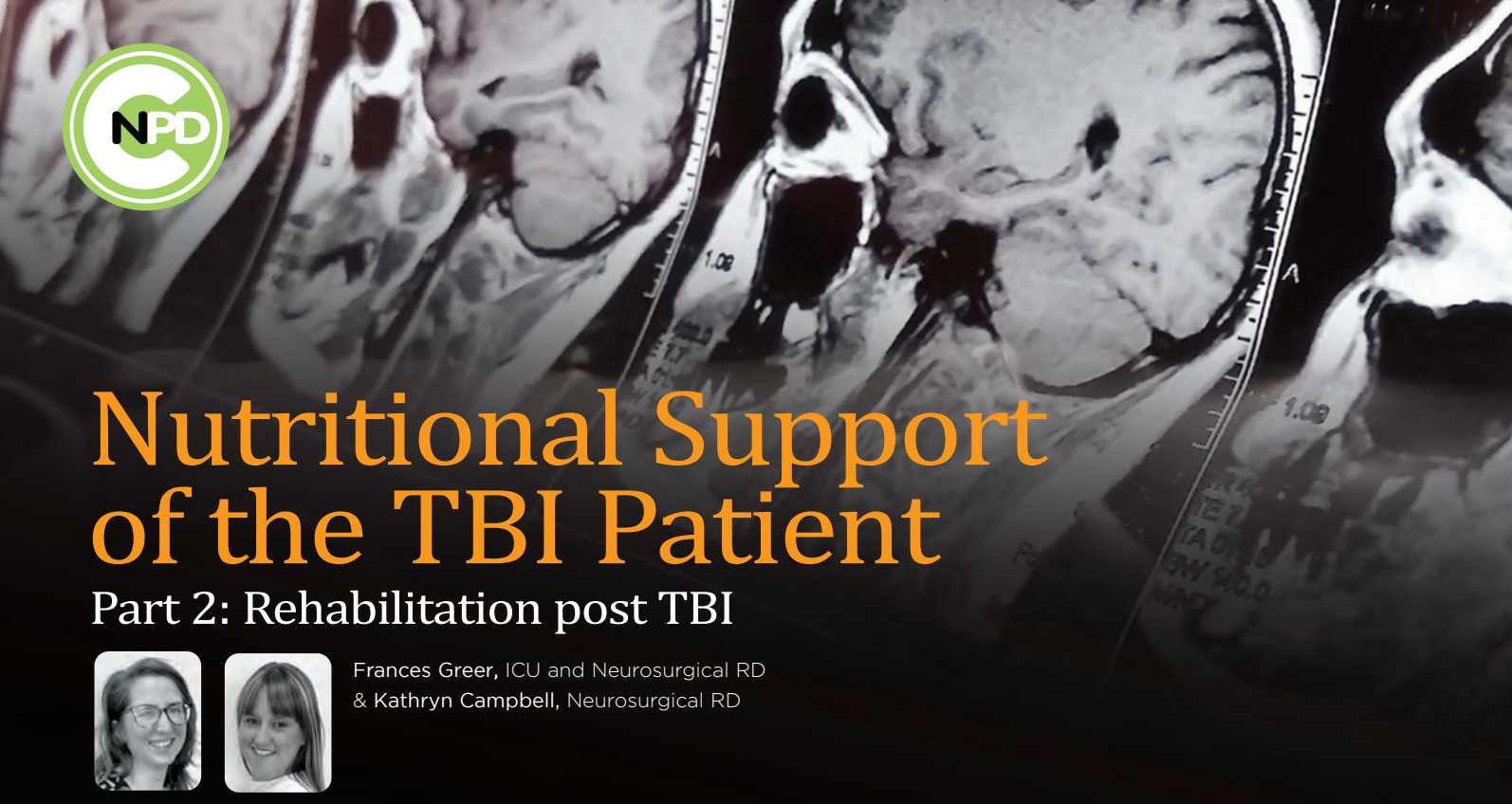

Nutritional Support of the TBI Patient

Part 2: Rehabilitation post TBI

Frances Greer, ICU and Neurosurgical RD
& **Kathryn Campbell,** Neurosurgical RD

Rehabilitation post traumatic brain injury (TBI) aims to support individuals to achieve the best physical and psychological outcome post injury. Whilst the brain is not able to repair itself, adaptation can occur post TBI to allow for some function to be restored. The aim of rehabilitation is to support the development of new nerve pathways to undamaged cells of the brain.[1]

