I. Literature Review

Overview of Guillain-Barré Syndrome

Guillain-Barré syndrome (GBS) is an inflammatory disorder that affects the peripheral nervous system[1]. An immune-mediated attack of peripheral nerve cells occurs during GBS onset characterized by infiltration of white blood cells. The immune attack results in inflammation of nervous tissue, demyelination of nerve cells, and axonal degeneration[2].

The peripheral nervous system (PNS) is composed of nerves located outside the brain and spinal cord and functions to connect the central nervous system to the limbs and organs. Neurons of the PNS are surrounded and protected by glial cells, which are non-neuronal cells that maintain homeostasis and form myelin. The principal glial cells of the PNS are Schwann cells. Myelinating Schwann cells wrap around axons of motor and sensory neurons to form the myelin sheath. Myelin is a specialized fatty insulation that envelops axons, the part of nerve cells involved in signal conduction[3] (Figure 1). In GBS, the immune system mounts an attack against myelin. When the neuron is demyelinated its ability to conduct signals is severely impaired, resulting in a neuropathy characterized by muscular weakness and distal sensory loss[4]. In GBS and other medical conditions where PNS nueron myelin sheaths are injured or degraded, nerves cannot transmit signals efficiently, and muscles begin to lose their ability to respond to commands relayed from the central nervous system.

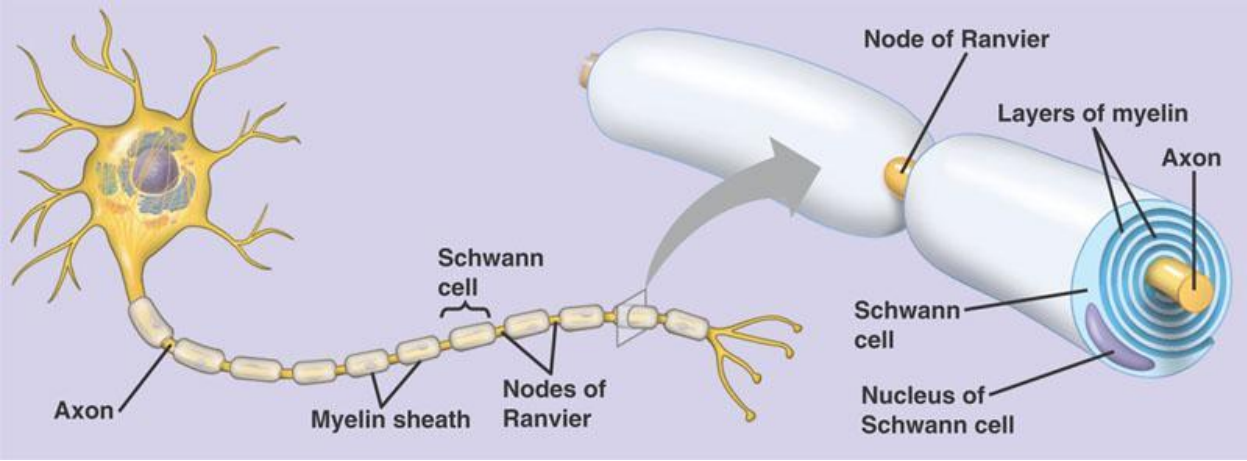

Figure 1 Structure of a Schwann Cell. Adapted from *Campbell Biology*. Copyright 2014 Pearson Education, Inc.

One or two cases of GBS per 100,000 people are reported each year[2]. GBS affects males more frequently than females, and its incidence increases with advancing age. GBS has been characterized as a syndrome rather than a disease due to lack of a specific disease-causing agent[1]. The symptoms can be varied, making it difficult to diagnose GBS in its early stages. GBS is often diagnosed by a clinical pattern of sudden onset weakness on both sides of the body and elevated protein in the cerebrospinal fluid without increase of white blood cells[5]. Protein

elevation is thought to be due to inflammation of nerve cells leaving the central nervous system. Autonomic nervous system involvement occurs in many cases and can cause urine retention, ileus, tachycardia, hypertension, arrhythmia, and orthostatic hypotension[6].

