomy

Early tracheostomy (4–6 days post injury), or ultra-early tracheostomy (1–3 days post injury) have been claimed as advantageous in reducing the use and duration of sedation, duration of mechanical ventilation and in preventing pulmonary and laryngeal complications. It may also accelerate safe discharge from ICU to a lower level of care and so enhance early physical rehabilitation. However, this is a controversial area and most studies have failed to show any effect on outcome. In general intensive care, the debate on early versus late (>10 days) tracheostomy got in favour of the latter after Tracman trial, a randomized controlled trial comparing this two timings found no difference in outcome in both mortality and length of stay. Even though it included patients with acute brain injuries, this study was mainly targeted to medical patients, therefore not powered to detect any difference in our population of interest. These findings cannot then be generalized to acute brain injured patients as a recent meta-analysis found a benefit on long-term mortality and ICU length of stay with early tracheostomy, with moderate to good quality small trials. Optimal timing of tracheostomy in TBI patients still needs to be identified.

See the ACE Courses module on Airway management.

In text References

(Rizk et al. 2011; Griffiths et al. 2005; Young et al. 2013)

References

- Young D, Harrison DA, Cuthbertson BH, Rowan K; TracMan Collaborators., Effect of early vs late tracheostomy placement on survival in patients receiving mechanical ventilation: the TracMan randomized trial., 2013, PMID:23695482
- Wang L, Li X, Yang Z, Tang X, Yuan Q, Deng L, Sun X., Semi-recumbent position versus supine position for the prevention of ventilator-associated pneumonia in adults requiring mechanical ventilation., 2016, PMID:26743945
- Rizk EB, Patel AS, Stetter CM, Chinchilli VM, Cockroft KM., Impact of tracheostomy timing on outcome after severe head injury., 2011, PMID:21786043
- Griffiths J, Barber VS, Morgan L, Young JD., Systematic review and meta-analysis of studies of the timing of tracheostomy in adult patients undergoing artificial ventilation., 2005, PMID:15901643

1. 3. Gastrointestinal system – nutrition and stress ulceration

Feeding is important to reduce catabolic effects and maintain immunological competence. There is evidence that the catabolic response in head-injured patients may be more profound and/or prolonged than in multiple trauma patients without head injury. Nutrition should be started shortly after admission with the intention of reaching full nutritional intake by day 3 in ICU since evidence suggests an improved outcome in terms of mortality and disability.

Enteral route is more physiological, less expensive and is associated with fewer complications than parenteral nutrition. It may also be associated with accelerated normalisation of nutritional status. However, there is a minor increased risk of reflux and potential for aspiration as gastric pH is higher (more neutralised) when enteral nutrition is established. Despite, the airway has a significant degree of protection against aspiration due to the use of cuffed tubes. Despite its perceived physiological benefits, enteral nutrition has not shown clear outcome benefit from randomized clinical trials.

The incidence of stress ulcers in patients with head injuries has been documented to be approximately 10%, twice the rate when compared with other patient groups. While the incidence of stress ulcers has decreased generally, severe TBI is an accepted risk factor for stress ulceration. Early enteral feeding and adequate use of analgesics and sedation are perhaps the most effective prophylaxis. H_2 antagonists, which reduce

acid production, is an established therapy and proton pump inhibitors are also used. However, with such agents there may be an increased risk of healthcare associated infections such as pneumonia and clostridium difficile colitis.

See the ACE Courses on Nutrition Part I to Part V.

In text References

(Curtis. 2011; Perel et al. 2006)

References

- Curtis L., Early, high quality enteral nutrition significantly improves outcome in head trauma patients., 2011, PMID:21846247
- Perel P, Yanagawa T, Bunn F, Roberts I, Wentz R, Pierro A., Nutritional support for head-injured patients., 2006, PMID:17054137

1. 4. Metabolic function

1. 4. 1. Disturbances of sodium balance

Disturbances of serum electrolytes are observed in approximately 60% of all patients with severe TBI. Changes of serum sodium concentration are the most common, therefore its importance to manage appropriately. Rapid recognition and management of such disturbances is dependent on serial clinical examination and careful monitoring: hourly urine output, blood pressure and central venous pressure measurements as well as frequent measurement of serum electrolytes and osmolarity and urine specific gravity and osmolality.

