A Critical Nursing Challenge: Patients with End-Stage Liver Disease

Christopher Fowler, PhD, RN, ACNP, is clinical director of the Liver, Kidney, and Pancreas Transplant Programs at Methodist Dallas Medical Center and Assistant Clinical Professor of Nursing at The University of Texas at Arlington. His research focus is on health-related quality of life in individuals with chronic and life-threatening illness.

Disclosure Statements: *The planning committee and author report no relevant financial relationships or conflicts of interest. The author does not intend to discuss any unapproved or off-label use of any product. There is no commercial support for this educational activity. Accredited status does not imply real or implied endorsement by the provider, Texas Nurses Association, or ANCC's COA of any product, service, or company referred to in this educational activity.*

Purpose: The purpose of this article is to enhance the professional nurse's recognition of the signs, symptoms and complications of end-stage liver disease, as well as the critical nursing assessment and intervention approaches.

Objectives: At the completion of this educational activity, the nurse should be able to:

1. Identify the physiologic changes of liver failure.
2. Recognize the signs and symptoms of ascites, spontaneous bacterial peritonitis, hepatic encephalopathy and variceal bleeding.
3. Describe the key components of nursing assessment with end-stage liver disease patients.

Requirements for Successful Completion:

1. Read the article.
2. Complete the post test questions and program evaluation by circling the selected responses on the post test.
3. Fill out the registration form.
4. Send registration form, post test, and a check for $12.00 to:

Continuing Nursing Education
The University of Texas at Arlington
Box 19197
Arlington, TX 76019-0197

5. A passing score is 80% to receive 1.0 Contact Hour. If you pass, your CE certificate will be forwarded to you. If you do not pass, you will be notified and may repeat the test once at no cost.
6. Send before May 15, 2011.

Prior to the development of safe methods for liver transplant, most patients with acute or chronic liver failure died within months to years. Despite increased technology and medical advances, end-stage liver disease remains the 12th leading cause of death in the United States (Friedman, 2009). Almost 16,000 individuals are currently waiting for a liver transplant. With the average national waiting time being slightly more than one year before receiving a liver transplant, individuals with end-stage liver disease will continue to be hospitalized for acute illness related to their disease (Scientific Registry of Transplant Recipients, 2009). The most common admissions are related to ascites, hepatic encephalopathy, and variceal bleeding. Each of these conditions requires accurate identification and treatment for successful outcomes.

Nursing care of the patient with chronic liver disease is becoming increasingly complex. The nursing challenges include close monitoring of coagulopathy, intravascular volume, renal function, electrolyte balance, cardiovascular status, and nutrition. The focus of assessment and physical examination will be on detection of signs and symptoms of altered liver function.

Physiologic Changes of Liver Failure

Normal liver function involves protein synthesis, glucose homeostasis, bilirubin excretion, and toxin removal. The major physiologic changes that occur as a result of cirrhosis include loss of hepatocellular synthetic and metabolic functions and the development of portal hypertension as a result of increased resistance to flow through the liver secondary to fibrous tissue and regenerative nodules. There is also active intrahepatic vasoconstriction that accounts for 20-30% of the increased intrahepatic resistance. Altered liver function can affect the heart and circulatory system, the brain, the lungs, the immune system, and the kidneys (Haranath, Enestvedt, Haranath, & Conjeevaram, 2006).

With 75% of the liver's blood supply from the portal vein, increased pressure can be problematic (Haranath, Enestvedt, Haranath, & Conjeevaram, 2006). The backflow of blood from the portal hypertension leads to the formation of portosystemic collaterals. Despite the development of these collaterals, however, portal hypertension persists because of an increase in portal venous inflow resulting from splanchnic arteriolar vasodilatation and insufficient portal decompression as the collateral vessels have a higher resistance than that of the normal liver (Garcia-Tsao et al, 2007).