A number of subtypes of GBS are recognized including acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN). However, many patients experience symptoms that overlap subtypes, and classification is often difficult. In addition, all subtypes can occur in partial forms[5]. The most common subtype in Europe and North America is acute inflammatory demyelinating polyneuropathy. In the AIDP subtype, the immune system reacts to antigenic determinants present on Schwann cells or myelin that lead to immune attack and result in demyelination[2]. The etiology is not completely understood, but it is believed to have an autoimmune origin which is triggered by an infection that stimulates anti-ganglioside antibody production in most cases[1]. Numerous case reports have identified possible infections and other events as antecedents of GBS. Case control studies have consistently confirmed *Campylobacter jejuni* and cytomegalovirus as the most common precipitants of GBS[6]. When GBS is preceded by an infection, it is thought that the virus changes the nature of neurons so the immune system treats them as foreign. Another thought is that the virus alters the immune system so it is less discriminating towards its own cells, allowing immune cells to infiltrate[5].

GBS reveals itself in a matter of days. The common clinical pattern is an ascending paralysis first noticed as loss of tendon reflexes in the lower extremities, which evolves over hours to days with numbness in all extremities. Lower limbs are affected more than upper limbs, and facial paralysis occurs in 50% of individuals[1]. The clinical course is similar regardless of subtype, although AIDP GBS after *Campylobacter* infection tends to lead to more severe symptoms than other identified antecendents[4]. The maximum severity of symptoms is usually seen in 2-4 weeks, often referred to as the acute stage[1]. Vital capacity and swallowing may also rapidly deteriorate to a point where intensive care becomes necessary[4].

AIDP in particular has been identified as an inflammatory process involving a large portion of the PNS. Activated white blood cells migrate across a delicate layer of connective tissue surrounding the myelin sheath and attract macrophages. Macrophages then penetrate Schwann cells and digest the myelin, leaving demyelinated axons. The location of neuron degradration accounts for the distribution of associated symptoms[6]. Demyelinating forms of GBS are characterized not only by macrophage-induced demyelination, but also by T-cell and plasma cell infiltration into peripheral neurons[7].

Activation of T-cells is evident in GBS by the increased number of circulating cells that bear activation markers, as well as increased concentrations of the interleukin-2-receptor expressed on the surface of immune cells. Inflammation seen in AIDP may be a consequence of the inflammatory mediators secreted by activated macrophages or T cells. One of the most powerful cytokines, TNFα, has often been proposed to have a direct role in mediating demyelination.

TNFα has been found in high concentrations in serum during the acute stage of GBS[6]. The release of TNFα during the acute phase of GBS is also consistent with a systemic stress response[8].

No cure exists for GBS, but available therapies can lessen symptoms and accelerate recovery. The most common therapies are plasma exchange (commonly called plasmapheresis or PLEX therapy) and high-dose immunoglobulin therapy. Both are effective, but immunoglobin therapy is easier to administer. In high-dose immunoglobulin therapy, a patient receives intravenous injections of immunoglobulin proteins. High doses lessen immune attack on the nervous system by saturating receptors on macrophages, suppressing antibodies, and suppressing inflammatory mediators. The alternative therapy is plasmapheresis, a method by which blood is removed from the body and processed. Red and white blood cells are separated from the plasma, and blood cells are returned to the patient while the plasma is not. Plasmapheresis is thought to be effective because antibodies and other immune cell-derived factors that contribute to nerve damage in GBS are removed[5].

Nutritional Needs in Guillain-Barré syndrome

During the first 1-2 weeks of acute onset, the metabolic response in GBS is similar to the stress response seen after a neurotrauma. When energy needs are determined by indirect calorimetry, the range may be as high as 40-45 kcal/kg compared with the typical requirement of 25-30 kcal/kg. In addition, protein needs could be as much as twice the normal amount because of accelerated protein breakdown[4]. The inability to protect body protein stores is seen in other inflammatory diseases as well. Impaired utilization of ketones for energy production during GBS is thought to be mediated by cytokines such as TNFα and interleukin-1beta[9].

