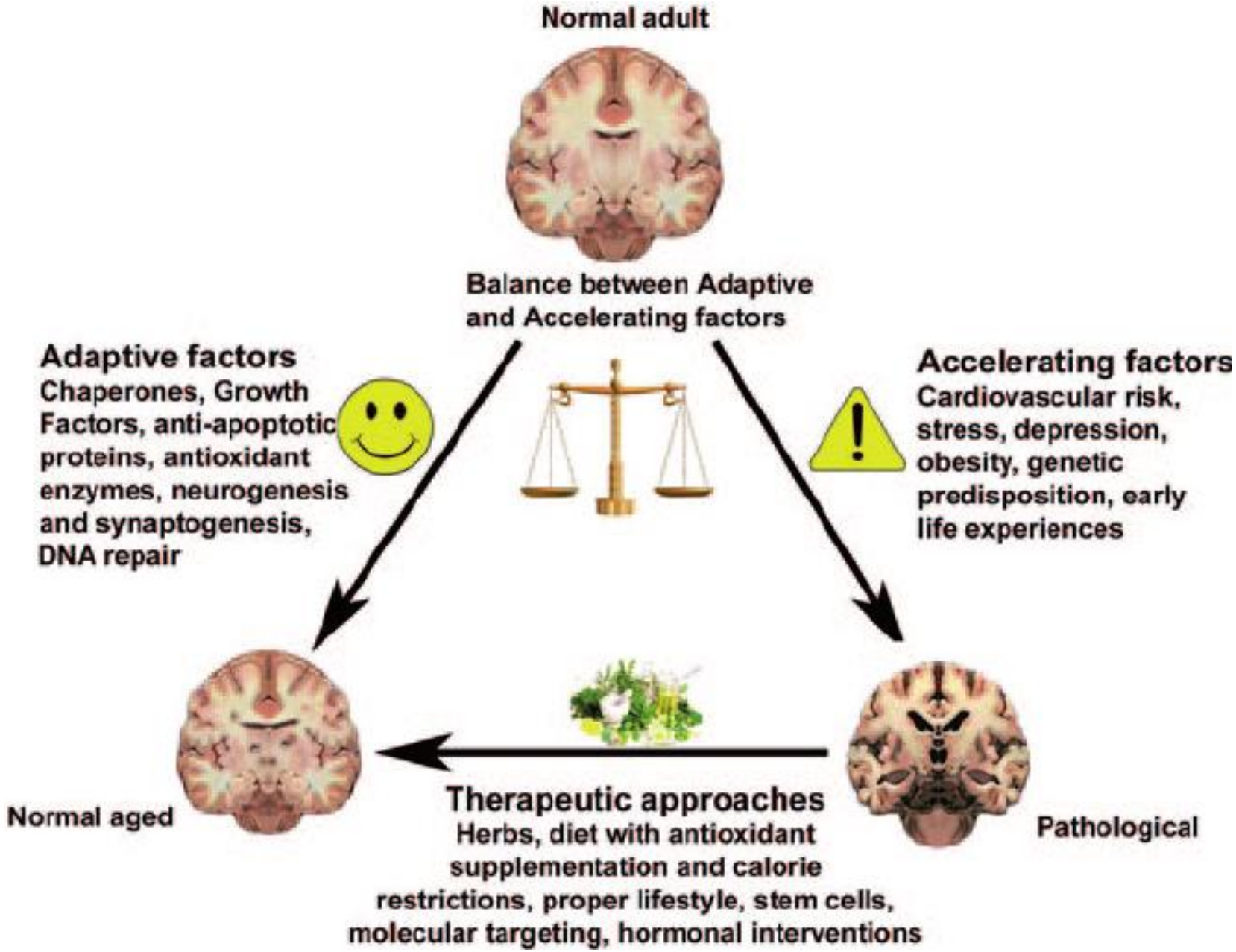
TATALAKSANA GIZI PADA KASUS NEUROPSIKIATRI

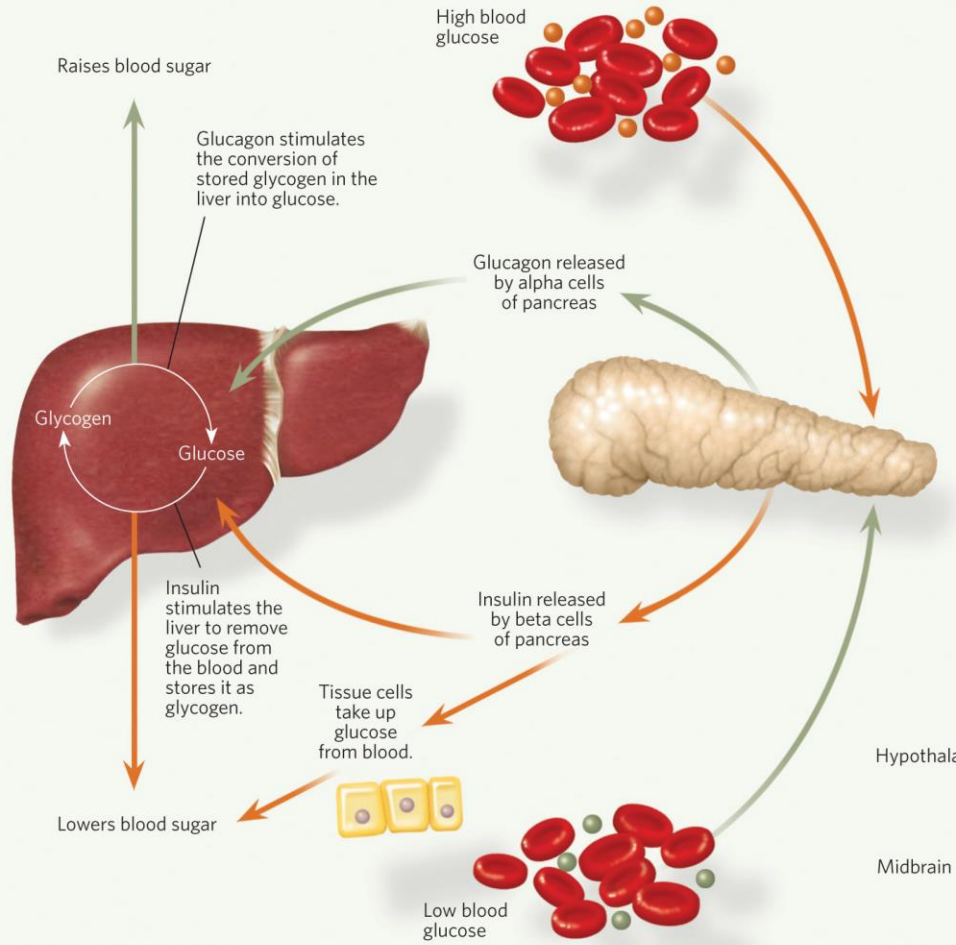
TIRTA PRAWITA SARI

PENYAKIT NEURODEGENERATIVE ADALAH SUATU PROSES INFLAMASI


Normal adult
Balance between Adaptive and Accelerating factors
Adaptive factors
Chaperones, Growth Factors, anti-apoptotic proteins, antioxidant enzymes, neurogenesis and synaptogenesis, DNA repair
Accelerating factors
Cardiovascular risk, stress, depression, obesity, genetic predisposition, early life experiences
Normal aged
Therapeutic approaches
Herbs, diet with antioxidant supplementation and calorie restrictions, proper lifestyle, stem cells, molecular targeting, hormonal interventions
Pathological

INSULIN'S ROLE IN BODY AND BRAIN

Insulin, long recognized as a primary regulator of blood glucose, is now also understood to play key roles in neuroplasticity, neuromodulation, and neurotrophism, the process of neuronal growth, stimulated by neuronal differentiation and survival.



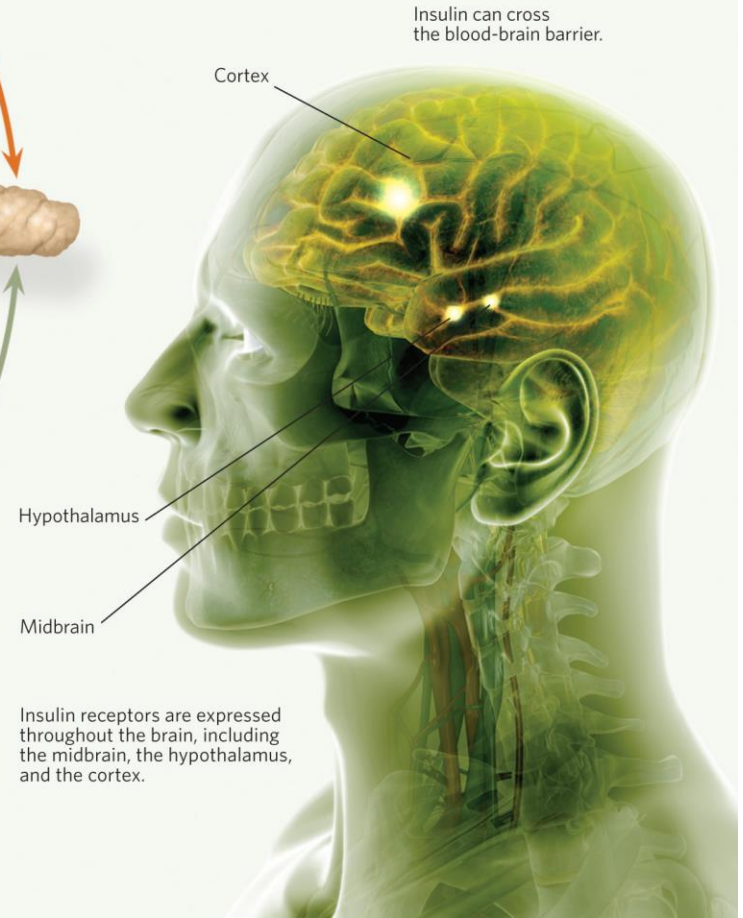
PRECISION GRAPHICS; HEAD © 3D4MEDICAL.COM/CORBIS

METABOLIC INFLUENCE

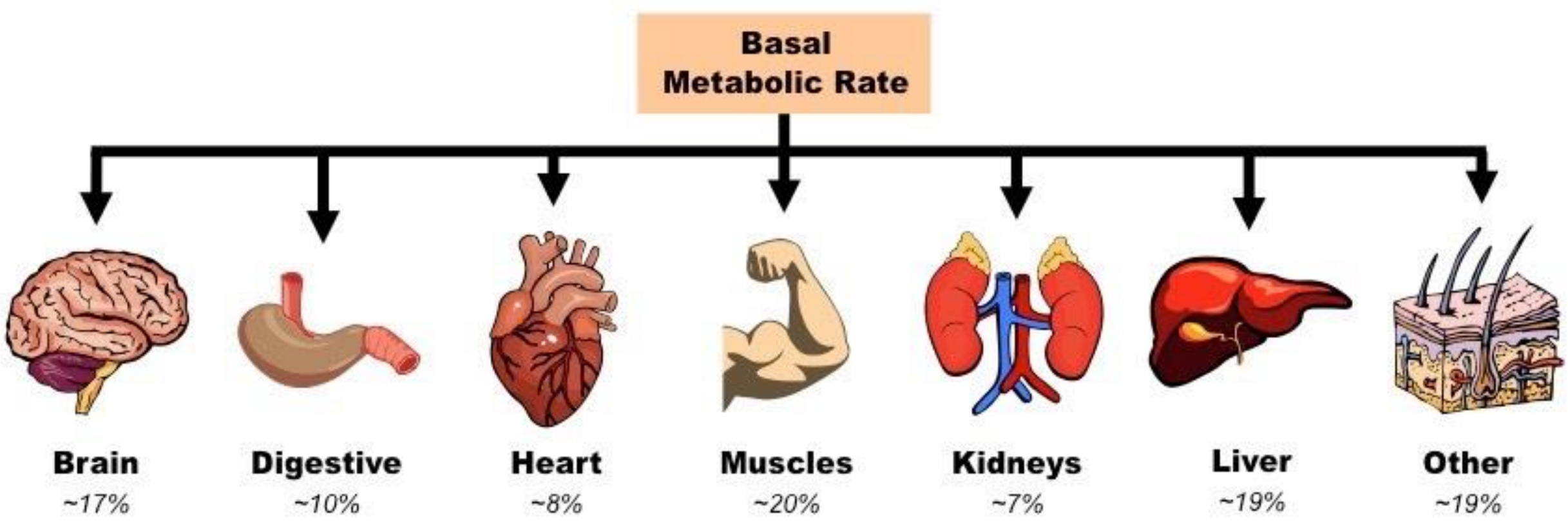
Insulin is one of the primary hormones involved in blood glucose regulation. Its dysregulation is associated with obesity and diabetes.

