TCHP Education Consortium

GI, Endocrine & Renal Critical Care Primer

This educational activity expires December 31, 2017.

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.

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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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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

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

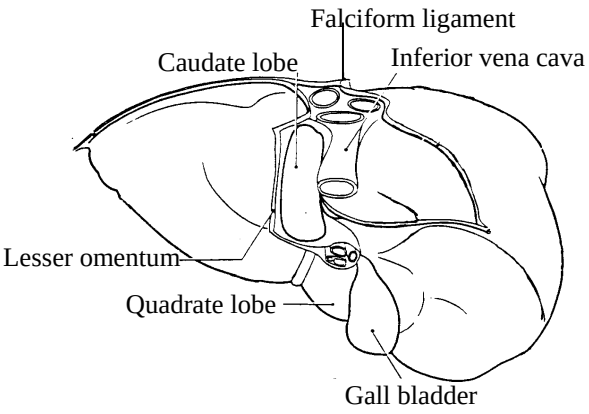
In mechanical obstruction, the gut is physically blocked, preventing nutrients from reaching those parts of the intestine that digest and absorb food and liquid. There is typically hyperperistalsis as the bowel attempts to force nutrients and fluids past the area of obstruction.

- **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.



The liver receives approximately 400 ml of blood each minute from the hepatic artery and the portal vein. This blood oxygenates the liver tissues, and passes through the lobules of the liver to be processed. Blood that has been

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 (Laennecs)** 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.

Carbohydrate metabolism: About 70% of ingested glucose is taken up into the peripheral tissues (mostly muscle). The remaining 30% is taken up by the splanchnic organs, mostly the liver. The liver stores about 70-80% of the glucose ingested as glycogen. The liver also synthesizes glucose from lactic acid, amino acids, and glycerol. Increased levels of circulating glucose result in the patient who is receiving nutrition, decreased levels of glucose result when the patient is not receiving any nutrition.

Fat metabolism: The liver forms lipoproteins and converts carbohydrates and proteins to fat in starvation; synthesizes cholesterol and forms ketones from fatty acids.

Protein metabolism: Proteins are broken down into amino acids -- the liver de*amin*ates these acids, forming ammonia and conjugating the ammonia into urea, which can be excreted.

Synthesis of plasma proteins: The liver is the only organ that can make albumin, as well as other plasma proteins, as a result of conjugation of the amino acids. A decrease in the amount of plasma proteins leads to edema formation because of the loss of oncotic pressure inside the blood vessels.

Synthesis of clotting factors: Fibrinogen, prothrombin, and factors V, VII, IX, and X are all formed by the liver. In the absence of proper function, coagulation times increase because of the lack of clotting factors.

Storage of minerals and vitamins: The liver normally stores minerals and fat soluble vitamins. Without adequate function, the body cannot store or use these vitamins and minerals.

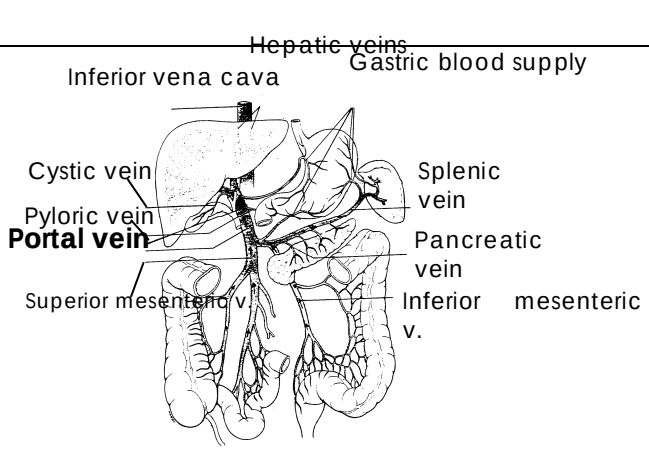
Filtration of blood and removal of bacteria and particulate matter: With the normal function of the Kuppffer cells destroyed, the patient is at higher risk for infection related to active bacteria and particulate matter.

Portal hypertension

In the normal system, blood is delivered to the liver via the hepatic artery (400 ml/min) and the portal vein (1,000 ml/min). Venous blood is drained from the liver via the hepatic vein.

