Introduction/Purpose Statement

Gastrointestinal (GI), endocrine, and renal problems may occur in any critically ill adult. The purpose of this home study is to review the anatomy and physiology of the GI, renal, and endocrine systems with practical, concise information that will help you form a foundation for understanding the pathophysiology of some of the diseases critical care nurses see. GI bleeding, diabetic ketoacidosis, hyperglycemic-hyperosmolar, non-ketotic coma, renal insufficiency and failure, liver failure and other metabolic problems will be addressed.

Target Audience

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

Content Objectives

1. Describe the pathophysiology of selected GI problems.
2. Describe the pathophysiologic process of cirrhosis and hepatic failure.
3. Identify the pathophysiologic process of renal insufficiency and failure.
4. Define hemorrhagic pancreatitis.
5. Differentiate between DKA and HHNK.
6. Identify the temperature at which critical hyperthermia and profound hypothermia occur.

Disclosures

In accordance with ANCC requirements governing approved providers of education, the following disclosures are being made to you prior to the beginning of this educational activity:

Requirements for successful completion of this educational activity:

In order to successfully complete this activity you must read the home study, complete the post-test and evaluation, and submit them for processing.

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**Expiration Date for this Activity:**
As required by ANCC, this continuing education activity must carry an expiration date. The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

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**Contact Hour Information**

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<th>For completing this <strong>Home Study and evaluation</strong>, you are eligible to receive:</th>
<th>2.0 MN Board of Nursing contact hours / 1.66 ANCC contact hours</th>
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**Criteria for successful completion:** You must read the home study packet, complete the post-test and evaluation and submit them to TCHP for processing.

The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center’s Commission on Accreditation.

Please see the last page of the packet before the post-test for information on submitting your post-test and evaluation for contact hours.
Gastrointestinal Problems

Mrs. Sylvia Scotch comes into the Emergency Room with extreme nausea. She has had one bright red blood emesis of 250 cc. Her husband states that she vomited bright red blood three to four times at home. She is at 17 weeks gestation, and reportedly has been severely nauseated throughout her pregnancy.

What are the causes of GI bleeding?

Eighty-five to ninety percent of all GI bleeding occurs in the upper GI tract. Erosive gastritis (25%), gastric or duodenal peptic ulcer (25%), esophageal varices (10%), Mallory Weiss tear, and aortointestinal fistula are all problems that can result in GI bleeding.

The remaining percentage of GI bleeding occurs in the lower GI tract. Problems resulting in bleeding are:

- Diverticulosis
- Neoplasm: carcinoma, polyp
- Inflammatory bowel disease such as ulcerative colitis or Crohn’s disease
- Ischemic colitis
- Angiodysplasia or AV malformation
- Meckel’s diverticulum
- Hemorrhoids
- Aortointestinal fistula

A Mallory-Weiss tear was diagnosed in Mrs. Scotch’s case. This type of GI bleeding occurs when persistent or violent vomiting occurs, tearing the junction between the esophagus and stomach (GE junction). Arterial blood vessels are exposed and torn.

Why is she vomiting bright red blood?

Bleeding manifests in different ways related to the physiologic processes the blood undergoes.

1. **Bright red blood** = has not undergone any chemical degradation. The site of bleeding is very close to the site of exit (hemorrhoids), or the bleeding is very fast (i.e. arterial bleed).
2. **Maroon/dark red blood** = has been through at least one chemical process, such as degradation by hydrochloric acid in the stomach or enzymes in the intestine.
3. **Maroon/dark red blood with clots** = has been through a chemical process and has coagulated.
4. **Black/tarry blood** = has been through multiple chemical processes. Excreted as *melena* after passing through the large intestine where water is removed.

How is the gastrointestinal system supplied with blood?

The heart, via the abdominal aorta, supplies the GI system with arterial blood. The branches of the abdominal aorta are responsible for certain organ systems:

- Celiac artery: supplies the esophagus, stomach, spleen, and pancreas. The celiac artery branches into the hepatic artery, which supplies the liver with blood.
- Superior mesenteric artery: supplies the pancreas, small intestine, and colon
- Inferior mesenteric artery: supplies the colon

The liver is supplied differently than the remainder of the GI system. The liver receives arterial blood through the hepatic artery, but also receives venous blood from the portal vein. The portal vein receives blood from the gastric, splenic, superior and inferior mesenteric veins. The liver is in charge of processing this venous blood and extracting its nutrients. The hepatic vein drains the processed blood into the inferior vena cava.

Bowel Obstruction

There are two general types of obstruction in the GI tract: functional obstruction and mechanical obstruction. Either type of obstruction may lead to ischemia, necrosis, and perforation of the bowel.

Functional Obstruction

In functional obstruction, the gut is unable to provide absorption, motility, or secretion to digest food.

Adynamic Ileus

One of the most common occurrences in the critically ill patient, an adynamic (paralytic) ileus occurs after surgical manipulation, trauma, or shock states. Peristalsis is greatly diminished; peristalsis is the mechanism by which nutrients and fluids are moved down the GI tract. This leads to a build-up of secretions and gas inside the GI tract. The bowel becomes distended and painful. Nausea and vomiting usually occur, potentially leading to fluid and electrolyte imbalances.

Peritonitis

GI, Endocrine & Renal Critical Care Primer
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The peritoneum is a serous membrane that covers the abdominal wall and abdominal organs. It encloses a normally sterile environment and contains only a small amount of lubricating fluid. In peritonitis, substances have entered into the peritoneum, either through the bowel, accessory organs, genitourinary tract, or from outside of the body.