At times, it is important to quantify the degree of portal hypertension. The preferred way to measure portal pressure is the wedged hepatic venous pressure (WHVP) measurement which is obtained by placing a catheter in the hepatic vein and wedging it into a small branch or by inflating a balloon and occluding a larger branch of the hepatic vein. To account for any increases in intra-abdominal pressure, the free hepatic venous pressure (FHVP) is subtracted from the WHVP. The result is the hepatic venous pressure gradient (HVPG). The normal HVPG is 3-5mm Hg (Garcia-Tsao et al, 2007).

Table 1

Child-Turcotte-Pugh Scoring System			
Clinical Parameter	**Numerical Score**		
	1	**2**	**3**
Ascites	None	Slight	Moderate to Severe
Encephalopathy	None	Slight to Moderate	Moderate to Severe
Bilirubin (mg/dL)	<2.0	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin Time (seconds increased)	1-3	4-6	>6

Assessment of Significance of Liver Dysfunction

Two methods are commonly used to predict mortality in patients with liver disease. The Child-Turcotte-Pugh score (see table 1) is used to determine risk in individuals undergoing surgery. Although variations of the scoring system exist, most incorporate three biochemical markers (prothrombin time, albumin level, and bilirubin level) and two clinical features (presence of ascites and encephalopathy) into an overall score. CTP scores range from 5-15 with individuals scoring 5-6 being in Class A, individuals scoring 7-9 being in Class B, and individuals scoring 10-15 being in Class C. Class A individuals undergoing surgery can expect a 10% peri-operative morbidity and mortality rate while class B and C individuals have a 30% and 82% mortality, respectively (Friedman, 2009; Haranath, Enestvedt, Haranath, & Conjeevaram, 2006).

A modified version of the Model for End-Stage Liver Disease (MELD) scoring system is used to prioritize need for patients on the liver transplant waiting list. The score is based on a patient's serum total bilirubin, creatinine, and INR. Possible scores range from 6 to 40, with 6 indicating less illness and 40 indicating severe illness (Haranath, Enestvedt, Haranath, & Conjeevaram, 2006; Murray & Carithers, 2005).

Ascites

Ascites is the most common of the three major complications of cirrhosis and results from portal hypertension, hypoalbuminemia, splanchnic vasodilation, and increased secretion coupled with impaired liver inactivation of aldosterone. Approximately 50% of patients with compensated cirrhosis develop ascites within ten years. Approximately 50% of patients with ascites die within two years (Runyon, 2004). Individuals admitted to the hospital with ascites should undergo a diagnostic paracentesis. Rou-

tine ascitic fluid studies include cell count with differential, total protein, and albumin.

The serum-ascites albumin gradient (SAAG) categorizes ascites into that associated with portal hypertension and ascites not associated with portal hypertension. Calculating the SAAG involves measuring the albumin concentration of serum and ascitic fluid specimens obtained on the same day and subtracting the ascitic fluid value from the serum value. If the SAAG is ≥1.1 g/dL, the patient has portal hypertension with approximately 97% accuracy. The mainstay of treatment for high-SAAG ascites includes a dietary sodium restriction of 2 grams/day and oral diuretics. Fluid restriction is not necessary in treating most patients with cirrhosis and ascites unless significant hyponatremia is present (Friedman, 2009; Runyon, 2004).

In patients who do not respond to sodium restriction alone, spironolactone may be added. The initial dose is 100 mg/day and may be increased to a maximum of 400 mg/day. Diuresis is further augmented and hyperkalemia is often prevented with the addition of a loop diuretic. Furosemide dosing ranges from 40 to 160 mg/day. The goal of weight loss in the ascitic patient should be no more than 1-1.5 lbs/day (Friedman, 2009). The oral diuretic doses can be increased simultaneously at 3-5 day intervals while maintaining the 100:40 mg ratio if weight loss and natriuresis are inadequate. Amiloride 10-40 mg/day can be used in patients who cannot tolerate spironolactone due to side effects such as tender gynecomastia. Intravenous furosemide can be used but may cause an acute reduction in renal perfusion with subsequent azotemia.

