

Morgan Cox

**Case Study Number 4: Cirrhosis of the Liver with
Resulting Hepatic Encephalopathy**

November 1, 2012

FCS 420 – Medical Nutrition Therapy I

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I. Understanding the Disease and Pathophysiology

1. The liver is an extremely complex organ that has a particularly important role in nutrient metabolism. Identify three functions of the liver for each of the following:

a. Carbohydrate metabolism

- i. Glycogenolysis- the metabolic pathway through which glycogen is converted to glucose.
- ii. Gluconeogenesis – the metabolic pathway through which glucose is formed from noncarbohydrate sources.
- iii. Glycolysis – the anaerobic enzymatic conversion of glucose to lactate or pyruvate, which results in the production of energy in the form of ATP (adenosine triphosphate).

b. Protein metabolism

- i. Synthesis of serum proteins
- ii. Synthesis of prothrombin
- iii. Synthesis of urea

c. Lipid metabolism

- i. Lipogenesis – the synthesis of triglyceride from carbohydrates and proteins.
- ii. Fatty acid oxidation
- iii. Formation of lipoproteins (complexes of lipids and proteins that play a role in the transport and distribution of lipids).

d. Vitamin and mineral metabolism

- i. Formation of acetyl CoA from pantothenic acid
- ii. Hydroxylation of vitamin D to 25-OH D₃, which is the active form of vitamin D, also known as calcitriol.
- iii. Formation of coenzyme B₁₂.

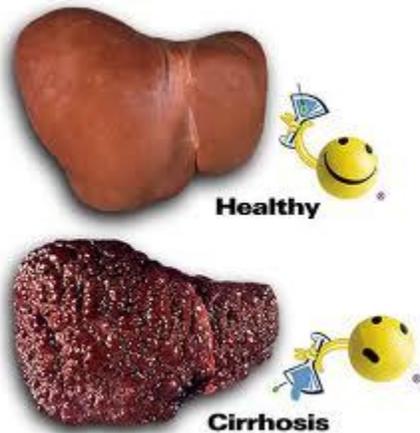
(Nelms, Sucher, Lacey, Roth, 2011).

2. The CT scan and liver biopsy confirm the diagnosis of cirrhosis. What is cirrhosis?

Cirrhosis is any pathological condition where fibrous connective replaces the healthy tissue in an organ. This is usually a consequence of inflammation or other injury (Nelms et al., 2011).

Rolfes, Pinna, and Whitney (2012) describe cirrhosis as an advanced stage of liver disease that includes the replacement of healthy liver tissue with massive amounts of scarring. This, in turn,

impairs the liver's ability function correctly and could ultimately result in liver failure (Rolfes, Pinna, Whitney, 2012). Appearance wise, a cirrhotic liver is firm and fibrous. It appears to look yellow and nodular. Within the cirrhotic liver, there is biliary obstruction. This means that there is an obstruction of bile flow (McCance et al., 2010). In the image below, you will see a healthy liver and a liver that is a cirrhotic liver (usermeds, 2012)



3. The most common cause of cirrhosis is alcohol ingestion. What are additional causes of cirrhosis? What is the cause of this patient's cirrhosis?

The most common causes of cirrhosis include the following:

- Hepatitis C
- Alcoholic liver disease
- Hepatitis C plus alcoholic liver disease
- Cryptogenic causes
- Hepatitis B, which may be concurrent with hepatitis D
- Genetic factors can increase susceptibility
- Other causes include:
 - Nonalcoholic steatohepatitis (NASH)
 - Autoimmune hepatitis
 - Primary biliary cirrhosis
 - Secondary biliary cirrhosis (associated with chronic extrahepatic bile duct obstruction)
 - Primary sclerosing cholangitis
 - Hemochromatosis
 - Cystic fibrosis
 - Wilson's disease
 - Alpha-1 antitrypsin deficiency

- Granulomatous disease (e.g. sarcoidosis)
- Type IV glycogen storage disease
- Drug-induced liver disease (e.g. methotrexate, alpha methyl dopa, amiodarone)
- Venous outflow obstruction (e.g. Budd-Chiari syndrome, veno-occlusive disease)
- Chronic right-sided heart failure
- Tricuspid regurgitation

The cause of this patient's cirrhosis is due to chronic hepatitis C. Hepatitis C (HCV) occurs when an individual is exposed to blood or body fluids from an infected person. The most common cause of this exchange is by sharing needles or being a receiver of blood clotting factors before 1987. Hemodialysis patients and infants born to infected mothers are also at an increased risk. Chronic hepatitis means that the virus lasts longer than six months. Some of the symptoms are fatigue, nausea, anorexia, and pain in the upper right quadrant (Nelms et al., 2011).

Hepatitis C is the most common cause of chronic liver disease. Unfortunately, there are no vaccines available to shield from HCV infection. The most recent findings of the HCV infections involve individuals who contracted the hepatitis C many, many years ago (Rolfes et al., 2012).

4. Explain the physiological changes that occur as a result of cirrhosis.

Initial signs of cirrhosis of the liver include nausea, vomiting, anorexia, and distension of the abdomen.

- a. Jaundice – causes the body tissues to have a yellow tint. This is usually the result of elevated bilirubin (pigment from hemoglobin resulting from the taking apart of red blood cells) concentration in the extracellular fluids, either unconjugated (also called indirect) or conjugated (direct) bilirubin. In other words, bilirubin is not conjugated and excreted into the bile; therefore, it is released into the body tissues which causes the tissues and whites of the eyes to turn yellow. (Nelms et al., 2011). Below is an image of a jaundice patient. Notice the yellowing of the eyes (iahealth, 2012).