1. 4. 1. 1. Hypernatraemia

The most common causes of hypernatraemia (serum sodium >150 mmol/L) in patients with TBI are: central or neurogenic diabetes insipidus (DI) and osmotic diuresis (mannitol infusion). Central or neurogenic DI occurs when the brain does not secrete adequate levels of antidiuretic hormone. The diagnosis is probable once these signs are present:

- High urine output (>3 mL/kg BW/h)
- Low urine specific gravity (1001–1005)
- Low urinary osmolality <150 mOsmol/kg and a
- Urinary sodium below 50 mmol/L.

Increase in serum sodium completes the diabetes insipidus picture. Treatment includes parenteral desmopressin acetate or argipressin administered as soon as the diagnosis has been confirmed. Afterwards any depletion of fluid and potassium should be corrected. Administration of additional water via the enteral route may be necessary to

replace the calculated free water deficit. In a patient with hypernatremia, the first priority is to correct the cause and the correction of the serum sodium per se should be done carefully. The transmembrane water transfer works more on sodium gradients than on absolute values. Chronic sodium derangements are less dangerous than acute ones. In severely affected patients, the main aim should be to avoid a further sodium increase.

1. 4. 1. 2. Hyponatraemia

In general medicine, severe hyponatraemia is defined as a serum sodium level <120 mmol/L but in the TBI patient, different thresholds exist.

A sodium level of 140 mmol/L is often considered borderline hyponatraemia, while levels <135 mmol/L defined as hyponatraemia. Syndromic reasons for hyponatraemia are cerebral salt wasting syndrome (CWS) and syndromes of inappropriate ADH secretion (SIADH-syndrome, Schwartz-Bartter-Syndrome).

However, the commonest causes are:

- Low sodium intake
- Iatrogenic hyponatraemia (use of hypotonic solution, excessive correction of hypernatraemia)
- Increased sodium losses (e.g. via the gastric tube)

In young patients managed with arterial pressure levels higher than their physiological values with the aim to preserve CPP, natriuresis can occur due to a compensatory mechanism, called ‘aldosterone escape’. In such patients consider targeting CPP according to their physiology and reduce arterial values to more appropriate ones.

A definitive diagnosis of the underlying cause of hyponatraemia is sometimes difficult to obtain. While in both syndromes (SIADH and CWS) urinary sodium is elevated, conversely the volume of urinary output is completely different, low in SIADH and high in CWS. In patients with sodium depletion due to poor intake, the urinary sodium is low. Treatment depends on the cause. In the case of SIADH, this includes fluid restriction and the use of loop diuretics while in cases of CWS treatment involves the administration of fludrocortisone acetate (starting 0.1-0.2 mg per day) and hypertonic intravenous sodium solutions with close monitoring of serum sodium and water/sodium balance.

In text References

(Fraser and Stieg. 2006; Paiva et al. 2011; Brimioulle et al. 2008)

1. 4. 2. Control of blood sugar levels

Extreme hyperglycaemia is dangerous for the brain. However, routinely active control of glycaemia has not been shown to improve patient outcome, probably because of the dramatic consequences of the increased incidence of hypoglycaemia associated with such protocols. Today tight control of glycaemia is not recommended.

In the specific field of neurotrauma there are further concerns that a strict glycaemic control could further impair glucose diffusion in the area at risk. injured areas e.g. the contusional perilesional area, are affected by critical values of rCBF, and frequently, by an accelerated glucose metabolism (hyperglycolysis) and a reduction of serum glucose that could further impair glucose availability. If a centre wishes to obtain the theoretical benefit from a tight glucose control, complex cerebral monitoring such as cerebral microdialysis, should be used. In brain-injured patients, microdialysis studies have shown that glucose measured in the cerebral extracellular fluid and blood glucose are correlated with a positive linear relationship, as in normal subjects, but with a curve shifted down in the most metabolically disturbed patients. Thus, blood glucose concentrations in the lower range of normal (4-10) can result in cerebral glucose below a critical threshold for depletion.
The Brain Trauma Foundation guidelines have not issued recommendations on glucose control due to lack of strong evidence. Yet in TBI patients, it's been suggested to maintain glycaemia in the 150 mg/dL to 200 mg/dL (8.3– 11 mmol/L) range. This is supported by a recent meta-analysis by Hermanides et al showing no association between intensive glicemic control and mortality reduction, but increased risk of hypoglycaemia.