Body weight, orthostatic symptoms, serum electrolytes, BUN, and creatinine should be monitored. Measurement of urinary sodium and potassium excretion is helpful when weight loss is less than desired. A random urinary sodium concentration greater than urinary potassium concentration correlates with a 24-hour sodium excretion greater than 78 mmol/day with ~90% accuracy. If the patient is excreting more than 78 mmol/day of sodium they are consuming more than 2 grams of sodium daily and should be counseled further about sodium restriction. Encephalopathy, significant hyponatremia despite fluid restriction, or serum creatinine >2.0 mg/dL should lead to a temporary cessation of diuretics (Runyon, 2004).

Refractory ascites is defined as fluid overload that is unresponsive to sodium-restriction and high-dose diuretic treatment or recurs rapidly after therapeutic paracentesis. Options for patients with refractory ascites include serial paracenteses, liver transplantation, transjugular intrahepatic portosystemic shunt placement, or peritoneovenous shunt creation. Once patients develop refractory ascites, 50% die within six months and 75% die within one year (Runyon, 2004).

A single 5-liter paracentesis can be performed safely without the need for colloid infusion. Larger volumes can be removed with the administration of intravenous albumin at a dose of 8-10 g/L of fluid removed to maintain an adequate circulating intravascular volume (Friedman, 2009; Runyon, 2004).

A transjugular intrahepatic portosystemic shunt (TIPS) can also be used for refractory ascites. The procedure and complications are described later. The 1-year survival rate for patients who have a TIPS placed for ascites is 48-76% (Boyer & Haskal, 2005).

Spontaneous Bacterial Peritonitis

Patients with ascites are at significant risk for developing spontaneous bacterial peritonitis (SBP). Symptoms of SBP are typically mild and include abdominal pain, worsening ascites, fever, and progressive encephalopathy. The diagnosis of SBP is made when there is a positive ascitic fluid bacterial culture and an absolute polymorphonuclear (PMN) cell count ≥ 250 cells/mm^3 in the ascitic fluid without evidence of a surgically treatable intra-abdominal source of infection (Runyon, 2004).

Bacterial growth occurs in approximately 80% of instances when there is a PMN count ≥250 cells/mm^3 when the fluid is inoculated into blood culture bottles at the bedside (Friedman, 2009; Runyon, 2004). Patients with ≥250 PMNs but a negative bacterial culture are labeled as having culture-negative neutrocytic ascites. These patients have similar signs, symptoms, and mortality rates as patients with positive cultures and warrant empiric antibiotic therapy. Broad-spectrum antibiotic therapy should be initiated based on the high PMN count and not delayed until a positive culture is obtained. Third generation cephalosporins are the treatment of choice for SBP as they cover 95% of the flora, including the three most common causes of SBP (escherichia coli, klebsiella pneumonia, and pneumococci). Quinolone antibiotics may also be used.

Follow-up paracentesis with ascitic fluid analysis is not usually required but may be helpful if the symptoms and response are atypical. The risk for SBP recurrence remains present even after effective treatment, especially in those patients with ascitic fluid protein concentrations <1.0 g/dL, serum bilirubin >3.0 mg/dL, variceal hemorrhage, and a prior episode of SBP. Those at risk for SBP should be treated prophylactically with a quinolone or trimethoprim/sulfamethoxazole (Friedman, 2009; Runyon, 2004).

Hepatic Encephalopathy

Hepatic encephalopathy results from nitrogenous substances from the gut adversely affecting brain function by producing alterations of neurotransmission that affect consciousness and behavior resulting in a mild confusional state and progressing to coma. These nitrogenous substances come from portosystemic shunting as a result of portal hypertension (Blei & Cordoba, 2001).