Normally, blood from all of the gastrointestinal organs comes through the liver to be processed. In portal hypertension, there is a mechanical blockage in the liver, due to cirrhosis, scarring, hepatitis, or sarcoidosis. This blockage causes the incoming low pressure blood to "back up"because the blood does not have the pressure to overcome the elevated pressure in the portal vein. The result of portal hypertension is distended veins throughout the GI system – hemorrhoids, varices, caput medusae, and leakage of fluid into the peritoneum – ascites and edema.

Disorders of the Kidney



Definitions

- *Glomerular filtration rate (GFR):* Rate at which solutes are filtered from the glomerulus into the nephron. Measured by the creatinine clearance.
- *Acute renal failure (ARF):* A broad term used to denote a rapid decrease in glomerular filtration rate (GFR) as a result of insult to renal parenchyma.
- *Acute tubular necrosis (ATN):* A specific form of ARF in which insult to renal parenchyma (e.g., renal ischemia, hemorrhage, drug effects) results in necrosis of renal tubules. There is an abrupt decrease in GFR. Regeneration of renal function can take weeks to months following removal of insult.
- *Azotemia*: Presence of nitrogenous waste products (urea, creatinine) in the blood at elevated levels.
- *Uremia***:** Toxic condition in which patient develops clinical symptoms resulting from nitrogenous waste build-up.

Classification of ARF

There are three types of conditions which may cause acute renal failure:

1. Pre-renal
2. Intra-renal
3. Post-renal

Pre-Renal ARF

In acute renal failure caused by pre-renal etiologies, there is a decrease in renal blood flow that results in decreased GFR. There are three major causes of pre-renal ARF:

1. *Decreased intravascular volume* from hemorrhage, sepsis, and extra-cellular volume depletion/dehydration

2. *Cardiac dysfunction with decreased cardiac output*, such as MI, arrhythmias, tamponade, cardiogenic shock, CHF, and afterload reduction therapy
3. *Obstruction of flow to the kidney*, such as by a renal artery embolus

Intra-Renal ARF

A severe parenchymal insult from disease or nephrotoxic agent resulting in damage to glomerulus and/or tubules may cause acute renal failure. There are many etiologies of this type of ARF, including:

1. *Glomerulonephritis* (5-10%)
2. *Hypertension*
3. *Diabetes mellitus*
4. *Vasculitic diseases*, such as polyarteritis nodosa, Wegener's granulomatosis, scleroderma, lupus
5. *Acute tubular necrosis* from ischemia, nephrotoxic agents (drugs, contrast media, heavy metals), myoglobins and hemoglobin in the urine
   - *Heme pigment ATN*, which is an episode of hemolysis resulting in the release of heme pigment, seen in transfusion reaction, venous snake/insect bites, extracorporeal circulation, and faulty heart valves.
   - *Nephrotoxic ATN*: drugs implicated in nephrotoxicity:

Acetaminophen	Dextran
Aminoglycosides	Lithium
Amphotericin B	Methotrexate
Analgesics	NSAIDS
Cephalothin	Penicillins
Cisplatin	Plicamycin
Contrast media	Tetracyclines
Cyclosporin	

Post-Renal ARF

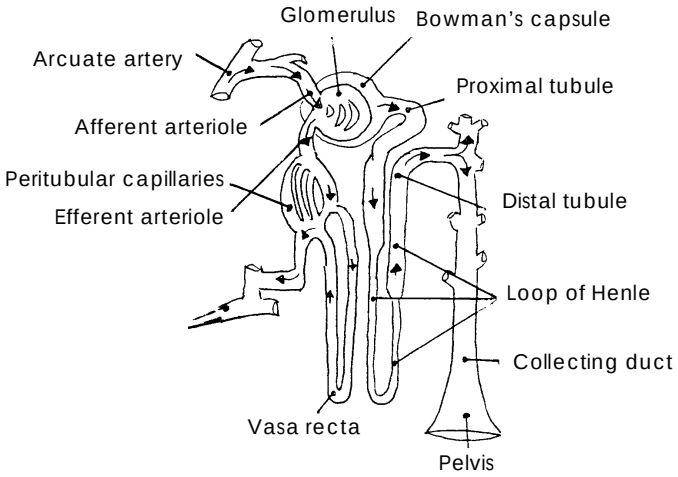
Post-renal ARF is caused by an obstruction anywhere along the urinary tract (renal pelvis to urethra). This obstruction results in the blockage of flow of urine and ultimately, damage to renal parenchyma secondary to hydronephrosis.

