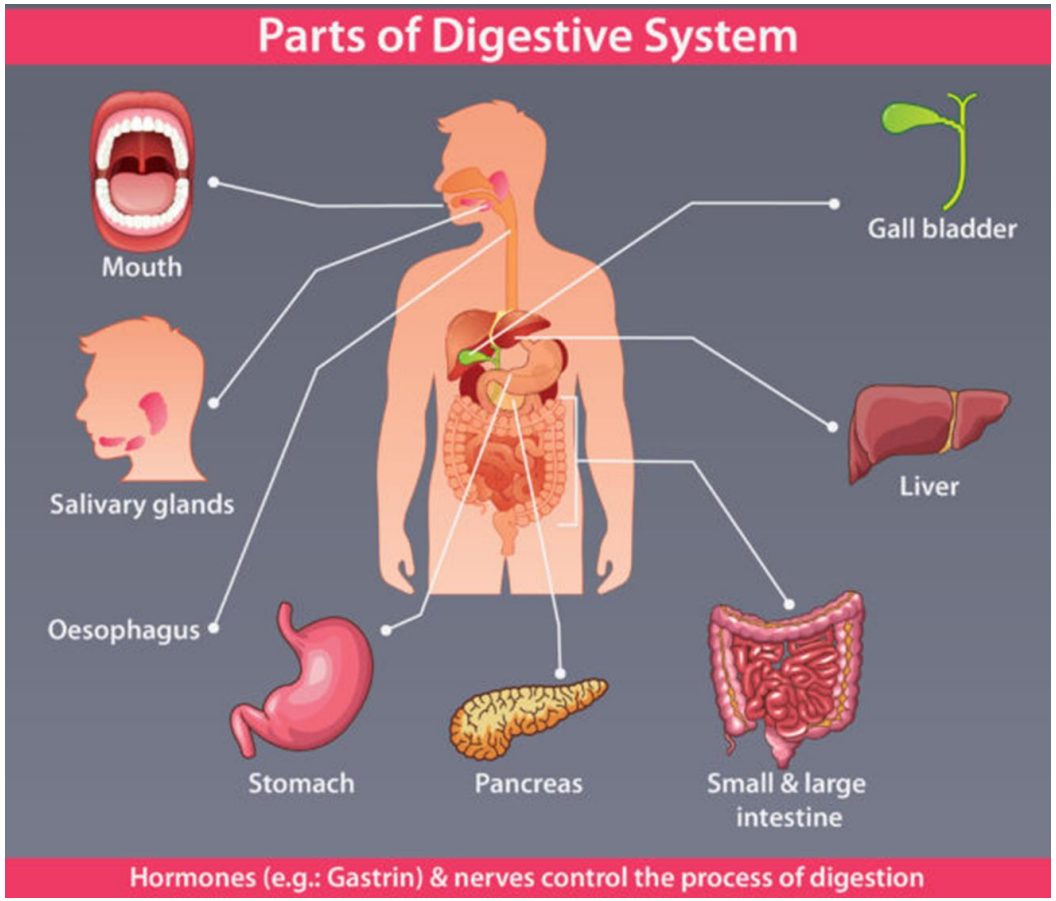
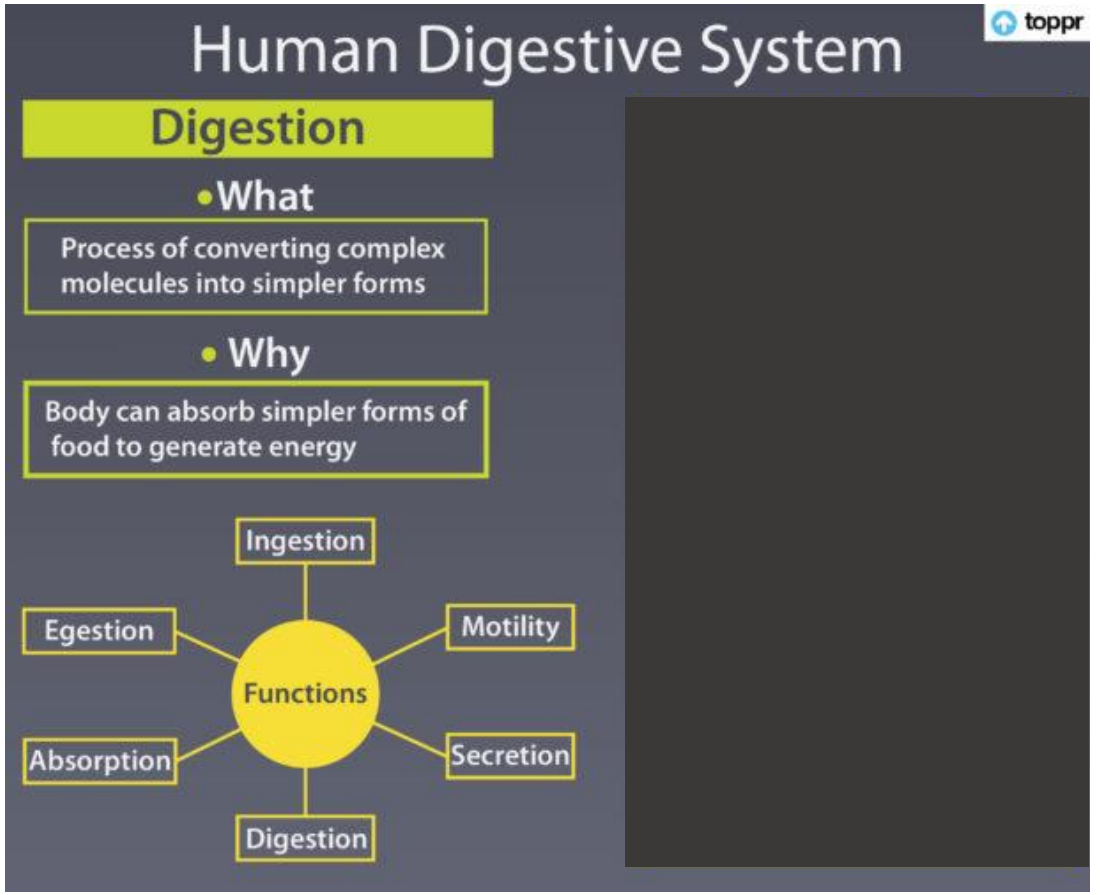
TERAPI GIZI PADA PENYAKIT GASTROINTESTINAL

TIRTA PRAWITA SARI

Updated 2023


Parts of Digestive System
Mouth
Gall bladder
Salivary glands
Liver
Oesophagus
Stomach
Pancreas
Small & large intestine
Hormones (e.g.: Gastrin) & nerves control the process of digestion

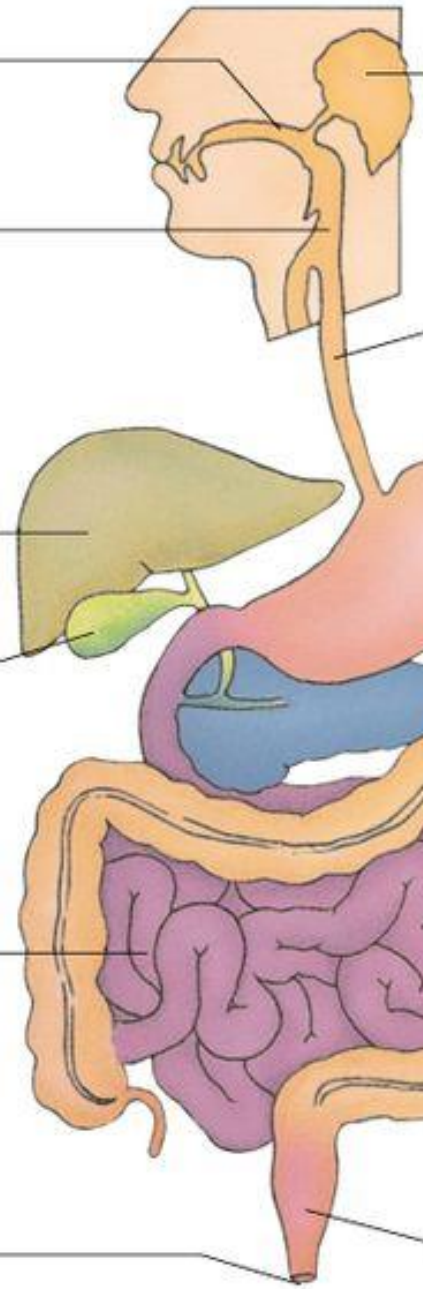

toppr
Human Digestive System
Digestion
•What
Process of converting complex molecules into simpler forms
• Why
Body can absorb simpler forms of food to generate energy
Ingestion
Egestion
Motility
Functions
Absorption
Secretion
Digestion