One of the most well-known nitrogenous substances is ammonia, although other toxins have been proposed in the etiology of hepatic encephalopathy (Blei & Cordoba, 2001; Friedman, 2009). An initial evaluation of venous ammonia levels may be helpful but serial levels are not necessary for follow-up as evaluation of the patient's mental status frequently does not correlate with serum ammonia levels (Blei & Cordoba, 2001).

Table 2

Stage	Physical Exam Findings
Stage 1	Trivial lack of awareness. Shortened attention span. Impaired addition or subtraction. Hypersomnia, insomnia, or altered sleep-wake patterns. Euphoria or depression. Asterixis may be detected.
Stage 2	Lethargy or apathy. Disorientation. Inappropriate behavior. Slurred speech. Obvious asterixis.
Stage 3	Gross disorientation. Bizarre behavior. Semi-stupor to stupor.
Stage 4	Coma.

Hepatic encephalopathy is frequently precipitated by infection, diuretic use, metabolic alkalosis, constipation, CNS depressant usage, hypoxia, sepsis, azotemia, electrolyte abnormalities, gastrointestinal bleeding, and acute deterioration of liver function. Treatment of hepatic encephalopathy must include identification and eradication of the precipitating cause (Blei & Cordoba, 2001; Haranath, Enestvedt, Haranath, & Conjeevaram, 2006). It remains important to rule out other causes of altered mental status.

Patients with minimal hepatic encephalopathy have no recognizable clinical symptoms but demonstrate mild cognitive and psychomotor deficits and attention deficit. The stages of overt encephalopathy range from mild confusion to coma and are described in table 2.

Treatment of hepatic encephalopathy includes reducing the nitrogenous load from the gut. This includes catharsis and the use of non-absorbable disaccharides and/or antibiotics. Colonic cleansing reduces the luminal content of ammonia, decreases colonic bacterial counts, and lowers serum ammonia levels in cirrhotic patients. Administration of enemas may be necessary in the patient with severe impairment of consciousness (Blei & Cordoba, 2001).

Lactulose is not broken down by intestinal disaccharidases and thus reaches the colon. In the colon, bacteria metabolize the sugar to acetic acid and lactic acid. This acidification results in the formation of non-absorbable ammonium instead of ammonia. Lactulose also causes a change in bowel flora so that fewer ammonia-forming organisms are present (Friedman, 2009). Lactulose has an excessively sweet taste and can cause flatulence and abdominal cramping. For acute or significant encephalopathy, lactulose 45ml PO is followed by dosing every hour until bowel evacuation occurs. The initial dose of lactulose for chronic encephalopathy is 30mL PO three to four times per day. Dosing is then adjusted to maintain 2-3 soft bowel movements per day (usually 15-45 ml q8-12h). Lactulose may also be given via enema (300 ml of lactulose in 700 ml of water) and retained for 1 hour (Blei & Cordoba, 2001; Friedman, 2009).

Neomycin and metronidazole can be used to neutralize ammonia-producing colonic bacteria. Acute hepatic encephalopathy requires neomycin 3-6g/day PO for 1-2 weeks. Chronic hepatic encephalopathy requires neomycin 1-2g/day PO. Renal and auditory function should be closely monitored. The non-absorbable agent rifaximin 400mg PO TID can be used without the systemic effects of neomycin (Friedman, 2009). Metronidazole should be started at a dose of 250mg BID PO (Blei, Cordoba, 2001).

The initiation of a protein-restricted diet remains controversial but may be used in patients with acute or refractory hepatic encephalopathy. Long-term protein restriction, however, may contribute to malnutrition in this chronically ill population. The increased catabolic effects of cirrhosis require a protein intake of 1-1.2 g/kg/day in those with compensated cirrhosis and 1.5 g/kg/day in those with malnutrition (Friedman, 2009). Vegetable and dairy sources of protein are preferable to animal protein as they provide a higher calorie:nitrogen ratio. Additionally, vegetable protein provides non-absorbable fiber.