The impression that GBS patients are well nourished frequently delays the initiation of nutrition support. Typically, however, GBS patients have already experienced protein depletion at the time of admission. Depletion has been found to depend on the severity of the syndrome[9]. In addition, the muscles of the throat are affected in a small percentage of patients, leading to dysphagia. Difficulty swallowing further indicates the necessity of nutritional intervention for patients with GBS [4].

Providing adequate nutrition during GBS is important, specifically to abate losses of respiratory skeletal muscle. The main cause of death in GBS is adult respiratory distress syndrome, which can necessitate ventilator support. When nutrition support is inadequate, patients are more susceptible to respiratory compromise due to decreased immunocompetence, muscle atrophy, and reduced respiratory muscle endurance. Inadequate nutrition support also increases the risk for pressure ulcers, fluid and electrolyte abnormalities, and infections[9].

Hypermetabolism and hypercatabolism decline over the course of the disease, and recovery is expected for most patients[9]. Although full motor recovery is expected in ~60% of patients, more than 15% of patients have residual moderate-to-severe weakness 1 year after disease onset, and

10% die despite intervention and treatment. Many patients experience sensory deficits and do not return to their previous functional level. When a patient fully recovers, recurrence of GBS is less than 5%[7].

II. Description of the patient

The patient was a 57-year-old Caucasian male. He was single and lived in a home he owned. Before his illness, he worked as a security guard at a correctional facility for 20 years. Prior to this, he was a professional wrestler. In general, the patient was physically active, and he often commented on how difficult the onset of GBS was due to his previous lifestyle. The patient drank 6-12 beers each night at home but did not experience overt withdrawal symptoms during the course of his hospital admission. The patient had a medical history of anxiety, tachyarrhythmia, and arthritis but was not taking any routine medications prior to admission.

III. Course of the patient

A week prior to hospital admission, the patient experienced urinary retention. An outpatient physician placed a temporary catheter, and the patient was given Flomax to relax bladder muscles and ease urination. After a few days, the patient noticed right arm numbness, which progressively spread to the rest of his body. A week after his outpatient visit, he was unable to walk due to weakness, so he called EMS personnel who took him to the emergency room. His spinal fluid protein was mildly elevated, giving him a clinical picture consistent with GBS, so he was transferred to Mission Memorial Hospital Medical/Surgical Intensive Care Unit (MSICU). No GBS subtype or antecedent infection was identified for the patient.

The day after admission the patient began intravenous immunoglobin (IVIG) therapy, the typical intervention for patients who present with GBS. IVIG was administered daily for 5 consecutive days. When admitted to the MSICU, the patient felt numb throughout his body and was unable to move his legs. Speech therapy was consulted on day 2 of admission to assess if he was able to swallow due to muscle weakness and numbness. A bedside swallow evaluation indicated that he had normal swallow function, and he was cleared for a regular oral diet and a full range of liquids for meals.

Five days after admission, the patient felt weaker and complained of difficulty swallowing. A videoflouroscopic swallow study (VFSS) was ordered to assess swallowing capacity. The VFSS did not show any differences from the prior bedside swallow study, so a regular diet and full range of liquids was still provided. On day 5, the patient also had an extremely low serum sodium level of 119 mEq/L (normal range 135-145 mEq/L), which was suspected to be due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH), a side effect commonly seen during GBS onset. Intravenous hypertonic saline was administered to raise sodium levels.

After 5 days of IVIG therapy, little to no changes were observed in the patient's functional capacity, so the treatment was extended to 7 days. On day 5, the patient was transferred out of

the MSICU to a step down unit within the hospital because he was medically stable and did not require critical care.

