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NURS1005 FINAL EXAM STUDY GUIDE Q&AS

BIOMOLECULES

Identify the products that macromolecules (fat, protein, carbohydrates) are broken down into during catabolism.

CARBOHYDRATES (polysaccharides → glucose)

- provides raw energy needed for energy production + fuels a number of processes
- catabolism occurs during digestion
- monosaccharide, disaccharide, polysaccharides (used as storage product)

FATS → fatty acids + glycerol

- **Triglycerides + Phospholipids**
  - protect internal organs + insulate + store energy fuels
  - triglyceride → phospholipid (cell membrane)
- **Cholesterol**
  - stabilize cell membrane (fluid mosaic model)
  - synthesizes → sex hormones, vitamin D, bile

PROTEINS → amino acids

- responsible for many physiological features
- 20 amino acids building blocks
- **Primary Structure** → sequence of amino acid (polypeptide chain)
- **Secondary Structure** → polypeptide chain either pleats (beta pleated sheets) OR folds (alpha helix)
  - held by hydrogen bond = weak + easily broken
- **Tertiary Structure** → conformation → side chains of amino acids fold, curls (stronger than hydrogen bond)
  - factors affecting bonds → pH, temperature, chemical environment, concentration
- **Quaternary Structure** → two or more polypeptide chains binds to form complete unit
- **enzyme**: special proteins that speed up bodily functions

DISCUSS WHY HOSPITALIZED PATIENTS ARE MORE PRONE TO MALNUTRITION THAN THE GENERAL POPULATION.

Malnutrition: lack of nourishment from adequate number of kilojoules (calories), proteins, vitamins, minerals + caused by improper diet, alterations in digestion or absorption, or combination of these factors

- malnutrition seen in hospital → combination of **cachexia** (disease related body wasting) + **malnutrition**
- illnesses that lead to reduced intake of food e.g. old age, malignant and chronic diseases
    - Old age → number of taste buds decline, eating itself is a strenuous activity, salivary secretions decrease, decrease in motility of esophagus + stomach
    - state of mental health
    - less mobility → weight loss
    - GI disorders
    - hospital food does not always necessarily taste good

GI SYSTEM

DESCRIBE THE PHYSICAL ALTERATIONS THAT LEADS TO VOMITING.

Vomiting: forceful emptying of stomach and intestinal contents (chyme) through the mouth

- protective response to a number of factors that may be potentially harmful to the body
- coordinates sensory stimuli → directs motor output to muscle involved

Stimuli → distention (stretching) stomach + duodenum, severe pain, unpleasant sight + odors, fear, chemoreceptor trigger zone (from GI tract e.g. contaminated food, toxic substances, chemotherapy)

Clinical Manifestation

- deep inspiration → diaphragm lowered = abdominal muscle contraction (glottis closes)
- leads to increased abdominal pressure
- lower esophageal sphincter relaxes + reverse peristalsis of duodenum (chyme forced from stomach/duodenum into esophagus)
- contractions of the abdominal muscles extremely strong → forces diaphragm high into thoracic cavity (retching)
- upper esophageal sphincter forced to open → chyme expelled from mouth
- upper part of esophagus contracts → force remaining chyme back into the stomach + lower esophageal sphincter closes

Physical Alterations

- alterations to GI tract → motility slows down
- trauma

OUTLINE THE CLINICAL MANIFESTATIONS THAT YOU MAY OBSERVE IN A PATIENT WHO HAS EXPERIENCED SEVERE VOMITING FOR SEVERAL DAYS

- disturbances in hydration, electrolytes and acid-base balance can become severe consequences of of vomiting
- retching
- nausea

DISCUSS THE COMPLICATIONS THAT CAN RESULT FROM UPPER GASTROINTESTINAL BLEEDING

UPPER GASTROINTESTINAL BLEEDING: bleeding in the esophagus, stomach or duodenum (first part of small intestine)

- bright red bleeding affected by stomach acids
- bleeding varicose veins in esophagus, peptic ulcers

