มหาวิทยาลัยราชภัฏนครปฐม

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

NURSING EDUCATION

Pathophysiology *of GI Disorders*

Comprehensive overview of gastrointestinal function, mechanisms of disease, and clinical nursing assessment.

Asst.Prof. Jutatip Tepsuwan

Introduction to GI System & Homeostasis

The Gateway for Nutrient Acquisition and Internal Equilibrium

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

What is the GI System?

A continuous muscular tube (Alimentary Canal) approximately 9 meters long, extending from the mouth to the anus. It acts as a specialized interface between the external environment and the internal milieu.

Maintaining Homeostasis

- **Fluid Balance:** Reabsorption of water (up to 9L/day processed) to prevent dehydration.
- **Electrolyte Regulation:** Precise uptake of Na+, K+, and Ca2+ essential for cellular function.
- **Immune Defense:** Gut-Associated Lymphoid Tissue (GALT) protects against ingested pathogens.

The Digestive Imperative

Digestion
Mechanical breakdown (chewing/churning) and Chemical degradation (enzymes/acid) of complex molecules.

Absorption
Movement of micronutrients (glucose, amino acids, lipids) across the epithelial lining into blood or lymph.

Elimination
Excretion of indigestible substances and metabolic waste products from the body.

STEP 1 Ingestion › STEP 2 Digestion › STEP 3 Absorption › STEP 4 Defecation

Functional Anatomy Review

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Mouth & Pharynx
- Mastication (Mechanical)
- Salivary Amylase (Chemical)
- Bolus formation & Swallowing

Esophagus
Muscular conduit using peristalsis to move food to the stomach. Contains Upper & Lower Sphincters.

Stomach
- **HCl & Pepsin:** Protein digestion
- **Mixing:** Chyme production
- **Intrinsic Factor:** Vit B12 absorption

Small Intestine
DUODENUM | JEJUNUM | ILEUM
- 90% of chemical digestion
- Primary site for nutrient absorption
- Villi/Microvilli increase surface area

Large Intestine
- Water & Electrolyte reabsorption
- Microbial fermentation (Vitamin K)
- Fecal storage and elimination

Rectum & Anus
Control of defecation via internal (involuntary) and external (voluntary) sphincters.

Accessory Organs
LIVER:
Bile production, metabolic processing, and detoxification.

PANCREAS:
Exocrine secretion of digestive enzymes (Lipase, Protease) & Bicarbonate.

GALLBLADDER:
Concentration and storage of bile for fat emulsification.

Clinical Integration
Functional health requires **synchronized motility**, **precise secretion**, and **intact mucosal surface**. Disruption at any anatomical point leads to specific pathophysiological manifestations observed in clinical practice.

INGESTION › PROPULSION › DIGESTION › ABSORPTION › DEFECATION

Enteric Nervous System (ENS)

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

"The Second Brain" of the Human Body

What makes it unique?

The ENS contains over 100 million neurons and can function ***independently*** of the Central Nervous System (CNS).

- Local reflex arcs (peristalsis)
- Sensory perception of gut wall stretch
- Regulation of local blood flow

The Two Major Networks

Myenteric Plexus (Auerbach's)
Located between muscle layers; controls **GI Motility**.

Submucosal Plexus (Meissner's)
Located in the submucosa; regulates **Secretion & Blood Flow**.

Autonomic Nervous System (ANS) Modulation

Parasympathetic

"REST AND DIGEST"
Stimulates GI activity via the **Vagus Nerve** (Cranial Nerve X).

↑ Motility & Secretion

Sympathetic

"FIGHT OR FLIGHT"
Inhibits GI activity; diverts blood flow away from the gut to skeletal muscles.

↓ Motility & Secretion

Hormonal Control of GI Function

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Coordination of digestive secretions and motility through the endocrine system.

STOMACH PHASE

Gastrin

- **Source:** G cells of the Gastric Antrum.
- **Stimulus:** Distension, protein, high pH.

Primary Action:

*Stimulates parietal cells to secrete **Gastric Acid (HCl)** and increases gastric motility.*

INTESTINAL PHASE

Secretin

- **Source:** S cells of the Duodenum.
- **Stimulus:** Low pH (acidic chyme) in duodenum.