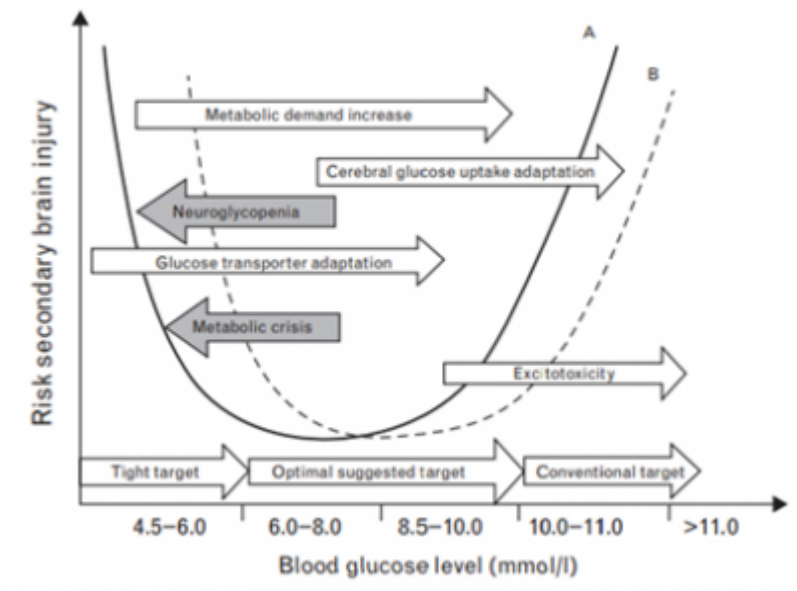

Figure 1:Suggested optimal target of blood glucose levels in acute brain injury. Adapted from Godoy et al.

Figure 1: *Suggested optimal target of blood glucose levels in acute brain injury. To convert to mg/dl, multiply by 18.(a) Basal state; (b) during injury, the demand of glucose increases, so the curve shifts to the right. Pathophysiological processes that affect glucose metabolism are depicted in arrows. Adapted from Godoy et al.*

In text References

(Chieregato. 2010; Qaseem et al. 2011; Hermanides et al. 2018; Magnoni et al. 2012; Godoy, Behrouz and Di Napoli. 2016)

References

- Fraser JF, Stieg PE., Hyponatremia in the neurosurgical patient: epidemiology, pathophysiology, diagnosis, and management., 2006, PMID:16883162
- Paiva WS, Bezerra DA, Amorim RL, Figueiredo EG, Tavares WM, De Andrade AF, Teixeira MJ., Serum sodium disorders in patients with traumatic brain injury., 2011, PMID:21941440
- Brimioulle S, Orellana-Jimenez C, Aminian A, Vincent JL., Hyponatremia in neurological patients: cerebral salt wasting versus inappropriate antidiuretic hormone secretion., 2008, PMID:17952405
- Chieregato A., Permissive mild to moderate hyperglycemia is safer for damaged brains., 2010, PMID:21102386
- Qaseem A, Humphrey LL, Chou R, Snow V, Shekelle P; Clinical Guidelines Committee of the American College of Physicians., Use of intensive insulin therapy for the management of glycemic control in hospitalized patients: a clinical practice guideline from the American College of Physicians., 2011, PMID:21320941
- Hermanides J, Plummer MP, Finnis M, Deane AM5 Coles JP6 Menon DK., Glycaemic control targets after traumatic brain injury: a systematic review and meta-analysis., 2018, PMID:29351760
- Magnoni S, Tedesco C, Carbonara M, Pluderi M, Colombo A, Stocchetti N., Relationship between systemic glucose and cerebral glucose is preserved in patients with severe traumatic brain injury, but glucose delivery to the brain may become limited when oxidative metabolism is impaired: implications for glycemic control., 2012, PMID:22610183
- Godoy DA, Behrouz R, Di Napoli M., Glucose control in acute brain injury: does it matter?, 2016, PMID:26866521

1. 5. Haematology/coagulation – venous thromboembolic disease

Patients with TBI are certainly at high risk of deep venous thrombosis (DVT). The prophylaxis of DVT with heparin is in constant conflict with the risk of inducing new intracerebral bleeding or increasing the volume of core injured areas. The risks of DVT in trauma patients are well defined. It has been increasingly suggested that in patients with TBI.