NEUROLOGIC INFLUENCE

Insulin activates insulin receptors and downstream signaling molecules in the brain and spinal cord, as well as insulin-sensitive glucose transporters in the peripheral insulin-sensitive tissues (liver, muscle, fat). Through these mechanisms, insulin participates in feeding behavior, reward pathways, whole body metabolism, and normal emotional and cognitive brain functions. The dysregulation of insulin-mediated signaling pathways in the brain is implicated in neurodegenerative diseases such as Alzheimer's and psychiatric disorders such as schizophrenia.

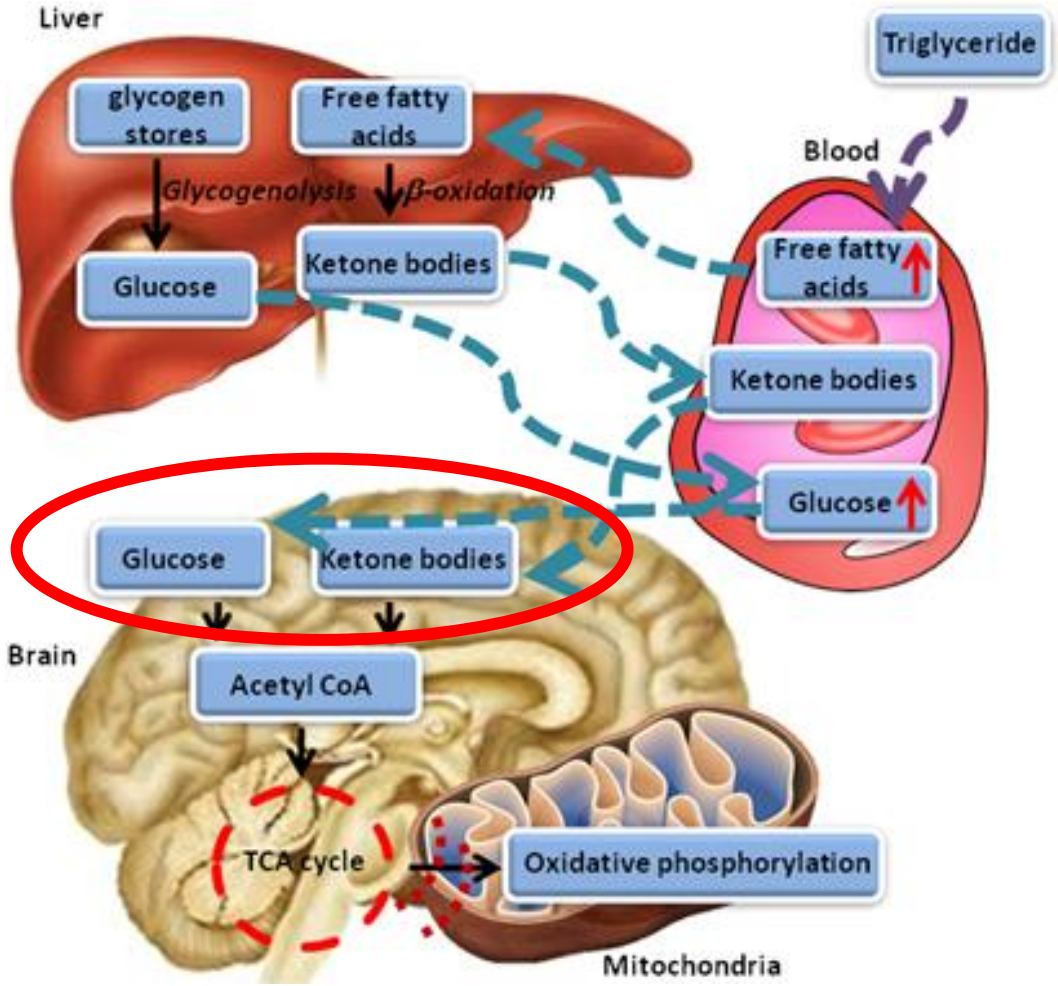


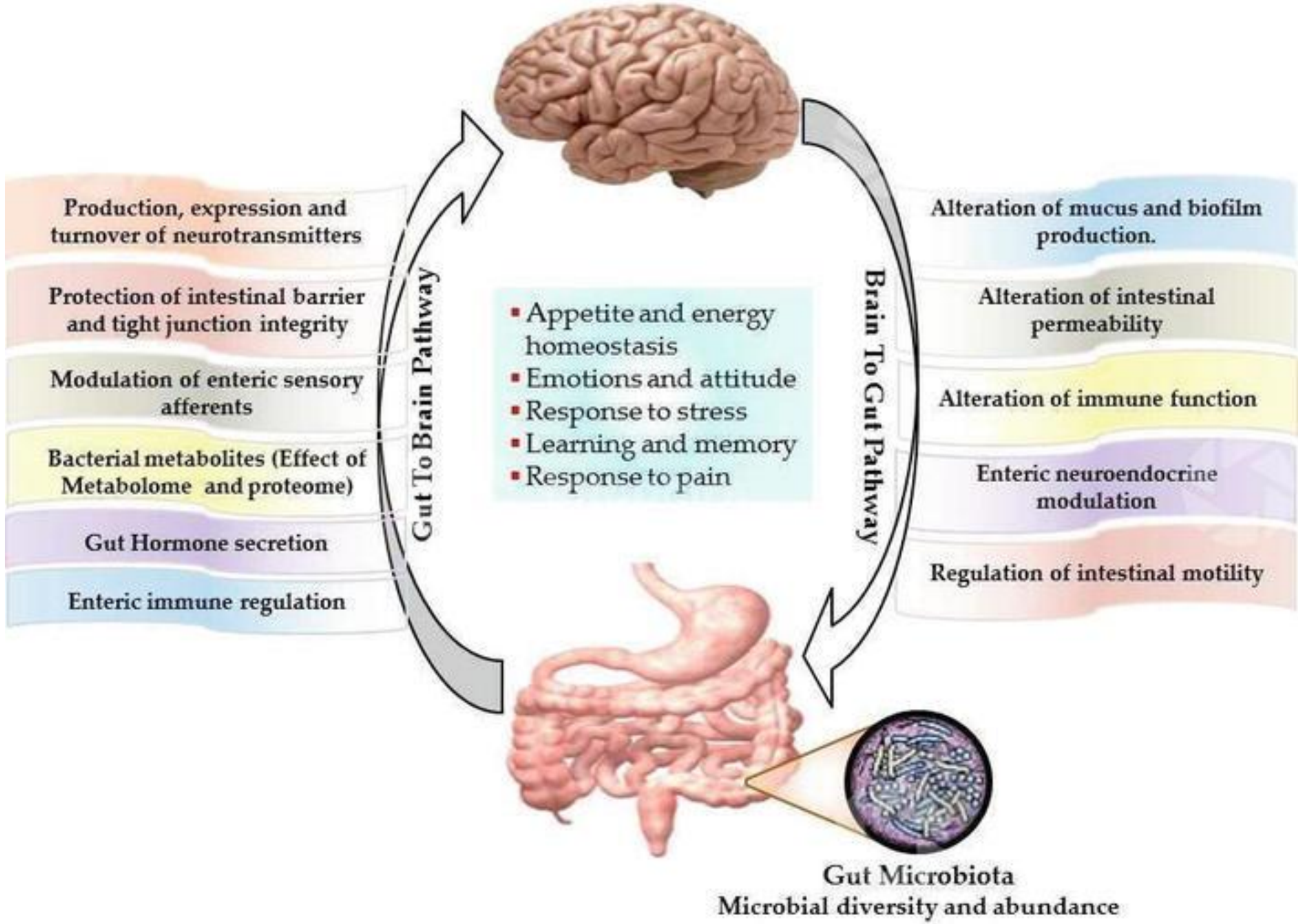
Insulin receptors are expressed throughout the brain, including the midbrain, the hypothalamus, and the cortex.


Basal
Metabolic Rate
Brain
~17%
Digestive
~10%
Heart
~8%
Muscles
~20%
Kidneys
~7%
Liver
~19%
Other
~19%