Zinc, a cofactor of urea cycle enzymes, may be deficient in cirrhotic patients, especially if associated with malnutrition. Zinc supplementation improves the activity of the urea cycle and deficiency can be repleted with zinc sulfate 600mg/day in divided doses (Blei & Cordoba, 2001; Friedman, 2009).

Variceal Bleeding

Gastroesophageal varices are present in approximately 50% of patients with cirrhosis, with hemorrhage occurring at a yearly rate of 5-15%. Their presence correlates with the severity of liver disease as only 40% of CTP class A patients have varices but 85% of CTP class C patients have varices. Bleeding from esophageal varices ceases spontaneously in up to 40% of patients but is associated with 20% mortality at six weeks (Boyer & Haskal, 2005; Garcia-Tsao et al, 2007).

Gastroesophageal variceal rupture is the most common lethal complication of cirrhosis. Cirrhotic patients who develop gastroesophageal varices have a hepatic venous pressure gradient of at least 10-12 mmHg. A decrease in HVPG leads to a decrease in variceal wall tension, reducing the risk of rupture. Also, a reduction in HVPG >20% from baseline decreases the risk of re-bleeding (Garcia-Tsao et al, 2007). Patients who survive acute variceal hemorrhage have a high risk of re-bleeding and death. The median re-bleeding rate in untreated individuals is 60% within 1-2 years with a mortality rate of 33%.

Patients with suspected acute variceal hemorrhage are usually admitted to the intensive care unit. The patient's airway should be maintained and venous access obtained. Emergent tracheal intubation may be required for airway protection, especially in patients with hepatic encephalopathy.

Vigorous saline resuscitation should be avoided as it can precipitate recurrent hemorrhage and can worsen or precipitate accumulation of ascites (Garcia-Tsao et al, 2007).

As all coagulation factors except for von Willebrand factor are produced in the liver, individuals with gastroesophageal bleeding frequently have a coagulopathy. Treatment may involve replacing vitamin K, administration of fresh frozen plasma, platelets, cryoprecipitate, and DDAVP (Haranath, Enestvedt, Haranath, & Conjeevaram, 2006). Transfusion of fresh frozen plasma and platelets should be considered in patients with significant coagulopathy or thrombocytopenia. Blood volume resuscitation should be used to maintain hemodynamic stability and a hemoglobin of approximately 8g/dL. Replacing all lost blood results in a portal pressure that is higher than baseline and results in re-bleeding and increased mortality (Garcia-Tsao et al, 2007).

Balloon tamponade immediately controls hemorrhage in over 80% of patients. Its use, however, is associated with potentially lethal complications of aspiration, migration, and necrosis with perforation of the esophagus with mortality rates up to 20% (Garcia-Tsao et al, 2007).

Pharmacologic therapy consists of splanchnic vasoconstrictors and venodilators. Splanchnic vasoconstrictors reduce portal venous inflow. Venodilators decrease intrahepatic and/or portocollateral resistance and cause hypotension (Garcia-Tsao et al, 2007). Vasopressin is the most potent splanchnic vasoconstrictor; unfortunately, side effects of vasopressin can include cardiac and peripheral ischemia, arrhythmias, hypertension, and bowel ischemia. Vasopressin is administered via continuous intravenous infusion of 0.2-0.4 units/minute and can be increased to 0.8 units/minute. Somatostatin and analogues such as octreotide also cause splanchnic vasoconstriction. An initial bolus of 50mcg of octreotide is followed by a continuous infusion of 50 mcg/hr (Garcia-Tsao et al, 2007). Non-selective beta-blockers prevent bleeding in more than one-half of patients with medium or large varices. Beta-blockers, however, should be used cautiously in the acute setting due to the risk of hypotension and a blunting of the increased heart rate associated with bleeding.

Cirrhotic patients with gastrointestinal bleeding have a high risk of developing severe bacterial infections that are associated with early recurrence of variceal hemorrhage and a greater mortality. The use of short-term prophylactic antibiotics in cirrhotic patients with or without ascites decreases the rate of bacterial infections and increases survival. Norfloxacin should be administered at a dose of 400mg PO BID X7 days. This antibiotic decreases the amount of gram-negative bacteria in the gut. Other quinolones may also be used (Garcia-Tsao et al, 2007).

