REVIEW ARTICLE

Nutrition in the neurocritical care unit

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Abstract

The aim of intensive care is to support the physiology of the body till the treatment or the reparative process of the body kicks in to the rescue. Maintaining an adequate nutrition during this period is of vital importance to counteract the catabolic effect of the critical disease process. The guidelines for nutritional care in the neuro intensive care unit (ICU) are sparse. This article collates the current evidence and best practice recommendations as applicable to the critically ill patient in the neuro ICU. The use of screening tests to identify patients at a risk of malnutrition and related complications is presently recommended for all patients with an emphasis on early initiation of caloric support. Over-aggressive feeding in an attempt to revert the catabolic effects of critical illness have not proven beneficial, just as the attempts to improve patient outcomes by altering the routes of nutrition administration. Special patient population such as traumatic brain injury, stroke, subarachnoid haemorrhage or spinal cord injury may have varying nutritional requirements; individualised approach in the neurocritical ICU with the help of the intensivist, nutritionist and pharmacology team may be of benefit.

Key words: Neurointensive care, nutrition, nutrition screening tools

INTRODUCTION

Critical illness renders a patient prone to malnutrition. Factors seen commonly in the intensive care unit (ICU)—loss of appetite, nausea, vomiting, altered sensorium, difficulty in swallowing, gastroparesis and mechanical ventilation exacerbate the effects of starvation. The energy deficit of critical illness has a more pronounced effect than natural fasting. It is accompanied by endocrine and inflammatory stress response to the underlying disease process resulting in immunosuppression, generalised weakness and greater morbidity. Malnutrition in critically ill patients has been linked to increased morbidity and mortality.[1-3]

The main purpose of nutritional therapy during critical illness is to assess, detect, prevent and treat malnutrition (pre-existing or ongoing) and related complications—these may exist in up to 40% of the critically ill population, more so in special subgroups.[2] Nutritional supplementation in patients with acute critical illness (ACI), prolonged acute critical illness (PACI) and chronic critical illness (CCI) has been well-studied in general critical care patients; target-oriented nutritional support protocols have shown clear benefits.[3] A patient in the neurocritical care unit is, in addition, prone to have a prolonged in-hospital and post-hospital recovery from critical illness phase (RCI) in which the risks of overnutrition-related obesity and consequent adverse effects increase.[4,5]

Data and recommendations for nutritional care in the neuro ICU is sparse, except perhaps in traumatic brain injury patients. The aim here is to present an evidence based account of the current practise and recommendations for nutritional therapy with emphasis on the common types of patients encountered in the neuro ICU. A search for literature was made in PubMed, Embase and Google scholar data bases. The search terms included 'nutrition', 'feeding', 'enteral', 'parenteral', 'neurocritical care', 'traumatic brain injury (TBI)', 'stroke', 'spinal cord injury (SCI)' and other specific neurocritical care-related diagnoses. Where good quality recommendations were not

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available for neurocritical care patients, general ICU patient data was referred to.

THE METABOLIC RESPONSE TO INJURY: WHERE IT ALL BEGINS

Critical illness is characterised by a specific injury or insult progressing to involve various organ systems. This relationship between tissue injury and whole-body metabolic changes was first described by Cuthbertson.[6] He suggested that the stress response of the body to an injury was an adaption necessary for the subsequent recovery of the critically ill physiology. This response consists of 'ebb' and the 'flow' phases characterised by a period of hypoperfusion, followed by post-resuscitation hypermetabolism. A period of anabolism follows this hypercatabolic phase and may last for months [Figure 1].[7]

The changes associated with this stress response affect all the organ systems of the body [Table 1]. The quality, quantity and time frame of the stress response may vary depending on the severity of insult, type of insult and the baseline patient characteristics. The clinician needs to recognise and direct diagnostic and treatment interventions depending on the course of this stress response pattern.