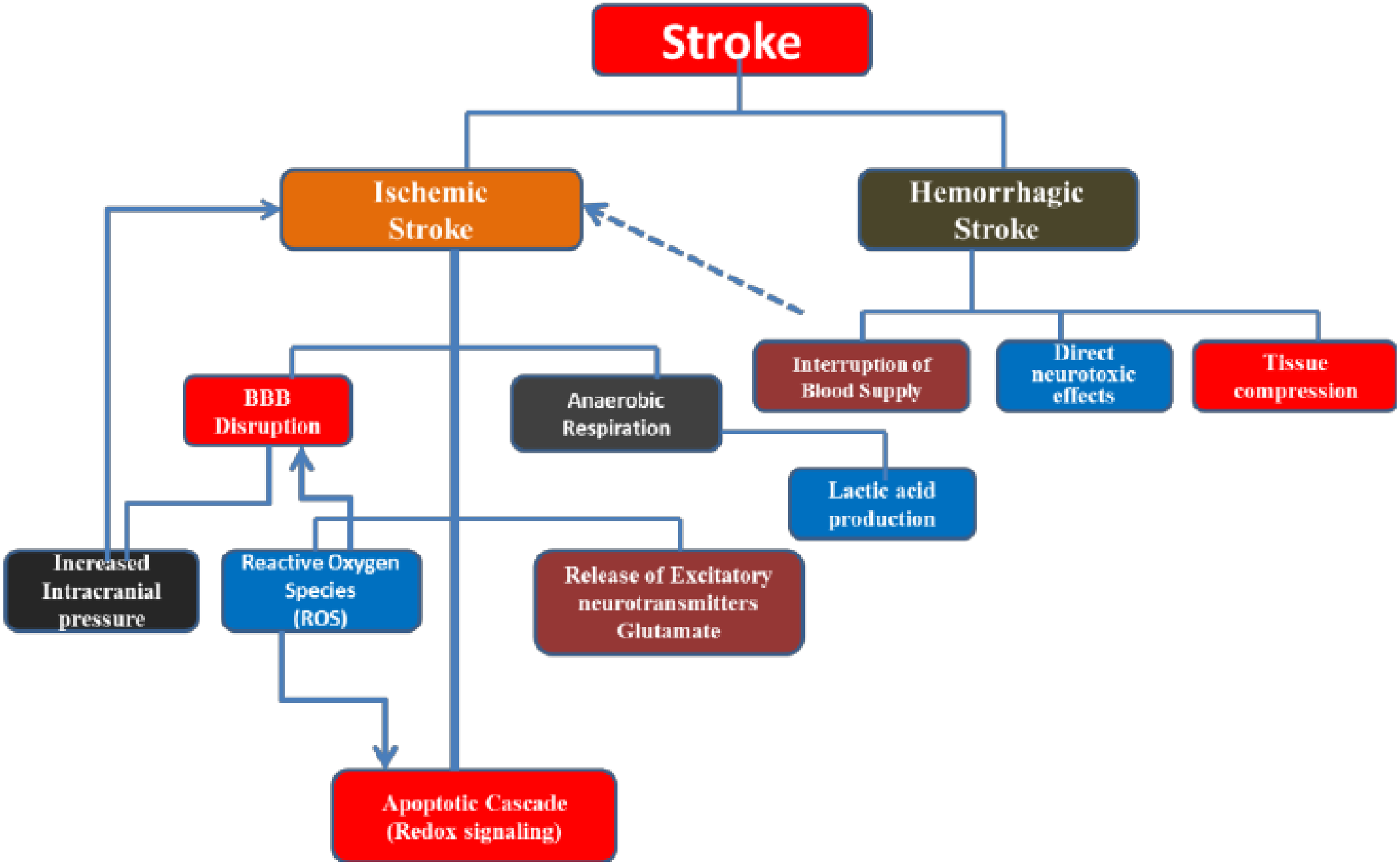
BRAIN ENERGY METABOLISM

ENERGY SOURCE

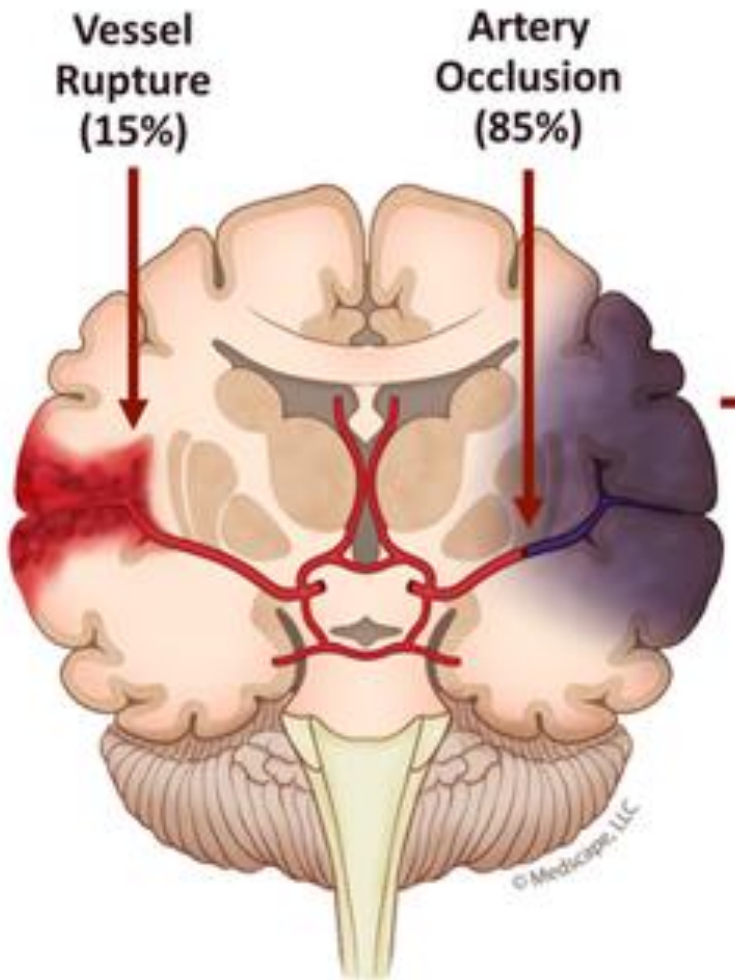



Production, expression and turnover of neurotransmitters
Protection of intestinal barrier and tight junction integrity
Modulation of enteric sensory afferents
Bacterial metabolites (Effect of Metabolome and proteome)
Gut Hormone secretion
Enteric immune regulation
Gut To Brain Pathway
Appetite and energy homeostasis
Emotions and attitude
Response to stress
Learning and memory
Response to pain
Brain To Gut Pathway
Alteration of mucus and biofilm production.
Alteration of intestinal permeability
Alteration of immune function
Enteric neuroendocrine modulation
Regulation of intestinal motility
Gut Microbiota
Microbial diversity and abundance

STROKE, TIPE APAPUN MERUPAKAN PENYAKIT YANG MENIMBULKAN RESIKO MALNUTRISI YANG TINGGI SEHINGGA MEMBUTUHKAN TATALAKSANA GIZI YANG ADEKUAT UNTUK PROSES PENYEMBUHAN PASIEN


Stroke
Ischemic Stroke
Hemorrhagic Stroke
Interruption of Blood Supply
Direct neurotoxic effects
Tissue compression
BBB Disruption
Anaerobic Respiration
Lactic acid production
Increased Intracranial pressure
Reactive Oxygen Species (ROS)
Release of Excitatory neurotransmitters Glutamate
Apoptotic Cascade (Redox signaling)

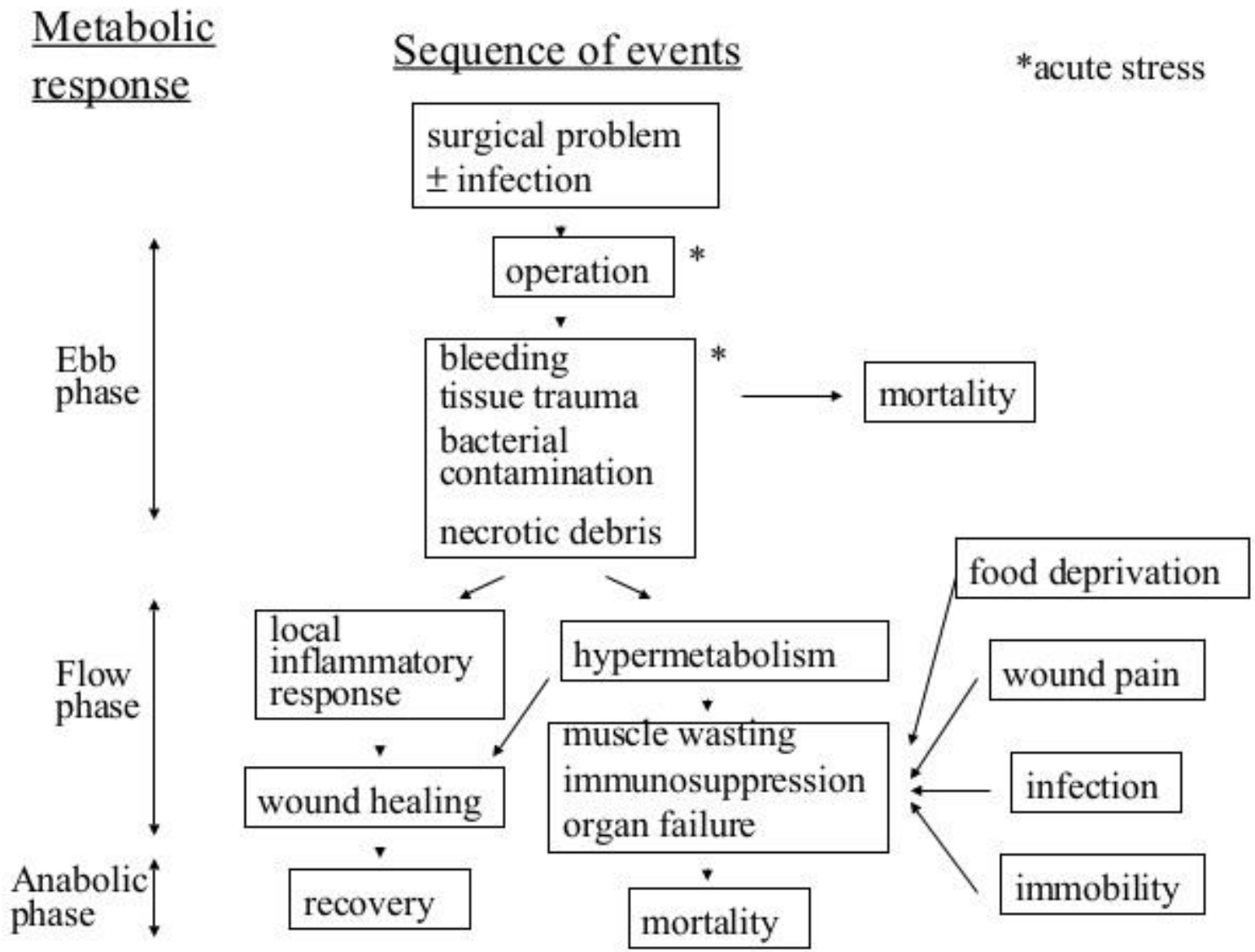
Stroke Etiologies

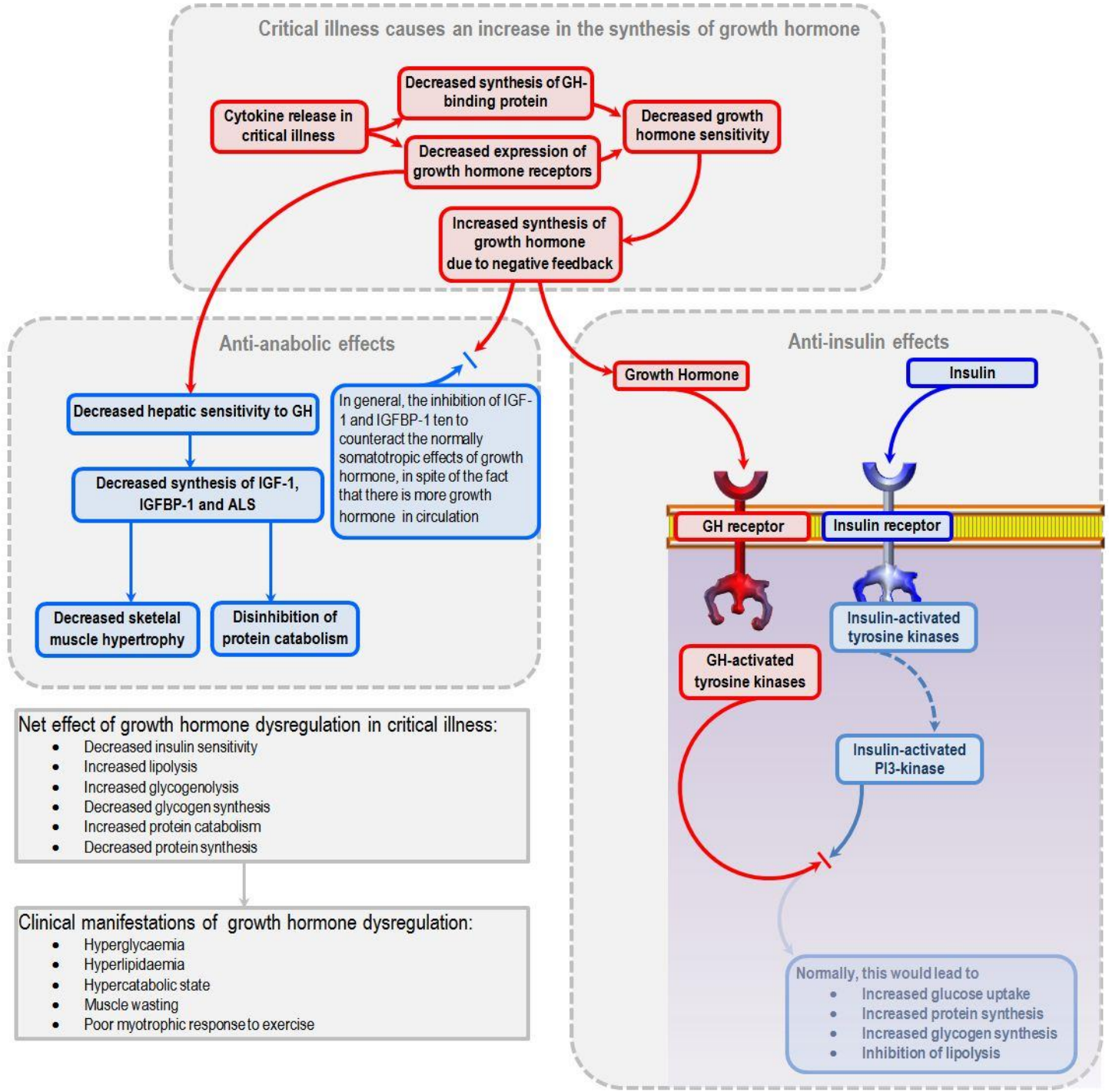


Etiology	%
Atherothrombotic Stenotic artery feeding area of infarction	25-30
Cardioembolic A thrombus or other material dislodges from the heart or aortic arch	20
Lacunar/Small Vessel Small; deep infarct	15-20
Other/Uncommon	5-10
Cryptogenic Unknown cause	25-30

Adams HP Jr, et al. *Stroke*. 1993;24:35-41; Foulkes MA, et al. *Stroke*. 1988;19:547-554.

