A pressure ulcer is defined as a localized injury to the skin, the underlying tissue, or both, usually over a bony prominence, that develops as a result of pressure or pressure in combination with shear. Pressure ulcers are staged according to the degree of damage observed clinically (Table 1). Pressure ulcers are a clinical concern in all patients, including critical care patients, and affect an estimated 2.5 million patients annually, increasing both a patient’s risk for serious infections and utilization of health care services. In 2011, the HealthGrades Patient Safety in American Hospitals study cited pressure ulcers as the second most common adverse patient safety event in hospitalized patients, with
attributable estimated health care costs of $9.1 billion to $11.6 billion. In 2008, the Centers for Medicare and Medicaid Services deemed hospital-acquired stage III/IV pressure ulcers “never events,” thus restricting reimbursement to hospitals for care associated with these ulcers. The prevalence rates of pressure ulcers in critical care patients are the highest among hospitalized patients, ranging from 14% to 27%. In 2009, severe hospital-acquired pressure ulcers defined as stage III or stage IV, unstageable, or suspected deep tissue injury occurred in 3.3% of critical care patients.

Development of a pressure ulcer is a multifactorial phenomenon. Critical care patients have a multitude of risk factors for these ulcers, including altered mobility, longer length of stay in the intensive care unit (ICU), emergent admission to the ICU, use of vasopressor agents, and comorbid conditions such as diabetes mellitus, infection, and cardiovascular or vascular disease.

Optimal nutrition is fundamental to wound healing. Current guidelines for the prevention and treatment of pressure ulcers support nutritional screening and nutritional interventions as important aspects of care for patients who are at risk for or have pressure ulcers. In the general hospitalized population, malnutrition has been significantly associated with the development of pressure ulcers, however, the exact causal relationship between nutrition and the development of these ulcers remains largely unknown and understudied. For critical care patients, the evidence is sparse; the role of nutrition in development and treatment of pressure ulcers has been specifically investigated in only 2 studies. In a study of 100 critical care patients with acute lung injury who were receiving mechanical ventilation, development of pressure ulcers was significantly less in patients given an enteral formula enriched with micronutrients, eicosapentaenoic acid, and γ-linolenic acid than in patients given a control enteral formula lacking these nutritional components (χ² = 3.5; P < .05). In a study on the impact of nutrition on healing of pressure ulcers in critical care patients, the progression of stage II or higher pressure ulcers was significantly less in patients given a feeding....

### Table 1 Pressure ulcer staging system

<table>
<thead>
<tr>
<th>Type of pressure ulcer</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suspected deep tissue injury</td>
<td>Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler than adjacent tissue.</td>
</tr>
<tr>
<td>Stage I</td>
<td>Intact skin with nonblanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; the color of the ulcer may differ from that of the surrounding area.</td>
</tr>
<tr>
<td>Stage II</td>
<td>Partial-thickness loss of dermis manifested as a shallow open ulcer with a red pink wound bed, without slough. May also appear as an intact or open/ruptured serum-filled blister.</td>
</tr>
<tr>
<td>Stage III</td>
<td>Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss.</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound. Often includes undermining and tunneling.</td>
</tr>
<tr>
<td>Unstageable</td>
<td>Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) or eschar (tan, brown, or black) in the wound bed.</td>
</tr>
</tbody>
</table>

Based on information from the National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel.

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**Authors**

Jill Cox is an assistant professor of nursing at Rutgers University, Newark, New Jersey. She maintains a clinical practice as an advanced practice and certified wound, ostomy, and continence nurse at Englewood Hospital and Medical Center, Englewood, New Jersey.

Louisa Rasmussen is the registered dietician for the critical care service at Englewood Hospital and Medical Center.

Corresponding author: Jill Cox, RN, PhD, APN-C, CWOCN, Englewood Hospital, 350 Engle St, Englewood, NJ 07631 (e-mail: jillm.cox@rutgers.edu).

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formula enriched with fish oil than in patients given an isocaloric control formula ($P = .02$). Nevertheless, best practice dictates nutrition as a fundamental aspect of a comprehensive plan of care in the prevention and treatment of pressure ulcers for all populations of patients and in all care settings.\(^1,\(^{18}\)

In this article, we define malnutrition, describe the pathophysiological effects of malnutrition, and discuss nutritional screening in critical care patients. We discuss the impact of nutrition on wound healing and present the current nutritional practices and guidelines associated with prevention and treatment of pressure ulcers. Finally we examine the nursing care considerations associated with the delivery of enteral feedings in critical care patients who are at risk for or who have a pressure ulcer.

