Monitoring Nutrition and Glucose in Acute Brain Injury

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And the Participants of the International Multi-disciplinary Consensus Conference on Multimodality Monitoring**

Abstract The metabolic response to injury is well described; however, very little is understood about optimal markers to measure this response. This summary will address the current evidence about monitoring nutritional status including blood glucose after acute brain injury (ABI). An electronic literature search was conducted for English language articles describing the testing, utility, and optimal methods to measure nutritional status and blood glucose levels in the neurocritical care population. A total of 45 articles were included in this review. Providing adequate and timely nutritional support can help improve outcome after ABI. However, the optimal content and total nutrition requirements remain unclear. In addition, how best to monitor the nutritional status in ABI is still being elucidated, and at present, there is no validated optimal method to monitor the global response to nutritional support on a day-to-day basis in ABI patients. Nitrogen balance may be monitored to assess the adequacy of caloric intake as it relates to protein energy metabolism, but indirect calorimetry, anthropometric measurement, or serum biomarker requires further validation. The adverse effects of hyperglycemia in ABI are well described, and data indicate that blood glucose should be carefully controlled in critically ill patients. However, the optimal frequency or duration for blood glucose monitoring after ABI remains poorly defined. There are significant knowledge gaps about monitoring nutritional status and response to nutritional interventions in ABI; these need to be addressed and hence few recommendations can be made. The optimal frequency and duration of blood glucose monitoring need further study.

Keywords Nutritional assessment · Nitrogen balance · Energy expenditure · Glucose · Monitoring · Brain injury

The Participants of the International Multi-disciplinary Consensus Conference on Multimodality Monitoring are listed in the Appendix.

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Introduction

Neurological injuries induce a cascade of sympathetic nervous system activation and inflammatory response resulting in a well-described hypermetabolic response [1]. The hypermetabolic response is characterized by protein catabolism and altered gluconeogenesis, weight loss, and disruption of mitochrondrial function in the brain and systemic tissues. Subsequently, increased energy expenditure can have significant impact on substrate utilization and lead to a malnourished state. The primary rational for nutritional therapy is to prevent malnutrition, maintain glucose homeostasis, and prevent the myriad associated complications by providing the appropriate doses of nutrients to meet the calculated or measured needs. Unfortunately, there is sparse data available on optimal methods to monitor nutritional requirements in the neurocritical care setting, limiting the ability to understand the adequacy of nutritional support and minimize the impact of malnourishment.

The adverse effects of hyperglycemia in neurocritical care patients are well described [2]. Altered glucose metabolism is one of the most common clinically observed metabolic abnormalities in the critically ill patient. Serum glucose is frequently elevated and the response to a glucose load demonstrates the problem of insulin resistance commonly seen during acute illness. Elevations in serum glucose levels appear to result primarily from sympathetic activation, inflammation and resultant increased hepatic glucose production rather than decreased glucose utilization. The rate of glucose oxidation in the periphery increases after injury when a stable circulation is maintained; however, the hepatic glucose output increases dramatically. Under the influence of increased concentration of catecholamines, glucagon and cortisol, glucose is synthetized in the liver using lactic acid, pyruvic acid, and amino acid as substrates [3]. Data indicate that blood glucose should be carefully controlled in critically ill patients; however, to what level and how need further clarification [2]. Central to this management is a strategy to accurately assess glucose levels to understand the response to injury and intensive insulin therapy.

Methods

This systematic review was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [4].

Search

Using the PubMed database, a systematic review was performed (1966–August 2013) using the flowing MESH terms: nutrition, nutritional support, nitrogen balance, catabolism, energy expenditure, hypermetabolism, and glucose AND monitoring. We restricted our search to articles in the English and describing critically ill patients with at least one of the following: traumatic brain injury (TBI), SAH, intracranial hemorrhage, ischemic stroke, coma after cardiac arrest, seizures.

Study Selection and Data Collection

The authors independently reviewed citations, abstracts and full-text articles to select eligible studies. Unpublished data or congress presentations/abstracts were not considered. We excluded: a) review articles; b) case reports or case series with ≤5 patients; c) experimental studies; d) study on pediatric ICU populations (<18years); e) studies that were not conducted on ICU patients; f) studies dealing with patients in brain death. Data were abstracted using a predefined abstraction spreadsheet, according to the PICO system.