Eight days after admission, the patient experienced acute hypoxemic respiratory failure and returned to the MSICU for observation. Respiratory distress occurred when the patient felt a buildup of mucus in his esophagus that he was unable to clear after consuming liquid through a straw. In the MSICU, speech therapy was consulted again due to respiratory difficulty arising after swallowing. At this stage no differences were observed in swallowing capacity, but the speech therapist warned that the patient was at risk for swallow difficulty due to progressive weakness that could eventually affect throat muscles. On day 9 of hospitalization, the patient was no long able to lift his arms, but he continued to have functionality of his head and neck.

Day 11 of admission, hypertonic saline was discontinued because his sodium level returned to the normal range. Before the patient returned to the step down unit on day 11, the speech therapist repeated a VFSS, which showed mild pharyngeal dysphagia, and the texture of his diet was modified to chopped meats. Speech therapy saw the patient on day 15 and noted that the patient was tolerating the recommended modified diet but required full assistance while eating due to full paralysis of his arms.

2 weeks after admission, after 7 days of IVIG therapy and no marked improvement to the patient's functional capacity, plasmapheresis (PLEX) therapy began. PLEX took place every other day for a total of 5 treatments. The patient again experienced respiratory difficulty on the day of PLEX initiation and complained of shortness of breath and trouble swallowing. He aspirated on water, was no longer deemed safe for an oral diet, and was intubated and transferred to the MSICU. Upon his return to the ICU, enteral nutrition was initiated. The patient remained in the MSICU for the remainder of his hospitalization due to need of respiratory support.

Day 19 after admission, a tracheotomy tube was placed to assist with his ongoing difficulty breathing so the endotracheal tube placed on day 14 could be removed. In addition, a percutaneous endoscopic gastrostomy tube (PEG) was placed on day 20 of admission due to the expected long-term need for nutrition support. After PLEX treatment was completed on day 24, the patient showed some minor improvements in the motor function in his limbs. However, he continued to experience respiratory difficulty and was unable to swallow.

Eventually, the patient was accepted by the long-term acute care hospital (LTACH) at Mission Hospital, where he continued to receive occupational and physical therapy, enteral nutrition, and respiratory support.

IV. Nutritional evaluation and treatment

The Clinical Nutrition Team (CNT) and the Metabolic Support Service (MSS) were consulted frequently throughout the patient's hospitalization to provide nutrition recommendations and support.

The patient was first seen by the CNT 5 days after admission, after transfer out of the MSICU. His weight was 124.3 kg and height was 190.5 cm. The Mifflin St. Jeor equation (x 1-1.1 activity factor) estimated his caloric needs at 2162-2378 kcal/day. His protein needs were estimated to be 99-134 g/day based on 0.8-1 g/kg body weight. At that time the patient was consuming 75-100% of the meals provided and reported that he did not have any issues with swallowing. The patient was on a regular oral diet, which provided around 1800 kcals and 91 grams protein per day. Because the patient was eating well, the CNT unfortunately did not implement any dietary interventions.

After the patient transferred to the MSICU on day 8, nutrition care was transferred to a MSS dietitian. On day 9, his weight was 110.1 kg, an 11% weight loss in 9 days. The MSS dietitian diagnosed the patient as moderately malnourished due to weight loss and a presumed energy intake less than his estimated needs in the setting of acute illness according to ASPEN malnutrition criteria[10] (Table 1).

	Acute Illness/Injury				Chronic Illness				Social or Environmental Circumstances			
Clinical Characteristics	Moderate Malnutrition		Severe Malnutrition		Moderate Malnutrition		Severe Malnutrition		Moderate Malnutrition		Severe Malnutrition	
Energy Intake	<75% of estimated energy needs for >7 days		≤50% estimated energy needs for ≥ 5 days		<75% of estimated energy needs for ≥1 month		≤75% of estimated energy needs for ≥1 month		<75% of estimated energy needs for ≥3 months		≤50% of estimated energy needs for ≥1 month	
Weight Loss	1-2%	1 wk	>2%	1 wk	5%	1 mo	>5%	1 mo	5%	1 mo	>5%	1 mo
	5%	1 mo	>5%	1 mo	7.5%	3 mos	>7.5 %	3 mos	7.5%	3 mos	>7.5 %	3 mos
	7.5%	3 mos	>7.5 %	3 mos	10%	6 mos	>10%	6 mos	10%	6 mos	>10%	6 mos
					20%	1 yr	>20%	1 yr	20%	1 yr	>20%	1 yr
Body Fat	Mild		Moderate		Mild		Severe		Mild		Severe	
Muscle Mass	Mild		Moderate		Mild		Severe		Mild		Severe	
Fluid Accumulation	Mild		Moderate to Severe		Mild		Severe		Mild		Severe	
Reduced Grip Strength	n/a		Measurably reduced		n/a		Measurably reduced		n/a		Measurably reduced	
**A minimum of 2 of the 6 characteristics is recommended for diagnosis of either severe or moderate malnutrition												