The open, warm, and moist environment of the peritoneum is ideal for the spread of contaminants throughout the peritoneum and also into the bloodstream. The peritoneum begins to “weep” or cause serous fluid to enter into the peritoneum, potentially leading to hypovolemia. When contaminants enter the peritoneum, a thick, sticky mucus is secreted to “wall off” the opening that is spilling the contaminants. Sympathetic nervous system stimulation causes a decrease in bowel motility, which will decrease the spread of contamination.

Untreated peritonitis can quickly lead to sepsis, septic shock, and death.

Disturbances in the nutritional state occur because of decreased bowel motility, increased caloric need from the inflammatory response, and the nausea and vomiting that normally accompany peritonitis.

**Narcotic use**

The most commonly used narcotic analgesics, including morphine (IV or po), meperidine (IV or po), and codeine can cause a decrease in bowel motility, leading to constipation. The decreased bowel motility, combined with bowel distention from the constipation, can lead to malabsorption of nutrients.

**Bowel ischemia**

Bowel ischemia can result from either a systemic or local decrease in blood flow to the intestine. The most common cause is shock. In shock, the sympathetic nervous system vasoconstricts the mesenteric arterial bed in order to get the most circulating volume into the main blood vessels. Ischemia results in a hypotonic, edematous bowel with little or no motility.

**Inflammatory bowel disease**

Crohn’s disease, diverticulitis, and ulcerative colitis are all diseases in which the bowel is inflamed and irritable. The bowel becomes hypertonic and hypermotile, leading to frequent small, painful diarrheal stools. The inflammation on the intima of the bowel causes a decreased absorption of nutrients.

**Mechanical Obstruction**

In mechanical obstruction, the gut is physically blocked, preventing nutrients from reaching those parts of the intestine that digest and absorb food and liquid. There is typically hyperperistalsis as the bowel attempts to force nutrients and fluids past the area of obstruction.

- **Adhesion**: A stricture of the bowel caused by scar tissue which wraps around the bowel and connects to another organ or the peritoneum.
- **Volvulus**: A twisting of the bowel.
- **Tumors**: A tumor anywhere in the GI tract can cause mechanical obstruction; the larger the tumor, the more potential blockage occurs.
- **Intussusception**: Telescoping of the bowel onto itself.

**Liver Failure**

**Anatomy of the Liver**

The liver is located directly below the lung on the right side of the thorax. It weighs approximately 3 pounds in the adult, and is composed of two lobes: the right and the left. Normally, the liver is protected by the rib cage and is not palpable. The liver is protected by a tough, fibrous coating called Glisson’s capsule.
processed is collected in the sinusoids, where it is passed through the central hepatic vein and into the vena cava.

Functions of the Liver
- Production of bile salts
- Elimination of bilirubin
- Metabolism of steroid hormones
- Metabolism of drugs (90% of alcohol)
- Carbohydrate metabolism
- Fat metabolism
- Protein metabolism
- Synthesis of plasma proteins
- Synthesis of clotting factors
- Storage of minerals and vitamins
- Filtration of blood and removal of bacteria and particulate matter

Pathophysiology of Hepatitis
Hepatitis is an inflammation of the liver caused by either a reaction to drugs or toxins (such as alcohol), by infections such as malaria, mononucleosis, or salmonellosis, or by a virus. Patients with hepatitis are generally not seen in the critical care areas unless they are in the acute stage of fulminant hepatitis.

Pathophysiology of Cirrhosis
In cirrhosis, the liver architecture has been altered through a diffuse process of fibrosis and scarring into structurally abnormal nodules. There are three types of cirrhosis:
1. **Postnecrotic cirrhosis** is characterized by nodules of fibrous tissue rather than normal liver nodules. It can be a result of viral hepatitis, an auto-immune disease, or as a toxic response to drugs and chemicals.
2. **Biliary cirrhosis** results when the bile is obstructed from flow, either through a primary or secondary pathology. Regardless of the cause, bile is unable to flow from the liver, backing up into the liver and causing lobule damage.
3. **Portal or alcoholic cirrhosis (Laennec’s)** occurs as a result of chronic and heavy ingestion of alcohol. The stages of development are:
   - **Fatty liver changes**: the alcohol replaces fat for fuel in liver metabolism, leading to a build-up of fat on and in the liver.
   - **Alcoholic hepatitis** occurs, causing inflammation and necrosis of liver cells.

- **Cirrhosis** is the end result of the fatty liver changes and hepatitis. In cirrhosis, the liver becomes yellow-orange, fatty, and is filled inside and out with scar tissue. The blood flow that normally goes through the liver is blocked, causing further damage to the liver.

Regardless of the cause, cirrhosis always leads to hepatic failure.

Pathophysiology of Liver Failure
Although only 10% of the liver is needed to survive, damage beyond that will cause total hepatic failure. Each of the functions that the liver normally performs fails, leading to multi-focal patient problems.

*Production of bile salts*: Bile salts are not produced, leading to inadequate or absent breakdown of fat in the intestine. Fatty, odorous stools (steatorrhea) are produced.

*Elimination of bilirubin*: Bilirubin is a breakdown product of the heme unit in hemoglobin. Normally, bilirubin is transferred to the liver, where it is conjugated with glucuronide and is excreted into the bile. With liver failure, bilirubin is not conjugated in the liver, and therefore cannot be excreted.

*Metabolism of steroid hormones*: The liver should bind the steroid hormones to proteins, rendering the hormones inactive. This does not occur in liver failure, leading to a build-up of hormones, including aldosterone, ADH, estrogens, and glucocorticoids.

*Metabolism of drugs, including alcohol*: One of the primary functions of the liver is to metabolize drugs, including alcohol. When the liver is not functioning, drugs are not metabolized or excreted. This leads to a build-up of certain drugs, or abnormal metabolism of others, which can cause serious damage to other organs.