Primary Action:

*Stimulates **Bicarbonate** release from pancreas to neutralize stomach acid.*

INTESTINAL PHASE

CCK

CHOLECYSTOKININ

- **Source:** I cells of the Duodenum/Jejunum.
- **Stimulus:** Fatty acids and Amino acids.

Primary Action:

*Triggers **Gallbladder contraction** (bile) and Pancreatic enzyme secretion.*

Nursing Insight: These hormones work via **Negative Feedback**. As pH drops in the duodenum, Secretin release increases to restore balance, ensuring optimal enzyme activity and mucosal protection.

GI Motility & Peristalsis

Mechanical Processing and Propulsive Forces

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University
PHYSIOLOGY UNIT

Patterns of Motility

1. Peristalsis (Propulsion)

Adjacent segments of alimentary tract organs alternately contract and relax, moving food distally.

2. Segmentation (Mixing)

Nonadjacent segments of alimentary tract organs contract and relax, moving food forward then backward for mixing.

The Pacemaker: Cells of Cajal

Interstitial Cells of Cajal (ICC) are specialized cells located between nerve endings and smooth muscle.

- Generate **Slow Waves** (Basic Electrical Rhythm - BER).
- Coordinate the frequency and direction of contractions.
- Act as electrical "bridges" for the Enteric Nervous System.

ELECTRICAL TO MECHANICAL CASCADE

ICC Activity
Slow Wave Generation

→

Action Potential
Threshold Reached

→

Smooth Muscle
Physical Contraction

Dysphagia: Swallowing Disorders

Classification, Causes, and Clinical Implications

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Definition

Dysphagia is difficulty swallowing that can occur at any stage of the swallowing process. It affects nutritional status, increases aspiration risk, and significantly impacts quality of life, particularly in elderly and neurologically compromised patients.

Oropharyngeal Dysphagia

Location

Difficulty in the **oral and pharyngeal phases** of swallowing (mouth and throat)

Common Causes

Neurological: Stroke, Parkinson's disease, dementia
Muscular: Myasthenia gravis, muscular dystrophy
Structural: Head/neck surgery, radiation therapy

Clinical Signs

- Coughing/choking during meals
- Drooling, food pocketing
- Nasal regurgitation
- Voice changes (wet/hoarse)

Esophageal Dysphagia

Location

Difficulty in the **esophageal phase** - food transport from pharynx to stomach

Common Causes

Mechanical: Strictures, tumors, rings
Motility: Achalasia, esophageal spasm
Inflammatory: GERD, esophagitis

Clinical Signs

- Food "sticking" sensation
- Chest pain during swallowing
- Regurgitation of undigested food
- Progressive dysphagia

Nursing Priorities & Complications

Aspiration Risk: Silent aspiration can lead to pneumonia, especially in neurological patients.

Nutritional Compromise: Weight loss, dehydration, and malnutrition from inadequate intake.

Psychosocial Impact: Social isolation, anxiety, and depression from eating difficulties.

Dyspepsia & Heartburn

Understanding Upper GI Distress and Acid Reflux Mechanisms

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Dyspepsia (Indigestion)

A clinical syndrome of upper abdominal symptoms including bloating, fullness, and epigastric pain. Often associated with delayed gastric emptying or visceral hypersensitivity.

Heartburn (Pyrosis)

A retrosternal burning sensation radiating toward the neck. The cardinal symptom of GERD, caused by the reflux of gastric acid into the sensitive esophageal mucosa.

Key Mechanisms

- **LES Dysfunction:** Inappropriate relaxation of the Lower Esophageal Sphincter allows acid backflow.
- **Acid Pocket:** Post-prandial unbuffered acid layer formed at the gastroesophageal junction.
- **Hyperalgesia:** Increased sensitivity of esophageal nerves to low levels of acid or distension.

COMMON RISK FACTORS

- Fatty/Spicy Foods
- Caffeine & Alcohol
- Tobacco Use
- Obesity
- NSAIDs & Meds
- Pregnancy
- Lying Down Post-Meal
- Stress & Anxiety

Nursing Tip: Always assess for 'Alarm Symptoms' like weight loss or dysphagia.

