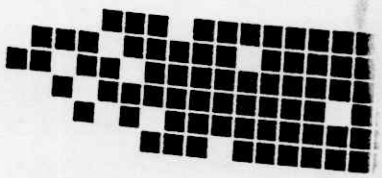
...igh-Protein Nutritional Support



In this article the authors describe nutritional and metabolic support for the postinjury critically ill patient. Some of the therapeutic modalities used are new and have not been studied extensively, and their effect on morbidity and mortality has not yet been demonstrated. Nevertheless, the authors believe that sufficient data justify their use in the selected, critically ill population they treat in the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems (MIEMSS). A detailed description of the nutritional protocol with comments on the monitoring techniques is presented, followed by three clinical examples. The differences between the metabolism of starvation and metabolism of trauma or sepsis are examined and the rationale of the therapeutic choices is explained. The final section consists of comments on the particular clinical situation of brain injury. The discussion has been focused intentionally on protein metabolism and quantity of amino acid (AA) infusion. Physicians at the MIEMSS Shock Trauma Center commonly administer parenteral and enteral formulas containing more than 2.5 gm protein/kg body weight/day.

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Routine Nutrition Assessment

Nutritional assessment of the critically ill trauma patient is a multidisciplinary, dynamic, step-by-step process requiring frequent monitoring and changing of the nutrition prescription throughout the hospitalization.

Screening

The first step in any nutrition assessment program is to identify those patients requiring nutritional support. To do this, the medical records are reviewed and the following noted:

- Diagnosis
- Nutritional support regimen (Patients who are eating will be followed with different protocols that are not discussed in this article.)
- Height/weight (Weight cannot be measured accurately most of the time because of the presence of casts, external fixators, or other devices. The patient may also be edematous after fluid resuscitation and "third-spacing." An estimate of the premorbid weight obtained from the family or by direct observation is used instead.)

The second step in screening is to obtain a thorough medical history.

Assessment

For the patients at risk for the acute malnutrition of trauma or those in whom chronic malnutrition is detected, the following are obtained:

- Metabolic profile: serum glucose, electrolyte, phosphorus, magnesium, cholesterol, triglyceride, and albumin levels and liver function test results
- Respiratory and cardiovascular parameters: oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory quotient (RQ), cardiac output, and serum lactate level
- In-depth injury list and list of procedures and surgeries
- Medication list: narcotics, sedatives, paralytics, inotropes, antibiotics, insulin, and steroids

The nutritional prescription is then formulated as follows:

- Estimate energy requirements (kcal per day) using one of the following methods:
  1. Harris-Benedict equation (HBE)* × 1.2 – 1.3
     (a) Use actual body weight (ABW) for normal body weight individuals.
     (b) Use ideal body weight (IBW) for ABW <0.9 × IBW.
     (c) Use (actual + ideal)/2 for ABW >1.2 × IBW.
  2. $\dot{V}O_2$ ml O_2/min × 7 ($\dot{V}O_2$ from Fick method, using cardiac outputs determined with thermodilution technique from a pulmonary artery catheter); used only to verify predicted requirements because only four measurements are obtained in 24 hours.
  3. Breath-by-breath indirect calorimetry; this is the preferred method when available (see below).
- Estimate protein requirements. Initially, we give 1.5 to 2.5 gm protein/kg/day based on injuries, level of stress, and renal function. Then the amount of protein is adjusted according to nitrogen losses.

*Men: 66 + (13.7 × Wt) + (5 × Ht) – (6.8 × A); women: 655 + (9.6 × Wt) + (1.8 × Ht) – (4.7 × A), where Wt = Weight in kilograms, Ht = height in centimeters, A = age in years.

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- Determine the dextrose to lipid ratio:
  1. Administer 4-5 mg glucose/kg/min (maximum).
  2. Administer 50% to 75% of calories as glucose, 25% to 50% as fat depending on patient tolerance of the two substrates.
  3. Choose between tube feeding (TF) and total parenteral nutrition (TPN). TF is the method of choice, but often it is not feasible:
     (a) If a transpyloric tube has been placed, we use it. If not, placement is recommended.
     (b) For gastric feedings, the decision to feed is based on the presence or absence of bowel sounds, gastric output, stools, flatus, and abdominal distension.
     (c) Our goal is to begin nutritional support within 48 hours of injury. TPN is often started first, with TF initiated as soon as possible.