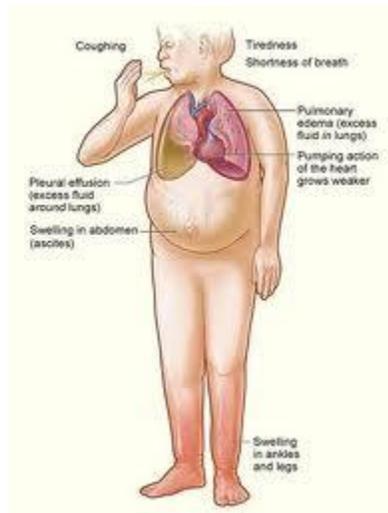


- b. Portal Hypertension – elevated blood pressure in the portal vein which is the vein in the abdominal cavity that drains blood primarily from the gastrointestinal tract and spleen. The blood flow is obstructed by the

nodular regeneration of the liver. This regeneration can cause the blood to “back up” and/or be redirected into smaller veins and capillaries that cannot withstand the high blood pressure and could, in turn, rupture and cause hemorrhaging. The veins affected are that of the esophagus and facial veins. It is usually defined as a portal pressure gradient (the difference in pressure between the portal vein and the hepatic veins) of 5 mm Hg or greater (Nelms et al., 2011). Portal hypertension contributes to congestive splenomegaly by increasing intrasplenic blood pressure (McCance, Huether, Brashers, Rote, 2010).

- c. Ascites – this is a primary symptom and complication due to portal hypertension. Ascites is the accumulation of fluid within the peritoneal cavity of the abdominal cavity (Nelms et al., 2011). Ascites is also a consequence of the kidneys retaining sodium and water. Also, there is a reduction of albumin synthesis within the diseased liver (Rolfes et al., 2012). There is an increase in renal absorption of sodium. This is the result of increased aldosterone production and decreased inactivation of aldosterone. A reduction in renal blood flow and the consequent decrease in renal filtration rate play a role in the body’s retention of sodium (Nelms et al., 2010).

Below is a model of a human body with ascites (Adam, 2012)



- d. Encephalopathy – Hepatic encephalopathy is a syndrome of impaired mental status and abnormal neuromuscular function that results from major liver failure. Important contributing factors are the degree of liver failure, the diversion of portal blood through the venous systemic circulation, bleeding from varices, and exogenous factors such as

sepsis. Another contributing factor includes a buildup of nitrogen in the blood because the liver cannot detoxify the body of nitrogenous waste produced by exogenous protein and send it to the urea cycle (Nelms et al., 2011).

- e. Collaterals and Gastroesophageal Varices – blood that is diverted to the smaller blood vessels around the liver develop into collaterals. These could be located in the gastrointestinal tract and in other regions that are located near the abdominal wall. Pressure begins to build and the collateral vessels become enlarged and engorged. They become varices and they develop in the esophagus and in the stomach. These varices that develop could rupture because they have very thin walls. These bulging walls sometimes swell into the lumen and if they rupture, it could cause extensive bleeding that could ultimately end in death. The blood loss is intensified by the liver’s reduction in the making of blood-clotting factors (Rolfes et al., 2012).
- f. Elevated Blood Ammonia Levels – This is due to the fact that the liver is unable to process the ammonia that is produced by bacterial action on unabsorbed dietary protein within the colon. In a normal situation, the liver would extract this ammonia from the blood and convert it to urea which would be excreted by the kidneys. Ammonia could be deposited within brain tissues and potentially cause encephalopathy (see “d” above) (Rolfes et al., 2012).
- g. Malnutrition and Wasting – Patients suffering from cirrhosis could potentially develop protein energy malnutrition (PEM). This could cause the patient to waste away. This could be caused by a reduction of nutrient intake (i.e. anorexia, altered mental status, early satiety due to ascites), malabsorption or nutrient loss (i.e. diarrhea, fat malabsorption), and altered metabolism or an increase in nutrient needs (i.e. hypermetabolism, impaired protein synthesis) (Rolfes et al., 2012).
- h. Easy bruising – due to a decrease in blood clotting factors because of a diseased liver (Mahan & Escott-Stump, 2008)
- i. Hepatorenal syndrome (HRS) which is the development of renal failure in patients with advanced chronic liver disease.
- j. Hyponatremia – occurs because of a decreased ability to excrete water resulting from the persistent release of antidiuretic hormone, sodium losses via paracentesis, excessive diuretic use, or overly aggressive sodium restriction (Mahan, Escott-Stump, 2008).
- k. Glucose alterations – some patients suffer from hyperinsulinism and hypoglycemia (Mahan, Escott-Stump, 2008).

1. Fat malabsorption – this occurs because of liver disease because there is a reduction in the secretions of bile salts that aid in the digestion of fat (Mahan, Escott-Stump, 2008).

5. List the signs and symptoms of cirrhosis, and relate each of these to the physiological changes discussed in question 4.

- a. Jaundice – yellowish tint to the body tissues as a result of elevated bilirubin concentration in the extracellular fluids (Nelms et al., 2011).
- b. Portal Hypertension – elevated blood pressure in the portal vein. The primary symptom is ascites which is the accumulation of fluid within the abdominal cavity and is recognized as a distension of the abdomen (Nelms et al., 2011).
- c. Hepatic Encephalopathy – Lethargy, fatigue, confusion, disorientation, changes in mental status and personality, and neuromuscular changes (Nelms et al., 2011).
- m. Collaterals and Gastroesophageal Varices - The varices that develop could rupture because they have very thin walls. These bulging walls sometimes swell into the lumen and if they rupture, it could cause extensive bleeding that could ultimately end in death. The blood loss is intensified by the liver's reduction in the making of blood-clotting factors (Rolfes et al., 2012). These varices could be caused by Hepatorenal syndrome as well (Nelms et al., 2011).
- d. Elevated Blood Ammonia Levels – This is due to the malfunctioning of the liver and a sign of elevated blood ammonia levels would include a chemistry lab value of Ammonia above 33 U/L.
- e. Malnutrition and Wasting – the body could become malnourished and steatorrhea and deficiencies of the fat-soluble vitamins and some minerals could occur. Other nutrient deficiencies could occur as well. Additional nutrient losses could be the result from diarrhea, vomiting, and gastrointestinal bleeding (Rolfes et al., 2012).
- f. Easy bruising – due to a decrease in blood clotting factors because of a diseased liver (Mahan & Escott-Stump, 2008)
- g. Itching
- h. Hypoglycemia and hyperinsulinism

6. After reading the patient's history and physical, identify her signs and symptoms that are consistent with the diagnosis.