Table 1 Clinical Characteristics to Support a Diagnosis of Malnutrition. Adapted from *Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition).* Copyright 2012 Journal of Parenteral and Enteral Nutrition.

The MSS dietitian added a multivitamin with minerals to the patient's diet in order to meet his micronutrient needs. In addition, to provide additional calories and protein due to excessive weight loss, the MSS dietitian ordered oral supplements of Ensure Plus shakes three times a day with meals, as well as Core Power high protein shakes. Each Ensure Plus potentially provided

the patient with an extra 350 kcal and 13 grams of protein, while Core Power provided 240 kcal and 26 g protein. Unfortunately, consumption of both meals and supplements were not well documented after the patient left the MSICU the second time. At that time the patient also required assistance feeding himself due to weakness and numbness of both arms.

When the patient experienced respiratory difficulty day 9, he was put a full liquid diet for 5 days. The full liquid diet consisted of liquids as well as solid foods that turn to liquid at room temperature, providing minimal calories. On day 13, a regular oral diet was provided once again, but with the texture modification of chopped meats following the VFSS on day 11, which showed mild pharyngeal dysphagia. After transfer out of the MSICU he was seen again by the CNT on day 13. The CNT recommended frequent weight checks as well as skin integrity checks due to his rapid weight loss and immobility. The dietitian also educated the patient on the need for consistent meal intake as well as high protein intake due to risk of muscle loss and skin breakdown.

When the patient returned to MSICU a third time 15 days after admission, tube feedings were initiated by the MSS dietitian because the patient was intubated and no longer able to swallow. Caloric needs were calculated based on the Penn State 2003 equation. Estimation indicated that the patient would need 2215 kcal/day. Protein needs were estimated to be 1-1.2 g/kg, or 110-132 g per day, based on his most recent weight of 110.1 kg. Promote, an enteral nutrition formula, was administered at 70 mL/h to provide 1680 kcal and 105 g protein/day. A protein module, Pro-Stat, was administered once daily to provide an additional 100 kcal and 15 g of protein[11] (Figure 2). The dietitian aimed to provide lower than his estimated needs due to the infusion of propofol, a drug used for sedation and administered in a lipid emulsion, providing 1.1 kcal/mL as fat[12].

I saw the patient on the 18th day of admission when he was in the MSICU. Propofol had been discontinued because the patient no longer required sedation and was not meeting his estimated calorie or protein needs due to the loss of calories provided by this drug. I calculated his energy needs based on his most recent weight of 116 kg and used the Penn State 2003 equation. His estimated energy needs were 2375 kcal/kg. His BMI was 31.96, suggesting obesity. ASPEN guidelines recommend permissive underfeeding for intubated obese patients (22-25 kcals/kg ideal body weight)[13]; however, because he was formerly a wrestler and was an avid weight lifter, I used clinical judgment and provided a tube feeding based on the estimated needs determined with the Penn State 2003 equation. Due to his previous rapid weight loss, I also estimated his protein needs as 1.3-1.5 g/kg, or 151-174 g protein/day. I changed his enteral formula to Osmolite 1.2 at 70 mL/hr plus the protein module Pro-Stat 3 times a day to provide 2316 kcals and 138 g protein (Figure 2).