**Definition of Malnutrition and Nutritional Screening in Critical Care Patients**

**Malnutrition**

Malnutrition is defined as a condition in which an imbalance of energy, protein, and other nutrients leads to adverse effects on tissue and body structures.\(^17\) Malnutrition in adults is considered undernutrition and is categorized in the context of one of the following: acute injury or illness, chronic illness, and social and environmental circumstances.\(^24\) Malnutrition is further classified as either nonsevere or severe on the basis of the presence and degree of 2 or more of the following characteristics: insufficient energy intake, weight loss, loss of muscle mass, loss of subcutaneous fat, localized or generalized fluid accumulation that can mask weight loss, and diminished functional status as indicated by hand grip strength.\(^24\) In critically ill patients, malnutrition is defined in the context of acute illness or injury. To be considered severely malnourished, a patient must have 2 of the 6 clinical characteristics of malnutrition outlined in Table 2.

Protein calorie malnutrition is a form of malnutrition associated with factors such as decreased oral intake of both protein and calories, unexplained or unintentional weight loss, and being underweight.\(^17,\(^25\) In critically ill patients, severe protein calorie malnutrition is common because of impaired intake and the hypercatabolic-hypermetabolic response to injury or severe illness.\(^26,\(^27\) The hypermetabolic response results in increased caloric needs and a disproportionate increase in protein requirements.\(^17\) Calories are pulled from glycogen stores first in an effort to meet end-organ energy needs.\(^28\) Elevated cortisol production by the adrenal cortex results in protein catabolism, amino acid mobilization, and the production of hepatic glucose. In addition, the concomitant activation of biochemical mediators associated with the inflammatory and immune responses, including cytokines, interleukins 1 through 6 (proinflammatory cytokines), and tumor necrosis factor $\alpha$, contribute to anorexia, malaise, muscle wasting, cachexia, and impaired albumin synthesis.\(^28,\(^29\) Serum proteins that decrease during the acute phase of the inflammatory response are termed negative acute-phase reactants and include albumin and prealbumin.\(^17\) With resolution of the inflammatory state, the levels of these serum proteins usually return to normal. Positive acute-phase reactants such as C-reactive protein, fibrinogen, and protein S dominate the acute inflammatory response and are essential to a successful inflammatory response.\(^17\) These physiological responses combined contribute to impaired use of nutrients, weight loss, and malnutrition.\(^17\)

**Nutritional Screening**

Nutritional screening is essential to identify the risk for malnutrition and unintentional weight loss, both of which may contribute to the development of pressure ulcers. Currently, no ideal laboratory tests for detecting malnutrition exist.\(^17\) Traditional nutritional markers include serum levels of proteins such as albumin, prealbumin,

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### Table 2

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Severe malnutrition defined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake</td>
<td>≤50% of estimated energy requirements for ≥5 days</td>
</tr>
<tr>
<td>Weight loss</td>
<td>&gt;2% in 1 week</td>
</tr>
<tr>
<td></td>
<td>&gt;5% in 1 month</td>
</tr>
<tr>
<td></td>
<td>&gt;7.5% in 3 months</td>
</tr>
<tr>
<td>Body fat</td>
<td>Moderate loss of subcutaneous fat (triceps, intercostal muscles)</td>
</tr>
<tr>
<td>Muscle mass</td>
<td>Moderate muscle loss (pectoralis, deltoids, quadriceps, gastrocnemius)</td>
</tr>
<tr>
<td>Fluid accumulation</td>
<td>Moderate to severe (generalized or local)</td>
</tr>
<tr>
<td>Reduced grip strength</td>
<td>Measurably reduced (not practical to test in an unresponsive critical care patient)</td>
</tr>
</tbody>
</table>

*Based on information from White et al.\(^24\)*
transferrin, and retinol-binding protein in addition to anthropometric measures such as height, weight, and body mass index. Although they have not been validated for use in critical care patients, these markers are used by some clinicians in clinical practice as indicators of a patient’s nutritional status. Critical illness and the inflammatory response alter these nutritional markers, producing misleading results that can lead to an inaccurate diagnosis of malnutrition. Serum albumin is not a sensitive indicator of malnutrition because levels of the protein are influenced by many nonnutritionally related factors, such as protein-losing states, hepatic function, acute infection, and inflammation. Fluid shifts and hydration status influence body weight in addition to serum albumin and prealbumin levels. If fluid volume is low, serum levels of albumin and prealbumin may be falsely elevated, and in patients with fluid volume overload, the serum levels may be low because of hemodilution. Compared with the serum concentration of albumin, the serum concentration of prealbumin provides more contemporaneous information about a patient’s protein stores because of prealbumin’s relatively short half-life of 72 hours. However, as previously discussed, serum levels of prealbumin can be decreased in patients with severe inflammation and thus can lead to a potentially false indication of malnutrition and result in unnecessary overfeeding.