Mouth
Breaks up food particles
Assists in producing spoken language

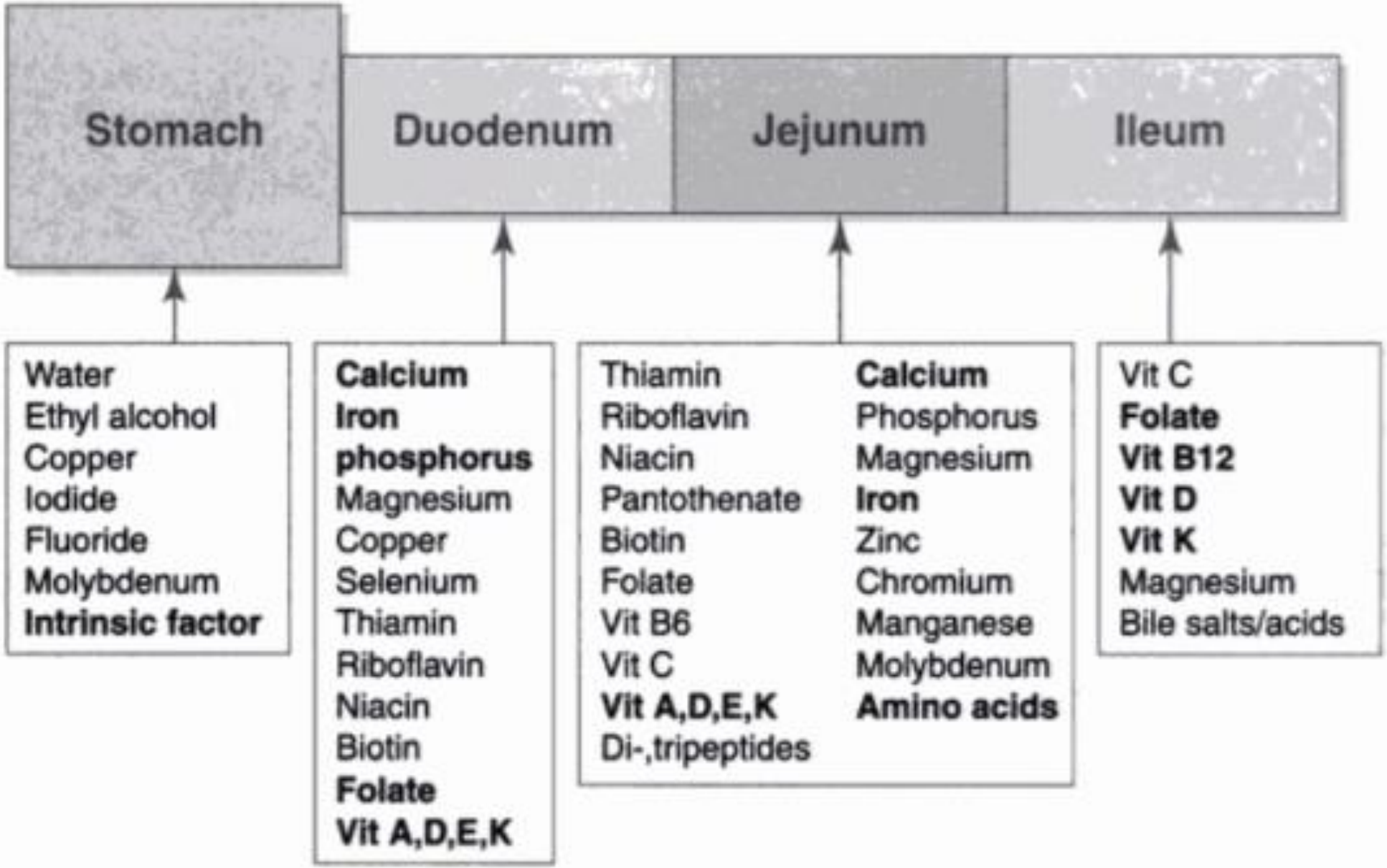
Pharynx
Swallows

Salivary glands
Saliva moistens and lubricates food
Amylase digests polysaccharides

Esophagus
Transports food



Sites of Nutrient Absorption in the GI Tract



Digestive hormones in the GI tract

HORMONE	LOCALIZATION	MAIN PHYSIOLOGIC ACTIONS
Gastrin	Gastric antrum, duodenum (G cells)	**-stimulate secretion of gastric acid and intrinsic factor from parietal cells** **-stimulate secretion of pepsinogen from chief cells** -promotes gastric and intestinal motility, mucosal growth
Cholecystokinin (CCK)	Duodenum, jejunum (I cells)	**-stimulate gallbladder contraction** **-stimulates release of pancreatic enzymes** **-relaxes sphincter of Oddi for release of bile and enzymes** -role in inducing satiety
Secretin	Duodenum, jejunum (S cells)	**-stimulate secretion of HCO_3 from pancreas** -inhibits gastrin and gastric acid secretion
Vasoactive intestinal peptide (VIP)	Enteric nerves	**-increases water and electrolyte secretion from pancreas and gut** -relaxes smooth muscles (via nitric oxide) of the gut
Gastric inhibitory polypeptide (GIP)	Duodenum, jejunum (K cells)	**-reduces gastric acid secretion** and intestinal motility **-stimulates insulin release**
Motilin	Throughout the gut (Mo cells and ECL cells)	**-increases small bowel motility (MMC during fasting) and gastric emptying**
Somatostatin	Stomach, small intestine, and pancreas (D cells)	**-inhibits secretion and action of many hormones, including all of the above**

Enzyme Name	Source	Substrates	Products	Notes (if applicable)
Aminopeptidase	Small intestine	Amino acid at amino end of peptides	Amino acids & peptides	n/a
Carboxypeptidase	Pancreatic acinar cells	Amino acid at carboxyl end of peptides	Amino acids & peptides	Activated from procarboxypeptidase by trypsin; optimum pH varies
Chymotrypsin	Pancreatic acinar cells	Proteins	Peptides	Activated from chymotrypsinogen by trypsin; optimum pH 7.8
Deoxyribonuclease	Pancreatic acinar cells	DNA	Nucleotides	n/a
Dipeptidase	Small intestine	Dipeptides	Amino acids	n/a
Elastase	Pancreatic acinar cells	Proteins	Peptides	Activated from proelastase by trypsin
Enterokinase	Small intestine	Trypsinogen	Trypsin	n/a
Gastric lipase	Stomach chief cells	Triglycerides	Fatty acids & monoglycerides	Optimum pH 4-5
Lactase	Small intestine	Lactose	Glucose & galactose	n/a
Lingual lipase	Lingual glands in tongue	Triglycerides (fats & oils) & other lipids	Fatty acids & diglycerides	Optimum pH 4.5-5.5
Maltase	Small intestine	Maltose	Glucose	n/a
Nucleosidases & phosphatases	Small intestine	Nucleotides	Nitrogenous bases, pentoses, phosphates	n/a
Pancreatic amylase	Pancreatic acinar cells	Starches	Maltose, maltotriose, α-dextrins	Optimum pH 6.7-7.0
Pancreatic lipase	Pancreatic acinar cells	Triglycerides emulsified by bile salts	Fatty acids & monoglycerides	Optimum pH 8.0
Pepsin	Stomach chief cells	Proteins	Peptides	Activated from pepsinogen by pepsin & HCl; optimum pH 1.5-1.6
Ribonuclease	Pancreatic acinar cells	RNA	Nucleotides	n/a
Sucrase	Small intestine	Sucrose	Glucose & fructose	n/a
Salivary amylase	Salivary glands	Starches	Maltose, maltotriose (trisaccharide), α-dextrins	Optimum pH 6.7-7
Trypsin	Pancreatic acinar cells	Proteins	Peptides	Activated from trypsinogen by enterokinase; optimum pH 7.8-8.7
α-Dextrinase	Small intestine	α-dextrins	Glucose	n/a