Esophageal Disorders

Gastroesophageal Reflux Disease (GERD) & Achalasia

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

GERD

HYPER-REFLUX

Pathophysiology:

- **LES Incompetence:** Lower Esophageal Sphincter is too relaxed or weak.
- **Acid Exposure:** Gastric contents reflux into the esophagus, causing mucosal injury.

CLINICAL IMPACT ON SWALLOWING:

Odynophagia (painful swallowing) and Heartburn (pyrosis). Risk of strictures and Barrett's Esophagus.

Achalasia

MOTILITY FAILURE

Pathophysiology:

- **LES Obstruction:** Failure of the LES to relax during swallowing.
- **Aperistalsis:** Loss of esophageal wave-like contractions (denervation).

CLINICAL IMPACT ON SWALLOWING:

Progressive Dysphagia for both **solids and liquids**. Regurgitation of undigested food.

Feature	GERD	Achalasia
LES Tone	Decreased (Open)	Increased (Closed)
Main Symptom	Heartburn & Regurgitation	Dysphagia & Weight Loss
Esophageal Body	Normal Motility	Loss of Peristalsis

Nursing Alert: Chronic GERD increases the risk of esophageal adenocarcinoma via metaplasia (Barrett's Esophagus).

Nausea & Vomiting Mechanisms

Neuro-Control Centers and Pathophysiological Consequences

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

The Brain Centers

Vomiting Center

Located in the **Medulla Oblongata**. Coordinates the muscular act of vomiting (reverse peristalsis).

CTZ (Chemoreceptor Trigger Zone)

Located in the **Area Postrema**. Outside the Blood-Brain Barrier; detects toxins and drugs in the blood.

Key Neurotransmitters

- Dopamine (D2)
- Serotonin (5-HT3)
- Histamine (H1)
- Substance P

Trigger Pathways

GI Tract

Vagus Nerve (Distention, Irritation)

Vestibular System

Motion Sickness, Inner Ear (H1, M1)

Cerebral Cortex

Smells, Sights, Anticipatory Anxiety

Systemic Toxins

Chemotherapy, Uremia, Opioids (CTZ)

Clinical Impacts

Metabolic Alkalosis

Loss of Hydrogen (H^+) and Chloride (Cl^-) from gastric acid.

Dehydration (Hypovolemia)

Significant fluid loss leading to reduced tissue perfusion.

Electrolyte Imbalance

Hypokalemia (low K^+) and Hyponatremia (low Na^+).

Nursing Tip: Continuous vomiting leads to metabolic alkalosis, while severe diarrhea often leads to metabolic acidosis.

Abdominal Pain: Types & Assessment

Understanding the pathophysiology behind patient complaints

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Visceral Pain

- Origin: Stretching or distention of abdominal organs.
- Character: Dull, aching, or cramping.
- Localization: Poorly localized, often felt at the midline.

Parietal Pain

- Origin: Inflammation of the parietal peritoneum.
- Character: Sharp, intense, and steady.
- Localization: Well-localized; aggravated by movement/ coughing.

Referred Pain

- Origin: Pain felt at a site distant from source organ.
- Mechanism: Shared spinal nerve pathways.
- Example: Biliary colic felt in the right shoulder.

Nursing Assessment: PQRST Framework

P	Q	R	S	T
Provocation / Palliation	**Quality**	**Region / Radiation**	**Severity**	**Timing**
What makes it better or worse?	Sharp, dull, burning, or cramping?	Where is it? Does it move?	Scale of 0-10?	When did it start? Constant or intermittent?

Clinical Tip: Sudden, severe parietal pain may indicate "Acute Abdomen" and requires immediate surgical evaluation.

Bowel Habit Alterations

Pathophysiological mechanisms of Constipation and Diarrhea

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Constipation

Decreased frequency or difficult defecation.

Mechanism:
Delayed transit time leading to excessive water reabsorption and hard stools.