Causes of post-renal failure include:

1. ureteral and pelvic blood clots, stones, and fungus balls;
2. ureteral and pelvic malignancy and retroperitoneal fibrosis;
3. bladder stones, blood clots, carcinoma;
4. urethral strictures or prostatic hypertrophy
5. neurogenic bladder with inadequate emptying

What anatomical structures are involved in renal failure?

The kidneys have an extraordinarily different system of perfusion. The renal system receives 20-25% of the cardiac output (about 1200 ml/min) at any given time. The descending aorta is the first blood vessel involved in delivering arterial blood to the kidneys. The aorta gives off a branch called the renal artery, which enters the kidney beside the ureter.



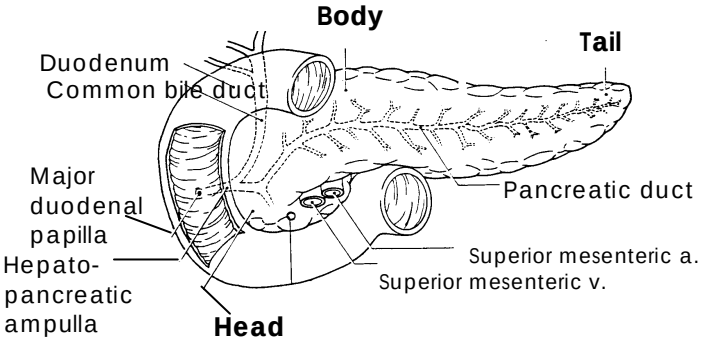
The afferent arteriole branches to form the capillary network. This network sits inside "Bowman's capsule." The afferent (incoming) and efferent (outgoing) capillaries, endothelial membrane and Bowman's capsule make up the glomerulus.

Blood flows through the kidneys at a regulated rate; the renal vasculature has an autoregulating mechanism. As the blood flows through the glomeruli, osmotic, hydrostatic, and electrical gradients will determine the elements to go into the urine. Blood will flow through the efferent arteriole after water products and/or water have been excreted and will exit through the interlobular vein into the arcuate vein, the interlobar vein and finally into the renal vein.

Disorders of the Pancreas

Functions of the Pancreas

There are two major functions of the pancreas: the exocrine and endocrine. The **exocrine pancreas** is made up of acinar cells which secrete pancreatic enzymes. These enzymes are designed to enter the duodenum and begin the initial breakdown of fats (by lipase), carbohydrates (by amylase), and proteins (by trypsin, chymotrypsin, and others). The enzymes are secreted through the pancreatic duct into the Ampulla of Vater. The common bile duct also enters into the Ampulla of Vater. The **endocrine pancreas** is composed of the islet



of Langerhans, which has four types of cells:

- ***Alpha*** cells: secrete glucagon, which breaks down glycogen into glucose, adipose tissue to triglycerides and stimulates gluconeogenesis
- ***Beta*** cells: secrete insulin, which facilitates entrance of glucose into the cells
- ***Delta*** cells: secrete somatostatin, which inhibits insulin, glucagon and growth hormone secretion
- ***PP*** cells: secrete a polypeptide, which causes hypermotility

Pancreatitis

Mr. George Statin is a 47-year-old male with a history of gallbladder disease. He is admitted to the unit with severe flank pain, accompanied by nausea, vomiting, diaphoresis, and pallor. The initial diagnosis is pancreatitis.

What is the connection between gallbladder disease and pancreatitis?

Acute pancreatitis can be caused by a number of different problems, including:

- Blunt and penetrating trauma
- Infections such as infectious mononucleosis, mumps, viral hepatitis
- Drugs, such as thiazides, estrogens, sulfonamides, tetracycline
- Hyperlipidemia, hyperparathyroidism, hypercalcemia
- Ischemia
- Operative injury / ERCP diagnostic test

The most common causes of pancreatitis are alcohol use and gallbladder disease.

The pancreatic duct, which carries the enzymes to the duodenum, connects with the same outlet (the Ampulla of Vater) as the common bile duct. A blockage in the Ampulla of Vater from a gallstone, or sludgy bile from the gallbladder can also block the pancreas.

What happens in pancreatitis?

There is a blockage, either through inflammation or mechanical obstruction, of the path through which the pancreatic enzymes travel through to the intestine. The enzymes build-up in the pancreas and in the pancreatic duct, leading to activation of their proteolytic action. The enzymes begin to "digest" the protein base of the pancreas. If the enzymes destroy tissues around and through the blood vessels of the pancreas, the pancreatitis is called **hemorrhagic.**

What are the complications of pancreatitis?