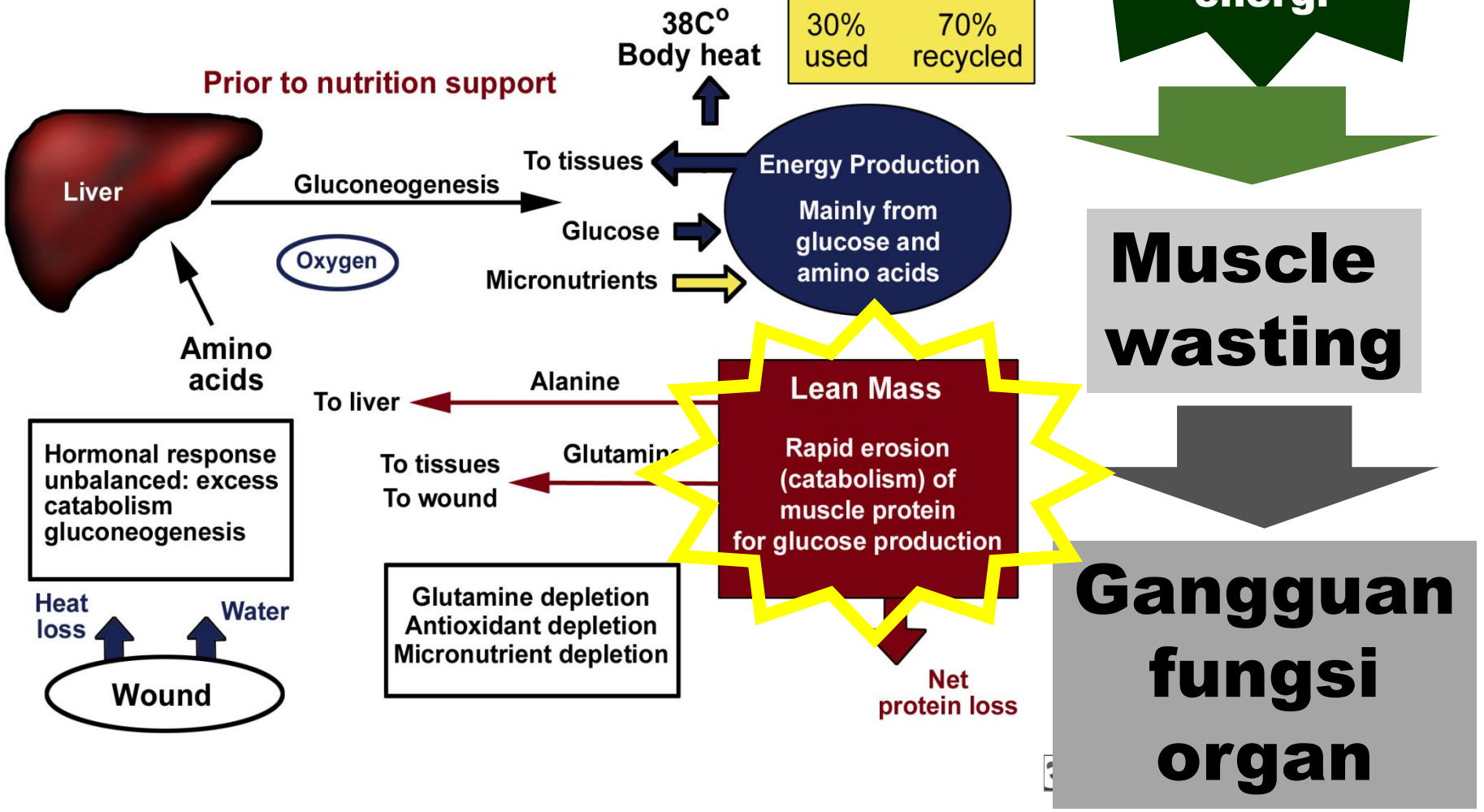
STROKE MENYEBABKAN STRESS METABOLIK YANG BESAR PADA PASIEN YANG BERUJUNG PADA KATABOLISME, YANG BILA TIDAK DIATASI AKAN MENYEBABKAN MALNUTRISI


Metabolic response
Sequence of events
*acute stress
surgical problem ± infection
operation
*
bleeding
tissue trauma
bacterial contamination
necrotic debris
*
mortality
Ebb phase
Flow phase
Anabolic phase
local inflammatory response
hypermetabolism
food deprivation
wound pain
infection
immobility
wound healing
muscle wasting
immunosuppression
organ failure
recovery
mortality


Critical illness causes an increase in the synthesis of growth hormone
Cytokine release in critical illness
Decreased synthesis of GH-binding protein
Decreased expression of growth hormone receptors
Decreased growth hormone sensitivity
Increased synthesis of growth hormone due to negative feedback
Anti-anabolic effects
Decreased hepatic sensitivity to GH
Decreased synthesis of IGF-1, IGFBP-1 and ALS
Decreased sketelal muscle hypertrophy
Disinhibition of protein catabolism
In general, the inhibition of IGF-1 and IGFBP-1 ten to counteract the normally somatotropic effects of growth hormone, in spite of the fact that there is more growth hormone in circulation
Anti-insulin effects
Growth Hormone
Insulin
GH receptor
Insulin receptor
Insulin-activated tyrosine kinases
GH-activated tyrosine kinases
Insulin-activated PI3-kinase
Normally, this would lead to
Increased glucose uptake
Increased protein synthesis
Increased glycogen synthesis
Inhibition of lipolysis
Net effect of growth hormone dysregulation in critical illness:
Decreased insulin sensitivity
Increased lipolysis
Increased glycogenolysis
Decreased glycogen synthesis
Increased protein catabolism
Decreased protein synthesis
Clinical manifestations of growth hormone dysregulation:
Hyperglycaemia
Hyperlipidaemia
Hypercatabolic state
Muscle wasting
Poor myotrophic response to exercise

Catabolic Insult-Induced Protein-Energy Malnutrition (Protein and Energy Production Abnormal)

- No adaptive responses activated
- Increase metabolic rate 35-40 kcal/kg/d
- Increase glucose production in excess of need
- Increase use of protein for fuel (glucose)
- Inefficient use of fat for energy