Primary Causes:
- Low-fiber diet & inadequate fluid intake
- Sedentary lifestyle (decreased motility)
- Medications (Opioids, Antacids)
- Neurogenic disorders (Hirschsprung disease)

Diarrhea

Increased frequency, fluidity, and volume of stool.

1. Osmotic Diarrhea

Non-absorbable solute in the lumen draws water via osmosis.

Clinical Hint: Stops when patient is NPO (fasting).

Example: Lactose intolerance, Sorbitol intake.

2. Secretory Diarrhea

Excessive mucosal secretion of fluid and electrolytes.

Clinical Hint: PERSISTS even when fasting.

Example: Bacterial toxins (Vibrio cholerae), IBD.

Nursing Priority: *Monitor for fluid-electrolyte imbalance (Hypokalemia, Metabolic Acidosis) and dehydration.*

GI Bleeding: Upper vs. Lower

Anatomical Boundaries and Clinical Presentation

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

DIVIDER: LIGAMENT OF TREITZ

↑ Upper GI Bleeding (UGIB)

Clinical Presentation:

Hematemesis: Bright red blood or "coffee-ground" emesis.

Melena: Black, tarry, foul-smelling stool (digested blood).

Common Causes:

- Peptic Ulcer Disease (PUD) - 50% cases
- Esophageal Varices
- Mallory-Weiss Tears

↓ Lower GI Bleeding (LGIB)

Clinical Presentation:

Hematochezia: Bright red or maroon blood per rectum.

Usually associated with lower abdominal cramping.

Common Causes:

- Diverticulosis
- Inflammatory Bowel Disease (IBD)
- Hemorrhoids / Anal Fissures

NURSING ALERT: HEMODYNAMIC STABILITY ASSESSMENT

Monitor for **Hypovolemic Shock:** Tachycardia (HR >100), Hypotension (BP <90/60), Orthostatic changes, and Altered Mental Status.

Pulse BP

Gastric Disorders

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Gastritis

Inflammation of the gastric mucosa. Can be **Acute** (erosive, often due to drugs/alcohol) or **Chronic** (non-erosive, often H. pylori or autoimmune).

Peptic Ulcer Disease (PUD)

Excavation (ulceration) in the mucosal wall of the stomach, pylorus, or duodenum.

AGGRESSIVE FACTORS

- Gastric Acid (HCl)
- Pepsin
- H. pylori Infection
- NSAIDs use

DEFENSIVE FACTORS

- Mucus secretion
- Bicarbonate layer
- Mucosal blood flow
- Prostaglandins

Key Risk Factors

H. pylori Infection

Spiral-shaped bacteria that weaken the protective mucus coating, allowing acid to reach the sensitive lining beneath.

NSAIDs & Aspirin

Inhibit prostaglandin synthesis, which is essential for maintaining the gastric mucosal barrier.

Lifestyle Factors

Excessive alcohol consumption, smoking, and severe physiological stress (Stress Ulcers).

Zollinger-Ellison

Hypersecretion of gastric acid due to gastrin-secreting tumors (rare but severe).

"Pathophysiology: Imbalance between mucosal insult and mucosal defense."

Intestinal Obstruction

Mechanical Blockage vs. Functional Failure (Ileus)

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

PATHOPHYSIOLOGY

Mechanical Obstruction

Physical barrier preventing movement of contents.

- **Adhesions:** Most common (post-surgical scarring).
- **Hernias:** Protrusion through weakened wall.
- **Tumors:** Intrinsic or extrinsic compression.
- **Volvulus/Intussusception:** Twisting or telescoping.

Functional (Paralytic Ileus)

Failure of peristalsis without physical blockage.

- **Post-operative:** Temporary handling of bowels.
- **Electrolyte Imbalance:** Hypokalemia (low K+).
- **Inflammation:** Peritonitis or severe infection.
- **Medications:** Opioids or anticholinergics.

Pathophysiological Cascade

1. **Accumulation**
   Gas and fluid build up proximal to the obstruction.
2. **Distension**
   Intestinal wall stretches, increasing intraluminal pressure.
3. **Vascular Compromise**
   Pressure reduces capillary flow, leading to edema and ischemia.
4. **Systemic Crisis**
   Perforation risk, peritonitis, and severe fluid/electrolyte shifts.