Based on Figure 1, current evidence indicates a provision of nutrition to the critical care population tailored to the phase of injury. Patients at a risk of malnutrition may be optimized prior to an elective surgery, with enteral (EN) or parenteral (PN) as tolerated; arginine supplementation may reduce infections and length of hospital stay. Early acute phase of critical illness may benefit from reduced non-protein calories, and greater (upto 2 gm/kg/day) protein supplementation. Measurements of early sepsis and critical care energy expenditures have shown decreased energy spent as

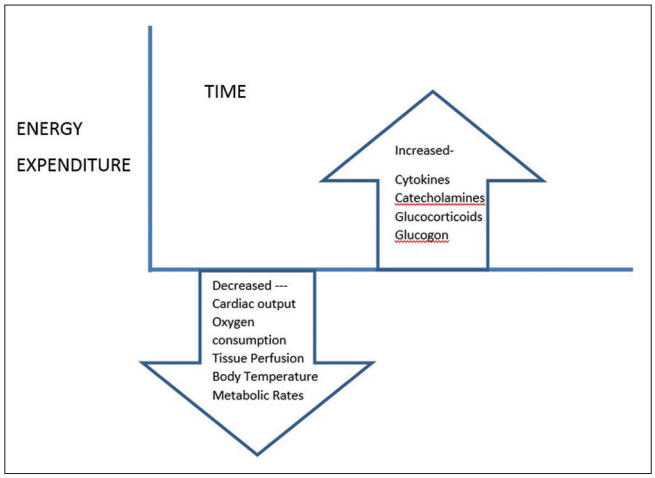

Figure 1: Biphasic metabolic response to tissue injury

the disease severity increases; this goes on to increase exponentially in the chronic or recovery phase of critical illness. In this phase, increased non-protein calories with adequate amount of proteins should be supplemented. The role of growth hormone, anabolic steroids and aggressive physical therapy in the chronic phase of critical illness is still under research.[8]

SCREENING FOR RISK OF MALNUTRITION IN THE ICU

A screening tool is used for rapid and simple evaluation of patients at risk of malnutrition. Various screening tools have been validated in the ICU. Most of these tools use the patients height (or a surrogate like length of ulna), weight at admission, history of recent acute weight loss and acuteness of disease or serum albumin as factors for calculating risk of malnutrition [Table 2]. Traditional screening tools albeit good as research tools are beneficial in improving awareness but do not uniformly identify patients at risk. The time spent in collecting data manually may not justify their routine use in the ICU.[9,10] We have compared two common screening tools in elderly critically ill patients in Eastern India—both the geriatric nutrition risk index (GNRI) and malnutrition universal screening tool (MUST) showed a high sensitivity. MUST was more practical to use due to a higher specificity and greater applicability (lesser missing values).[11] Screening for the risk of malnutrition should be followed by a systematic assessment of the nutritional status. This involves measurements of various indices: anthropometric measurements like fat and muscle mass, laboratory values like serum albumin, transferrin and body composition like by bioelectrical impedance analysis. The magnitude of fluid electrolyte shifts in critically ill patients renders these measurements redundant, however.

Recently the nutrition risk in the crtitically ill (NUTRIC) tool has been described for ICU patients- developed specifically for the critically ill patient; it incorporates age, Acute Physiology And Chronic Health Evaluation (APACHE) II score, Sequential Organ Failure Assessment (SOFA) score, number of co-morbidities, days of hospital stay prior to ICU admission and interleukin 6 (IL6) levels. It is used to quantify the risk of patients developing adverse events that may be modified by aggressive nutritional therapy.[12]

Nutrition assessment will usually place the patient in one among these groups—over-nourished or obese, well-nourished, under-nourished or severely undernourished. Once done, management guidelines or local policy may be used to develop care plans suited to the particular ICU and patient population.

Table 1: Effect of critical illness physiology on various organ systems of the body