Endoscopic variceal ligation (EVL) is the preferred form of endoscopic therapy for acute esophageal variceal bleeding (Garcia-Tsao et al, 2007). EVL should be repeated at 7-14 day intervals until the varices are obliterated (usually in 2-4 sessions). Once varices are eradicated, endoscopic examination is usually repeated at three to six month intervals to evaluate for recurrence. The most common complication of EVL is transient dysphagia and chest discomfort. Shallow ulcers at the site of each ligation result and may bleed. A proton pump inhibitor should be used in patients treated with EVL due to the risk of ulcer formation (Garcia-Tsao et al, 2007).

TIPS Procedure

Despite urgent endoscopic and/or pharmacological therapy, variceal bleeding cannot be controlled or recurs early in about 10-20% of patients. Shunt therapy has proven clinical efficacy for patients who fail to respond to endoscopic or pharmacologic therapy (Garcia-Tsao et al, 2007).

A transjugular intrahepatic portosystemic shunt (TIPS) procedure is performed under conscious sedation or general anesthesia. The purpose of the TIPS is to decompress the portal venous system, thus preventing bleeding from varices and reducing the formation of ascites. This decompression is achieved by inserting an expandable metal stent into a branch of the hepatic vein and extending into the portal vein. The success rate with TIPS for the decompression of the portal vein is >90%.

Absolute contraindications to placement of a TIPS include congestive heart failure, uncontrolled systemic infection, and severe pulmonary hypertension. Relative contraindications include hepatoma, portal vein thrombosis, severe coagulopathy with an INR>5, platelet count <20,000, and moderate pulmonary hypertension. Hepatic encephalopathy can limit the effectiveness of the TIPS as new or worsening encephalopathy following TIPS is 20-31%. Risk factors for encephalopathy after TIPS include female sex, hypoalbuminemia, increasing age, and a past history of encephalopathy. The 1-year survival for TIPS placed for bleeding varices is 48-90% (Boyer & Haskal, 2005).

Complications of TIPS include shunt stenosis or occlusion due to thrombosis or hyperplasia of the intima; it can occur within 24 hours of TIPS creation (Friedman, 2009). The cause of the thrombosis may be leakage of bile into the shunt, hypercoagulable syndromes, or inadequate coverage of the TIPS tract with sufficient stents. Thrombosis or stenosis of the TIPS is identified using doppler ultrasound and patency can re-established through repeat catheterization (Boyer & Haskal, 2005).

Conclusion: Take on the Challenge!

The frequency with which patients with end-stage liver disease will be admitted to the acute care setting will increase as medical advances continue and patients continue to wait for liver transplantation. The challenges of caring for these patients are complex and require that nurses recognize the key complications and associated diagnoses, including ascites, hepatic encephalopathy, and gastroesophageal variceal bleeding. All of these patients will require astute nursing assessment, emotional support and encouragement, and multiple interventions to prevent or reduce morbidity and mortality.

References available online at nurseslounge.com

Registration Form and Post Test for **Continuing Education Credit**

"A Critical Nursing Challenge: Patients with End-Stage Liver Disease**"**

Purpose: The purpose of this article is to enhance the professional nurse's recognition of the signs, symptoms and complications of end-stage liver disease, as well as the critical nursing assessment and intervention approaches.

Objectives: At the completion of this educational activity, the nurse should be able to:
1. Identify the physiologic changes of liver failure.
2. Recognize the signs and symptoms of ascites, spontaneous bacterial peritonitis, hepatic encephalopathy and variceal bleeding.
3. Describe the key components of nursing assessment with end-stage liver disease patients.