FLUID SHIFT
Dehydration & Hypovolemia

PH IMBALANCE
Metabolic Alkalosis/Acidosis

SEPSIS RISK
Bacterial Translocation

Malabsorption Syndromes

Failure of the Intestinal Mucosa to Absorb Vital Nutrients

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Pathophysiological Basis

Malabsorption occurs when there is an interference with **digestion** (intraluminal), **absorption** (mucosal), or **transport** of nutrients into the systemic circulation.

Celiac Disease

- **Mechanism:** T-cell mediated autoimmune response to **Gluten**.
- **Pathology:** Villous atrophy & crypt hyperplasia.
- **Result:** Reduced surface area for absorption.

Lactose Intolerance

- **Mechanism:** Deficiency of the enzyme **Lactase**.
- **Pathology:** Undigested lactose stays in lumen.
- **Result:** Osmotic diarrhea & gas production.

Nutritional Consequences

Weight Loss & Muscle Wasting

Failure to absorb calories (Carbs/Proteins).

Anemia

Malabsorption of Iron, Vit B12, or Folate.

Bone Disorders

Osteoporosis due to Vitamin D & Calcium deficiency.

Steatorrhea

Fatty, foul-smelling stools due to fat malabsorption.

Nursing Tip: *Monitor for signs of dehydration and specific vitamin deficiencies (e.g., night blindness - Vit A, bruising - Vit K).*

Inflammatory Bowel Disease (IBD)

Chronic, relapsing immune-mediated inflammatory disorders

Crohn's Disease

- **Location:** Any part of GI (Mouth to Anus). Mostly terminal ileum & cecum.
- **Depth: Transmural** (affects all layers of the bowel wall).
- **Pattern: Skip Lesions** (healthy tissue between diseased areas).

Ulcerative Colitis

- **Location:** Limited to **Colon and Rectum**. Starts distally.
- **Depth: Mucosal** (affects inner lining: Mucosa & Submucosa).
- **Pattern: Continuous** inflammation without skip areas.

Feature	Crohn's Disease	Ulcerative Colitis
Clinical Hallmark	Abdominal pain, Malabsorption	Bloody diarrhea, Tenesmus
Morphology	Cobblestone appearance, Strictures	Pseudopolyps, Friable mucosa
Complications	Fistulas, Abscesses, Perforation	Toxic Megacolon, Colorectal Cancer

Both conditions significantly increase the risk of systemic extra-intestinal manifestations (e.g., Uveitis, Arthritis).

Irritable Bowel Syndrome (IBS)

A Functional Disorder of the Gut-Brain Axis

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Key Pathophysiology

Gut-Brain Dysregulation:
Altered signaling between the CNS and enteric nervous system.

Visceral Hypersensitivity:
Lowered pain threshold to intestinal stretching/distension.

Post-Infectious Changes:
Low-grade inflammation or dysbiosis after GI infection.

Clinical Presentation

Rome IV Criteria highlights:

- Abdominal pain related to defecation
- Change in stool frequency
- Change in stool form (appearance)

IBS vs. IBD: Critical Distinctions

Feature	IBS (Syndrome)	IBD (Disease)
Type	Functional	Structural/Organic
Inflammation	Absent (Normal mucosa)	Present (Visible damage)
Diagnostic Tests	Normal imaging/ Biopsy	Abnormal (Ulcers/Strictures)
Systemic Signs	None (No fever/ weight loss)	Fever, weight loss, anemia
Risk of Cancer	No increased risk	Increased risk (Colorectal)

⚠ RED FLAGS (NOT IBS)
Rectal bleeding, nocturnal diarrhea, progressive pain, or onset after age 50 require immediate investigation for organic disease.

Note: IBS is a diagnosis of exclusion. Nursing focus is on symptom management and dietary education (FODMAP).