Among the complications of acute pancreatitis are:

- **Chronic pancreatitis**
- **Pseudocyst** formation: an accumulation of pancreatic enzymes in a membranous sac that protrudes from the pancreas
- **Abscess** formation: either in, on, or around the pancreas
- **Respiratory insufficiency** as a result of the changes in capillary permeability from the enzymatic digestion; can result in Adult Respiratory Distress Syndrome (ARDS).

Diabetic Ketoacidosis (DKA)

Joshua Springer is a 16-year-old male who was playing basketball when he suddenly fainted. The paramedics were called when Joshua was difficult to arouse. When he entered the emergency room, he was found to have a blood sugar of 1600 mg/dL and an arterial pH of 7.03. His parents stated that he had had flu-like symptoms for several days. Joshua was previously undiagnosed with diabetes.

What is Diabetic Ketoacidosis?

DKA is a condition in which the body is not supplied with and does not manufacture insulin with which glucose can enter the cells. It occurs in people with uncontrolled Type I diabetes mellitus, with severe metabolic stress, and in people who have not been diagnosed with Type I diabetes.

What is the pathophysiologic process of DKA?

There are two main problems associated with DKA:

1. There is too much circulating glucose.
2. There is not enough glucose in the cells.

The amount of circulating glucose rises because of (1) continued dietary intake and (2) the attempt by the liver to increase the amount of glucose by gluconeogenesis and glycogenolysis. The liver does not understand that the body is unable to **utilize** the glucose; it is only stimulated because the tissues are not **receiving** glucose. The high amount of glucose causes:

- **Osmotic diuresis**: the osmotic pressure inside the vascular is much higher than in the interstitium and the cells, causing fluid to be pulled into the blood from the cells and tissues. This leads to increased urine output, resulting in hypovolemia.

- **Increased catabolism of proteins.**

- **Increased lipolysis**, leading to overproduction of free fatty acids.

The lack of glucose entering the cells leads to the production of ketoacids, which can act as an energy source for the cells. This leads to:

- **Metabolic acidosis** from the overabundance of ketoacids.

- **Hyperkalemia:** as the potassium leaves the cells and hydrogen enters the cells to try to buffer the metabolic acidosis.

- **Decreased level of consciousness**: the brain can **use only glucose** for metabolism; it cannot store glucose and will not use any other substrate for energy.

Hyperglycemic, Hyperosmolar NonKetotic Coma (HHNK)

Mrs. Cecelia Post is an 89-year-old female nursing home patient who is brought by ambulance into the hospital because of decreasing level of consciousness and anuria. Her serum glucose is 1200 mg/dL, her pH is 7.34, and her bicarbonate level is 23 mEq/L. The initial diagnosis of HHNK is made.

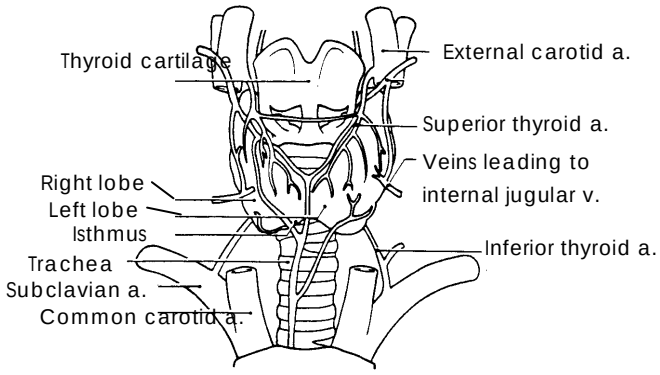
How is HHNK different from DKA?

In DKA, there is **no** insulin production or supply; in HHNK, there is a relative lack of insulin. There is just enough insulin in the patient with HHNK that ketoacids are not manufactured; thus, metabolic acidosis does not occur. The steady increase in glucose levels, however, causes the same osmotic diuresis as in DKA, leading to a large volume deficit. There is a gradual decrease in the level of consciousness in the patient with HHNK, rather than the acute neuro changes of DKA.

What will cause HHNK?

HHNK occurs in patients with known or undiagnosed Type II (non-insulin dependent) diabetes. The majority of the patients with HHNK are elderly; the ability to manufacture "normal" insulin diminishes, as does the attention to increased thirst and increased urine output. This type of hyperglycemia is often misdiagnosed in the elderly as a gradual decline in their physical state.