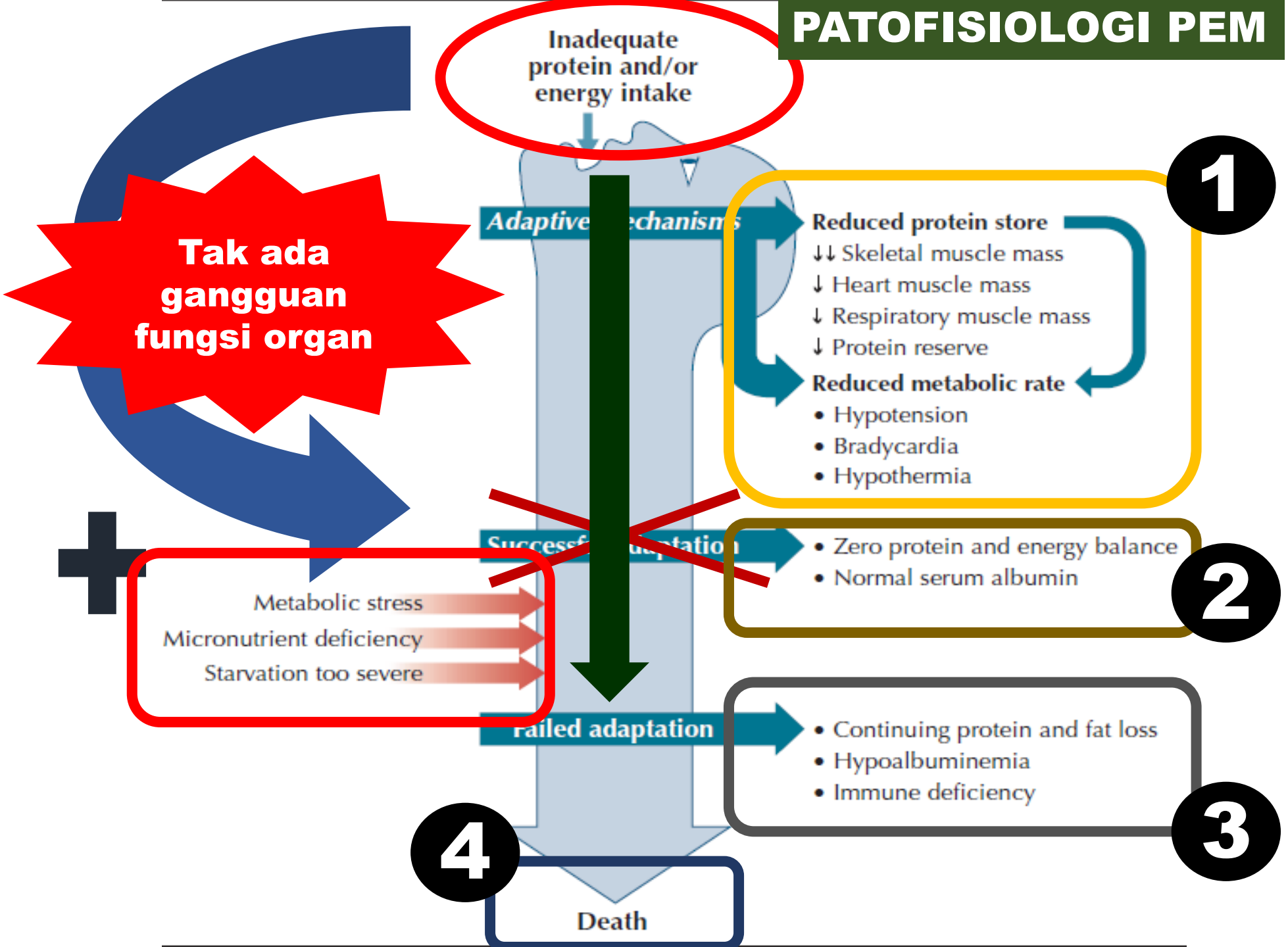


Meningkat katabolisme protein (otot) utk sumber energi

Muscle wasting

Gangguan fungsi organ

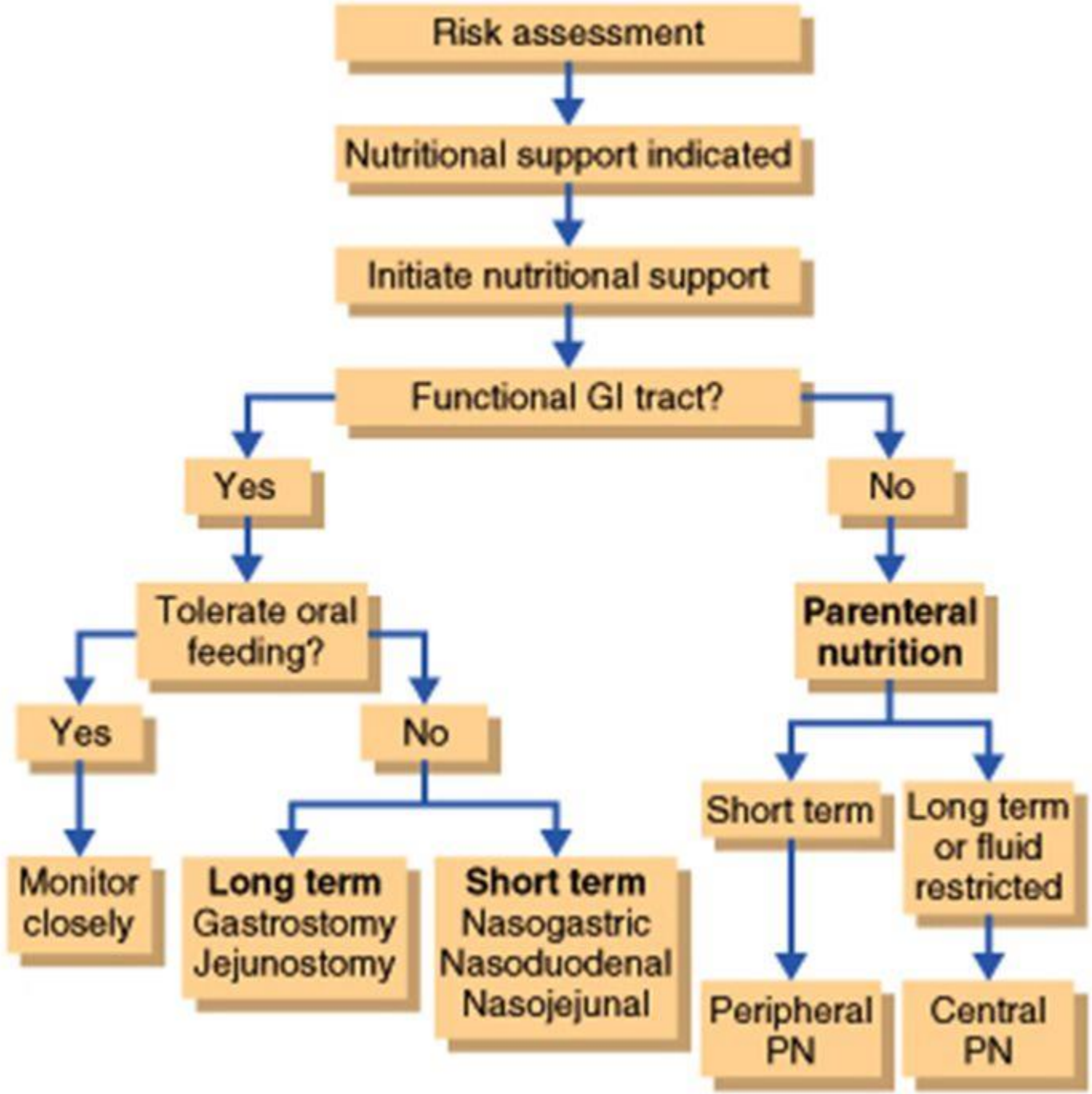
PATOFISIOLOGI PEM

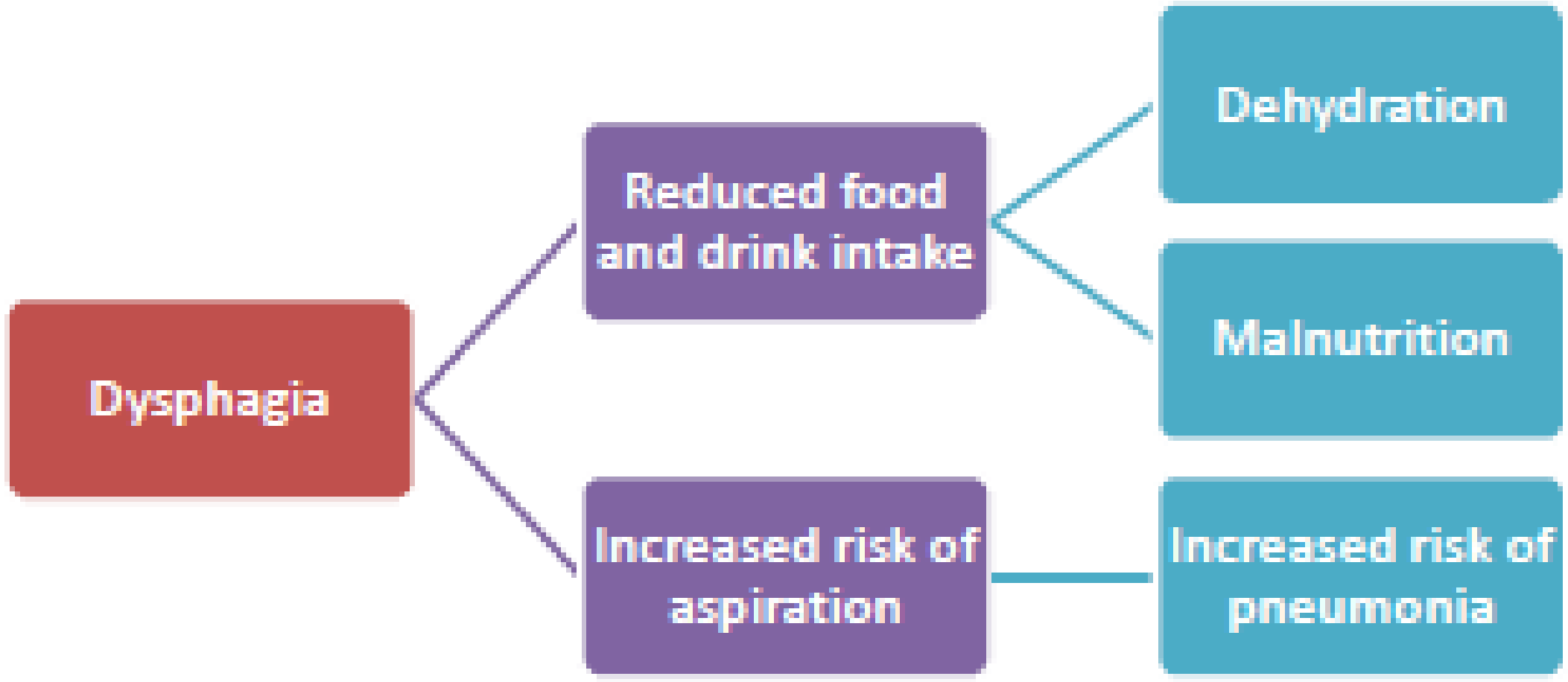


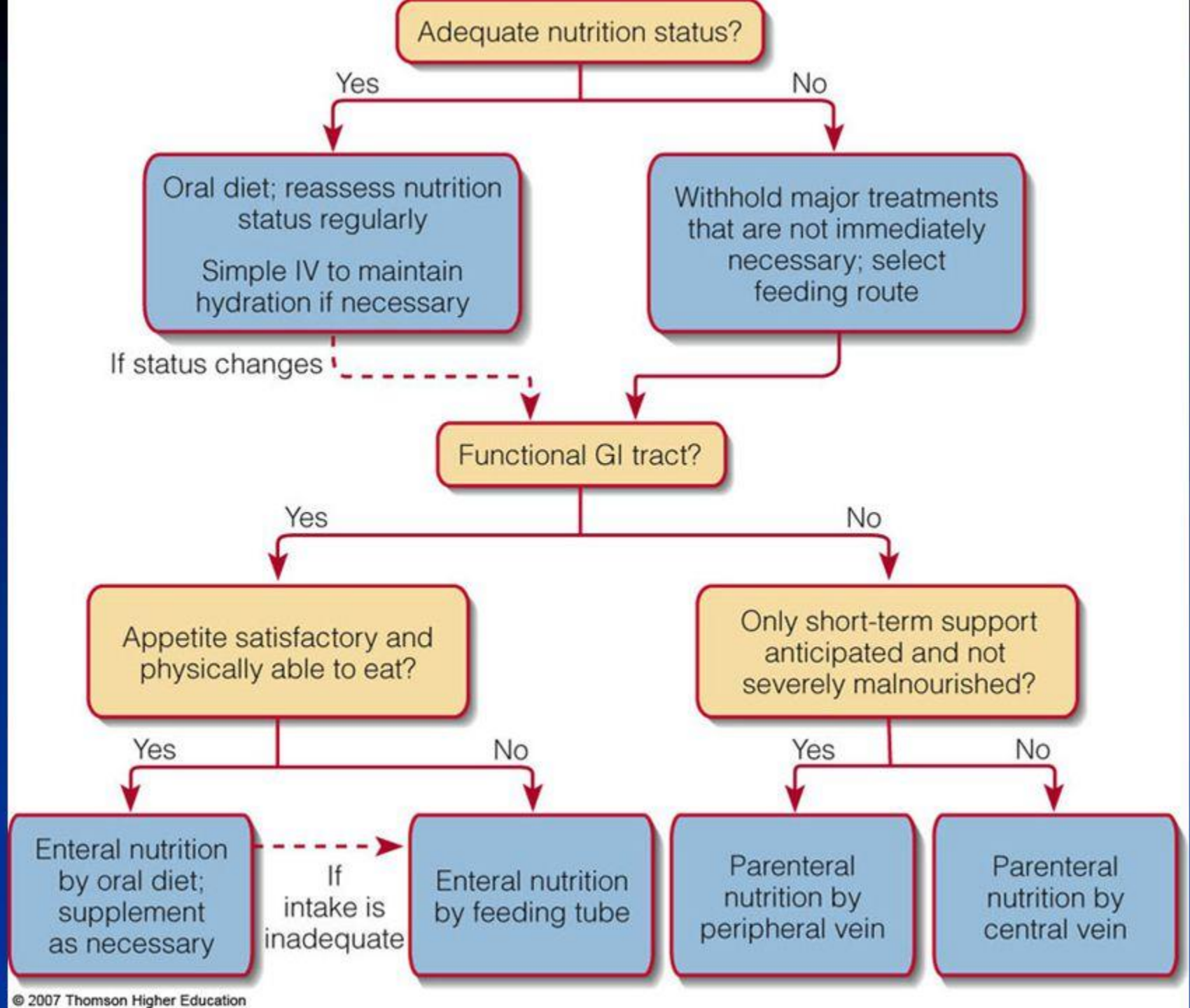
Metabolic response during stress

Organ	Response
liver	↑ glucose production , AA uptake , acute-phase protein synthesis trace metal sequestration
Central nervous system	Anorexia , fever
Circulation	↑ Glucose , TG ,urea ↓ AA, iron, zinc
Skeletal muscle	↑ AA efflux (especially glutamine) leading to loss of muscle mass
Intestine	↓ AA uptake from both luminal and circulating sources , leading to mucosal atrophy
Endocrine	↑ ACTH, cortisol , GH, epinephrine , norepinephrine , glucagon , insulin

KESULITAN TATALAKSANA GIZI ADALAH PADA SITUASI KRITIS DAN HAMBATAN PADA JALUR PEMBERIAN NUTRISI, SEHINGGA DIPERLUKAN ASSESSMENT YANG TELITI UNTUK MENENTUKAN TATALAKSANA


Risk assessment
Nutritional support indicated
Initiate nutritional support
Functional GI tract?
Yes
No
Tolerate oral feeding?
Yes
No
Monitor closely
Long term
Gastrostomy
Jejunostomy
Short term
Nasogastric
Nasoduodenal
Nasojejunal
Parenteral nutrition
Short term
Long term or fluid restricted
Peripheral PN
Central PN

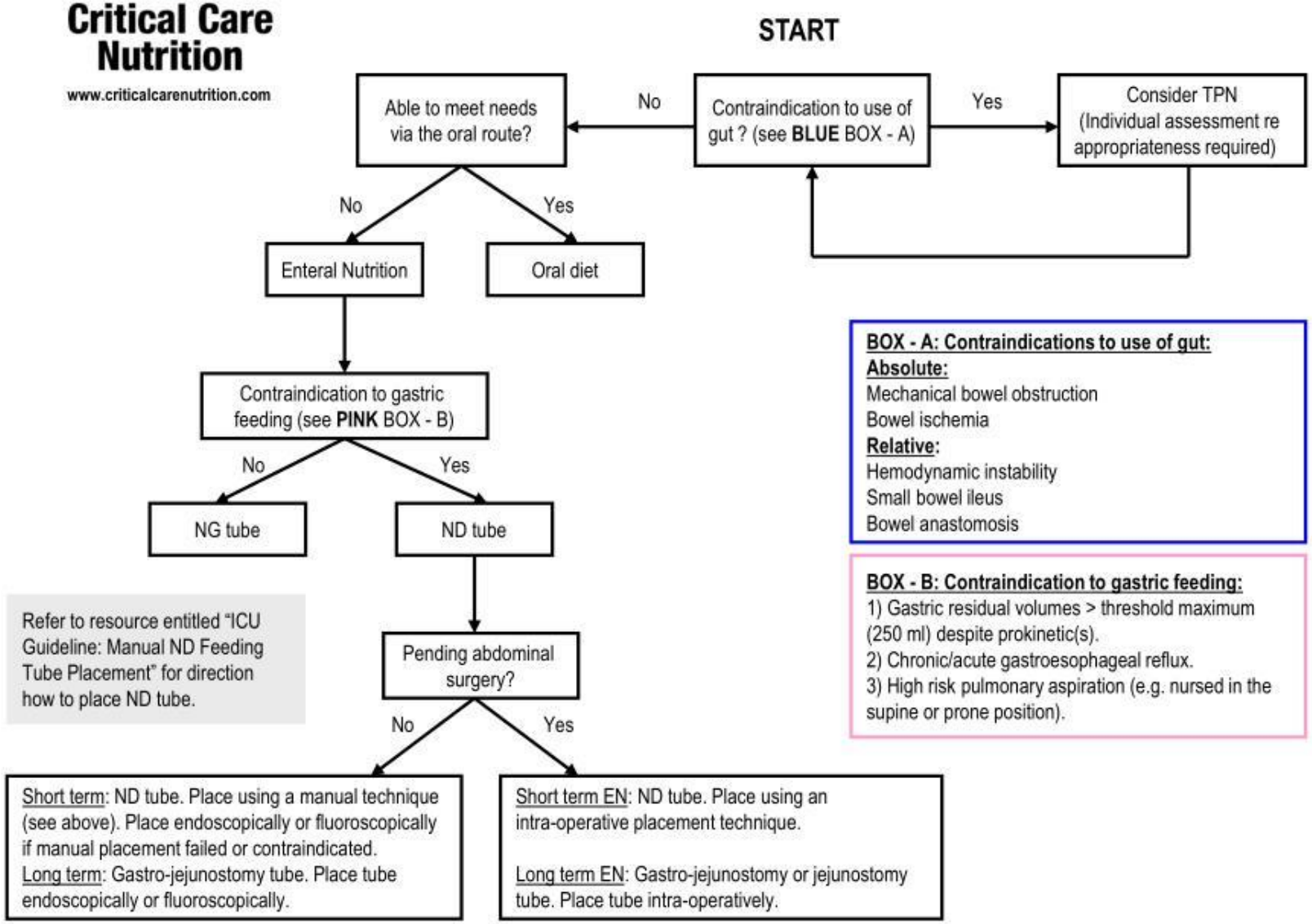

Dysphagia
Reduced food and drink intake
Increased risk of aspiration
Dehydration
Malnutrition
Increased risk of pneumonia