Review end-points

The end-points of this review were to answer the following questions about monitoring of nutritional status and systemic glucose levels in acute brain injuries (ABI).

Nutritional Status

1. Can measuring energy expenditure with indirect calorimetry be used to monitor the nutritional requirements in neurocritical care patients?
2. What methods are useful when monitoring the response to nutritional interventions?
3. Is there utility in monitoring gastric residuals in patients receiving enteral nutrition?

Blood Glucose

1. Should monitoring of serial blood glucose values be performed routinely during the critical care after acute brain injury?
2. How should glucose monitoring be performed in the acute critical care period after brain injury?
3. What duration of frequent glucose monitoring is required after brain injury?
4. Is there an optimal point of care method to monitor blood glucose levels?

Literature Summary

The search identified 7,747 studies; 5,549 of these were selected because they were studies completed in humans. From this list, 91 articles were found to address the questions proposed in the PICO approach. Ultimately, 45 published articles were used in the analyses. After selection, the evidence was classified and practical recommendations developed according to the GRADE system.

Nutritional Status and Assessment

Although it is recognized that providing adequate, timely nutritional support improves outcomes in neurocritical care populations [5], both optimal content and total nutrition requirements remain unclear. Very few data are available about the appropriate parameters to monitor nutritional status. The majority of published studies investigate

nutritional status after traumatic brain injuries (TBI). Much of this literature also is more pertinent to the rehabilitation phase of injury and not the critical care setting. Consequently, many commonly used strategies to monitor nutrition progress, such as body weight and other anthropormetric measures are not easily applicable in the intensive care unit (ICU) setting since there is concomitant use of sedatives or therapeutics, inflammatory responses and sympathetic activation that directly impact the metabolic rate.

Can Measuring Energy Expenditure with Indirect Calorimetry be used to Monitor the Nutritional Requirements in Neurocritical care Patients?

We were not able to identify any studies that answered this question in our review. Indirect calorimetry (IDC) assesses the CO_2 production and O_2 consumption directly from the patient and using the modified Weir equation, computes the resting energy expenditure (REE) [6]. IDC is the gold standard by which to measure the REE in critically ill patients. Several studies, most in TBI, have used IDC values to determine the REE after brain injury [7]. In four TBI studies, singular REE measurements were used to determine the amount of calories required for experimental nutritional interventions [8–11]. These studies failed to identify an optimal timing, duration, or frequency for REE measurements. In 3 studies in non-TBI brain hemorrhage patients, REE was measured as a marker of hypermetabolism and not to guide nutritional therapy [12–14]. No study has assessed the accuracy of repeated measures of REE during the acute injury to monitor the ongoing nutritional requirements in the neurocritical care patient population.

We identified 4 studies that examined whether any estimating equations adequately assess nutritional requirements acutely after neurological injury? Predictive equations, such as the Harris-Benedict equation, have long been used to determine a patient's predicted energy expenditure to set a caloric target. So-called activity and stress factors are used to improve the equation's accuracy and numerous estimating equations have been developed for specific critical care populations [15]. Predictive equations have been validated to REE but not necessarily to actual nutritional requirements. There are four studies in TBI and one in ischemic stroke that utilized specific predictive equations to test accuracy to REE measurements [16–20]; however none of these studies determined the accuracy of these equations to define nutritional requirements or monitor ongoing level of nutritional support. No studies in other neurocritical care patient populations were identified.

What Methods are Useful when Monitoring the Response to Nutritional Interventions?

Thirteen studies that addressed this question were reviewed. Protein energy metabolism is commonly assessed by measurement of nitrogen balance derived from the difference between daily nitrogen intake and daily nitrogen output. Nitrogen intake is easily calculated from the volume and the composition of all the fluids administered to the patient. Nitrogen loss is measured by collecting urine, feces, and drainage fluids and by determining their nitrogen, or alternatively by only measuring urine urea nitrogen values and using an adjustment factor for nitrogen losses elsewhere. The nitrogen balance is the only biomarker for protein energy metabolism widely reported in the neurocritical care population [8, 9, 16, 18, 21]. RCTs measuring nitrogen balance or the degree of nitrogen loss as a surrogate of outcome have been performed and suggest that less than 50 % of administered nitrogen is retained after TBI [22–24]. In ischemic stroke and non-traumatic brain hemorrhage, nitrogen balance has been shown to be associated with severity of injury and infectious complications [13, 25] but not as a marker of appropriate nutritional therapy. The majority of studies reported nitrogen balance once during the first week of injury. The acceptable amount of nitrogen loss relating it to functional recovery has not been subjected to clinical studies.

