Metabolic Stress and Trauma: Open Abdomen
Case Study 2
MNT I Fall 2013
Due November 21, 2013

“The Best of Times Hokies”:
Maggie Wilcox
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Understanding the Disease and Pathophysiology

1. J.P. underwent an exploratory laparostomy upon admission to the hospital due to a gunshot wound to the abdomen. This procedure results in the fascia of the abdomen left purposefully open, leaving the peritoneal contents exposed to the environment. Leaving the wound open allows for quick a transition if a follow-up surgery is required and prevents pressure build up that may lead to abdominal compartment syndrome\textsuperscript{1,2}. It is necessary to take all possible steps in preventing abdominal compartment syndrome post-op because this condition leads to the patient becoming hemodynamically unstable and experiencing respiratory, renal, and neurological abnormalities\textsuperscript{2}. Due to the decision to leave the abdomen open, the patient’s medical team has employed the use of a wound VAC to manage care and promote the closure of the wound. A wound VAC, or vacuum-assisted closure, involves the placement of an adhesive material dressing over the open abdomen. This dressing connects to a vacuum via a tube, and the vacuum is able to collect discharge from the wound while stimulating a negative pressure across the open abdomen. The placement of a VAC helps to prevent the buildup of positive pressure within the body of patients who have an open abdomen and the negative pressure aids in the reduction of tissue edema\textsuperscript{1,3}. A wound VAC is beneficial in preventing the adhesion of the peritoneal viscera to the anterior abdominal wall as fascial closure is achieved incrementally via several operations. The VAC also contributes to the natural closure of the fascia by providing a constant force that is able to draw the fascia together\textsuperscript{1}.

2. Patients who undergo gastric resection often experience symptoms of early satiety, epigastric fullness, and dumping syndrome. Early satiety and/or epigastric fullness often leads to the diagnosis of inadequate oral intake, and steps should be taken to ensure the patient is meeting energy requirements. Dumping syndrome is thought to occur due to the decrease in the amount of gastric space available to hold nutrients before release into the intestines and an accelerated movement of hyperosmolar particles into the proximal small bowel\textsuperscript{2,4}. Some professionals suggest an anti-dumping diet, which includes smaller, more frequent meals throughout the day, limiting foods high in sugar (i.e. soda, fruit juices, jams/jellies, cakes and candy), consumption of fluids before meals instead of during, and consumption of high protein food sources with each meal\textsuperscript{4}.

The jejunum comprises the upper 40% of the small intestine past the duodenum. The majority of nutrients are absorbed in the jejunal portion of the small bowel; therefore resection of the proximal jejunum may decrease the patient’s physical ability to absorb nutrients through the GI tract\textsuperscript{5}. The jejunum is thought to adapt more quickly than the other sections of the small bowel, and early feeding via PO or enteral nutrition is thought to aid in recovery and adaptation\textsuperscript{5}.

Digestion and absorption of lipid following gastric and small bowel resection is problematic. Multiple factors contribute to fat malabsorption including the increased rate of nutrients moving through the GI tract preventing enzyme and bile action and a decrease in enzyme production. In addition, the patient’s liver hemorrhage may result in a decreased production of bile, a requirement for the digestion and absorption of dietary fats\textsuperscript{6}.
3. In response to critical illness or severe injury, the body undergoes a metabolic response consisting of 3 phases termed ebb, flow, and recovery. The ebb phase initiates immediately following injury, and lasts up to 36 hours. This phase is the body’s immediate reaction to stress resulting in hypovolemia, shock, tissue hypoxia, decreased cardiac output, decreased metabolic rate, decreased body temperature, decreased oxygen consumption, a fall in insulin levels and a rise in glucagon. The elevation of glucagon and the subsequent fall of insulin results in a stimulation and increased rate of hepatic glucose production via gluconeogenesis and glycogenolysis. The acute flow phase begins immediately following the ebb phase and lasts for 3-4 days post injury. The acute flow phase is characterized by an increase in cardiac output, oxygen consumption, body temperature, energy expenditure, and protein catabolism due to fluid resuscitation and restoration of oxygen transport ability combined with an increased need for oxygen by the tissues. The hormones insulin, epinephrine, norepinephrine, glucagon, and cortisol increase leading to an increase in glucose and free fatty acid production. As catabolism decreases and anabolism increases the hormonal response decreases and the hypermetabolic rate begins to normalize. This leads to the ability of the body to store lean tissue mass, and is termed the recovery phase, or adaptive flow phase. This phase typically lasts 9-14 days after the injury. The recovery phase is characterized by a decrease in hypermetabolic rate, a gradually diminishing hormonal response, restoration of body protein, hemodynamic stability, and possible wound healing if nutrient intake is adequate. The goals during this phase are to minimize catabolism and promote anabolism, provide recommended energy requirements without overfeeding the patient, provide estimated micro and macronutrient requirements, and establish and maintain fluid and electrolyte balance. J.P. was most likely in the ebb phase from the date of his admission on 3/22 until 3/24 or 3/25. There is no lab data from these individual days; therefore, exact length of the ebb phase for this patient cannot be determined. His flow phase followed the end of the ebb phase and lasted at least until 3/29. The lowering glucose lab values shown on 4/1 suggest the patient is entering the recovery phase.