Critical Care Nutrition

www.criticalcarenutrition.com

ICU GUIDELINE: ROUTES OF NUTRITION SUPPORT



BOX - A: Contraindications to use of gut:

Absolute:
Mechanical bowel obstruction
Bowel ischemia

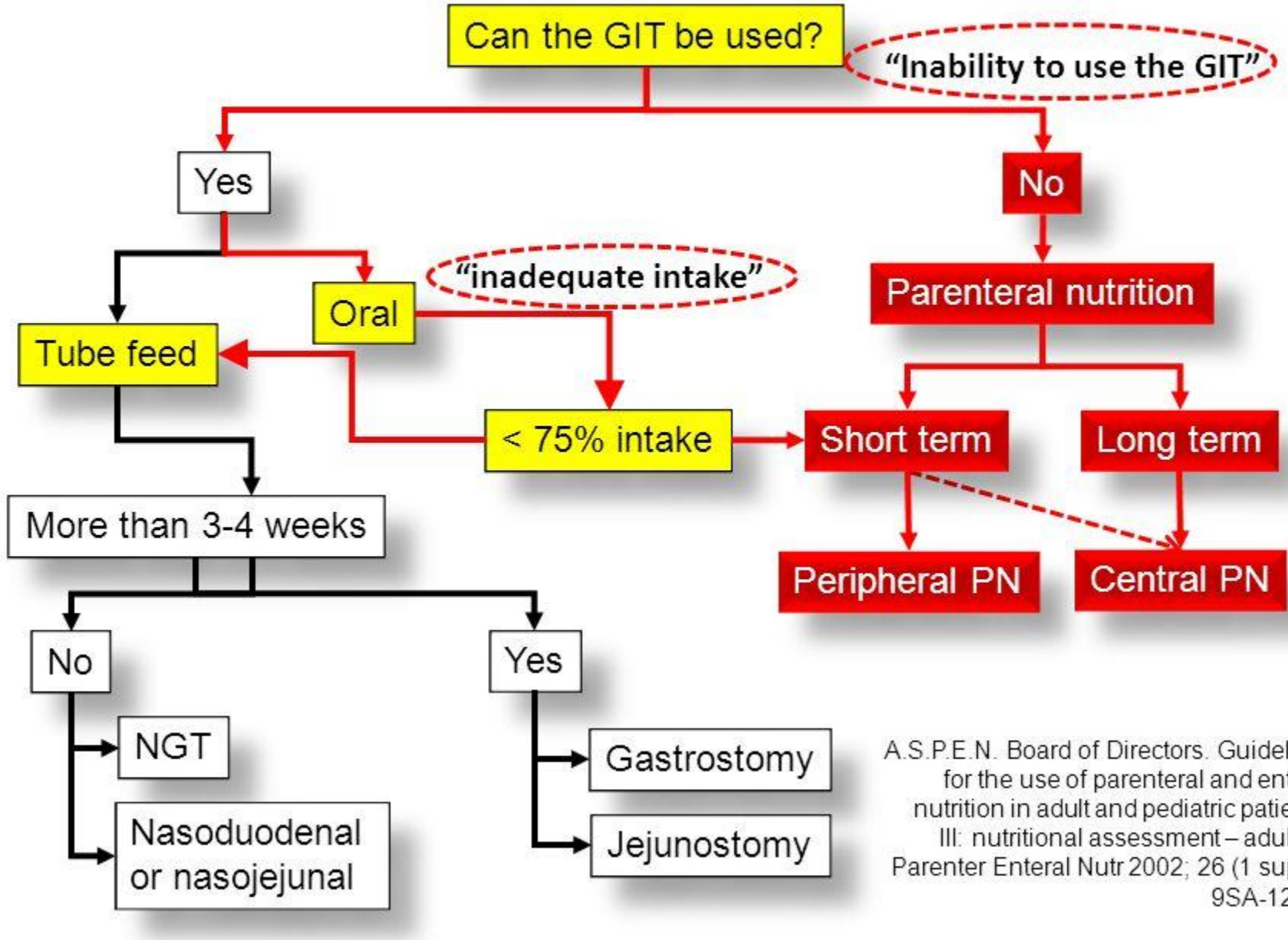
Relative:
Hemodynamic instability
Small bowel ileus
Bowel anastomosis

BOX - B: Contraindication to gastric feeding:

1) Gastric residual volumes > threshold maximum (250 ml) despite prokinetic(s).
2) Chronic/acute gastroesophageal reflux.
3) High risk pulmonary aspiration (e.g. nursed in the supine or prone position).

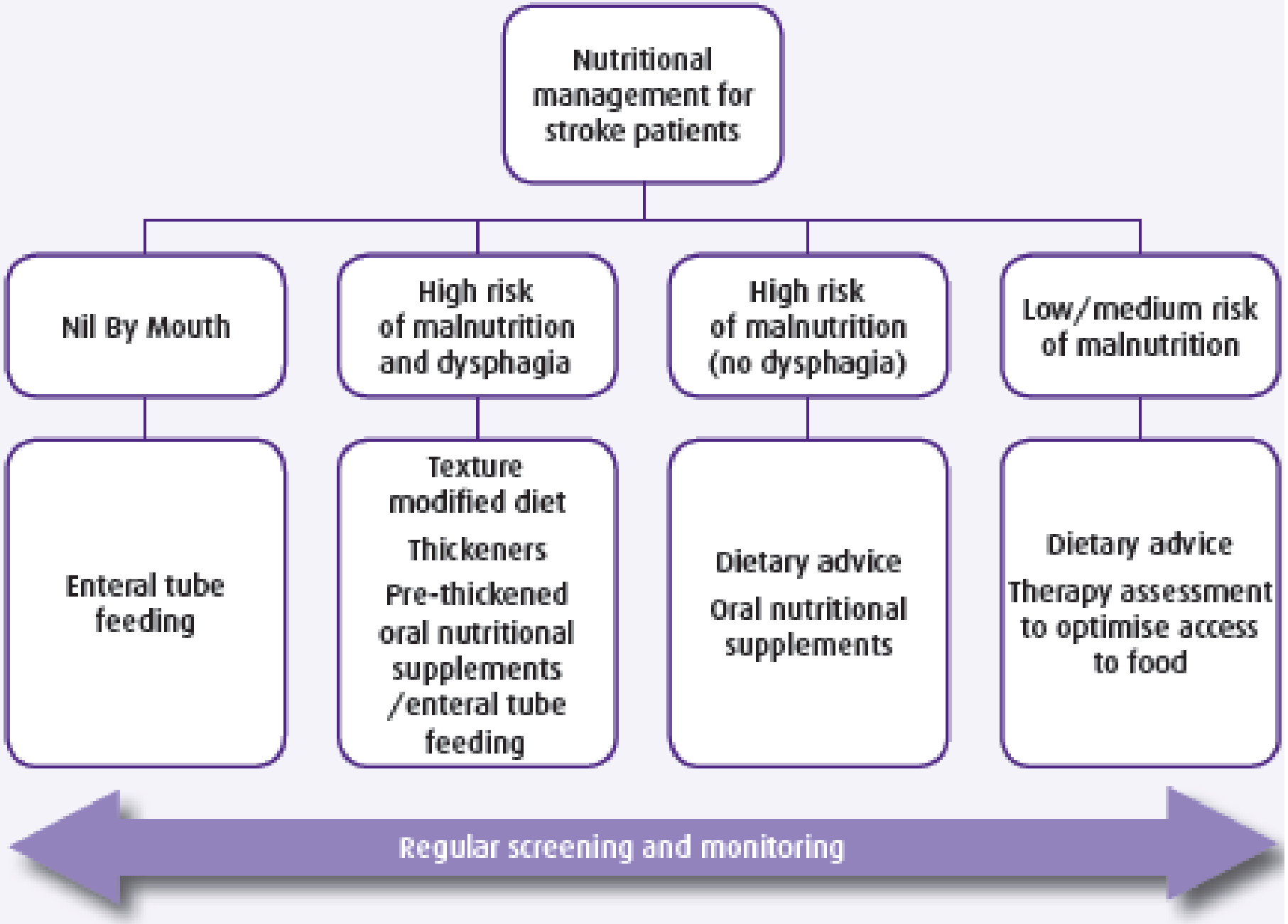
Developed by: J. Greenwood, RD. Critical Care Program – Vancouver Coastal Health Authority (Update 7/4/2010). Used with permission.