Follow-up

After the initial assessment, frequent (usually daily) monitoring of critically ill patients is required.

- Nutrition orders are checked for accuracy and appropriateness.
- Metabolic profile and indirect calorimetry are checked daily to assess patient tolerance to nutrition regimen and changes in caloric requirements caused by alterations in stress, sepsis, or drugs.
- Gastrointestinal function and tolerance to TF are checked every day. Actual and goal intake are compared, and infusion rate is changed as needed.
- New developments in clinical conditions are checked every day and support is changed as indicated.
- After 7 days of full, unchanged nutrition support:
  1. We obtain 24-hour urine collection for measurement of urinary urea nitrogen (UUN).
  2. We calculate nitrogen (N_2) balance: N_2 balance = N_2 input − N_2 output, where N_2 input = gm protein infused/6.25; N_2 output = UUN + nonurea nitrogen (NUN); NUN = (UUN × 0.25)* + 0 to 5 gm†
  3. If N_2 balance is grossly negative (i.e., more than −3 gm), we increase N_2 load or caloric load to achieve a +3 gm N_2 equilibrium.
  4. We note the serum transferrin and prealbumin levels. Decreased transferrin (normal levels 250 to 300 mg/dl) or prealbumin (normal levels 16 to 30 mg/dl) in severe trauma or sepsis indicates whether reprioritization of protein synthesis has occurred.[1] These changes are predictive of stress level; not necessarily as a marker of malnutrition).
  5. After 7 days of the new regimen, we repeat N_2 balance, transferrin, and prealbumin measurements.
- The patient progresses from TPN to TF to oral feeding as dictated by clinical condition.

Indirect Calorimetry in Trauma

Every critically ill patient admitted to our intensive care unit (ICU) is connected to a light spectrometer that continuously measures O_2 and CO_2 concentration in the expired and inspired gases. These data are then matched with instantaneous flow measured at the end of the endotracheal tube, and the O_2 consumption ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$) are computed.

*Nonurea urinary nitrogen.
†Nonurine nitrogen loss through skin defects, stool, and drainage.

The measurements are performed for a minute at 10-minute intervals or less. This respiratory monitoring system (RMS) potentially can measure gas exchange 250 times in a 24-hour period and give a mean value for 24 hours.[2] Values outside the mean ±3 SD are discarded. Values for $\dot{V}O_2$ and $\dot{V}CO_2$ are used to compute the actual caloric expenditure with the following formula: Energy expenditure (kcal) = (3.9 $\dot{V}O_2$ + 1.1 $\dot{V}CO_2$) × 1.44. The ratio between $\dot{V}CO_2$ and $\dot{V}O_2$ (RQ) varies with the fuel mixture being oxidized.

Stoichiometrically, the RQ of lipid oxidation is 0.7, protein oxidation 0.8, carbohydrate oxidation 1.0, and lipogenesis 8.7. However, in biologic systems all four processes can occur simultaneously; therefore, the RQ derived from gas analysis is a net result of oxidation and storage of all substrates and varies between 0.7 and 1.3.

Monitoring the RQ serially allows the clinician to assess the predominant fuel being oxidized as well as whether a net lipogenesis is occurring. Net lipogenesis is an energy-consuming process and infers a low output of adenosine triphosphate per mole of $\dot{V}O_2$ and $\dot{V}CO_2$; therefore, this state is deleterious in patients with marginal O_2 delivery or CO_2 removal capacity. Another benefit of monitoring RQ is the determination whether infused lipids are being oxidized or simply stored. Routine serial monitoring of RQ is required.