There is no validated way to monitor the global response to nutritional support on a day-to-day basis. A short-term biochemical parameter that could reliably monitor response to nutrition within the same timeframe as the nutrition therapy would be very valuable in clinical practice. Both serum albumin and transthyretin levels are widely used markers of nutritional therapy in critical illness [26]. There are no studies investigating the utility of monitoring either albumin or transthyretin as markers of responsiveness to nutritional therapy in the neurocritical care patient population. Body weight is the most commonly used indicator in assessing nutrition status in non-critical care population [26], but in the ICU setting is more likely a marker of fluid balance than of nutritional status and not a reliable measure of responsiveness to nutritional therapy.

Is there utility in monitoring gastric residuals in patients receiving enteral nutrition?

Early enteral nutrition is the standard of care for all mechanically ventilated critically ill patients. However, enteral nutrition often is not adequately delivered because of concern for aspiration and ventilator associated pneumonia (VAP) related to gastrointestinal intolerance, gastroesophageal reflux and regurgitation or vomiting. We

identified one study, a prospective RCT conducted in a mixed critical care population of which 15 % were admitted with primary neurological illnesses that examined the question above. The No reduction in VAP in mechanically ventilated patients who had gastric residuals routinely monitored was observed [27]. In addition patients who did not undergo routine monitoring of gastric residuals were more likely to receive 100 % of their caloric goal. We did not identify any studies that analyzed the utility of monitoring gastric residuals in non-ventilated ABI patients.

Blood Glucose

Recent studies of glycemic control with intensive insulin therapy (IIT) in critical care have resulted in consensus guidelines that recommend relatively higher targets for blood-glucose levels than initially indicated by the original randomized clinical trial (RCT) published in 2001 [28]. Utilizing appropriate modalities to monitor blood glucose levels during IIT is crucial. However, there are very few studies to guide clinicians as to the optimal method to monitor blood glucose levels. Central laboratory blood glucose measurements in plasma are considered the standard since these measures best approximate the physiologic activity of glucose [29]. However, because of convenience and rapidity of results, point-of-care (POC) tests are commonly used to measure blood-glucose concentrations in critically ill patients. Many of these monitors were not designed to monitor patients in the ICU, and as a result may not be a good measure of the physiologic burden of glycemic levels after injury nor sufficiently accurate to guide IIT [30].

Should Monitoring of Serial Blood Glucose Values be Performed Routinely During the Critical care After Acute Brain Injury?

Acute hyperglycemia is common after TBI and other neurologic disorders. Serum glucose concentrations often are increased at admission to hospital and for many days of intensive care. The increase in serum glucose is thought to result from a generalized increase in sympathetic catecholamine tone. The extent of hyperglycemia at admission is correlated with the severity of injury [31, 32] and with poor outcome after TBI [31, 33, 34], with glucose values >300 mg/dl (16.6 mmol/L) uniformly associated with fatal TBI in one study [34]. In particular admission hypergblycemia is associated with poor prognosis [34, 35]. However the relationship of of delayed hyperglycemia >200 mg/dL (11.1 mmol/L) with poor outcome is less certain [32]. Experimentally, immediate but not persistent

post-traumatic hyperglycemia may exacerbate final lesion volume, but this is not well studied in humans. Furthermore it is unclear whether one-time or persistent hyperglycemia has a greater negative impact on brain injury. The association between hyperglycemia and poor outcome also is observed in ischemic stroke, intracerebral hemorrhage and subarachnoid hemorrhage [36–38]. Despite these repeated associations, it is not clear whether hyperglycemia is an independent factor or a marker of disease severity.