After reading Mrs. Wilcox's history and physical, the signs and symptoms that are consistent with her diagnosis of cirrhosis that is secondary to chronic hepatitis C include the following:

- Tiredness
- Fatigue/weakness
- Yellowing of the skin (possible Jaundice)
- Anorexia
- Nausea and Vomiting
- Weight loss of 10 pounds
- Bruising of skin not related to injury on lower arms and legs
- Enlarged esophageal veins
- Telangiectasias on chest
- Mild distension of the right upper quadrant of the abdomen
- Splenomegaly (spleen enlargement) without hepatomegaly (liver enlargement)
- Family history of cirrhosis (paternal grandfather)

7. Hypoglycemia is a symptom that cirrhotic patients may experience. What is the physiological basis for this? Is this a potential problem? Explain?

Cirrhosis of the liver increases an individual's energy expenditure because of vasodilation and an expansion in blood volume. Because of this phenomenon, blood sugar levels can be erratic in patients with cirrhosis of the liver. When cirrhosis reaches levels at which 80% of hepatocytes (liver cells) are not functional, hypoglycemia is a common incidence due to hyperinsulinemia (high amounts of insulin in the blood). The potential problem is the lack of sufficient amounts of glycogen reserves as a result of liver damage may cause hypoglycemia after an overnight fast and may also prompt recruitment of amino acids from the skeletal muscles for gluconeogenesis (the metabolic pathway through which glucose is formed from noncarbohydrate sources). Low-blood glucose store could pose a problem because this could cause a patient to suffer from fatigue, heart palpitations, tremors, and sweating. This is the body's reactions to hypoglycemia because it is slowing down its processes. However, 40%-50% of all patients with end-stage liver disease suffer from insulin-resistant diabetes mellitus which means hyperglycemia may also be apparent (Nelms et al., 2011).

Fasting hypoglycemia can occur because of the decreased ability of glucose from glycogen in addition to the failing gluconeogenic capacity of the liver (Mahan, Escott-Stump, 2008).

8. What are the current medical treatments for cirrhosis?

For the management of cirrhosis (no cure exists for cirrhosis) the following is recommended:

- Minimize further deterioration of liver function through the withdrawal of toxic substances, alcohol, and drugs.
- Correction of nutritional deficiencies with vitamins and nutritional supplements and a high-calorie and moderate-to high-protein diet.
- A mechanically soft diet to avoid rupturing varices in esophagus.
- Treatment of ascites and fluid and electrolyte imbalances.
 - Restrict sodium and water intake, depending on the amount of fluid retention
 - Bed rest to aid in diuresis.
 - Diuretic therapy, frequently with spironolactone, a potassium-sparing diuretic that inhibits the action of aldosterone on the kidneys. Furosemide (Lasix), a loop diuretic, may also be used in conjunction with spironolactone to help balance potassium depletion.
 - Abdominal paracentesis to remove fluid and relieve symptoms; ascetic fluid may be ultra-filtered and reinfused through a central venous access.
 - Administration of albumin to maintain osmotic pressure
- Transjugular intrahepatic portosystemic shunt (TIPS), an interventional radiologic procedure, may be performed in patients whose ascites are resistant to other forms of treatment. TIPS is a percutaneously created connection within the liver between the portal and systemic circulations. A shunt is placed to reduce the portal pressure in patients with complication related to portal hypertension.
- Symptomatic relief measures, such as pain medication and antiemetics.
- Treatment of other problems associated with liver failure and/or liver cancer. Administration of lactulose or neomycin for hepatic encephalopathy.
- Orthotropic liver transplantation may be necessary.
(Nettina, 2006).

9. What is hepatic encephalopathy? Identify the stages of encephalopathy and outline the major theories regarding the etiology of this condition.

Hepatic encephalopathy is a condition that is usually caused by cirrhosis of the liver and portal hypertension in which toxins produced by the gut pass into the systemic circulation and damage brain cells. This results in impaired cognition, tremor, and a decreased level of consciousness (McCance et al., 2010).

Staging Scales for Hepatic Encephalopathy

West Haven Scale		
Stage	West Haven Criteria	Adapted-West Haven Criteria
0	<ul style="list-style-type: none"> No abnormality detected 	Alert and attentive (oriented in time and space) without signs of encephalopathy (neither dysarthria, ataxia, flapping tremor or obvious decrease in the speed of mental processing)
1	<ul style="list-style-type: none"> Trivial lack of awareness Euphoria or anxiety Shortened attention span Impairment in performance of addition 	Alert and attentive, but with at least one of the following signs: <ul style="list-style-type: none"> Dysarthria (motor or speech disorder) Ataxia (loss of full or partial bodily movements) Flapping tremor Obvious decrease in the speed of mental processing
2	<ul style="list-style-type: none"> Lethargy or apathy Minimal disorientation for the time or place Subtle personality change Inappropriate behavior Impaired performance of subtraction 	Awake but inattentive: disoriented, somnolent, easy to distract, unable to perform easy mental tests (addition, subtraction, remember a list of numbers). Patient's speech is easy to understand.
3	<ul style="list-style-type: none"> Somnolence (drowsiness) to semi-stupor but responsive to verbal stimuli Confusion Gross disorientation 	Marked somnolence or psychomotor agitation. Speech is difficult to understand.
4	<ul style="list-style-type: none"> Coma (unresponsive to verbal or noxious stimuli) 	Coma – The patient does not speak and does not follow simple commands (such as raising an arm or opening the mouth).

(Nelms et al., 2011).

The pathogenesis of hepatic encephalopathy is unknown, though research points to the inability of the liver to metabolize products that are toxic to the brain. There are three major hypotheses that try to explain the impairment of the neurotransmission:

1. The ammonia hypothesis
2. The synergistic neurotoxin hypothesis
3. The false neurotransmitter

(Nelms et al., 2011).

