study smarter method

Medical Nutrition Therapy

How to use this study guide

Note to reader: Many RD2Be's want to start studying for the RD exam with MNT. However, MNT makes up a very small portion of the RD exam. If you have already studied for the exam, I encourage you to focus instead on Management Concepts, Research Concepts, and Food Service, while using practice questions for clinical scenarios.

As you review MNT considerations for each of the following states, think about how and why energy and nutrient needs change from standard pediatric or adult recommendations to the recommendations for each nutrition-related disorder. I recommend having a stack of blank paper or a blank notebook to use as you work through this study guide. There are study guide questions throughout that have you critically assess the information you've read. You can also use the MNT worksheet template available in the RD Exam Toolkit (https://www.allaccessdietetics.com/rd-exam-toolkit/) to organize the information in this section. **Be sure to read the recommended readings, many of which are position and practice papers. CDR considers these resources fair game for the exam.**

To help you organize the information, always come back to big picture questions. For example, if protein and calorie needs are elevated for a disease, know WHY. HOW does the underlying pathophysiology of the disease manifest in signs and symptoms, including lab test results? HOW might you expect the planned nutrition intervention to influence those signs and symptoms? Turn information that you read into questions you can answer, so that you aren't overwhelmed by details so that you learn the information in the same way you'll be asked on the test – in question form. You need to be able to connect details together into a bigger picture rather than trying to simply memorize the details. The study guide questions throughout are "big picture questions" and after a few sections you may be able to think of your own study guide questions.

You'll notice that most conditions have a nutrition therapy and MNT table at the beginning of the section. The format of this table varies from acute to chronic conditions. For example, some conditions, such as burns, may have specific energy, protein, and fluid recommendations. For other conditions, such as celiac disease, where the estimated needs are the same for someone without celiac disease, there is minimal change. I recommend keeping a table of the DRIs handy as you go through this document so that you can reinforce what the typical energy and macronutrient needs are with attention to changes that should be made for patients of certain conditions, such as a gluten-free nutrition prescription for patient with celiac disease. While this study guide mentions aspects of the Nutrition Care Process, it is not intended to replace review of the Nutrition Care Process. You can learn about the NCP in the Nutrition Care study guide or online for free at https://www.ncpro.org/nutrition-care-process.

If you like the format of this study guide, you will like the other Study Smarter Method study guides. The other study guides also include more extensive lists of study guide questions (open-ended questions throughout this document in italics) as well as multiple-choice practice questions. A full list of study guides available is below. You can get these 10 study guides in Pass the Exam Prep (powered by the Study Smarter Method) at www.allaccessdietetics.com/Pass-the-exam-prep

- Calculations and Formulas
- Normal Nutrition, Digestion, and Absorption
- Enteral and Parenteral Nutrition Support
- Kidney Function, Electrolytes, and Dehydration
- Food Service Logistics
- Food Science
- Food Safety
- Nutrition Care
- Research Concepts
- Management Concepts

Terms of use

medical nutrition therapy study guide

FOR THE REGISTERED DIETITIAN NUTRITIONIST EXAMINATION

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Reference (where not otherwise specified)

- Mahan LK, Escott-Stump S. and Raymond JL. **Krause's Food and the Nutrition Care Process.** 2012. Philadelphia, Pa.; Edinburgh: Elsevier Saunders. 13th ed. Print.

Critical Care & Hypermetabolic States

Trauma activates a systemic response with physiologic and metabolic changes that can lead to shock and organ dysfunction. The metabolic response to injury has an ebb phase and a flow phase (also called "adaptive flow response"). The ebb phase is characterized by hypovolemic shock, with poor tissue perfusion, depressed metabolic rate, low oxygen consumption, and decreased blood pressure and body temperature. The acute flow response follows the ebb phase after fluid resuscitation and oxygen transport restoration. The acute phase is characterized by lean body mass catabolism, acute phase proteins, and increased metabolic rate with impaired utilization of fuel. During the adaptive flow response, the body returns to positive nitrogen balance (anabolism) and the hormonal response decreases.

Pull out a sheet of paper and divide it in half. Write ebb phase at the top of one side and flow phase at the top of the other. As you go through this section, list the features of each.

Nutrition therapy should account for catabolic protein losses and provide sufficient calories and protein for healing while also recognizing the social and emotional function of food. For example, a patient may need enteral nutrition support to meet increased calorie and protein needs but it is important for the RDN to consider the use of oral intake (if medically appropriate) as a comfort measure.

Nutrition Therapy	**MNT Goals[1]**

• Energy: ideally use indirect calorimetry
  ○ General: 25-35 kcal/kg
  ○ Penn State equation
• Protein: >1.2 g/kg/day
• Fluid: Variable
• Micronutrients: meet at least DRIs
	• Improve outcomes (infection rate, ventilator days, days in ICU)
	  ○ Initiate early enteral nutrition in a safe manner if necessary
	  ○ Avoid overfeeding (possibly through mild underfeeding)
	  ○ Tightly controlling blood glucose
	  ○ Providing immunonutrients if appropriate
	• Minimize catabolic loss of body protein

Before going into specifics for burns, bone fractures, surgery, and pressure ulcers, here are some general considerations for critically ill patients.

In the absence of indirect calorimetry resources, the RDN can estimate energy needs in mechanically ventilated patients using the Penn State equations for both nonobese and obese critically ill patients. The Penn State equations modify the RMR estimate from the Mifflin-St Jeor formula with coefficients for minute ventilation (L/min) and max daily temperature (°C) for mechanically ventilated patients. You don't need to know the Penn State equations for the RDN exam. You can read the 2012 Evidence Analysis Library Critical Illness Guideline here: https://www.andeal.org/topic.cfm?cat=4840.

Energy needs aren't typically met in critically ill patients and there is controversy around whether it is desirable to meet 100% of energy needs in the early-stage critically ill patient. ASPEN

recommends aiming for 65-70% of energy needs determined via indirect calorimetry. [2] If not using indirect calorimetry or the Penn State equations, a weight-based equation of 25-30 kcal/kg/day can be used. Note that in severe sepsis or burns, up to 35 kcal/kg may be used. For protein, general recommendations are 1.2 – 2 g protein/kg/day. If a patient is receiving continuous renal replacement therapy (CRRT) for acute kidney injury (AKI), protein should be increased to 1.5 – 2.5 g/kg/day. Fluid needs depend on the actual physiologic state of the critically ill patient, including the need for volume resuscitation, the extent of capillary leakage and endothelial injury, as well as cardiac and renal function (acute or chronic).

ASPEN recommends hypocaloric, high-protein nutrition support for critically ill patients with obesity (including patient with burns) in order to preserve lean body mass, utilize adipose stores, and minimize overfeeding complications. [2] **Energy** estimates may be calculated as 20-25% total energy estimate, 11 – 14 kcal/kg actual weight if BMI 30 - 50, or 22-25 kcal/kg IBW for BMI >50.[2] The table below summarizes **protein** recommendations for critically ill patients with obesity. *Compare and contrast the protein needs of a critically ill patient with obesity to one without obesity.*

Table 1. Protein Needs for the Obese Critically Ill Patient

Protein	Situation
1.2 – 2 g/kg/day actual body weight	BMI < 30
2 g/kg/day IBW	BMI 30 – 40 with hypocaloric feeding
2.5 g/kg/day IBW	BMI >40 with hypocaloric feeding

As nutrition support is often used in the critically ill patient, be sure to review information on enteral and parenteral nutrition support. Use your own resources, such as Krause or class day materials from your internship. You can also download the Enteral and Parenteral Nutrition Support Study guide at www.studysmartermethod.com. Extensive nutrition support explanations and calculations are beyond the scope of this free resource. You may also find these free tutorials helpful:

- Enteral nutrition: http://www.csun.edu/~cjh78264/tubefeeding/introduction.html
- Parenteral nutrition: http://www.csun.edu/~cjh78264/parenteral/index.html

Burns

Nutrition Therapy	MNT Goals[3]
• Energy: Increase 20 – 30% up to 2 x REE • Protein: 20-25% kcal • CHO: 60% • Vitamins & minerals	• Wound healing • Infection prevention • Meet nutritional needs • Weight maintenance • Fluid and electrolyte balance

Burns are classified by 3 things: percentage of total body surface area (TBSA), depth (1st, 2nd, 3rd, or 4th degree) and etiology. The first 24-48 hours after a large burn is the “ebb” or shock phase and is characterized by capillary leakage leading to edema, decreased cardiac output, decreased tissue perfusion, and depressed metabolic rate. Treatment during this stage is focused on large volume fluid resuscitation. The acute, or ‘flow’ phase, occurs after resuscitation and is the period of increased metabolism due to a catecholamine-driven stress response. This results in increasing oxygen consumption, gluconeogenesis, and proteolysis, leading to loss of lean body mass and negative nitrogen balance. The flow phase is where we focus on feeding.

How are burns classified?

Calorie needs are increased due to fever, sepsis, trauma, wound healing, stress of surgery (if applicable), and physical therapy (if applicable). Carbohydrates should make up 60% of total calories.

The diet should be high calorie, high protein, high fluid, and high in vitamin C and zinc. Calorie needs should increase by 20 – 30% and may be as high as 2 x REE (but should not exceed 2 x REE). Ideally, indirect calorimetry is used to estimate REE but in the absence of that technology, the Harris-Benedict equation or the Curreri formula can be used Table 2.

Protein requirements are increased in burn patients because of wound healing and anabolism but how much they increase depends on the extent of the burns. A rule of thumb is to have protein make up 20-25% of total calories or 1.5 to 2 g/kg of body weight. Protein needs may be as high as 2.5 g/kg/day after surgery and up to 4 g/kg/day during the flow phase.

For the exam you should be familiar with the Harris-Benedict Equation.

Table 2. Harris-Benedict Equation to estimate RMR

Men	*66 + Weight(13.7) + Height(5) – Age(6.8)*
Women	*655+ Weight(9.6)+ Height(1.7) - Age(4.7)*

Weight in kg, height in cm, and age in years.

The RDN should monitor BUN, creatinine, and hydration status when using high protein diets. In the first 24-48 hours following injury, fluids and electrolytes should be restored. When fluid status is stabilized, feeding can begin. Enteral or parenteral nutrition support may be required to meet nutrient and fluid needs particularly in acute stages of treatment, with other medical conditions, and for large surface area burns. ASPEN recommends initiating EN within 4-6 hours of the injury.[2]

Surgery

Nutrition Therapy	**MNT Goals**
• Energy: Increase • Protein: Increase • GI-specific modifications • Vitamins & minerals	• Optimize nutritional status pre-op • Recovery after surgery • Promote healing • GI-specific goals

Optimizing nutritional status before surgery (if time allows) is important to prepare the body for the stress of the surgery and to help with recovery following surgery. In the postoperative period, good nutrition promotes healing. If the surgery involves the GI tract, the patient typically remains NPO for up to 48 hours and then progresses from clear liquids to soft, digestible foods, to a regular diet that is high in protein, vitamins, and minerals. *For GI-specific modifications and goals, think about if a patient had a bowel resection, a gastrectomy, or has trouble swallowing. What food/nutrient-delivery interventions would you need to think about?*

Pressure Ulcers

Nutrition Therapy	MNT Goals[4]
• Energy: Meet increased needs (30-40 kcal/kg/day) • Protein: Increase (1.25 – 2 g/kg/day)* • Fluid: 1 mL/kcal/day** • Vitamins & minerals if indicated per labs • Assess medication list *See* Table 3 *for more details.*	• Maintain adequate nutritional status through optimal nutrient and fluid levels • Identify and treat causes of poor nutritional intake • Monitor weight status routinely to detect weight loss • Select nutrition interventions that improve/maintain nutritional status

*Excessive protein intake (beyond 1.5 g/kg/day) may not necessarily lead to increased protein synthesis and may increase risk of dehydration.[5] **Fluid needs are increased with higher protein intake, if using an air-fluidized bed, or with additional fluid losses.

Pressure ulcers are defined as a "localized injury to the skin and/or underlying tissue over a bony prominence, as a result of pressure or pressure in combination with shear".[6] Pressure ulcers develop due to continuous pressure impeding capillary blood flow to the skin and underlying tissue. Tissue is compressed and blood flow is restricted, causing ischemia and necrosis. Shear is force parallel to the skin that stretches and distorts internal tissues, leading to ischemia and necrosis. Poor or impaired mobility, paralysis, incontinence, rigidity, and sensory losses contribute to the risk and severity of pressure ulcers. Malnutrition and undernutrition can contribute to the formation of pressure ulcers as well as delay healing. Patients that are bedridden are at particular risk for pressure ulcers.

The National Pressure Ulcer Advisory Panel classification system has 6 categories, or "stages" of pressure ulcers.[6] Staging is based on observation and palpation of the tissue and when pressure ulcers heal, they do not 'reverse' stage. Stage I and Stage II are partial thickness ulcers while Stage III, Stage IV, and unstageable ulcers are full thickness ulcers. Suspected deep tissue injuries, sometimes abbreviated sDTI, fall somewhere in between. See Table 3 for detailed descriptions and a summary of energy and protein needs. Access the PowerPoint from Dr Karen Zulkowksi's Agency for Healthcare Research and Quality (AHRQ) webinar on wound classification for graphic photos of pressure ulcers at each stage.

- AHRQ Webinar PowerPoint on Wound Classification form Dr Karen Zulkowski: https://bit.ly/3vaB7EK

Some terms used in Table 3 include eschar, slough, undermining, and tunneling. Eschar is black, brown, or tan necrotic tissue that adheres firmly to the wound bed or edges of the ulcer. Slough is yellow or white tissue, liquified or wet, that adheres to the ulcer bed in rings, thick clumps, or is mucinous. Undermining means that the tissue damage extends under the edge of the visible wound bed while tunneling refers to tracts that extend out from the wound. Suspected deep tissue injuries may be covered in thin eschar, and unstageable wounds are unstageable because they are covered in a thick layer of eschar. Slough may be present in Stage III and Stage IV ulcers. Stage IV ulcers may have slough and/or eschar. The AHRQ webinar has graphic photos of these 4 terms.

Table 3. Description and MNT Energy/Protein Needs by Pressure Ulcer Stage

	Partial thickness		**Suspected deep tissue injury**
	Stage I	Stage II	
Description	• Intact skin • Localized area of non-blanchable redness • Usually over a bony prominence • Painful, firm, soft, warmer, or cooler than adjacent tissue • May be difficult to detect in patients with dark skin tones	• Loss of dermis • Shiny or dry shallow ulcer without slough or bruising • Red-pink wound bed *or* ruptured blister filled with serum	• Purple or maroon localized area of intact skin or blood-filled blister • May become covered with thin eschar • Evolution can be rapid, exposing additional tissue layers • May be difficult to detect in patients with dark skin tones
Nutrition Therapy	Energy: 30 - 35 kcal/kg/day Protein: 1.25 – 1.5 g/kg/day		Energy: 30 kcal/kg/day Protein: 0.8 - 1 g/kg/day
	Full thickness		
	Stage III	Stage IV	Unstageable
Description	• Open ulcer exposing subcutaneous fat or tissue but not bone, tendon, or muscle • Can be shallow in areas without subcutaneous tissue such as nose, ear, occiput, malleolus, and bridge of nose • Slough may be present	• Open ulcer exposing bone, tendon, or muscle • Often includes undermining and tunneling • Slough or eschar may be present • Can extend to muscle and supporting structures leading to osteomyelitis • Can be shallow in areas without subcutaneous tissue	• Ulcer covered by slough or eschar, preventing ascertainment of wound depth and stage
Nutrition Therapy	Energy: 35 - 40 kcal/kg/day Protein: 1.5 – 1.75 g/kg/day	Energy: 35 - 40 kcal/kg/day Protein: 1.75 – 2 g/kg/day	

Source: National Pressure Ulcer Advisory Panel, 2019

Both energy and protein needs are increased in patients with pressure ulcers. Routine micronutrient supplementation is not needed; however, if lab tests reveal depleted levels, they should be supplemented. Micronutrient supplementation in absence of true deficiency remains controversial as the evidence of benefit is inconclusive.[5] Micronutrients you may see referenced include vitamin A, vitamin C, zinc, and arginine. The medication list should also be reviewed for any medications that may impede wound healing, appetite, or nutrient absorption. Fluid needs may be higher than normal if a patient is on an air-fluidized bed, has additional fluid losses, or is at risk of dehydration due to high protein intake.[5] Patients with conditions previously requiring fluid restrictions (e.g. severe kidney disease or heart failure) should continue to have fluid restrictions.

How would you determine the stage of a partial thickness pressure ulcer? Full thickness? What calorie and protein would you prescribe for each stage?

Chronic Wounds

Nutrition Therapy	MNT Goals[7]
• Energy: 30 - 35 kcal/kg/day • Protein: 1.25 - 1.5 g/day • Fluid: 30 ml/kg or 1 – 1.5 mL/kcal	• Meet energy, protein, and fluid needs through oral intake or nutrition support • identify and address causes of poor intake • Prevent unintended weight loss or increase weight, if appropriate • Correct dehydration • Correct any nutrition deficiencies contributing to delayed wound healing • Control glucose levels in patients with diabetes

The 3 phases of wound healing are inflammatory, proliferation, and maturation or remodeling. A chronic wound is one with a skin defect persisting >6 weeks, or a wound with frequent recurrence.[7] Chronic wounds are more common in patients with conditions that slow wound healing or prevent wound healing (Table 4). Wound healing cannot be ignored in patients, even if they are being treated for other factors. Factors associated with nonhealing wounds include poor circulation, compromised immune system (for example, due to cancer, cancer treatment, HIV/AIDS, immunosuppressive medications), some medications (glucocorticoid steroids, NSAIDs, chemotherapy), older age (≥65 years), dehydration, immobility, neuropathy, spinal cord injuries, obesity, certain diseases, stress, tobacco smoking, malnutrition, and certain nutrient deficiencies.[7] Conditions associated with poor wound healing include diabetes, uremia, keloids, fibrosis, jaundice, alcoholism, heart disease, respiratory diseases, and hereditary healing disorders. Many of these factors and conditions co-exist or contribute to one another. For example, a patient with diabetes may have poor wound healing due to poor circulation, neuropathy, and obesity. Obesity contributes to poor wound healing due to local wound conditions (e.g. increased wound tension, venous hypertension, decreased vascularity to tissue), associated diseases or conditions (e.g. mobility problems, heart disease, dyslipidemia, hypertension, stroke, respiratory issues), and factors altering immune and inflammatory responses (e.g. adipokine, cytokine, and chemokine concentrations).[8] Regarding nutrition status, malnutrition (protein and/or energy) can affect collagen synthesis, contribute to prolonged inflammation, decrease phagocytosis, lead to immune cell dysfunction, and decrease the skin's mechanical strength – all of which affect wound healing.[9] Some micronutrient deficiencies are associated with poor wound healing because those micronutrients are necessary for wound healing.

Table 4. Factors and Diseases Associated with Poor Wound Healing

• Age ≥ 65 years	• Dyslipidemia	• Neuropathy
• Alcoholism	• Fibrosis	• Nutrition status
• Cancer	• Hereditary healing disorders	• Obesity
• Compromised immune system (cancer, cancer treatment, AIDS, immunosuppressive medications)	• Hypertension	• Poor circulation
	• Immobility	• Respiratory dysfunction
	• Jaundice	• Smoking
• Coronary heart disease	• Keloids	• Spinal cord injury
• Dehydration	• Medications (steroids, NSAIDs, chemotherapy)	• Stress
• Diabetes		• Uremia

Adapted from Guo & Dipietro 2010.[8]

Nutrition diagnoses for wound healing may include inadequate protein intake, increased nutrient needs (protein and energy), or increased nutrient needs (protein), among others. Calorie needs range from 30 to 35 kcal/kg/day for nonobese adults. Older patients and obese patients need an individualized approach such as modified predictive equations or indirect calorimetry to best determine REE. Increased protein is needed for wound healing in order to synthesize enzymes and to support cell proliferation and connective tissue formation. Protein of 1.25 – 1.5 g/kg/day is recommended. Excessive protein, such as 2 g/kg/day may lead to dehydration particularly in older adults and those with reduced kidney function. *How is kidney function measured?*

Nutrition interventions to help with poor oral intake include providing snacks or nutritional supplements in between meals, using calorie- or protein-dense foods, and providing feeding assistance or modified equipment at meal time.

What are the 3 phases of wound healing?

What factors and conditions are associated with nonhealing wounds?

Look at the following patient examples. Are their wounds chronic? Are they at risk of chronic wounds?

Eating Disorders

"Eating disorders" is an overarching term currently used to describe "abnormal and maladaptive eating and related behaviors with psychological and biological underpinnings."[10] Because the underlying cause(s) of eating disorders have not been identified, medical diagnosis is made using signs, symptoms, and behaviors. Also, because the underlying cause(s) have not been identified, individualization of eating disorder treatment is important, because patients with similar symptoms may have different etiologies and different disease processes.[10] There are many different types of eating disorders and all are defined and described in the *Diagnostic and Statistical Manual of Mental Disorders (DSM).*[11] Some types include anorexia nervosa, bulimia nervosa, binge eating disorder, avoidant/restrictive food intake disorder or ARFID, night eating syndrome, and purging disorder. Night eating syndrome, purging disorder, "atypical" anorexia, bulimia of "low frequency or limited duration", and binge eating disorder "of low frequency or limited duration" are grouped together in OSFED, or "other specified feeding and eating disorders" because they are variations of other eating disorders but not as well-defined.

Recommended reading:

- Position of the American Dietetic Association: **Nutrition Intervention in the Treatment of Anorexia Nervosa, Bulimia Nervosa, and Other Eating Disorders**. J Am Diet Assoc. 2006. 106:2073-2082. https://doi.org/10.1016/j.jada.2006.09.007

RDNs may be the first to recognize an eating disorder and because nutritional rehabilitation is a "cornerstone of eating disorder recovery", RDNs play a key role in treating individuals with eating disorders. An RDN should not rule out an eating disorder diagnosis solely based on gender, economic status, weight, age, appearance, mental capacity, or any other single factor. While there may be patterns in who presents with eating disorders, research is limited, with small sample sizes and the majority of studies are in women. There is no "typical" eating

disorder patient. In other words – eating disorders occur at every weight and size; eating disorders occur at similar rates across race and ethnicity group; and persons of all genders can develop eating disorders.

What signs and symptoms are common across several eating disorders? Which are distinct?

AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER (ARFID)

Avoidance or restriction of food intake manifesting as failure to meet nutrient (including energy) needs through oral intake of food due to lack of interest in eating or in food, avoidance based on food characteristics, concern about consequences of eating (e.g., choking, vomiting)

Onset typically in infancy or childhood[11]

Nutrition-related Medical Issues
- Significant weight loss or faltering growth (in children)
- Micronutrient deficiencies
- Malnutrition

Nutrition Therapy
- Address nutrition-related medical issues
- Normalizing eating patterns

Signs & Symptoms
- Growth faltering (children)
- Significant weight loss or low BMI
- S&S of micronutrient deficiencies
- Low body temperature
- Bradycardia
- Anemia

BINGE EATING DISORDER

Recurrent episodes of binge eating at least 1x/week for 3 weeks with feelings of loss of control during the episode, without compensatory behaviors

BMI may be normal or indicate overweight/obesity[11]

Nutrition-related Medical Issues
- Health risks associated with overweight/obesity

Nutrition Therapy
- Normalizing eating patterns

Signs & Symptoms
- Gastrointestinal symptoms
- Weight fluctuation
- Difficulty concentrating

ANOREXIA NERVOSA

Restricted eating leading to a significantly low body weight (BMI < 18.5), intense fear of gaining weight even though at a low weight, and distorted perception of body image[11]
- Two subtypes: restrictive and binge/purge

Nutrition-related Medical Issues
- Protein-calorie malnutrition
- Micronutrient deficiencies
- Fluid and electrolyte imbalances

Nutrition Therapy
- Address nutrition-related medical issues
- Normalizing eating patterns
- Gradual caloric adjustment

Signs & Symptoms
- Low bone density, osteoporosis
- Muscle wasting, weakness
- Low magnesium, phosphorous
- Low potassium with refeeding syndrome
- Menstrual irregularity or amenorrhea
- Orthostatic hypotension
- Bradycardia (low HR)
- Lanugo (fine body hair)
- Lowered body temperature
- Decreased thyroid function
- Delayed gastric emptying
- Decreased immune function
- Difficulty concentrating

BULIMIA NERVOSA

Repeat episodes of binge eating, recurrent inappropriate compensatory behaviors to prevent weight-gain (at least 1x/week for 3 months), self-evaluation heavily influenced by body shape and weight;[11] often 18.5 ≤ BMI < 30

Inappropriate compensatory behaviors to prevent weight gain include self-induced vomiting, misuse of enemas, diuretics, laxatives, fasting, excessive exercise[11]

Nutrition-related Medical Issues

- Fluid and electrolyte imbalances

Nutrition Therapy

- Address nutrition-related medical issues
- Normalizing eating patterns

Signs & Symptoms

- Menstrual irregularity or amenorrhea
- Low potassium, chloride, magnesium
- Gastrointestinal symptoms (reflux, gas, bloating)
- Cardiac arrhythmias
- Dental erosion due to vomiting
- Difficulty concentrating

What is refeeding syndrome?
A shift in fluid and electrolytes that can lead to cardiovascular failure, respiratory failure, seizures, and death.[12,13]

Why does it occur?
In a starvation state, the catabolism of muscle and fat leads to electrolyte losses (notably potassium, magnesium, and phosphate) which may or may not manifest in low lab values.[12,13] When normal nutrition intake resumes, electrolytes shift into cells, causing low blood levels that manifests in test results and serious medical consequences.[12,13]

When does it occur?
Refeeding syndrome can occur when an oral, enteral, or parenteral diet is initiated in a very malnourished person. Nutritional rehabilitation should be performed under medical observation to reduce the risk of refeeding syndrome or to manage its occurrence.[12]

Who is at highest risk?[13]
Persons at risk of refeeding syndrome include those with BMI < 16, weight loss of 15% UBW in past 3 – 6 months, little to no food intake over the past 10 days, or low phosphorus, potassium, or magnesium levels in the blood. Among patients with diagnosed anorexia nervosa or avoidant/restrictive food intake disorder, a BMI < 18.5, weight loss of 10% UBW in past 3 – 6 months, little to no food intake over past 5 days, or a history of alcoholism, insulin use, chemotherapy treatment, diuretic use, or antacids increases the risk of refeeding syndrome.

What are the clinical signs & symptoms?[12]

- **Phos**: Very low phosphorus (hypophosphatemia) can lead to confusion, heart failure, cardiac arrhythmias, and seizures.
- **Mg**: Very low magnesium (hypomagnesemia) can lead to cardiac arrhythmias, tachycardia, diarrhea, seizures, and low calcium.
- **K**: Very low potassium (hypokalemia) can lead to cardiac arrest, cardiac arrhythmias, paralysis, respiratory depression, and ileus.
- **Glucose**: High blood glucose (hyperglycemia)
- **Fluid**: Fluid excess (rapid weight gain, change in sodium, increased blood pressure, increased heart rate)

Can you think of a mnemonic to remember the S&S of refeeding syndrome?

The role of the RDN in treating eating disorders includes the following:[10]

- Evaluate the patient's current eating patterns
- Offer active learning activities when appropriate (e.g. cooking, eating, grocery shop) to help teach new behaviors and acceptance of food-related tasks and environments
- Identify dysfunctional and detrimental thoughts and feelings around food, eating, and body size, and knowledge and skill deficits that prevent the patient from implementing recommendations
- Develop an individualized plan for improvement to replenish nutritional deficiencies and promote optimal nutrition and growth
- Teach group nutrition classes to patients, their families and caregivers and lead group nutrition discusses that address dysfunctional eating and promote improved nutrition
- Educate patients and caregivers regarding eating disorders and nutrition as they relate to the treatment plan and recovery needs
- Explain the role of proper nutrition and eating in physical and mental well-being and provide education to challenge inaccurate beliefs about food
- Help the patient determine how to implement needed nutritional recommendations
- Share findings with other team members
- Communicate frequently with other members of the interdisciplinary treatment team including family members and significant others
- Refer information about underlying life stressors to a mental health professional

During the nutrition assessment, the RDN should identify eating disorder symptoms and behaviors. Important data to collect include anthropometric measurements (height and weight history, growth patterns and maturation in patients ≤ 20 years); biochemical data (with attention to refeeding syndrome); evaluate eating patterns and core attitudes regarding weight, body shape, and eating; and assess behavioral-environmental symptoms such as food restriction, bingeing, preoccupation, rituals, secretive eating, affect, impulse control, purging, excessive exercise. Then, the RDN should identify and prioritize the nutrition problems using PES statements.

The nutrition intervention should focus on the etiology when possible, but otherwise on signs and symptoms, including calculating energy and macronutrient intake needs to achieve expected rate of weight change and to meet health goals, while setting goals *with* the patient, focusing on normalizing eating patterns and weight restoration or maintenance if appropriate. Nutrition interventions for eating disorders may include food and/or nutrient delivery, nutrition-related medications, nutrition education, nutrition counseling, and should always include coordination of care, as eating disorder treatment require interdisciplinary care. Specific Nutrition Intervention examples include increasing the amount and/or variety of foods consumed, achieving normal perceptions of hunger and satiety, modifying beliefs about supplement use, providing psychosocial support and positive reinforcement, providing a structured refeeding plan, counseling individuals and their family members (as appropriate) on food selection while considering the patient's health history, individual preferences, physical factors, psychological factors, and resources.

Nutrition Monitoring and Evaluation should include monitoring rate of weight gain and adjusting food intake to maintain weight and communicating progress with the care team. Regarding care

coordination, the RDN should act as a resource to other health care professionals, as well as the patient and their family, to provide education and raise awareness. The RDN should also advocate for evidence-based treatment and work collaboratively to communicate the patient's nutrition needs across a continuum of settings such as inpatient, day treatment, and outpatient settings. The RDN may seek additional training in cognitive behavioral therapy, dialectical behavior therapy, and motivational interviewing and seek supervision from a licensed mental health professional to expand abilities in treating patients with eating disorders.[14]

Food Allergies, Sensitivities & Intolerances

Recommended Readings

- Practice Paper: **Role of the Registered Dietitian Nutritionist in the Diagnosis and Management of Food Allergies**. J Acad Nutr Diet. 2016;116:1621-1631. PMID: 27671759. http://dx.doi.org/10.1016/j.jand.2016.07.018
- Read more about food allergies from the FDA. PDF: https://bit.ly/2LmS7pN
- Read more about celiac disease at https://celiac.org/.
- Nowak-Wegryzn A et al. International consensus guidelines for the diagnoses and management of protein-induced enterocolitis syndrome: Executive summary – Workgroup Report of the adverse Reactions to Foods Committee, American Academy of Allergy, Asthma and Immunology. J Allergy Clin Immunol. 2017. 1111-1126.e4. PDF: https://bit.ly/3fJ0ZBK

For the RD exam you should know the difference between food allergies, food sensitivities, and food intolerances as well as examples of each. You should be aware of the importance of food allergens in foodservice, in counseling patients, and be aware of the first steps in addressing potential allergies in infants.