PHYSIOLOGIC CHANGE	DISORDERS RELATED TO CHANGE	NURSING INTERVENTIONS	RATIONALES
Stomach			
Atrophy of the gastric mucosa is characterized by a decrease in the ratio of gastrin-secreting cells to somatostatin-secreting cells. This change leads to decreased hydrochloric acid levels (hypochlorhydria).	Decreased hydrochloric acid levels lead to decreased absorption of iron and vitamin B_{12} and to proliferation of bacteria. Atrophic gastritis occurs as a consequence of bacterial overgrowth.	Encourage bland foods high in vitamins and iron. Assess for epigastric pain.	Bland foods help prevent gastritis. Assessment helps detect gastritis.
Large Intestine			
Peristalsis decreases, and nerve impulses are dulled.	Decreased sensation to defecate can result in postponement of bowel movements, which leads to constipation and impaction.	Encourage a high-fiber diet and 1500 mL of fluid intake daily (if not contraindicated). Encourage as much activity as tolerated.	These interventions increase the sensation of needing to defecate.
Pancreas			
Distention and dilation of pancreatic ducts change. Calcification of pancreatic vessels occurs with a decrease in lipase production.	Decreased lipase level results in decreased fat absorption and digestion. Steatorrhea, or excess fat in the feces, occurs because of decreased fat digestion.	Encourage small, frequent feedings. Assess for diarrhea.	Small, frequent feedings help prevent steatorrhea. Diarrhea may be steatorrhea. Excessive diarrhea can lead to dehydration.
Liver			
A decrease in the number and size of hepatic cells leads to decreased liver weight and mass. This change and an increase in fibrous tissue lead to decreased protein synthesis and changes in liver enzymes. Enzyme activity and cholesterol synthesis are diminished.	Decreased enzyme activity depresses drug metabolism, which leads to accumulation of drugs—possibly to toxic levels.	Assess for adverse effects of all drugs.	Assessment can help detect drug toxicity.

Assessment of nutritional status

Methodology in clinical practice

First Stage Assessment

Nutrient intake
Compared to estimated requirements

Clinical Signs
External signs- skin, hair, eyes..

Anthropometry
Height, weight, circumference, skinfold

Second Stage Assessment

Biochemistry and Haematology
Blood and/or urine tests for protein status, vitamin, mineral and trace element status

Third Stage or Research protocols

Body Composition
Distribution of fat, lean, water and minerals

Functional Tests
Neurological function
Developmental tests

Review > Gastroenterol Clin North Am. 2018 Mar;47(1):1-22. doi: 10.1016/j.gtc.2017.09.002. Epub 2017 Dec 7.

Malnutrition in Gastrointestinal Disorders: Detection and Nutritional Assessment

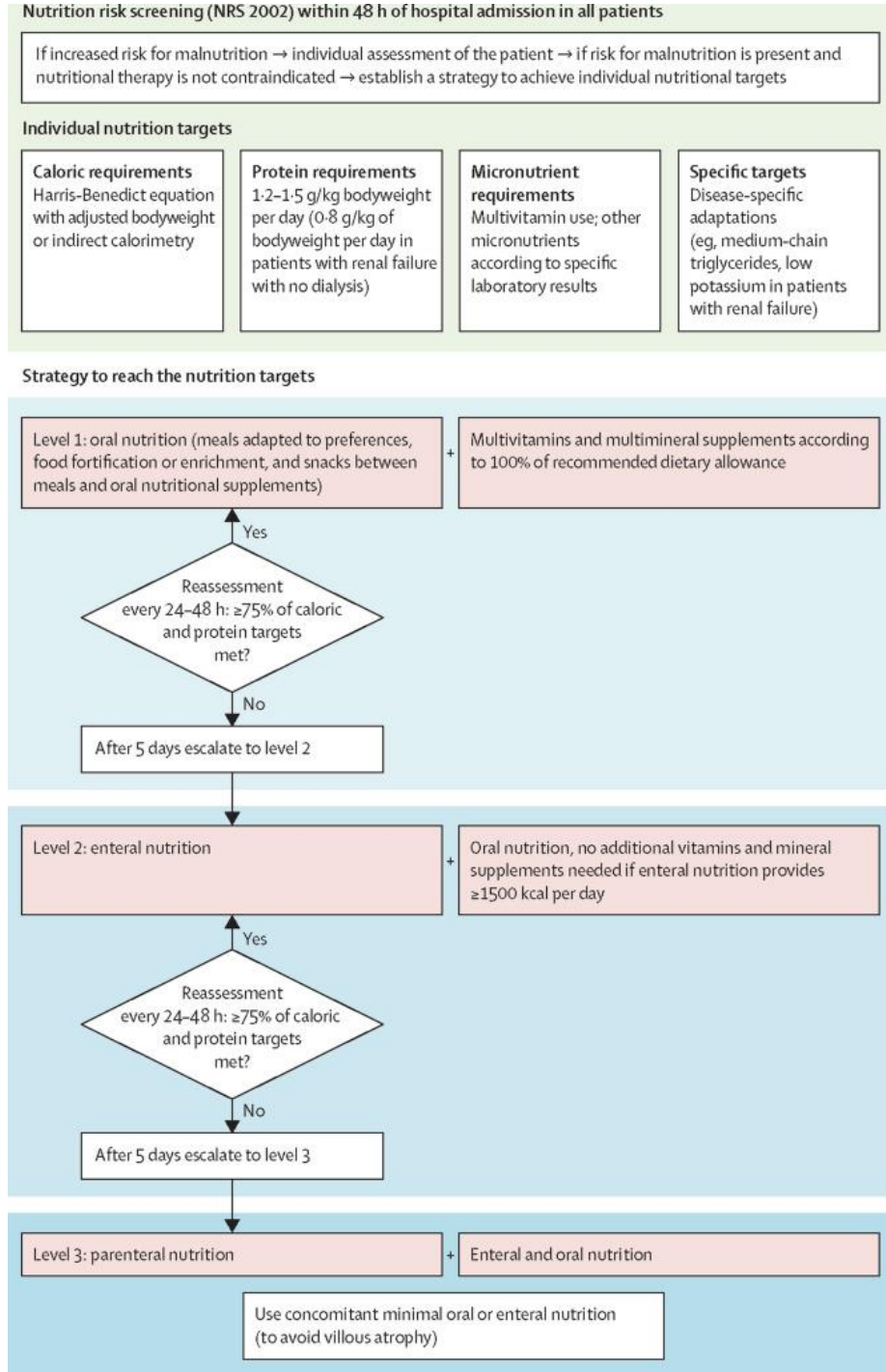
Khursheed N Jeejeebhoy [1], Donald R Duerksen [2]