10. Protein-energy malnutrition is commonly associated with cirrhosis. What are the potential causes of malnutrition in cirrhosis? Explain each cause.

Malnutrition is common for individuals with cirrhosis of the liver. Patients with cirrhosis may be unable to consume adequate amounts of food due to early satiety caused by the side effects of ascites on the stomach's capacity to expand. They may also experience impaired nutrient digestion and absorption due to portal hypertension, decreased pancreatic enzyme production and/or secretion, and villus atrophy. Important micro and macro nutrients may not be absorbed if the patient is suffering from diarrhea and nausea/vomiting. Cirrhosis increases energy expenditure because of vasodilatation and expanded blood volume (Nelms et al., 2011).

There is a decrease in fat absorption in patients with cirrhosis. There is a decrease in the secretion of bile salts which aid in the digestion of food, especially fat. This causes the stool to become greasy and it will float in water. Thinking critically, fat is not water-soluble, so that means that it does not mix with water. If fat comes into contact with water, it will float and remain separate from the water. This is why the stool will float; it contains fat that was not absorbed (Mahan, Escott-Stump, 2008).

Studies have shown that individuals with diseased livers need more protein because they suffer from an increase of nitrogen loss (Mahan, Escott-Stump, 2008).

In summary, the following are etiologies that ultimately lead to the malnutrition of a patient with cirrhosis of the liver:

- Maldigestion or malabsorption
 - Restricted diets
 - Altered metabolism
 - Nausea and vomiting
 - Early satiety or dysgeusia
 - Anorexia (inadequate oral intake)
- (Mahan, Escott-Stump, 2008).

II. Understanding the Nutrition Therapy

11. Outline the nutrition therapy for the following stages of cirrhosis with the rationale for each:

Diagnosis	Sodium	Potassium	Protein	Micronutrients	Fluid	Other Modifications
Stable cirrhosis	Low sodium diet is recommended – 1,000 mg or less.	Potassium intake should be monitored if a potassium-wasting diuretic (such as furosemide) is used.	0.8 to 1g/kg per day is the mean protein requirement to achieve nitrogen balance in patients with stable cirrhosis.	Vitamin and mineral deficiencies are common due to effects of other illnesses that accompany cirrhosis. For best results, a multivitamin may be administered, but the fat soluble vitamins should be in water soluble form. In some cases, fat-soluble vitamins may need to be increased 2-fold.	Fluids are not usually restricted.	Alcohol strictly prohibited. Moderate fat intake in attempt to prevent steatohhrea.
Cirrhosis with acute encephalopathy	Low sodium diet is recommended – 1,000 mg or less	Potassium intake should be monitored if a potassium-wasting diuretic (such as furosemide) is used.	Protein should only be restricted with severe forms of encephalopathy. Greater amounts of protein from vegetable and dairy sources have been	Vitamin and mineral deficiencies are common due to effects of other illnesses that accompany cirrhosis. For best results, a multivitamin may be administered, but the fat soluble vitamins should be in water	Fluids are not usually restricted.	Probiotics and synbiotics(good sources of bacteria and fermentable fibers) can be used to treat hepatic encephalopathy. Alcohol strictly prohibited.

			<p>recommended for patients with mild encephalopathy. Ideally, protein intake should be no more than 30 to 50 grams. If coma is approaching, it is recommended for patient to consume 1 to 15 grams of protein.</p>	<p>soluble form. In some cases, fat soluble vitamins need to be increased 2-fold.</p>		<p>Moderate fat intake in attempt to prevent steatohhrea.</p>
<p>Cirrhosis with ascites and esophageal varices</p>	<p>Sodium restriction of 2 grams per day</p>	<p>Potassium intake should be monitored if a potassium-wasting diuretic (such as furosemide) is used.</p>	<p>Extensive low-protein exchange lists have been developed to treat liver failure and cirrhosis.</p>	<p>Use of a multivitamin/mineral supplements as indicated by nutrition assessment and blood chemistry information is appropriate. Patients with esophageal varices may find it easier to ingest supplements that are in liquid form. In some cases, fat soluble vitamins needs to be consumed 2-fold.</p>	<p>Fluid restriction may be required when ascites is accompanied by a low concentration of serum sodium. Sodium levels below 128 milliequivalents per liter = 1200-1500 mL/day. Sodium levels below 125 milliequivalents per</p>	<p>For esophageal varices, a mechanically soft diet may be recommended as a preventive measure to reduce risk of bleeding. Alcohol strictly prohibited. Moderate fat intake in attempt to prevent steatohhrea.</p>

					liter = 1000-1200 mL/day.	
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(Lutz, Przytulski, 2011) (Rolfes et al., 2012) (Mahan, Escott-Stump, 2008) (Nelms et al., 2010).

III. Nutrition Assessment

12. Measurements used to assess nutritional status may be affected by the disease process and not necessarily be reflective of nutritional status. Are there any components of nutrition assessment that would be affected by cirrhosis? Explain.

Many of the methods often used for anthropometric and biochemical assessment of nutritional status are not reliable for individuals with cirrhosis. This could be the result of edema and ascites. With these two conditions, a patient's dry weight may not be able to be measured accurately or may not be able to be obtained all together. Electrical impedance analysis may also not be accurate. Because of hepatic dysfunction, many of the liver protein lab values that are used to determine visceral protein could be abnormal. In order to avoid these miscalculations, the European Society for Parenteral and Enteral Nutrition recommended that the following are used to determine a cirrhosis patient's anthropometrics accurately:

- Subjective Global Assessment (SGA)
- Measurement of mid-arm muscle circumference (MAMC)
- Measurement of mid-arm circumference (MAC)
- Triceps skin fold thickness test (TST)

(Nelms et al., 2010).