A **food allergy** (food hypersensitivity) is a severe immune response to a food antigen that causes a histamine release, such as a peanut allergy. The "Big Eight" are 8 foods that are responsible for 90% of food allergies: milk, eggs, tree nuts, peanuts, wheat, soy, fish, and shellfish.[15]

On April 23, 2021, the Food Allergy Safety, Treatment, Education, and Research (FASTER) Act was signed and declared sesame as the 9th major food allergen recognized in the US. Labeling requirements will be effective Jan 1, 2023.[16]

Food sensitivity is a general term for a person's adverse reaction (clinically abnormal response) to a food or additive and the term may be used when it's unclear if the reaction is a food allergy or a food intolerance. Food sensitivities can manifest as dermatologic, gastrointestinal, respiratory, or systemic symptoms (Table 5).

Table 5. Symptoms of Food Sensitivities

Dermatologic	Gastrointestinal
• Red skin • Itching • Rashes (including atopic dermatitis or contact dermatitis) • Hives (urticaria)	• Nausea, vomiting, gas • Swelling of lips, neck, throat • Stomach cramps, pain • Fecal blood loss • Malabsorption
Respiratory	**Systemic**
• Sneezing • Nasal congestion • Irregular breathing	• Anaphylaxis • Pallor, irritability, headaches • Low blood pressure, cardiac arrhythmia

A **food intolerance** is a food reaction that results because of how the body processes the food, not from an immune response. An example of food intolerance is lactose intolerance due to a person's inability to digest lactose. Food intolerances include idiosyncratic responses (hypersensitivity response without immune action), pharmacologic responses (reaction to a food additive or other chemical in food), metabolic reactions (enzyme deficiencies or GI disorders such as liver disease, pancreatic disease, gallbladder disease), inborn errors of metabolism, and toxic reactions (including food poisoning). **Error! Not a valid bookmark self-reference.** compares and contrasts food allergies and food intolerances.

Table 6. Food Allergies versus Food Intolerances

FOOD ALLERGIES (IMMUNE MEDIATED)	FOOD INTOLERANCES (NOT IMMUNE MEDIATED)
IgE-mediated • Anaphylaxis • Asthma • Latex allergy • Oral allergy syndrome • Hives **Mixed IgE- and non-IgE-mediated** • Eosinophilic esophagitis • Eosinophilic gastroenteritis **Cell-mediated** • Celiac disease • FPIES	**Food poisoning** **Pharmacologic response** • MSG, tartrazine, sulfites, nitrites, nitrates, artificial color, caffeine, histamine, tyramine, serotonin **Idiosyncratic response** **Metabolic reactions** • Enzyme deficiencies • GI disease **Inborn errors of metabolism**

Review the following terms and definitions before progressing to the main part of this section.

Antibodies are proteins that the human body creates in response to **antigens**. In the case of food allergies, the antigens are food proteins that stimulate an immune response.

Atopy is a genetic predisposition to excess immunoglobin E antibody production in response to certain antigens. IgE-mediated allergies may present in response to dander, pollen, foods, among other environmental factors, with symptoms such as eczema, food allergy, atopic conjunctivitis, atopic rhinitis, and asthma. Atopy is typically identified in infancy via skin-prick test. Immune-mediated food allergies that are not IgE-mediated include celiac disease and food protein-induced enterocolitis syndrome (FPIES).

Anaphylaxis is a specific and often severe reaction to an antigen the person was previously exposed to.

Anaphylactic reaction is an anaphylaxis-like food reaction but is a result of non-immune chemical mediators rather than an immune response.

Food poisoning involves ingesting food contaminated by a microorganism, toxins produced by that microorganism, or a naturally toxic food constituent.

The RDN should perform a complete nutrition assessment, including a nutrition-focused physical exam to look for physical symptoms and evaluate fat and muscle stores, and gather information about possible foods that trigger symptoms, including a description of recent symptoms, suspected foods, time of ingestion of those foods related to the symptoms, and the amount of food necessary to cause the reaction. For infants and children, information on prenatal history, early food exposures, and feeding practices is also helpful. Tools used and topics considered in MNT for food allergies and intolerances include the following bolded terms.

A **food symptom diary** is a useful tool for identifying food allergies and intolerances. It should be 7 to 14 days and include quantity and types of foods, meal timing, supplements, medications, and symptoms as well as stress level, physical exercise, and sleep patterns if possible

Food-elimination diets are used to diagnose and manage food allergies and intolerances. Suspect foods are eliminated for 4 to 12 weeks then re-introduced one at a time under careful monitoring. Elemental formulas and medical foods can be used to maintain nutritional adequacy if multiple foods are being eliminated from the diet.

Oral food challenges are done under medical supervision after symptoms have resolved and a patient is off anti-histamine medications. Foods may be presented openly, single-masked with a placebo, or double-masked with a placebo. Foods are often masked in another type of food and the quantity is increased until non-life-threatening symptoms manifest. When symptoms occur, medication is given and the patient continues under medical observation until they resolve.

Educating clients on the **avoidance of unsafe food** is an important part of MNT for food allergies. Total avoidance of the suspected foods is the only proven treatment for food allergy. Labeling laws require that food ingredients containing the Big Eight food allergens must be

clearly labeled. Clients should read labels regularly in case of recipe changes and inform staff at restaurants of their food allergies.

> Infants allergic to cow's milk or soy who are not exclusively breastfed can be fed partially hydrolyzed, extensively hydrolyzed, free amino-acid formula. Soy formula is not typically recommended for cow's milk allergy as the infant may also be allergic to soy. *You should know this for the exam.*

Gut healing and immune balance is an important nutrition goal for MNT for food allergies and intolerances. Seventy percent of immune cells are in gut-associated lymphatic tissue and damage to this tissue can lead to further immune issues. Measures to promote gut healing and restore immune balance include optimizing stomach acidity, adequate enzyme function, restoring gut microbiota levels, and repleting nutrition stores.

Nutritional adequacy is assessed on a regular basis by the RDN by monitoring food records, nutrition status, and growth (for infants, children, and adolescents) and weight (adults). Children with multiple food allergies are at higher risk of malnutrition and poor growth. The RDN should discuss the need for vitamin and mineral supplementation as well as strategies to help clients and families cope with food allergies to minimize disruption to the social aspects of eating.

HIV Infection and AIDS

Nutrition Therapy	MNT Goals
• Energy: maintain energy balance* • Protein: dependent on acute infection • Fluid: dependent on fever, vomiting, dehydration • Fat, fiber, and exercise to manage hyperlipidemia, lipodystrophy, and promote insulin sensitivity • Focus is on a high-quality complete diet *REE can be increased 5-17% in persons with HIV but activity levels can be decreased leading to a similar TEE to healthy individuals. Energy needs should be considered with regard to disease severity, stage, and secondary infection as well as effects of medications such as poor intake. On the flipside, the 12 months following antiretroviral therapy is associated with weight gain.[17]	• Restoration and maintenance of nutritional stores • Achieve healthy body weight and body composition • Support medication treatment goals while controlling/reducing symptoms • Prevent or delay advancement of HIV disease and onset of comorbidities (e.g. cardiovascular disease, diabetes) • Provide nutrition education related to food and water safety particularly those with low CD4 cell counts • Assess barriers to adequate and appropriate food intake and refer to relevant resources (e.g. food banks, meal delivery)

Recommended Reading
- Practice Paper: **Nutrition Intervention and Human Immunodeficiency Virus Infection**. J Acad Nutr Diet. 2018;118(3):486-498. PMID: 29477186. https://doi.org/10.1016/j.jand.2017.12.007

Human immunodeficiency virus (HIV) infection leads to immune system dysfunction. The disease process contributes to malnutrition and wasting with coinfection and inflammation leading to muscle wasting and additional nutritional risk. Malnutrition can worsen immune

suppression. The body's hormonal response to infection leads to changes in hormonal sensitivity that alter tissue catabolism and nutrient metabolism and can affect appetite and food intake.[18]

With successful medications that help prevent the progression of HIV disease to AIDS, many individuals with HIV are living longer but at higher risk for comorbidities such as cardiovascular disease and diabetes. Furthermore, while HIV medications key to viral suppression require adequate food and fluid intake to process, they can also have side effects that affect nutrient intake, such as nausea and vomiting. The RDN treating patients with HIV should be familiar with the interplay between HIV medications and effects on nutritional adequacy.

HIV can cause metabolic abnormalities including hyperlipidemia and insulin resistance. As part of the nutrition assessment, the RDN should evaluate not only CD4 cell count and viral load but also inflammatory markers such as CRP, lipids (cholesterol, LDL, HDL, triglycerides), fasting glucose and insulin, liver enzymes, BUN, creatinine, as well as anemia markers.[19] Food- and nutrition-related history is very important in the nutrition assessment of a patient with HIV, specifically food and nutrient intake, food and nutrition knowledge, beliefs, and attitudes, social network, psychosocial and economic factors, and access to food and nutrition programs are important.[20] The RDN should assess for food security as well as access to safe food and water, as individuals with HIV with low CD4 count are considered a vulnerable population with regards to food and water safety. Specifically, patients with HIV should avoid raw eggs, unpasteurized cheese, uncooked seafood including sushi, and raw/rare/undercooked meat.[21]

Malnutrition

Recommended Readings

- White JV, Guenter P, Jensen G, et al. Consensus statement: Academy of Nutrition and Dietetics and American Society for Parenteral and Enteral Nutrition: characteristics recommended for the identification and documentation of adult malnutrition (undernutrition). J Parenter Enteral Nutr. 2012;36(3):275-283. doi:10.1177/0148607112440285. PMID: 22535923 PDF: https://bit.ly/3sUdpLF

- Bharadwaj S, Ginoya S, Tandon P, et al. Malnutrition: laboratory markers vs nutritional assessment. Gastroenterol Rep (Oxf). 2016;4(4):272-280. doi:10.1093/gastro/gow013 PMID: 27174435. Full text at PMC (PMC5193064)

The Malnutrition Quality Improvement Initiative (MQii) is a collaboration between the Academy, Avelere Health, and other stakeholders to advance evidence-based malnutrition care across the US. The initiative resulted in creating of 4 electronic clinical quality measures (eCQM) that CMS uses in quality incentive programs and publicly reported quality data. Feasibility testing of the 4 eQMs occurred in 3 health systems and 3 EHR vendors. Validity and reliability testing took place in 2 health systems. The Journal of the Academy of Nutrition and Dietetics published an entire supplement issue focused on the MQii (https://jandonline.org/issue/S2212-2672(19)X0003-9#) Peruse the Table of Contents and read any articles that jump out at you.

The 4 eCQMs developed were:

1. Completion of malnutrition screen within 24 hours of admission
2. Completion of nutrition assessment for patients identified as “at risk” within 24 hours of screening
3. Nutrition care plan for patients identified as “malnourished” after a complete nutrition assessment
4. Appropriate documentation of a malnutrition diagnosis

In the clinical setting, albumin, prealbumin, and CRP lab test results were previously used to assess for malnutrition. Per the Nutrition Care Manual, the use of these metrics is no longer recommended to determine nutrition status.[22] Albumin, prealbumin, and CRP are negative acute phase reactants and thus typically abnormal in the hospital patient. Levels can also be altered due to fluid overload. CRP may be chronically elevated in patients with chronic disease, independent of nutritional status. However, you should be aware that these measures have previously been used and recognize that if you take the RD exam prior to its update, you may come across questions regarding these values. You should still be familiar with what these lab values reflect and that prealbumin was preferred over albumin for its shorter half-life.

What does the term “negative acute phase reactants” mean? Download and plan to fill out the free Lab Values worksheet if you have not done so already.

Malnutrition can coexist with other medical conditions that warrant biochemical evaluation. The RDN should examine lab results for acid-base balance, electrolytes, glycemia, inflammation, lipids, anemia, and renal function if warranted in the context of the patient’s medical condition and physical findings.[22]

Conducting a nutrition-focused physical exam is important when evaluating for malnutrition and determining the severity of malnutrition.[23] The RDN should look for loss of adiposity, muscle atrophy, abnormal bone prominence, edema, markers of micronutrient deficiency and dehydration, skin symptoms, wound healing and ask about GI symptoms.[24] See Table 7.The RDN should also review the physician’s exam for abnormalities in blood pressure, heart rate, and respiratory rate.

The table on the next page lists nutrition-focused physical findings that may be present with malnutrition (adapted from the Nutrition Care Manual). Use your class resources, textbooks, or online sources to define them as well as make notes of why they would be noticed in malnutrition and their significance. You can download the Malnutrition Worksheet for free at www.studysmartermethod.com/freebies for a printable version. Some of the items, such as location of bony prominence of loss of adiposity, do not need defining but rather you should be aware of them as things to look for in a nutrition-focused physical exam. Some findings will also be present in other conditions, such as peripheral edema in a heart failure patient. Note when other causes of a physical finding should be ruled out and what conditions would be ruled out. When you finish defining each physical finding, reorganize them. For example, identify physical findings that point to vitamin A deficiency and group them on a separate sheet of paper. Repeat for iron-deficiency anemia, etc. What findings point to essential fatty acid deficiency? Protein-energy malnutrition versus energy malnutrition? What measures would you look at in an infant or child rather than an adult? *These are active learning strategies that are explained extensively as part of the Study Smarter Method in the e-book available at www.studysmartermethod.com. If you would like to learn how to study for the RD exam both effectively and efficiently and apply these methods to other substantive areas covered on the exam, you can go online to purchase the book.*

Table 7. Nutrition-Focused Physical Exam Findings

Body Habitus and Muscles
Atrophy of orbital fat
Loss of subcutaneous fat over triceps, biceps, ribs
Abnormal bony prominence(s)
Muscle atrophy (general, specific)
Anasarca
Pitting edema

Hair	
Dry, brittle, sparse	Lanugo

Mouth	
Ageusia, dysgeusia	Beefy red tongue
Angular stomatitis (cheilosis)	Atrophy of tongue papillae
Oral candidiasis	Dry tongue
Cracked lips	Glossitis
Dry mucus membranes	Strawberry tongue
Hemorrhagic gingivitis	Macroglossia
Pale gums	Cracked tongue
Dental caries	Magenta tongue
Poor dentition	Pale tongue

GI System	
Abdominal distension	Diarrhea
Constipation	Poor appetite

Eyes	
Angular blepharitis	Night blindness
Bitot's spots	Pale conjunctiva
Keratomalacia	

Hands and Nails	
Beau's lines	Leukonychia
Flaking of nails	Muehrcke's lines
Trachyonychia	Koilonychia (spooning)
Nail clubbing	Russell's sign
Nail pitting	

Skin	
Decreased skin turgor	Petechiae
Dry skin	Pruritus
Ecchymosis	Purpura
Hyperpigmentation	Pellagrous dermatosis
Poor wound healing	

Vital Signs	
Hypotension	Bradypnea
Bradycardia	Arrhythmia

Some medical conditions can predispose patients to high nutritional risk and malnutrition. Furthermore, malnutrition can severely worsen prognosis in these conditions. Chronic medical conditions associated with malnutrition include Crohn's disease, short gut syndrome, cancer, pulmonary disorders including cystic fibrosis and COPD, cardiac disorders, and eating disorders.[25] You should also ask about surgical procedures, as some affect oral intake due to postoperative GI symptoms or changes to the GI tract. Surgeries to look for include cancer surgeries (i.e. gastrectomy due to stomach cancer), GI surgery (i.e. removal of bowel), trauma-related or injury-related surgery, and bariatric surgery.[25] Finally, assess medication use and previous medical interventions such as radiation and chemotherapy.[25]

Per the Nutrition Care Manual, there are 3 types or etiologies of adult malnutrition: starvation related, chronic disease related, and acute disease related.[26,28] All are marked by inadequate intake of protein and/or energy for a long enough period of time to result in the loss of fat and/or muscle mass. They differ in the degree of inflammation present and context. The context for chronic disease-related and acute disease-related malnutrition is clear. Context for starvation-related malnutrition may be due to environmental and/or social circumstances, such as food insecurity or knowledge and beliefs. Context for chronic disease-related malnutrition may be due to increased energy/nutrient needs with or without reduced intake. Finally, acute disease (or injury)-related malnutrition may occur due increased energy/nutrient needs due to a systemic inflammatory response, poor intake, and/or altered GI function (such as after GI surgery or due to a medical condition).

Table 8. Types of adult malnutrition

Malnutrition Type	Degree of Inflammation	Example	Example Context
Starvation-related	No apparent inflammation	Chronic starvation; anorexia nervosa	Depression; chronic alcohol abuse; food insecurity
Chronic disease related	Mild to moderate inflammation	Organ failure; pancreatic cancer; rheumatoid arthritis	COPD; cancer
Acute disease related	Marked inflammatory response	Major infection; burns; trauma; closed head injury; SIRS	Altered GI tract function (surgical or medical); increased nutrient needs

Adapted from Nutrition Care Manual, accessed 15 May 2021,[26]Jensen GL et al 2009,[27] Jensen G et al 2010.[28]

Protein-Calorie Malnutrition

Protein-calorie malnutrition, also called protein-energy malnutrition, is an umbrella term for malnutrition that results from poor intake of protein, calories, or both. It is a type of undernutrition and is considered to be *acute*. Kwashiorkor and marasmus are severe types of protein-calorie malnutrition.

Kwashiorkor

Kwashiorkor, or protein-deficiency malnutrition, occurs when a patient consumes sufficient energy but insufficient protein. The name means “the sickness the baby gets when the new baby comes”, and it often manifests at weaning age when the older child’s diet changes from breast milk to a high-carbohydrate diet poor in protein. Kwashiorkor is rare in developed nations, and prevalence in developing countries, particularly Southeast Asia, Central America, Puerto Rico, Jamaica, South Africa, and Uganda, rises during times of famine.[29]

Depressed visceral protein alters fluid stores, resulting in pitting peripheral edema. Abdominal distension is caused by hepatomegaly due to fatty liver. Other physical findings include muscle atrophy, rounded face, thin and dry skin with scaling and hyperpigmentation, growth retardation, as well as anorexia (poor appetite) and apathy.[29] The child’s immune system is severely compromised, and death is typically due to diarrhea, dehydration, or infectious disease.

Treatment includes treating or preventing electrolyte imbalances, dehydration, hypothermia, infection, micronutrient deficiencies while starting cautious feeding with the goal of achieving catch-up growth.[29] Fluid imbalance is corrected using ReSoMal (Rehydration Solution of Malnutrition) that differs from normal saline (which lacks potassium and has too much sodium).[29] Adequate follow-up is necessary for recovery and sensory stimulation and emotional support are important for the child’s recovery.[29]

Marasmus

Marasmus, or deficiency of both protein and energy, results in a wasted appearance and depresses somatic and visceral protein stores. The patient will have a low weight, muscle wasting, and low body temperature and/or cold intolerance.

Marasmus can occur in children and adults. The precipitating factors typically differ by age. For children, growth retardation can be as severe as weight-for-height 3 SD below age-sex values.[30] In adults, wasting can manifest as a low body weight, low BMI, as well as physical findings such as muscle wasting and loss of adipose stores. A patient with marasmus may also develop protein insufficiency, a condition called marsamic-kwashiorkor or marasmus-kwashiorkor mix.

Treatment involves resuscitation and stabilization, nutritional rehabilitation, and follow-up to monitor recovery and prevent recurrence (Table 9). Review the information on refeeding syndrome in the Eating Disorders section.

Table 9. Treatment and management of marasmus

Treatment Stage	Goals	Duration
Resuscitation and stabilization	Rehydrate, prevent infection, avoid refeeding syndrome	~1 week
Nutritional rehabilitation	Gradual increase in caloric intake, increased motor activity	2 – 6 weeks
Follow-up	Provide nutrition education, connect with community resources, monitor nutrition status	Ongoing

Adapted from Titi-Lartey and Gupta, 2021.[30]

Failure-to-thrive

Failure-to-thrive refers to inadequate intake or utilization of energy/nutrients in infants, children, and the elderly. If you haven't already, read the 2 position papers on malnutrition in pediatric and adult populations.

Please read the following two papers:

- Consensus Statement of AND/ASPEN: **Indicators Recommended for the Identification and Documentation of Pediatric Malnutrition (Undernutrition).** Nutrition in Clinical Practice. 2015. 30(1). PMID: 25422273. Free access at https://doi.org/10.1177/0884533614557642
- Position of the Academy of Nutrition and Dietetics**: Malnutrition Screening Tools for All Adults**. J Acad Nutr Diet. 2019. PMID: 31866359. https://doi.org/10.1016/j.jand.2019.09.011

Inborn Errors of Metabolism

While newborn screening varies from state to state, most states screen for the following metabolic diseases: phenylketonuria (PKU), methylmalonic acidemia (MMA), maple syrup urine disease (MSUD), citrullinemia, tyrosinemia, and medium chain acyl-CoA dehydrogenase (MCAD) deficiency.

Maple syrup urine disease (MSUD), or branched-chain ketoaciduria, is an autosomal recessive disorder that results from a defect in the branched chain α-ketoacid dehydrogenase enzyme complex.[31] Defects in these enzymes prevent metabolism of branched-chain amino acids (leucine, isoleucine, and valine). By 4 or 5 days after birth, the infant will have poor feeding behaviors, vomiting, lethargy, periodic hypertonia, and by 7 days, a sweet odor from urine and perspiration. Failure to treat MSUD leads to acidosis, neurologic damage, seizures, coma, and death. Acute management involves peritoneal dialysis and hydration. Plasma leucine levels are monitored in infancy and childhood with high leucine levels associated with acute infections. Incidence is around 1 in 200,000 infants.

Methylmalonic acidemia (MMA) is an organic acidemia disorder characterized by high levels or organic acids in the urine and high plasma ammonia levels. Several enzymatic defects can lead to MMA but the most common is a defect in methylmalonyl-CoA mutase apoenzyme. Low protein intake during infancy can be achieved by diluting standard formula to decrease protein content and adding a low protein formula. Specialty formulas low in threonine and isoleucine and acing methionine and valine may also be used. The child's growth rate, health, enzyme activity, energy intake, and protein intake should all be monitored. Medical treatment may include pharmacologic doses of vitamin B12, which is a cofactor for methylmalonyl-CoA mutase. Incidence is around 1 in 80,000 infants. Adequate fluid to correct electrolyte imbalances and to promote urinary excretion of abnormal metabolites is essential. IV fluids may be used. Metabolic acidosis episodes can occur due to high protein intake, infection, constipation, or other factors.

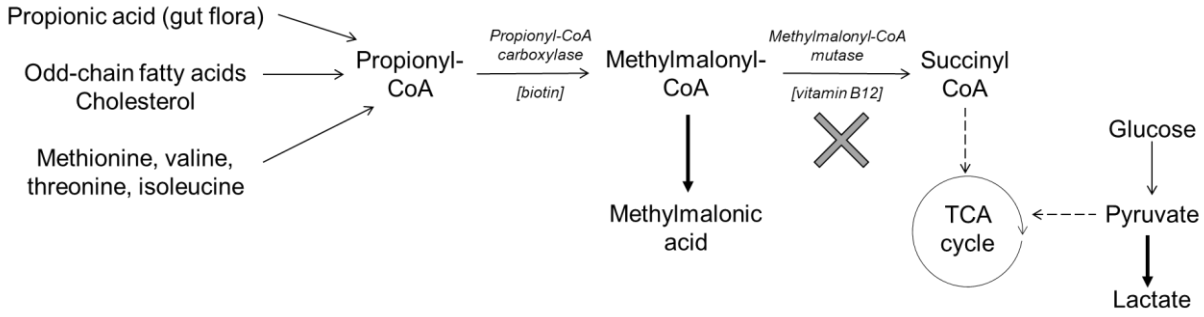


Figure 1. Simplified depiction of what goes wrong with methylmalonic acidemia (MMA). A defect in methylmalonyl-CoA mutase means that methylmalonyl-CoA cannot be adequately converted to succinyl CoA. Accumulation of methylmalonyl-CoA increases conversion to MMA, and on the other side of the TCA cycle, because pyruvate isn't being pulled into the TCA cycle, conversion to lactate increases. Bold arrows indicate an increase in conversion. Dashed arrows indicate a decrease. Image created by Bailey DeBarmore. Do not reproduce without permission.

Citrullinemia is a disorder of urea cycle metabolism with a deficiency in the enzyme argininosuccinic acid synthetase.[31] This enzyme metabolizes citrulline to argininosuccinic acid and thus a defect in the enzyme results in high levels of citrulline in the blood, which is used to diagnose the disorder. Other urea cycle metabolic disorders include ornithine transcarbamylase deficiency (OTC), argininosuccinic aciduria (ASA), and carbamyl-phosphate synthetase (CPS) deficiency. The incidence of all urea cycle metabolic disorders is 1 in 30,000 infants. All urea cycle metabolic disorders result in ammonia accumulation in the blood. Symptoms of high blood ammonia in infants includes poor feeding and recurrent vomiting. If untreated, symptoms progress to seizures, neurologic abnormalities, and coma. Medical nutrition therapy includes a low protein diet and formula with only essential amino acids.

Tyrosinemia type 1 is an amino acid disorder, like phenylketonuria, but involves a defect in the fumarylacetoacetate hydrolase enzyme.[31] Incidence is estimated at between 1 in 100,000 and 1 in 120,000 infants worldwide. Incidence is higher in Quebec, Canada (1 in 16,000 infants) and Norway (1 in 60,000 births).[32] Symptoms include vomiting, acidosis, diarrhea, failure to thrive, hepatomegaly, rickets (due to renal Fanconi syndrome), and high levels of tyrosine and methionine in the blood and urine. Medical nutrition therapy includes a low protein diet and specialty formulas lacking tyrosine, phenylalanine, and methionine. The buildup of tyrosine and its metabolites can lead to severe liver disease and liver cancer, kidney dysfunction, and central

nervous system dysfunction. Tyrosinemia type II and type III involve different enzymes and do not have liver abnormalities.

Medium Chain Acyl-CoA Dehydrogenase (MCAD) deficiency is a fatty acid oxidation disorder. If not identified via newborn screening, infants and children will present during fasting or illness with failure to thrive, episodic vomiting, and hypotonia.[31] They present with hypoglycemia but no urinary ketones. Hypoglycemia can progress quickly and be fatal. Treatment involves avoidance of fasting and in some cases, a low-fat diet (<30%) as fats are not well metabolized. The incidence is about 1 in 20,000 infants. There are also long chain, short chain, and very long chain acyl-coA dehydrogenase deficiencies. They are very rare.

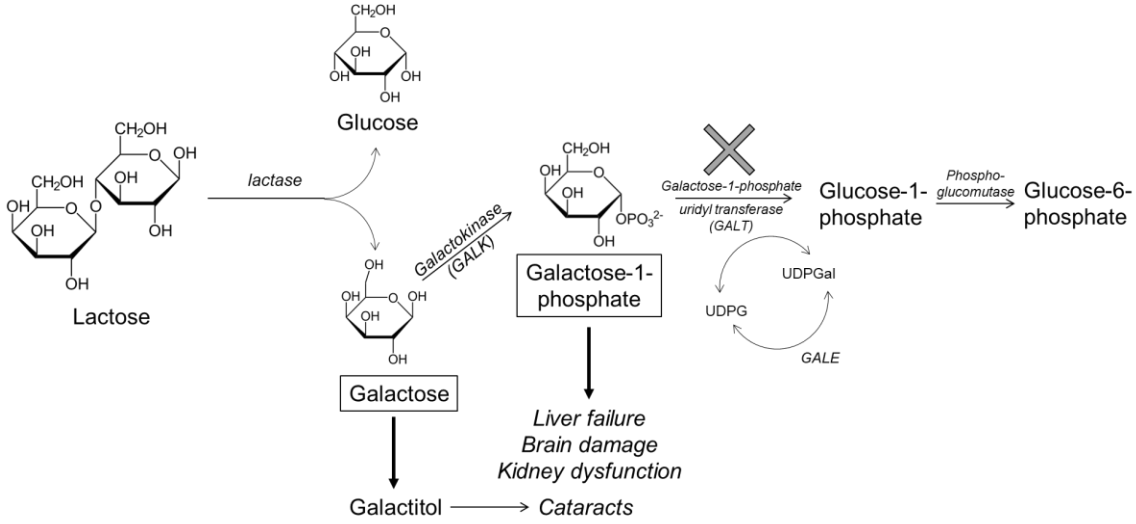


Figure 2. Simplified depiction of galactose metabolism with blocks at GALT, resulting in an accumulation of galactose and Gal-1-P (boxed) which leads to increased galactose → galactitol conversion (bold arrow) and complications (italicized). Image created by Bailey DeBarmore. Do not reproduce without permission.

Galactosemia is a disorder of carbohydrate metabolism and can present as seizures and sepsis in the newborn.[31] High levels of galactose-1-phosphate in the blood, galactose in the urine (galactosuria), and low levels of galactose-1-phosphate uridyl transferase (GALT) are used to diagnose the disorder. Because galactose metabolism cannot proceed normally, galactose and galactose-1-phosphate accumulate in body tissues (Figure 2). Increased galactose concentrations result in conversion to galactitol which accumulates in ocular tissues leading to cataracts. Accumulation of galactose-1-phospahte lead to hepatomegaly, liver failure, kidney dysfunction, and neurologic damage. Other symptoms include vomiting, diarrhea, lethargy, failure to thrive, and jaundice. Early treatment can result in normal physical and motor development but intellectual development may still be impaired. Treatment includes soy formula, because human breast milk and cow's milk contain galactose. Food remains low protein after weaning. Dysfunction in galactokinase (GALK) and uridine diphosphate glactose-4-epimerase (GALE) can lead to similar symptoms but are not as common as GALT dysfunction.[33]

Glycogen storage diseases occur when the body cannot metabolize glycogen into glucose, or cannot correctly form glycogen from glucose.[31] There are different types that correspond with several possible enzyme defects. Types I, III, and IV are the most common (Figure 3).

Symptoms include poor growth, hypoglycemia, hepatomegaly, and downstream effects of poor glycogen metabolism such as abnormal cholesterol and triglyceride levels.

Glycogen storage disease Type I (GSD1), also called von Gierke disease, is an autosomal recessive disorder with altered function of the glucose-6-phosphatase enzyme. The body cannot break down glycogen into glucose, resulting in excessive glycogen and fat storage in the liver and kidneys as well as metabolite accumulation (lactate, uric acid, lipids, triglycerides). Symptoms manifest around 3-4 months of age and include hypoglycemia, hepatomegaly, nephromegaly, elevated metabolite levels in the blood, and seizures due to hypoglycemia.[34] Regular episodes of hypoglycemia leads to poor growth (short stature), muscle weakness, and brain dysfunction. Nutrition therapy may consist of a diet low in lactose, fructose, and sucrose, low in fat, and high in complex carbohydrates. Cornstarch may be used as a supplement and continuous enteral feeds may be used overnight. Fasting should be avoided, as both glycogenolysis and gluconeogenesis is impaired.