4. The acute phase response is mediated by the release of low-molecular weight proteins called cytokines. Following surgery the cytokines interleukin-1 (IL-1), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6) are released from the macrophages and monocytes of damaged tissues and initiate an immune response. Cytokine release leads to the liver’s release of positive acute phase proteins, C-reactive protein (CRP), fibrinogen, and α2-macroglobulin in order to promote wound healing and produce an anti-inflammatory response. The mobilization of these proteins leads to a rapid loss of lean body mass and an increased negative nitrogen balance in patients who have experienced a high degree of stress and trauma from surgery and/or injury. This acute phase response will continue until the inflammatory response resolves. The breakdown of acute phase proteins results in an increased loss of potassium, phosphorus, and magnesium in the urine, and patients’ electrolytes should be monitored closely as a result.

CRP is a marker of a lessening of the hypermetabolic response that occurs due to stress and trauma. CRP increases in the initial stages of the stress response, and the amount CRP increases is indicative of the intensity of the body’s response to stress. Critically ill patient’s CRP levels should be monitored as a part of nutritional assessment and evaluation because decreasing CRP
levels indicate the patient has entered the anabolic stages of inflammatory response and may require more rigorous nutrition support.

Other acute phase proteins include albumin, transferrin, prealbumin, and retinol-binding protein. The inflammatory response due to injury results in decreased levels for all of these proteins, although retinol-binding protein may be less affected than albumin, prealbumin, and transferrin. The amount by which these proteins are decreased in the serum serves as an indicator of the severity of the injury and resulting stress response.

Understanding the Nutrition Therapy

5. The energy requirements of metabolically stressed patients are significantly increased, but the exact increase of energy requirements depends on the extent of the injury. Major surgery, trauma, extensive burns, and sepsis all result in a significant increase in a patient’s caloric requirements. Indirect calorimetry is the preferred method for determining a patient’s energy requirements, but when this method is not available a recommendation of 25-30 kcal/kg/day should be used in determining individual requirements.

Metabolism of nutrients is significantly altered due to stress and trauma resulting from extensive surgical procedures and traumatic injuries. This state of high bodily stress results in an increased flow of nutrient substrate because the increased needs to promote recovery. Unfortunately, the increased flow is countered by a decreased ability of the body to use carbohydrates, protein, lipids, and oxygen. In states of metabolic stress, there is an increased rate of proteolysis and catabolism of lean tissue mass, which manifests through the loss of muscle.

Hormonal changes cause a large portion of the alterations in nutrient metabolism that occur in states of metabolic stress. The purpose of these alterations in hormone production is the body’s increased need for the catabolism of nutrients due to increased energy needs, the retention of sodium and water to maintain fluid volumes, and to maintain cardiovascular homeostasis. This response is due to the evolutionary adaptation of the human body in order to increase survival rate until the body is healed and can consume and absorb adequate energy again.

Glucagon, a hormone released from the alpha cells of the pancreas, upregulates glycogenolysis and gluconeogenesis in the liver. This increased production and release of glucose from the liver results in the elevation of blood glucose levels.