During the initial stages of injury, it can be difficult to predict the extent of the TBI and how long the rehabilitation process will take. Rehabilitation typically begins in the acute setting and continues in the community, either in specialist neuro- rehab centres or in patients own homes. Whilst many patients will require neuro-specialist rehabilitation for months, or even years, post injury, it is known that the most significant improvements will be seen within the first 6 months.[1] During the rehabilitation journey there are many nutritional factors to be considered. These can vary significantly depending on severity of injury and stage of rehabilitation journey.

Acute rehabilitation

The start of the rehab journey begins once medically stabilised following the initial injury. For most patients, specialised neuro-rehabilitation begins after ICU discharge. Crucially, this means that many patients will be starting their rehab journey whilst still in a hypermetabolic state. As discussed in Part 1 of this series, a TBI is known to be hypermetabolic and can persist for at least 30 days post injury,[2] with some estimates suggesting ongoing hypermetabolism for up to 12 months, and hypercatabolism for 75 days.[2] Therefore, it is likely that significant lean mass loss has already occurred before rehabilitation can begin.[4]

Therefore, in practice, a comprehensive baseline nutritional assessment post ICU is important to establish the extent of lean mass loss and to support restoration where possible. However, there are many barriers to nutritional assessment at this stage of acute rehabilitation. It can be difficult to achieve accurate weight information as often patients will be immobile and unable to make use of standing scales on acute wards. Seated scales may be inappropriate if patients need specialised seats due to postural instability/abnormal muscle tone, and the use of hoists and hoist scales are contraindicated for those patients with an extra ventricular drain (EVD) in situ. These challenges can persist throughout rehab with reports of up to 66% of TBI patients not being weighed for the first 6 months of their rehabilitation.[5] The use of handgrip dynamometer is often inappropriate due to reduced alertness and impaired cognition, especially at the beginning of the rehabilitation journey. The use of skinfold callipers, mid-upper arm circumference (MUAC) or calf circumference measurements can be helpful, provided the patient is compliant. There is limited evidence for use of specific equations to calculate requirements in the acute and rehab setting, so clinical judgement is advised.

In addition to the expected significant weight loss, the beginning of rehabilitation is often fraught with feeding difficulties due to dysphagia and impaired cognition. The incidence of dysphagia in TBI patients post ICU can be high, with some estimates ranging from 25-93%.[6] Therefore, it is not surprising that many TBI patients will require enteral feeding, particularly at the beginning of their rehabilitation journey. Enteral feeding may be required to meet full nutritional requirements if dysphagia is severe or for supplementary feeding if oral intake is inadequate. This can be common post TBI due to high distractibility, agitation and post-traumatic amnesia (PTA).[6] As a result, the oral intake of food can be poor, with two small studies (n=84) suggesting median energy and protein intakes during admission post TBI were 52-83% and 41-75% respectively.[7,8]

Swallowing assessments and texture modified diets

Evaluation of swallow generally begins with a screening and/or bedside swallow assessment by a speech and language therapist (SALT). This may then, if appropriate, be followed with instrumental investigations – fibreoptic endoscopic evaluation (FEEs) and videofluroscopy swallowing study (VFSS) – which provide direct visualisation of the anatomy and physiology whilst swallowing. The goals of the swallowing assessment are to determine the optimal methods to support adequate nutrition and hydration whilst reducing any associated risks.[9] These investigations can help progress the patient onto an oral diet, which may initially include a texture modified diet +/- thickened fluids as described by the International Dysphagia Diet Standardisation Initiative (IDDSI) Framework (www.iddsi.org/framework). As well as generally lower nutritional content of these meals due to the addition of extra fluids to form the correct consistency,[10] the tolerance of texture modified meals can be variable. In practice, patients can often find the texture unpalatable and fail to take in sufficient amounts to warrant the cessation of enteral feeding. Patients with impaired cognition can also provide challenges to increasing oral intake due to decreased attention, increased agitation and short-term memory deficits.[11] In this instance, intervention, such as overnight/bolus feeding and supplementation with nutritionally complete food and drinks, can help increase oral intake and aid the transfer to a fully oral diet. Given the difficulties of impaired metabolism, dysphagia and impaired cognition, it is not surprising that rates of malnutrition at the beginning of TBI rehabilitation journey are high, with 35-68% of TBI patients considered to be at high risk of malnutrition according to 'Malnutrition Universal Screening Tool' ('MUST') scores or equivalent.[12, 13]

