Nutrition in Traumatic Brain Injury

Rowan Clemente, ICU Dietitian
North Bristol NHS Trust, UK

Approximately one million people visit accident and emergency each year as a consequence of a traumatic brain injury (TBI). Eighty-five per cent of these injuries are classified as minor, 10% moderate and 5% severe. Men are 2-3 times more likely to suffer a TBI, and in the age range of 15 to 29 years this increase fivefold.[1] However, the numbers of women requiring hospitalisation following TBI are rapidly rising.

The main causes of TBI:

• Road traffic collisions • Falls • Assaults • Injuries at work • Injuries during sport or leisure.

The term TBI encompasses not only the initial injury, but also the secondary injury cascade that occurs as a consequence of the primary injury. The primary injury is usually the result of a direct injury to the brain from a blunt force coming into contact with the head or penetration of an object into the skull and/or brain tissue. This type of injury can cause permanent brain damage due to direct tissue damage. **Table One** displays some of the most common classifications of this type of injury. The primary injury is often difficult to treat due to the speed at which the injury and resultant damage occurs. For the majority of cases the management of the primary injury is focused on treating intercranial hypertension which occurs due to the mass effect of a haemorrhage or obstruction in cerebrospinal fluid outflow. A useful way to think about this is that the skull effectively acts like a box that the brain sits within, this is usually a useful feature as the brain is protected during common bumps and trips. However, should damage occur to the brain itself, the bleeding and or swelling that occurs as a consequence of this damage has very little space to evolve, thus presses onto the brain itself causing further damage. Rapid neurosurgery to evacuate the haemotoma, haemorrhage, or to site extra ventricular drains, aims to reduce the pressure and try to prevent or minimise the damage that can occur. In contrast, secondary injury is caused by the natural inflammatory cascade, observed as brain oedema, brain cell metabolic dysfunction and, ultimately, cell death.

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Nutritional management

The majority of patients with a TBI admitted to the intensive care unit (ICU) will be held in a drug induced coma whilst the primary injury and secondary injury cascade is assessed and managed.

Energy requirements

TBI patients are some of the most hypercatabolic and hypermetabolic seen within the ICU setting, with authors quoting resting energy expenditure to be anywhere between 40 and 200% of the basal metabolic rate.[3] The degree of hypermetabolism is proportional to severity of injury and motor dysfunction[4] and is likely to persists for several weeks,[5] with some authors suggesting it is present for up to a year post injury.[6] This leads to significant inter and intra individual variations in energy requirements, which makes their estimation difficult. Many factors will interplay to affect the energy requirements of patients with a TBI and these need to be accounted and adjusted for when estimating nutritional requirements. **Table Two** contains some of the common factors that will increase and decrease a patient's energy expenditure, which need to be taken into consideration when estimating requirements.

The use of predictive equations can provide a useful guide to a patient's energy requirements, but it is important to assess an individual's requirements on a continuous basis, paying close attention to their current clinical condition and treatment programme.

Protein requirements

Due to the hypercatabolic state a TBI induces, patients with a TBI will experience high nitrogen losses; it is recognised that during this period protein losses can only be minimised and not prevented. A protein intake of 1.2-1.5 g/kg/d is commonly recommended.[7, 8] Although, more recently, some authors have increased the upper end of requirements – 1.5-2.5 g/kg/d[9] 2-2.5 g/kg/d.[10] As with energy requirements, the patients clinical condition and co-existence of sepsis, other trauma and organ failure will affect overall requirements.

Fluid and electrolytes

Careful monitoring of fluid and electrolytes are required post a TBI due to the important role they play in maintaining cerebral prefusion pressure and intracranial pressure. However, disturbances are regularly observed, many of the management techniques to prevent cerebral oedema involve dieresis, which can leave the patients vulnerable to developing hypomagnesaemia, hypophosphataemia and hypokalaemia.[11] Hypomagnesaemia, in particular, has been associated with a poorer outcome, therefore, serum magnesium levels are frequently checked and supplemented as indicated.[12] It is not uncommon for patients with TBI to experience difficulties with their sodium balance, this is generally attributable to either syndrome of inappropriate anti-diuretic hormone (SIADH), cerebral salt wasting, or diabetes insipidus. The clinical features of these and their nutritional management are described in **Table three**.

Table One: Common Head Injury Classifications

DAI	Diffuse axonal injury
EDH	Extradural haematoma
SDH	Subdural haematoma
SAH	Subarachnoid haemorrhage
ICH	Intracerebral haemorrhage
Contusions	
Concussion	

Table Two: Factors Which Increase and Decrease Energy Expenditure in TBI Patients[2]

Factors that will decrease energy expenditure

- Loss of body weight
- Sedation
- Paralysis – due to neuromuscular blocking agents

Factors that will increase energy expenditure

- Mode of ventilation/suctioning
- Spasticity
- Pyrexia/sepsis
- Activity or agitation
- Cerebral storming

Timing and route of feeding

Early initiation of enteral feeding (e.g. 24-48 hours) is thought to prompt many benefits from reduced length of stay,[10] reduced morbidity[13, 14, 15] and improved neurological outcome at three months.[16] A multicentre study by Härtl *et al.*[14] considered 797 patients from 22 different USA trauma centres and concluded delaying feeding by 5-7 days in severe TBI patients had a 2-4 fold increase likelihood of death respectively. In the first five days the amount of nutrition received was inversely associated with mortality – every 10 kcal/kg reduction in calorie intake was associated with 30-40% increase in mortality. However, enteral feeding in these patients is often complicated by delayed gastric emptying. It is suggested that gastroparesis occurs in about 80% of TBI patients with a Glasgow Coma Scale <12,[17] and this is observed to persist for longer than in other groups of trauma patients.[18] Several features of a TBI are thought to have a role in the development of gastroparesis, including sedation, neuromuscular blockade, metabolic state and raised intercarnial pressure. Intravenous metroclopramide is often prescribed as first line management of gastroparesis, proceeding to nasojejunal feeding in centres that have this option available to them, or parenteral nutrition when this fails.

The onward journey

ICU is often just the beginning of a very long recovery period for these patients. Many will experience long inpatients stays as their medical conditions stabilise before extended periods in a rehabilitation environment. It is interestingly to note that, at admission, 94% of patients will receive some form of enteral nutrition,[10] but at six months 92% will receive their food orally, with 84% having gained nutritional independence.[19] This progression seems to encapsulate these patients well in terms of the fact they need regular ongoing dietetic input to ensure adequate and appropriate nutrition.

Table Three: Common Sodium Disturbances Seen in TBI

	Clinical Features	Nutritional Management
SIADH (syndrome of inappropriate anti-diuretic hormone [ADH])	• Increased levels of ADH, causing the body to hold on to urine • Serum Na <13 • Urinary Na elevated >25 mmol • Serum osmolarity <280 Osm/l	• Fluid restriction • High energy low volume feeds
Cerebral salt wasting	• Excessive renal losses of Na • Serum Na <134 • Urinary Na high • Serum osmolarity – hypo-osmolar	• Slow IV saline replacement • Don't restrict fluid
Diabetes insipidus	• Lack of ADH, causing the body to produce lots of urine • Serum Na >155 • Urinary Na low • Serum osmolarity – hyper-osmolar	• Use of low sodium feeds if serum Na exceeds 155-160 mmol/l

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