A. Evaluation of Weight/Body Composition

13. Dr. Horowitz notes Ms. Wilcox has lost ten pounds since her last exam. Assess and interpret Ms. Wilcox's weight.

Ms. Wilcox's current body weight is 125 pounds. Her usual body weight (the weight at which she was at six months ago at her last doctor's appointment) was 135 pounds. With this information, we can state that Ms. Wilcox is at 93% of her usual body weight. ($125 \div 135 \times 100$).

Ms. Wilcox's ideal body weight is 145 pounds. She is currently twenty pounds too light.

Ms. Wilcox's current BMI is 18. This is considered underweight because her BMI is less than 18.5. Her usual BMI of 135 pounds is 20. This is a normal BMI that represents a healthy weight.

Ms. Wilcox's low weight could be due to her lack of appetite (anorexia) and possible malabsorption. All of this is due to the exacerbation of her cirrhosis of the liver (Rolfes et al., 2012).

14. Identify any nutrition problems using the correct diagnostic term.

Because Ms. Wilcox has not eaten in the past two days, her 24-hour recall diet only consists of sips of water and a Diet Coke. With the information provided, I would identify the nutritional problems as follows:

- Inadequate energy intake (NI – 1.4)
- Inadequate oral food/beverage intake (NI – 2.1)
- Inadequate fluid intake (NI – 3.1)
- Because she is suffering from hepatic encephalopathy, she should limit her protein intake. If she does consume protein, it should be plant and/or dairy bases. Inappropriate intake of amino acids (NI – 5.7.3) and inadequate protein-energy intake (NI – 5.3).
- It was stated that her general food intake consists of take-out at night. This type of food is loaded with sodium. A diet with a low amount of sodium is recommended. Excessive mineral intake of sodium (NI – 5.10.2). (Nelms et al., 2010).
- Underweight (NC – 3.1) and Involuntary weight loss (NC – 3.2)

B. Calculation of Nutrient Requirements

15. Calculate the patient's energy and protein needs.

BEE for adult women = $655 + (9.6 \times 57 \text{ kg}) + (1.8 \times 175 \text{ cm}) - (4.7 \times 26 \text{ y.o.})$.

BEE = 1,395 kcal

TDE = $1,395 \times 1.5$ activity factor = **2,093 kcal**

Add 500 kcal to TDE because underweight = **2, 593 kcal**

Protein needs not considering hepatic encephalopathy = $2,593 \div 200 \times 6.25 =$ **81 g protein**

Protein needs considering her hepatic encephalopathy (which will be lower) 0.8 g per kg of body weight each day = $0.8\text{g} \times 57 \text{ kg} =$ **46g protein per day**

16. What guidelines did you use and why?

I used the Harris-Benedict Formula. I used this formula because when I calculated using other formulas, I felt that they did not provide her with enough calories. She is underweight and the Harris-Benedict allowed me to add that factor in (Rolfes et al., 2012).

I considered calculating her required nitrogen by dividing the TDE by 200 for maintenance and then multiplying it by 6.25. When I saw this value, I considered her hepatic encephalopathy. According to Nelms et al (2010), a patient with this occurring should limit their protein intake (Nelms et al., 2010). Mahan and Escott-Stump (2008) informed me that the protein consumed should be plant or dairy based. Her protein intake should be lowered, so they gave me the equation - 0.8 grams of protein for every kilogram of body weight (Mahan, Escott-Stump, 2008).

C. Intake Domain

17. Evaluate the patient's usual nutritional intake.

Ms. Wilcox's diet was altered due to her lack of an appetite.

Ms. Wilcox's usual intake consisted of calcium-fortified orange juice for breakfast. This is an excellent choice because it provides her with a serving of fruit, vitamin C, and calcium. For lunch, she consumed soup and crackers and a Diet Coke. This provided her with a decent amount of carbohydrates. For supper, she could consume Chinese or Italian take-out. This would contain some protein that is needed for her diet. This is the positive outlook on her usual intake.

The negative outlook is she needs to consume more for breakfast. The orange juice is good, but it needs to be accompanied with some complex carbohydrates and a small amount of protein from dairy, like milk or yogurt. Her lunch contains a lot of sodium from the soup (especially if it's from a can) and the crackers (especially if they are saltine crackers). The take-out that she consumes is loaded with sodium. This is unhealthy for all individuals, especially for Ms. Wilcox. She would be better off sautéing a small amount of chicken and a lot of vegetables for dinner at night. By examining her beverages, she did not consume any water for her usual intake. She only consumed orange juice, and two Diet Cokes.

I would have Ms. Wilcox increase her intake of fruits, vegetables, dairy, and water. She must obtain her protein from vegetables and dairy. I would up her servings to help her gain the weight that she has lost.

The first order of business, though, is to help her obtain an appetite again.

18. Her appetite and intake have been significantly reduced for the past several days. Describe the factors that may have contributed to this change in her ability to eat.

Individuals with cirrhosis often suffer from nausea and vomiting (especially the patients with ascites and gastrointestinal symptoms). This greatly hinders their food intake. Cirrhotic patients sometimes suffer from fatigue. Fatigue could impede on their ability to prepare food and reduce their overall interest in food. Those who have been diagnosed with cirrhosis of the liver

experience abdominal pain, nausea, bloating, and are found to have altered gut motility, This leads to the development of functional dyspepsia. These are known to lead to an increased severity of gastrointestinal symptoms that are associated with weight loss (Nelms et al., 2010).

Mrs. Wilcox is suffering from fatigue, anorexia, nausea/vomiting, and weakness. From the information provided, I could conclude that she is too fatigued and weak to cook and/or eat, she is suffering from anorexia which means she has no appetite and no desire to eat, and she is suffering from nausea and vomiting which causes her to cease her food intake and possibly become malnourished (Rolfes et al., 2012).

19. Why was a soft, 4-g Na, high-kcalorie diet ordered? Should there be any other modifications?

A soft diet was called for because the food would be easy to swallow and digest. Her oral foods need to be easy to swallow because of the risk that cirrhotic patients have in developing esophageal varices, which are located in the esophagus and are vulnerable to rupture and bleeding (Mahan-Escott-Stump, 2008)(Rolfes et al., 2012).