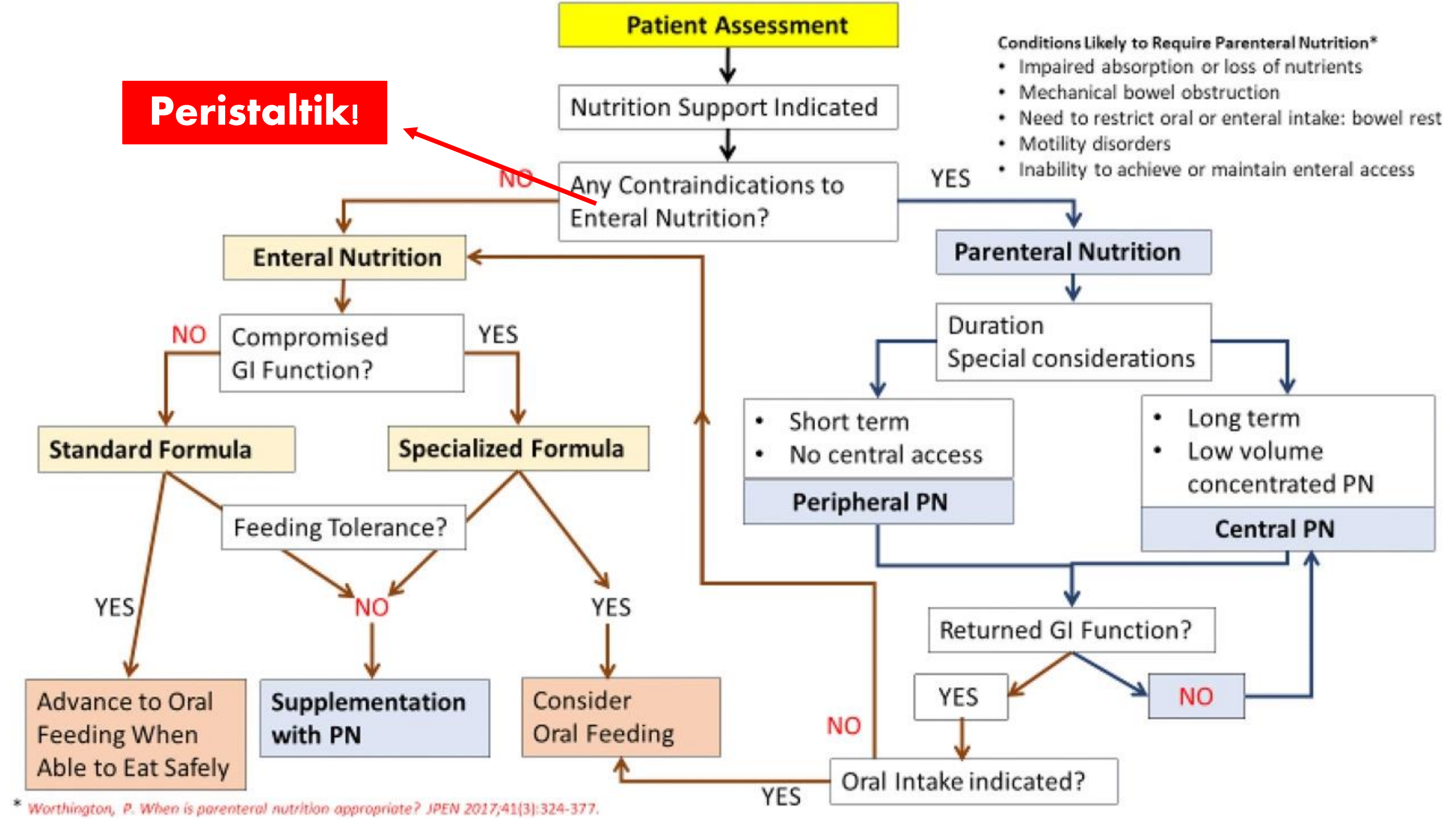
- All patients with significant gastrointestinal disease should be clinically assessed for protein calorie malnutrition by using the **Subjective Global Assessment**.
- Blood tests: **anemia, electrolytes, calcium, phosphorus, magnesium, ferritin, vitamin B_{12}, and folate** should be considered for assessment of major micronutrients.
- malabsorption or inflammatory bowel disease: **bone mineral density** using dual beam x-ray absorptiometry, **25-OH vitamin D levels**, and measurement of other vitamins and trace elements should be considered.
- in at-risk patients: vitamin and trace element clinical deficiency syndromes should be considered during patient assessment.

Normal lab value reference ranges differ between labs and institutions. Check with your facility for normal ranges.

Lab	Normal Value	Alteration	Potential Gastrointestinal Cause of Abnormal Value
Lipase	7-60 u/L	↑	Pancreatitis
Amylase	30-170 u/L	↑	Pancreatitis
Calcium	8.5-10.3 mg/dL	↓	Pancreatitis, malnutrition
Platelets	130-400 x 10^3/mm^3	↓	Liver dysfunction, cirrhosis, hepatitis, GI bleed
AST	< 42 u/L	↑	Liver dysfunction, cirrhosis, hepatitis
ALT	< 48 u/L	↑	Liver dysfunction, cirrhosis, hepatitis
Fibrinogen	200-400 mg/ dL	↓	Liver dysfunction, cirrhosis, hepatitis
Prothrombin Time (PT)	(PT) 10.0-12.5 sec	↑	Liver dysfunction, cirrhosis, hepatitis
Albumin	3.5-5.0 g/dL	↓	Liver dysfunction, cirrhosis, hepatitis, malnutrition
Bilirubin	≤ 1.3 mg/dL	↑	Liver dysfunction, cirrhosis, hepatitis, cholecystitis
Ammonia	0.17-0.80 mcg/mL	↑	Liver failure
Hemoglobin	12.0-17.2 g/dL	↓	GI bleed, hemorrhagic pancreatitis
Hematocrit	35-50%	↓	GI bleed, hemorrhagic pancreatitis
Electrolytes	variable	↑	Hemoconcentration in early GI bleed or hemorrhagic pancreatitis
BUN	7-30 mg/dL	↑	Hemoconcentration & absorption of protein (blood) in GI bleed hemorrhagic pancreatitis
WBC	3.8-10.8 x 10^3 /mm^3	↑	Infection of stress response of pancreatitis, GI bleed

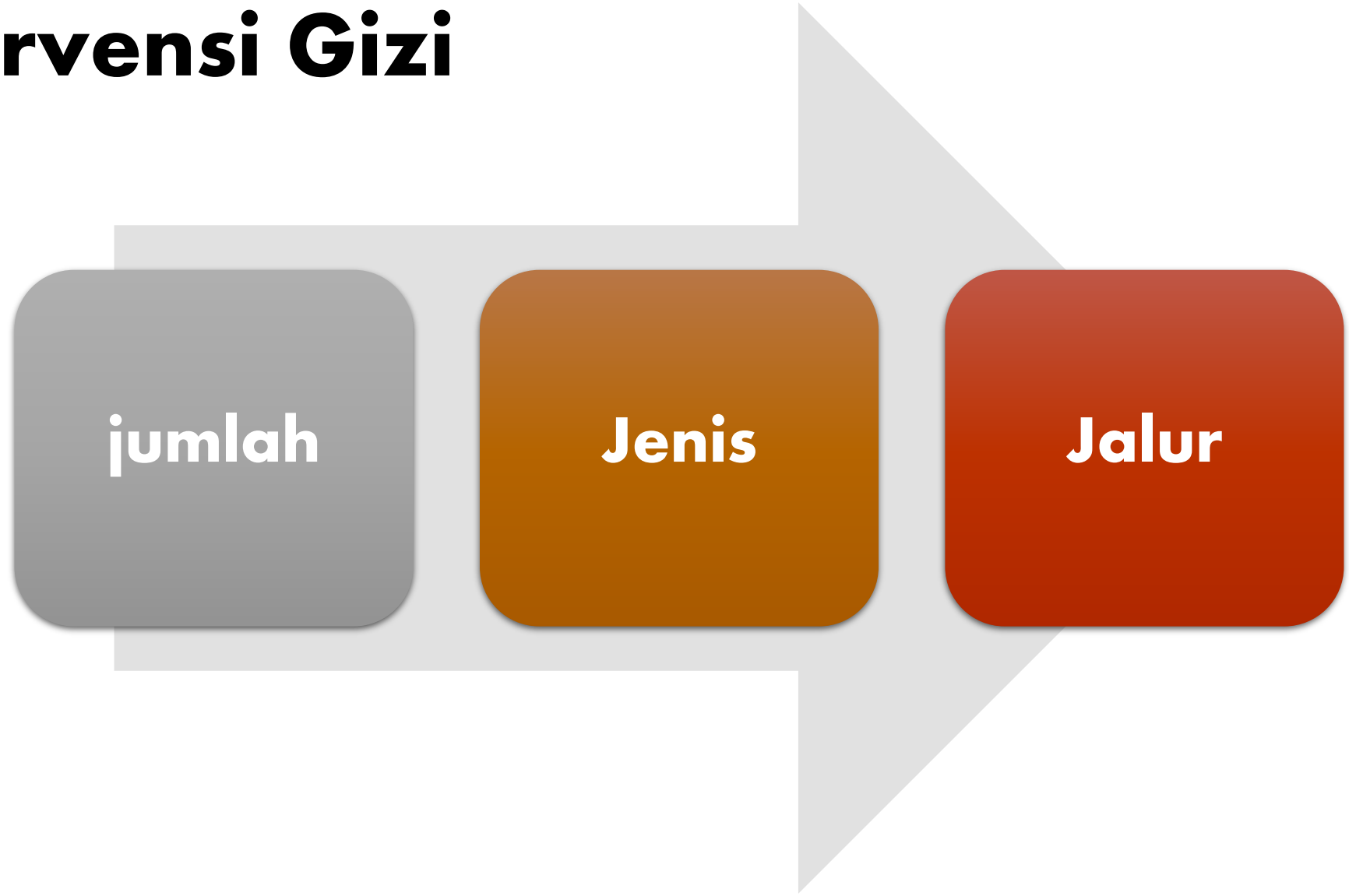
(Merck Manual Online, 2013)