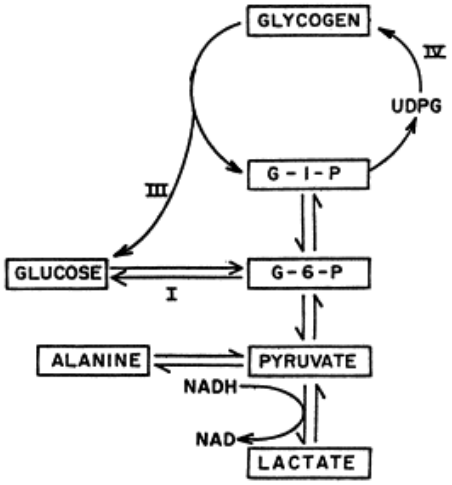


Figure 3. Simplified depiction of where glycogen enzyme defects occur in GSD Type I, Type III, and Type IV. Adapted from Goldberg T and Slonim AE. Nutrition Therapy for Hepatic Glycogen Storage Diseases. JADA. 1993. 93(12):1423-1430. https://bit.ly/2R0VpBK

Glycogen storage disease Type III (GSD3), also called Cori disease or Forbes disease, is caused by a defect in the glycogen debranching enzyme. Symptoms are very similar to those of GSD1 and include excessive glycogen storage in the liver and muscles, hypoglycemia, failure to thrive, and recurrent infections and during the first few years of life.[35] Ketone levels are high during fasting with GSD3, in contrast to GSD1, and ketonemia may precede hypoglycemia. Dietary management can be used to improve symptoms, though liver cirrhosis and carcinoma can occur in adulthood. Nutrition therapy for infants and children includes small frequent feedings of complex carbohydrates and protein (avoiding simple sugars) to prevent fasting and possibly continuous enteral feeds for overnight.[36] Cornstarch supplementation may be used.

Glycogen storage disease Type IV, also called Andersen disease, is caused by a defect in the glycogen branching enzyme (GBE) which causes glycogen to be stored in an abnormal form in the liver, muscle, and other tissues. Symptoms appear in the first few months and include failure-to-thrive and hepatosplenomegaly. Progressive hepatic cirrhosis and liver failure by age 5 can occur.[37]

To learn more about glycogen storage diseases, you can review this article (yes, it is old, but it is still relevant): Goldberg T and Slonim AE. Nutrition Therapy for Hepatic Glycogen Storage Diseases. JADA. 1993. 93(12):1423-1430. https://bit.ly/2R0VpBK PMID: 8245377

Phenylketonuria, or PKU, is a protein metabolism disorder resulting in an increase in the amino acid phenylalanine (PHE) in the blood. PKU is part of mandatory newborn screening in all 50 states and if treatment is started right away, much of the damage can be avoided. The incidence of PKU is estimated at around 1 in 10,000 persons of European descent, with most mutations occurring in the phenylalanine hydroxylase gene.[38] This enzyme is responsible for catalyzing PHE to tyrosine (Figure 4). When the body digests protein, PHE is absorbed. If it cannot be further catalyzed, it builds up in the blood and leads to structural and functional brain damage.

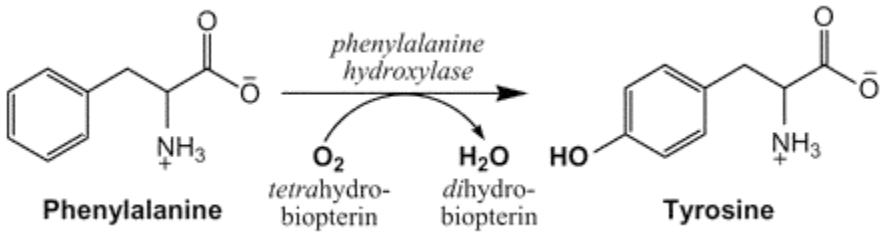


Figure 4. Conversion of phenylalanine to tyrosine via phenylalanine hydroxylase using cofactor BH4 (tetrahydrobiopterin). The treatment Kuvan is a form a BH4 that, in low doses, can increase the conversion of PHE to TYR and help manage blood PHE levels.

Nutrition Therapy[38]	**MNT Goals**[38]
Calculate: • total energy intake and percentage of energy from formula and medical foods • total protein intake and percentage of protein from formula and medical foods • PHE intake from foods • tyrosine intake from formula and medical foods • calcium and iron intake from formula and medical foods Compare calculated PHE intake to blood PHE levels and adjust intake to bring into treatment range	• Daily PHE restriction through use of medical PHE-free formula, specialty low-PHE foods, and some natural foods low in PHE • Ensure adequate energy, protein, and micronutrient intake while still meeting PHE restrictions • Monitor progress by growth trajectory, serum PHE, and serum tyrosine levels • Individualize nutrition therapy and tailor nutrition education to the patient

Treatment includes low protein diets and specialty medical foods that lack PHE. While limiting protein can keep blood PHE levels low enough to manage symptoms, low protein diets interfere with normal growth and development.[39] Medical PHE-free formulas are used to meet 75% of protein and essential nutrient needs and are supplemented by special low-PHE foods to meet energy needs.[39] These specialty products are fortified with L-tyrosine. Patients with PKU should be seen by specialty RDNs. Foods to avoid on the PKU diet include cheese, other dairy, nuts, seeds, dried beans and peas, peanut butter, eggs, poultry, meat, fish, and other seafood.[39] Foods that are allowed in small amounts (and must be monitored and measured) include breads, crackers, potato chips, popcorn, fruits, vegetables, juices, and special low-protein foods and cereals.[39]

Why would specialty products for PKU be fortified with tyrosine? Why do patients with PKU need to avoid foods containing aspartame? What foods typically contain aspartame?

Cancer

Nutrition Therapy[40]	MNT Goals[41]
Energy • 25-30 kcal/kg (non-ambulatory, sedentary) • 30-35 kcal/kg (hypermetabolic, weight gain, anabolic, 1st month after stem cell transplant) • ≥35 kcal/kg (severely stressed, acute graft-vs-host disease, during head & neck chemoradiation, malabsorption) Protein • 1 – 1.2 g/kg (non-stressed with cancer) • 1.2 – 1.5 g/kg (undergoing treatment) • 1.5 – 2 g/kg (stem cell transplant) • 1.5 – 2.5 g/kg (increased protein needs [e.g. enteropathy or wasting])	• Prevent or reduce micronutrient deficiencies • Preserve lean body mass and prevent weight loss • Minimize effect of nutritional impact symptoms • Enhance immune function by decrease risk of infection • Maximize quality of life, independence, and ability to perform ADLs through use of food as comfort and improving nutritional status • Inform patients regarding alternative and complementary diets using active listening and a nonjudgmental approach

Cancer prevention centers on *promoting healthy behaviors* and avoiding or *reducing harmful behaviors.* Examples from the CDC are listed in Table 10. Once diagnosed, cancer is staged using the Tumor, Node, Metastasis (TNM) classification system that identifies the extent of the tumor, its size, and the degree of its growth (to lymph nodes) and spread (metastasis).[42] Staging is used to inform decision making.

Table 10. A summary of measures used to prevent cancer across a lifetime

Healthy Behaviors	Harmful Behaviors
• Eat a healthful diet rich in fruits and vegetables • Engage in regular physical activity • Maintain a healthy weight • Use sun protection outdoors • Breastfeed • Get sufficient sleep • Manage chronic diseases (e.g. diabetes) • Test for infections (e.g. HCV, HPV)	• Avoid tobacco and secondhand smoke • Avoid intentional tanning • Limit alcohol consumption (<2 drinks/day for men, <1 drink/day for women) • Limit consumption of energy-dense, nutrient-poor foods and sugar-sweetened beverages • Avoid exposure to certain chemicals • Limit radiation doses during medical imaging

Adapted from "Preventing Cancer Across a Lifetime", Centers for Disease Control and Prevention. Accessed 16 May 2021.[43]

Cancer Treatment

As you work through this section, take out a sheet of paper and list the nutrition-related adverse effects of cancer treatment (also called nutrition impact symptoms). Define them if you are not familiar with the term. Next to them, list nutrition interventions that could be used. For the exam, you should be aware of the complex nature of cancer treatment and its nutrition-related adverse effects and you should be familiar with common nutrition interventions to address them. If you go on to certify as an oncology RD, you will be required to know additional information.

Cancer treatment can be intended to cure cancer, control cancer, or to be palliative.[42] Types of treatment include chemotherapy, hormonal therapy, biotherapies, radiation therapy, stem cell transplant, and surgery.

Chemotherapy is drug-based treatment. Some sources classify hormonal therapy and biotherapy as types of chemotherapy. The most common type of therapy referred to as chemotherapy is cytotoxic therapy. Cytotoxic therapy targets cells with high replication rates, which includes normal body cells like those in the gut and hair cells as well as tumor cells.[44] The severity and impact of chemotherapy is dependent on the duration and dose of the treatment and the specific agent or agents used.[42]

Hormonal therapy is used to target hormone-sensitive cancers. It blocks the tumor's ability to use endogenous hormones and reduces levels of hormones in the body that the tumor would otherwise use to grow.[42]

Biotherapies include immunotherapies and targeted therapies. Immunotherapy molecules include interferon, interleukin, and hematopoietic growth factors that work to restore the body's immune function. [44] Targeted biotherapies 'target' the cancer cells while sparing normal tissue and include monoclonal antibodies, signal-transduction inhibitors, and proteasome inhibitors. For example, monoclonal antibodies mark cancer cells for eradication, block tumor growth signals, or deliver radiation/drugs to the tumor.[42]

Radiation therapy causes localized effects to the specific area of the body being irradiated. [42] Radiation therapy may be curative, palliative, or prophylactic and is typically used along with other therapies. Ionizing radiation lyses cells, damaging cell structure, and preventing further growth. Patients with cancer of the head and neck and cancer of the GI tract are particularly susceptible to nutrition-related adverse effects following radiation therapy.[44]

Stem cell transplant involves conditioning the patient with high doses of chemotherapy and irradiation followed by a transfusion of healthy stem cells from the patient or a donor. Parenteral nutrition is often used to meet nutrition needs. [42] An **autologous transplant** is when cells are taken from the patient's bone marrow prior to chemotherapy and then replaced after conditioning. [44] **Allogenic transplant** involves harvesting and using stem cells from a donor who is a close match to the patient.[44]

Surgery is used for solid tumors and may be curative, palliative, or used to debulk the tissue prior to adjuvant treatment.[42] For example, a patient may receive neoadjuvant (preoperative) chemotherapy prior to surgery, and/or adjuvant (postoperative) chemotherapy with or without radiation.

It is important for the oncology RDN to anticipate nutrition-related adverse effects of treatment modalities and to prescribe nutrition interventions aimed at maintaining or restoring nutritional status. Poor nutritional status may adversely affect treatment because the patient may require a reduction in therapy dose or delays in treatment.[42] Cancer patients typically receive multiple therapies.

Symptoms and adverse effects that can impact nutrition include: anemias, neutropenia, leukopenia, thrombocytopenia, electrolyte imbalances, dehydration, constipation, diarrhea, malabsorption, dysphagia, stomatitis, mucositis, esophagitis, nausea/vomiting, anorexia, early satiety, dysgeusia, ageusia, xerostomia, fatigue, weight loss, pain, hepatic toxicity, renal toxicity,

neuropathy, headache, myalgia, dysosmia, fluid retention, and capillary leak syndrome (Table 11).[44]

Table 11. Nutrition Impact Symptoms Common in Patients Receiving Cancer Treatment

• ageusia	• early satiety	• myalgia
• anemias	• electrolyte imbalances	• nausea/vomiting
• anorexia	• esophagitis	• neuropathy
• capillary leak syndrome	• fatigue	• neutropenia
• constipation	• fluid retention	• pain
• dehydration	• headache	• renal toxicity
• diarrhea	• hepatic toxicity	• stomatitis
• dysgeusia	• leukopenia	• thrombocytopenia
• dysosmia	• malabsorption	• weight loss
• dysphagia	• mucositis	• xerostomia

Palliative Care and Hospice

Nutrition goals for advanced cancer and palliative care are focused on improving quality of life rather than reversing malnutrition and weight loss.[41] A liberalized diet, use of food as a comfort, and management of nutritional impact symptoms are example nutrition therapy goals.

Hospice is a care concept "designed to provide comfort and support to patients and families when a life-limiting illness no longer responds to cure-oriented treatments."[45] Nutrition therapy goals in hospice include improving quality of life, preserving dignity, controlling pain, and providing comfort. [42] Patients may prefer soft foods and clear liquids, and eating should be focused on pleasure rather than meeting nutrition needs.[42]

The goal of palliative care and hospice are both pain relief and symptom relief, however, they differ in the prognosis and treatment goals. Palliative care can occur at any stage of the disease and is comfort care at the same time as curative treatment while hospice services excludes curative treatment and is focused on comfort care alone.[46]

Nutrition-Related Adverse Effects

For the RD exam, you should be familiar with common nutrition-related symptoms and adverse effects of cancer treatment as well as general nutrition interventions to address them, such as small frequent meals, altering food composition or presentation to alleviate diarrhea, dysgeusia, and dry mouth, and recognize the types of nutrition education that the oncology RD would provide to a cancer patient and/or caregiver. An example question you may encounter is "Which of the following would be an appropriate nutrition intervention for a patient with dysosmia?"

Anorexia and Cachexia

Cancer-related anorexia/cachexia syndrome (CACS) is defined as anorexia and weight loss of both muscle mass and fat tissue.[47] Anorexia means a loss of appetite, or poor appetite. While cancer patients may have trouble meeting their nutrition needs orally, merely meeting nutrition

needs does not restore lean muscle mass with CACS (in contrast to normal starvation which is reversible with feeding).[48] The underlying mechanisms of CACS are complex. CACS is most common in patients with solid tumors, GI tract cancers, and patients with advanced cancer. In particular, cancer of the lung, head and neck, and pancreas are associated with CACS.[49]

Anorexia (a decrease or loss of appetite) can result due to GI tract obstruction, nausea/vomiting, dysgeusia, dysosmia, food aversions, pain, anxiety, and/or depression.[50] Some patients may be treated with pharmacologic agents to stimulate appetite, but none are FDA-approved specifically for CACS. Progesterone analogs (such as megestrol acetate and medroxyprogesterone acetate) and glucocorticoids (such as dexamethasone and prednisolone) are well-studied, however the side effects are not insignificant.[47] Side effects of progesterone analogs include thromboembolic events and edema/fluid retention.[47] Side effects of long-term glucocorticoid use include immunosuppression, hypertension, osteoporosis, adrenal suppression, myopathy, Cushing's syndrome, peptic acid disease, and induced hyperglycemia. [47,51]

Evidence is inconclusive for olanzapine, androgens and selective androgen receptor modulators (such as oxandrolone), growth hormone, ghrelin analogs, cyproheptadine, thalidomide, and mirtazapine.[47] Synthetic cannabinoids (such as dronabinol), TNF inhibitors (such as pentoxifylline, etanercept, infliximab), hydrazine sulfate, insulin, and melatonin have sufficient evidence to recommend against their use to treat CACS.[47]

The RDN should recognize appetite-stimulating medications used in patients with CACS. Nutrition interventions are typically used to achieve short-term benefits like improved appetite, increased oral intake, and a better sense of well-being rather than directly affecting the underlying causes of CACS or curing the cancer.[52] Sample nutrition interventions include frequent small meals of energy-dense food and fluids, creating a relaxing and pleasant environment at meal time, liberalizing any diet restrictions if medically feasible, use of easy-to-prepare meals and snacks, encouraging light physical activity with the goal of increasing appetite, addressing other causes of anorexia such as depression, constipation, or other medications, and finally, considering nutrition support if indicated.[52]

Fatigue

Cancer-related fatigue is a distressing and persistent sense of fatigue that may be physical, emotional, and/or cognitive and the patient may express it as 'tiredness' or 'exhaustion'.[53] It interferes with usual functioning and the degree of fatigue is not proportional to recent activity.

The RDN should recommend nutrition interventions that include ready-to-serve items or foods with minimal preparation, energy-dense foods that are easy to chew, and small frequent meals and snacks.[54] The largest meal should be eaten when both energy and appetite are at the best and the patient may want to keep snacks and beverages nearby for easy access, such as by their bed or favorite chair.

Constipation and Diarrhea

Nutrition recommendations for constipation that include fiber content in the diet can be difficult if the patient is also experiencing poor appetite and early satiety.[55] Rather than choosing high-fiber foods, the patient might instead consider fiber supplements (contraindicated in patients with potential bowel obstruction). The RDN can also recommend increasing fluid intake and over-the-counter gas and bloating medications, such as simethicone, and light exercise if tolerated to relieve gas accumulation.[55]

The RDN can assess for foods that may be contributing to diarrhea such as fatty foods, foods high in simple carbohydrates and sugar alcohols, caffeine, alcohol fiber, and/or dairy items and provide nutrition education on replacement foods for these.[56] Depending on severity of symptoms, the RDN may provide education on a clear liquid diet with gradual advancement to a low-fiber diet.[56] Banana flakes, apple powder, and other pectin foods can be eaten to thicken stool consistency.[57] The RDN should also provide education on the importance of restoring fluid balance, such as consuming 1 cup of water per loose stool movement or if warranted, the use of oral rehydration solutions.[56,57]

Mucositis and Stomatitis

Mucositis is the inflammation, irritation, and ulceration of the GI tract including the oral cavity and esophagus.[58] Mucositis of the mouth and lips is called stomatitis. Mucositis of the esophagus is esophagitis. Mucositis itself can produce additional nutrition-related symptoms including oral lesions, erythema, xerostomia, oral candidiasis, dysphagia, and odynophagia.

The RDN can advise the patient and/or caregivers on foods and liquids that limit pain from mucositis. These options include nutrient-dense, soft, moist, and easy-to-chew foods while avoiding acidic foods, spicy foods, and alcohol. Foods should not be too cold or too hot.[59] Finally, the patient should maintain good oral care with a soft tooth brush and control pain with pain medication or analgesic mouth rinses. The goal is to avoid the need for nutrition support.

Nausea and Vomiting

Nutrition intervention for nausea and vomiting is supportive (not curative) and specific interventions depend on the cause and severity of the nausea and vomiting, antiemetics prescribed by the physician, and whether the patient is currently undergoing chemotherapy.[60] The RDN can provide education on progressing from a clear liquid diet to bland foods as long as vomiting is controlled. Food suggestions include low-fat, non-odorous foods and frequent small amounts of dry food throughout the day.[60]

Neutropenia

Neutropenia is a low neutrophil count and is common during chemotherapy and classifies patients as a ‘vulnerable population’ with regards to food safety. Neutropenia increases the risk of serious life-threatening infections, particularly when coupled with impairment of other protective barriers such as the skin, GI tract, and other aspects of the immune system. The RDN should provide education on food safety to patients with neutropenia or low WBC counts

including the importance of good hand washing practices, proper cooking and storage temperatures, and avoiding raw and undercooked meats/fish/shellfish/poultry/tofu/breakfast meats/eggs, unpasteurized milk products and juice, aged cheeses, sprouts, unwashed produce, and untested well water.[61]

Think about the benefits of trophic feeds for a stem cell transplant patient in regards to maintaining the gut barrier.

Dysgeusia and Dysosmia

Dysgeusia (taste alterations) and dysosmia (smell alterations) can result in the patient not meeting energy needs.[62] Some patients experience more intense tastes, some experience poor or little taste sensing (ageusia), or a metallic taste. The RDN can recommend avoiding caffeine, chocolate, red meat, and citrus fruits and juices as these are common food aversions to patient with dysgeusia. Zinc deficiency and niacin deficiency, as well as poor oral hygiene, may also contribute to dysgeusia and dysosmia.[62] For patients experiencing altered smell, the RDN can recommend choosing cold foods (which are less odorous) and drinking from a covered cup with a straw to mask beverage smell. For foods that taste too sweet, sour sauces, salt, or lemon juice can be added. For a metallic taste, the patient can try eating with chopsticks or plastic utensils. For a bitter taste (common for red meats) the patient can try marinating the meat in sweet juice, such as fruit juice or wine. If red meat continues to be unpalatable, the patient may prefer poultry, fish, or vegetarian proteins.[63]

Xerostomia

Xerostomia is when a patient perceives dry mouth and it is particularly common in patients with head and neck cancer receiving radiation therapy.[64] Physical signs of xerostomia include cheilosis, tongue furrows, halitosis, food debris sticking to oral cavity, candidiasis, erythema, thick/ropey saliva, and dehydration (sunken eyes, poor skin turgor).[65] Nutrition interventions that can help with xerostomia include increasing fluid intake while eating, such as alternating bites of food with sips of liquid; sipping liquid frequently throughout the day; choosing soft and moist foods or adding sauces to moisten foods when eating; using sugarless chewing gum or hard candy to stimulate saliva flow; using liquid nutritional supplements to increase intake; limiting coffee, tea, and alcohol which can dry out the mouth; using a cool mist humidifier while sleeping; and providing nutrition education on good oral care.[66]

Anemias

Anemia is a blood condition resulting from insufficient healthy red blood cells (RBCs, erythrocytes). There may be issues with the number of RBCs, the size of the RBCs, or the amount of hemoglobin in the RBCs.[67] Iron, vitamin B12, and folic acid are key nutrients required for normal RBCs synthesis (erythropoiesis) and function.[67] Deficiencies in these nutrients, as well as hemorrhage, genetic conditions, chronic disease, and drug toxicity can lead to anemia. [67]

Anemia is an umbrella term. There are several types of anemia and they are classified based on the size of the RBCs (macrocytic, normocytic, microcytic) and the hemoglobin content of the

RBCs (hypochromic, normochromic). Anemia symptoms reflect limited exchange of O_2 and CO_2 between the blood and tissue cells and can include fatigue, tiredness, weakness, pale or yellowish skin, faintness, dizziness, increased thirst, sweating, weak but tachycardic pulse, tachypnea, dyspnea, lower leg cramps, and other heart-related symptoms such as arrhythmias and heart failure.[68]

Nutritional Anemias

Iron Deficiency Anemia

Iron insufficiency refers to a negative iron balance, progressing from iron depletion to iron-deficient erythropoiesis to iron deficiency anemia.[69] Note that microcytic anemia (iron deficiency anemia) is the last stage of iron deficiency and manifests clinically after a prolonged period of iron depletion.[67]

Iron is required to make hemoglobin, the protein in red blood cells that binds oxygen. During periods of growth, including pregnancy, the body produces more red blood cells and requires more iron. Not meeting the increased iron need can lead to iron deficiency anemia. Iron deficiency anemia can also occur with blood loss. See Table 12 for the main etiologies of iron deficiency anemia and examples. Groups at risk include infants, children, adolescents, persons who menstruate, persons with Crohn's disease, celiac disease, and kidney failure, persons who consume insufficient oral iron, and patients with internal bleeding.[68]

The bioavailability of dietary iron depends on:[70]

1) rate of absorption (increased with depleted iron stores)
2) form of iron (heme iron absorbed better than nonheme iron)
3) presence of dietary-enhancing factors (vitamin C)
4) presence of negative chelating factors (tannins [tea, coffee], fiber, phytates [legumes, whole grains, maize], oxalates [dark green leafy vegetables, berries, chocolate, coffee])[71]

Table 12. Etiologies for iron deficiency anemia

Etiology	Example
Inadequate dietary intake	Vegetarian or vegan meal plan with poor iron intake or supplementation; low-energy diet plans or skipping meals; excessive fiber, coffee, or tea consumption
Inadequate iron absorption	Diarrhea, achlorhydria, GI disease, gastrectomy, drug-nutrient interaction (H_2-blockers, tetracycline, antacids, antiretrovirals, cholestyramine, cimetidine, pancreatin)
Inadequate iron utilization	Chronic GI issues
Increased iron requirement	Infancy, adolescents, pregnancy, lactation
Increased iron excretion	Heavy menstrual flow, chronic blood loss*, acute blood loss from injury, parasites, malignancy
Insufficient release and/or use of iron from body stores	Chronic inflammation, chronic disease

Increased RBC hemolysis	Endurance athletes, soldiers (increased RBC hemolysis at beginning of vigorous training programs)

*Bleeding ulcer, hemorrhoids, esophageal varices, enteritis, ulcerative colitis. Adapted Krause's Food and the Nutrition Care Process.[69]

Persons with iron deficiency anemia absorb 20-30% of dietary iron compared to persons without iron deficiency absorbing only 5-10% of dietary iron. Heme iron (15% absorbable) is found in meat, fish and poultry. Nonheme iron is around 3-8% absorbable and is also found in meat, fish and poultry products as well as eggs, grains, vegetables, and fruits. Vitamin C (ascorbic acid) binds to iron and creates an easy-to-absorb complex. Thus, nutrition delivery interventions that pair vitamin C-rich foods with nonheme iron can improve the absorbability of nonheme iron. Consuming heme iron with nonheme iron will also improve absorbability. *Can you think of example meals that would improve absorbability of nonheme iron?*

New and recycled iron is transported in the blood attached to *transferrin*, a glycoprotein. Transferrin is a reverse acute-phase reactant and levels are decreased with inflammation (measured by total iron-binding capacity, TIBC).[69] Free iron is a harmful free radical, thus the body binds iron to protein both for transport and storage. Serum iron measures the amount of iron bound to transferrin; however, serum iron is not the most reliable measure of iron status. Iron is stored in the body as *ferritin.* Small amounts of ferritin leak from cells into the blood, with ~ 1 ng/mL of serum ferritin = 8 mg stored iron, in healthy adults. More details on iron digestion, absorption, storage, and transport are covered in the Normal Nutrition, Digestion, and Absorption study guide. The only information presented here relate directly to the biochemical labs used to evaluate for iron deficiency and microcytic anemia.

There are several biochemical measures that can be used to assess for iron deficiency (Table 13). The most sensitive markers of iron deficiency are ferritin level (serum or plasma) and transferrin saturation or TIBC as they change early on in developing iron deficiency and change only with true iron deficiency (rather than other nutritional or non-nutritional causes of anemia).[72] Several methods of iron status evaluation should be conducted, with a preference for ferritin, iron, and total circulating transferrin plus evaluation of RBC morphology. Hemoglobin levels are not used to diagnose iron deficiency anemia because levels only decrease late in the disease, do not distinguish between types of anemia, and normal hemoglobin values vary between individuals.[72]

Ferritin is an acute-phase reactant and levels rise with inflammation. Thus, ferritin will not accurately reflect iron stores in patients with acute inflammation, uremia, malignancy, chronic alcoholism, leukemia, Hodgkin's lymphoma, or liver disease. Anemia of chronic and inflammatory disease (ACD) must be distinguished from iron deficiency anemia to avoid unwarranted iron supplementation. Some patients with ACD have microcytic hypochromic RBCs with normal ferritin, low serum iron, and low TIBC. High ferritin may also indicate hemochromatosis or porphyria.[73]

If you haven't already, start working on the Lab Values Worksheet. It includes these lab values and prompts you to research when the values may be lower than normal as well as higher than normal.

Table 13. Biochemical Tests and Clinical Findings for Iron Deficiency Anemia

Biochemical Test	Result with Iron Deficiency	Clinical Findings	
		Early-Stage Iron Depletion	Iron Deficiency
Ferritin (plasma or serum)	Decreases	Poor muscle function (e.g., decreased exercise tolerance)	Pale skin and conjunctiva
Iron (plasma or serum)	Decreases	Reduced gastric acidity	Angular stomatitis
Total circulating transferrin (TIBC)	Increases	Neurologic changes (e.g., fatigue, anorexia, pica)	Atrophy of lingual papillae or glossitis
% saturation circulating transferrin	Decreases	Growth abnormalities in children	Oral erythema
Serum transferrin receptor (STFR)	Increases	Epithelial disorders	Gastritis with achlorhydria
Erythrocyte morphology	Microcytic, hypochromic	Reduced immune function (e.g. infections)	Thin, flat nails progressing to koilonychia

Adapted from Nutrition Care Manual[72] and Krause's Food and the Nutrition Care Process.[69]

The RDN should recognize and screen patients at high risk of iron deficiency and ideally identify early stages of iron depletion. The RDN should evaluate a patient's eating patterns to determine if they are meeting the RDA for iron and recommend nutrition interventions to increase iron intake. *Be familiar with the iron RDA for different life stages.*

Treatment for iron deficiency anemia is ferrous iron (typically as ferrous sulfate). Typical doses are 50 mg to 200 mg for adults and 6 mg/kg body weight for children. At these doses, ferrous (Fe^{2+}) iron absorption is more than 3 times that of ferric iron (Fe^{3+}).[74] Ideally, iron supplementation is done on an empty stomach but if the patient experiences gastric upset, nausea, heartburn, diarrhea, or constipation, the patient can take the iron with meals.[74] The RDN should also provide nutrition education on dietary sources of iron and ways to increase iron absorption.

Megaloblastic Anemia

Megaloblastic anemia is anemia due to vitamin B12 and/or folate deficiency. RBCs are macrocytic and may be oval in shape (macro*oval*ocytic). Symptoms of megaloblastic anemia include fatigue, pale skin, lethargy, dyspnea, dizziness, headaches, a sore and red tongue, palpitations, depression, poor appetite and weight loss, and forgetfulness. Symptoms unique to vitamin B12 deficiency include paresthesia (pins and needles) due to inadequate nerve myelinization, poor coordination, disturbed vision, and hallucinations. Symptoms unique to folate deficiency include reduced sense of taste or ageusia and diarrhea.

Drugs that can affect vitamin B12 status include paraaminosalicylic acid (TB antibiotic), colchicine (for gout), neomycin, metformin, and antiretrovirals. Intrinsic factor (IF) is a glycoprotein found in gastric juice that is necessary for absorption of vitamin B12 in the ileum.