Clinical Examples of Nutritional Support

Case 1

A 30-year-old man was admitted after a motorcycle accident. Injuries included a descending thoracic aorta tear and a splenic laceration. The aortic tear was repaired successfully with an interposition Gore-Tex graft (Gore & Associates Inc., Elkton, Md.). An attempt to coagulate the splenic lacerations with the

argon beam coagulator failed; therefore, the spleen was removed.

Anthropometric data included a height of 173 cm; weight, 73 kg; ideal body weight, 70 kg; HBE calculation, 1725 kcal/day.

On postoperative day 3 the patient was in the ICU maintained with a mechanical ventilator because of adult respiratory distress syndrome (ARDS). TPN consisted of 28.8 gm nitrogen/180 gm protein (2.45 gm/kg) and 2140 nonprotein kcal (HBE × 1.25). A 24-hour urine collection indicated UUN of 22.5 gm; the calculated NUN was 5 gm. N_2 balance was +1.1 gm. Prealbumin and transferrin levels were 12 mg/dl and 124 mg/dl, respectively.

On postoperative day 7, the patient had a septic complication (meningitis); his temperature rose to 40° C (104° F), and his white blood cell count was 32,000 cells/mm^3. A repeated UUN was 32.8 gm; the calculated NUN was 9 gm. N_2 balance was −13 gm. Data from the RMS showed an average caloric consumption of 2200 kcal/day (total) and an RQ of 0.72 (13 readings in 24 hours).

Postoperative day 10: TPN was changed to 38.4 gm nitrogen/240 gm protein (3.3 gm/kg) and 2700 nonprotein kcal (HBE × 1.56).

Postoperative day 12: TPN was changed to 26.4 gm nitrogen/165 gm protein (2.25 gm/kg) and 2500 nonprotein kcal (HBE × 1.45). Protein load was now provided by an AA mixture with 45% as branched-chain amino acids (BCAA). Their use was justified by the septic state. The overall protein load was reduced as a result of fluid constraints. Data from the RMS showed an average caloric expenditure of 1933 kcal/day and an RQ of 0.7 (41 readings over 24 hours).

A gastrointestinal bleed on postoperative day 13 prevented use of the gut for enteral feedings until postoperative day 19.

Postoperative day 16: Nonprotein calories were decreased to 2225 kcal/day.

Postoperative day 19: A 24-hour urine collection showed UUN of 30.65 gm; calculated NUN was 8.5 gm. N_2 balance was −11.95 gm. Tube feeding with high protein content (83 gm/L) was started, while TPN continued.

Postoperative day 21: TPN was discontinued and protein module enriched tube feedings were advanced to supply 2340 kcal and 41 gm N_2 per day.

Postoperative day 53: The patient's endotracheal tube was removed, and a soft diet started.

Case 2

A previously healthy 44-year-old man was admitted in shock after a stab wound to the chest. The wound extended from the right to the left anterior axillary lines. A transverse thoracotomy was performed and a right lung and hilar laceration was repaired.

Anthropometric data included a height of 183 cm; weight, 107 kg; ideal body weight, 87 kg; estimated lean body mass, 97 kg; HBE calculation, 1860 kcal.

Postoperative day 4: The postoperative course was complicated by ARDS. TPN consisted of 27 gm nitrogen/170 gm protein (1.75 gm/kg) and 1775 nonprotein kcal (HBE × 0.95). A 24-hour urine collection indicated UUN of 21 gm; calculated NUN was 5.2 gm. N_2 balance was −0.8 gm.

Postoperative day 8: The diagnosis of right lung abscess was made by computed tomography scan and clinical data. TPN and TF supplied 260 gm protein (2.7 gm/kg), 2780 nonprotein kcal (HBE × 1.5), and 41.5 gm nitrogen. Tube feeding was not tolerated because of high gastric residuals and therefore was discontinued. The actual N_2 intake was 16 gm for 24 hours. A 24-hour urine collection showed UUN of 32 gm; calculated NUN was 8.0 gm. N_2 balance was −24 gm.