Nutrition risk screening (NRS 2002) within 48 h of hospital admission in all patients
If increased risk for malnutrition → individual assessment of the patient → if risk for malnutrition is present and nutritional therapy is not contraindicated → establish a strategy to achieve individual nutritional targets
Individual nutrition targets
Caloric requirements
Harris-Benedict equation with adjusted bodyweight or indirect calorimetry
Protein requirements
1·2–1·5 g/kg bodyweight per day (0·8 g/kg of bodyweight per day in patients with renal failure with no dialysis)
Micronutrient requirements
Multivitamin use; other micronutrients according to specific laboratory results
Specific targets
Disease-specific adaptations (eg, medium-chain triglycerides, low potassium in patients with renal failure)
Strategy to reach the nutrition targets
Level 1: oral nutrition (meals adapted to preferences, food fortification or enrichment, and snacks between meals and oral nutritional supplements)
+
Multivitamins and multimineral supplements according to 100% of recommended dietary allowance
Yes
Reassessment every 24–48 h: ≥75% of caloric and protein targets met?
No
After 5 days escalate to level 2
Level 2: enteral nutrition
+
Oral nutrition, no additional vitamins and mineral supplements needed if enteral nutrition provides ≥1500 kcal per day
Yes
Reassessment every 24–48 h: ≥75% of caloric and protein targets met?
No
After 5 days escalate to level 3
Level 3: parenteral nutrition
+
Enteral and oral nutrition
Use concomitant minimal oral or enteral nutrition (to avoid villous atrophy)



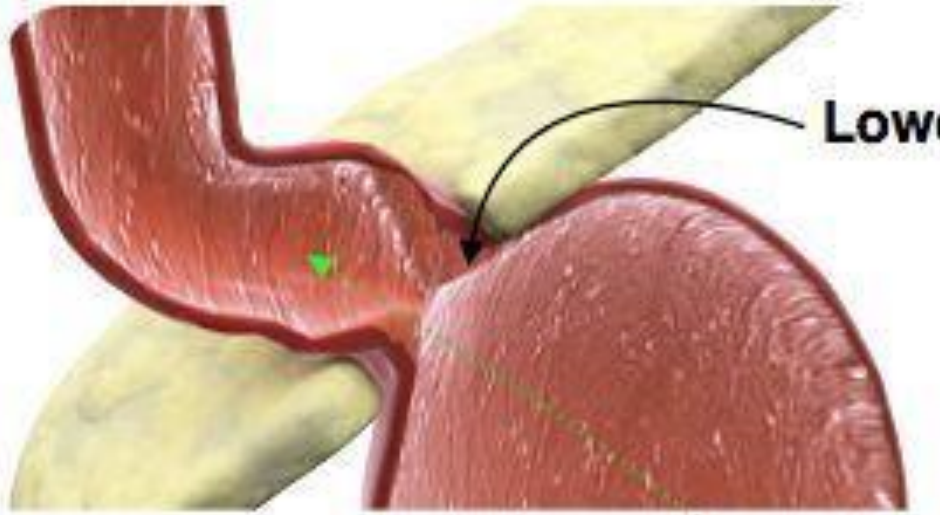
* Worthington, P. When is parenteral nutrition appropriate? JPEN 2017;41(3):324-377.

Intervensi Gizi



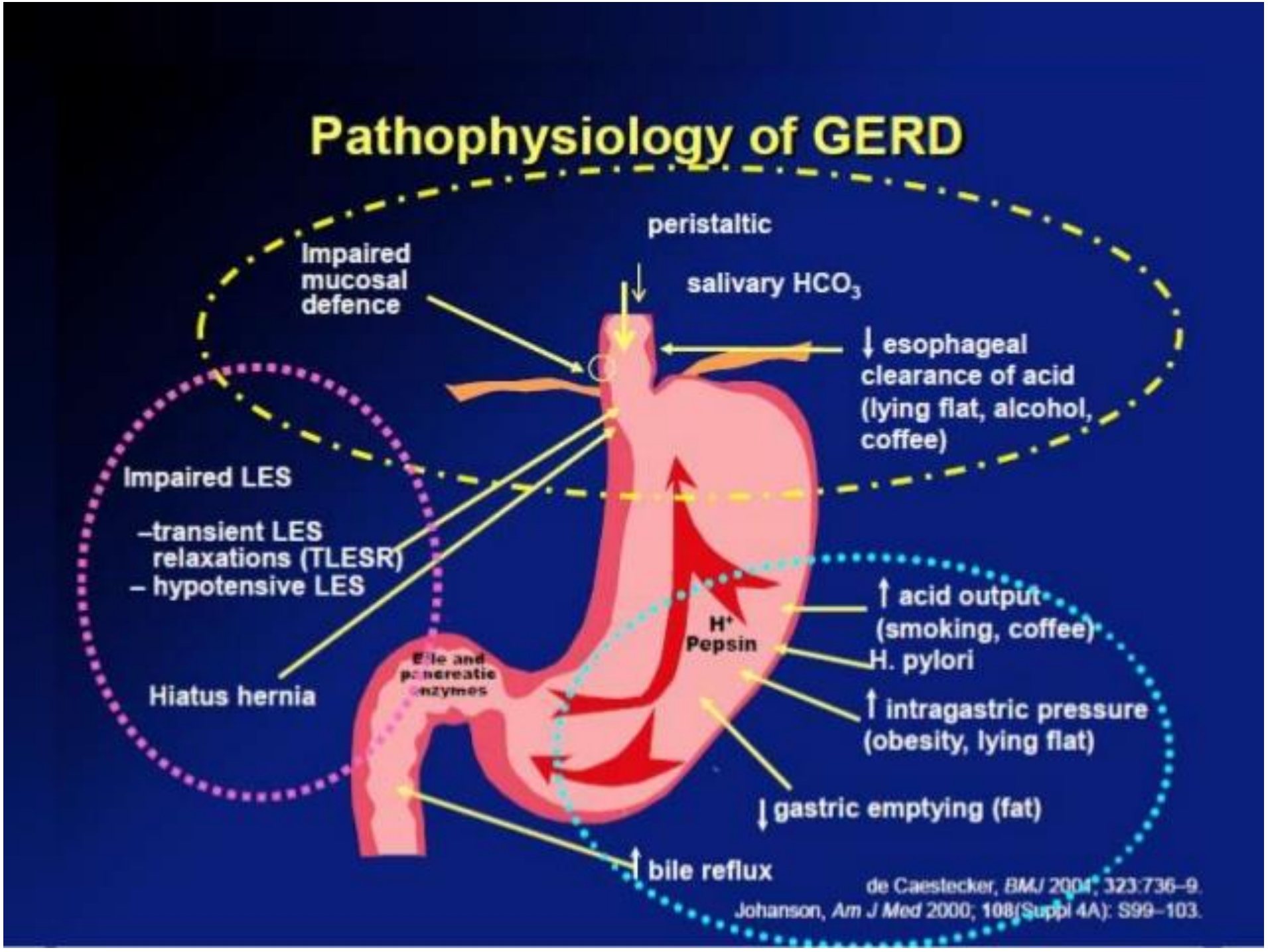
Sistem Gastrointestinal, Hepatobilier, dan Pankreas			
1	Mata kuning	15	Perut berbunyi
2	Mulut kering	16	Benjolan di daerah perut
3	Mulut berbau	17	Muntah
4	Sakit gigi	18	Muntah darah
5	Gusi bengkak	19	Sembelit atau tidak dapat berak
6	Sariawan	20	Diare
7	Bibir pecah-pecah	21	Berak berlendir dan berdarah
8	Bibir sumbing	22	Berak berwarna hitam
9	Sulit menelan	23	Berak seperti dempul
10	Cegukan/*hiccup*	24	Gatal daerah anus
11	Nyeri perut	25	Nyeri daerah anus
12	Nyeri ulu hati	26	Benjolan di anus
13	Perut kram	27	Keluar cacing
14	Perut kembung	28	Air kencing seperti teh