Table 14. Etiology of Megaloblastic Anemia

Etiology	Vitamin B12 Deficiency	Folate Deficiency
Inadequate dietary intake	Poor diet, veganism without supplementation, chronic alcoholism	Vitamin B12 or vitamin C deficiency, poor diet, chronic alcoholism
Inadequate absorption	Gastric disorders (*H pylori,* lack of IF), small intestinal disorders (celiac disease) or short bowel syndrome, pancreatic disease, HIV/AIDS, chronic alcoholism, calcium-chelating agents	Celiac disease, drug interactions, congenital defects, chronic alcoholism
Inadequate utilization	Enzyme deficiency, abnormal binding proteins (TCII), drug interactions	Vitamin B12 or vitamin C deficiency, poor diet, chronic alcoholism, drug interactions
Increased requirement	Hyperthyroidism, growth life stage, increased hematopoiesis	Growth life stage, increased hematopoiesis, increased metabolic activity, drug interactions, enzyme deficiency (MTHFR), chronic alcoholism
Increased excretion	Inadequate vitamin B12 binding protein, liver disease, kidney disease	Chronic exfoliative dermatitis, vitamin B12 deficiency, liver disease, dialysis, chronic alcoholism
Increased destruction	Pharmacologic doses of ascorbic acid	High levels of dietary oxidants, chronic alcoholism

H. pylori: Heliobacter pylori; IF: intrinsic factor; TCII: transcobalamin II. Adapted from Krause's Food and the Nutrition Care Process.[69]

When dietary vitamin B12 is consumed, gastric acid unbinds it from food proteins and the vitamin B12 binds to R-binder protein in a sufficiently acidic environment, like a healthy stomach. Pancreatic trypsin releases vitamin B12 from the R-binder in the small intestine and vitamin B12 binds with IF in an alkaline environment, attaching to the surface vitamin B12-IF receptors in the ileum in the presence of calcium. Vitamin B12 is released from IF and binds to holo-transcobalamin II (holo TCII) and the complex enters portal blood. Patients lacking TCII or with abnormal TCII develop megaloblastic anemia. Now that we've reviewed normal digestion and absorption of vitamin B12, we can identify multiple situations where the process could be disrupted: insufficient gastric acidity; gastritis (impaired IF production); overly acidic pH in the small intestine; consumption of calcium-chelating agents (chelated calcium cannot help absorb vitamin B12 in the ileum), among others.

Pernicious anemia refers to anemia from vitamin B12 deficiency. It is typically caused by an IF deficiency, rather than inadequate intake of vitamin B12, but can result from any of the etiologies presented in Table 14. Vitamin B12 deficiency is typically managed medically via intramuscular or subcutaneous vitamin B12 injection. In the case of IF deficiency, very large oral doses (1000 mcg) may be given, as ~1% will absorb via diffusion. When metformin is interfering with vitamin B12 absorption, increasing calcium intake can help reverse the malabsorption. After medical treatment, if appropriate, a high protein diet (1.5 g/kg) that includes green leafy vegetables (for iron and folic acid), meats, eggs, and dairy is useful. The Dietary Guidelines recommend older adults consume a vitamin B12 supplement or sufficient fortified cereals to limit the effects of age-related atrophic gastritis. *What is atrophic gastritis?*

Folate deficiency anemia does not have a specific term. Drugs that can affect folate status include anticonvulsants, barbiturates, cycloserine (TB and UTI antibiotic), sulfasalazine (anti-

inflammatory for ulcerative colitis and rheumatoid arthritis), cholestyramine, and metformin. An excess of glycine and methionine can also contribute to folate deficiency. Alcohol interferes with the enterohepatic transport and activation of folate and chronic alcoholism typically results in folate deficiency from all 6 etiologies listed in Table 14.

Folate is transported and stored as 5-methyl THFA (inactive) and requires vitamin B12 to be activated to THFA, which is then converted to the coenzyme form of folate used in DNA synthesis (including hematopoiesis). Thus, folate deficiency due to impaired utilization can arise from vitamin B12 deficiency due to the 'methylfolate trap' (Figure 5). Medically, folate deficiency is treated with oral folate though caution should be taken to further determine if there is also a vitamin B12 deficiency. Folate supplementation can reverse the hematopoietic consequences but not neurologic effects of vitamin B12 deficiency, which if untreated, may become permanent. After medical treatment, the patient should consume at least 1 serving of uncooked fruits or vegetables per day and/or fortified foods for folate deficiency.

Tests for vitamin B12 deficiency and folate deficiency include serum homocysteine, folate, vitamin B12, and methylmalonic acid (MMA). Folate, vitamin B12, and vitamin B6 are required to convert homocysteine to methionine. Elevated homocysteine levels point to genetic enzyme deficiencies or a deficiency in folate, vitamin B12, and/or vitamin B6. Folate is measured in plasma + whole blood and then again in serum. The difference between the two values is RBC folate concentration, which is considered by some to be the most reliable indicator of folate status. Reasons for folate malabsorption, such as bariatric surgery, celiac disease, chronic alcoholism, or drug-nutrient interactions should be considered if folate levels are low. Serum vitamin B12 levels are reflective of RBC vitamin B12 levels and are typically measured by TCII and TCII percent saturation. To differentiate between vitamin B12 deficiency anemia and folate deficiency anemia, a serum or urinary MMA assay should be done. Elevated MMA indicates a vitamin B12 deficiency, because elevated MMA occurs when the lack of vitamin B12 inhibits methylmalonyl CoA → succinyl CoA, resulting in a buildup of MMA. Figure 5 shows the role of vitamin B12, folate, and vitamin B6 in MMA and homocysteine metabolism.

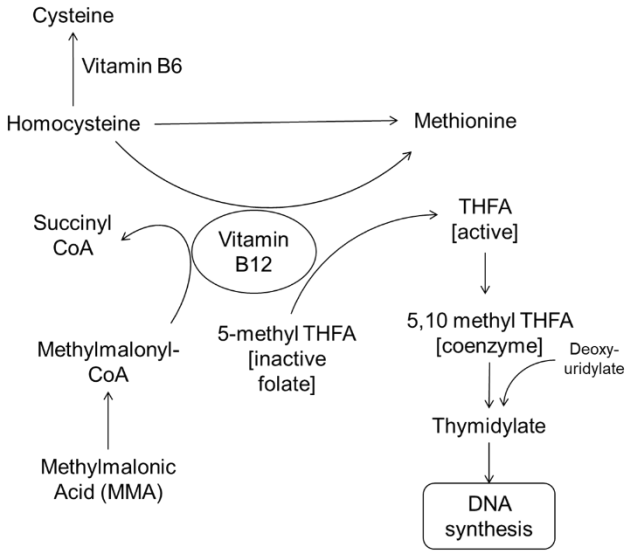
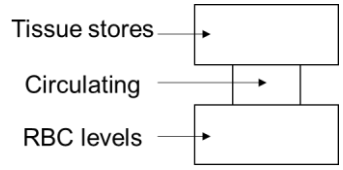


Figure 5. Conversion of homocysteine to methionine, requiring vitamin B12 and folate; conversion of MMA-CoA to succinyl CoA requiring vitamin B12, and the role of THFA (active folate) in downstream DNA synthesis. Created by Bailey DeBarmore. Do not reproduce without permission.

For the exam, you should be familiar with the link between erythropoiesis, folate, and vitamin B12; folate, vitamin B12 and homocysteine; and vitamin B12 and MMA.

Sometimes the pattern of depletion and deficiency for nutritional anemias is shown with an "I" shaped diagram (bottom right). The top represents tissue stores, the bottom RBC levels, and the middle small part represents circulating levels. Different biomarkers represent the different locations in the body iron, vitamin B12, or folate can be found, and the progression from depletion to deficiency differs between iron deficiency anemia and megaloblastic anemia. If this visualization of deficiency makes sense to you, take a look at Figure 6 below. If you find this confusing, skip over it – there is no need to try to memorize something that does not help you understand the material more. Instead, try to think of another way to visualize or discuss nutritional anemias. Perhaps a visual diagram with the locations where iron, vitamin B12, or folate is stored and the flow through depletion to deficiency with symptoms might make more sense to you.



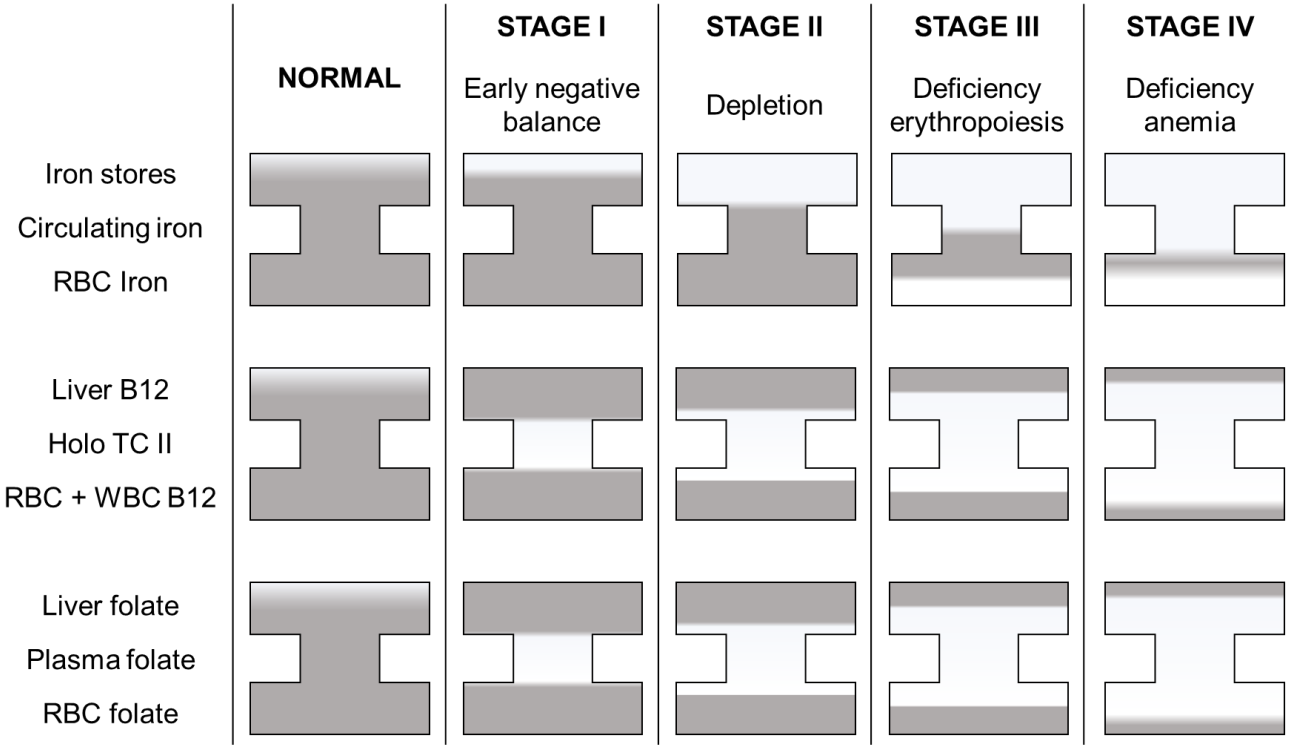


Figure 6. Stages of iron, vitamin B12, and folate depletion and deficiency.

Other Nutritional Anemias

Protein deficiency and **protein-energy malnutrition** can result in *normocytic* hypochromic anemia characterized by relatively fewer RBCs (due a decrease in oxygenation needs without change in blood volume) and low RBC hemoglobin content due to protein deficiency. If the etiology is poor intake, the patient may also have a deficiency in other nutrients like iron and folic acid.

Why does erythropoiesis scale down in a person with protein deficiency or protein-energy malnutrition?

Anemia of pregnancy is typically due to an increase in blood volume which results in a lower concentration of RBCs (and in this case would be a non-nutritional anemia). However, the RDN should evaluate a pregnant patient's typical food intake to ensure they are meeting the increased iron requirements.

Copper deficiency can result in low serum iron and low hemoglobin despite normal iron stores because ceruloplasmin, a copper-containing protein, is responsible for mobilizing iron stores.

Non-Nutritional Anemias

Hemolytic Anemia

Hemolytic anemia refers to a condition where healthy RBCs prematurely lyse due to membrane defects and oxidative damage. Early vitamin E deficiency manifests as early hemolysis which is effectively treated with vitamin E supplementation (and would therefore be a nutritional anemia).

Thalassemia and **sickle cell anemia** are inherited hemolytic anemias. Thalassemia is most common in persons of Mediterranean origin and is caused by defective hemoglobin synthesis and ineffective erythropoiesis. It is a severe disease with increased plasma volume and bone marrow expansion, progressive splenomegaly, facial deformities, and osteomalacia. Increased iron absorption results in damaging iron deposits in tissues leading to heart, liver, and endocrine dysfunction. These patients require both transfusions and chelation therapy as lifesaving therapy.

Sickle cell anemia is caused by homozygous inheritance of *hemoglobin S* which results in sickle shaped RBCs that do not move well through capillaries and do not carry oxygen efficiently. Patients experience acute pain episodes due to capillary occlusion and the hemolytic anemia and vasocclusion lead to liver and kidney dysfunction. Low concentrations of vitamin B6 can elevate serum homocysteine (Figure 5 in previous section) and patients can also have iron deficiency anemia. It is important for patient with sickle cell anemia to maintain hydration, limit risk of infections, and monitor spleen size.[75]

Serious complications include acute pulmonary injury, acute chest syndrome, pneumonia, pulmonary fat embolism, cerebral infarctions, and hemorrhagic stroke.[75] The RDN should assess for malnutrition and poor growth in infants, children, and adolescents. Patients have higher caloric needs and specialized energy estimation equations are used that incorporate hemoglobin levels.[76]

High-calorie and high-protein nutrition therapy can be used with regular nutrition monitoring. An overall balanced diet with adequate hydration should be emphasized. The RDN should also assess for micronutrient deficiencies (magnesium, zinc, iron, vitamin E, vitamin D, B vitamins, and vitamin C).[77] Specific nutrition problems, such as diarrhea, constipation, and high cholesterol should also be managed with MNT as appropriate.

Acquired hemolytic anemia can occur due to infection, certain medications, blood cancers, autoimmune disorders, hypersplenism, valvular cardiovascular disease, or blood transfusion reaction.[78]

Anemia of Chronic and Inflammatory Disease (ACD)

Anemia of chronic and inflammatory disease (ACD) is the second most common anemia, after iron deficiency anemia. Patients with infections, cancer, chronic kidney disease, heart failure, obesity, or autoimmune diseases like rheumatoid arthritis, lupus, vasculitis, sarcoidosis, or inflammatory bowel disease are at particular risk of ACD. Inflammation, infection, or malignancy can result in decreased erythropoiesis and/or dysfunctional iron metabolism. Because ferritin is an acute phase reactant, its levels increase with inflammation and no longer accurately reflect iron stores. Transferrin is a reverse acute phase reactant, and thus can be falsely low with ACD. This type of anemia is *typically* mild and normocytic and should be distinguished from iron deficiency anemia to avoid iron supplementation. Instead, recombination erythropoietin therapy (EPO) is typically given. If iron deficiency co-occurs, ferritin may be normal rather than low, because it is falsely "high" relative to iron stores due to inflammation.

Gastrointestinal Disorders

GERD

Nutrition Therapy	MNT Goals
• Trial food restrictions • Small frequent meals • Weight reduction • Smoking cessation • Lifestyle modifications	• Nutrition education related to foods triggering in GERD • Tailored nutrition therapy to address symptoms, inadequate oral intake, and unintended weight loss

Gastroesophageal reflux disease (GERD) puts patients at potential nutritional risk. GERD occurs due to lower esophageal sphincter dysfunction or pressure allowing gastric contents to reflux into the esophagus. The lower esophageal sphincter may dysfunction for a number of reasons. The signs and symptoms of GERD occur due to the esophageal mucosal damage from exposure to acidic gastric contents. In nutritional assessment, the RDN should assess for the presence of any contributing factors that would cause or exacerbate GERD. The complications of untreated or treatment-resistant GERD can be serious. Surgical treatment includes Nissen fundoplication (Figure 7).

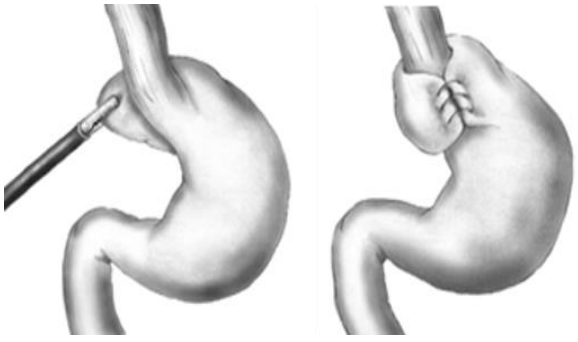


Figure 7. Nissen fundoplication (below the diaphragm) to tighten the lower esophageal sphincter. Source: WikiMedia Commons.

Nutrition therapy for GERD includes lifestyle and diet modifications to reduce frequency or severity of GERD symptoms. The RDN may guide the patient to restrict or eliminate foods that increase gastric acidity (pepper, caffeine,

alcohol) and/or foods that affect lower esophageal sphincter pressure (chocolate, high fat foods, peppermint, spearmint, certain fruits and vegetables like citrus and onions).[79]

Since obesity can contribute to increased abdominal pressure, nutrition therapy and education related to appropriate weight loss may be warranted. Lifestyle modifications include exercising at least 30 minutes most days of the week, wearing loose fitting clothes, eating small frequent meals, and eating in a calm environment, to help alleviate or prevent severe GERD symptoms. Waiting around 3 hours between eating and lying down as well as propping the head up 6-9 inches in bed can help prevent reflux. Finally, if a patient smokes, initiating a smoking cessation program should be prioritized.

Table 15. Summary of GERD risk factors, symptoms, complications, and treatment

Complications	**Symptoms**
• Dysphagia (ongoing) • Aspiration • Pneumonia, asthma • Ulceration • Esophageal perforation or stricture • Barrett's esophagus	• Dysphagia • Heartburn (may be severe and radiate to neck and jaw) • Increased salivation • Belching
Contributing factors	**Treatment**

• Increased hormone secretion
• Comorbidities (hiatal hernia, scleroderma)
• Cigarette smoking
• Obesity
• Medications (dopamine, morphine, theophylline)
• Foods with spearmint, peppermint, chocolate, alcohol, caffeine, or high in fat
	• Reduce gastric acidity to minimize symptoms and complications via medication and nutrition therapy
	  ○ Antacids, buffers
	  ○ Histamine blockers
	  ○ Prokinetics
	  ○ Proton pump inhibitors
	  ○ Mucosal protectants
	• Surgical intervention for severe GERD
	  ○ Nissen fundoplication

Why do we try to reduce the acidity of gastric contents to treat GERD? How could reduced gastric acidity affect digestion and lead to side effects?

Factors that increase abdominal pressure can exacerbate GERD symptoms. Examples include obesity, tight fitting clothes, as well as respiratory conditions because abdominal pressure increases with increased effort to breathe. What modifications could the RDN recommend to help with abdominal pressure? What other conditions should the RDN be aware of as a part of the interdisciplinary care team?

Go online and look up the medications listed under "Treatment" in Table 15. Make note of what they do, how they do it, and example medication names.

Inflammatory Bowel Disease

Nutrition Therapy[80]	MNT Goals
• Energy: 25-35 kcal/kg, but ideally determine via indirect calorimetry* • Protein: 1-1.5 g/kg/day • Micronutrients: B vitamins, vitamin C, vitamin E, vitamin D, vitamin K, iron, zinc, magnesium, selenium, potassium*** • Supplements: omega 3, glutamine, prebiotics, probiotics (not inulin)	• Address symptoms interfering with adequate oral intake • Compensate for malabsorption • Tailor nutrition therapy and education to help patient meet nutritional requirements, correct nutritional deficiencies, and compensate for increased nutritional losses

*Not all patients are hypermetabolic, though some may be due to infection and medical intervention such as surgery and healing processes. Energy needs depend on weight, disease severity, and nutritional deficits.
**Protein needs based on disease state, exacerbation vs remission, body weight, infection, and surgical healing
***Use DRIs as baseline recommendation and note that patient may need higher amounts of listed vitamins and minerals. Evaluate need for iron supplements.

Inflammatory bowel disease (IBD) is a chronic, inflammatory, autoimmune condition of the GI tract. The term IBD refers to a syndrome that includes ulcerative colitis (UC), Crohn's disease (Crohn's) and indeterminant colitis. These 3 diseases can be distinguished from each other through symptoms, GI involvement, biopsy, and antibody testing.[81] For example, UC primarily affects the lower bowel, particularly the colon and the rectum. The rectum is always affected in UC and damage is continuous, leaving no normal intestinal mucosa. In contrast, Crohn's can affect any area of the GI tract, from the mouth to the anus, and often skips over areas, leaving patches of normal mucosa. Another difference is that while UC affects only the GI mucosa, Crohn's affects all layers of the GI tract (it is 'transmural'), leading to penetrative fistulas that are replaced with fibrotic tissue as they heal, leading to strictures and bowel obstructions.

Current hypotheses are that environmental triggers cause an abnormal autoimmune response in the GI tract in genetically susceptible individuals.[81] Environmental factors associated with IBD include antibiotics, NSAIDs, infection, stress, diet, smoking and possibly oral contraceptive use.[81] Several chromosomal sites have been associated with IBD as well as a higher incidence in Jewish populations.[81] IBD can manifest outside of the GI tract, affecting other organ systems and leading to serious complications. Extraintestinal complications include osteopenia, osteoporosis, peripheral arthritis, ankylosing spondylitis, skin symptoms, eye symptoms, primary sclerosing cholangitis, and thromboembolic events.[81] Oxalate kidney stones are common in Crohn's disease. Other complications of Crohn's disease include abscesses, anal fissures, bowel obstruction and perforations requiring bowel resections (leading to short bowel syndrome), colon cancer, fistulas, hyperoxaluria, malnutrition, steatorrhea, bowel strictures, and ulcers.[81]

Review Table 16. If there are any terms unfamiliar to you, such as transmural versus mucosa only, or pyoderma gangrenosa, go online and look them up. Look up examples of drugs used in the treatment to be able to recognize generic names.

Table 16. Summary of Crohn's Disease and Ulcerative Colitis

	Crohn's Disease	Ulcerative Colitis
Parts of GI tract affected	Any portion (mouth to anus) • Ileum and colon most common • Often 'skips' areas Transmural	Lower bowel • Rectum • Continuous Mucosa only
Symptoms	Abdominal pain Diarrhea Fever Oral aphthous ulcerations Pyoderma gangrenosa	Abdominal pain Abdominal cramping Bloody diarrhea Nausea, vomiting Fever
Complications	Kidney stones See text	Toxic megacolon See text
Treatment	Anti-inflammatories (aminosalicylates, corticosteroids) Immunosuppressants Biologics Symptom management • Anti-diarrheal • Pain relievers • Vitamin-mineral supplements Bowel resection	Anti-inflammatories (aminosalicylates, corticosteroids) Immunosuppressants Biologics Symptom management • Anti-diarrheal • Pain relievers • Antispasmodics • Iron supplements Proctocolectomy with J-pouch or ileal stoma
Nutrition therapy	High calorie, high protein Oxalate restriction Nutrition therapy re: short bowel syndrome Nutrition support Low fiber diet Tailored to manage symptoms	High calorie, high protein Nutrition therapy re: proctocolectomy, J-pouch/ ileal stoma Nutrition support Tailored to manage symptoms

Any patient with an IBD diagnosis is considered at nutritional risk.[81] Nutrition indicators to look for include inadequate intake; nausea, vomiting, diarrhea, abdominal pain; symptoms of malabsorption (gas, bloating, fatty stool); weight loss; weight status; growth trajectory in children and adolescents; lean body mass; micronutrient deficiency including anemia; drug-nutrient interactions; and comorbidities.

Why would a patient with IBD have higher calorie and protein needs?

Why would knowing the specific locations of bowel resections affect the RDN's nutrition prescription?

Nutrition prescriptions during exacerbations are different than during remission. During an exacerbation, the patient may receive nutrition support, a modified diet, and vitamin-mineral supplements (vitamin D, zinc, calcium, magnesium, folate, vitamin B12, iron).[82] The modified diet is typically low-fat, low-fiber (low residue), and high protein, high calorie. Small frequent meals with the goal of returning to a normal, high-quality, complete diet is ideal. Fiber is only to be restricted during exacerbations or when there are bowel strictures. During remission, patients should follow an eating pattern that is tailored to their GI function while replenishing their nutrient stores and helping them maintain their weight. Patients with Crohn's disease may

restrict foods in high in oxalate to avoid oxalate kidney stones, may consider use of probiotics and prebiotics, and may supplement with omega-3 fatty acids and glutamine.

Nutrition management of IBD is complex and something that RDNs typically come to specialize in. For the RDN exam, be familiar with the differences and similarities between Crohn's disease and UC, and the underlying physiologic factors (inflammation, autoimmune response). Recognize that nutrition therapy relates to the disease itself but also the treatments, including surgical resection and medications like methotrexate and corticosteroids which can lead to other problems.

Irritable Bowel Syndrome

Nutrition Therapy[83]	**MNT Goals**
• Modified diet (low FODMAP diet) • Normalize eating patterns • Supplements to ensure adequate intake • Consider use of pre- and probiotics (but not inulin)	• Ensure adequate nutrient intake • Tailor diet for specific GI symptoms • Provide nutrition education on the potential role of different foods in managing symptoms

Irritable bowel syndrome (IBS) is likely caused my multiple factors which means intervention to manage the resulting symptoms must be individualized. It is primarily diagnosed by ruling out other conditions, such as celiac disease, and by symptom type and frequency.[84] Symptoms of anemia, rectal bleeding, bloody stools, tarry stools, and weight loss are symptoms that suggest a problem other than IBS.

The Rome III Criteria (2006) and updated Rome IV Criteria (2016) as well as the American College of Gastroenterology (ACG) Criteria are used to diagnosed IBS (Table 17).[85] The difference between the Rome III and Rome IV Criteria is the removal of 'abdominal discomfort' from the list and looking at a weekly average of stool consistency rather than a monthly average. You shouldn't need to know the specific criteria for the RDN exam.

Table 17. Diagnostic Criteria for Irritable Bowel Syndrome

Rome III Criteria	**Rome IV Criteria**
Recurrent abdominal pain or discomfort ≥3 days per month for last 3 months and that meets ≥2 of the following criteria: • Related to bowel movements • Related to change in bowel movement frequency • Onset associated with change in stool appearance	Recurrent abdominal pain on average ≥1 day per week in last 3 months associated with ≥2 of the following: • Related to bowel movements • Related to change in bowel movement frequency • Onset associated with change in stool appearance
ACG Criteria	
Abdominal pain or discomfort associated with altered bowel habits for at least 3 months	

In addition to abdominal pain, other symptoms include constipation, diarrhea, alternating constipation and diarrhea, bloating, mucus in stool, incomplete evacuation, excessive gas, fatigue, headaches, urinary incontinence, and chest discomfort.[84] IBS is often classified as IBD-

C (constipation predominant), IBD-D (diarrhea predominant), IBD-M (mixed), or IBD-U (unspecified). Patients may alternate between classifications.[86]

Dysmotility, visceral hypersensitivity, and brain-gut dysfunction are 3 factors that may contribute to IBS.[87] Dysmotility is the poor regulation of GI tract muscle contractions. Visceral hypersensitivity refers to greater sensitivity of nerves that attach to the GI tract. Brain-gut dysfunction refers to problems in how the brain and gut communicate.

You can read the 2009 Position Statement on Management of IBS from the American College of Gastroenterology here (https://bit.ly/3vjzhSg) if you'd like to learn more.

Unlike IBD, IBS does not cause persistent harm to the gut and there are no visual signs of disease, such as inflammation, in the gut. IBS may occur following abnormal inflammatory response to a GI infection, or may be due to a genetic predisposition or dietary intolerance. Because the cause of IBS is unknown and likely multifactorial, nutrition intervention should aim to minimize symptoms rather than address the underlying etiology. One type of nutrition intervention is a modified diet targeted at limiting FODMAPs that may contribute to symptoms.

FODMAPs

FODMAP stands for **F**ermentable **O**ligo-, **D**i-, and **M**ono-saccharides, **A**nd **P**olyols. The term was created by Australian researchers who hypothesized that foods with these length carbohydrates contribute to IBS symptoms. These short-chain carbohydrates are poorly absorbed in the small intestine, are osmotic (drawing water into the gut lumen) and are quickly fermented by gut microbiota leading to gas, distention, bloating, diarrhea, constipation, and cramping (IBS symptoms).

The **oligosaccharides** considered here ("O" in FODMAP) are **fructans** (ex: inulin), fructo-oligosaccharides (**FOS**), and galacto-oligosaccharides (**GOS**). Humans lack the digestive enzyme to cleave and absorb these molecules. Foods high in these oligosaccharides include cruciferous vegetables (cabbage, turnips, Brussels sprouts, broccoli), Liliaceae vegetables (garlic, asparagus, chives, shallots, onions, leeks), pulses (beans, peas, lentils, chickpeas), Poaceae (corn, rice, wheat, barley, oats, rye), beets, apples, peaches, watermelon, pistachios as well as foods labeled "high in fiber" or "prebiotic" as they typically contain inulin. The patient can check the ingredient list for inulin to be sure.

The **disaccharide** ("D" in FODMAP) considered is **lactose**, and only causes IBS symptoms when a person lacks sufficient lactase levels. Genetics, ethnicity, and coexisting gut disorders are associated with lactase insufficiency, or lactose intolerance. Lactase is the digestive enzyme that cleaves lactose into two monosaccharides which are then absorbed. *What two monosaccharides make up lactose?* Lactose is found in milk, yogurt, ice cream, custard, and cheese.

The "M" in FODMAP stands for **monosaccharide** and refers to **free fructose**. While fructose can be absorbed with glucose, when fructose concentration exceeds glucose concentration, the body can absorb fructose using specific fructose carriers. However, some individuals have few

of these fructose carriers and so when fructose concentrations are high, they experience fructose malabsorption symptoms. Free fructose is high in drupes (like apples, mangoes, and pears), watermelon, asparagus, artichokes, sugar snap peas, honey, and high fructose corn syrup.

The “P” in FODMAP stands for **polyols**, or **sugar alcohols**. The gut only absorbs about 1/3 the amount of polyols consumed. Sorbitol, mannitol, maltitol, and xylitol may be added to packaged foods and when consumed in large amounts can lead to IBS symptoms. High-polyol natural foods include drupes (apples, apricots, cherries, pears, nectarines, peaches, plums, prunes), watermelon, avocado, cauliflower, mushrooms, snow peas.

In working with a patient with suspected FODMAP intolerance, the nutrition intervention may involve avoiding all foods high in FODMAPS for 6-8 weeks and recording GI symptoms with a detailed food, supplement, and symptom diary. FODMAP foods may be slowly re-introduced, one FODMAP category at a time, to asses tolerance and threshold. In order to accurately identify foods culpable for GI symptoms, there should be regular follow-up. The goal is to help the patient achieve adequate nutrient intake without unnecessary food restrictions which can lead to nutrient deficiencies.[88]

Chronic Peptic Ulcer Disease

Nutrition Therapy	MNT Goals
• General healthful nutrition recommendations • Small frequent meals • Avoid eating before bed Special considerations with severe alcoholism and/or very poor or no intake for extended period (refeeding risk)	• Optimize nutritional intake to meet needs and correct deficiencies • Adopt dietary and lifestyle factors to minimize symptoms and promote healing

Peptic and duodenal ulcers can develop when the stomach produces excessive pepsin and gastric acid and/or when the mucosal lining of the stomach or duodenum is impaired and therefore more susceptible to damage from gastric acid and pepsin.[89] Duodenal ulcers are included in peptic ulcer disease because if the chyme entering the duodenum is more acidic than normal, or the duodenal mucosa is impaired, duodenal ulcers can occur.