In the hospital, initial nutritional screening and assessment of the risk for pressure ulcers are generally completed by a nurse during a patient’s admission by using a standardized assessment tool such as the Braden Scale. The Braden nutrition subscale is used to measure a person’s usual nutritional intake; however, usual nutritional intake may be difficult to ascertain in a patient who is critically ill. Because of their heavy burden of illness, most critical care patients are unable to articulate a dietary history, especially in the initial days of an ICU admission. Thus, the full spectrum of nutrition as a risk factor for pressure ulcers at the time of the ICU admission may not be adequately determined by using the Braden Scale.

A more complete nutritional screening and assessment, completed by a registered dietitian, is vital to ensure that a comprehensive nutritional plan is implemented according to the national guidelines and individualized to account for a patient’s critical illness and existing comorbid conditions, including pressure ulcers. The assessment should encompass an evaluation of the patient’s nutritional intake before admission, an evaluation of weight loss before admission, current level of disease severity, knowledge of the patient’s comorbid conditions, the functionality of the gastrointestinal tract, and the current risk level for pressure ulcers or the stage of existing pressure ulcers. Currently, no evidence-based nutritional screening tools specifically designed for use in critical care patients are available; therefore, critical care patients are screened for nutritional risk according to the same nutritional criteria allocated for the general hospitalized population.

Phases of Wound Healing and the Impact of Nutrition

Adequate nutrition is essential to optimize wound healing in patients with existing pressure ulcers; however, data on the optimal amount, combination, and timing of nutritional regimens are controversial, lack empirical rigor, and are based mostly on expert opinion. Suboptimal nutrition can alter immune function, collagen synthesis, and tensile strength—all essential elements in the wound-healing cascade. Patients who experience involuntary weight loss and protein calorie malnutrition are also at risk for delayed and impaired wound healing.

Wound healing occurs in 3 distinct, but overlapping, phases: the inflammatory phase, the proliferative phase, and the remodeling phase. Each phase is time limited and characterized by a distinct set of physiological events. Table 3 outlines the key events that occur in each phase of wound healing and the associated pathophysiological alterations that can occur as a result of nutritional deficits.

In patients with chronic wounds, such as pressure ulcers, the phases of wound healing can be delayed and do not follow an orderly and timely process. Chronic wounds can be characterized by a prolonged inflammatory response, low levels of growth factors, and high levels of bioburden (microorganisms) in the wound. Malnutrition is a common contributor to wound chronicity. Nutritionally, the long-term inflammatory state of chronic wounds can induce catabolic metabolism, protein calorie malnutrition, and dehydration.
In the management of a critical care patient who is at risk for pressure ulcers or has a pressure ulcer, nutritional provisions are essential and should be based on the nutritional guidelines specifically recommended for the critically ill. According to the nutritional guidelines\(^\text{25}\) of the American Society for Parenteral and Enteral Nutrition (ASPEN) and the Society for Critical Care Medicine, feedings for critical care patients should be initiated early, ideally within the first 24 to 48 hours after ICU admission. In the ASPEN guidelines,\(^\text{35}\) the preferred route for feeding critical care patients who are unable to consume nutrients orally is the enteral route, which offers many benefits, including maintenance of the functional integrity of the gastrointestinal tract (Table 4). In patients who cannot meet their nutritional needs via enteral feedings, the concomitant use of parenteral feedings should be considered, and parenteral feedings should be initiated early, ideally within the first 24 to 48 hours after ICU admission.