1. Insulin concentration in the blood is decreased due to an α-adrenergic inhibition of the pancreatic β-cells and the body’s own cellular inability to respond to insulin. This results in a state of insulin resistance and the primary reason for hyperglycemia in patients recovering from major surgery.
2. Cortisol secretion from the adrenal cortex results in an increase of skeletal muscle catabolism to provide the free amino acids required for hepatic gluconeogenesis and acute protein synthesis. Cortisol is partially responsible for the decreased ability of the peripheral tissues to take in glucose and contributes to the hyperglycemic state seen in many trauma patients. In addition, lipolysis is stimulated by cortisol, resulting in an increased concentration of free fatty acids in the blood. Fatty acids can be oxidized in the liver and muscle tissues for energy, converted to ketone bodies, or...
3. Aldosterone and vasopressin are increased during the body’s response to stress. Aldosterone leads to the retention of sodium and vasopressin resulting in an increased resorption of water in the renal tubules

The endocrine response manifests in an altered metabolism of the macronutrients:

- **Carbohydrate Metabolism:** The release of cortisol and catecholamines epinephrine and norepinephrine during surgery results in an increased glucose production from the liver via glycogenolysis and gluconeogenesis. However, because peripheral tissue glucose uptake decreases at this time blood glucose levels rise and patients often become hyperglycemic. The mechanisms to control blood glucose levels do not return post surgery because catabolic hormones continue to be increased while insulin release is subsequently suppressed. This results in a chronic state of hyperglycemia.

- **Protein Metabolism:** Cortisol is also the main hormone responsible for an increased catabolism of body proteins due to metabolic stress and trauma. Skeletal muscle and a small proportion of visceral proteins are broken down into their amino acid building blocks. These amino acids can then be used to produce other proteins that are in high demand, such as the acute phase proteins. They may also be catabolized in the liver to produce substrates for energy via gluconeogenesis.

- **Lipid Metabolism:** Metabolic stress from surgery and/or trauma stimulates lipolysis in order to provide free glycerol for hepatic gluconeogenesis. The fatty acids that were hydrolyzed from the glycerol molecules enter a fatty acid pool so that they can be oxidized via $\beta$-oxidation in the liver and skeletal muscle, converted to ketone bodies that can also be used for ATP production, or re-esterified to a new glycerol molecule.

6. The body’s response due to trauma results in the release of acute-phase proteins, which affects the serum levels of iron, phosphorus, and magnesium. The stress response causes a decrease of these micronutrients due to acute-phase protein action. When serum iron, phosphorus, and magnesium levels are low prior to the initiation of nutrition support, a patient is at an increased risk for refeeding syndrome. Refeeding syndrome occurs due to a rapid increase in carbohydrate intake in patients who have been relying on fat as their primary energy source. Carbohydrate stimulation of insulin secretion results in a rapid cell uptake of phosphorus, magnesium, and potassium that can be detrimental to a patient’s health outcomes.

The patient has a decreased ability to absorb micronutrients, sugars, and lipids due to his recent jejunal resection. This will result in a low level of electrolytes in the blood prior to the initiation of nutrition support. J.P. should be monitored in order to prevent the occurrence of refeeding syndrome by assessing lab values frequently.

Glutamine, Arginine, and Omega-3 fatty acids play a role in wound healing, protein synthesis, and the reduction of risk for the development of infection. Glutamine is the preferred fuel of the small intestinal cells, and is able to enhance mucosal adaptation of the gastrointestinal tract after gastric and/or small bowel resection. This will aid in the progression of nutritional support and
the patient’s ability to consume, digest, and absorb nutrients. Arginine is required to form nitric oxide and for the formation of other major mediators of the inflammatory response, and therefore is a necessary amino acid for patients who are under severe stress and trauma. Omega-3 fatty acids have been shown to counter the synthesis of inflammatory eicosanoids that are a product of omega-6 fatty acids. Omega-3 fatty acids synthesize prostanoids, which have an anti-inflammatory effect on the body. There should be an increase in the amount of omega-3 fatty acids given to the patient in order to counter inflammation that results from metabolic stress and trauma.

J.P. should receive 2-7 times the normal recommendation of Glutamine, 17-25 g of Arginine, and 1.6 of omega-3 fatty acids per day in order to promote recovery.