How to Earn One Contact Hour:
1. Read the article.
2. Complete the post test questions and program evaluation by circling the selected responses on the post test.
3. Fill out the registration form.
4. Send registration form, post test, and a check for $12.00 to:
Continuing Nursing Education
The University of Texas at Arlington
Box 19197
Arlington, TX 76019-0197
5. Send before May 15, 2011.

Within three weeks after receipt of your post test and registration, you will be notified of your results. A passing score is 80%. If you pass, your CE certificate will be forwarded to you. If you do not pass, you will be notified and may repeat the test once at no cost.

The University of Texas at Arlington Center for Continuing Nursing Education is an approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

Accredited status does not imply real or implied endorsement by the provider, Texas Nurses Association, or ANCC's COA of any product, service or company referred to in this educational activity.

Registration Information:

Name: _____

Address: _____

_____

City/State/ZIP: _____

State(s) of Licensure: _____

Telephone Number: _____

Email: _____

Post Test Questions for Continuing Education Credit

Please circle your response for each question.

1. Which of the following parameters is not included in the Child-Turcotte-Pugh scoring system?
   a. Prothrombin time
   b. Presence of ascites
   c. Creatinine
   d. Presence of encephalopathy
2. Which of the following SAAG measurements is consistent with ascites that has developed as a result of portal hypertension?
   a. 0.3 g/dL
   b. 0.5 g/dL
   c. 0.8 g/dL
   d. 1.2 g/dL
3. Which of the following is not a usual treatment for ascites in patients with end-stage liver disease?
   a. Fluid restriction
   b. Spironolactone
   c. Sodium restriction
   d. Furosemide
4. Ascites formation in patients with end-stage liver disease results from all of the following except:
   a. Increased hepatic clearance of aldosterone
   b. Hypoalbuminemia
   c. Portal hypertension
   d. Splanchnic vasodilation
5. Which of the following is indicative of spontaneous bacterial peritonitis?
   a. Polymorphonuclear cell count <100 cells/mm3
   b. Positive bacterial culture
   c. Elevated monocyte level
   d. Positive AFB smear
6. Which of the following is not a common etiology of spontaneous bacterial peritonitis?
   a. Eschericia coli
   b. Klebsiella pneumonia
   c. Pneumococcus
   d. Candida albicans
7. Which of the following is not an appropriate treatment for hepatic encephalopathy?
   a. Valium
   b. Lactulose
   c. Neomycin
   d. Rifaximin
8. Patients experiencing acute esophageal variceal hemorrhage require:
   a. Close observation and maintenance of their away
   b. Saline resuscitation to maintain systolic blood pressure >140 mmHg
   c. Transfusion of packed red blood cells to maintain Hgb >12
   d. Sedation with morphine to decrease portal pressure
9. Which of the following limits the use of the TIPS procedure in patients with refractory ascites?
   a. Hypotension
   b. Hepatic encephalopathy
   c. Dehydration
   d. Portal hypertension
10. Which of the following is an absolute contraindication to placement of a TIPS in patients with portal hypertension?
    a. Left ventricular ejection fraction of 70%
    b. Severe pulmonary hypertension
    c. E. coli urinary tract infection
    d. Platelet count of 30,000

Program Evaluation

Strongly Disagree Strongly Agree

Objective 1 was met.
1 2 3 4 5

Objective 2 was met.
1 2 3 4 5

Objective 3 was met.
1 2 3 4 5

The article was effective as a learning resource/tool.
1 2 3 4 5

The objectives were relevant to the overall purpose.
1 2 3 4 5

The activity met your expectations.
1 2 3 4 5

List two ways that you will integrate what you learned in this activity into your practice and/or work environment: _____
_____
_____
_____

The following were disclosed:

Requirements for successful completion
Yes No

Conflicts of interest
Yes No

Commercial support
Yes No

Non-Endorsement of Products
Yes No

Off-label use
Yes No

Did you perceive any bias that was not disclosed in this activity?
Yes No

If Yes, please describe
_____
_____
_____

State the number of minutes it took you to read the article, complete the test and evaluation
_____min.