To PEG or not to PEG

Of severe TBI patients that require prolonged enteral feeding, 37% remain on a restricted diet after 18 weeks of inpatient rehab.[6] The chances of returning to an unrestricted diet depends on the severity of the brain injury and can be predicted by factors including the Glasgow Coma Scale (GCS) score and functional oral intake at admission.[14] As discussed within Part 1 of this series, early enteral feeding is initiated via a nasogastric (NG) tube when appropriate post incident, but one of the barriers to this can be inadvertent tube loss. This can often be attributed to agitation and confusion; characteristic behaviours seen in this patient cohort. Continual tube dislodgement can lead to inadequate nutritional intake and early PEG (percutaneous endoscopic gastrostomy) insertions. Practices to reduce accidental tube loss include the use of mittens, but these can be very uncomfortable and frustrating, particularly for cognitively impaired patients, and the use of a 24 hour one-to-one care, which can prove costly. There is increasing evidence that nasal bridles are effective at preventing tube dislodgement. A meta-analysis of five studies by Bechtold *et al.* reported dislodged tubes in 14% of the patients in the nasal bridle group compared to 40% in the adhesive tape group, and as a result nasal bridles are now a common method to secure NG tubes.[15] A study by Lynch *et al.* concluded that nasal bridles may avoid PEG placement in patients with mild to moderate dysphagia.[16]

There are varying views on the most appropriate timing of PEG insertion following TBI. A recent retrospective review of TBI patients requiring PEG in an American trauma centre found that low admission GCS score and type of TBI had no impact on if a PEG was required.[17] This study reviewed 332 patients with severe TBI and found that of the 64 patients who required a PEG, they also required significantly more neurosurgical interventions (59% *vs* 26%) and tracheostomy (93.8% *vs* 3.7%).[17] Based on these findings, the authors' recommend that patients who are likely to be on a ventilator for a prolonged period should be considered for a PEG. However, it is also worth noting that the average time to PEG placement in this group was 9.42 days post admission, which compared to practice in the UK, is very early. There are limited guidelines pertaining to TBI patients available across Europe and the UK. The National Institute for Health and Care Excellence (NICE) guidelines advise gastrostomy feeding to be considered in people likely to need long-term (4 weeks or more) enteral tube feeding and in the acute setting; patients suffering with dysphagia should have an initial 2-4-week trial of nasogastric enteral tube feeding.[18] Predicting recovery in patients with severe TBI remains challenging, as patients can have different patterns of injury and the evolution of these can be variable.[17] It can be difficult to know when to place a PEG as no two patients present in the same way. ESPEN guidelines state that, as a general rule, PEG feeding should be considered if it is expected that the patient's nutritional intake is likely to be qualitatively or quantitatively inadequate for a period exceeding 2-3 weeks.[19] Prior to the insertion of an enteral feeding tube, each case should be considered on its own merits, taking into account the clinical situation, diagnosis, prognosis, ethical issues, the expected effect on the patient's quality of life and the patient's own wishes.[19] There is limited evidence regarding the benefits and risks associated with early versus late PEG insertion. It is standard practice within our hospital to be guided by the patient's potential clinical recovery and to liaise regularly with SALT regarding a patient's potential swallow improvements.

Electrolyte imbalance

Hyponatraemia can be a common electrolyte disturbance after TBI, and the cause can be attributed to syndrome of inappropriate antidiuretic hormone secretion (SIADH), cerebral salt wasting or diabetes insipidus. These conditions are usually a direct consequence of TBI and can be a short- or medium-term problem that can be managed with fluid electrolyte manipulation. Their clinical features and nutritional management are described in **Table 1**.