Adult Enteral Formulas									
Product name	Indications for use	Features	Nutrient values per	kcal/mL	PRO (g)	CHO (g)	FAT (g)	mL to meet 100% RDIs	Water (g)
Promote	Can be sole source; low caloric needs and those at risk for protein-energy malnutrition or pressure ulcers	Very-high-protein; 19% fat as MCT	1 L	1	62.5	130	26	1000	839
Osmolite 1.2	Can be sole source; increased protein and calorie needs; long-or short-term tube feeding	Concentrated calories, high protein, 20% fat as MCT	1 L	1.2	55.5	157.5	39.3	1000	820

Modular				
Product name	Nutrient Values	Kcal	PRO (g)	CHO (g)	FAT (g)
Pro-Stat Sugar Free 64	2 Tbsp (30 mL)	100	15	3	0

Table 2 Adult Enteral Formulas. Adapted from *Adult Enteral Nutrition Formulary*. Copyright 2012 Mission Hospital.

Due to the previous diagnosis of moderate malnutrition and excessive weight loss in a short time frame despite eating 75-100% of his diet order prior to intubation and placement of a feeding tube, I wondered if he had elevated calorie needs. To gain a more precise picture of his resting energy expenditure (REE), I ordered a metabolic cart study.

A metabolic cart determines REE using indirect calorimetry, the gold standard for determining energy requirements. The cart measures oxygen consumption and carbon dioxide production; every liter of oxygen consumed is equivalent to 5 calories of energy. The key to accurately determining REE is collecting all inspired and expired air. Metabolic cart studies are most accurate when the patient has consistent intake. A patient either needs to be NPO (nothing passed orally) or on a stable tube feeding when the cart study is performed. Studies are most easily performed when a patient is intubated, because his respiratory rate is steady. Because I had changed the formula of the tube feeding, I knew the cart study could only be performed in a small window of time. I spoke with the technician, who made an effort to see the patient before the new enteral formula was initiated. The metabolic cart study showed that instead of the estimated REE of 2375, the patient's actual REE was 3081 kcal.

I researched the metabolic process of GBS, and found that both hypermetabolism and hypercatabolism were major components of the syndrome, specifically within the first 2-4 weeks of onset. When a metabolic cart study is performed and a patient's BMI is above 30, guidelines recommend feeding the patient approximately 70% of his REE[13]. The enteral formula that I recommended provided the patient with 75% of his REE, and no further changes were made to his enteral nutrition order.

The week after I saw the patient, a urine urea nitrogen sample was collected and showed neither positive nor negative nitrogen balance. No net loss or gain of nitrogen in the urine indicated that the patient received an adequate amount of protein in his tube feeding, and no further muscle wasting occurred despite hypercatabolism.

V. Progress and prognosis of the patient

Early during his hospitalization, the patient became progressively weaker, beginning in his lower extremities followed by his upper extremities. Eventually, he experienced respiratory distress, was unable to clear secretions in his airways, and required intubation and enteral nutrition support.

The patient required ongoing respiratory support, so a tracheotomy tube was placed 19 days after admission. In addition, 20 days after admission, a PEG tube was placed for ongoing nutrition support. When the tracheotomy tube was placed, a culture of lung secretions showed that the patient had developed a hospital-acquired pneumonia. He was placed on several antibiotics.

The patient received typical treatments to alleviate the symptoms of GBS. Initially he was given a course of IVIG treatment, with no noted improvement. He then underwent 5 PLEX treatments. After PLEX concluded, limited improvement was noted. He was able to wiggle his toes and move his arms but was still unable to feed himself and had difficulty breathing on his own.

After enteral nutrition support was initiated, the patient did not continue to lose weight. Due to his prolonged intubation and his loss of swallowing functionality, he remained on tube feedings during the rest of his hospitalization.

The physicians remained hopeful that the patient would slowly regain his strength. When he was stable in the MSICU, he worked extensively with physical and occupational therapists. Due to his expected prolonged recovery and need for multiple allied health professional support, he was transferred to the LTACH at Mission Hospital. He was placed at LTACH one month after admission.