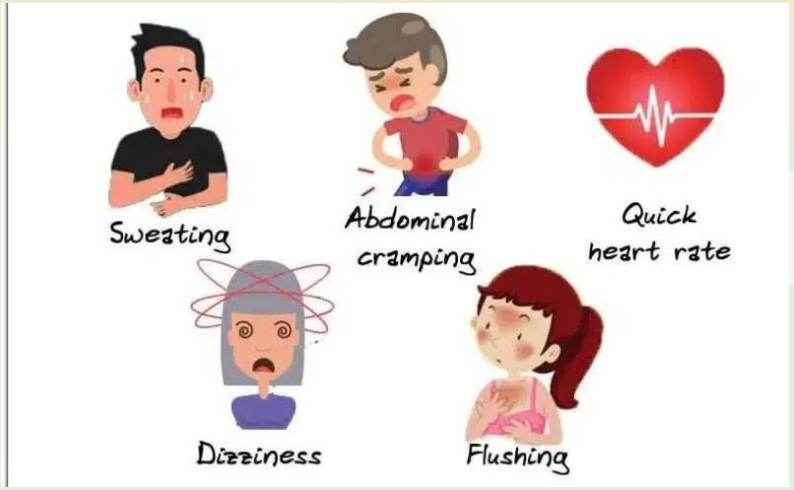
Dumping Syndrome

Rapid Emptying: Occurs when food moves too quickly from the stomach to the small intestine, often after gastric surgery.

Phased Symptoms: Early (10-30 mins) includes nausea and cramps; Late (1-3 hours) involves hypoglycemia and shakiness.

Management

Controlled through small, frequent meals, high protein/fiber intake, and avoiding simple sugars.



Liver Cirrhosis & Complications

End-stage chronic liver disease characterized by diffuse fibrosis and nodular regeneration.

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Pathophysiological Progression

01 Chronic Hepatocyte Injury

Alcohol, Hepatitis B/C, or NAFLD leads to inflammation.

02 Fibrosis & Scarring

Activation of Stellate cells; healthy tissue replaced by collagen.

03 Structural Distortion

Nodules compress vascular and biliary channels.

04 Portal Hypertension

Increased resistance to blood flow through the liver.

Portal Hypertension Effects

- Esophageal Varices
- Splenomegaly
- Encephalopathy
- Caput Medusae

Ascites Formation

Accumulation of fluid in the peritoneal cavity due to:

HYDROSTATIC	ONCOTIC
↑ Portal Pressure	↓ Albumin Synthesis

Result: "Third Spacing" & Severe Edema

Nursing Alert: Observe for signs of GI bleeding (hematemesis/melena) and altered mental status (ammonia toxicity), as these represent life-threatening emergencies in cirrhotic patients.

Jaundice & Bilirubin Metabolism
Hyperbilirubinemia: Pathophysiology and Classification

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

CLINICAL SIGN

What is Jaundice?

A yellowish discoloration of the skin, mucous membranes, and sclera (icterus) caused by elevated serum bilirubin levels (usually > 2.5–3.0 mg/dL).

SIMPLIFIED METABOLISM PATHWAY

Heme (RBCs) *Breakdown* → **Unconjugated** Indirect (Lipid Soluble) → **Conjugated** Direct (Water Soluble) → **Excretion** *Bile / Urine / Feces*

Classifying by Cause

Pre-Hepatic

"Too much produced"

- **Cause:** Excessive hemolysis (RBC destruction).
- **Bilirubin:** High levels.
- **Examples:** Sickle cell anemia, transfusion reactions, hemolytic disease.

Intra-Hepatic

"Liver cannot process"

- **Cause:** Hepatocellular damage or impaired uptake.
- **Bilirubin:** Mixed (Both Unconjugated & Conjugated).
- **Examples:** Hepatitis, Cirrhosis, Liver Cancer, Neonatal jaundice.

Post-Hepatic

"Bile cannot flow out"

- **Cause:** Obstruction of the biliary drainage system.
- **Bilirubin:** High levels.
- **Examples:** Gallstones (Cholelithiasis), Pancreatic tumor, Bile duct stricture.

NURSING ALERT: Observe for dark (tea-colored) urine and clay-colored stools, which often indicate Post-Hepatic obstruction.

Cholelithiasis &Cholecystitis

Gallstone Formation and Gallbladder Inflammation

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

BILIARY DISORDERS

Cholelithiasis

DEFINITION

Presence of stones (calculi) in the gallbladder or biliary duct system.