CT assessment of increase of haemorrhagic or intraparenchymal lesions is unlikely after the first 3 days post injury. Prophylaxis with low molecular weight heparin after the first 3 day post injury in combination with the application of mechanical compression

devices to the leg has been proposed. However, no large randomized controlled trials have evaluated the hazard of thromboprophylaxis in these patients. As such, an individualized approach seems to be reasonable to balance the risk of DVT versus risks of increased brain lesions.

See the ACE Courses on Bleeding and Thrombosis.

In text References
(Phelan. 2012)

References

- Phelan HA., Pharmacologic venous thromboembolism prophylaxis after traumatic brain injury: a critical literature review., 2012, PMID:22651698

1. 6. Infections – prevention and management

Patients in coma are at high risk of pneumonia. Indeed, some may have pulmonary aspiration before leaving the scene of trauma or reaching hospital. There is a general agreement that pulmonary infection severity in such patients is minimised by early intubation and ventilation. Pulmonary complications can be expected 3–7 days post injury, usually starting with a moderate increase of temperature, a deterioration in the purulence and volume of tracheal secretions and a decline in oxygenation. X-ray changes can vary from reduced ventilation at the lung bases to, infrequently, frank pneumonic changes. Various strategies of antimicrobial prophylaxis and therapy have been recommended. Wide spectrum prophylaxis is not acceptable due to increasing rate of multiresistent bacteria. It is reasonable to use hospital VAP protocols to guide diagnosis and initial therapy but specific microbiological/Infectious Diseases advice may be required for refractory infections where the patient may be a carrier of resistant organisms. Fever leading to an increase of ICP should be treated with antipyretics however continuous use of antipyretic drugs can mask infection. Specific treatments may increase the risk of severe pneumonia, including hypothermia and barbiturate use.

In the presence of a functioning gastrointestinal tract, parenteral nutrition should be avoided since it increases the risk of catheter related infection/bacteraemia. Almost all TBI patients will require central lines/catheters for infusion of catecholamines and sedation. Good practice advice is that central venous catheters should be reviewed daily and removed when not strictly needed to reduce the risk of catheter associated infections.

Urinary infections (and by extension sepsis) are also a significant risk due to the need for long-term indwelling urinary catheters in TBI patients. Diagnosis should be made by monitoring blood leukocyte count and systemic signs of infection, together with colony

count on urine culture. Appropriate antibiotics should be administered intravenously unless absorption from the GI tract is assured. Use of a urinary catheter care bundle may reduce the risk of urinary sepsis in TBI patients.

See ACE Courses on Sepsis and Septic Shock Part I to Part IV, Pyrexia (for diagnosis of catheter related infection; CRI) and Infection Prevention and Control.

In text References

(Ricard et al. 2012; Lissauer et al. 2012; Clarke et al. 2013)

References

- Ricard JD, Conti G, Boucherie M, Hormann C, Poelaert J, Quintel M, Rubertsson S, Torres A., A European survey of nosocomial infection control and hospital-acquired pneumonia prevention practices., 2012, PMID:22771420
- Lissauer ME, Leekha S, Preas MA, Thom KA, Johnson SB., Risk factors for central line-associated bloodstream infections in the era of best practice., 2012, PMID:22673242
- Clarke K, Tong D, Pan Y, Easley KA, Norrick B, Ko C, Wang A, Razavi B, Stein J., Reduction in catheter-associated urinary tract infections by bundling interventions., 2013, PMID:23220761

1. 7. Management of other injuries

TBI patients may have several extracranial priorities. Patients with any type of cord lesion should be stabilised surgically as soon as possible to allow patient mobilisation and reduce pulmonary complications. Patients with cervical spine lesions may have had anterior stabilisation and consequently tracheostomy should be delayed to allow the surgical wound to repair. Patients with pulmonary contusion and severe gas impairment may need an approach which recognises the conflict between strategies to improve gas exchange and those required for ICP control.

Abdominal surgery, haemoperitoneum, post-traumatic or post-surgical paralytic ileus may all increase intra-abdominal pressure (IAP) and contribute to ICP elevation.

Patients with pelvis and long bone fractures of lower limbs have a higher risk of DVT. These fractures may cause pain and limitation of mobilisation. Management wise, they are best stabilised as soon as possible, preferably with external devices, to reduce any further haemodynamic impairment and to allow early mobilisation.