7. The American Society for Parenteral and Enteral Nutrition (ASPEN) Guideline A4 states “enteral feeding should be started early within the first 24-48 hours following admission,” and “feedings should be advanced toward goal over the next 38-72 hours.” In patients who have suffered blunt or penetrating trauma, early initiation of enteral nutrition has been shown to decrease the patient’s risk for infection and prevent gut atrophy. When assessing patient tolerance for EN in the ICU setting, lack of bowel sounds and the passage of stool is not required for EN initiation, and when the patient shows evidence of gastric intolerance, feeding of the small bowel should be the method of enteral nutrition.

All methods of enteral nutrition should be assessed and the benefits should clearly outweigh the risks before parenteral nutrition is initiated. ASPEN guideline B3 specifically addresses patients who undergo major upper GI surgery and states, “PN should not be initiated in the immediate postoperative period, but should be delayed 5-7 days (should EN continue not to be feasible.). However, this guideline also states that if the PN is expected to be administered for less than 5-7 days, the risks may outweigh the benefits and recommends that administration of PN be reserved for patients who are estimated to require it for more than 7 days. If the patient has not been able to tolerate full enteral nutrition for greater than 7-10 days TPN or a combination of TPN and TEN should be initiated immediately. A patient placed on TPN should be monitored for return of GI function and steps should be taken to initiate and increase nutritional support from enteral nutrition while reducing the energy provided from TPN. When a patient is able to tolerate and meet >60% of estimated energy needs enterally, TPN should be stopped and all nutrient delivery should be provided through TEN.

J.P. was unable to eat orally due to a state of sedation. Additionally, the resection of the patient’s proximal jejunum, the discontinuity of the gastrointestinal tract, and open abdomen indicate the need for parenteral nutrition. Parenteral nutrition was administered on day 7, which is in accordance with ASPEN guidelines, and a distally placed jejunostomy tube was placed on day 7 for transition to enteral nutrition as soon as the patient establishes tolerance. On day 10, it has been determined the patient’s GI function is returning and the decision to initiate a transition to enteral nutrition has been made.

Nutrition Assessment
9. Trauma patients require fluid resuscitation, which leads to a false increase in weight throughout time when the body is responding to stress. It is essential initial weight be obtained as soon as possible upon admission to the hospital because this is likely to be the most accurate usual body weight of the patient. If possible the weight upon admission should be compared to a usual body weight given by the patient or by a family member. A possible alternative to scale weight assessment may be the use of mid-arm circumference within 48 hours of admission, however this method may not serve as a good method to assess an alteration in muscle mass in critically ill patients. Therefore mid-arm circumference should only be used for initial weight assessment and should not be used to monitor nutritional status while patients are in the hospital. The pt’s 4+ edema upon admission indicates fluid status would effect the assessment of weight. The pt’s 24 hour I/O indicate the patient is holding on to fluid based on a net value of +888ml in the 24 hours and +3721 since admission. In addition to increased fluid retention, the patient’s current state of sedation, J-P drainage tubes, and the wound VAC make it difficult to assess scale weight due to the patient’s inability to stand and/or remain awake while in the ICU.

10. According to the Nutrition Care Manual, the Penn State equation, the Ireton-Jones equation, or the Swinamer equation should be used when estimating energy requirements.

- In order to use the Penn State Equation, we must first use the Mifflin-St.Jeor equation.
  Calculations: 10W + 6.25H - 5A + 5
  $10(102.7 \text{ kg}) + 6.25(177.8 \text{ cm}) - 5(29) + 5 = 1998$ kcal/day
- Inserting 1989 kcal into the Penn State Equation, his estimated energy requirement is 2353 kcal/day
  Calculations: Mifflin(0.96) + V_e(31) + T_{max}(167) – 6212
  $1998(0.96) + (3.5 \text{ L/min})(31) + (39.2)(167) – 6212 = 2361$ kcal
- The Ireton Jones Energy Equation for ventilator dependent pts incorporates the increased energy needs of stress and trauma. IJEE estimates the pts needs to be 2462 kcal/day.
  Calculations: $1784 – 11(A) + 5(W) + 244 (S) + 239 (T) + 804 (B)\$
  $1784 – 11(29) + 5(102.7) + 244(1) + 239(1) + 804(0) = 2462$ kcal

Both equations appear to underestimate the patient’s energy requirements, and we found it necessary to use an additional method for comparison.