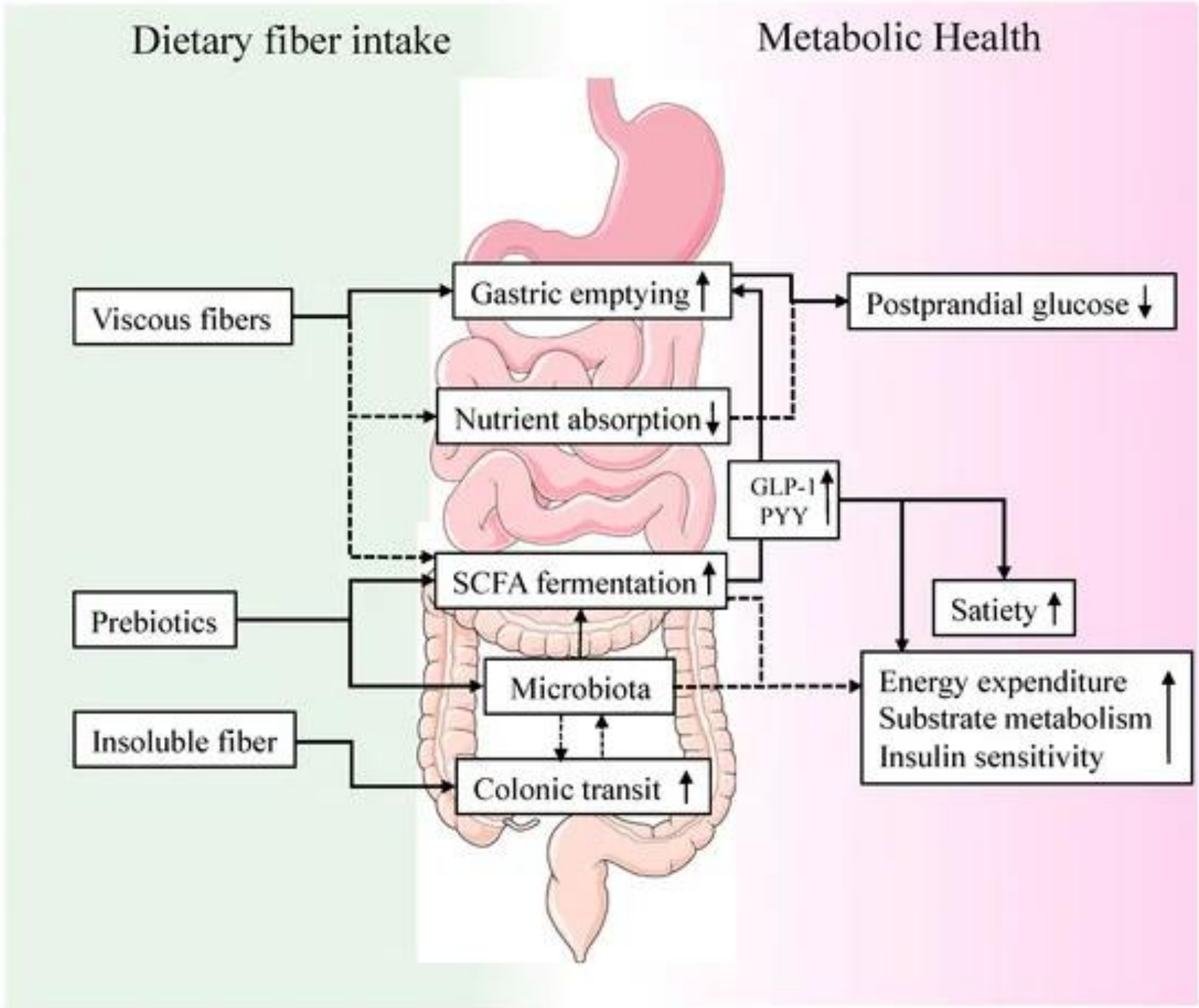
Gastroesophageal Reflux Disease



The 3 Dominant Pathophysiologic Mechanisms causing GERD

1. Transient lower esophageal sphincter relaxations
2. A hypotensive lower esophageal sphincter
3. Anatomic disruption of the gastroesophageal junction


Pathophysiology of GERD
peristaltic
Impaired mucosal defence
salivary HCO_3
↓ esophageal clearance of acid (lying flat, alcohol, coffee)
Impaired LES
–transient LES relaxations (TLESR)
– hypotensive LES
↑ acid output (smoking, coffee)
H. pylori
H^+
Pepsin
Hiatus hernia
↑ intragastric pressure (obesity, lying flat)
↓ gastric emptying (fat)
↑ bile reflux
de Caestecker, BMJ 2004; 323:736–9.
Johanson, Am J Med 2000; 108(Suppl 4A): S99–103.


Dietary fiber intake
Metabolic Health
Viscous fibers
Prebiotics
Insoluble fiber
Gastric emptying
Nutrient absorption
SCFA fermentation
Microbiota
Colonic transit
GLP-1
PYY
Postprandial glucose
Satiety
Energy expenditure
Substrate metabolism
Insulin sensitivity