System involved	Physiology	Pathology	End organ effect
Neurologic	Increased substrate metabolism including amino acids	Increased levels of aromatic amino acids; decreased global cerebral function	Encephalopathy of critical illness-delirium, coma
		Hyperdynamic circulation	Myocardial injury, oedema
Cardiovascular	Increased oxygen requirements and delivery		
Fluids and electrolytes	Increased body water expansion of extracellular compartment with contraction of intracellular space	Deranged electrolyte balance	Hypokalemia, hypomagnesemia and hyperphosphatemia during recovery of stress response
	Catabolic state Inflammation	Increased oxygen consumption and carbon dioxide production; increased capillary permeability	Tachypnea, respiratory failure, ARDS
Pulmonary	Increased protein turnover	Atrophic villi	Gastroparesis
	Inflammation, Peripheral vasodilation	Organ oedema	Bowel obstruction
	Incompletely understood	Renal hypoperfusion	Oliguria
			AKI
Gastrointestinal	Upregulation of catabolic; downregulation of anabolic hormone signalling	Suppressed cell mediated immunity	Increased susceptibility to infection
Renal	Increased energy production, gluconeogenesis, lipolysis	Increase Cathecholamine, glucagon, and cortisol	Stress hyperglycemia, sick euthyroid syndrome, altered sleep wake cycle
Immunologic	Triglyceride reesterification	Decrease GH pulsatility and quantity, thyroxine peripheral low levels	Weight gain, mostly water
	Protein catabolism	Increased production of acute phase proteins, mobilisation and recomposition of fat stores	Loss of lean body mass
Endocrine			
Nutrition			

ASSESSMENT

Caloric requirement, time of initiation and the EN vs PN debate

How is caloric requirement estimated?

Intensivists and nutritionists have traditionally used predictive equations to estimate a patient's basal energy expenditure (BEE), and to set caloric target for patients. These equations may be derived originally from normal volunteers such as the Harris-Benedict equation (HBE) and Mifflin St Jeor or from hospitalised patients such as the Ireton–Jones equation. Based on static variables, these equations do not account for the dynamic changes in the condition of the patient, making them unreliable.[13] None of the equations have been validated in the neuro ICU. A recent comparison of 23 predictive equations and 11 fixed weight-based factors such as 25 calories/kg/day) showed an overall accuracy of only 40% (predicted energy expenditure from equations within 10% of measured resting energy expenditure (REE) by indirect calorimetry).[14]

Indirect calorimetry, *done repeatedly* over a period of ICU stay may estimate energy expenditure more accurately. It is important to note that using extrapolated values from a singular indirect calorimetry may result in calculations that are more erroneous than using a predictive equation.[13]

The newer emerging strategies of nutritional therapy in the ICU emphasize on identifying patients already malnourished or at risk for developing malnutrition and in setting a daily caloric goal for them. This helps to recognize the (not uncommon) patient in the neuro ICU (stroke, spinal cord injury and amyotrophic lateral sclerosis) who may be hypometabolic and in avoiding the stress of over-feeding in them. Some patient groups at specially high risk of having unpredictable caloric and metabolic requirements who will benefit most from

such a time and energy intensive tailored therapy are the elderly, obese, difficult to wean, children and those previously malnourished.[13-16]

The healthcare professional should ensure that the total estimated nutrient support includes appropriate quantities of energy, protein, fluid, electrolyte, mineral, micronutrients and fibre. This has to take into account the patients' stage in the disease [Figure 1], gastrointestinal tolerance, likely duration of nutritional support and the risk of developing re-feeding syndrome.[17]

As an example, an average build middle-aged patient admitted to the ICU after a road trauma injury may need 25–30 K Cal/kg/day. This will include calories from the carbohydrates and proteins provided in the EN or PN formula. Extra calories from intravenous fluids or propofol infusion (1.1 Kcal/ml) must be accounted for to prevent over-feeding. Proteins (complex or as amino acids, depending on the patient's tolerance) should be administered at 0.8–1.5 gm/kg/day. In haemodynamic stable patients, fibres may be added to the blenderised hospital diet provided in most Indian ICUs or a commercially available EN feed may be chosen which will provide 1 Kcal/ml of feed absorbed. Total fluid of 30–35 ml/kg/day and replacement of vitamins, minerals and trace elements have to be done especially in patients on prolonged formula feeds. An example for calculating or advising PN feed for the same patient is illustrated in Table 3.