The low-sodium (4 grams per day) diet order was because of the risk of cirrhotic patients developing ascities. Ascites is an accumulation of fluid within the abdominal cavity. Along with the fluid buildup, there is also an increase in sodium retention. Reducing the sodium intake will keep the fluid buildup and sodium retention from getting out of control (Nelms et al., 2010).

The high-kcalorie diet was orderd because Mrs. Wilcox is approximately 20 pounds underweight. She has lost 10 pounds since her last doctor's visit six months ago. This higher-kcalorie diet will assist her in putting weight back on and keep her from losing more weight.

Other modifications that I would make would be to inform her that her protein should come mostly from vegetable and dairy sources because these sources are higher in branch-chain amino acids. I would also modify the 4-gram sodium (Na) recommendation because it is suggested that patients with cirrhosis consume less than two grams of sodium per day. I would maintain the high calorie recommendation due to her current weight loss of ten pounds. I would make sure to recommend that Ms. Wilcox obtain no more than 30 percent of those calories from fat (Nelms et al., 2010).

20. This patient takes multiple dietary supplements. Identify the possible rationale for each and identify any that may pose a risk for someone with cirrhosis.

Vitamin E is thought to act as an antioxidant and protect red blood cell membranes against hemolysis.

Calcium aids in bone and teeth health. It also reduces the total acid load in the GI tract, elevates gastric pH in order to reduce pepsin activity, strengthens gastric mucosal barrier, and increases esophageal sphincter tone.

Vitamin D encourages absorption and utilization of calcium and phosphate which helps to regulate homeostasis.

Multivitamin/mineral supplements stimulate retinal function, bone growth, reproduction, and aids in the integrity of the epithelial and mucosal tissues.

(Drug Handbook, 2006).

Patients that have cirrhosis of the liver experience an impairment of the absorption of many fat-soluble vitamins such as A, D, E, and K due to the reduction of intraluminal bile salt concentrations. Ms. Wilcox is taking a supplement that provides her with extra Vitamin D and E which can be beneficial with her recent diagnosis. With her new multivitamin/ mineral supplement, the recommendation is that she needs to be sure that she does not exceed her recommended daily intake of potassium (Lutz and Przytulski, 2011).

Milk Thistle aids with dyspepsia, liver damage from chemicals, supportive therapy for inflammatory liver disease and cirrhosis, and for the loss of appetite. The patient taking milk thistle should not take the herb for liver inflammation or cirrhosis before seeking appropriate medical evaluation because doing so may delay the diagnosis of a potentially serious medical condition (Drug Handbook, 2006). Though milk thistle is generally well tolerated and is an alternative medicine for cirrhosis, it can have a laxative effect on the body. It can also cause an allergic reaction. It was stated in Ms. Wilcox's patient admission database that she suffers from allergies; this could be a culprit of her allergy symptoms (Nelms et al., 2010).

Chicory contains inulin, which may help with weight loss, constipation, improving bowel function, and overall general health. Chicory has been associated with overall liver health as well (Find a vitamin..., 2012).

Ginger helps with the treatment of liver toxicity. It is also an anti-inflammatory and antispasmodic herb (Drug Handbook, 2006).

D. Clinical Domain

21. Examine the patient's chemistry values. Which labs support the diagnosis of cirrhosis? Explain their connection to the diagnosis.

Albumin, total protein, and prealbumin are all low and could point to a protein deficiency and/or malnutrition/malabsorption. Her blood glucose is high. Cirrhosis increases energy expenditure because of vasodilation and expanded blood volume. This can cause blood sugar levels to be erratic. Ms. Wilcox's ammonia level was on the high-side of normal. This lab value is increased in cirrhotic patients, especially ones with hepatic encephalopathy. Her bilirubin was high. Patients with hepatitis and/or cirrhosis, the liver is unable to process bilirubin and blood levels of this substance are elevated. A high level of bilirubin is also indicative of biliary obstruction and red blood cell hemolysis. Alkaline phosphate (Alk phos) is an increased lab value in Ms. Wilcox. Increased alk phos activity occurs in hepatic disease and in chronic obstruction of the biliary duct. Her aspartate aminotransferase (AST) is increased. This lab value is increased due to liver disease as well. Her alanine aminotransferase (ALT) is increased. This lab value, when tested, is the most sensitive test to detect hepatocellular injury secondary to exacerbation of infectious hepatitis and in acute hepatocellular damage (Nelms et al., 2010). Ms. Wilcox's creatine phosphokinase (CPK) was elevated. CPK is increased in when creatine kinase is released from damaged cells in which it is store, so conditions that affect the brain, heart, or skeletal muscle and cause cellular destruction can result in and increased CPK lab value. Ms. Wilcox's HDL cholesterol was low. This is the "good" cholesterol, so when this is lowered, it can signify an increased risk of developing heart disease. Finally, Ms. Wilcox's TG, or thyroglobulin, was high. A high TG level indicates tumor recurrence and is related to disorders such as inflammation, structural damage, and cancer (Van Leeuwen, Poelhuis-Leth, Bladh, 2011).

22. Examine the patient's hematology values. Which are abnormal, and why?

Ms. Wilcox's red blood cell count was low. This could be due to a chronic inflammatory disease, hemorrhage, or a nutritional deficit. Her hemoglobin was low. This lab value could be decreased due to cirrhosis, chronic disease, fluid retention, splenomegaly, anemia, or nutritional deficit. In Ms. Wilcox's situation, her low hemoglobin level is more than likely die to cirrhosis. Ms. Wilcox's hematocrit level was decreased. A decreased hematocrit could be the result of cirrhosis, chronic disease, fluid retention, splenomegaly, nutritional deficit, and anemia. The low hemoglobin and hematocrit levels could cause the body to feel weak and fatigued. Ms. Wilcox's MCV, or mean corpuscular hemoglobin, was elevated. This lab is used to evaluate the size, shape, weight, and hemoglobin concentration of the cell. It is used to diagnose and monitor the therapy for diagnoses such as iron-deficiency anemia. This lab value is increased in individuals with liver disease, alcoholism, pernicious anemia, and antimetabolic therapy, which inhibits vitamin B₁₂ and folate. Ms. Wilcox's ferritin was low. This lab value is low due to conditions like hemodialysis and iron-deficiency anemia that decrease iron stores. The last abnormal lab value was prothrombine time (PT). This was increased. This lab value can be increased due to biliary obstruction, disseminated intravascular coagulation, prolonged liver disease (cirrhosis), and/or vitamin K deficiency. We can conclude that many of the abnormal values listed are high

or low due to the exasperation of Mr. Wilcox’s cirrhosis and/or malnutrition/malabsorption of vital micro and macro nutrients – especially protein (Van-Leeuwen et al., 2011).