Postoperative day 15: TPN consisted of 41 gm nitrogen/255 gm protein (2.6 gm/kg) and 2520 nonprotein kcal (HBE × 1.35). A 24-hour urine collection assessed UUN at 38.4 gm; calculated NUN was 12 gm. N_2 balance was −10 gm. Data from the RMS showed an average caloric expenditure of 2447 kcal/day (total) and RQ = 0.71 (80 readings over 24 hours).

Postoperative day 21: The patient was clinically improved. TPN consisted of 44 gm nitrogen/275 gm protein (2.8 gm/kg) and 2830 nonprotein kcal (HBE × 1.52). The 24-hour urine collection indicated UUN of 27.9 gm; calculated NUN was 9 gm. N_2 balance was +6.5 gm. Average caloric expenditure was 2594 kcal/day (total) and RQ = 0.72 (138 readings in 24 hours), according to the RMS.

Postoperative day 31: The patient was maintained with tube feeding (three quarter-strength high-protein tube feeding plus nine scoops of protein module per liter), supplying 220 gm protein and 2340 kcal. A 24-hour urine collection showed UUN of 27 gm; calculated NUN was 8.75 gm. N_2 balance was +1.3 gm.

Postoperative day 84: The patient was discharged to a rehabilitation facility and was able to eat a regular diet.

Case 3

A 26-year-old man was admitted after an assault in which he sustained a left open parietal skull fracture with severe brain injury. The patient was immediately taken to the operating room for debridement and closure of the wound. The admission Glasgow Coma Scale score was 3/15.

Anthropometric data included a height of 185 cm; weight, 77 kg; IBW = 77 kg; HBE = 1875 kcal/day.

Postoperative day 2: The patient was not receiving any nutrition. A 24-hour urine collection indicated UUN of 22 gm; calculated NUN was 5.5 gm. N_2 balance was −27.5 gm.

Postoperative day 3: The diagnoses of pneumonia and ARDS were made.

Postoperative day 13: TPN consisted of 30.5 gm nitrogen/190 gm

protein (2.45 gm/kg) and 2630 nonprotein kcal (HBE × 1.4). The 24-hour urine collection indicated UUN of 45.4 gm; calculated NUN was 12 gm. N_2 balance was −26.9 gm. RMS data demonstrated RQ = 0.7 and actual energy expenditure as 2213 kcal/day (102 readings in 24 hours).

Postoperative day 14: Prealbumin = 13 mg/dl; transferrin = 168 mg/dl.

Postoperative day 19: Prealbumin = 14 mg/dl; transferrin = 150 mg/dl.

Postoperative day 20: The patient was still septic by clinical and laboratory data (i.e., positive blood cultures). TPN consisted of 40.8 gm nitrogen/255 gm protein (3.3 gm/kg) and 2445 nonprotein kcal (HBE × 1.3). A 24-hour urine collection assessed UUN at 42.2 gm; calculated NUN was 12 gm. N_2 balance was −13.7 gm. Prealbumin = 13 mg/dl; transferrin = 140 mg/dl.

Postoperative day 24: TPN consisted of 43 gm nitrogen/270 gm protein (3.5 gm/kg) and 2445 nonprotein kcal (HBE × 1.3). A 24-hour urine collection indicated UUN of 36.5 gm; calculated NUN was 8.75 gm. N_2 balance was −4.3 gm.

Postoperative day 30: Tube feeding was started, which the patient did not tolerate. Total nitrogen intake was 17 gm/24 hr. A 24-hour urine collection showed UUN of 35.2 gm; calculated NUN was 8.75 gm. N_2 balance was −27 gm.

Postoperative day 33: The same TPN as postoperative day 24 was restarted. A 24-hour urine collection indicated UUN of 27 gm; calculated NUN was 8.75 gm. N_2 balance was +7.65 gm. The prealbumin concentration was 10 mg/dl and transferrin, 106 mg/dl.

Postoperative day 79: The patient was tolerating tube feeding. Total intake was 14.7 gm of nitrogen. A 24-hour urine collection assessed UUN at 9.35 gm; calculated NUN was 4.35 gm. N_2 balance was +1 gm. The prealbumin level was 25 gm/dl; the transferrin level was 156 mg/dl. The patient was discharged to a rehabilitation facility and was maintained with gastrostomy tube feeding.