- Using the Mifflin-St Jeor equation with an activity factor of 1.2 and an injury factor of 1.6 the patient’s energy requirements are estimated to be 3819 kcal/day.
  Calculations: 10W + 6.25H - 5A + 5
  $10(102.7 \text{ kg}) + 6.25(177.8 \text{ cm}) - 5(29) + 5 = 1998$ kcal/day
  $1998 \text{ kcal} (1.2) (1.6) = 3836$ kcal/day

Because of the pt’s increased energy needs due to the stress of surgery, the Mifflin-St Jeor estimation of energy needs should be used to determine an estimate of total calories required.

Estimation of the pt’s protein requirements should be calculated using adjusted body weight because the pt is greater than 120% of his IBW. Protein requirements are increased due to the pt’s recent surgeries and state of stress. A factor of 2.0-2.5 g/kg ABW was used based on the patient’s decreased prealbumin and a recently determined Nitrogen Balance of -39.36.

- J.P.’s protein requirements are 165-205 g/day
  $82.3 \text{ kg} (2.0-2.5 \text{ g/kg}) = 165-205$ g/day
11. Indirect calorimetry measures the energy expenditure of an individual by measuring the amount of oxygen consumed and amount of carbon dioxide produced over a given period of time. Measurement of energy requirements via indirect calorimetry employs a ventilated hood in order to capture carbon dioxide expired by the patient. The oxygen and carbon dioxide values are measured and quantified using a metabolic cart, which will provide an estimated energy expenditure of the patient that can be used to determine individual needs. This method of determining energy needs is the preferred method for the critically ill patient population because patient needs are much higher and individual needs are unique to severity and intensity of the patient’s stress response. Limitations to using indirect calorimetry can arise with patients who have high oxygen requirements, a chest tube, acidosis or are using supplemental oxygen because these factors can create invalid results.

12. The Penn State and Ireton Jones Energy Equations predicted an estimated energy need of 2361 and 2462 kcal/day respectively for the patient. Although the Penn State Equation has been suggested to be approximately 70% accurate in predicting energy needs of critically ill patients, we did not believe this estimation to be enough for the amount of stress the patient’s body was under. Therefore, the Mifflin-St Jeor equation was employed with an activity factor of 1.2 and an injury factor of 1.6. This resulted in an estimated energy expenditure of 3819 kcal/day and was similar to the indirect calorimetry results of 3836 kcal/day.

13. RQ indicates the type of substrate being metabolized for energy by the body. The patient’s RQ value of 0.76 indicates his body’s major and preferred source of energy is currently from the metabolism of fatty acids.

14. The patient has undergone multiple, consecutive surgeries due to a gunshot wound, and his abdomen has been left open. This has placed the patient’s body under metabolic stress and resulted in a state of hypermetabolism. The nutrient requirements for an open abdomen may account for 25-30% of the patient’s total energy needs, and when combined the other surgical and trauma injuries J.P.’s body is in a state of catabolism in order to provide enough energy to promote healing.

15. J.P. was prescribed Dextrose:140 CAA: 60 FAT/L:20 at a goal rate of 135 mL/hr. This prescription provides 2448 kcal/day and 194.4 g protein. With the additional 924 kcal from the propofol, the total amount of energy provided is 3372 kcal/day. The total volume provided on the follow-up day 7 was 3.312 L based on the pt’s I/O for this day. The calculated total volume was 3.24 L, however there was likely an addition of micronutrients and other components such as fluid or insulin, leading to a value greater than what is calculated based solely on carbohydrates, protein, and lipids.

Calculations:

At a goal rate of 135 mL/day x 24hr = 3.24 L total volume/day
- 3.24L x 20g/L lipid = 64.8 g lipids x 2kcal/g= 129.6 kcal from lipids TPN + 924 kcal from lipids via Propofol
- 3.24L x 60g protein=194.4g x 4 kcal/g = 778 kcal from protein
- 140g CHO/L x 3.24L = 453.6g CHO x 3.4 kcal/g = 1540.2 kcal from CHO
16. On day 4, the metabolic cart measured the pt’s energy requirements to be 3657 kcal. This aligns well with the amount of energy provided through TPN, and unless there is reason to believe the patient is losing weight, energy provided through nutrition support does not need to be altered. A major restraint to altering the nutrition support composition is the pt’s current requirement for Propofol, which contributes both total calories and fat that must be accounted for. When pts are on Propofol, there is a risk for overfeeding and adjustment of nutrition support goal rates can be difficult because both the feed rate and the propofol infusion rate will have to be adjusted.