23. Does she have any physical symptoms consistent with you findings?

Ms. Wilcox does have physical symptoms that are consistent with my findings. She is fatigued and weak. This is evident through her low albumin, total protein, prealbumin, and glucose lab values. The lab values also consistent with fatigue and weakness that points toward anemia include her red blood cell count, hemoglobin, mean corpuscular hemoglobin, and hematocrit value. She is protein-energy malnourished. Her bilirubin is high, which is why there is a yellowing of her skin (Jaundice). Her liver panel shows the worsening of her cirrhosis, which explains the anorexia and nausea and vomiting, which are signs and symptoms of cirrhosis of the liver (Van-Leeuwen et al., 2011).

24. What signs and/or symptoms would you monitor to determine further liver decomposition?

The patient should be observed for tolerance to feedings, amounts of nutrients that are consumed, weight changes or fluctuations, laboratory values, and cognitive status (Nelms et al., 2010). It is important to observe Ms. Wilcox and make sure that she does not develop steatorrhea, which is the presence of fat in the stool. This happens in cirrhosis patients, especially if there is disease involving the injury to bile ducts and/or obstruction within the liver (Mahan, Escott- Stump, 2008). It is very important to make sure Ms. Wilcox does not become malnourished. Sometimes, enteral or parenteral feedings must be considered (Nelms, et al., 2010). A very important lab value that must be monitored is her ammonia level. This could ultimately put her in a coma. That is the last stage of hepatic encephalopathy. The first signs and symptoms that must be observed are mild confusion, agitation, irritability, sleep disturbance, and decreased attention span (Mahan, Escott-Stump, 2008).

25. Dr. Horowitz prescribes two medications to assist with the patient’s symptoms. What is the rationale for these medications, and what are the pertinent nutritional implications of each?

Rationale for Rx	Nutritional Implications
Spironolactone – The purpose of this medication is to increase the excretion of sodium and water from the body. It is also a potassium-sparing diuretic and an aldosterone receptor antagonist. .	This medication may increase BUN and potassium levels. It may decrease sodium levels. This drug may interact with potassium-containing salt substitutes and potassium-rich foods, like citrus fruits and tomatoes. This drug may increase the risk of developing hyperkalemia, which

	means there is too much potassium within the blood stream. It is recommended that there be a decrease in calories and sodium intake. Natural licorice should be avoided. There could possibly be an increase in thirst, nausea, vomiting, diarrhea, and gastritis.
Propranolol – This drug is a nonselective beta blocker that reduces the cardiac oxygen demand by blocking catecholamine-induced increases in heart rate, blood pressure, and the overall force of myocardial (heart) contraction. This drug also suppresses rennin secretion and prevents the vasodilation of the cerebral arteries.	This drug may increase BUN levels.

(Drug Handbook, 2006)

26. If the patient’s condition worsens (e.g., acute varices, bleeding, progression to hepatic encephalopathy), the following medications could be used. Describe each drug classification and mechanism.

Drug	Classification	Mechanism
Vasopressin	Antidiuretic Hormone	Increases the permeability of the renal tubular epithelium to adenosine monophosphate and water. The epithelium promotes reabsorption of water and produces concentrated urine.
Lactulose	Laxative, antihyperammonemic, a synthetic sugar	Produces an osmotic effect within the colon. This results in distention and promotes peristalsis. It also decreases ammonia, probably as a result of bacterial degradation, which lowers the pH of the contents of the colon.
Neomycin	Antibiotic	Interferes with the protein synthesis of

		bacteria
Ferrous sulfate	Iron supplement	Provides elemental iron, which is an essential component in the formation of hemoglobin.
Bisacodyl	Laxitive	Stimulant laxative that increases peristalsis. It has a direct effect on smooth muscle of the intestine. This could be completed by irritating the muscle or stimulating the colonic intramural plexus. This drug also promotes fluid accumulation in colon and small intestine.
Docusate	Stool softener	Stool softener that reduces the surface tensions of interfacing liquid contents of the bowel. This promotes the incorporation of additional liquid into stools. This forms a softer mass.
Diphenhydramine	Antihistiamine	Competes with histamine for H ₁ -receptor sites on effector cells. Prevents, but doesn't reverse, histamine-mediated responses.

E. Behavioral-Environmental Domain

27. What is the recommendation regarding alcohol intake when cirrhosis is caused by the hepatitis C virus?

The point eliminating alcohol intake in individuals with hepatitis C is to reduce the strain on the liver as much as possible and supply it with as many beneficial nutrients as one can. The recommendation regarding alcohol intake is to avoid it all together. By doing so, this would further reduce damage to the liver (Nelms et al., 2010).

IV. Nutrition Diagnosis

28. Select two high-priority nutrition problems and complete the PES statements for each.

- (1) Underweight due to lack of appetite and nausea/vomiting as evidenced by a current body mass index (BMI) of 18.
- (2) Involuntary weight loss due to anorexia and nausea/vomiting as evidenced by a weight loss of ten pounds in the last six months.

V. Nutrition Intervention

29. Ms. Wilcox is discharged on a soft, 4-g Na diet with a 2-L fluid restriction. Do you agree with this decision?

I would modify the 4-gram sodium (Na) recommendation because it is suggested that patients with cirrhosis consume less than two grams of sodium per day in order to reduce the risk of the patient of developing ascites (Mahan, Escott-Stump, 2008).