Table 2: Malnutrition screening tools used in the intensive care unit population

Screening test	History	Clinical data
Subjective global assessment[1]	Weight change over 2 weeks-6 months	Subcutaneous fat
	Change in diet intake (amount and type)	Muscle wasting
	Gastrointestinal symptoms	Oedema
	Functional capacity (change in)	Ascitis
Malnutrition universal screening tool[2]	Unplanned weight loss over 3-6 months	Body mass index
		Acute disease effect
Nutritional risk screening (2002)[3]	Acute weight loss in 3-6 months	Body mass index
	Reduced dietary intake in last week	Severe illness (e. g., ICU)
Mini nutrition assessment[4]	Food intake	Body mass index
	Weight loss	Mobility

1-Detsky *et al.* JPEN, 1984, 2-BAPEN, 3-Kondrup *et al.* Clin Nutr 2003, 4-Vellas *et al.* Nutrition 1999. ICU: Intensive care unit population

Table 3: Initiating parenteral nutrition in the neuro intensive care unit

Steps for initiating parenteral feed in the ICU
Reconsider the indication for choosing PN over EN
Decide on total caloric goals (ICU protocol based, HB equation or indirect calorimetry)
Consider risk of refeeding syndrome (malnourished, chronic alcoholic, starving for last 4-5 days)-if present initiate at 50% of daily caloric requirement, with attention to serum electrolytes, minerals and vitamins
Consider comorbidities like diabetes mellitus, renal or hepatic failure
Calculations to order PN
25% dextrose 1000 ml=250 gm dextrose=850 kcal; (1 gm dextrose=3.4 kcal)
20% lipid 200 ml=40 gm lipid=400 kcal; (1 gm lipid emulsion with glycerol=10 kcal approximately)
Protein requirements (1-2 gm/kg body weight) in gm×4=protein calories (1 gm protein=4 Kcal)
Osmolarity (mosm/litre)=dextrose/litre (gm)×5+proteins/litre (gm)×10+350
For example, for a 'normal' patient 70 kg body weight with no comorbidity to supply approximately 1750 non-protein calories and 70 gm protein (25 kcal/kg and 1.0 gm protein/kg) in 2500 ml fluid
1500 ml 25% dextrose=1275 kcal
250 ml/day 20% lipid=500 kcal
Total=1775 non-protein kcal and 1750 ml fluids
10% AA 100 ml=10 gm protein; 700 ml=70 gm
Osmolarity of this prescription if given as a mixture=250×5+10×10+350=1700 mosm/l

ICU: Intensive care unit; PN: Parenteral Nutrition; EN: Enteral nutrition; HB: Harris- Benedict

To administer as estimated or not?

The benefit of providing calories as per the BEE (estimated either from equations or the indirect calorimetry) has itself been debated. The data which had demonstrated the adverse effects of energy debt in the ICU population was mainly from observational studies,[18,19] and has been challenged by studies which show either no harm or benefit from intentional under-feeding.[20-22]

The landmark Enteral Nutrition in Adult Critically Ill Patients (EPaNIC) trial has led to renewed discussion about the adequacy of calories and proteins that should be supplemented to the critically ill patient. In the largest such multicentre study of critical care nutrition in 4,640 patients, Caesar *et al*., showed that matching caloric requirements to BEE by supplementing EN with parental feeds in patients who could not meet caloric goals by EN feeds alone, led to worse outcomes in terms of time to recovery and complications.[23]

The newer critical care nutrition pundits emphasize on 'sufficient' feeding for each patient, with greater focus on identifying and treating those at risk for malnutrition (either under-nourished or hypometabolic at risk of over-feeding).

Blood sugar control-how tight is tight enough?

As the debate about tight sugar control is slowly settling down for patients in the general critical care units after the landmark Normoglycemia in Intensive Care Evaluation and Surviving Using Glucose Algorithm Regulation (NICE SUGAR) trial[24] and other studies with similar results, neurointensivists are asking if the same results can be extrapolated to their patients.

A systematic review and meta-analysis was conducted by Kramer *et al*., to answer this question. Sixteen randomised controlled trials (RCTs) (1,248 neurocritical care patients with TBI, SCI, stroke, infection and anoxic encephalopathy) were included.[25] The investigators found no mortality benefit of intensive glucose control; hypoglycemia episodes were higher in the intensive sugar control group. Tight sugar control improved neurologic outcome in patients with TBI, intracranial haemorrhage (ICH) and subarachnoid haemorrhage (SAH) but not in ischemic stroke—a finding similar to the lack of benefit of glucose-potassium-insulin (GKI) infusions seen in ischemic stroke patients.[26] This improvement in outcome was apparent when tight control was compared with a lose control (Random blood sugar > 200 mg%); benefit was lost when compared to more intermediate sugar levels (110–180 mg%). The authors concluded that although some benefits cannot be excluded among special subgroups of patients in the neurocritical care unit, intensive sugar control increases the risk of hypoglycaemia greatly and does not carry any mortality benefit against a moderate sugar control strategy of 110–180 mg%. Blood sugar levels > 200 mg% in the neuro ICU should be avoided.