17. Intravenous administration of propofol results in the sedation of critically ill patients, and is commonly prescribed to mechanically ventilated patients to maintain a relaxed and sedated state in the hospital intensive care unit. Propofol provides 1.1 kcal/ml at 35 ml/hr from lipid, increasing the patient’s total energy intake. The additional 924 kcal provided from the medication must be considered when calculating the nutrition support energy and nutrient requirements in order to ensure the patient is not overfed. Before administration of Propofol, the patient or a family member should be questioned about possible allergies to eggs and/or soy as these are components of the medication and may produce an allergic reaction.

18. Trickle or trophic feeding involves the provision of a small amount of enteral nutrition (~10-20 ml/hr to provide 15-30% of pt needs) to the small bowel of a patient receiving TPN. The energy provided through EN is not a sufficient amount to be considered in the calculation of energy provided to the patient. The intention of the initiation of trickle/trophic feeding is to provide stimulation to the gut in order to preserve some GI function in patients receiving TPN. This will ideally make the transition to enteral and oral nutrition more easily tolerated.

J.P. is experiencing metabolic stress due to trauma and an open abdomen. Pivot 1.5 tube feed formula is developed for patients in these conditions because it contains a higher concentration of calories and high peptide-based proteins (25% of kcal from protein) in order to promote wound healing. In addition to the increased energy and protein, Pivot 1.5 provides glutamine, arginine, and increased levels of β-carotene, Vitamin C, and Vitamin E to promote immune function.

18. Pertinent Abnormal Lab Values

<table>
<thead>
<tr>
<th>Lab Result</th>
<th>Concern</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1C 7.1%</td>
<td>Elevated</td>
<td>Indicative of diabetes – carbohydrates need to be assessed</td>
</tr>
<tr>
<td>ALT/AST 435 U/L / 190 U/L</td>
<td>Elevated</td>
<td>Due to liver hemorrhage – pt has increased protein needs</td>
</tr>
<tr>
<td>Test</td>
<td>Value</td>
<td>Status</td>
</tr>
<tr>
<td>-------------------------</td>
<td>---------</td>
<td>----------</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>245 mg/dL</td>
<td>Elevated</td>
</tr>
<tr>
<td>Prealbumin</td>
<td>3.0 mg/dL</td>
<td>Elevated</td>
</tr>
<tr>
<td>Albumin</td>
<td>1.4 g/dL</td>
<td>Decreased</td>
</tr>
<tr>
<td>Glucose</td>
<td>164 mg/dL</td>
<td>Elevated</td>
</tr>
<tr>
<td>BUN</td>
<td>23 mg/dL</td>
<td>Elevated</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>540 U/L</td>
<td>Elevated</td>
</tr>
</tbody>
</table>

20. A primary goal of nutrition support is the achievement and/or maintenance of a positive nitrogen balance. This goal is difficult to achieve in patients with an open abdomen because patients have a hypermetabolic response to stress that results in an increased catabolism of body proteins. In addition, the maintenance of an open abdomen in these patients results in fluid, electrolyte, and protein losses through the exposed abdominal viscera. The amount of protein lost through abdominal fluid losses is an estimated 15-30 g/L. When the increased protein loss of these patients is not considered in calculation of nitrogen balance, the result is underestimation of the total nitrogen loss and overestimation of sufficient protein administration to meet patient needs. This lapse leads to underfeeding and inadequate nutrition support. In critically ill patients, underfeeding consequently decreases wound healing, increases the occurrence of infection, and decreases the patient’s survival outcomes.