I would lower her fluid restriction to 1.5 L (1500mL). This, along with a low-sodium diet, will reduce her risk in developing ascites (Nelms et al., 2010).

30. Ms. Wilcox asks if she can use a salt substitute at home. What would you tell her?

I would tell her that salt substitutes should be used with caution. Salt substitutes that contain potassium chloride should be steered clear from because these can increase potassium levels in the body; this could become dangerous. I would encourage her to utilize spices such as basil, dill pepper, and vinegar that would add flavor to her foods instead of adding salt. Also, Spironolactone is a potassium-sparing diuretic and can interact with potassium-containing salt substitutes (Insel, Ross, McMahan, Bernstein, 2013).

31. What suggestion might you make to assist with compliance for the fluid restriction?

First, I would encourage her to record everything that she eats and drinks. This would allow her to keep track of everything that she puts into her body. It can also help to identify problematic foods. I would also encourage her to cut out her alcohol consumption all together and to limit her sodium intake as much as possible. If she follows her diet restrictions (2 –g Na and 1.5 L fluid), this would help manage the symptoms of her diagnosis.

VI. Evaluation and Monitoring

32. When you see Ms. Wilcox 1 month later, her weight is now 140lbs. She is wearing flipflops because she says her shoes do not fit. What condition is she most probably experiencing? How could you confirm this?

The condition that Ms. Wilcox is experiencing is called edema. Edema is the extreme buildup of fluid within the interstitial spaces (McCance et al., 2010). This is evident in Ms. Wilcox because her shoes no longer fit. There is an accumulation of fluid around her ankles. Her ankles are the first to swell because of the force of gravity. I could confirm this by pressing lightly on her upper ankle and if my fingerprint remains, edema is present. The picture below is an example of edema:



33. Her diet history is as follows: Breakfast: 1 slice toast with 2 tbsp peanut butter, 1 c skim milk; Lunch: 2 oz potato chips, grilled cheese sandwich (1oz American cheese with 2 slices of whole-wheat bread; grilled with 1 tbsp margarine), 1 c skim milk; Supper: 8 barbeque chicken wings, French fries 1 cup, 2 c lemonade. What changes might you make to her nutrition therapy? Identify foods that should be eliminated and make suggestions for substitutions.

First, I would have her eliminate the potato chips, French fries, margarine, and the barbeque wings. These foods are high in sodium. This diet has way more than 2 grams of sodium. She could replace the potato chips with baby carrots or celery sticks with peanut butter and raisins. If she is set on having a starch, she could consume no-salt-added saltine crackers. The French fries can be substituted with oven-baked potatoes. The only stipulation is that she cannot add salt to them. Instead, add something like Mrs. Dash Seasoning Blend, Salt-Free, All Natural, and no



MSG added. A picture of the Mrs. Dash product I am referring to: . In replacement

of the margarine, she could use unsalted butter. I would replace the barbeque wings with a small portion of grilled lemon-pepper chicken.

34. Over the next 6 months, Terri's cirrhosis worsens. She is evaluated and found to be a good candidate for a liver transplant. She is placed on a transplant list and, 20 weeks later, receives a transplant. After the liver transplant, what diet and nutritional recommendations will the patient need for discharge? For the long term?

	Immediate Posttransplant (First 2 Months)	Long-Term Posttransplant
Kcal	120-130% of BEE. Identify if weight needs to be gained.	Maintenance 120-130% of BEE
Protein	1.3-2 g per kg each day	Consider activity level and the body's needs, but around 1 gram per kilogram of body weight.
Fat	30% of calories	<30% of total calories
CHO	50-70% of calories	50-70% of calories
Sodium	Less than 2 grams	Less than 1,500
Fluid	Between 1.2 and 1.5 L	1.2 L
Calcium	1,200- 1,500 mg/day	1,500 mg/day. Evaluate patient to see if Vitamin D needs to be added for calcium absorption.
Vitamins	Multivitamin/mineral supplement as recommended by her doctor	Multivitamin/mineral supplement as recommended by her doctor

(National Institute of Health, 2012).

Resources

Insel, P., Ross, D., McMahon, K., Bernstein, M. (2013). *Nutrition: MyPlate Update* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Lutz, C., Przytulski, K. (2011). *Nutrition and Diet Therapy* (5th ed.). Philadelphia, PA: F.A. Davis Company.

Mahan, L., Escott-Stump, S. (2008). *Krause's Food & Nutrition Therapy* (12th ed). St. Louis, MO: Elsevier.

McCance, K., Huether, S., Brashers, V., Rote, N. (2010). *Pathophysiology: The Biologic Basis for Disease in Adults and Children* (6th ed.). Maryland Heights, MO: Elsevier.

National Institute of Health. Retrieved from www.nih.com. Accessed on November 7, 2012.

Nelms, M., Sucher, K., Lacey, K., Roth, S. (2011). *Nutrition therapy and pathophysiology* (2nd ed.). Belmont, CA: Cengage Learning.

Nettina, S. (2006). *Lippincott Manual of Nursing Practice* (8th ed.). Ambler, PA: Lippincott Williams & Wilkins.

Nursing 2006: Drug handbook (26th ed.). (2006). Ambler, PA: Lippincott Williams & Wilkins.

Rolfes, S., Pinna, K., & Whitney, E. (2012). *Understanding Normal and Clinical Nutrition* (9th ed.). Belmont, CA: Cengage Learning.

WebMD. “ Find a Vitamin Supplement: Chicory” (2012). <http://www.webmd.com/vitamins-supplements/ingredientmono-92CHICORY.aspx?activeIngredientId=92&activeIngredientName=CHICORY> . Accessed on November 6, 2012.

Van Leeuwen, A., Poelhuis-Leth, D., Bladh, M. (2011). *Davis's Comprehensive Handbook of Laboratory Diagnostic Tests with Nursing Implications* (4th ed.). Philadelphia, PA: F.A Davis Company.