ACID / ALKALINE FOOD COMPARISON CHART

MORE ACIDIC - EAT LESS

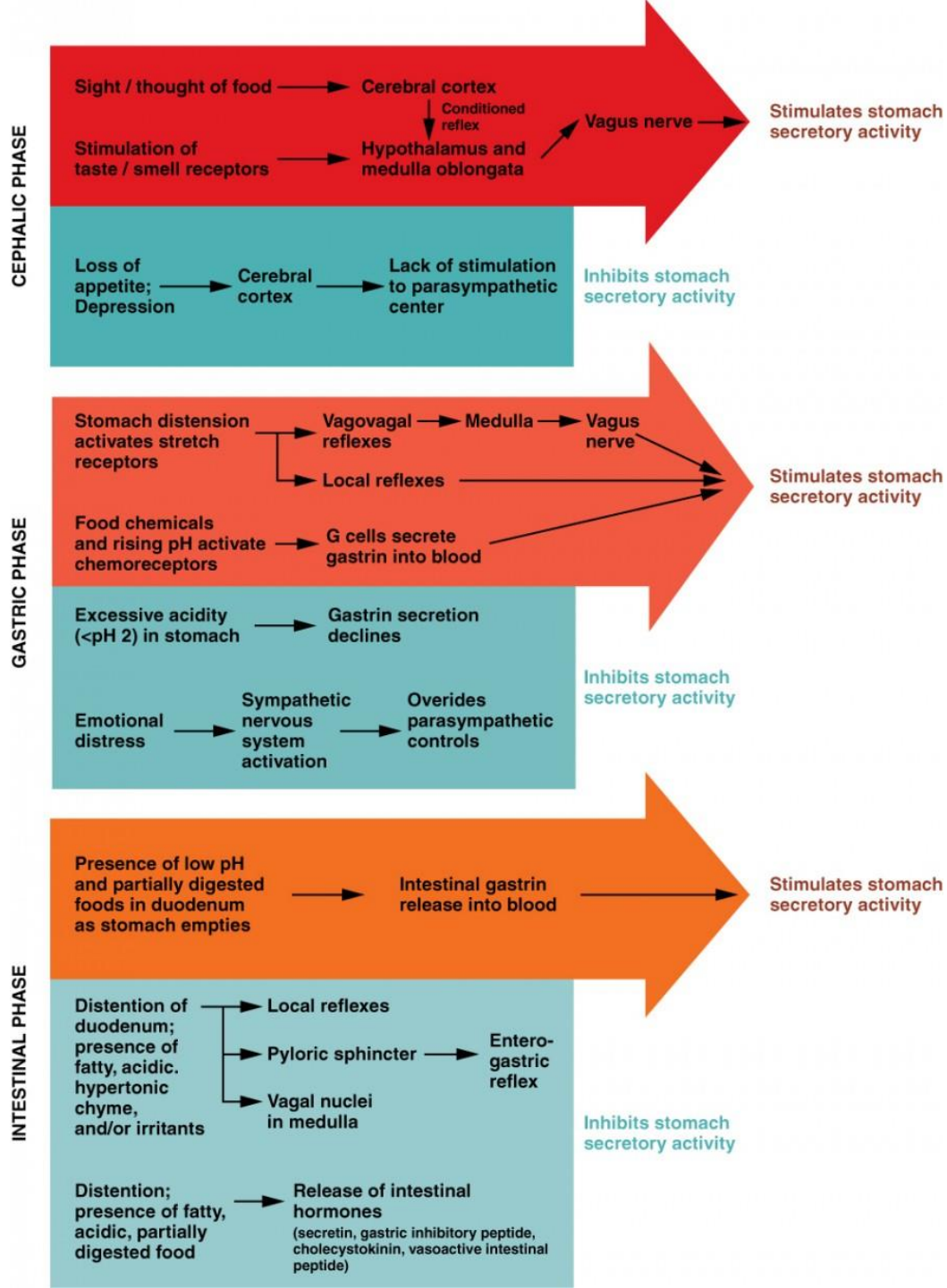
NEUTRAL

MORE ALKALINE - EAT MORE

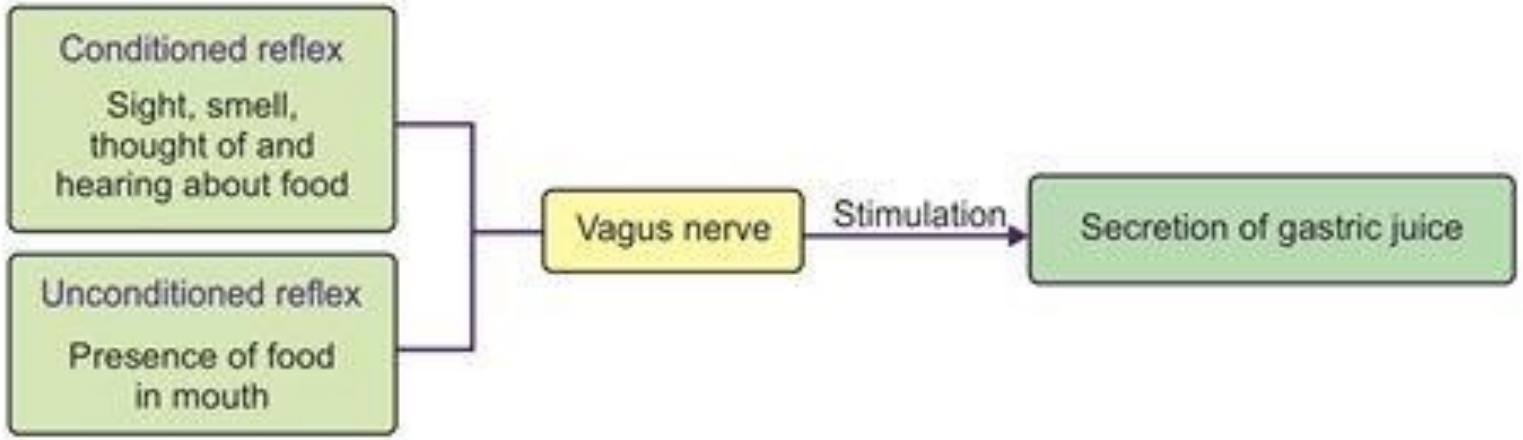
⊖⊖⊖⊖	⊖⊖⊖	⊖⊖	⊖		⊕	⊕⊕	⊕⊕⊕
Soft Drinks Energy Drink Carbonated Drinks ***Processed & Refined Food**	Popcorn Cream Cheese Buttermilk Pastries Pasta Cheese Pork Beef Beer, Wine Black Tea Pickles Roasted Nuts Vinegar Sweet & Low Equal, Nutra Sweet	Most Purified Water Distilled Water Coffee Chocolate Sweetened Fruit Juice Pistachios White Bread Peanuts Nuts Wheat	Fruit Juices Most Grains Eggs Fish Tea Soy Milk Coconut Lima Beans Plums Brown Rice Cocoa Oats Oysters Salmon	Most Tap Water Most Spring Water River Water	Apples Almonds Tomatoes Grapefruit Corn Mushrooms Turnip Olives Peaches Bell Pepper Radish Pineapple Cherries Wild Rice Apricot Strawberries Bananas	Avocados Green Tea Lettuce Celery Peas Sweet Potatoes Egg Plant Green Beans Beets Blueberries Pears Grapes Kiwi Melons Tangerines Figs Dates Mangoes Papayas	pHresh greens® Spinach Broccoli Artichoke Brussel Sprouts Cabbage Cauliflower Carrots Cucumbers Lemons Limes Seaweed Asparagus Kale Radish Collard Greens Onion ***Raw / Uncooked**

Note that a food's acid or alkaline-forming tendency in the body has nothing to do with the actual pH of the food itself. For example, lemons are very acidic, however the end-products they produce after digestion and assimilation are very alkaline so lemons are alkaline-forming in the body. Likewise, meat will test alkaline before digestion but it leaves very acidic residue in the body so, like nearly all animal products, meat is very acid-forming.