Nitrogen Balance Study:
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NB = \left[ \frac{\text{Protein intake (g)}}{6.25} \right] - (\text{UUN} \times 1.2) + 2 - 4
\]
\[
[194.4 \ g / 6.25] - (42 \ g \times 1.2) - 20
\]
\[
NB = -39.36
\]
21. Nutrition Diagnoses

- NC 1.4: Altered GI function R/T gastrointestinal surgery resulting in a shortened jejunal limb and open abdomen AEB absence of stool output, lack of bowel sounds, and moderate abdominal distention
- NI 2.1: Inadequate oral intake R/T sedation and recovery from GI surgery AEB current nutrition support of TPN and placement of a distal jejunostomy tube
- NI 5.1: Increased energy and protein needs R/T protein catabolism and hypermetabolic stress response to surgery AEB negative nitrogen balance of -39.36, elevated BUN (23 mg/dL) and metabolic cart measurement of energy expenditure of 3657 kcal

22. Nutrition Intervention

Transition to Enteral Nutrition from Parenteral Nutrition (ND 2.1 and ND 2.2)

- Increase continuous TF Pivot 1.5 to rate 20 ml/hr providing 660 kcal (6.4 kcal/kg BW), 41.3 g protein (0.4 g/kg BW), and 334 ml free fluid.
- Increase TF rate 10 ml/hr q 12 hours as tolerated until goal rate of 83 ml/hr to provide 2733 kcal (26.6 kcal/kg BW), 171 g protein (1.7 g/kg BW), and 1386 ml free fluid with free water flushes of 197 ml q 2 hours.
- Decrease TPN by 25% @ TF 20 ml/hr
- Decrease TPN by 50% @ TF rate 40 ml/hr
- Stop TPN @ FT rate 70 ml/hr (>75% pt needs met by TF)
- Initiate free water flushes of 107 ml q 2 hours @ TF goal rate of 70 ml/hr.
- Physicians should make alterations to D5 0.45 IV fluid prn

Nutrition Monitoring and Evaluation

23. The monitoring of daily weight, urine glucose, acetone, and protein levels are important for a patient receiving TPN. The site of IV insertion should be carefully monitored for infection. Lab values that should be monitored include electrolytes, BUN, creatinine, glucose, magnesium, calcium, phosphorus, cholesterol, blood triglycerides, albumin, transferrin, prealbumin, LFTs, and nitrogen balance. The function of organs and signs of organ failure should be closely monitored and a patient’s acid/base balance should be assessed. Potential drug-nutrient interactions should be identified and signs and symptoms of DNI should be assessed. The patient’s metabolic cart measurement should be monitored for when catabolism transitions to anabolism. GI tolerance for the use of enteral nutrition support should be assessed frequently. A patient’s daily weight via dry weight assessment is essential to monitor actual vs. prescribed nutrient intake and evaluate the need to increase or decrease total energy provided through nutrition support.

24. Hyperglycemia indicates the patient’s decreased ability to maintain glycemic control/glucose homeostasis. There are multiple reasons hyperglycemia occurs in the critically ill patient: Glucose provided by nutrition support, increased gluconeogenesis, insulin resistance in combination with increased glucagon and catecholamines all lead to increase of blood glucose levels, especially when combined with the impaired ability of peripheral tissues to take glucose
into the cells due to the inflammatory response. Hyperglycemia is a concern for the critically ill patient population because it acts as an immunosuppressant, leading to potential decreased wound healing and a longer recovery time. Evidence suggests that in the critically ill patient population an acceptable blood glucose range may be higher than the reference range used in normal conditions and blood glucose goal of 100-150 mg/dl may be more applicable for this population. The patient’s hyperglycemia is being managed through the administration of insulin via the sliding scale method. Lab results from 4/1 indicate this method is successful due a decrease in blood glucose levels from previous labs to an acceptable value of 140 mg/dl. The patient’s blood glucose levels should continue to be monitored and necessary adjustments should be made to nutrition support or amount of sliding scale insulin provided as/If lab values change.
Nutrition Services
4/1/2013
8:00am

Assessment:
J.P., a 29-year-old male was admitted to the ER due to a GSW to the abdomen and underwent an exploratory laparotomy, gastric repair, control of liver hemorrhage, and resection of the proximal jejunum, leaving his GI tract in discontinuity. The pt returned to surgery 3 times following initial admission in order to reestablish continuity of the bowel, place and change a wound VAC, shorten the jejunal limb, and insert a distal jejunostomy feeding tube. Patient is sedated. No known allergies.