Helicobacter pylori infection, aspirin use, NSAID use, alcohol, and steroid use can affect mucosal integrity while smoking, stress, and blood vessel injury can decrease blood supply to the mucosa affecting tissue integrity.[89] Acid secretion can be stimulated by certain foods like pepper, alcohol, caffeine, by stress, by certain conditions such as Zollinger-Ellison syndrome, or rapid gastric emptying can lead to overly acidic chyme entering the duodenum.[89]

Patients with chronic peptic ulcer disease may have unexplained weight loss, inadequate nutritional intake, and nutritional deficiencies such as calcium, iron, and vitamin B-12 absorption due to long-term use of anti-acid medications and/or blood loss. With persistent vomiting, patients may also have electrolyte imbalances.

Symptoms include abdominal pain that may be exacerbated or relieved by food consumption. If H. pylori is responsible for the ulcers, treatment with antibiotics, bismuth, and an acid-pump inhibitor is typical.[89] Other medications include antacids, proton pump inhibitors, histamine-blocking agents, prokinetics, and mucosal protectants.

Compare the symptoms and treatment for peptic ulcers and GERD. Could a patient have both?

What is Zollinger-Ellison syndrome?

Could there be risk of nutritional deficiencies with excessive use of medications to lower gastric acidity? Why or why not?

Gastric Surgery

Nutrition Therapy	MNT Goals
• Energy: adequate for postoperative healing • Protein: adequate for postoperative healing • Liquid multivitamin and mineral supplements • Vitamin B-12 injections • Modified diet and eating behaviors to prevent dumping syndrome	• Promote postoperative healing • Tailor nutrition education to promote optimal nutrition intake while minimizing symptoms of malabsorption and/or maldigestion • Prevent or correct nutritional deficiencies

Gastric surgery may be performed as surgical treatment for malignancy, peptic ulcer disease, abscesses, sepsis, or GI bleeding. Common procedures include vagotomy, gastric resection, and pyloroplasty. Reconstruction following the latter two include Billroth I (gastroduodenostomy), Billroth II (gastrojejunostomy) or Roux-en-Y. See Table 18 and Figure 8. Indicators for nutritional risk following gastric surgery include unexplained weight loss, anemia, micronutrient deficiencies, dumping syndrome, hypoglycemia, food intolerance and inadequate nutritional intake.[90]

Table 18. Description of Common Gastric Surgeries and Reconstructive Procedures

Gastric Surgery	Description
Vagotomy	Interrupts vagus nerve innervation to parietal cells in order to decrease acid production and response to gastrin
Pyloroplasty	Interrupts vagus nerve innervation to parietal cells and gastric emptying; enlarges pyloric sphincter
Billroth I	Partial gastrectomy or pyloroplasty; anastomosis of proximal duodenum to distal stomach
Billroth II	Partial gastrectomy; anastomosis or proximal jejunum to distal stomach
Roux-en-Y	Partial gastrectomy; anastomosis of jejunum to upper portion of stomach

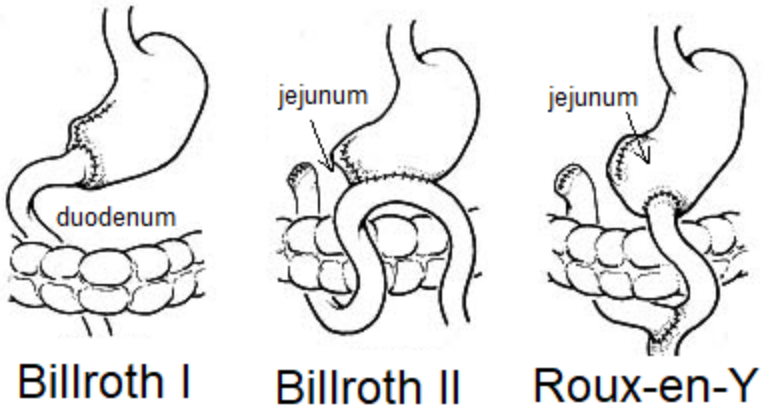


Figure 8. Depiction of Billroth I (gastroduodenostomy), Billroth II (gastrojejunostomy), and Roux-en-Y.

Gastric resection alters normal digestive mechanisms while the decreased gastric volume and changes in gastric emptying can increase nutritional risk. Nutritional issues may include dumping syndrome, hypoglycemia, malabsorption, anemia (iron deficiency, pernicious, or megaloblastic), lactose intolerance, and micronutrient deficiencies (vitamin A, vitamin D, thiamin, copper, calcium).[91] Tolerance to high-fat foods, lactose, caffeine, and sorbitol should be assessed.[92] Biochemical indicators of anemia, malnutrition, fluid and electrolyte status, and micronutrient deficiency should be assessed and monitored.

Dumping Syndrome

When stomach contents enter the small intestine too quickly, the high osmolar load and high volume draws fluid into the small intestine resulting in cramping, abdominal pain, dizziness, weakness, tachycardia, and diarrhea (early dumping syndrome, 10-20 minutes after eating).[91] Late dumping syndrome occurs 1-3 hours after eating and is characterized by hypoglycemia. Contents in the small intestine stimulate insulin release but with dumping syndrome, there is increased GI motility so by the time insulin is there, there is no substrate to act upon, resulting in hypoglycemia.[91] Symptoms of hypoglycemia include shakiness, sweating, confusion, and weakness. Finally, if undigested food enters the colon, fermentation can lead to gas, abdominal pain and cramping, and diarrhea.

To prevent dumping syndrome, patients should avoid all simple sugars initially. Initial meals during the recovery period should include 1 or 2 two foods at a time and with a goal of 5-6 smalls meals per day.[93] Liquids should be consumed 30-60 minutes after consuming solid food (rather than at the same time) and patients should lie down after eating to slow gastric emptying. Functional fiber may help with delaying gastric emptying and preventing diarrhea.

Celiac Disease

Nutrition Therapy	MNT Goals
Gluten-free diet Consider gluten-free vitamin/mineral supplement	• Provide nutrition education re: gluten-free nutrition prescription (what foods must be avoided, what grains are allowed), food labeling, access to gluten-free products, non-food sources of gluten, cross-contamination • Create a healthful eating plan with the patient that addresses nutritional risks of a gluten-free prescription

Celiac disease is an immune-mediated disorder that primarily manifests as chronic inflammation in the small intestine due to consumption of gluten, a prolamin found in wheat, barley, and rye.[94] The damage to the small intestine leads to malabsorption. GI symptoms may include bloating, diarrhea, constipation, gas, lactose intolerance, steatorrhea, nausea, vomiting, and abdominal pain. If celiac disease manifests in children (a life cycle stage critical to growth and development) symptoms include delayed puberty, damage to tooth enamel, failure-to-thrive, irritability, short stature, slow growth, and weight loss.[95] There are non-GI symptoms of celiac disease, including *dermatitis herpetiformis,* fatigue, joint pain, peripheral neuropathy, seizures, canker sores, and infertility, among others.

Go online and look up dermatitis herpetiformis.

There is no single definitive diagnostic test for celiac disease. Instead, physicians will consider medical history, family history, physical symptoms, a dental exam, blood tests, and small intestine biopsies. Blood tests include immunoglobin A (IgA), antihuman tissue transglutaminase, and IgA endomysial antibody immunofluorescence.[96] The patient may also be tested for genetic markers (DQ2, DQ8).

Patients with type 1 diabetes or other autoimmune disorders, patients who have relatives with celiac disease, and patients with Turner syndrome, Down syndrome, or Williams syndrome may be at higher risk of celiac disease.[97]

Go online and look up Turner syndrome, Down syndrome, and Williams syndrome.

If you have a patient with both type 1 diabetes and celiac disease, what considerations would you need to make in their eating plan and nutrition education?

Damage to the small intestine can result in vitamin/mineral deficiencies, particularly calcium, vitamin D, iron, and folate, which can manifest as osteoporosis, osteomalacia, and anemia.[98] In addition to these consequences of celiac disease, the patient can also experience chronic malnutrition, nervous system problems (dizziness, neuropathy), as well as reproductive and fertility problems. Rare complications include adenocarcinoma, liver damage, and lymphoma.[95]

When symptoms continue or return despite following a strict gluten-free diet, the condition is deemed ‘refractory celiac disease’. The small intestine is not able to heal and continues to be severely damaged, leading to chronic malabsorption and malnutrition. The patient may require parenteral nutrition to meet nutrient needs.

You should be familiar with the gluten-free labeling laws of the Food Allergen Labeling and Consumer Protection Act of 2004. For example, if a food contains semolina, which a consumer may not recognize as wheat or as gluten-containing, the term "Wheat" must appear somewhere on the label. The law applies to foods, dietary supplements, infant formulas, and medical foods. Read about gluten-free labeling laws in the US on the FDA website: https://www.fda.gov/food/food-labeling-nutrition/gluten-free-labeling-foods

A gluten-free nutrition prescription traditionally involves an eating plan based on grains and plant foods that are naturally gluten-free (corn, rice, amaranth, quinoa, millet, sorghum, arrowroot, buckwheat, flax, sago, potato, soy, legumes, tapioca, wild rice, yucca, cassava, nuts, seeds).[98] However, there is a Codex standard for gluten-free foods that allows foods including gluten-containing grains to be "rendered gluten-free" and qualify for gluten-free labeling if they contain less than 20 ppm gluten.[98] As the RDN, you should provide nutrition education on the sources of gluten in both foods and nonfood sources (such medications and supplements), teach the patient how to read food labels, and make the patient aware of the variety of foods that may contain gluten (seasoning, sauces, candy, cold cuts), including beverages (beer, some spirits, malt beverages).

Many gluten-free cereal foods are not enriched with B vitamins and iron like other grains in the food supply are. The patient should choose whole grain gluten-free products (teff, millet, quinoa, buckwheat) when possible, increase intake of noncereal sources of B vitamins and iron, and consider a gluten-free multivitamin mineral supplement. [98]

Table 19. Gluten-containing Foods and Beverages

Grains	Wheat, barley, rye, cross-bred varieties (triticale), oats, and processed foods containing wheat, barley, or rye
Ingredients	Flour (white, plain, bromated, enriched, phosphate, self-rising, durum, graham), farina, semolina
Other products	Beer, ale, porters, stout, matzo, bouillon cubes, brown rice syrup, cold cuts, other processed meats, seasonings and sauces

Ostomies

Colostomy

Nutrition Therapy	MNT Goals
• Consider underlying disease leading to ileostomy • Diet modifications to address odor and GI symptoms • Fluid, electrolytes: increased losses • Micronutrients: monitor B vitamins and vitamin K • Restrict foods high in oxalates • Assess for lactose intolerance and fat malabsorption	• Maintain fluid and electrolyte balance • Provide nutrition education on strategies to reduce odor in ostomy output if needed • Work with patient to alleviate GI symptoms such as odor, gas, and diarrhea

Surgical resection of the rectum requires formation of a colostomy, where the end of the colon is surgically attached to a stoma or used to form a pouch in place of the rectum. A patient may undergo removal of part of the colon and have it attached to the rectum (if not removed) or may have a colostomy even if the rectum isn't removed. Nutrition therapy should consider the underlying disease that required the bowel resection such as IBD, colon cancer, diverticulitis, or trauma.[99]

Nutrition therapy following colostomy should consider postoperative healing, adequate nutrient absorption, electrolyte and fluid balance, monitoring B vitamins and vitamin K, and alleviation of symptoms such as odor, gas, and diarrhea. Postoperatively, patients with colostomy begin with clear liquids, progress to a low-fiber nutrition diet, and then return to an individualized but normal diet where foods may be added one at a time to establish tolerance.[100]

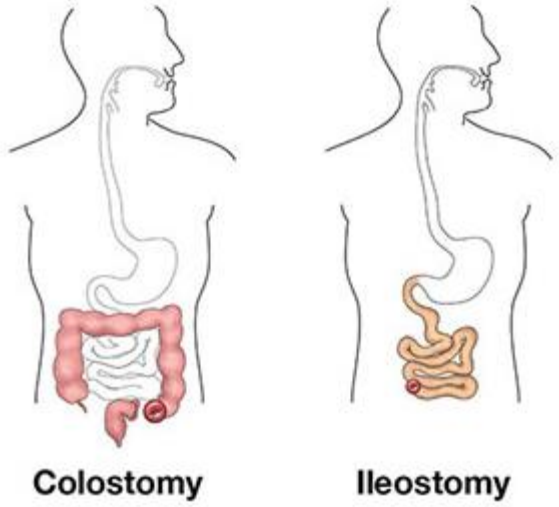


Figure 9. Simplified depiction of colostomy and ileostomy.

Patient with colostomy or ileostomy may be concerned about excessive gas formation and stool odor. Foods that can cause gas and odor include alcohol, asparagus, beans, broccoli, Brussels sprouts, cabbage, cauliflower, eggs, fish, and onion.[101] Intolerances to beans, legumes, fiber, high-fat foods, lactose, caffeine, and sorbitol should be assessed. Some eating and drinking practices can contribute to increased swallowed air, such as chewing gum, using straws, smoking, chewing tobacco, eating quickly, and carbonated beverages.[101] Foods that may help decrease stool odor include buttermilk, parsley, yogurt, kefir, and cranberry juice. Foods that may thicken stool include banana flakes, applesauce, pectin, pasta, potatoes, and cheese.[102]

Ileostomy

Nutrition Therapy	MNT Goals
• Consider underlying disease leading to ileostomy • Diet modifications to address odor and GI symptoms, including malabsorption due to GI motility • Fluid, electrolytes: increased losses • Micronutrients: specific to portion of GI tract resected (i.e. how much ileum was resected) ○ Monitor vitamin D, vitamin B12, other B vitamins, vitamin K • Restrict foods high in oxalates • Assess for lactose intolerance and fat malabsorption	• Maintain fluid and electrolyte balance • Provide nutrition education on strategies to reduce odor in ostomy output if needed • Work with patient to alleviate GI symptoms such as odor, gas, and diarrhea

Surgical resection of the colon and rectum (proctocolectomy) requires the formation of an ileostomy, where the end of the ileum is surgically attached to a stoma or used to form a pouch in place of the rectum. Resection of the terminal ileum can result in electrolyte, vitamin, and mineral deficiencies. The ileocecal valve controls movement from small intestine to the large intestine and when removed, GI motility can be faster than normal which can inhibit normal absorption.[103]

Nutrition therapy should consider the underlying disease that required the bowel resection as well as the extent of bowel resection. Underlying diagnoses may include IBD, radiation enteritis, GI malignancy, and/or ischemic bowel.[104] Nutrition therapy following ileostomy should consider postoperative healing, adequate nutrient absorption, electrolyte and fluid balance, monitoring

vitamin B12 and vitamin D status, and alleviation of symptoms such as odor, gas, and diarrhea. Postoperatively, patients with ileostomy begin with clear liquids, progress to a low-fiber nutrition diet, and then return to a normal diet with small frequent meals, limiting co-intake of fluids at meals.[105] Foods high in oxalate should be restricted and the patient should be educated on signs and symptoms of lactose intolerance and fat malabsorption. Oral rehydration beverages and adding salt to food may be helpful to maintain fluid and electrolyte balance.

Read this case study write-up by Meagan Bridges, Roseann Nasser and Carol Rees Parrish: High Output Ileostomies: the Stakes are Higher than the Output. Practical Gastro. 2019. Volume XLIII, Issue 9. https://practicalgastro.com/2019/09/23/high-output-ileostomies-the-stakes-are-higher-than-the-output/

Cardiovascular Disease

The 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease emphasizes 10 take-home messages, most of which emphasize lifestyle changes that are under the RDNs purview:[106]

1) Promoting a healthy lifestyle throughout life is the most important way to prevent cardiovascular disease.
2) Clinicians should use a team-based care approach and evaluate social determinants of health to inform treatment decisions.
3) Adults aged 40-75 should undergo 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimation and have a clinician-patient discussion before starting pharmacological therapy. Clinicians should also assess for other risk-enhancing factors such as coronary artery calcium (CAC) scanning that could guide decisions about preventive intervention in some individuals.
4) All adults should consume a healthful diet that emphasizes vegetables, fruits, nuts, whole grains, lean vegetable or animal protein, fish, and minimizes trans fats, red meat, processed meats, refined carbohydrates, and sugar-sweetened beverages. Adults with overweight and obesity should receive counseling and education on a reduced calorie diet to achieve and maintain weight loss.
5) Adults should engage in ≥150 minutes of moderate intensity physical activity per week (or 75 minutes vigorous intensity physical activity).
6) Lifestyle changes for adults with type 2 diabetes mellitus are crucial to manage cardiovascular risk. If indicated, metformin should be used as first-line therapy followed by SGLT2 or GLP1 receptor agonists.
7) Clinicians should inquire about tobacco use at every healthcare visit and strongly advise tobacco users to quit.
8) There is a lack of net benefit in using aspirin as routine primary prevention of ASCVD and its use should be infrequent.
9) Patients with LDL-C ≥ 190 mg/dL, those with diabetes mellitus, adults between 40 and 75 years, and those at sufficient ASCVD risk should be prescribed statin therapy for primary prevention.
10) All adults with elevated blood pressure or hypertension should be advised on nonpharmacological interventions.

You can read the guideline here: Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines.

Circulation. 2019;140(11):e596-e646. doi:10.1161/CIR.0000000000000678 PMID: 30879355 PMCID: PMC7734661. Journal Link: https://www.ahajournals.org/doi/10.1161/CIR.0000000000000678

You should be familiar with the modifiable and nonmodifiable cardiovascular risk factors and have a general understanding of their role in increasing ASCVD risk (next page)

Table 20. Cardiovascular Risk Factors

Risk Factor	Comment
Increased age	Men: increased risk ≥ 45 years; Women: increased risk after menopause
Environment and occupation	Exposure to air pollution, toxins, radiation, or other hazards, sedentary occupation, high stress occupation (working > 55 hours per week, shift work, and night shifts that disrupt sleep)
Family history of early heart disease	Early heart disease defined as diagnosis before age 55 (men) or age 65 (women)
Genetics	Apolipoprotein E (apoE), Apolipoprotein B (apoB), MTHFR
Lifestyle Risk factors	
Physical inactivity	Can contribute to or worsen dyslipidemia, hypertension, diabetes and prediabetes, and overweight and obesity
Insufficient sleep	Poor sleep and sleep apnea associated with increased cardiovascular risk
Tobacco use or secondhand exposure	Particularly cigarettes
Stress	Can contribute to coronary microvascular disease and may lead to smoking or poor diet
Unhealthy eating patterns	Unvaried diets, diets high in saturated fats and refined carbohydrates can contribute to overweight and obesity, dyslipidemia, and atherosclerosis
Other Medical Conditions	
Atherosclerosis	Build-up of fatty plaques in arteries
Autoimmune and inflammatory diseases	Crohn's disease, ulcerative colitis, psoriasis, lichen planus, pemphigus, histiocytosis, lupus, rheumatoid arthritis
Chronic kidney disease	eGFR < 60 mL/min, microalbuminuria
Congenital coronary artery defects	Aortic stenosis, atrial septal defect, coarctation of the aorta, Ebstein anomoly, patent ductus arteriosus, patent foramen ovale, tetralogy of Fallot, truncus arteriosus, ventricular septal defect
Diabetes	Fasting blood glucose ≥126 mg/dL (among other diagnostic criteria – see Diabetes section).
Dyslipidemia	High blood LDL-cholesterol, high triglycerides, and/or high total cholesterol
Hypertension	Untreated high blood pressure can damage blood vessels and organs. See Hypertension section for detailed definitions
HIV/AIDs	Particularly among older adults with some of the risk due side effects of HIV treatments
Mental health conditions	Anxiety, depression, and PTSD
Overweight and obesity	Defined as BMI >25 kg/m^2
High waist circumference	Men > 40 in, Women > 35 in
Sleep disorders	Sleep apnea, sleep deprivation, and sleep deficiency
Sex-specific conditions	Endometriosis, gestational diabetes, polycystic ovary syndrome, preeclampsia, early menopause

Adapted from Coronary Heart Disease, National Heart, Lung, and Blood Institute. https://www.nhlbi.nih.gov/health-topics/coronary-heart-disease Accessed 18 May 2021

Biochemical data the RDN should review includes a fasting serum lipid panel, vitamin D, fasting blood glucose, hemoglobin A1C, CRP, liver function tests, coagulation profile, BUN, creatinine, sodium, potassium, and when available, lipoprotein(a), apo A1 and apoB, Lp-PLA2, and insulin sensitivity measures.[107] Common cut-offs for lipid values are presented in Table 21.

Table 21. Cholesterol and Triglyceride Lab Values

	Total cholesterol	LDL-C	HDL-C	Triglycerides	
Desirable	<130	<100	≥40 (men); ≥50 (women)	Normal	<150
Above desirable	130 – 159	100 – 129			
Borderline high	160 – 189	130 – 159		Borderline high	150 – 199
High	190 – 219	160 – 189		High	200 – 499
Very high	≥ 220	≥ 190		Very high	≥ 500

All values in mg/dL. LDL-C: LDL cholesterol. HDL-C: HDL-cholesterol. Adapted from Nutrition Care Manual.[107]

Hyperlipidemia

The “cardioprotective dietary pattern” is used to manage blood lipid levels and reduce risk of coronary heart disease.[108,109]

- Saturated and trans fats < 7% energy intake
- Total fat 25-35% of total energy intake
- Cholesterol < 200 mg/day
- Plant stanols and sterols (2-3 g/day)
- Reduced calorie diet for weight loss if indicated
- Antioxidant-rich foods (whole grains, nuts, fruits, vegetables)
- Limited refined carbohydrates and added sugars
- Omega-3 fatty acids and/or monounsaturated fats (in the form of fatty fish or plant-derived omega-3 fatty acid foods)
- Emphasis on fiber (25-38 g (men) and 21-25 g (women) total fiber per day) with half from soluble fiber
- Alcohol limited to <2 drinks/day for men and <1 drink/day for women
- Resistance exercise ≥2 days per week and 30 mins moderate-intensity physical activity for 30 mins on most if not all days

A physician may recommend omega-3 fatty acid supplements.

Hypertension

The 2017 ACC/AHA Guidelines on Hypertension published new guidelines for hypertension classification (normal, elevated, stage 1 hypertension, and stage 2 hypertension) (see Table 22).[110] The previous 2003 JNC7 classification included ‘normal’, ‘prehypertension’, stage 1, and stage 2 hypertension with different cut-offs (Table 22).

Table 22. Blood Pressure Cut-offs for 2003 JNC7 and 2017 ACC/AHA Hypertension Guidelines

SBP		DBP	2003 JNC7	SBP		DBP	2017 ACC/AHA
<120	and	<80	Normal	<120	and	<80	Normal
120 – 139	or	80 - 89	Prehypertension	120 – 129	and	<80	Elevated
140 – 159	or	90 – 99	Stage 1	130 – 139	or	80 – 89	Stage 1
≥ 160	or	≥ 100	Stage 2	≥ 140	or	≥ 90	Stage 2

How would the following patients be classified according to the 2003 versus 2017 hypertension guidelines?

- *Patient A: 120 / 85*
- *Patient B: 135 / 75*
- *Patient C: 122 / 70*
- *Patient D: 145 / 85*
- *Patient E: 165 / 90*
- *Patient F: 145 / 100*

The Dietary Approaches to Stop Hypertension (DASH) Eating Plan emphasizes increased fruits, vegetables and whole grains; and limiting fats and/or replacing saturated fats with unsaturated fats (Table 23). It is very similar to the cardioprotective dietary pattern discussed in the hyperlipidemia section previously. Specifically, the DASH Eating Plan limits sodium to 1,500 – 2,300 mg per day with an emphasis on foods high in potassium, calcium, magnesium, and fiber.[109]

The RDN should provide nutrition education on suggested servings of food groups that meet the DASH guidelines for different calorie levels (guides available in the USDA Dietary Guidelines). The patient should decide what goals they want to focus on and which changes they will implement to make small changes over time. For example, a patient may begin with a low-sodium diet and progress to the major dietary changes introduced by the DASH Eating Plan.

Table 23. The Dietary Approaches to Stop Hypertension (DASH) Eating Plan

Food Group	Servings	Food Group	Servings
Grains	6-8 daily	Meats, poultry, fish	6 or less daily
Vegetables	4-5 daily	Fruit	4-5 daily
Dairy (low-fat, fat-free)	2-3 daily	Fats and oils	2-3 daily
Fats and oils	2-3 daily	Sodium	1,500 – 2,300 mg daily
Nuts, seeds, dry beans, peas	4-5 weekly	Sweets	5 or less weekly

Adapted from DASH Eating Plan, National Heart, Lung, and Blood Institute. https://www.nhlbi.nih.gov/health-topics/dash-eating-plan Accessed 18 May 2021.

Heart Failure

Nutrition Therapy[111]	MNT Goals
• Energy: determine via indirect calorimetry when possible • Protein: 1.12 g/kg (normally nourished) – 1.37 g/kg (stable depleted patient) • Fluid: <2 L/day depending on clinical symptoms	• Provide nutrition education on limiting dietary sodium in the diet (food choices, nutrition labels, seasoning substitutes) and on the DASH diet • Provide education on fluid restrictions • Recommend small frequent meals for patients with early satiety, dyspnea, and/or fatigue

Heart failure is a condition when the heart can't pump enough blood for what the body needs. The heart may be too weak to pump properly or it may not fill up with enough blood.[112] Heart failure can be acute or chronic with or without acute exacerbations. Symptoms include fatigue, dyspnea, fluid overload, poor appetite, nausea, and malnutrition. The condition is progressive and life-threatening. Targeted nutrition therapy aims at providing sufficient calories and protein while typically limiting fluid and sodium.

The New York Heart Association (NYHA) functional classification of heart failure consists of 4 classes based on symptom severity and exercise intolerance (Table 24).

Table 24. New York Heart Association (NYHA) Heart Failure Classification

	Class I	Class II	Class III	Class IV
Symptoms	Ordinary physical activity does not cause fatigue, palpitation, dyspnea	Comfortable at rest. Ordinary physical activity causes fatigue, palpitation, dyspnea	Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea	HF symptoms at rest
Exercise	No limitation	Slight limitation	Marked limitation	Unable to carry on any physical activity without discomfort

Patients with heart failure are at risk of cardiac cachexia and malnutrition, both of which are associated with poor prognosis.[113] They can also experience rapid weight changes due to excess fluid. Energy estimates using predictive equations should use an estimated dry weight. MNT for heart failure can reduce rehospitalizations, with recommended frequency of encounters ranging from monthly to twice a month depending on disease severity.[114]

Medical management may include angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blocker (ARB), β blocker, diuretic, as well as medications for dyslipidemia, hypertension, diabetes, thyroid dysfunction, and/or kidney disease, if present.[115] If the patient is prescribed an anticoagulant the RDN should provide nutrition education regarding dietary sources of vitamin K, if relevant.

Coronary Heart Disease

Nutrition Therapy[109]	**MNT Goals**[116]

• Energy: RMR* x 1.3 (or reduced for weight loss)
• Protein: 0.8 – 1 g/kg
• Therapeutic Lifestyle Changes
  ○ Fat: 25 – 35% total energy needs
  ○ Fiber: 25-30 g/day
  ○ Saturated fat: <7% total energy needs
  ○ Cholesterol: <200 mg/day
  ○ Alcohol: ≤2 drinks/day (men), ≤1 drink/day (women)
  ○ Sodium: ≤ 2,000 mg/day
	• Promote healing and recovery following surgery (if applicable) and emphasize importance of lifestyle changes to maintain artery health and prevent further disease progression[117]
	• Support modification of risk factors (LDL-C, blood pressure)
	• Provide nutrition education about Therapeutic Lifestyle Changes, food label reading, healthful food choices, and physical activity

*RMR estimated with Mifflin-St. Jeor equation. Activity factor of 1.3 for sedentary.

Table 25. Mifflin-St. Jeor equation to estimate RMR

Men	(10 x Weight) + (6.25 x Height) – (5 x age) + 5
Women	(10 x Weight) + (6.25 x Height) – (5 x age) - 161

Weight in kg, height in cm, age in years

Coronary heart disease, also called coronary artery disease, occurs when the arteries that deliver oxygen and nutrients to the heart muscle (coronary arteries) is partially or fully blocked due to atherosclerosis. Clinically, coronary heart disease can manifest as chest pain, a myocardial infarction, or sudden cardiac arrest. It can also progress without symptoms.[118] Treatment includes heart-healthy lifestyle changes, medications, and/or surgery such as coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI).

Therapeutic Lifestyle Changes (TLC) Eating Plan include:[119]

- Saturated fat <7% daily total energy intake
- Cholesterol <200 mg/day
- Fat 25-35% total energy intake, emphasizing unsaturated fats, particularly omega-3 fatty acids
- Carbohydrates 50-60% total energy intake, with 25-30 g/day fiber (50% as soluble)
- Protein 15% total energy intake
- Adequate energy intake to maintain a healthy body weight and prevent weight gain
- Moderate physical activity to expend at least 200 kcal per day
- Plant stanols and sterols 2-3 g/day

For patients recovering from surgery, additional nutrition interventions may be required.

Respiratory Disease

Nutrition Therapy	MNT Goals
Energy: 25 – 35 kcal/kg*	• Individualize calorie prescription and macronutrient composition based on nutrition assessment (energy intake compared to estimated needs, and weight status)

Medical nutrition therapy to improve patient outcomes (such as body weight status, quality of life, exercise capacity, and body composition) is recommended for patients with chronic respiratory disease like COPD and cystic fibrosis.[120] The RDN should assess current energy intake compared to estimated needs, assess and monitor weight status, and individualize nutrition prescription to optimize patient outcomes.[121] High-fat, low-carbohydrate diets and enteral formulas for acute respiratory disorders are not recommended.

COPD

Nutrition Therapy[122]	MNT Goals[122]
• Energy: ~30 kcal/kg • Protein: 0.8 g/kg/day (DRI) • Vitamin D supplementation if 25(OH)D ≤10 ng/mL • Fluid: Modify DRI for treatment and environment, monitor for fluid overload (I&Os, weight change, S&S, labs)	• Maintain or restore nutrition status using food and beverage intake with or without supplements. • Prevent weight loss even in overweight patients and maintain or restore lean body mass • Liberalize diet order or develop individualized eating plan to meet energy and nutrition needs with small frequent small meals and snacks with foods that are easy to chew, swallow, and digest.

I&O: intake and output records, S&S: signs and symptoms.

Chronic obstructive pulmonary disease (COPD) is a term used to describe several respiratory disorders characterized by progressive obstruction and inflammation of the airways. The patient experiences increased airway resistance due to reduced lung elasticity, increasing the work required to breathe.