Metabolic Disorders



The thyroid gland is located immediately beneath the larynx on either side of the trachea. It is made up of two lobes, which are made up of two different cell types: the follicular cells, which produce triiodothyronine (T3) and thyroxine (T4); and the parafollicular cells, which produce thyrocalcitonin.

The thyroid hormones initiate and increase metabolic activity in all cells and tissues. T3 is the most active of the hormones, and is converted from T4. The actions of the thyroid hormones are to:

1. Increase the metabolic activity, oxygen consumption, rate of chemical reactions, and heat production of all cells.
2. Promote growth by working with insulin, growth hormone, and the sex steroids.
3. Stimulate metabolism of carbohydrates, fats, and proteins.
4. Increase metabolism and clear hormones and drugs

Thyrotoxicosis (Thyroid Storm)

In thyrotoxicosis, or thyroid storm, the actions of the hormones are **greatly accentuated.** This is a life-threatening condition which may be caused by decompensation of a preexisting hyperthyroid state, insufficient provision of antithyroid therapy, or administration of an iodine load to a patient who has an underlying thyroid disease.

Alterations in Temperature

There are three zones of temperature in the human body: (1) the superficial zone, (2) the intermediate zone, and (3) the core zone. The superficial zone is the temperature of the skin; the intermediate zone is the temperature of the muscles; and the core zone is the temperature of organs such as the brain, heart, and liver. The oral temperature is normally between 36.0 and 37.0 degrees Celsius.

Heat is produced through cellular metabolism. The ingestion and metabolism of food, basal metabolic rate, muscle activity, hormones, and sympathetic nervous system all produce heat. Heat production is increased during higher metabolic rate periods, such as with exercise, excessive food intake, and the release of catecholamines. Less heat than normal is produced when the patient has a lower metabolic rate than normal, such as in starvation or with a lack of thyroid hormones.

The hypothalamus is in charge of regulating heat production and responding to heat loss through the thermoregulatory center. The possible responses of the hypothalamus are to cause vasodilation or vasoconstriction, shivering, and activating the neuroendocrine response to balance heat production and heat loss.

Hypothermia

Hypothermia is a body temperature below 37 degrees C (98.6 degrees F). Hypothermia can be either accidental or elective. Elective hypothermia is frequently used in the OR for cardiovascular and neurosurgery; it may be indicated for multiple trauma, cerebral ischemia, and graft preservation. Accidental hyperthermia is related to a number of problems:

- Near-drowning
- Exposure or prolonged exposure to cold temperatures
- Rapid infusion of cold intravenous solutions or blood products
- Dysfunction of thermoregulatory mechanisms
- Drugs, such as alcohol, antidepressants, antipyretics, sedatives, and barbiturates

The patient with mild hypothermia (34 to 36.5 degrees C) may have an increased risk for shivering, altered mentation, a clouded LOC, and altered electrodynamics. The patient experiencing moderate hypothermia (28 to 33.5 degrees C) will experience atrial dysrhythmias, a 50% decrease in body metabolism, a 50% degree in heart rate, and nonreactive pupils. Deep hypothermia is a temperature between 17 and 27.5 degrees C, and is characterized by ventricular fibrillation, inactivation of thermoregulatory centers, absent muscle reflexes, and cardiac and respiratory arrest. The last stage, profound hypothermia, is when the patient's body temperature is between 0 and 16.5 degrees C. The chance of survival is slim at this stage.

Hyperthermia

Hyperthermia is defined as a body temperature that is above the normal physiologic level of 37.2 degrees Celsius. In mild hyperthermia (37.2 to 38.8 degrees C), the patient has a potential for dehydration and dysrhythmias. In moderate hyperthermia (38.8 to 40 degrees C), the patient may be confused and experiencing dysrhythmias and dehydration. With a temperature above 40.5 degrees, the patient is experiencing critical hyperthermia. At this stage, the patient has increased dysrhythmias, altered mentation, confusion, and is at risk for seizures and cardiac arrest.

Malignant hyperthermia is a specific kind of critical hyperthermia, characterized by a rise of 0.5 degrees C every 15 minutes to 42.7 degrees C (109 degrees F). This type of hyperthermia tends to be genetic in origin and is related to general anesthesia.