VI. Summary

This patient presented interesting challenges due to my limited knowledge of the effect of GBS on metabolism. The patient lost excessive weight during his hospitalization, meaning that he was underfed during his stay. This was due to receiving inadequate calories while consuming the regular hospital diet, and additionally from receiving a full liquid diet for 5 days. A regular oral diet at Mission Memorial Hospital on average provides 1800 calories and 91 grams of protein. Even when the patient was eating 100% of his meals, he could not meet his REE 3081 calories, causing him to lose weight rapidly. Working on this case underscored, for me, the importance of understanding disease processes, obtaining weekly weights of patients, and abating unnecessary weight loss during prolonged hospitalization.

References

1. Sudulagunta, Sreenivasa Rao, Sodalagunta, Mahesh Babu, Sepehrar, Mona, Khorram, Hadi, Bangalore Raja, Shiva Kumar, Kothandapani, Shyamala, Nidsale Sudarshan, Ranjitha. (2015). Guillain-Barré syndrome: clinical profile and management. http://doi.org/10.3205/000220
2. Nyati, K. K., & Prasad, K. N. (2014). Role of Cytokines and Toll-Like Receptors in the Immunopathogenesis of Guillain-Barré Syndrome. *Mediators of Inflammation*, *2014*, 1–10. http://doi.org/10.1155/2014/758639
3. Reece, J. B., Urry, L. A., Cain, M. L., Wasserman, S. A., Minorsky, P. V., Jackson, R. B., & Campbell, N. A. (2014). *Campbell biology* (Tenth edition). Boston: Pearson.
4. Mahan, L. K., Escott-Stump, S., Raymond, J. L., & Krause, M. V. (Eds.). (2012). *Krause's food & the nutrition care process* (13th ed). St. Louis, Mo: Elsevier/Saunders.
5. Guillain-Barré Syndrome Fact Sheet. (n.d.). Retrieved November 14, 2015, from http://www.ninds.nih.gov/disorders/gbs/detail_gbs.htm
6. Hughes, R. A., Hadden, R. D., Gregson, N. A., & Smith, K. J. (1999). Pathogenesis of Guillain-Barré syndrome. *Journal of Neuroimmunology*, *100*(1-2), 74–97.
7. Chiang, S., & Ubogu, E. E. (2013). The role of chemokines in guillain-barré syndrome: Chemokines in GBS. *Muscle & Nerve*, *48*(3), 320–330. http://doi.org/10.1002/mus.23829
8. Coleman, R. A. (2014). *Fasting and Starvation* (PowerPoint slides). Retrieved from https://sakai.unc.edu/portal
9. Roubenoff, R. A., Borel, C. O., & Hanley, D. F. (1992). Hypermetabolism and hypercatabolism in Guillain-Barré syndrome. *JPEN. Journal of Parenteral and Enteral Nutrition*, *16*(5), 464–472.
10. White, J. V., Guenter, P., Jensen, G., Malone, A., Schofield, M., Academy Malnutrition Work Group, … and the A.S.P.E.N. Board of Directors. (2012). Consensus Statement: Academy of Nutrition and Dietetics and American Society for Parenteral and Enteral Nutrition: Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition). *Journal of Parenteral and Enteral Nutrition*, *36*(3), 275–283. http://doi.org/10.1177/0148607112440285
11. *Adult Enteral Nutrition Formulary* [Brochure]. (2012) Asheville, NC: Mission Hospital
12. DeChicco, R., Matarese, L., Hummell, A. C., Speerhas, R., Seidner, D., & Steiger, E. (1995). Contribution of Calories from Propofol to Total Energy Intake. *Journal of the American Dietetic Association*, *95*(9), A25. http://doi.org/10.1016/S0002-8223(95)00438-6
13. Gottschlich, M. M., DeLegge, M. H., Guenter, P., & American Society for Parenteral and Enteral Nutrition. (2007). *The A.S.P.E.N. nutrition support core curriculum: a case-based approach : the adult patient*. Silver Spring, MD: American Society for Parenteral and Enteral Nutrition.