THE "5 FS" RISK FACTORS

Female	**Fat** (BMI > 30)
Forty+	**Fertile**
Fair	

CAUSES

Cholesterol supersaturation, bile stasis, and biliary sludge formation.

Cholecystitis

PATHOGENESIS

Acute or chronic inflammation of the gallbladder wall, usually triggered by cystic duct obstruction.

The Obstruction Cycle:

1. Gallstone blocks the duct.
2. Bile builds up (pressure increases).
3. Chemical irritation & ischemia.
4. Secondary bacterial infection.

Impact on Digestion

Bile Deficiency
Lack of bile salts in the duodenum prevents fat emulsification.

Steatorrhea
Undigested fat excreted in stool (greasy, foul-smelling, floating).

Vitamin Malabsorption
Deficiency in fat-soluble vitamins (A, D, E, and K).

1 **STONE FORMATION** *Bile Stasis* → 2 **DUCT OBSTRUCTION** *Pressure Increase* → 3 **INFLAMMATION** *Pain & Fever* → 4 **FAT INTOLERANCE** *Nausea & Steatorrhea*

Pancreatitis: Acute &Chronic

Inflammation and Autodigestion of the Pancreas

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

GI PATHOLOGY

Acute Pancreatitis

SUDDEN & POTENTIALLY LIFE-THREATENING

- **Primary Causes:** Gallstones (obstruction) and Alcohol abuse.
- **Mechanism:** Premature activation of enzymes inside the pancreas.
- **Symptoms:** Severe epigastric pain radiating to the back, nausea, vomiting.

Chronic Pancreatitis

CONTINUOUS & IRREVERSIBLE

- **Primary Cause:** Long-term heavy alcohol consumption (most common).
- **Mechanism:** Progressive fibrosis and calcification of pancreatic tissue.
- **Outcome:** Permanent loss of exocrine and endocrine functions.

Pathophysiology: The Autodigestion Cycle

TRIGGER
Biliary obstruction or alcohol toxicity

→

ENZYME ACTIVATION
Trypsinogen converts to Trypsin prematurely

→

AUTODIGESTION
Proteolysis and Lipolysis of pancreatic tissue

→

NECROSIS
Hemorrhage, edema, and systemic inflammation

Exocrine Failure
Lipase deficiency leads to Steatorrhea (fatty stools).

Malabsorption
Inability to digest proteins and fats; Weight loss.

Secondary Diabetes
Destruction of Islets of Langerhans (Insulin loss).

Pancreatic Cancer: Symptoms by Location

Head of Pancreas

Early Jaundice: Yellowing of skin and eyes due to bile duct obstruction.

Other Signs: Dark urine, pale stools, and persistent itching (pruritus).

Body and Tail

Silent Growth: Often asymptomatic until the tumor becomes large.

Key Symptoms: Deep abdominal pain radiating to the back and rapid weight loss.

Infectious Enterocolitis

Pathogen Type	Pathophysiology	Clinical Presentation
Rotavirus	Destroys villi tips directly; reduces absorption area	Severe watery diarrhea; common in children
Bacterial	Toxins stimulate fluid secretion or tissue invasion	Mucobloody stools; caused by E. coli or Salmonella

**Bacterial infections often involve direct damage to the intestinal lining.*

Colorectal Cancer (CRC)

Pathogenesis

Develops from polyps over 10-15 years via APC and p53 gene mutations.

Risk Factors

Age, red meat consumption, smoking, and chronic IBD history.

Warning Signs

Bowel changes, bloody stools, abdominal pain, and unexplained weight loss.

Early detection leads to a survival rate higher than 90%.

Clinical Assessment & Physical Examination

Bridging Pathophysiology to Bedside Practice

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

NURSING SKILLS

Standardized Abdominal Sequence

1. Inspection
Contour, symmetry, skin changes, or pulsations.

2. Auscultation
Bowel sounds (always before palpation to avoid false results).

3. Percussion
Identify gas, fluid, or solid masses (Tympany vs. Dullness).

4. Palpation
Light for tenderness; Deep for organs or masses.

Critical Diagnostic Signs

Rebound Tenderness (Blumberg's Sign)
Pain upon sudden release of deep pressure
Patho: Suggests peritoneal inflammation (Peritonitis/Appendicitis).