Discussion

The first two patients had no brain injury but a complicated hospital course. Initially, their nitrogen balance was positive or only slightly negative while they received approximately 2.5 gm protein/kg/day. With development of sepsis or ARDS, the same amount of protein was not sufficient to maintain a positive nitrogen balance. Clearly, the elevated excretion of nitrogen was not a result of an inadequate amount of calories, as demonstrated by the indirect calorimetry data. Basal energy expenditure predicted by the HBE plus 25% (to adjust for increased energy expenditure caused by trauma) was a reasonably good estimate of caloric expenditure in both cases.

Note that an attempt to start tube feeding was made on postoperative day 8 in the second patient. The attempt failed because of high gastric residuals, as often is seen in patients with sepsis.

The third patient had an isolated brain injury. Initial nitrogen balance, determined while the patient was not receiving any nutritional support, revealed a nitrogen loss higher than expected based on published reports. High protein intake and administration of an adequate amount of calories were initiated; however, the UUN increased but the net nitrogen balance was essentially unchanged in comparison with the first measurement. Between the first and second N_2 balance studies, a serious septic complication developed: we could not discern if the increased N_2 losses were caused by the sepsis or the increased metabolism caused by protein load. The amount of protein was then increased above 2.5 gm/kg/day: the gap between nitrogen intake and output first narrowed and finally closed with abatement of the septic process. Total nitrogen excretion reached a plateau on postoperative day 13 and was not increased further by changing the protein load from 190 to 255 gm/day.

Changes in metabolism induced by stress

Patients with trauma or sepsis have completely different metabolism in comparison to healthy individuals after overnight fast. The fasted trauma patient exhibits a markedly increased rate of muscle proteolysis, AA clearance by the liver, glycogenolysis, gluconeogenesis, and hepatic protein synthesis. Reliance on lipid as an energy source is greater in the fasted trauma patient compared with the fasted healthy person.

The hallmark of protein metabolism in stress is mobilization of peripheral (muscle) protein and transport to the central (primarily hepatic) protein pool for acute phase protein synthesis, gluconeogenesis, and ureagenesis.

In *muscle,* net proteolysis occurs as a result of a mild increase in protein synthesis, but the increase in protein catabolism is much greater, leading to rapid erosion of muscle mass, which is often clinically evident. Amino acids are released into the circulation at an increased rate (threefold to fivefold higher than normal). BCAA originating from the muscle are mostly deaminated, oxidized locally, and partly released to the circulation. Alanine and glutamine are released from the muscle in higher concentrations than their proportions in the tissue. Alanine is formed by the amination of pyruvate, glutamine by the amination of α-ketoglutarate. The reactions thus recycle carbohydrate intermediates back to the liver for gluconeogenesis and detoxify the ammonia formed by BCAA oxidation, allowing transport to the liver for ureagenesis.

Alanine and glutamine are the most important substrates for hepatic gluconeogenesis. Glutamine is also the principal energy source for

fibroblasts and lymphocytes. Fat and glucose are oxidized in the muscle at the normal rate, while leucine catabolism is increased threefold.

In the *liver,* on the other hand, AA clearance from the serum is increased fivefold above normal. About half of amino acid uptake in the liver is used for protein synthesis. The rest is used for gluconeogenesis or oxidized.

Protein synthesis is increased overall, with a 2 to 1000 times increase of the secretion of the so-called acute phase reactants (C-reactive protein, α_2-macroglobulin, α_1-antitrypsin, fibrinogen, complement factors, haptoglobin, and ceruloplasmin). Albumin synthesis is decreased by half. Synthesis of transferrin, prealbumin, and other transport proteins is also decreased. This "reprioritization" of synthesis is related to the injury rather than the nutritional status. Thus, the utility of albumin, prealbumin, and transferrin as nutritional markers in stress states is questionable.

Glycogenolysis and gluconeogenesis are both increased, but only the first can be normalized by exogenous glucose infusion at 4 mg/kg/min.