Symptoms include dyspnea, wheezing, persistent cough (with sputum), increasing respiratory rate (tachypnea; >20 breaths/min at rest), lung hyperinflation (barrel chest), diminished breath sounds with or without crackles and wheezes, and cyanosis.[123]

Patients are at increased nutritional risk due to diminished appetite, diminished ability to shop for food and prepare meals, inability to sleep, exhaustion unrelated to lack of sleep, and breathlessness. All of these factors contribute to chronic and acute malnutrition due to coexisting inadequate intake with increased energy needs due to inflammation and increased work breathing.[124]

COPD patients with a BMI < 20 kg/m^2 or those who experience significant weight loss have a higher risk of COPD-related death compared to COPD patents with a BMI between 25 and <30 kg/m^2.[124,125] MNT should focus on preventing weight loss and accounting for increased resting energy expenditure. Patients with COPD may undergo a bone density screening due to increased risk of vertebral fractures and osteoporosis. In the nutrition assessment, the RDN should evaluate lab values for hemoglobin, hematocrit, serum iron, serum electrolytes, serum

proteins, pH, pO2, PaCo2, immunologic testing results, creatinine height index, nitrogen balance, and serum 25(OH)D levels.

Patients on oxygen should be observed during meal time, as food consumption may be leading to oxygen desaturation.[126] Nutrition interventions include small, frequent, small meals focusing on foods that are easy-to-chew, swallow, and digest as well as nutrient-dense foods or medical food supplements to help meet nutrient and energy needs orally. If medically feasible, previous diet orders should be liberalized to promote oral intake. Enteral nutrition would be considered if oral intake is inadequate and not expected to improve soon or if the patient requires intubation or artificial ventilation.[122] Oral intake should be adjunctive to enteral nutrition when possible.

ARDS

Nutrition Therapy	MNT Goals[128]
• Energy: 25 – 35 kcal/kg* • Protein: 1.5 – 2 g pro/kg • Enteral formula with dietary fish oil (EPA) and borage oil (GLA)	• Provide adequate energy and protein via enteral nutrition to promote nutritional status and prevent weight loss, even in overweight and obese patients • Transition to high-calorie/high-protein diet upon ventilation weaning and improved respiratory and mental status

*Perform indirect calorimetry to determine calorie need and avoid both underfeeding and overfeeding. EPA: eicosapentaenoic acid; GLA: gamma-linolenic acid.

Acute respiratory distress syndrome (ARDS) is a serious lung disease. A less severe form is called “acute lung injury” (ALI). Both involve lung parenchyma inflammation, pulmonary edema, and increased permeability of the pulmonary capillaries, which impairs gas exchange. Because most patients with ARDS and ALI are mechanically ventilated and, due to their respiratory distress, these patients require nutrition support.[127]

While most patients are hypermetabolic, the RDN should not risk overfeeding these patients as the excessive CO_2 produced can prevent respiratory recovery and ventilator weaning. That being said, ICU patients receiving enteral nutrition do not typically receive all of their prescribed formula, and so the RDN should closely monitor enteral nutrition delivery to ensure the patient is not being underfed. The systemic inflammatory response means that ARDS and ALI patients have a higher protein requirement than a typical adult. To monitor protein status, check urinary urea nitrogen (UUN) levels.

While the general recommendation for enteral nutrition for critically ill patients is to *not* use an immune-enhancing formula, the recommendation differs for patients with ARDS and ALI.[128] For these patients, an enteral formula enriched with dietary fish oil (EPA), borage oil (GLA), and enriched levels of antioxidant vitamins should be started within 24 hours of ICU admission.

Cystic Fibrosis

Nutrition Therapy	MNT Goals[129]
• Energy and protein: increased (~1.2 - 2 x DRI) • Fat-soluble vitamins • Zinc supplementation Monitor growth in children and adolescents – some CF patients may have normal growth with RDA for calories and protein Energy needs may increase during exacerbation but may be offset by reduced activity level	• Increase caloric and fat intake through high-calorie and high-fat foods and nutrition supplements. • Supplement oral intake with enteral nutrition if BMI <19 kg/m^2 or <10th percentile [calorically dense formula >1 kcal/mL with pancreatic enzyme replacement, or semi-elemental formula] • Provide patient education on meeting increased nutritional requirements, correcting or preventing nutritional deficiencies, and compensating for increased losses of fat and salt

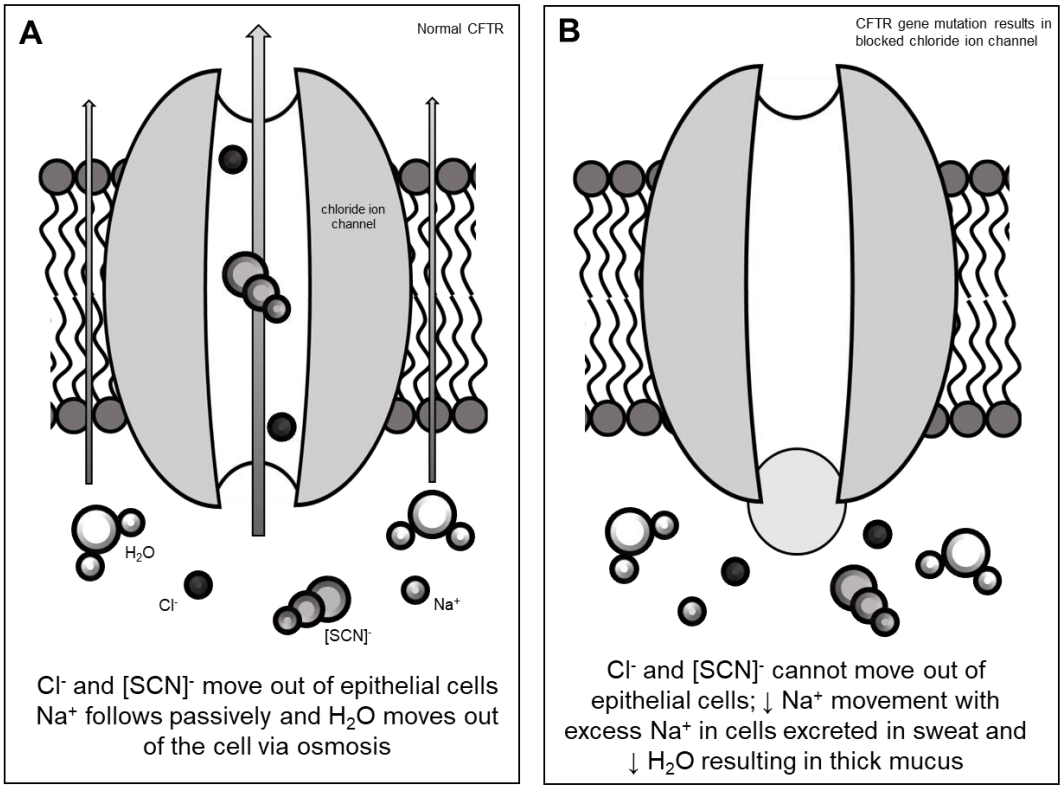


Figure 10. The CFTR gene codes for chloride ion channels which is found in the epithelial cells of the lungs, liver, pancreas, digestive tract, skin, and reproductive tracts. The chloride ion channel allows for negatively charged chloride (Cl-) and sodium thiocyanate (SCN-) to move out of epithelial cells. Sodium (Na+) follows passively and water moves out of the cell via osmosis (Panel A). The CFTR gene mutation results in a dysfunctional chloride ion channel and without the negative ion movement, excess sodium builds up in the cells and is excreted in sweat and insufficient water results in thick sticky mucus that presents with respiratory and pancreatic symptoms (Panel B). Figure created by Bailey DeBarmore. Do not reproduce without permission.

Cystic fibrosis (CF) is caused by gene mutations in the CFTR gene (cystic fibrosis transmembrane conductance regulator). This gene codes for a chloride ion channel that creates sweat, digestive juices, and mucus but the defect means that the mucus produced in the lungs and GI tract is unusually thick and sticky, because chloride ions and water cannot move through

the blocked channels (Figure 10).[130] Respiratory symptoms include a chronic cough and frequent respiratory infections. Digestive symptoms include maldigestion and malabsorption due to inability to produce necessary digestive enzymes, either at all or in sufficient amounts, as well as difficulty in transporting these digestive enzymes to the duodenum. Thickened secretions can also lead to liver damage and pancreatic damage (pancreatic insufficiency, CF-related diabetes). As lung function continues to decline, patients may receive a double-lung transplant.

CF is diagnosed by analyzing the patient's sweat, with an elevated sweat chloride level on at least 2 separate occasions considered confirmatory. In the nutrition assessment, the RDN should assess for poor oral intake, diarrhea, steatorrhea, or change in stool appearance, macronutrient and micronutrient content of the patient's diet, abdominal pain, weight loss, trouble gaining weight, poor growth, and reduced skeletal muscle mass (skin-fold measurements).[131]

MNT is focused on increasing calorie and fat intake using energy-dense foods. Energy and protein needs are increased (about 1.2 to 2 times estimated REE) due to increased work of breathing as well as maldigestion/malabsorption. The RDN should provide nutrition education on how to meet these increased requirements while also correcting or preventing nutritional deficiencies and counseling on how to compensate for fat and salt loss.

If the patient has low weight (BMI <19 kg/m^2) or poor growth (<10th percentile), enteral nutrition may be used, typically through a G-tube. Calorically dense enteral formulas (>1 cal/mL) are typically available only as complete formulas and the patient should use them with pancreatic enzyme replacements before and after dispensing enteral nutrition.[129] Alternatively, the patient can use a semi-elemental formula, which is not calorically dense but does not require pancreatic enzyme replacement. An RDN that works regularly with CF patients would come to specialize in this area, beyond what you would need to know for this exam.

Pneumonia

Nutrition Therapy[133]	**MNT Goals[133]**
• Energy: 25 – 35 kcal/kg • Protein: 1 – 1.5 g pro/kg (1.6 – 2 g/kg if septic) • Monitor UUN levels • Avoid overfeeding	• Provide adequate energy and protein via liberal diet order, small frequent snacks, and foods that are easy to chew, swallow, and digest • Promote nutritional status and prevent weight loss, even in overweight patients

Pneumonia is a general term used for lung inflammation and is most commonly caused by bacteria or viruses. Symptoms include cough, chest pain, fever, dyspnea, poor appetite, as well as nausea/vomiting.[132] When symptoms do not resolve at home with oral antibiotics, rest, and fluids, or in patients with pre-existing breathing problems or comorbidities, hospital admission is typically required.[132] Patients in the hospital may develop hospital-acquired pneumonia. Pneumonia is diagnosed based on symptoms and physical findings, including chest X-ray results.

Malnutrition and weight loss plays a role in the development of and recovery from pneumonia. While most patient with pneumonia are hypermetabolic, their protein needs are not substantially

increased, particularly compared to patients with ARDS. However, if the patient has another stressed condition, such as sepsis, then protein needs are increased up to 2 g/kg. Pneumonia symptoms like loss of appetite, shortness of breath, nausea, and vomiting can interfere with oral intake.[133] The RDN should recommend frequent small meals/snacks and foods that are easy-to-chew, swallow, and digest. If respiratory function deteriorates to the point that the patient requires mechanical intubation or ventilation then enteral nutrition should be initiated. Formula choice should depend on the digestive and absorptive ability of the GI tract.[133]

Liver Disease

Nutrition Therapy[134]	**MNT Goals**[135]
Energy: Estimate with Mifflin-St. Jeor using dry weight + 20% Some patients may require 30 – 35 kcal/kg Protein: 1 – 1.2 g/kg End-stage liver disease may require food restrictions	• Maintain or approach healthful weight while providing adequate balance of macronutrients and micronutrients • Address feeding problems • Monitor symptoms, lab values, and/or comorbidities that may require changes in the nutrition prescription

Table 26. Mifflin-St. Jeor equation to estimate RMR

Men	(10 x Weight) + (6.25 x Height) – (5 x age) + 5
Women	(10 x Weight) + (6.25 x Height) – (5 x age) - 161

Weight in kg, height in cm, age in years

Hepatitis refers to liver inflammation and can be due to alcohol toxicity, infection (hepatitis A, B, C, D, or E), medications, or fatty liver. Hepatitis can progress to fibrosis and then cirrhosis. Acute hepatitis refers to hepatitis caused by one of the hepatitis viruses. Infection with hepatitis B or C in particular, is associated with progression to fibrosis and cirrhosis. Hepatitis can also be chronic. See Table 27 for a description as well as signs and symptoms of each stage of liver damage/disease.

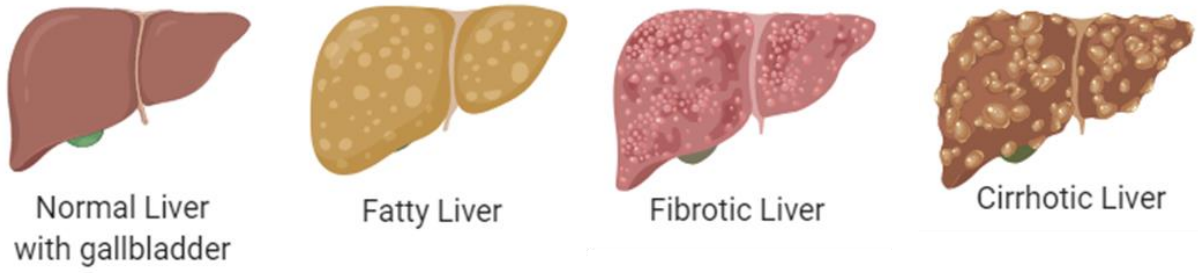


Figure 11. Different stages of liver disease. Images courtesy of www.biorender.com.

The liver plays a central role in nutrient metabolism and regulation, including producing bile to help absorb dietary fats, cholesterol, and fat-soluble vitamins; storing nutrients filtering blood; enzyme production and activation; protein synthesis (e.g. albumin); and creating blood clotting proteins. Hepatitis and liver disease impair these functions and can lead to weight loss and muscle loss due to inadequate nutritional intake, and/or abnormal nutrient digestion, absorption,

and/or metabolism of macronutrients and micronutrients, particularly vitamins ADEK, thiamin, folic acid, pyridoxine, zinc, magnesium, copper, and iron.[136] Inadequate nutritional intake may be due to loss of appetite, early satiety (particularly with ascites), fatigue, nausea, vomiting, abdominal pain, or food restrictions. Nutrition interventions should consider the multiple factors that may contribute to malnutrition in patients with hepatitis or liver disease.

Table 27. Stages of Liver Damage and Liver Disease

Stage		Description	Signs and Symptoms
Hepatitis	Inflammation	Injury to normal liver results in chronic inflammation; injury can include damage from alcohol, infection (like hepatitis viruses), nonalcoholic fatty liver disease, or from metabolic disorders, genetic disorders, or autoimmune disorders	Flu-like symptoms, fatigue, dark urine, abdominal pain, poor appetite, jaundice, weight loss, *acholic* stool (pale stool due to lack of bile)
Fibrosis	Scarring	Inflammation leads to scarring and nodules, and damaged tissue doesn't work as well. Fibrosis can regress to a health liver or progress to cirrhosis and liver failure.	Poor appetite, jaundice, ascites, edema, nausea, weight loss
Cirrhosis	Extensive scarring	Liver function deteriorates as scarring increases. The liver shrinks and hardens leading to portal hypertension.	Portal hypertension, esophageal varices, spider angiomas, confusion, jaundice, poor appetite, ascites, edema, nausea, weight loss, gastritis, pancreatitis

Food and fluid restrictions may be required to manage help symptoms for patients with end-stage liver disease. These may include <2,000 mg sodium, <30% energy as fat (if patient has steatorrhea), and any modifications required for hyperglycemia. **Error! Bookmark not defined.** Due to n ausea, fatigue, and early satiety, 4-6 small frequent feedings including a bedtime snack can help promote adequate energy and protein intake and minimize loss of lean body mass.**Error! B ookmark not defined.**

What types of feeding issues might a patient with liver failure have?

Pancreatitis

Nutrition Therapy[137]	**MNT Goals[138]**
Energy: use indirect calorimetry when possible, particularly for patients with obesity, shock, multiple organ failure, and/or mechanical ventilation Increased energy and protein needs with moderate – severe pancreatitis • Energy: 25 – 35 kcal/kg • Protein: 1.2 – 1.5 g/kg Early enteral nutrition (within 48 hrs) for: • severe acute pancreatitis • those with significant malnutrition or with inadequate oral intake for 5-7 days	Acute • Progress from NPO to oral feeds without symptom exacerbation • Provide adequate energy and protein to prevent deficiency and maintain or improve nutritional status • Prevent weight loss • Initiate early enteral nutrition if indicated Recovery • Replenish nutritional deficiencies that occurred during acute phase • Encourage avoidance of alcohol and excessive fat intake to prevent recurrence of symptoms

Pancreatitis can be acute or chronic. The main risk factors for acute pancreatitis are gallbladder disease (biliary tract obstruction) and alcoholism.[139] Other risk factors include obesity and certain drugs, such as furosemide and tetracycline. Risk factors for chronic pancreatitis include cystic fibrosis, hypertriglyceridemia, hypercalcemia, renal failure, and infections. With chronic pancreatitis, repeated inflammation in the pancreas leads to tissue damage and can ultimately result in diabetes due to impaired endocrine function. Malnutrition and micronutrient deficiency are important concerns in chronic pancreatitis.

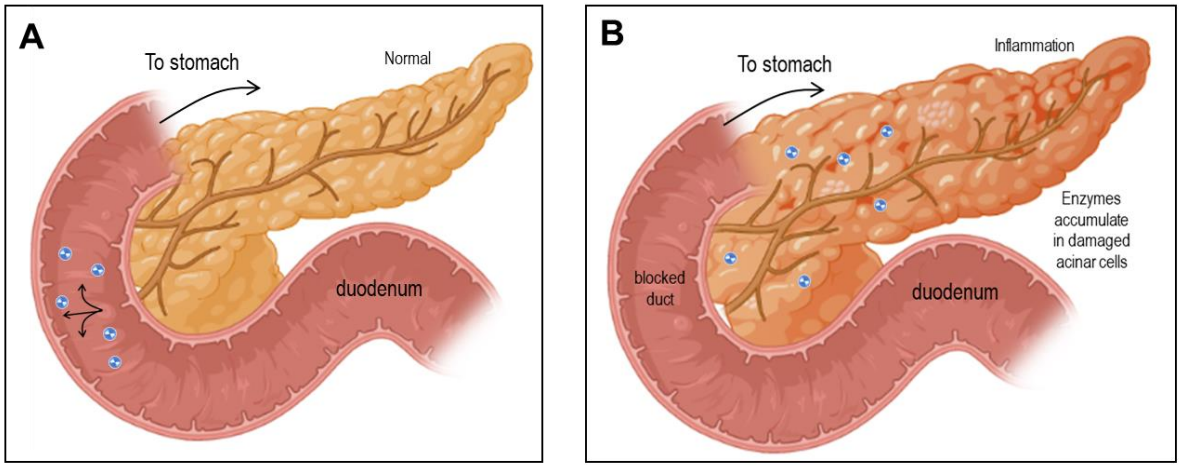


Figure 12. Simplified illustration of normal pancreas releasing enzymes into duodenum (Panel A) and inflamed pancreas with accumulated enzymes in damaged acinar cells (Panel B). Images adapted from www.biorender.com.

Treatment for pancreatitis is divided into the acute exacerbation phase and recovery phase and is also divided by severity (mild, moderate, and severe). During the acute phase, the goal is to prevent weight loss and maintain nutritional status as much as possible. The recovery stage should be used to replenish any nutritional deficiencies as well as encourage avoidance of exacerbating factors, such as alcohol and excessive fat intake.[138]

Current guidelines recommend that mild to moderate pancreatitis cases remain NPO and then progress to an oral diet as tolerated (Figure 13). For patients that present with malnutrition, or are unable to progress to an adequate oral diet within 5 – 7 days should begin enteral nutrition within 48 hours of admission (early enteral nutrition).[138] Patients with severe pancreatitis should also be started on early enteral nutrition, once hemodynamically stable.

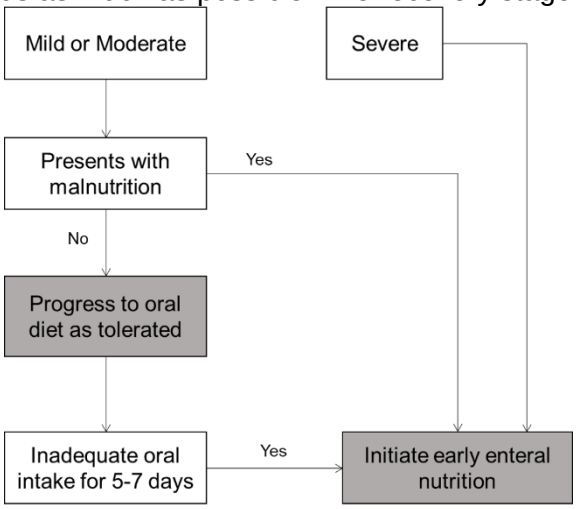


Figure 13. Simplified flow chart for deciding initial MNT for patients with pancreatitis. Patients with mild or moderate pancreatitis who present without malnutrition should progress to an oral diet as tolerated, while those who are malnourished should receive early enteral nutrition support (within 48 hours of admission). If patients do not achieve adequate oral intake within 5 – 7 days they should progress to enteral nutrition. Patients with severe pancreatitis should initiate early enteral nutrition, once hemodynamically stable.

Kidney Disease

Acute Kidney Injury

Acute kidney injury (AKI) is an *acute* change in kidney function (within 48 hours) defined as an absolute increase in serum creatinine of at least 0.3 mg/dL, a percentage increase in serum creatinine of 50% or more, or documented oliguria < 0.5 mL/kg/h for > 6 hours.[140] *If you have difficulty digesting large chunks of text, break out lists and definitions into visuals like Figure 14 for AKI.*

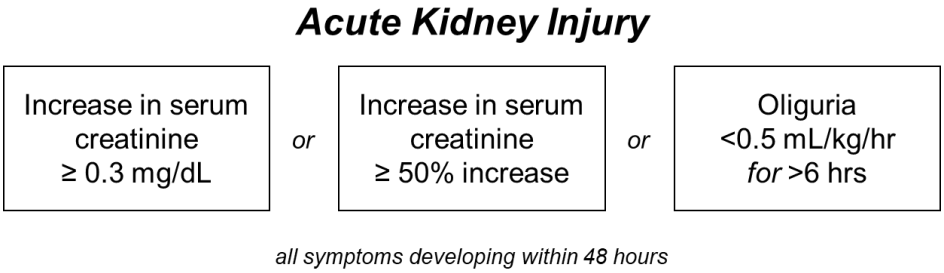


Figure 14. Diagnostic criteria for acute kidney injury, based on acute change in kidney function with acute = 48 hours.

Patients with AKI may be hypercatabolic, particularly when the cause of AKI is shock, sepsis, rhabdomyolysis, infection, hypotension, surgery, or the patient had inadequate protein or energy intake prior to AKI or preexisting malnutrition.[141] Energy and protein requirements increase with the degree of catabolism and with treatment (hemodialysis or continuous renal replacement therapy).[142] *For the exam, you should be familiar with how AKI is defined, and how it differs from chronic kidney disease.*

Chronic Kidney Disease

Chronic kidney disease is defined by glomerular filtration rate (GFR) in 5 stages per the National Kidney Foundation. The KDIGO 2012 categories include a crosstab of eGFR and albuminuria. *Albuminuria* is albumin in the urine. Healthy kidneys don't allow albumin to pass into the urine (or only in small amounts) so increasing amounts of albumin in the urine can be used as an indicator of kidney damage.

Nephrotic syndrome refers to damage to the kidney's filtering units, evidenced by proteinuria, including albuminuria. It is a contributing factor to chronic kidney disease and other types of renal disorders.[143] Note that normal eGFR is ≥ 60 (seen in Stage 1 and Stage 2), and thus kidney disease at that stage would be diagnosed by other signs of kidney damage, like albuminuria or physical damage.

	Stage 1	Stage 2	Stage 3A	Stage 3B	Stage 4	Stage 5
eGFR*	≥ 90	60 – 89	45 – 59	30 – 44	15 – 29	< 15

Estimated glomerular filtration rate in units of mL/min/1.73m^2

The kidneys have several important functions in the body, including stimulating erythropoiesis, and maintaining acid-base balance, electrolyte balance, mineral balance, and fluid balance, among others. In CKD, these functions are disturbed and treatment for CKD is often aimed at correcting this dysfunction.

Nutrition Therapy[144]	**MNT Goals**[145]
<u>CKD Stage 3-5</u> • Protein: 0.55 – 0.6 g/kg (0.6 – 0.8 g/kg with diabetes) • Energy: 25 – 35 kcal/kg • Potassium modifications (high or low) • Limit phosphorus and sodium intake • Oral nutrition supplements if indicated • Manage blood pressure and glucose <u>CKD Stage 5D (dialysis)</u> • Protein: 1 – 1.2 g/kg • Energy: 25 – 35 kcal/kg • Potassium modifications (high or low) • Limit phosphorus and sodium intake • Fluid restriction if indicated • Calcium modifications (↑ with diet, supplements, or ↓ with Ca^{2+}-binders) • Oral nutrition supplements if indicated • Manage blood pressure and glucose	• Provide nutrition education on a healthful diet with emphasis on balanced food options that still meet potassium, phosphorus, and sodium modifications • Assess readiness to change nutrition-related behaviors as well as health literacy and exposure to misinformation • Connect patient with community nutrition resources if indicated

Patients with CKD may have anemia due to iron deficiency and/or due to reduced production of erythropoietin (EPO), a hormone synthesized by the kidney that stimulates bone marrow erythropoiesis.[145] Patients may receive EPO-stimulating agents, oral or IV iron, iron-based phosphate binders, or other iron-related medications.

Excessive potassium in the blood is dangerous for the heart. The RDN should provide nutrition education to the patient with CKD on low-potassium food choices while still maintain a balanced diet. The patient may also be prescribed potassium-removing agents, such as certain diuretics, that increase potassium excretion. Note that some patients may have low potassium due to high doses of these medications and might need to increase dietary intake of potassium.[145]

CKD-bone mineral disease occurs due to altered mineral metabolism, including phosphate, calcium, and vitamin D. The patient may be prescribed phosphate binders or niacinamide to decrease GI phosphorus absorption and the medications should be taken with meals.[145] Phosphate binders may be iron-based or calcium-based. Calcimimetic agents bind to calcium receptors on the parathyroid gland, reducing PTH levels which contribute to bone mineral disease.[145] The patient may also receive vitamin D supplementation. *Review the role of PTH, vitamin D, phosphate, and calcium in bone health.*

Patients with CKD may present with cachexia due to uremia and/or malnutrition, sarcopenic obesity, bone pain or injuries, cardiovascular abnormalities including dyspnea, bradypnea or tachypnea, bradycardia or tachycardia, and fluid retention that can lead to heart failure and trouble breathing.[146] Patients with CKD may have a number of GI symptoms due to kidney

dysfunction, uremia, medication side/effects and/or fluid overload. These symptoms may include poor appetite, early satiety, diarrhea, constipation, abdominal pain and/or bloating, heartburn, nausea, and vomiting.[146] Excess sodium, fluid, and hyperglycemia can result in peripheral and orbital edema. Patients may also experience "clouded consciousness" due to uremia, dizziness, trouble chewing or swallowing, hair and nail changes, dry skin and pruritis, and dysgeusia.[146]

What is uremia? What inborn error of metabolism also leads to uremia?

What types of nutrition-impact symptoms might a CKD patient have? What food/nutrient-delivery interventions could be prescribed to address these symptoms?

Organize the information in the previous few paragraphs (nutrition-related impact of CKD) into a table or a diagram.

Renal Replacement Therapy

Renal replacement therapy includes continuous renal replacement therapy (CCRT), hemodialysis, and peritoneal dialysis. CCRT is primarily used to treat patients with acute renal failure (acute kidney injury) who are hemodynamically unstable (and thus need dialysis at bedside rather than having intermittent hemodialysis).[147] Hemodynamically stable patients with AKI may receive in-hospital hemodialysis.

Hemodialysis and peritoneal dialysis are used to treat CKD. Hemodialysis is most common and is usually performed at a dialysis center several times a week. Home dialysis or daily in-center dialysis is also used. Vascular access for hemodialysis may be a central line catheter, separate vein- and artery-access points in the forearm, or the patient may undergo surgery to have an AV fistula or graft created in their forearm. Hemodialysis uses machines with pumps, special membranes, and built-in safeguards to protect the patient from air bubbles and other dangers. Blood is removed from the body, 'cleaned' in the dialyzer, and returned to the body.

Peritoneal dialysis involves infusing dialysate into the patient's peritoneum through a surgically placed catheter (Figure 15). The concentration of dextrose (glucose) in the dialysate creates an osmotic gradient that draws excess fluid and toxins from the body into the dialysate, which is then discarded. Glucose absorption from the dialysate should considered when estimating energy requirements and intake, and varies by dextrose concentration, dwell time, and the number and volume of exchanges. Peritoneal dialysis can be done manually during the day, as shown in the figure, or it can be done at night using a machine.