Enteral or Parenteral?

The current evidence-based teaching in this regards is to 'Use the gut where possible and as soon as possible'. The absence of bowel sounds has no predictive value regarding the extent of ileus or the success (or failure) of EN feeding.

Studies addressing EN versus PN in the general ICU (including traumatic brain injury patients in the neuro ICU) have undergone meta-analyses.[27-29] Based on the results, we know that EN is associated with fewer infectious complications, greater feasibility and lower costs than PN. Both EN and PN may have associated complications, which the caregiver must be vigilant for [Table 4].

When to feed the patient? Timing of initiation of EN or PN

EN or PN, 'Earlier, the better'.

The timing of feeding initiation may be of greater importance than the route and it affects outcome; early initiation of EN (by 24 to 48 hours) has been shown to reduce mortality in trauma patients.[30]

Doig *et al*., started early PN in 1,372 patients who were expected to have late initiation of EN due to various indications.[31] They reported no differences in infections, adverse events or 60-day mortality attributed to PN between the groups. The PN group had lesser days of mechanical ventilation (ICU and length of stay (LOS) in hospital were similar) and reported better quality of life at 60 days.

Can the patient be fed orally? Which type of EN tube?

Patients in the neuro ICU are more prone to swallowing difficulties requiring long term placement of EN tubes. Dysphagia has been associated with increased morbidity and mortality in neurocritical care population and is the most important cause resulting in malnutrition after ischemic stroke.[32,33] The neuro ICU staff must identify patients at risk of dysphagia and initiate EN tube feeding in them. It is as important to decide when to remove the EN tube and institute oral feeds—this would avoid complications of aspiration pneumonia (early removal) or sinusitis, strictures, tracheooesophageal fistulae, etc., (prolonged placement). Elaborate swallow tests done by experienced staff and video fluoroscopic swallow studies have been described for detection of dysphagia; simple bedside algorithms using National Institutes of Health Stroke Scale (NIHSS) and Glasgow Coma Scale (GCS) scores may also be used with good success.[34,35]

Table 4: Complications of enteral and parenteral feeding

Problems with enteral feeding
Tube related
Insertion problem: Misplaced into respiratory tract, trauma; rarely peritonitis, colonic perforation or necrotising fasciitis with PEG insertion
Accidental removal
Ulceration, tissue necrosis any point along the pathway of the tube
Gastrointestinal complications
Nausea, bloating
Diarrhoea/altered bowel habits
Gastric colonisation and infection
Aspiration
Metabolic problems
Hyperglycaemia
Re-feeding syndrome
Overhydration
Electrolyte imbalance
Mechanical
Problems with parenteral feeding
Insertion of central venous access: Pneumothorax, vascular/neural injury, arrhythmias; venous thrombosis
Infectious
Sepsis at the site of CVC
Metabolic
Early-volume overload, hyperglycaemia, re-feeding syndrome, hypokalaemia, hypophosphatemia, hypomagnesaemia, hyperchloremic acidosis, fluid electrolyte imbalance
Late-deficiency of vitamins, minerals, trace elements. Steatosis, hepatic cholestasis and acute pancreatitis. Bone demineralisation, thrombocytopenia

PEG: Percutaneous endoscopic gastrostomy, CVC: Central venous catheter

Studies have shown no significant benefits of feeding via the nasojejunal route or via percutaneous endoscopic gastrostomy (PEG) as compared to the simpler and cost effective nasogastric route in the general and neuro ICUs.[36,37]

Residual volumes—how much is too much?