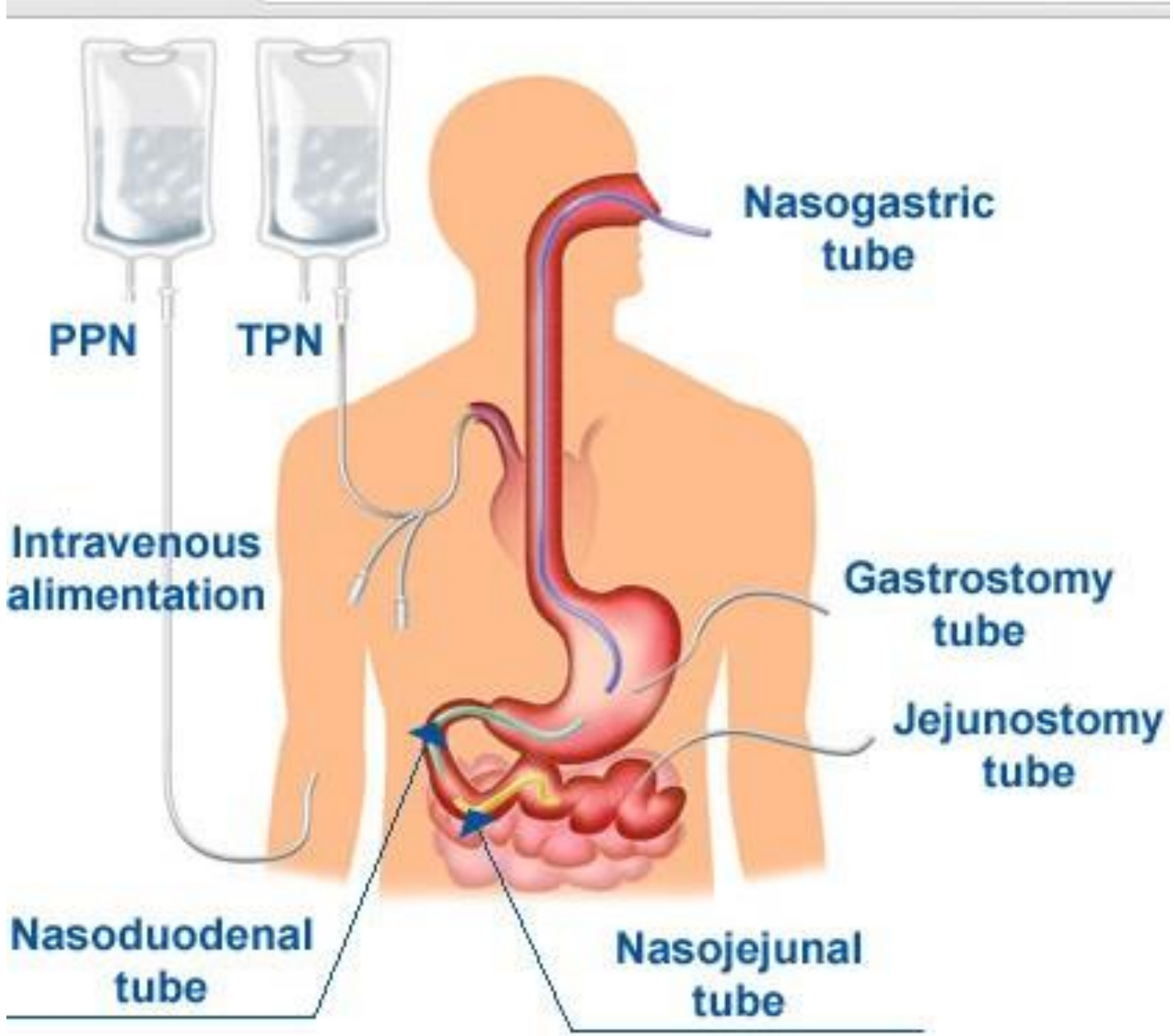
Feeding Pathways



A.S.P.E.N. Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients, III: nutritional assessment – adults. J Parenter Enteral Nutr 2002; 26 (1 suppl): 9SA-12SA.

An example of a nutritional management pathway for stroke patients:




PPN
TPN
Nasogastric tube
Intravenous alimentation
Gastrostomy tube
Jejunostomy tube
Nasoduodenal tube
Nasojejunal tube

Comparing enteral feeding tubes

This table lists types of enteral feeding tubes along with their features.

Tube type	Features
	PREPYLORIC
Nasogastric tube	• Can be placed at bedside by qualified nurse • With weighted tube (Dobhoff), fluoroscopic or radiologic confirmation of placement required before stylet removal • For short-term use (4-6 weeks); longer use poses risk of nasal mucosal damage or sinusitis
Gastrostomy tube	• Inserted surgically • Terminates in stomach • Poses risk of implantation in stomach wall • Allows administration of crushed medications
Percutaneous endoscopic gastrostomy tube	• Inserted endoscopically • Minimally invasive • Allows administration of crushed medications

	Postpyloric
Nasojejunal tube	• Terminates in jejunum • Commonly placed in radiology lab under fluoroscopic guidance; can be placed at bedside with radiographic confirmation • For short-term use (4-6 weeks); poses risk of nasal mucosal damage or sinusitis with longer use
Gastric-jejunal tube	• Terminates in small intestine • Can be used in patients requiring both stomach drainage and intestinal feeding at same time • Poses risk of jejunal extension becoming clogged from inappropriate medication administration or from attempt to rotate tube (as with G tube), causing it to curl back into stomach or protrude out through skin
Percutaneous endoscopic jejunal tube	• Terminates in small intestine • Preferred for patients who need single tube for feeding into small bowel • Required for gastrectomy or esophagectomy patients with gastric pull-up

Selection of Enteral Formula

TYPES OF FORMULA	
Standard Formulas	•For people who can digest and absorb nutrients. •They contain intact protiens or protein Isolates. Carbohydrate sources are modified starch, Glucose polymer etc.
Elemental Formulas	•For patients with compromised digestive and absorptive functions. •Contain protein s or CHO that have been broken partially or fully broken down into fragments for easy digestion. •The formulas are low in fat and may contain MCT.
Specialized formulas	•Also called disease specific formulas are designed to meet the specific nutrient needs of patients with particular Illness.
Modular Formulas	Created from individual macronutrient preparations for patients who require specific nutrient combinations to treat their illness.

Clinical Enteral Feeding Complications

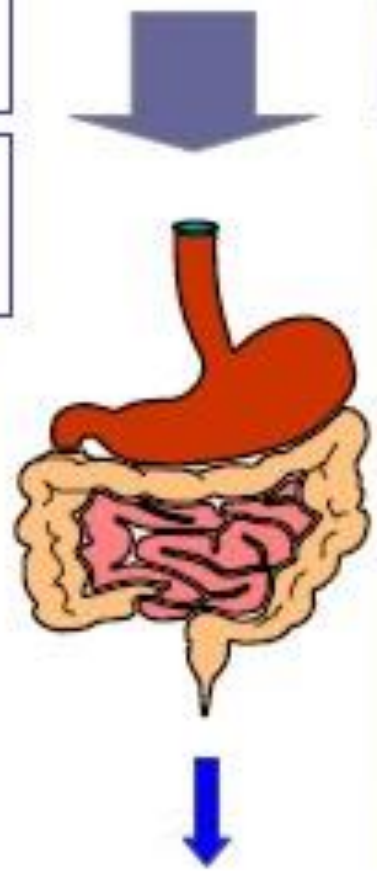
Gastrointestinal
Diarrhea, nausea, vomiting, bloating, abdominal distension

Technical
tube and/or stoma placement and maintenance

Metabolic
fluid, glucose and electrolyte imbalance

Infective
gastroenteritis, septicemia

Psychological
oral aversion, altered body self-image



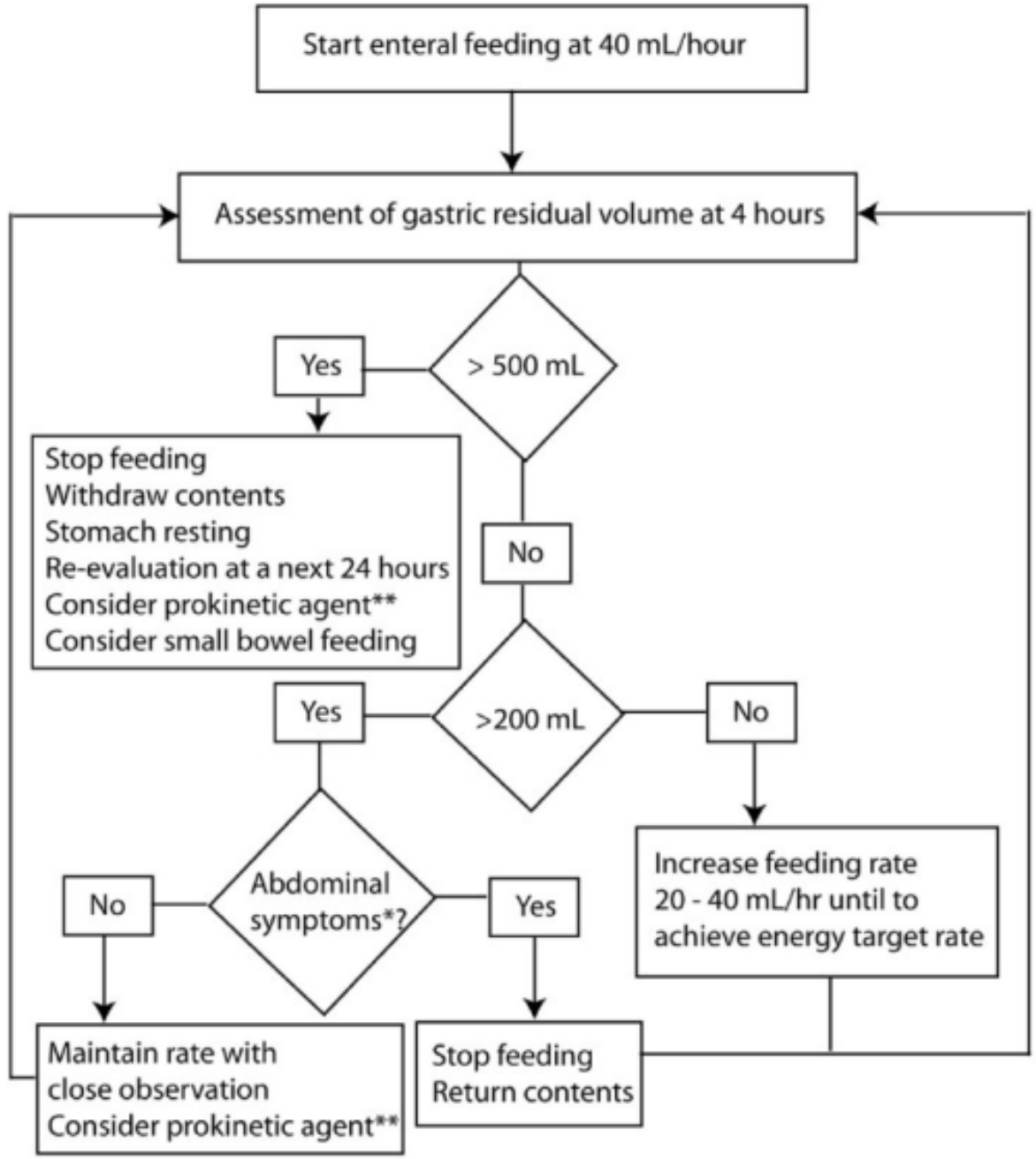
Formula selection & feeding techniques (modes)

Delivery site & delivery route
stomach vs intestine
tubes
gastro/jejunostomies

Functional & Morphologic state (Disease)
Requirements
Digestion
Absorption
Specific metabolic demands

Types of parenteral nutrition

Central	Peripheral
• Amino acids (> 5%) • Dextrose (> 20%) • Lipids • Includes vitamins, minerals, and trace elements • Carrier of pharmaconutrients like glutamine or omega-3-fatty acids • Osmolality (> 700 mOsm/kg H_2O) • Volume restriction	• Total kcal limited by concentration and ratio to volume being administered (usually delivers between 1000 to 1500 kcal/day) • The current formulations can now deliver the daily requirements of macro and micronutrients • Osmolality < 700 mOsm/kg • No volume restriction



*Abdominal symptoms including abdominal tenderness, distention, nausea, vomiting, or abdominal discomfort
**Prokinetic agent was prohibited during the study period

Prokinetics drugs

Drugs that promote gastrointestinal motility without purgation

Muscrinic agonist	• Bethanechol • Neostigmine
Peripheral cholinergic stimulants	• Cisapride (prepulsid)
Dopamine antagonist	• Metoclopramide • Domperidone

Prokinetic agent	Dosage
Metoclopramide	10-20 mg IV, 4-6 hourly (5 mg in case of renal failure)
Erythromycin	200 mg or 70 mg IV, 12-hourly
Naloxone	8 mg, 6-hourly
Tegaserod	6 mg, 12-hourly
Neostigmine	0.4-0.8 mg/h infusion
Insufficient data	
Alvimopan	6 mg, 12-hourly
Mitemcinal	10-30 mg, 12-hourly
Domperidone	10-20 mg, 6-hourly
Ghrelin	10 pmol/kg/min infusion
Dexloxiglumide	200 mg, 8-hourly

Monitoring of enteral nutrition

Feed administration	*daily*
Fluid balance	*daily*
Laboratory tests	
Na, K, Glucose	*initially daily*
P, Ca, Urea, Creatinine, ALT, Blood count	*initially twice/week*
Nutritional status	*weekly/every 2nd week*
Weight, albumin, Bioimpedance analysis	
Functional status	*weekly*
Hand grip strength	

Table 5

Recommendations for Monitoring PN in Hospitalized Patients

Monitoring parameter	Initial frequency	Frequency when more stable
Body weight	Daily	Every other day
Inputs and outputs	Daily	Daily
Vital signs	3–4 times daily	1–2 times daily
Serum electrolytes	Daily	2–3 times weekly
Blood urea nitrogen, creatinine	Daily	2–3 times weekly
Blood glucose	1–4 times daily	Daily
Triglycerides	Daily	Weekly
Liver function tests	2 times weekly	Weekly
International normalized ratio	Weekly	Weekly
Complete blood count	Weekly	Weekly
Albumin, prealbumin	Weekly	Weekly
Nitrogen balance	Weekly	Weekly

ASPEN guidelines on critical care

Table 1. Summary of Characteristics of Enteral Formulations and Recommendations for Use.