Table 1: Common sodium disturbances after TBI

	Clinical features	Nutritional management
SIADH	• Increased levels of ADH resulting in reduced urination • Lower serum sodium levels	• Fluid restriction • High energy, low volume feed
Cerebral salt wasting	• Excessive renal losses of Na • Lower serum sodium levels	• Slow IV saline replacement • No fluid restriction
Diabetes Insipidus	• Low levels of ADH, resulting in increased urine production • High serum sodium levels	• Low sodium feed if serum sodium > 155-160 mmol/l

"Hyponatraemia can be a common electrolyte disturbance after TBI, and the cause can be attributed to syndrome of inappropriate antidiuretic hormone secretion (SIADH), cerebral salt wasting or diabetes insipidus"

In practice, hypercalcaemia is a rare consequence of TBI, with unknown incidence reported in literature. The mechanism for this is still not fully understood, but it likely due to rapid bone turnover, following severe TBI and corresponding long-term immobilisation. This is mainly managed medically, but dietetic treatment consists of increased provisions of fluid. Antidotally, often in excess of 3 litres of fluid is needed to help manage this.

Community rehabilitation

Following the management of the acute issues discussed above, patients become suitable for hospital discharge and specialist community neuro rehabilitation. Typically, this occurs several weeks to months post injury. At this stage in their rehab, weight loss should have plateaued for most patients. Several studies have a described weight stability at 2-3 months post injury, with most patients experiencing a slow weight gain towards baseline pre-morbid weight.[12, 20]

In fact, for many patients excess weight gain during rehabilitation is a greater problem. Several European studies have found that 42-90% of TBI patients experienced weight gain, leading to a body mass index defined as overweight or obese after 12 months in rehabilitation.[21, 22, 23] One possible explanation for weight gain is that during inpatient rehabilitation TBI patients are encouraged to have autonomy with food choices. As a result, there are reports of low intake of fruit and vegetables (i.e. less than 3 portions a day) and high intake of foods high in fat and sugar.[23] Other factors contributing to weight gain include reduced mobility, impaired ability to exercise, disinhibited behaviours relating to food and emotional eating.[21] Elevated weight places acquired brain injury patients at increased risk of developing chronic conditions, such as diabetes, hypertension and cardiovascular disease.[24]

Furthermore, there have been reports of increased risk of sarcopenia, particularly sarcopenic obesity in TBI patients,[23] which has been demonstrated to adversely affect outcomes and quality of life.[25] Provision of high protein, healthy menu options and weight management/exercise plans may be needed to support patients who are experiencing unhealthy weight gain.

Vitamin D deficiency is another common problem during rehabilitation, with some estimates as high as 80% in TBI patients.[26] Particularly for severe TBI injuries, many patients will have been a hospital inpatient for several months, with limited exposure to sunlight, making them high risk for deficiency. Routine screening and supplementation should be considered, as deficiency is associated with impaired cognitive function and depression symptoms.[26]

Conclusion

The rehabilitation journey can be long for many TBI patients and can be fraught with many nutritional issues. At the start of rehabilitation, often significant weight and lean mass loss post ICU is common. However, with increasing time, most patients will regain independence with eating and reliance on enteral nutrition will decline. For those patients who require long-term feeding, the decision around PEG placement timing can be complex.

For many patients, weight gain during community rehab can be common and strategies to minimise excessive weight gain should be considered. An increasing awareness should be given to risk of sarcopenic obesity and vitamin D deficiency in long stay rehabilitation patients.