Thus in summary, a massive mobilization of AA occurs in the muscle; most are released in the bloodstream, except for leucine, isoleucine, and valine, which are in part oxidized in the muscle and in part released in the circulation. The liver uses these AA for its increased protein synthesis and for gluconeogenesis. The latter cannot be suppressed even if glucose is given in excess of caloric requirements.

In patients with sepsis, the release of AA from the muscle can be lowered significantly by infusing TPN with adequate calories and approximately 2 gm protein/kg/day.[3] The same effect is not seen with "inadequate TPN." The central pool of some AA such as phenylalanine, tyrosine, methionine, and glycine is significantly larger than normal, although the opposite is true for glutamine and the BCAA. This relationship suggests that central utilization of BCAA and glutamine is increased.[3]

Several authors have found statistically significant differences in serum levels and hepatic clearance of AA in traumatized patients with sepsis versus traumatized patients without sepsis. These differences were also noted between survivors with sepsis versus nonsurvivors with sepsis. Similar differences have been observed in patients with cirrhosis who survive surgery compared with those who die.[4-7] The data available suggest that the uptake of AA from the liver is reduced, early in the course, in patients who are not going to survive.

Hormonal response to trauma and sepsis

Attempts have been made to relate changes in metabolism to the hormonal response to stress. Secretion of glucagon, cortisol, and epinephrine is increased in trauma and sepsis; however, infusing these hormones in normal volunteers at rates that will produce comparable plasma concentrations produces only a modest negative nitrogen balance, hyperinsulinemia, hyperglycemia, and peripheral leukocytosis.

A peptide containing 33 AA, with a molecular weight of approximately 4274 daltons, has been isolated from the serum of patients under variable degrees of stress.[8] This substance has been shown, in vitro, to increase AA release from rat muscle.

Proteolytic activity is higher in patients with trauma or sepsis compared with individuals undergoing "clean" general surgery who have shown only a mild increase compared with normal volunteers.[8]

Rationale for the use of high protein infusion

The hormonal and cytokine changes induced by trauma and sepsis are responsible for the mobilization of skeletal muscle protein and consequent lean body weight loss. Mortality approaches 100% if the loss of lean body mass reaches 40%.

The synthesis of particular protein is essential for wound healing, immunologic response, and tissue repair. Several investigators have demonstrated that a high nitrogen diet accompanied by adequate calories will increase protein synthesis and that maximal synthesis rate is obtained with intakes of 1.5 to 2.5 gm protein/kg/day. Improvement of immunologic parameters has also been demonstrated with high protein intake.[9] Although aggressive protein replacement causes an increase in whole body protein catabolism (WBPC), this apparent increase is probably generated by deamination of exogenous AA rather than increase in muscle proteolysis. High protein loads do stimulate whole body protein synthesis (WBPS), so that the net effect will result in WBPS that matches WBPC, which is the goal of protein replacement in trauma patients.

We administer glucose and fat emulsions to our patients, relying, whenever possible, on indirect caloric measures to guide the level and types of oxidative fuels. This approach avoids complications of overfeeding and maximizes the nitrogen-sparing effect of glucose and fat. If indirect calorimetry is not available, we rely on the HBE with appropriate stress/activity correction factors (usually 1.25 to 1.35 times the basal energy expenditure). Once we are reasonably sure that energy balance has been achieved, we measure N_2 balance. If this value is profoundly negative (more than 3 gm), we increase the protein load and repeat the N_2 balance determination after 7 days. Sometimes we must infuse up to 3 to 3.5 gm protein/kg/day in some patients (see third clinical case). Our routine is to start at 2.5 gm/kg/day in the most severely injured, meet energetic requirements, and then adjust according to the 24-hour urinary urea nitrogen.

Some authors have pointed out that N_2 balance techniques are fraught with errors and are therefore of little value.[10] We believe that, in the absence of a better clinical method, N_2 balance, if done serially, is a reasonable monitoring tool. Our protocol is to continue to increase protein load, accepting the increased N_2 output, as long as the net balance becomes less negative and problems related to high protein intake do not develop. An elevation of the blood urea nitrogen level is common but rarely is of any clinical significance unless the patient has a significant degree of renal dysfunction.