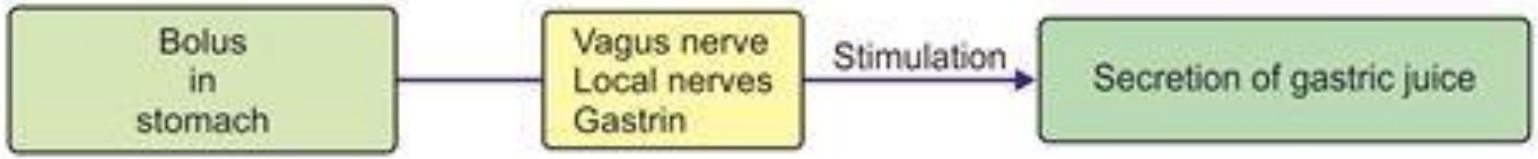
***Eat less processed and refined foods and more raw and uncooked greens and fruits.**


CEPHALIC PHASE
Sight / thought of food
Cerebral cortex
Conditioned reflex
Stimulation of taste / smell receptors
Hypothalamus and medulla oblongata
Vagus nerve
Stimulates stomach secretory activity
Loss of appetite; Depression
Cerebral cortex
Lack of stimulation to parasympathetic center
Inhibits stomach secretory activity
GASTRIC PHASE
Stomach distension activates stretch receptors
Vagovagal reflexes
Medulla
Vagus nerve
Local reflexes
Stimulates stomach secretory activity
Food chemicals and rising pH activate chemoreceptors
G cells secrete gastrin into blood
Excessive acidity (<pH 2) in stomach
Gastrin secretion declines
Inhibits stomach secretory activity
Emotional distress
Sympathetic nervous system activation
Overides parasympathetic controls
INTESTINAL PHASE
Presence of low pH and partially digested foods in duodenum as stomach empties
Intestinal gastrin release into blood
Stimulates stomach secretory activity
Distention of duodenum; presence of fatty, acidic. hypertonic chyme, and/or irritants
Local reflexes
Pyloric sphincter
Entero-gastric reflex
Vagal nuclei in medulla
Inhibits stomach secretory activity
Distention; presence of fatty, acidic, partially digested food
Release of intestinal hormones
(secretin, gastric inhibitory peptide, cholecystokinin, vasoactive intestinal peptide)

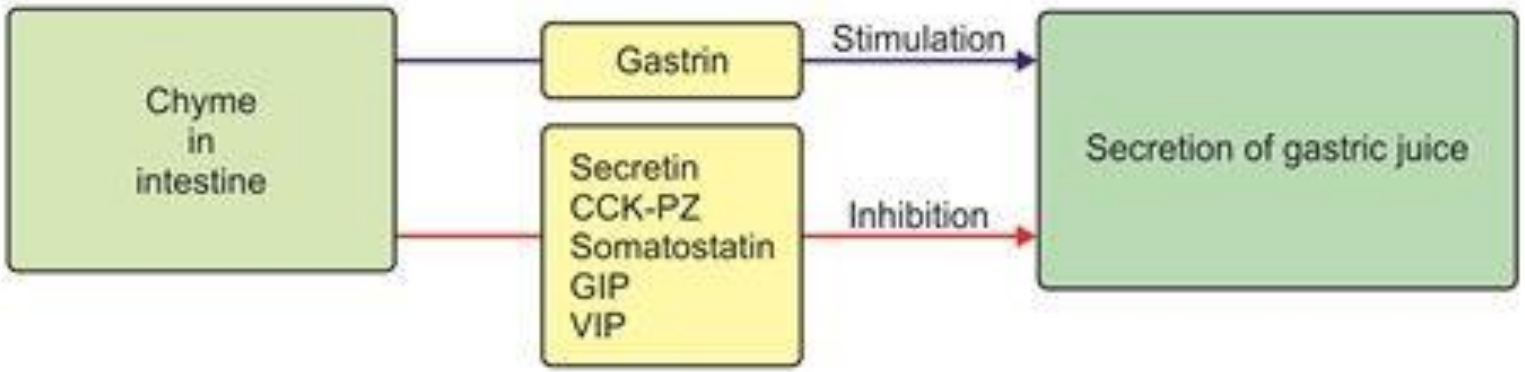
Cephalic phase: Only nervous



Gastric phase: Nervous and hormonal



Intestinal phase: Mostly hormonal



Trigger Foods

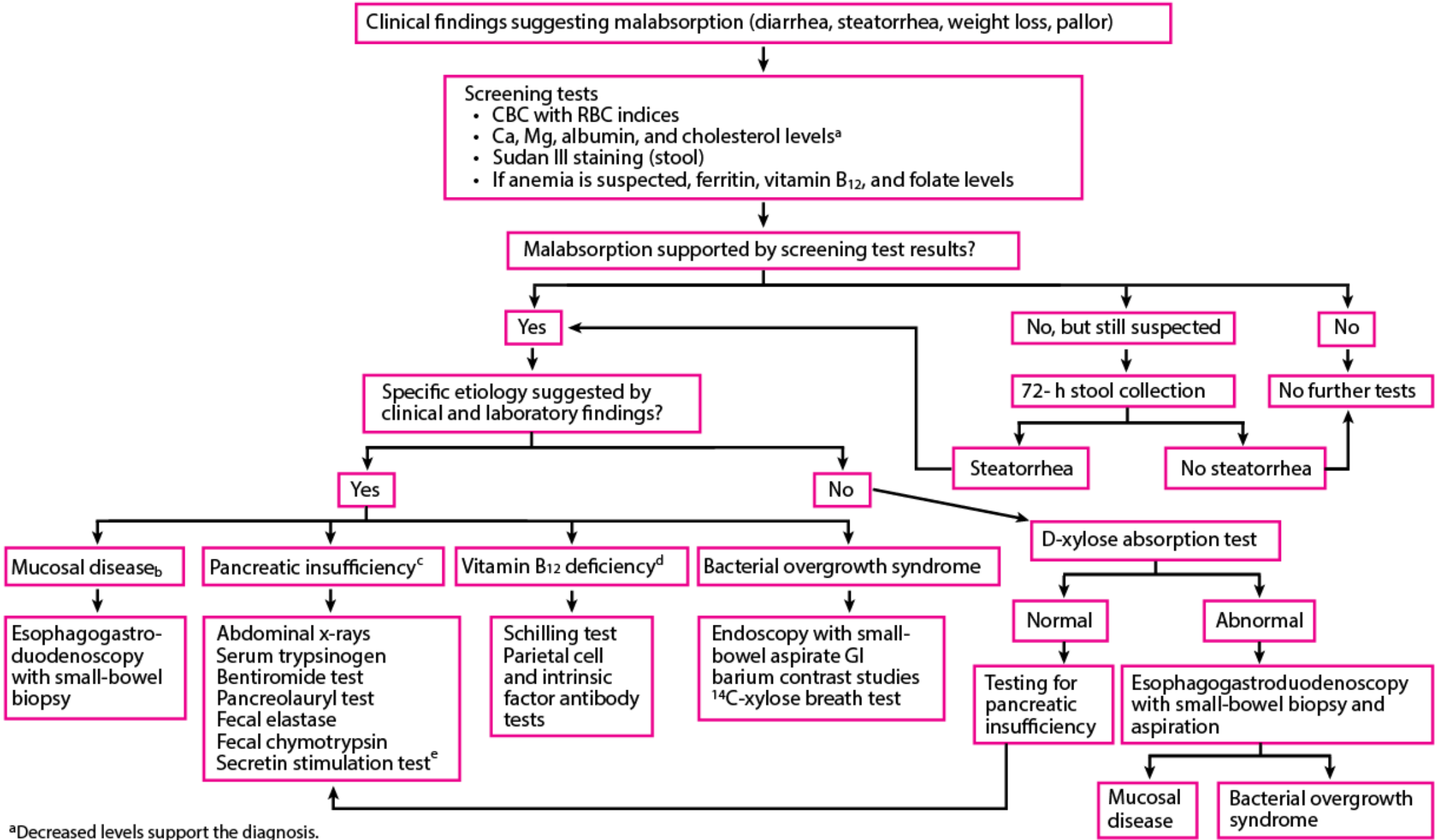
Coffee and caffeinated beverages can relax the Lower Esophageal Sphincter.

Peppermint, garlic and onions can relax the Lower Esophageal Sphincter (LES) causing acid reflux.

Chocolate contains chemical similar to caffeine, which can relax the Lower Esophageal Sphincter

Fizzy drinks causes bloating which can increase pressure in the Lower Esophageal Sphincter.

Fatty, spicy or fried foods can relax the LES as well as delay stomach emptying.



[a]Decreased levels support the diagnosis.
[b]Eg, celiac sprue, tropical sprue, Whipple's disease, lymphangiectasia, amyloidosis.
[c]Eg, chronic pancreatitis, pancreatic cancer, hereditary pancreatitis, cystic fibrosis.
[d]Eg, pernicious anemia, pancreatic insufficiency, bacterial overgrowth.
[e]Available at only a few centers.