References: **1.** Headway (2007). Rehabilitation after brain injury. Accessed online: www.headway.org.uk/media/4001/rehabilitation-after-brain-injury-e-booklet.pdf (Sep 2022). **2.** Krakau K, *et al.* (2006). Metabolism and nutrition in patients with moderate and severe traumatic brain injury: a systematic review. Brain Inj.; 20(4): 345-367. **3.** Foley N, *et al.* (2008). Hypermetabolism following moderate to severe traumatic acute brain injury: A systematic review. J Neurotrauma.; 25(12): 1415-1431. **4.** Kurtz P, Rocha EEM. (2020). Nutrition Therapy, Glucose Control, and Brain Metabolism in Traumatic Brain Injury: A Multimodal Monitoring Approach. Front Neurosci.; 14: 190. **5.** Krakau K, *et al.* (2010). Resources and routines for nutritional assessment of patients with severe traumatic brain injury. Scand J Caring Sci.; 24(1): 3-13. **6.** Lee WK, *et al.* (2016). Characteristics of Dysphagia in Severe Traumatic Brain Injury Patients: A Comparison with Stroke Patients. Ann Rehabil Med.; 40(3): 432-439. **7.** Chapple L, *et al.* (2017) Longitudinal changes in anthropometrics and impact on self-reported physical function after traumatic brain injury. Critical Care Resusc.; 19(1): 29-36. **8.** Abdullah MI, *et al.* (2020) Determination of calorie and protein intake among acute and sub-acute traumatic brain injury patients. Chin J Traumatol.; 23(5): 290-294. **9.** Thibault R, *et al.* (2021) ESPEN guidelines on hospital nutrition. Clin Nutr.; 40(12): 5684-5709. **10.** Wright L, *et al.* (2005). Comparison of energy and protein intakes of older people consuming a texture modified diet compared with a normal diet. J Hum Nutr Diet.; 18(3): 213-219. **11.** Mackay LE, Morgan AS, Bernstein BA. (1999). Factors affecting oral feeding with severe traumatic brain injury. J Head Trauma Rehabil.; 14(5): 435-447. **12.** Krakau K, *et al.* (2007) Nutritional treatment of patients with severe traumatic brain injury during the first six months after injury. Nutrition. 23(4): 308-317. **13.** Campbell G, *et al.* (2015) Does the nutritional status of Acquired Brain Injury patients on admission to rehabilitation affect their functional outcome at discharge? Clin Nutr ESPEN.; 10(5): E198. **14.** Hansen TS, Engberg AW, Larsen K. (2008). Functional oral intake and time to reach unrestricted dieting for patients with traumatic brain injury. Arch Phys Med Rehabil.; 89(8): 1556-1562. **15.** Bechtold ML, *et al.* (2014) Nasal bridles for securing nasoenteric tubes: a meta-analysis. Nutr Clin Pract.; 29(5): 667-671. **16.** Lynch A, *et al.* (2018) A systematic review of the effectiveness and complications of using nasal bridles to secure nasoenteral feeding tubes. AJO.; 1(1): 8. **17.** Rendel R, *et al.* (2022) Gastrostomy Versus Non-gastrostomy Enteral Access for Severe Traumatic Brain Injury. Am Surg.; 88(8): 1940-1942. **18.** NICE (2006). Nutrition support for adults: oral nutrition support, enteral tube feeding and parenteral nutrition. Accessed online: www.nice.org.uk/guidance/cg32 (Sept 2022). **19.** Loser C, *et al.* (2005) ESPEN guidelines on artificial enteral nutrition-percutaneous endoscopic gastrostomy (PEG). Clin Nutr.; 24(5): 848-861. **20.** Chapple L, *et al.* (2016) Energy and protein deficits throughout hospitalization in patients admitted with a traumatic brain injury. Clin Nutr.; 35(6): 1315-1322. **21.** Crenn P, *et al.* (2014) Changes in weight after traumatic brain in-jury in adult patients: A longitudinal study. Clin Nutr.; 33(2): 348-353. **22.** Odgaard, L, *et al.* (2020) Using clinical quality databases to monitor the quality of fundamental care: Example with weight status after severe traumatic brain injury. J Clin Nurs.; 29(11-12): 2031-2038. **23.** Vazquez-Lorente H, *et al.* (2022) Need of nutritional assessment and monitoring in a population with acquired brain injury: an analytical cross-sectional study. Nutr Neurosci.; 1-10. **24.** Driver S, *et al.* (2012) Creating an effective physical activity-based health promotion programme for adults with a brain injury. Brain Inj.; 26(12): 1482-1492. **25.** Wahlen B, *et al.* (2020) Impact of sarcopenia in trauma and surgical patient population: A literature review. Asian J Surg.; 43(6): 647-653. **26.** Jamall OA, *et al.* (2016). Prevalence and correlates of vitamin D deficiency in adults after traumatic brain injury. Clin Endocrinol.; 85(4): 636-644.