Use of BCAA

On the basis of theoretic and experimental evidence, several authors have suggested the use of BCAA-enriched solutions. The plasma pool of BCAA is decreased in trauma and sepsis, while their utilization is increased in the muscle to meet energy requirements. This situation would cause a relative BCAA deficiency for protein synthesis in the liver, which could be corrected by a BCAA-enriched solution.

Some have also postulated that BCAA, in particular leucine, have a regulatory effect on protein synthesis toward anabolism, which cannot be explained on the basis of their availability as an alternate energy substrate. A recent study (comparing two groups of patients receiving equivalent TPN solutions except for BCAA content) demonstrates better nitrogen retention, elevation of total lymphocyte count, reversal of anergy to skin test, and higher plasma transferrin in the group receiving 50% of protein as BCAA.[9]

Another study confirmed the same finding of better nitrogen retention but failed to demonstrate any positive effect on outcome.[11]

A prospective trial was conducted in our institution.[12] A group of 16 severely injured patients was given standard TPN after injury. After sepsis was diagnosed, the patients were randomized to two groups that received either standard TPN with 15% BCAA or an enriched mixture with 46.6% BCAA. Total calories and total nitrogen administered were comparable in the two groups; lipid calories were 27% in the controls and 29% in the study group. Patients receiving the BCAA-enriched mixture had lower urea N_2 excretion and proteolysis; higher serum levels of fibrinogen, ceruloplasmin, transferrin, α_1-antitrypsin, and α_2-macroglobulin; shorter prothrombin times; and lower serum levels of C-reactive protein. These results suggest that enriched BCAA mixture reduced protein catabolism as well as the reprioritization of hepatic plasma protein release. Currently our protocol is to administer standard AA solutions to patients with uncomplicated trauma and use BCAA only in cases of sepsis where hyperglycemia or hypertriglyceridemia indicate nonutilization of the conventional fuel substrates.

Special problems in patients with brain injuries

Patients with isolated brain injuries have urinary nitrogen excretion higher than most polytrauma cases, as shown in the cases presented and as reported in the literature.[13] Typically, as in polytrauma, caloric and protein requirements in the first week are difficult to meet with gastric feeding. For this reason we start TPN within 24 to 48 hours of admission and make the transition to duodenal or gastric feeding as soon as possible. The early use of TPN in this patient population has been shown to improve outcome.[14] Parenteral nutrition can be a problem because of the large amount of fluid administered and the need to limit the volume of free water to reduce brain tissue edema.

In our experience, TPN can be administered safely and serum osmolality maintained at an appropriate level (295 to 305 mOsm/L) if 140 to 160 mEq of sodium per liter is added to the solution and total fluid amount is administered judiciously. For the same reason, we add sodium to commercial feeding formulas when tube feeding is used. Finally, monitoring caloric needs in patients treated with high doses of barbiturates is crucial because these drugs induce a marked decrease in metabolism and overfeeding may occur if the intake is not corrected. However, urinary excretion may be quite significant.

Conclusions

The goal of nutritional support in critically ill trauma patients is to minimize skeletal muscle and visceral protein loss, reverse immunologic anergy, facilitate tissue repair, and prevent multiple organ failure syndrome. Protein loads of 2.5 gm/kg/day are commonly administered along with a caloric load 25% to 30% in excess of HBE calculations to maximize the protein-sparing effect of glucose and lipids. Most authors have not advocated higher amounts of protein because they are unable to decrease endogenous protein catabolism below a critical level. In our opinion, the goal of high protein nutritional support is to raise protein synthesis to the point at which it matches protein losses and a slight positive nitrogen balance is obtained. With this approach, a positive N_2 balance was obtained in most of the patients with brain injury studied by Twyman et al.[13] Also, Alexander et al.[15] found an improved outcome in a controlled, randomized, prospective study conducted in burned children receiving an average of 4.9 gm protein/kg/day. Further studies are necessary to define the validity of this approach in the critically ill adult trauma patient.

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