Hyperglycemia is thought to worsen outcome after ABI through downstream metabolic effects. It is generally thought that hyperglycemia will result in increased anaerobic metabolism and lactate production under conditions of brain ischemia. Post-traumatic brain ischemia, which is thought to be a common phenomenon in the hours after TBI, potentially could be made worse by hyperglycemia. Two recent reports support this theory. In the first, Diaz-Parejo et al. [39] demonstrated increased extracellular lactate during episodes of profound hyperglycemia, with serum glucose >15 mmol/L (270 mg/dL), but not with moderate hyperglycemia in which serum glucose was 12–15 mmol/L (216–270 mg/dL). In the second study, Zygun et al. [40] confirmed that profound hyperglycemia is associated with worsened tissue acidosis in TBI. In addition, hyperglycemia is associated with other adverse effects in critically ill patients, such as increased infection rates, poor wound healing, and increased mortality. Thus, the preponderance of evidence suggests that hyperglycemia has deleterious effects after ABI and hence there is value to its assessment and monitoring.

However there are no studies that specifically address with what frequency or for how long should blood glucose be monitored after brain injury. The majority of studies of the impact of serum glucose on outcome have monitored serum glucose or blood capillary levels at a frequency of every 1–8 h [31]. The frequency of monitoring is driven by IIT protocols. In prospective and retrospective studies of IIT, the usual frequency is every 1–2 h. There is no evidence to suggest a specific frequency for monitoring that is a more reliable marker of the physiologic impact of blood glucose levels after brain injury. Beyond admission, the majority of studies have indicated that glucose levels for the next 48 h and through the period of critical care remain important predictors of long term outcome [31]. However, the duration of critical care is different across studies, and therefore optimal duration cannot be determined.

How Should Glucose Monitoring be Performed in the Acute Critical care Period After Brain Injury?

Common methods used to measure blood glucose include arterial measurements from arterial blood gas analyzers

(ABGs) or glucose meters that use either arterial or capillary blood. There are no studies specifically performed in neurocritical care populations; however, we identified 13 studies prospective and retrospective studies in general critical care populations that examine point of care testing to set glycemic targets. In summary these studies demonstrate blood-glucose monitoring with ABG analyzers tend to be more accurate than that by glucose meters with arterial blood. Additional studies have noted greater overall accuracy with arterial blood samples versus capillary blood samples for blood-glucose measurements in adult critically ill patient [41–53]. It is important to note that the accuracy of blood-glucose measurements is variable regardless of the type of device, indicating that all POC devices require routine calibration with central laboratory glucose measurements to ensure continued accuracy. Finally, seven studies noted that POC testing of any kind is less accurate during episodes of hypoglycemia or hemodynamic shock [42, 45–50].

Limitations to the Literature

Although studies have outlined the importance of early, adequate nutritional support and blood glucose monitoring after brain injury [5, 44], there is a paucity of studies assessing the natural history of the metabolic response to acute neurological injury. Developing metabolic profiles of different types of neurological injury will better inform the overall approach towards overall nutritional support and targeted blood glucose monitoring. This will necessitate the use of newer analytical techniques that allow for better description of the response to injury at the cellular level. Any new nutritional biomarker that is developed will need to be validated in a clinical setting where there is a significant interaction between inflammation and the sympathetic activation and the metabolic response. This area of monitoring also is ideally suitable to understand the effects of substrates and substrate utilization on gene expression, and should represent an area for future investigation. Finally, there is a need for indicators of nutrition progress that can be monitored sequentially, where the change in the indicator over time is predictive of an improved nutritional therapy related outcome. The most commonly utilized methods, indirect calorimetry and nitrogen balance, have predominantly been studied as singular assessments of physiological state, rather than as markers of responsiveness to nutritional therapies. This may be due to the inability of these markers to change significantly within the timeframe of a short-term response to nutrition therapy. This lack of sensitivity renders current monitoring techniques useless when assessing whether the nutrition provided is optimal.

With glucose monitoring, further work is needed to improve the accuracy of POC tests and to better define the duration of close monitoring that coincide with the greatest risk for secondary injury. An area of interest is determining the relationship between blood and interstitial brain glucose levels. Future investigations in nutritional and blood glucose monitoring should evaluate the impact the systemic inflammatory response and/or therapies may directly impact metabolism, such as fever, targeted temperature modulation or sedation, into the assessment of the nutritional requirements of ABI patients.

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