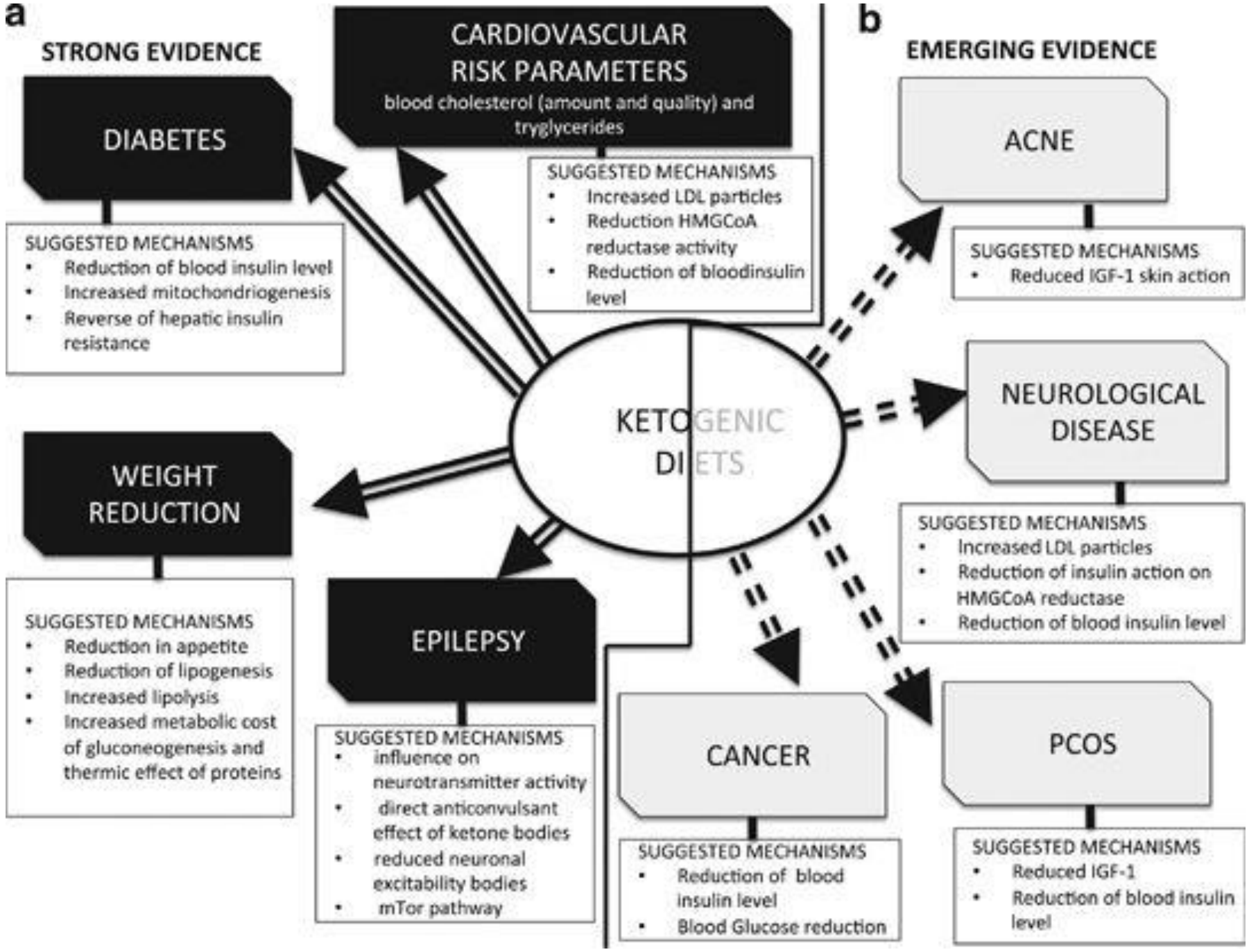
Formula Type	Summary of Characteristics	Recommendations for Use
Polymeric	• Contain macronutrients as nonhydrolyzed protein, fat, and carbohydrate • Range in concentration from 1–2 kcal/mL • 1–1.5 liters usually meets RDA for vitamins and minerals • May be disease specific and/or contain pre- and probiotics	• Intended for use among patients without severe malabsorptive disorders
Fiber containing[5–16]	• Fiber content intended to improve the health of the GI tracts regulating frequency and/or consistency of stool by maintaining healthy GI flora • Fiber content is typically well below total daily fiber recommendations • May contain prebiotics in the form of fructooligosaccharides, oligofructose, or inulin • May also contain probiotics	• Recommended for use among patients with diarrhea and/or to promote/maintain gut microbiota
Whole food/blenderized[17]	• Blenderized whole foods designed to allow patients to receive qualities of food not found in standard enteral formulas, such as phytochemicals	• Only considered for use in medically stable patients with a healed feeding tube site and no signs of infection • Best suited for patients with safe food practices and tube maintenance techniques • Should be provided as bolus feeds to maintain safe food practices (hang time ≤2 hours) • RD should be involved in development of feeding composition to ensure adequate nutrient delivery

Diabetes/glucose intolerance[18–25]	• Intended to reduce hyperglycemia with macronutrient composition of 40% carbohydrate, 40% fat, and 20% protein • Fat and soluble fiber content may slow gastric emptying and prevent elevated blood glucose	• Use of DM-specific enteral formulas is not currently supported by strong research; instead, efforts should be made to prevent overfeeding
Renal[9,26–32]	• Fluid restricted	• Standard enteral formula should be the first line for patients with renal insufficiency
	• Contain lower amounts of electrolytes, specifically potassium and phosphorous to prevent excessive delivery to patients with renal insufficiency	• If significant electrolyte abnormalities exist or develop, a renal formula should be considered until electrolytes stabilize
	• Protein content varies	• Standard, high-protein formulas without fluid restriction should be used among critically ill patients receiving dialysis; if electrolyte abnormalities exist without dialysis, renal formulas should be considered
Hepatic[9,33–39]	• Contain lower protein content with higher percentage of branched-chain amino acids, lower aromatic amino acids to prevent hepatic encephalopathy	• Standard EN formula should be administered as first line among patients with hepatic encephalopathy
	• Low protein content may result in inadequate protein delivery	• Reserve only for use among encephalopathic patients in whom standard therapy with luminal acting antibiotics and lactulose does not improve encephalopathy
	• Fluid and sodium restricted to attenuate effects of ascites	
Bariatric[9,40–49]	• Contain approximately 37% kcal from protein in efforts to maintain positive nitrogen balance, modest carbohydrate content for glucose control, and EPA/DHA in efforts to modulate inflammatory response	• Intended for patients with BMI >30 kg/m^2

Table 1. (continued)

Formula Type	Summary of Characteristics	Recommendations for Use
		• Goal enteral delivery should not exceed 60%–70% of target energy requirements, but provide adequate protein
Elemental/semi-elemental[52–55]	• Macronutrients are hydrolyzed to maximize absorption	• Intended for use among patients with malabsorptive disorders; not intended for routine use
Pulmonary/fish oil[56–73]	• In efforts to reduce carbon dioxide production, these formulas are contain >50% total calories from fat, with lower carbohydrate (<30%) and similar protein content (16%–18%)	• Efforts to prevent excessive EN delivery should be employed to reduce complications associated with overfeeding
	• Typically also contain ω-3 fatty acids derived from fish oil to increase delivery of anti-inflammatory properties of EPA/DHA	• Pulmonary formulas should be used with caution among septic, critically ill patients
Immunonutrition/ immune modulating[66–67,70–71,73–88]	• Contain pharmacologically active substances, such as arginine, glutamine, ω-3 fatty acids, γ-linolenic acid, nucleotides, and/or antioxidants in efforts to modulate immune function	• Administration of immune-modulating substances as components of EN are potentially beneficial when used for patients undergoing elective surgery; however, research is not sufficient to recommend immune-modulating formulas for routine use among critically ill patients

BMI, body mass index; DHA, docosahexaenoic acid; DM, diabetes mellitus; EN, enteral nutrition; EPA, eicosapentaenoic acid; GI, gastrointestinal; RD, registered dietitian; RDA, recommended dietary allowances.


a
STRONG EVIDENCE
DIABETES
SUGGESTED MECHANISMS
• Reduction of blood insulin level
• Increased mitochondriogenesis
• Reverse of hepatic insulin resistance
CARDIOVASCULAR RISK PARAMETERS
blood cholesterol (amount and quality) and tryglycerides
SUGGESTED MECHANISMS
• Increased LDL particles
• Reduction HMGCoA reductase activity
• Reduction of bloodinsulin level
KETOGENIC DIETS
WEIGHT REDUCTION
SUGGESTED MECHANISMS
• Reduction in appetite
• Reduction of lipogenesis
• Increased lipolysis
• Increased metabolic cost of gluconeogenesis and thermic effect of proteins
EPILEPSY
SUGGESTED MECHANISMS
• influence on neurotransmitter activity
• direct anticonvulsant effect of ketone bodies
• reduced neuronal excitability bodies
• mTor pathway
b
EMERGING EVIDENCE
ACNE
SUGGESTED MECHANISMS
• Reduced IGF-1 skin action
NEUROLOGICAL DISEASE
SUGGESTED MECHANISMS
• Increased LDL particles
• Reduction of insulin action on HMGCoA reductase
• Reduction of blood insulin level
CANCER
SUGGESTED MECHANISMS
• Reduction of blood insulin level
• Blood Glucose reduction
PCOS
SUGGESTED MECHANISMS
• Reduced IGF-1
• Reduction of blood insulin level

THE KETOGENIC FOOD PYRAMID

Carbohydrates

Keep carbohydrates to a maximum of 5% of your total daily calorie intake. Making up of mostly green cruciferous vegetables. **Avoid all sugars, starches, grains, bread, pasta, fruits** (except avocado).

5%

Protein

Protein is essential for muscle retention and muscle building but **too much protein can keep you out of Ketosis.** Limit your protein intake to 25% of your daily calorie intake. Excellent sources of protein are: Fatty cuts of meat, eggs, full fat cheeses. Avoid milk, fat reduced cheeses and creams.

25%

Fat

Fats will make up a dominant portion of a Ketogenic Diets macronutrients. When fat intake is high and carbs are low the body will resort to using fat as fuel through **Ketosis** (put simply). When possible your fat intake should come from **Saturated Fats** (Butter, Coconut Oil etc) & **Monounsaturated Fats** (Avocado, Macadamia Nuts etc). Ensure you get ample Omega-3's in your diet as well.

70%

TABLE 3

Sample 1,500-kcal Menu for Classic Ketogenic Diet

MEAL COMPONENTS	APPROXIMATE HOUSEHOLD MEASUREMENT
Breakfast	
Heavy whipping cream: 65 g	2 oz heavy cream
Protein: 25.5 g eggs and 10 g bacon	0.5 egg, 2 strips bacon
Fat: 10 g butter and 13.8 g mayonnaise	2.5 pats butter, 1 packet mayonnaise
Carbohydrate: 6 g peaches	1 tsp peaches
Lunch	
Heavy whipping cream: 65 g	2 oz heavy cream
Protein: 6.6 g macadamia nuts, 19.6 g deli ham, 8.4 g American cheese	2.5 macadamia nuts, 1.5 slices deli ham, 0.5 slice American cheese
Fat: 10.9 g mayonnaise and 4 g oil	1 packet mayonnaise, 1 tsp oil
Carbohydrate: 10.4 g applesauce	2 tsp applesauce
Dinner	
Heavy whipping cream: 65 g cream	2 oz heavy cream
Protein: 48.5 g hot dog	1 hot dog
Fat: 11.4 g mayonnaise and 4 g oil	1 packet mayonnaise, 1 tsp oil
Carbohydrate: 14.2 g broccoli	1 T broccoli

Note: Meal plan provides 1,500 kcal, 26 g protein, and 11 g carbohydrates