Read the following articles from the National Institutes of Diabetes and Digestive and Kidney Diseases:

- Peritoneal Dialysis: https://www.niddk.nih.gov/health-information/kidney-disease/kidney-failure/peritoneal-dialysis
- Hemodialysis: https://www.niddk.nih.gov/health-information/kidney-disease/kidney-failure/hemodialysis

For the RD exam, you should know that while a protein-restricted diet is used for CKD Stage 3+, once a patient starts dialysis, their protein requirement increases. *Why?*

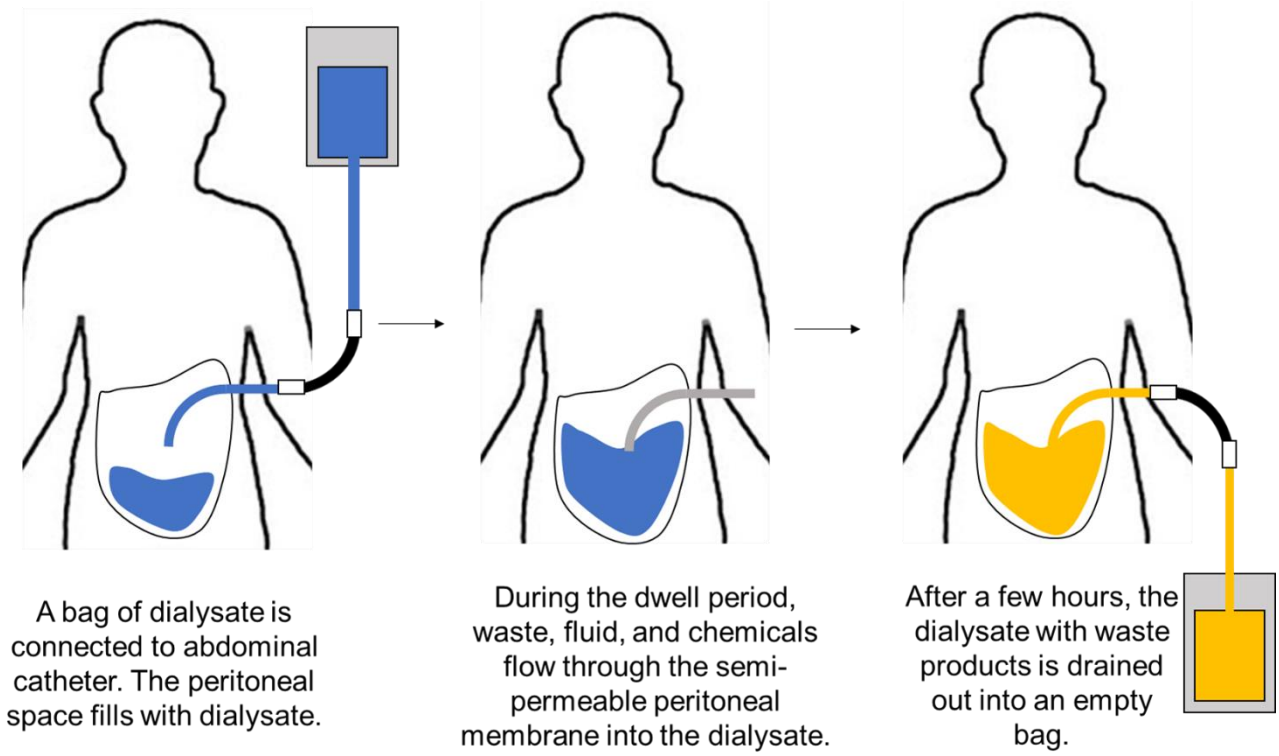


Figure 15. Schematic representation of peritoneal dialysis. Patients treated with peritoneal dialysis have a permanent catheter placed in their abdomen that coils into their peritoneal space, which surrounds the abdominal organs. The peritoneal membrane is semipermeable. A fresh bag of dialysate is hung at the beginning of a session and the abdomen fills with fluid. The dialysate is allowed to "dwell" for a certain amount of time, during which waste products, fluid, and chemicals flow from the blood through the semipermeable membrane and into the dialysate. The dialysate contains dextrose to create an osmotic gradient. After the dwell period, the dialysate with waste products is drained into an empty bag. Figure created by Bailey DeBarmore. Do not reproduce without permission.

Kidney Stones

Kidney stones, or urolithiasis, are the presence of *calculi* in the urinary tract. The calculi, or kidney stones, may be made of calcium oxalate, calcium phosphate, uric acid, ammonium urate, struvite, or cystine. Calcium oxalate stones are the most common in the US.[148]

Risk factors for developing kidney stones include: high sodium intake, high oxalate intake, high animal protein intake, low calcium intake, low fluid intake, history of UTIs, gout, obesity, bowel disease, sedentary lifestyle, restricted eating patterns or eating disorders, chronic diarrhea due to malabsorption or short bowel syndrome, excessive vitamin C consumption, and history of previous kidney stones (**Error! Reference source not found.**).[149]

Table 28. Risk factors for Developing Kidney Stones (Urolithiasis)

Risk Factors for Urolithiasis	
Diet	High sodium, high oxalate, high animal protein, low calcium
Conditions	History of UTIs, history of kidney stones, gout, obesity, bowel disease
Lifestyle	Sedentary, low fluid intake, eating disorder or restricted eating pattern
Other factors	Chronic diarrhea due to malabsorption, excessive vitamin C supplementation

How would chronic diarrhea due to malabsorption increase the risk of kidney stones?

The RDN should inquire about sodium, oxalate, animal protein, calcium, and fluid intake as well as medical conditions or surgeries that can impact risk of kidney stones. For example, excessive use of salt or soy sauce, regular intake of processed foods or salted snack foods, daily intake of high-oxalate foods, >8 oz animal protein daily, little to no dairy, and inadequate fluid (especially water) intake are dietary risk factors.[150]

Nutrition intervention for kidney stones depend on the urinary risk factor and suspected cause. Patients with kidney stones may also need MNT for diabetes, CKD, CVD, or other conditions and the RDN should provide nutrition education that synthesizes urolithiasis MNT with nutrition therapy for those conditions.[151]

Neurological and Cerebrovascular Disease

Epilepsy

Recommended readings:

- Roehl K and Sewak S. Practice Paper of the Academy of Nutrition and Dietetics: Classic and Modified Ketogenic Diets for Treatment of Epilepsy. J Acad Nutr Diet. 2017;117:1279-1292. DOI: 10.1016/j.jand.2017.06.006 PMID: 28754198. PDF: https://bit.ly/3fp0P20
- Randall R and Groveman S. The Ketogenic Diet for Epilepsy. Today's Dietitian. May 2016. Vol 18:5; 46. https://www.todaysdietitian.com/newarchives/0516p46.shtml

If you have not already, read the Practice Paper on the use of ketogenic diets for treatment of epilepsy (cited above).

Epilepsy is a chronic neurologic disorder that causes seizures, and is diagnosed as 2 or more unprovoked seizures at least 24 hours apart.[152] Physicians have long observed that periods of fasting are effective at treating epilepsy. In the early 1900s, physicians observed that ketosis without malnutrition was effective at treating epilepsy (rather than prolonged fasting or starvation). The "classic ketogenic diet" proposed by Dr Wilder of Mayo Clinic and his colleagues consists of 1 g protein/kg, 10-15 g carbohydrate/day and the remaining calories from fat. When phenytoin, an anti-epileptic medication, was discovered in 1938, the use of ketogenic diet to treat epilepsy declined. The use of modified ketogenic diets, easier in compliance than the classic diet, became popular once again in the 1990s and includes the Modified Atkins Diet and the low glycemic index treatment (LGIT).

A ketogenic diet is any diet that includes a dietary composition that would result in a ketogenic state, and is typically high-fat, low-carbohydrate, and moderate protein so that the body

metabolizes fat instead of glucose to mimic the metabolic state of fasting.[153] RDNs are instrumental in implementing a ketogenic diet to ensure that sufficient nutrient intake is achieved to ensure healthy growth and development of children with epilepsy. Ketogenic diets are often described in terms of their ratio of fat (g):non-fat (g) where non-fat refers to combined protein and carbohydrate. For example, a classic ketogenic diet might be a 3:1 ratio with 3 g fat:1 g protein and carbohydrate while the Modified Atkins Diet is typically a 1:1 ratio.[153] While the classic ketogenic diet requires precise measurement of all food and beverages consumed, the Modified Atkins Diet and LGIT utilize portion sizes and are thought to be easier to implement and adhere to. [153]

The interdisciplinary team of health care practitioners needed to meet the needs of a patient with epilepsy on a ketogenic diet includes a neurologist and nurse as well as a dietitian who specializes in ketogenic diets. [153] Pharmacists, case workers, and social workers as well as community-based health care practitioners are also valuable on the interdisciplinary team.

Ketogenic diet therapy is typically *not* the first-line treatment for epilepsy but rather it is used in children with seizures not well-controlled on current medications.[154] Risks of ketogenic diet therapy include hypoglycemia, acidotic dehydration, hypokalemia, kidney stones, hyperlipidemia, hyperuricemia, constipation, GERD, and pancreatitis.[154] Vitamin and mineral supplementation with carbohydrate-free fillers are used to ensure micronutrient intake is adequate, particularly of iron, calcium, vitamin C, vitamin D, vitamin B complex, and selenium.[155] The diet is considered a complex meal regimen and is typically initiated with a hospital admission to monitor for side effects. [154] Ketogenic diet therapy is primarily used in children but its use in adolescents and adults it being explored.

Nutrition Therapy	**MNT Goals**
• Ketogenic diet if indicated Have family complete a 3-day diet record before starting ketogenic diet therapy and use to determine daily calorie requirements along with RDAs for age and weight	• Assess for conditional contraindications to a ketogenic diet and discuss with interdisciplinary team • Provide detailed nutrition education on a classic or modified ketogenic diet if agreed to by team and patient • Provide nutrition education on the risks of ketogenic diet therapy and how to reduce risks • Use self-monitoring data to monitor blood glucose readings and hunger levels • Monitor weight gain and linear growth in children and adolescents • Discuss micronutrient supplementation regimen

Absolute contraindications to a ketogenic diet include defects in fatty acid metabolism (see Figure 3 in the Practice Paper[153]). Considerations that warrant further workup before initiating a ketogenic diet include failure to thrive, dysphagia, GI issues, limited food acceptance (picky eating), extreme dyslipidemia, cardiomyopathy, renal disease, liver disease, baseline metabolic acidosis, and social constraints such as access to food/kitchen, multiple caregivers and/or unstable home environment and caregiver support and compliance (see Figure 4 in the Practice Paper[153]).

Read the Today's Dietitian article listed in the Recommended Readings. Complete the 10 multiple choice questions at the end of the article.

Stroke and TIA

Nutrition Therapy	MNT Goals[156]
• Energy: for weight maintenance or weight loss if overweight or obese • Fat: healthful, unsaturated fat sources	• Provide adequate nutrition via oral or enteral nutrition to optimize recovery and reduce likelihood of future cardiovascular events • Utilize food and/or nutrient delivery interventions that prevent aspiration and compensate for dysphagia • Provide nutrition education on food sources of vitamin K and other foods that may interfere with medications • Provide nutrition education on reading nutrition labels, identifying foods high in saturated fat, cholesterol, and sodium, and recipe modification

Cerebrovascular accidents (CVAs) include hemorrhagic stroke, ischemic stroke, and transient ischemic attacks (TIA). Hemorrhagic stroke occurs when a brain blood vessel bursts. High blood pressure, smoking, use of illegal drugs, and medications that cause bleeding are risk factors for hemorrhagic stroke.[157]

Ischemic stroke occurs when there is a lack of blood flow to the brain due to an embolus or thrombus. Atherosclerosis involves plaques in the arteries of the body. A thrombotic stroke occurs when a cerebral arterial plaque ruptures and obstructs the narrowed artery. An embolic stroke occurs when a plaque ruptures in a non-cerebral artery and the resulting clot travels to the brain, blocking blood flow. When brain ischemia is *transient* rather than permanent, the event is called a 'transient ischemic attack' or mini-stroke. Risk factors for ischemic stroke and TIA are the same as risk factors for other manifestations of cardiovascular disease, including hypertension, diabetes, smoking, and dyslipidemia.[157]

Some patients have limited or no disability following a stroke while others may have severely limited ability to feed themselves, consume a normal diet, or consume sufficient nutrition, often due to motor issues and/or dysphagia. Texture-modified diets, nutrition supplements, and in some cases, enteral nutrition may be indicated. [157]

Nutrition therapy involves addressing modifiable risk factors and interventions that modify food/nutrient delivery if indicated. Modifiable risk factors include diet high in sodium, saturated fat, cholesterol; smoking; hypertension; dyslipidemia; lack of physical activity; obesity; diabetes; and alcohol use (>2 drinks/d for men and >1 drink/d for women).[158]

Patients taking blood thinners, such as warfarin, should be educated on food sources of vitamin K.[156] Patients taking certain statins and blood pressure lowering medications should be educated on the potential interaction of grapefruit juice with their medication.[159]

Alzheimer's Disease and Dementia

Dementia is a collective term for brain disorder symptoms. The death of nerve cells and/or the loss of communication between nerve cells leads to dementia, manifesting as impaired intellectual functioning. Dementia is not a normal part of aging and can affect adults of any

age.[160] Alzheimer's disease is the most common form of dementia. Early-onset dementia refers to the disease in adults less than 50 years of age.

To diagnose dementia, there must be "significant impairment" of ≥ 2 intellectual functions: memory, language skills, perception, cognitive skills (reasoning, judgement).[160] Dementia can affect any area of the brain, and the area affected manifests in accordance to the function of that brain location. All changes affect the ability the eat food and meet nutrient needs.

Alzheimer's disease onset is typically gradual and is described in five stages. As memory, language skills, loss of time and space perception, thinking, and behavior irreversibly decline, activities of daily living are impaired and various behavioral and psychiatric symptoms appear.[161]

Nutrition interventions should address the identified nutrition problems. Examples include modified consistency diets if there is difficulty chewing or swallowing, liberalization of therapeutic diets to promote food intake, and tailored interventions specific to the issues that patient is experiencing.[162]

Read the article, 'Caring for Dementia Patients' by Laura Hilliard, MS RD LDN CSG CDP from the August 2013 issue of Today's Dietitian.
https://www.todaysdietitian.com/newarchives/080113p64.shtml

Metabolic and Endocrine Disorders

Obesity

Nutrition Therapy	MNT Goals
Comprehensive weight management program including diet, physical activity, and behavior therapy MNT for at least 6 months or until weight loss goals are achieved with weight maintenance program afterwards	• Individualize approach with goal of losing up to 10% of initial weight in first 6 months, basing energy needs on indirect calorimetry or Mifflin-St. Jeor using actual weight • Provide nutrition education on portion control, food selection, reading labels, recipe modification, and discourage extremes and fad diets

Recommended Reading: Position of the Academy of Nutrition and Dietetics: **Interventions for the Treatment of Overweight and Obesity in Adults**. J Acad Nutr Diet. 2016;116:129-147. PMID: 26718656. http://dx.doi.org/10.1016/j.jand.2015.10.031

You should be familiar with the Mifflin-St Jeor equation to estimate resting metabolic rate. Multiply the resulting RMR by an activity factor of 1.3 for sedentary individuals, or higher for more active individuals. Subtract 500 kcal per day for 1 lb/week weight loss goal.

Table 29. Mifflin-St. Jeor equation to estimate RMR

Men	(10 x Weight) + (6.25 x Height) – (5 x age) + 5
Women	(10 x Weight) + (6.25 x Height) – (5 x age) - 161

Weight in kg, height in cm, age in years

Current research demonstrates that energy balance (energy intake and expenditure) does not fully account for weight regulation in patients who struggle with obesity.[163,164] Energy homeostasis is also affected by the nervous system (hypothalamus, autonomic nervous system) and hormones. Adipose tissue releases hormones and cytokines (angiotensinogen, PAI-1, leptin, adiponectin, resistin, TNF-α, and interleukin-6). Eating behaviors are influenced by our environment, both family and societal (portion sizes, healthful cooking versus eating out, cost of whole versus processed foods, access to healthy foods).[164] The RDN must consider all factors contributing to overweight and obesity, including previous attempts at weight loss.

Overweight and obesity are defined using body mass index. However, the RDN should be aware that BMI does not distinguish between lean mass and fat mass. A very muscular person may have a BMI that classifies them as obese while another person with disproportionate central adiposity may have a normal BMI but still be at higher metabolic risk due to a high waist circumference. In addition to measuring BMI, the RDN should also monitor waist circumference.

What are the high-risk waist circumference cut-offs for men and women?

Table 30. Body Mass Index and Waist Circumference Classifications

BMI Classification	BMI (kg/m^2)
Underweight	< 18.5
Normal weight	18.5 - 24.9
Overweight	25 - 29.9
Obesity (Class 1)	30 - 34.9
Obesity (Class 2)	35 - 39.9
Extreme obesity (Class 3)	> 40

Waist Circumference	
Men	≥ 40 in (102 cm)
Women	≥ 35 in (88 cm)

The RDN should be aware of mental health issues such as clinical depression, bulimia nervosa, binge eating disorder, and anorexia nervosa before recommending a weight loss intervention.[165] Treatment in these scenarios emphasizes the need for an interdisciplinary care team and specialized training. In the nutrition assessment, the RDN can screen for clinical depression using the Center for Epidemiologic Studies Depression Scale (CES-D), as well as screen for binge eating disorder and other eating disorders before recommending a nutrition intervention.[165] If any of these screens are positive, the patient should be referred back to a physician before proceeding with a nutrition intervention.[165]

As part of a comprehensive weight management program that addresses diet, physical activity, and behavior therapy, the RDN should utilize multiple behavior therapy strategies such as self-monitoring, stress management, stimulus control, problem solving, contingency management, cognitive restricting, and social support. A reduced calorie diet should be implemented by developing a healthful eating plan *with* the patient that distributes energy intake throughout the day in conjunction with individualized physical activity goals (after approval by physician on the healthcare team). Other considerations such as bariatric surgery and use of FDA-approved weight loss medications should factor into the Nutrition Care Process.

For a patient who regularly consumes sugar-sweetened beverages, developing strategies with the patient to replace these with water or calorie-free beverages would be a key step in

developing an individualized plan. For a patient that struggles with portion control, meal replacements can be used to substitute 1-2 daily meals or snacks.[166] For a patient who enjoys cooking, the RDN can provide nutrition education on food label reading and recipe modification such as choosing low-fat dairy, lean meats and replacing solid fats with heart-healthy oils.

More information related to the Nutrition Care Process, energy need estimation, and nutrition interventions is provided in the Nutrition Care study guide.

You should be familiar with the Mifflin-St Jeor equation to estimate resting metabolic rate. Multiply the resulting RMR by an activity factor of 1.3 for sedentary individuals, or higher for more active individuals. Subtract 500 kcal per day for 1 lb/week weight loss goal.

Table 31. Mifflin-St. Jeor equation to estimate RMR

Men	(10 x Weight) + (6.25 x Height) – (5 x age) + 5
Women	(10 x Weight) + (6.25 x Height) – (5 x age) - 161

Weight in kg, height in cm, age in years

Bariatric Surgery

Nutrition Therapy	MNT Goals
• Energy: Measure with indirect calorimetry or use Mifflin-St. Jeor with actual body weight and activity factors* • Protein: 1.1 - 1.5 g pro/kg IBW • Micronutrients: Tailor to prevent or correct deficiencies using DRIs as baseline	• Address preoperative and postoperative nutrition needs • Provide nutrition education related to postoperative bariatric diet • Prevent or correct micronutrient deficiencies

*Significant reduction in calorie needs compared to presurgery RMR – ranging from 12-21% reduction in RMR[167]

Review the section on Gastric Surgeries in addition to this section on bariatric surgery as many of the nutritional therapies and symptoms, such as dumping syndrome, are similar. Not all bariatric surgeries are gastric resections, however. You should be familiar with the indications for bariatric surgery, recognize the types of surgeries, know if they are successful in reducing comorbidities and weight loss, and know the general nutritional considerations for post-operative care.

Specific eligibility criteria for bariatric surgery are related to surgery candidacy as well as insurance coverage. The American Society for Metabolic and Bariatric Surgery (ASMBS) summarizes 3 common qualifications:[168]

1. BMI ≥ 40 or more than 100 pounds overweight
2. BMI ≥ 35 with ≥1 obesity-related comorbidity (T2DM, hypertension, sleep apnea, nonalcoholic fatty liver disease, osteoarthritis, lipid abnormalities, GI disorders, heart disease)
3. Inability to achieve healthy weight loss sustained for a period of time with prior weight loss efforts

The ASMBS does not provide additional specifics on how “>100 pounds overweight” is determined, thus it could be 100 lbs over ideal body weight (IBW), a weight that corresponds to a normal BMI, etc.

Contraindications to bariatric surgery include any contraindications to receiving general anesthesia, as well as severe heart disease, end-stage lung disease, active cancer treatment, drug and/or alcohol dependency, portal hypertension, and for some surgeries, Crohn’s disease.[169]

Table 32. Common Types of Bariatric Surgery Performed in the United States.

Bariatric Surgery	Description	Characteristics
Laparoscopic Adjustable Gastric banding (LAGB)	Places an adjustable band around the top of the stomach, limiting food passage from the stomach into the intestine and increasing feelings of fullness	Restrictive
Roux-en-Y gastric bypass (RYGB)	Creates a stomach pouch ~ size of an egg and creates a roux limb of the small intestine to bypass a large portion of the small intestine	Restrictive, malabsorptive
Sleeve gastrectomy	Removes 80% of the stomach leaving a “banana shaped” pouch	Restrictive
Biliopancreatic diversion ± duodenal switch (BPD, BPD-DS)	Removes part of the stomach and attaches the remnant stomach to the ileum, with the rest of the small intestine used to pass pancreatic and bile juices	Restrictive, malabsorptive

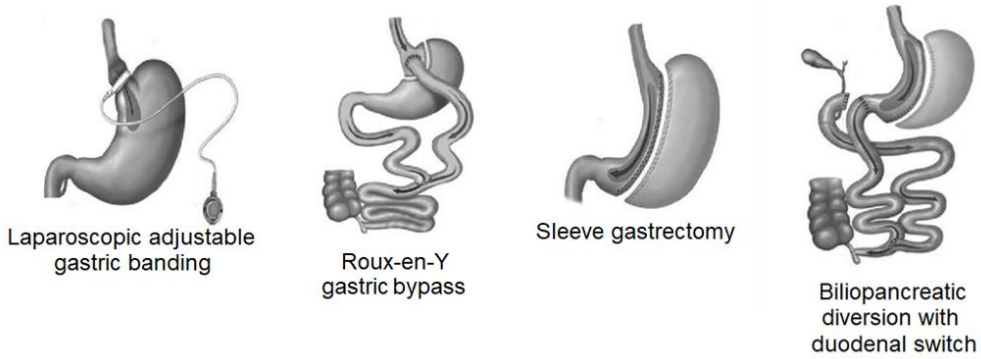


Figure 16. Common types of bariatric surgery. Adapted from Shaui X, Tao K, Mori M, and Kanda T. Bariatric Surgery for Metabolic Syndrome in Obesity. Metab Syndr Relat Disord. 2015. 13(4):149-160. PMID: 25715110

Weight loss seems to be highest in Roux-en-Y gastric bypass and lowest in the adjustable gastric banding.[170] Adjustable gastric banding requires frequent follow-up for band adjustment and behavioral modification for success such as mindful eating, thorough chewing, and avoiding foods that can obstruct the band outlet.

Immediately after surgery, many patients see resolution or improved severity of diabetes and hypertension. Hyperlipidemia and sleep apnea also improve. Weight loss following surgery can improve nonalcoholic fatty liver disease and joint pain.

All bariatric patients should be assessed for micronutrient deficiencies preoperatively in order to minimize postoperative deficiencies. Some micronutrient deficiencies are associated with obesity due to inflammation, medications, hyperinsulinemia, or adipose tissue activity.[171,172]

Read the American Society or Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient, 2016 Update on Micronutrients here: https://bit.ly/3sRgrAe or via PMID: 28392254

Read about Bariatric Surgery by clicking through the pages on the National Institute of Diabetes and Digestive and Kidney Diseases website: https://www.niddk.nih.gov/health-information/weight-management/bariatric-surgery/definition-facts

Nutrition therapy and education after surgery may include addressing inadequate weight loss, weight regain, dehydration, reactive hypoglycemia (dumping syndrome), pregnancy, constipation, diarrhea, dysphagia, and micronutrient deficiencies.[173]

Diabetes Mellitus

Recommended reading: Position of the Academy of Nutrition and Dietetics: **The Role of MNT and RDNs in the Prevention and Treatment of Prediabetes and Type 2 Diabetes**. J Acad Nutr Diet. 2018;118:343-353. PMID: 29389511. https://doi.org/10.1016/j.jand.2017.11.021

Type 1 Diabetes

Nutrition Therapy	MNT Goals[174]
DRIs • Energy: meet needs • Protein: 0.8 g/kg/day • Fluid: ~1 mL/kcal/day	• Develop eating plan and insulin regimen that fits with patient's food and physical activity preferences • Respect the patient's wishes, willingness, and motivation to change • Ensure adequate energy in eating plan for normal growth and development (youth), optimal outcomes (pregnant and lactating), and appropriate weight (adults) • Provide self-management education related to hypoglycemia, acute illness, and exercise • Prevent and treat chronic complications of diabetes (dyslipidemia, CVD, hypertension, nephropathy, neuropathy)

While Type 1 diabetes can manifest at any age, it is most commonly diagnosed in patients under 30 years of age. Patients with type 1 diabetes require insulin to prevent diabetic ketoacidosis (DKA) and death and the first MNT priority is to identify an eating plan that integrates an insulin regimen into the patient's typical eating habits and physical activity schedule.[175] Around half of a patient's insulin is given as basal, or background, insulin and the other half is bolus insulin given at meal times. To calculate how much insulin should be bolused, patients can use carbohydrate counting with insulin:carbohydrate ratios or an insulin scale. Carbohydrate counting can be used with exchange lists for meal planning (Table 33), where the RDN can provide nutrition education on serving sizes of various food groups that equal the approximately 12 – 15 g. Conversely, the patient can read nutrition labels to determine what serving size corresponds to ~12 – 15 g carbohydrate.

Type 1 diabetes is often diagnosed during acute illness and/or due to a DKA episode, requiring hospitalization. For a newly diagnosed type 1 diabetes patient, typically while in the hospital the RDN only has time to conduct a nutrition assessment and provide basic nutrition education.[176] In the hospital, MNT goals are to optimize glycemic control, meet metabolic demands through sufficient energy, and to include a plan for follow-up MNT care in the discharge plan. In the outpatient setting, at least 3 – 4 MNT encounters in the first 6 months should be conducted, with additional follow-up at least once a year for adults, but typically more frequently for children and adolescents due to changing nutrient needs with growth.

Table 33. Exchange List for Meal Planning

Food Group	Carbs	Protein	Fat	Calories	Example Serving Sizes
Starch	15 g	0 – 3 g	0 – 1 g	80 cal	½ cup cooked cereal, grain, or starchy vegetable; ½ cup cooked rice or pasta; 1 oz bread (1 slice, ½ hotdog bun), ¾ - 1 oz snack food
Fruits	15 g	-	-	60 cal	½ cup canned or fresh fruit, ½ cup fruit juice, 4 oz fresh fruit (1 small apple), 2 T dried fruit
Milk					½ pint, 8 fluid oz, 1 cup Ex: 1 cup yogurt, 1 cup milk Cheese listed with meat and meat substitutes; cream listed in fats
Whole	12 g	8 g	8 g	160 cal	
2%	12 g	8 g	5 g	120 cal	
Skim, 1%	12 g	8 g	0 – 3 g	100 cal	
Sweets	15 g	Varies	Varies	Varies	Includes sugar-sweetened beverages, cakes, cookies, pies, puddings, candy, condiments, pastries, frozen desserts, granola bars
Non-starchy vegetables	3+ cups of raw or 1 ½ + cups cooked vegetables count as 1 starch				½ cup cooked vegetables, ½ cup vegetable juice, 1 cup raw vegetables
Meat & meat substitutes					
High-fat meat	-	7 g	8+ g	100 cal	2 slices bacon, 1 oz cheese, 1 hot dog
Medium-fat meat	-	7 g	4 – 7 g	75 cal	1 oz medium-fat meat, 1 oz cheese (feta, mozzarella, low-fat cheese), ¼ cup ricotta cheese, 1 egg
Lean meat	-	7 g	0 – 3 g	45 cal	1 oz lean meat or fish, 1 oz cheese (<3 g fat/oz), 2 egg whites, ¼ cup cottage cheese
Plant-based protein	Varies	7 g	Varies	Varies	Some count as starch + meat combos depending on carbohydrate and fat content
Fats	-	-	5 g	45 cal	1 ½ tsp nut butters, 1 oz avocado, 1 tsp butter, 1 slice bacon, 2 T cream, 2 T sour cream, 1 tsp mayo
Alcohol	Varies	-	-	100 cal	½ oz absolute alcohol has ~100 cal; 12 fl oz beer counts as 1 alcohol + 1carb; 5 fl oz wine counts as 1 alcohol, 1.5 oz distilled spirits count as 1 alcohol
"Free" foods	≤ 5 g			< 20 cal	Includes some raw fruits and vegetables, condiments, seasonings, and drinks/mixes

Adapted from Exchange Lists for Meal Planning, Krause's Food and the Nutrition Care Process. 2012.

Pathophysiology

The majority of patients with type 1 diabetes have *immune-mediated diabetes mellitus* meaning that the etiology of the disease is due to genetic predisposition and autoimmune destruction of pancreatic islet β cells.[177] The other form of type 1 diabetes is idiopathic type 1 diabetes, where idiopathic means no known etiology.

In contrast to type 2 diabetes, type 1 diabetes has an abrupt onset of clinical signs and symptoms of hyperglycemia (polyuria, polydipsia, polyphagia, weight loss) and if left untreated develops into dangerous DKA.

"Latent autoimmune diabetes of adults" is the term used for adult-onset type 1 diabetes. These patients are typically not obese, over the age of 35, and positive for one of the five autoantibodies that contribute to β cell destruction.[177] These patients don't typically present with weight loss or ketoacidosis and can be managed on oral glucose-lowering medications rather than insulin, initially. However, compared to patients with type 2 diabetes (who are negative for the autoantibodies), they typically progress to insulin therapy quicker. [177]

Insulin deficiency affects not only carbohydrate metabolism but also protein and lipid metabolism. Insulin prevents tissue breakdown and promotes energy storage and helps cells uptake nutrients.[177] In Table 34, the Normal column describes insulins actions. The Type 1 Diabetes column describes what happens when there is not insulin.

Table 34. Comparing the role of insulin and the effect of insulin efficiency in type 1 diabetes

	Normal	Type 1 Diabetes
Carbohydrate	Glucose uptake by cells ↑ glycogenesis (liver, muscle) Excess glucose stored in adipose as triglycerides (lipogenesis)	↑ gluconeogenesis (liver) ↑ glycogenolysis (liver, muscle)
Protein	↓ blood amino acid levels ↑ tissue synthesis ↓ gluconeogenesis	↑ gluconeogenesis (liver) ↑ proteolysis
Lipid	Inhibits lipolysis ↑ lipogenesis (liver, adipose tissue)	↑ ketogenesis (liver) ↑ lipolysis (adipose tissue)

Adapted from the Nutrition Care Manual, Academy of Nutrition and Dietetics. Accessed 13 May 2021.

To learn more about the effect of type 1 diabetes in the body, review the "How the Body Processes Sugar" module from the UCSF Diabetes Education Center: https://dtc.ucsf.edu/types-of-diabetes/type1/understanding-type-1-diabetes/how-the-body-processes-sugar/.

Take out a sheet of paper. Write down and then define the following terms: gluconeogenesis, glycogenesis, glycogenolysis, proteolysis, ketogenesis, lipogenesis, lipolysis. When do they typically occur in the body?