LOWER GASTROINTESTINAL BLEEDING: bleeding in jejunum, ileum, colon or rectum

- caused by polyps, inflammatory disease, cancer

Complications

- **hematemesis** (vomiting blood)
- **hypovolemic shock** (severe blood loss) - best way to look for it is measuring blood pressure
- **oliguria** (low urine output) → diminished blood flow to kidneys
- **diarrhea** → accumulation of blood in GI tract is irritating and increases peristalsis
- **melaena** → black or tarry stools that are sticky + foul odor → result from partial digestion of blood components
- **anemia** → lack of healthy red blood cells

DISCUSS THE PHYSICAL ALTERATIONS THAT CAN LEAD TO INCREASED GASTRIC ACID PRODUCTION

PEPTIC ULCER DISEASE: break or ulceration in the protective mucosal lining of the stomach or duodenum

- exposes submucosal area to gastric secretions + auto-digestion (digestion of gut mucosa by the body's secretions)

Factors that increase gastric acid production

- increase in the number of chief (pepsinogen - digestive enzyme) and parietal cells (secrete hydrochloric acid)
- decrease in the inhibition of gastric secretions
- increased sensitivity to food/other stimuli e.g. caffeine, histamine
- excessive vagal stimulation (stress)

DISCUSS THE TREATMENTS THAT COULD BE USED TO ALLEVIATE SYMPTOMS OBSERVED IN CELIAC DISEASE AND LACTOSE INTOLERANCE (NUTRITIONAL DISORDERS)

CELIAC DISEASE: loss of mature intestinal villi caused by hypersensitivity to gluten (protein component of cereal, grains) → malabsorption

- diarrhea early symptom
- abdominal pain
- can lead to malnutrition
- **Treatment**:
    - immediate + permanent institution of a diet free of cereal grains e.g. wheat, rye, barley oats
    - may need vitamin D, iron, folic acid supplements to treat deficiencies (infants)
    - patient education → can result in lactose intolerance (destruction fo villi where enzyme lactase is located)

LACTOSE INTOLERANCE: deficient of enzyme (lactase) necessary for digesting lactose, a sugar (disaccharide) found in milk

- when lactose cannot be digested → not absorbed across intestinal wall
- bacterial fermentation occurs → gas + abdominal pain
- irritation + osmotic diarrhea
- dehydration may also occur (fluid going into feces)
- **Treatment**:
    - avoiding milk + adhering to lactose-free diet
    - oral lactase supplement

EXPLAIN HOW THE STOMACH PROTECTS ITSELF FROM ITS OWN GASTRIC ACID

Stomach: muscular organ that stores food during eating, secretes digestive juices, mixes food with these juices + propels chyme (partially digested food) into small intestine

- stomach has a protective mucosal barrier - coating of alkaline (bicarbonate ions - neutralize harsh acids)
- inner mucus layer → acts as barrier for hydrochloric acid + impervious to bacteira
- prostaglandins → stimulate secretion of mucus and bicarbonate + inhibiting secretion of acid
- mucosal blood flow important to maintaining mucosal protective functions

COMPARE AND CONTRAST THE CAUSES AND PATHOPHYSIOLOGY OF OSMOTIC AND SECRETORY DIARRHEA

Diarrhea: increase in frequency of defecation and the fluidity and volume of feces

- **OSMOTIC DIARRHEA**: non-absorbable substance in intestine draws excess water into intestine → increase stool weight + volume → large-volume diarrhea
    - can be found in lactose-intolerant (no lactase enzyme to digest lactose) - nutritional disorder
        - bacterial fermentation occurs → gas → intestines stretch → abdominal pain
        - undigested lactose causes change in osmotic gradient
    - synthetic sugar (non absorbable) → when excessively ingested
- **SECRETORY DIARRHEA**: excessive mucosal secretion of fluid and electrolytes produces large-volume diarrhea (more mucous passes with the chyme than usual)
    - bacterial enterotoxins (e coli)
    - neoplasms (cancer)
    - inflammatory bowel disease

Systematic effects → dehydration, electrolyte imbalance, metabolic acidosis, weight loss

RESPIRATORY SYSTEM

DESCRIBE THE PATHOPHYSIOLOGY, CLINICAL MANIFESTATIONS AND AGE OF ONSET FOR:

PNEUMOTHORAX: presence of air or gas in the pleural space caused by a rupture in the visceral pleura (surrounds lung) or parietal pleura and chest wall

- destroys negative pressure → essential for preventing the lung from collapsing/disrupt equilibrium between elastic recoil forces of the lung and chest wall