Summary

Although not as "glamorous" as other systems in the body, the gastrointestinal, endocrine, and renal systems can certainly cause as significant of a critical illness as any other system. In this program, you learned about the anatomy and physiology of these organ systems and the pathophysiology of selected GI, endocrine, and renal diseases.

Recommended Reading

1. Brozenee S, Russell SS. (1999). *Core Curriculum for Medical-Surgical Nursing*, 2nd ed. Academy of Medical-Surgical Nurses, Janetti NJ.
2. Dickson, S. (1995, Oct.). Understanding the oxyhemoglobin dissociation curve. Critical Care Nurse, pp. 54-58.
3. Phipps WJ, Sands JK, Marek JF, eds. (1999)..*Medical-Surgical Nursing: Concepts & Clinical Practice*, 6th ed. St. Louis: Mosby, Inc.
4. Seidel HM, Ball JW, Dains JE et al, eds.(2002) *Mosby's Guide to Physical Examination*, 5th ed. St. Louis: Mosby, Inc.
5. Stillwell, S. (2002). *Mosby's Critical Care Nursing Reference*. 3rd ed. St. Louis, Mo: Mosby/Elsevier.
6. Smeltzer SC, Bare BG, eds. (2002) *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 10th ed. Philadelphia: Lippincott William and Wilkins.
7. Wiegand, D.J.L. & Carlson, K.K. (eds.) (2005). *AACN Procedure Manual for Critical Care*. 5th ed. Philadelphia: Elsevier.

Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. If you are completing this home study as pre-reading for a TCHP class, please bring your post-test and evaluation to class with you for processing. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

HealthEast, HCMC, & MVAMC Employees

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for $12.00. Please make check payable to **TCHP Education Consortium** and mail to:

TCHP Education Consortium
Capitol Office Building
525 Park Street, Suite 120
St. Paul, MN 55103

Your post-test will be returned to you with the certificate of completion.

GI, Endocrine & Renal Critical Care Primer Post-Test

Please print all information clearly and sign the verification statement:

Name_____
(please print legal name above)

Birth date (required)

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

For HealthEast, HCMC, or MVAMC, employees only:

Hospital _____ Unit_____

Personal verification of successful completion of this educational activity (required):

I verify that I have read this home study and have completed the post-test and evaluation.

_____
Signature

1) What is the most common source of GI bleeding?
   a) Duodenal peptic ulcer
   b) Mallory Weiss tears
   c) Diverticulosis
   d) Hemorrhoids

2) An adynamic ileus can result from:
   a) surgical manipulation
   b) starvation
   c) fever
   d) mechanical ventilation

3) Which of the following will NOT cause decreased bowel motility?
   a) morphine
   b) Darvocet
   c) Codeine
   d) Meperidine

4) A volvulus is:
   a) twisting of the bowel
   b) telescoping of the bowel onto itself
   c) stricture of the bowel
   d) connection of one abdominal organ to another

5) Azotemia refers to:
   a) elevated serum creatinine and urea levels
   b) itching caused by bile salt deposition
   c) body edema caused by renal failure
   d) toxic condition in which patient develops symptoms from high creatinine levels

6) Uremia refers to:
   a) toxic condition where the patient has symptoms from high creatinine and urea levels
   b) elevated serum creatinine
   c) itching caused by bile salt deposition
   d) body edema caused by renal failure

7) Which of the following differentiates DKA from HHNK?
   a) elevated blood glucose
   b) the synthesis of ketones for energy
   c) presence of ketones
   d) osmotic diuresis

8) Hypothermia in the clinical setting may be caused by:
   a) rapid infusion of cold IV fluids
   b) open surgical procedures
   c) drugs such as barbiturates
   d) all of the above

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.

Evaluation: GI, Endocrine, & Renal Critical Care Primer

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
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.					
7. The teaching / learning resources were effective. *If not, please comment:*					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).	Yes	No
8. Notice of requirements for successful completion, including purpose and objectives		
9. Conflict of interest		
10. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
11. Sponsorship or commercial support		
12. Non-endorsement of products		
13. Off-label use		
14. Expiration Date for Awarding Contact Hours		
15. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? *If yes, please describe the nature of the bias:*		

16. How long did it take you to read this home study and complete the post test and evaluation:
_____hours and _____minutes.

17. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?
____Yes
____No, more contact hours should have been offered
____No, fewer contact hours should have been offered.

Expiration date: December 31, 2017