Height: 177.8 cm
Admission Weight: 102.7 kg
Current Body Weight: 109 kg
Usual Body Weight: N/A
BMI: 32.1
IBW: 75.3 kg
% IBW: 136%
ABW: 82.3 kg

Current Nutrition: NPO, TPN
- TPN rx- Dextrose 140, CAA 60, Lipid: 20 at a goal rate of 135 ml/hr
- Pivot 1.5 trickle feeds at 5 ml/hr

Estimated Energy Requirements (Mifflin-St Jeor, AF 1.2, IF 1.6): 3836 kcal/day
Energy Expenditure determined by indirect calorimetry: 3657 kcal/day, RQ 0.76

Protein Requirements (2.0-2.5 g/kg ABW): 165-205 g/day

Fluid Requirements (1 ml/kcal): 3657-3836
- Receiving D5 0.45 IV Fluid at 75 ml/hr

Nutrition Focused Physical Findings:
- Abdomen: moderately distended w/ wound VAC
- Bowel Function: no stool output
- Bowel Sounds: absent

Labs: WNL except:
- Elevated HbA1C: 7.1%
- Elevated ALT/AST: 435 U/L / 190 U/L
- Elevated C-reactive protein: 245 mg/dL
- Elevated Prealbumin: 3.0 mg/dL
- Decreased Albumin: 1.4 g/dL
- Elevated BUN: 23 mg/dL
- Elevated Glucose: 164 mg/dL
- Alkaline phosphatase: 540 U/L
Nitrogen Balance Study: -39.36

Medications: Propofol (35 ml/hr)- addition of 924 kcal

*Diagnosis:*
NC 1.4: Altered GI function R/T gastrointestinal surgery resulting in a shortened jejunal limb and open abdomen AEB absence of stool output, lack of bowel sounds, and moderate abdominal distention

NI 2.1: Inadequate oral intake R/T sedation and recovery from GI surgery AEB current nutrition support of TPN and placement of a distal jejunostomy tube

NI 5.1: Increased energy and protein needs R/T protein catabolism and hypermetabolic stress response to surgery AEB negative nitrogen balance of -39.36, elevated BUN (23 mg/dL) and metabolic cart measurement of energy expenditure of 3657 kcal

*Intervention:*
Transition to Enteral Nutrition from Parenteral Nutrition (ND 2.1 and ND 2.2)
- Increase continuous TF Pivot 1.5 to rate 20 ml/hr providing 660 kcal (6.4 kcal/kg BW), 41.3 g protein (0.4 g/kg BW), and 334 ml free fluid.
- Increase TF rate 10 ml/hr q 12 hours as tolerated until goal rate of 83 ml/hr to provide 2733 kcal (26.6 kcal/kg BW), 171 g protein (1.7 g/kg BW), and 1386 ml free fluid with free water flushes of 197 ml q 2 hours.
- Decrease TPN by 25% @ TF 20 ml/hr
- Decrease TPN by 50% @ TF rate 40 ml/hr
- Stop TPN @ FT rate 70 ml/hr (>75% pt needs met by TF)
- Initiate free water flushes of 107 ml q 2 hours @ TF goal rate of 70 ml/hr.
- Physicians should make alterations to D5 0.45 IV fluid prn

*Monitoring and Evaluation:*
Monitor GI tolerance of enteral nutrition support
- Return and maintenance of bowel sounds and stool output
- Continued decrease in abdominal distention and returned tenderness of the abdomen

Monitor Nitrogen Balance for adequate protein intake to account for total protein losses
- NB should become for positive as catabolism decreases and anabolism increases

Monitor weight daily via dry weight assessment to assess actual vs. prescribed nutrition support intake
- Increase or decrease nutrition support as needed

Monitor urine glucose, acetone, and protein levels
- Glucose (Neg)
- Acetone (Neg)
- Protein (Neg)
Monitor blood glucose lab values to assess effectiveness of insulin administration with enteral nutrition support
  • Blood glucose within 100-150 mg/dl

Monitor lab values:
  • Electrolytes, BUN (0.1-1.0 mg/dl), creatine (0.7-1.6 mg/dl), magnesium (1.7-2.4 mEq/L), calcium (8.1-10.7 mg/dl), phosphorus (2.6-4.8 mg/dl), cholesterol (160-200 mg/dl), blood TG (28-235 mg/dl), albumin (3.5-5.0 g/dl), transferrin (215-365 mg/dl), prealbumin, (15-36 mg/dl), LFTs (ALP 30-120 U/L and AST 0-35 U/L)

Reassess metabolic cart energy expenditure for indication of anabolism
  • RQ 0.85

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Bibliography