Ascites Assessment
Fluid Wave & Shifting Dullness.
Patho: Accumulation of fluid due to portal HTN or low albumin (Cirrhosis).

Murphy's Sign
Inspiratory arrest during RUQ palpation
Patho: Gallbladder inflammation (Cholecystitis).

Nursing Assessment (History)

Pain Characteristics
Location (quadrants), radiation, quality (cramping vs. sharp).

Bowel Habit Changes
Frequency, consistency, color (Melena/Hematochezia), and odor.

Associated Symptoms
Anorexia, nausea, vomiting, dysphagia, or early satiety.

Always assess the painful area last during palpation to maintain patient comfort and accurate findings.

Diagnostic Tests Interpretation

Evaluating GI Integrity, Structure, and Function

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Endoscopy

Upper GI (EGD)

Visualizes esophagus, stomach, duodenum. Detects ulcers, GERD, and H. pylori.

Lower GI (Colonoscopy)

Identifies polyps, tumors, and IBD lesions in the colon and rectum.

Note: Biopsy capability is the "Gold Standard" for malignancy diagnosis.

Imaging

- **Abdominal CT/MRI:** Cross-sectional view for tumors, pancreatitis, or abscesses.
- **Ultrasound:** Preferred for gallstones (Cholelithiasis) and liver parenchyma assessment.
- **KUB X-Ray:** Rapid screening for intestinal obstruction (air-fluid levels) or perforation (free air).

Stool Analysis

Occult Blood (OB)	Bleeding
Fat Content	Malabsorption
Ova & Parasites	Infection
Calprotectin	IBD Flare

CLINICAL SIGNIFICANCE

Pre-procedure preparation (NPO, Bowel prep)

Post-procedure monitoring (Vitals, Perforation signs)

Correlation of lab results with clinical symptoms

Case Studies & Clinical Application

Bridging Theory to Bedside Nursing Practice

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

Scenario: Hepatic Dysfunction

Clinical Case: Mr. Somsak

PROFILE
54-year-old Male

HISTORY
Chronic Alcohol Use, Cirrhosis

Physical Assessment Findings:

- **Abdomen:** Distended with visible caput medusae; positive fluid wave test (Ascites).
- **Integumentary:** Scleral icterus (jaundice) and spider angiomas on the chest.
- **Neurological:** Mild confusion, "flapping" hand tremors (Asterixis).

Lab Alert!

Serum Albumin: 2.1 g/dL (Low) **Total Bilirubin: 4.8 mg/dL (High)** **Ammonia: 110 µmol/L (High)**

Critical Thinking

"Based on the Starling's Law principles learned in Section 5, explain why this patient developed Ascites and Peripheral Edema?"

Keywords: Oncotic Pressure, Albumin, Portal Pressure.

Summary & Key Points

Essential takeaway for nursing practice and exams

มหาวิทยาลัยราชภัฏนครปฐม
Nakhon Pathom Rajabhat University

CONCLUSION

Pathophysiology Core

- Understand the balance between **Aggressive factors** (acid/enzymes) and **Protective factors** (mucus/bicarb).
- The **ENS** and **Hormones** (Gastrin, CCK) regulate motility and secretions.
- Inflammation (IBD) vs. Functional disturbances (IBS).

Clinical Red Flags

- **GI Bleeding:** Hematemesis (Upper) vs. Melena/ Hematochezia (Lower).
- **Obstruction:** Distension, vomiting, and absence of flatus.
- **Pancreatitis:** Severe epigastric pain radiating to the back.

Nursing Priorities

- **Assessment:** Inspection, Auscultation, Percussion, and Palpation (IAPP order).
- **Fluid Balance:** Monitor for dehydration in vomiting/diarrhea cases.
- **Nutrition:** NPO status for acute conditions and tailored diets for IBD/GERD.

Next Step: Apply these concepts to the clinical simulations in the lab!

Questions?

มหาวิทยาลัยราชภัฏนครปฐม