Monitoring residual volumes during EN feeding is a traditional practise based on low-grade evidence (suggesting higher residuals may cause aspiration pneumonia). Recent evidence suggests that not monitoring residual volumes does not increase the risk of ventilator associated pneumonia (VAP) and may result in achieving higher caloric goals as compared to routine residual volume measurement.[38]

Immune enhancing diets

Diseases encountered in the neurocritical care setting including ischemic and hemorrhagic stroke and traumatic brain injury may increase oxidative stress and the production of oxidizing reactive species—decreasing serum levels of endogenous antioxidants.[39] It has been suggested that this oxidative stress may be countered by restoring endogenous antioxidants and supplementing with exogenous nutrients known to have antioxidant properties.[40]

The term immunonutrients includes glutamine, arginine, omega-3 fatty acids, vitamins and nucleotides. Uniform recommendations are lacking for all neurocritical care patients; the critically ill trauma population has been studied extensively, however, and immunity enhancing diet have resulted in decreased infectious complications and reduced length of hospital stay.[40,41]

Dietary Management in Special Circumstances

Renal failure

Patients with acute or chronic renal failure who are not on dialysis, will need special nutrition tailored to their urine output, serum urea and electrolyte levels. Renal feeds are specially formulated to be calorie dense (up to 2 kcal/ml), lower in protein, potassium, magnesium and phosphorus. In patients undergoing renal replacement therapy (RRT), especially continuous, venovenous haemodialysis, renal formulae are not always necessary as protein requirements are higher and fluid restriction is not needed. If serum electrolytes are normal, patients on RRT may continue on a standard high-protein diet.[42]

Liver failure

Patients with liver failure who are not clearly in hepatic encephalopathy do not need protein or calorie restriction; protein intake may be decrease to 0.6gm/kg/day if encephalopathy is present. The evidence for branched chain amino acids (BCAA), calorie or lipid restriction in this group of patients is not clear.

Acute respiratory distress syndrome and acute respiratory failure

Excess calories than required may cause increased production of carbon dioxide production from calories being supplemented. It is recommended to continue a balanced diet (without preferentially decreasing carbohydrates or increasing lipids) but decrease total calories to 80% to prior load in these patients. Feeding with diet enriched in omega-3 fatty acids may improve respiratory function and decrease length of stay in the ICU.[43]

Specific Patient Population in the Neuro ICU

Traumatic brain injury

The hypermetabolic response generated in TBI matches that of burns (20–40% of body surface area) and septic

shock. Excessive protein catabolism and negative nitrogen balance ensue. The adverse effects of hyperglycaemia in brain injury are well-described and blood glucose should be carefully controlled in these patients. There are significant knowledge gaps about monitoring nutritional status and response to nutritional interventions in acute brain injury.[44]

Hartl *et al*., conducted a multicentre study recruiting 797 patients of severe TBI in 22 centres. They found that both the timing of initiation of EN feeds and the calories delivered affected the outcomes in TBI patients (controlling for other factors affecting mortality). A delay of 5 and 7 days increased the mortality 2 and 4 fold, respectively. Every 10 kcal/kg decrease in intake increased mortality by 30–40%.[45]

Ketones can provide energy more efficiently to the brain than glucose. Scientists have associated carbohydrate free diet to result in lower lactate levels and better nitrogen balance. Although well-demonstrated in animal models, only small human trials justify the use of ketone diets or other special diets like zinc-enhanced nutrition in TBI.[46-49]

Cerebrovascular disease

Ischemic stroke: Nutritional support among patients with ischemic stroke has been well studied and robust evidence is available from the Feed or ordinary food (FOOD) trial. Dennis *et al*., performed three linked trials; two enrolled stroke patients with dysphagia—one evaluated for difference in early feeding or no feeding within 7 days and the other evaluated the benefits of nasogastric feeding over PEG feeding, if any. The randomised controlled multicentre study enrolled 859 patients in the first trial and concluded that a survival benefit of early EN feeding may exist in stroke patients, but is statistically insignificant and might be at the expense of outcome (greater survival with poor outcomes). PEG feeding in the second trial was found not superior to nasogastric feeding among the 321 patients studied.[50]

The third part of the multicentre FOOD trial enrolled over 4,000 stroke patients (14% recruitment from Indian hospitals) who were able to swallow and randomised them into two groups—those receiving normal hospital diet or a protein calorie supplemented diet.[51] It found no mortality benefit of routine administration of supplementation. Of note here, however, would be the fact that only 8% of their patient population was undernourished at the time of being enrolled for the study.