Insulin and Insulin Analog Therapy

Insulin is divided into categories based on how quickly it acts: rapid-acting or prandial, short-acting, intermediate-acting (regular), and basal (long-acting or ultra-long acting). Patients may also have access to pre-mixed insulin (combination of rapid-acting and intermediate-acting insulin) that spreads insulin throughout the day. See Table 36 for a summary of the insulin types. Finally, some medications combine pre-mixed insulin with GLP-1 receptor agonists, such as Degludec/Liraglutide (Xultophy®) and Glargine/Lixisenatide (Soliqua™). These insulin/GLP-1 receptor agonist combinations seem to have less weight gain and hypoglycemia adverse effects compared to prandial + basal insulins.[178]

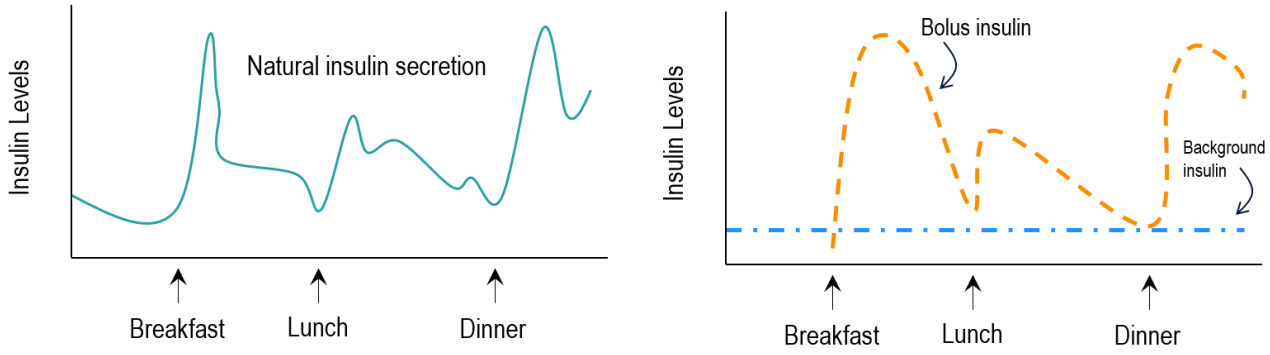


Figure 17. Intensive insulin therapy (basal-bolus) attempts to duplicate the body's natural pattern of insulin secretion. background, or basal, insulin is used to provide a low steady level of insulin overnight, while fasting, and between meals, with a rapid surge of insulin at mealtime

Insulin onset, peak, and duration are dependent on the insulin dose given with a larger dose of resulting in a longer duration of action. Prandial insulin is typically used to refer to rapid-acting insulin, with an onset of 5 to 15 min, peaking at 1 hr, and lasting 3 to 5 hours. Ultra-rapid acting insulin is also technically a prandial insulin, with an onset of 2.5 minutes, peaking at 1 hr, and lasting 5 – 6 hrs. It should be taken at the beginning of a meal or within 20 minutes of beginning to eat. Short-acting, or regular, insulin has an onset of 1 hr, peaks at 2-4 hrs, and lasts 5-8 hrs. Compared to rapid-acting insulin, short-acting insulin has a higher risk of hypoglycemia.

Intermediate-acting insulin (NPH), unlike the others mentioned so far, is cloudy. It has an onset of 1 to 2 hours, with a peak around 8 hours and lasts up to and sometimes beyond 14 hours. It is typically given before breakfast and again at bedtime. Patients using NPH may need an afternoon snack to avoid hypoglycemia at the 8 hour peak. Pre-mixed insulin, such as NPH/Regular 70/30 and Lispro Protamine/Lispro 50/50, combine insulin for two peaks so that the patient does not need to give multiple injections. Onset is between 15 and 1 hour and there may be two peaks corresponding to the two types of insulins. Note that the onset, peak, and duration provided in the table are generalizations. If

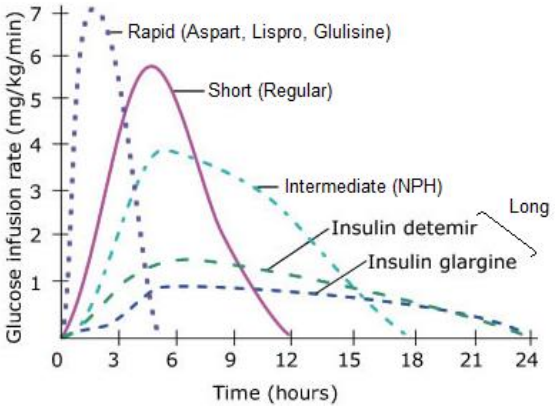


Figure 18. Activity profiles of different types of insulin. Adapted from Diabetes Teaching Center (UCSF). Accessed 12 May 2021.

you find a resource that says the peak is 1 – 4 hrs when the table below says 1 – 3 hrs, do not fret. The important thing to know for the exam is that prandial has a peak of *about 1 – 3 hrs* compared to *regular insulin* which has a slightly longer peak. If you go on to work frequently with diabetes patients, you will likely earn an advanced credential and will 1) be much more knowledgeable about insulins used and 2) be able to work with your client to understand their onsets, peaks, and durations so they can individualize their management plan.

Glycemic targets for nonpregnant patients with type 1 diabetes should be individualized to age and health status with the goal of maintaining normal growth and development (in youth) and minimizing risk of both severe hyperglycemia and hypoglycemia. Glycemic control is measured using hemoglobin A1c, which is the amount of glycosylated hemoglobin present in red blood cells. Because red blood cells have a lifespan of 2-3 months, hemoglobin A1c represents glycemic control over the past 2-3 months in contrast to a fasting or random glucose test that measures recent glucose control. Patients without diabetes have an A1c between 4 and 6%. Research has shown that for patients with type 1 diabetes, an A1c < 7% reduces the risk of macrovascular and microvascular complications.[179] Some patients may have a target as low as 6%.**Error! Bookmark not defined.** Hemoglobin A1c targets are set with the physician.

Table 35. Recommended Hemoglobin A1c Targets

Patient Group	Hemoglobin A1c Target
< 18 years	< 7.5%
Adults 18 – 65 yrs	< 7.0%
Older adults (> 65 yrs)*	
Healthy	< 7.5%
Complex	< 8.0%
Poor health	< 8.5%

*Healthy older adults refers to an older adult without comorbidities. As the number of comorbidities increases or other health factors contribute to 'poor health', the recommended hemoglobin a1C target is increased. Adapted from the Nutrition Care Manual, Academy of Nutrition and Dietetics. Accessed 13 May 2021.

Which patient group has the "tightest glucose control" recommendation? Why do you think the A1c target for youth is higher than for adults?

What are the macrovascular and microvascular complications associated with diabetes?

The RDN should provide nutrition education regarding hypoglycemia. Hypoglycemia severity is defined by symptoms rather than blood glucose. Mild hypoglycemia manifests as sweating, trembling, difficulty concentrating, and dizziness and these symptoms can quickly be alleviated (within 10-20 mins) by drinking fruit juice or soft drinks, or consuming foods with carbohydrates like glucose tablets, glucose gel, honey, syrup, or crackers for about 15 – 20 g glucose/carbohydrate (which raises glucose 45 – 50 mg/dL).

Other topics important for nutrition education with patients who have type 1 diabetes include meal planning approaches, such as carbohydrate counting or simplified approaches, learning to read food labels, learning about the benefits of physical activity and the

Table 36. Types of Insulin and Insulin Analogs

Type	Generic	Brand	Onset	Peak	Duration
Ultra-Rapid Acting	Aspart	Fiasp®	2.5 min	1 hr	5 – 6 hr
Rapid-Acting (Prandial)	Aspart, Lispro, Glulisine	Novolog®, Humalog®, Admelog®, Apidra®	5 – 15 min	1 – 3 hrs	3 – 5 hr (~4 hrs)
Short-Acting	Regular	Humulin® R, Novolin® R	30 min - 1 hr	2 – 4 hr	6 – 8 hr
Intermediate-Acting (cloudy)	NPH insulin	Humulin® N, Novolin® N	1 - 2 hr	4 – 12 hr	14 – 24 hr
Pre-Mixed (cloudy)	NPH / Regular 70/30 Lispro Protamine / Lispro 50/50	Humulin® 70/30 Humalog® Mix 50/50	30 min – 1 hr 5 – 20 min	2 – 12 hr 1 – 2 hr	10 – 16 hr
Long-Acting	Detemir Glargine	Levemir® Lantus®, Basaglar®	1 – 4 hr 3 – 4 hr	Relatively flat	12 - 24 hr 20 – 24 hr
Ultra-Long Acting	Degludec	Tresiba®	1 hr	9 hr (or no peak)	Up to 42 hr

Sources: Injectable Insulin Medications, Cleveland Clinic Drugs, Devices & Supplements. https://my.clevelandclinic.org/health/drugs/13902-injectable-insulin-medications; Diabetes Education Center from the University of California San Francisco Diabetes Teaching Center. https://dtc.ucsf.edu/ ; Insulin and Combination Insulin/GLP-1 Receptor Agonist Products, Infographic from Dietitians on Demand (free). www.dietitiansondemand.com

Type 2 Diabetes

Nutrition Therapy	MNT Goals[180]
• Energy: reduced if overweight or obese DRIs • Protein: 0.8 g/kg/day • Fluid: ~1 mL/kcal/day	• Individualize eating plan to duration of disease, comorbidities, age, culture, SES, and *personal preferences* • Educate on potential food-drug interactions and nutrition-related adverse effects • Educate on carbohydrate management strategies and the role of protein intake in diabetes management • Educate on blood glucose self-monitoring and use data to adjust therapy • Support weight loss (if overweight or obese) using evidence-based guidelines • Encourage an individualized physical activity plan

The Academy's Nutrition Practice Guideline for Diabetes in Adults[180] recommend that, in collaboration with other members of the interdisciplinary care team, RDNS ensure that all at-risk overweight and obese adults are screened for type 2 diabetes; and that all adults with type 2 diabetes are referred for MNT with three to six MNT encounters in first 6 months and additional encounters past 6 months based on individualized assessment as well as at least 1 follow-up MNT encounter each year.

The RDN should also discuss herbal, vitamin, and mineral supplements with patients with diabetes. For example, some supplements like cinnamon, curcumin, and aloe vera are advertised as useful for glycemic control.

Major risk factors for type 2 diabetes that should prompt you to screen a patient for diabetes mellitus include: having a 1st degree relative with type 2 diabetes, identifying as a race/ethnicity that, as a population, has higher prevalence of type 2 diabetes mellitus (African American, Latinx, Native American, Asian American, Pacific Islander), history of CVD, hypertension or on blood pressure lowering medication, elevated triglycerides, low HDL, physical inactivity, and clinical conditions associated with insulin resistance such as PCOS, severe obesity, and *acanthosis nigricans*.[181]

What is acanthosis nigricans?

In addition to weight status, you should consider location of excess weight, as excess central (abdominal) fat is associated with a greater risk of metabolic diseases. A waist circumference ≥ 40 in (102 cm) for men and ≥ 35 in (88 cm) for women is considered high risk. Note that lower cut-offs are used for Asian men (≥ 35 in [90 cm]) and women (≥ 31.5 in [80 cm]).[182]

To diagnose diabetes, two abnormal test results are required; however, in those with overt hyperglycemia symptoms such as polydipsia, polyuria, or unexplained weight loss, one random plasma glucose of ≥ 200 mg/dL can be used to confirm a type 2 diabetes diagnosis.[183] The random plasma glucose with symptoms is often used to diagnose type 1 diabetes, as screening does not usually capture type 1 diabetes. See the table on the next page.

Table 37. Diagnostic tests used for prediabetes and diabetes mellitus

	Fasting Plasma Glucose Test	2-Hr Plasma Glucose Test	Hemoglobin A1C
Prediabetes	100 – 125 mg/dL	140 – 199 mg/dL	5.7 – 6.4%
Diabetes	≥ 126 mg/dL	≥ 200 mg/dL	≥ 6.5%

In addition to managing blood glucose levels, the RDN should assess and help the patient manage CVD risk factors and CKD risk factors.

Why are patients with diabetes at a higher risk of CVD and kidney disease compared to those with normal fasting blood glucose?

Treatment goals for patients with diabetes, and those at even higher CVD risk (i.e. with diagnosed cardiovascular disease), are presented in the table below. These goals apply to patients with type 1 and patients with type 2 diabetes.

Table 38. Cardiovascular risk factor goals for patients with diabetes

	LDL-C	HDL-C	Triglycerides	Blood Pressure
Patients with diabetes	< 100 mg/dL	> 40 mg/dL (men) > 50 mg/dL (women)	< 150 mg/dL	< 140 SBP < 90 DBP
Patients with diabetes and diagnosed CVD	< 70 mg/dL			< 130 SBP < 80 DBP

CVD: cardiovascular disease; LDL-C: LDL cholesterol; HDL-C: HDL cholesterol; SBP: systolic blood pressure; DBP: diastolic blood pressure.

The Diabetes Teaching Center at the University of California San Francisco has a Diabetes Education Online portal that goes in-depth on medications and therapies for type 1 and type 2 diabetes. You can also complete self-assessment quizzes. Go to: https://dtc.ucsf.edu/types-of-diabetes/

How do the blood pressure goals in the table above compare to the hypertension guidelines presented in the Hypertension section?

Non-Insulin Therapies

I recommend studying the non-insulin therapies in terms of action (insulin secretagogues, euglycemics, etc.). Be able to name the different medication classes within each action group and be able to at least name a few generics for each class. You do not need to be familiar with the typical dosage or delivery mechanism (oral, SQ, IM, etc) for the RD exam.

See Table 39 for a summary of non-insulin medications used to treat diabetes. Insulin secretagogues stimulate insulin secretion from the pancreas, and include sulfonylureas, meglitinides, and glinides. Biguanides are a euglycemic medication, meaning that they bring blood glucose into the normal range through various mechanisms but should not cause hypoglycemia on their own. Review the table for more information as well as your class materials and reputable online sources, such as the UCSF Diabetes Education Online portal mentioned above.

Table 39. Summary of non-insulin therapies for diabetes mellitus

Medication Class	Example	Mechanism of Action	Nutrition-related AEs	Weight
Sulfonylureas (SFU)	Tolbutamide (Orinase®), Glimepiride (Amaryl®), Glipizide (Glucotrol®), Glyburide (Micronase®, DiaBeta®)	Stimulate insulin secretion	Hypoglycemia	↑
Meglitinides and Glinides	Repaglinde (Prandin®), Nateglinide (Starlix®),	Stimulate insulin secretion	Hypoglycemia	↑
Biguanides	Metformin (Glucophage®)	↓ hepatic glucose production; ↓ intestinal glucose absorption, may reduce insulin resistance by ↑ glucose uptake	N/V, diarrhea, gas	~ or ↓
Thiazolidinediones	Pioglitazone (Actos®), Rosiglitazone (Avandia®)	↑ peripheral insulin sensitivity (muscle and fat tissues)	Fluid retention, anemia, bone loss, fractures Contraindicated with heart and liver problems	↑
GLP-1 receptor agonist (GLP-1 analog)	Exenatide (Byetta®), Liraglutide (Victoza®), Albiglutide (Tanzeum®), Dulaglutide (Trulicity®),	↑ glucose-dependent insulin secretion; ↑ satiety and ↓ gastric emptying; suppress postprandial glucagon secretion*	N/V, diarrhea, abomdinal pain, headache, hypoglycemia if taken with SFU or insulin, pancreatitis (rare)	↓
Dipeptidyl peptidase-4 inhibitors	Alogliptin (Nesina®, Sitagliptin (Januvia®), Saxagliptin (Onglyza®), Linagliptin (Tradjenta®)	↑ effect of GLP-1 and GIP to increase insulin secretion and reduce hepatic glucose production	Runny nose, sore throat, headache, upper respiratory infections, pancreatitis (rare)	~
α-glucosidase inhibitor	Acarbose (Precose®)	Delay CHO absorption	Nausea, diarrhea, gas	~
Amylin agonist	Pramlintide (Symlin®)	↓ glucagon production*, ↓ gastric emptying	N/V, headache, hypoglycemia (particularly if taken with insulin)	↓
Sodium-glucose cotransporter-2 inhibitors (SGLT)	Canagliflozin (Invokana®), Dapagliflozin (Farxiga®), Empaglifozin (Jardiance®)	↓ glucose absorption, ↑ glucose excretion in urine	↑ urinary frequency, ↑ urinary/ genitourinary infections, hypo-tension, increase blood K+ Contraindicated with kidney problems	↓

AE: adverse effect. N/V: nausea/vomiting. K+: potassium. *Suppressing glucagon production and secretion helps decrease mealtime hepatic glucose release and reduce postprandial hyperglycemia. **Sources**: Diabetes Education Center from the University of California San Francisco Diabetes Teaching Center. https://dtc.ucsf.edu/types-of-diabetes/type2/treatment-of-type-2-diabetes/medications-and-therapies/type-2-non-insulin-therapies/table-of-medications/ Non-Insulin Medications for Diabetes, Infographic (Free). Dietitians on Demand. www.dietitiansondemand.com . Overview, Type 2 Diabetes. Nutrition Care Manual. Academy of Nutrition and Dietetics.

Gestational Diabetes

Nutrition Therapy[184]	MNT Goals[185]
Same or similar to pregnancy recommendations. See Nutrition Care study guide for details. Energy: 2nd trimester: non-pregnant EER + 340 kcal 3rd trimester: non-pregnant EER + 452 kcal Protein: 1.1 g/kg/day (pregnancy) Carbohydrate: 175 g/day Fluid: 3 L/day Micronutrients: Prenatal vitamin/mineral Be familiar with IOM/ACOG guidelines regarding recommended weight gain during pregnancy https://www.acog.org/clinical/clinical-guidance/committee-opinion/articles/2013/01/weight-gain-during-pregnancy	• Maintain glucose values within target goal ranges before and after meals • Provide adequate energy intake for appropriate fetal growth and gestational weight gain without maternal ketosis • Ensure safe and adequate (but not excessive) nutrient intake for maternal and fetal health • Individualize nutrition interventions to help patents with gestational diabetes succeed in meeting their goals • Encourage patient to talk about concerns of guilt, anger, fear, and overwhelming stress using techniques such as motivational interviewing • Provide nutrition education on sources of carbohydrate, portion sizes, carbohydrate counting (if necessary), food labels, and the impact of carbohydrate intake on blood glucose

Gestational diabetes is diabetes diagnosed in the 2nd or 3rd trimester of pregnancy (that was not clearly preexisting type 1 or type 2 diabetes).[183] During these last 2 trimesters, an increase in insulin-antagonistic hormones leads to insulin resistance. If a pregnant person's body can't overcome this insulin resistance, it manifests as gestational diabetes. For many pregnant people, fasting blood glucose levels return to normal after delivery but those who had gestational diabetes are at a higher risk of type 2 diabetes later on.[186] Untreated maternal hyperglycemia increases the risk of neonatal macrosomia and neonatal hypoglycemia, because in utero, the fetus is accustomed to increased glucose crossing the placenta and its pancreas releases excess insulin to maintain normal glucose concentrations. After birth, when maternal glucose is no longer transferred via the placenta, the neonate's pancreas continues to release high levels of insulin, resulting in hypoglycemia.[186] Gestational diabetes is also associated with maternal hypertension, preeclampsia, and polyhydramnios.[186]

What is preeclampsia? What is polyhydramnios?

Read about Gestational Diabetes on the CDC website. www.cdc.gov/diabetes/basics/gestational.html

Pregnant persons with type 2 diabetes risk factors (see previous section) should be tested for type 2 diabetes at their first prenatal visit. If they meet the standard diagnostic criteria, they should be diagnosed with type 2 diabetes rather than gestational diabetes.[187] Untreated hyperglycemia due to type 2 diabetes is also associated with negative maternal and neonatal outcomes.

If testing negative, screening for gestational diabetes mellitus will occur between 24 and 28 weeks.[187] Gestational diabetes can be diagnosed using a one-step or a two-step method. The one-step approach includes a 75-g 2-hour oral glucose tolerance test (OGTT) with plasma glucose measured at fasting, and at 1 and 2 hours. The two-step method uses involves a 50 g

glucose load challenge (nonfasting) and if the plasma glucose measurement at 1 hour is > 130 mg/dL*, the patient proceeds to fasting 100 g 3-hour oral glucose tolerance test with measurements at 1, 2, and 3 hours. You can read more about gestational diabetes in the Academy's Evidence Analysis Library: https://bit.ly/3y9W3ht

**There is no consensus on the cutoff for the first step of the 2-step method, with the ACOG recommending that an OB practice select 130 mg/dL, 135 mg/dL, or 140 mg/dL and use it consistently in their practice. An OB may select a certain cut-off based on community prevalence of gestational diabetes among their patients.*

Table 40. Diagnostic Criteria for Gestational Diabetes Mellitus

	Description		Diagnostic Criteria
One-Step[1]	2-hour fasting OGTT with 75 g glucose		Any of the following: • Fasting: > 92 mg/dL • 1 hour: > 180 mg/dL • 2 hour: > 153 mg/dL
Two-Step[2]	STEP 1	Nonfasting glucose challenge with 50 g glucose	Proceed to 3-hour fasting OGTT if 1-hour plasma glucose exceeds >130, >135, or >140 mg/dL*
	STEP 2	3-hour fasting OGTT with 100 g glucose	Any 2 of the following:** • Fasting: > 95 mg/dL • 1 hour: > 180 mg/dL • 2 hour: > 155 mg/dL • 3 hour: > 140 mg/dL

[1] International Association of Diabetes and Pregnancy Study Groups Consensus. [2] NIH consensus. * There is no consensus on the cutoff for the first step of the 2-step method, with the ACOG recommending that an OB practice select 130 mg/dL, 135 mg/dL, or 140 mg/dL and use it consistently in their practice. An OB may select a certain cut-off based on community prevalence of gestational diabetes among their patients. **Carpenter/Coustan cutoffs are presented. The national Diabetes Data group use 105, 190, 165, and 145 mg/dL cut-offs. BE aware that there are different cut-offs to choose from and that 2 of the 4 criteria must be met to diagnose gestational diabetes. Adapted from the Nutrition Care Manual (Academy of Nutrition and Dietetics).

Diagnosing gestational diabetes may seem a bit more complicated than diagnosing type 2 diabetes but don't fret over all of the details. Instead, think through a scenario of a pregnant person being screened for type 2 diabetes at their first prenatal visit, and what would happen if they test negative versus positive for type 2 diabetes. In which scenario would they go on to be screened for gestational diabetes? When would they be screened? If they are screened with the one-step method, what would the process be like? How would they be diagnosed? If they are screened with the two-step method, what would the process be like and how would they be diagnosed?

Risk factors for gestational diabetes include gestational diabetes during a previous pregnancy, history of large birthweight infants, maternal overweight classification, family history of type 2 diabetes, age between 25 and 35 years, maternal PCOS, and pregnancy with twins.[188] In addition, pregnant women in some race/ethnicity groups may be at higher risk of gestational diabetes compared to non-Hispanic whites, including some Asian subgroups (Indian, Chinese, South Asian, Filipin), Mexican and Mexican-Americans, Pacific Islanders and Native Hawaiians, and American Indians and Alaska Natives. [188,189]

MNT for patients with gestational diabetes includes nutrition education and motivational interviewing to support the patient, as a gestational diabetes diagnosis can add additional stress to an already stressful time. Work with the patient to set individualized nutrition goals and provide them with the knowledge and support they need to succeed. Example patient goals include eating regularly throughout the day (3 meals, 2-3 snacks), eating no more than 45 g carbohydrates in one sitting, choosing whole foods rather than processed foods, avoiding juice/soda and limiting sweets, and attending follow-up MNT visits. [190] Food records, self-monitoring of fasting and postprandial glucose levels, fetal growth, and maternal weight gain can be used as indicators for nutrition intervention effectiveness. The RDN should evaluate patient acceptance and understanding, monitor successful behavior changes and additional needs for skills or information, and anticipate adherence.[191]

Metabolic Syndrome

Nutrition Therapy	**MNT Goals[192]**
• Energy: reduced for weight loss • Cardioprotective diet: increase whole grains and fiber, reduce intake of sugar, saturated fats, and trans fats	• Promote a healthful, cardioprotective eating pattern with reduced energy intake for a goal of reducing body weight 7 – 10 % in the first year • Encourage at least 30 mins of physical activity 5 days a week • Provide ongoing support with counseling and nutrition education to address nutrition-related knowledge deficits and treatment goals

The co-occurrence of abdominal obesity, hyperglycemia, dyslipidemia, and hypertension has been referred to as “metabolic syndrome”. However, critics of the term ‘metabolic syndrome’ note that the CVD risk of metabolic syndrome is not greater than the sum of the individual components and as each component is treated individually, they argue against the idea of a ‘syndrome’.[194,193] A patient that meets 2 of the 3 criteria would still be treated for the individual criteria.

The International Diabetes Federation, jointly with several other societies) updated their definition of metabolic syndrome in 2009 to require at least 3 of the following:[194]

- Elevated fasting glucose (≥ 100 mg/dL) or diagnosed diabetes
- Low HDL-cholesterol (≤ 40 mg/dL for men and < 50 mg/dL for women) or drug treatment
- Elevated triglycerides (≥ 150 mg/dL) or drug treatment
- Elevated waist circumference (≥ 37 in [94 cm] for men, ≥ 31 in [80 cm] for women)
- Elevated blood pressure (≥ 130/85 mmHg) or drug treatment

Previously definitions required an elevated waist circumference (with race/ethnicity specific cut-offs) and then additional criteria. The updated definition allows for the ‘diagnosis’ of metabolic syndrome without requiring elevated waist circumference.

The National Cholesterol Education Program ATP III criteria for metabolic syndrome (last updated in 2005 with the American Heart Association and National Heart, Lung, and Blood Institute) include any 3 of the 5 criteria:

- Elevated fasting glucose (≥100 mg/dL) or glucose-lowering medication
- Low HDL-cholesterol (≤ 40 mg/dL for men and <50 mg/dL for women) or drug treatment
- Elevated triglycerides (≥ 150 mg/dL) or drug treatment
- Elevated waist circumference (≥ 102 cm [40 in] for men, ≥ 88 cm [35 in] for women; ≥ 90 cm (men) or ≥ 80 cm (women) for Asian patients)
- Elevated blood pressure (≥ 130/85 mmHg) or drug treatment

MNT for metabolic syndrome involves working with the patient to develop a healthful and cardioprotective eating plan that facilitates weight loss and helps meet treatment goals, such as reduced triglycerides, normal blood pressure, and normal fasting glucose. The RDN should prioritize nutrition diagnoses, work with the patient to develop patient-oriented goals, and identify necessary resources and knowledge deficits.[195] Counseling support to facilitate lifestyle changes should combine behavior theory and cognitive behavioral therapy, use motivational interviewing techniques, and asses client's readiness for change based on the Stages of Change model.[195]

Gout

Nutrition Therapy	**MNT Goals[196]**
• Energy: reduced if needed for weight loss • Fluid: 8 – 16 cups fluid/day with at least half as water • Protein: from low-purine sources, limit meat fish and poultry to 4-6 oz/day	• Provide nutrition education regarding the connection between hydration, diet, and alcohol with acute gout episodes • Address comorbidities such as hyperlipidemia, diabetes, renal insufficiency, and hypertension if present • Have patient discuss abstaining from alcohol with physician • Create a healthful eating plan with the patient considering their culture, SES, and preferences

Gout is a type of crystal-induced arthritis ("gouty arthritis") characterized by chronic hyperuricemia due to deposition of monosodium urate crystals in the body's joints and tissues. Gout can present as acute episodes of a single joint (usually the joint of the big toe) but can become chronic and affect multiple joints, such as the ankle and knee. In addition to joint pain, acute episodes are characterized by fever, chills, and malaise and typically subside within 3 to 10 days even without treatment. Repeated acute episodes can lead to permanent joint damage and chronic pain as well as urinary tract stones and interstitial nephropathy.[197]

Risk factors for gout include genetic predisposition, older age, hypertension, renal insufficiency, medications that impair uric acid secretion, high alcohol intake, dietary excess (particularly alcohol and purine-rich animal foods), and obesity.[197] Renal insufficiency can lead to hyperuricemia because the major route of uric acid excretion is through the kidneys. Hyperuricemia *without* joint pain due to uric acid crystals is not gout. Diagnosis of gout is made by analyzing synovial fluid in the affected joint for presence of uric acid crystals. Medications that can affect uric acid excretion include thiazide, loop diuretics, low-dose aspirin, cyclosporine (to prevent transplant rejection), niacin, tuberculosis drugs, some chemotherapy drugs, and didanoside (HAART).[198] Research shows a high risk of acute gout episodes following consumption of beer, more so than other alcohols, as well as animal foods like organ and glandular meats (liver [including foie gras], kidney, sweetbreads), red meat, and seafood. Dehydration can also contribute to crystal formation.

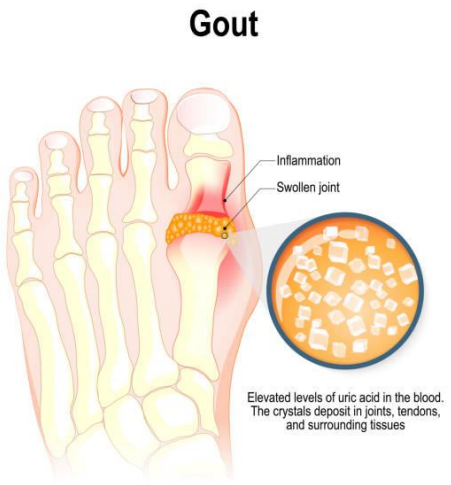


Figure 19. Illustration of acute gout episode with inflamed joint of big toe with uric acid crystals.

PCOS

Nutrition Therapy[199]	**MNT Goals**
Support medical care plan to achieve regular menstrual function, reduce androgen and insulin levels, improve skin symptoms (follicular keratosis), reduce or maintain weight, and prevent long-term complications	• Prioritize energy needs for weight maintenance or weight loss of up to 10% in first 6 months • Provide nutrition counseling to effect behavior change and provide nutrition education related to a comprehensive weight management program

Polycystic ovarian syndrome (PCOS) is an endocrine disorder characterized by increased androgens (produced by adrenals or ovaries) and irregular menstrual cycles due to ovarian cysts. High androgen levels manifest as hirsutism, alopecia, and acne. Up to 70% of people with PCOS have insulin resistance with compensatory hyperinsulinemia. The latte r promotes central adiposity, trouble losing weight, food cravings, and hypoglycemic events.[200] People with PCOS are at increased risk of impaired glucose tolerance, type 2 diabetes, kidney disease, and cardiovascular disease.[201]

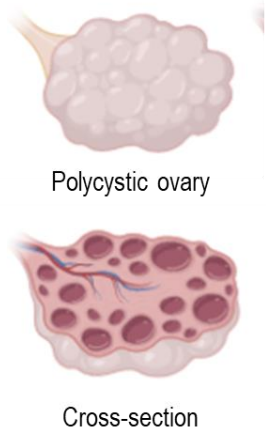


Figure 20. Illustration of polycystic ovary (www.biorender.com)

Biochemical data to review during the nutrition assessment include fasting glucose, results of glucose tolerance test, hemoglobin A1C, lipid profile, serum 25(OH)D levels, hemoglobin and hematocrit.[202] Nutrition intervention is focused on lifestyle behavior management for weight management and is the same for overweight and obese patients that do not have PCOS.[203]

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