Non-traumatic intracranial haemorrhage: In the neurocritical care patients with subarachnoid haemorrhage, energy requirements are increased in the early post-bleed stage. The requirements increase over time: Throughout this period the HBE underestimates BEE calculations as compared with indirect calorimetry (IC).[52] Recent brain microdialysis studies have shown that after subarachnoid haemorrhage, over time, the interstitial milieu becomes hyperglycolytic and cerebral glucose consumption increases. Enteral nutrition and insulin administration to control plasma glucose levels may increase metabolic stress in these patients by causing brain interstitial hypoglycaemia and increase in lactate and pyruvate levels.[53] Optimisation of nutritional support and blood glucose control after SAH requires further research.

Spinal cord injury

As follows any severe trauma, energy expenditure in patients with SCI increases markedly after injury. Factors such as muscle paralysis and steroid administration cause nitrogen loss in the urine: The degree of loss increasing with the severity and level of injury. The negative nitrogen balance starts at one week post injury, peaks at 3 weeks and may last for 7 weeks despite the provision of sufficient quantities of proteins and calories.

In the chronic phase of SCI, hypotonicity and disuse atrophy of muscles, decreased lean body mass and increased bone resorption all result in decreased BEE and electrolyte imbalance. Over-feeding may result in predominantly central or abdominal obesity and its associated complications.[54] Two thirds of SCI patients might be malnourished—under-nourished or obese, by the time they reach the rehabilitation centre from the neuro ICUs. A well-planned nutrition therapy is necessary for these patients.

Other special patient groups and nutritional considerations in the neuro ICU

Patients with Guillain Barre syndrome have a prolonged period of hospitalisation and may spend an equally long time in rehabilitation. Mechanical ventilation and infections cause hypercatabolic response offsetting the reduced metabolic demands of the generalised muscle weakness. Isolated recommendations suggest high-energy (40 to 45 non-protein kcal/kg), high-protein (2.0 to 2.5 g/kg) nutrition support for these patients. In the absence of better evidence, a balanced approach is suggested, based on guidelines for general critical care patients.[55,56] Ketogenic diet has proved beneficial in terminating seizures in patients with refractory status epilepticus.

The possibility of pre-existing starvation in some patients (which may result in the constellation of electrolyte abnormalities and organ dysfunction known as the 're-feeding syndrome') must be kept in mind before initiating nutrition support in the neuro ICU.[57]

The risk of the neuro ICU patient acquiring Clostridium difficile infection and diarrhoea have been found to be lower than the general ICU population.[58] It is recommended to continue EN feeds as tolerated in these patients, unless ileus, toxic megacolon or colonic perforation occur. A high index of suspicion, rapid toxin analysis of the stool samples, prompt patient isolation and treatment are required.[59]

Neurocritical care and the chronically critically ill patient

A subgroup of patients (up to 10%) admitted to the ICU land in a chronic state of illness characterised by re-admissions, prolonged ventilation and multiple episodes of sepsis and shock. Although a unanimous definition of CCI has still not been agreed on, duration of tracheostomy and mechanical ventilation (for more than 21 days) are the defining factors. CCI is typically associated with the presence of inflammation and adult Kwashiorkor-like malnutrition, requiring active metabolic and nutritional assessment cum intervention. This subgroup of patients represent a great burden at the individual, ICU and societal level.[5]

Recent evidence links neurological dysfunction in a critically ill patient with greater risk to becoming CCI.[56] The intensivist in the neuro ICU, therefore must stay highly vigilant and use his clinical acumen for early screening and intervention in the high-risk patient population. High on the list of suggested interventions to avoid or treat the CCI patient is appropriate nutrition care instituted by a multidisciplinary team approach- involving the neurointensivist, the endocrinologist and the dietician or ICU nutritionist.

CONCLUSION

Evidence suggests that identifying those patients who are at a higher risk of malnutrition and paying special attention to their feeding improves outcomes. Early initiation of feeding is more important than the route chosen. Over-feeding and under-feeding are both to be avoided; estimating caloric requirements is best done by repeated indirect calorimetry, but if unavailable, setting daily caloric goals on the basis of the patient's clinical situation may be equally prudent, especially if decided in a multidisciplinary approach. Blood sugars kept < 180 mg% as in the general ICU population is safe. The role of antioxidants and immune modulating diets needs to be studied further before concrete recommendations can be made in the neuro ICU.

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