<table>
<thead>
<tr>
<th>Phase</th>
<th>Duration</th>
<th>Key events</th>
<th>Impact of nutritional deficiencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory</td>
<td>Begins at the time of injury or within 4-6 days</td>
<td>Activation of the coagulation cascade after hemostasis Release of proinflammatory cytokines Increased vascular permeability, vasodilation Phagocytosis of bacteria by neutrophils</td>
<td>Vitamin A: Alterations in immune function (T- and B-cell function/antibody response); increased risk of infection Vitamin C: impaired immune response Iron: impaired immune response (T-cell and phagocytic function) Zinc: decreased immunity, increased susceptibility to pathogenic organisms</td>
</tr>
<tr>
<td>Proliferative</td>
<td>Begins on day 3 or 4 and continues 2-3 weeks</td>
<td>Removal of debris and secretion of growth factors by macrophages Macrophage recruitment of fibroblasts Angiogenesis Fibroblast proliferation Fibroblast: collagen synthesis Collagen deposition and cross-linking Development of granulation tissue, extracellular matrix Epithelialization: epithelial resurfacing Wound contraction: closing of wound by myofibroblasts begins</td>
<td>Vitamin C, iron, copper, zinc, manganese deficiencies: impaired tensile strength and collagen synthesis Hypoproteinemia: impaired fibroblast proliferation and collagen synthesis Vitamin C deficiency: increased capillary wall fragility and angiogenesis, increased risk of wound hemorrhage</td>
</tr>
<tr>
<td>Maturation/remodeling</td>
<td>Begins around day 21 and continues up to 2 years</td>
<td>Collagen maturation and stabilization into the organized matrix, increased tensile strength Scar tissue formation</td>
<td>Vitamin A: impaired collagen synthesis Vitamin C: reduced tensile strength Zinc: Impaired wound strength; decreased fibroblast proliferation, collagen synthesis, and epithelialization rate</td>
</tr>
</tbody>
</table>

**Table 3** Phases of wound healing and impact of nutritional deficits\(^a\)  
\(^a\) Based on information from Rote,\(^\text{29}\) Stechmiller,\(^\text{32}\) and Doughty and Sparks-DeFriese.\(^\text{34}\)

**Table 4** Overview of enteral nutrition\(^a\)  
\(^a\) Based on information from Brantley.\(^\text{35}\)

**Benefits**  
Maintains integrity of gastrointestinal tract  
Prevents bacterial translocation through continued production of immunoglobulin A  
Allows continued metabolism and efficient use of nutrients  
Reduces risk of cholecystitis  
Reduces complications such as pneumonia, intravenous catheter–associated sepsis, sepsis, and intra-abdominal abscess  
Is cost-effective  

**Contraindications**  
If expected duration of enteral nutrition is <5-7 days in malnourished patients or <7-9 days in adequately nourished patients  
Severe short bowel (<100 cm remaining)  
Severe gastrointestinal bleeding  
Severe gastrointestinal malabsorption  
Distal, high-output fistulas  
Intractable vomiting  
Diarrhea (not responsive to medical management)  
Paralytic ileus  
Mechanical obstruction  
Inability to gain access
feedings should be strongly considered for all patients for whom enteral feedings are contraindicated.25

Enteral formulations are most often classified into 4 categories: standardized (polymeric); elemental or semi-elemental; modular; and disease-specific, such as renal or pulmonary formulations36 (Table 5). Selection of an enteral feeding from any of these categories takes into consideration the following factors: ability of the gastrointestinal tract to absorb the nutrients provided in the feeding, comorbid conditions, and feeding tolerance.35,36 In critical care patients, feeding tolerance may be reduced because gastric emptying can be adversely affected by age, severity of illness, immobility, medications, and type of illness.35

Enteral feeding preparations to promote pressure ulcer healing should be calorically dense, protein-rich, and provide the recommended daily requirement of micronutrients.28,32,37

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standardized (polymeric)</td>
<td>Contain intact proteins, complex carbohydrates, and mainly long-chain triglycerides</td>
</tr>
<tr>
<td>Elemental</td>
<td>Contain individual amino acids, glucose polymers, and are low fat, with only about 2% to 3% of calories derived from long-chain triglycerides</td>
</tr>
<tr>
<td>Semi-elemental</td>
<td>Contain peptides of various chain lengths, simple sugars, glucose polymers or starch and fat, primarily as medium-chain triglycerides</td>
</tr>
<tr>
<td>Modular</td>
<td>Consist of a singular macronutrient; provide additional protein and calories and can be used to augment enteral formulations</td>
</tr>
<tr>
<td>Disease-specific (ie, renal and pulmonary, immunomodulating formulations)</td>
<td>Formulated to meet nutrient requirements for patients with specific medical diseases; can include products containing branched-chain amino acids, high-fat formulas, essential amino acids as protein sources, and formulas high in omega fatty acids</td>
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</tr>
</tbody>
</table>

Macronutrients

The provision of adequate energy and protein is fundamental for cell metabolism, collagen formation, and nitrogen maintenance, all of which are required for wound healing.28,32,37 Energy is provided via the ingestion and administration of the macronutrients: carbohydrates, proteins, and fats.28 According to the guidelines1 of the National Pressure Ulcer Advisory Panel/European Pressure Ulcer Advisory Panel (NPUAP/EUPAP), the provision of 30 to 35 Cal/kg of body weight, to a maximum of 40 Cal/kg, is recommended for patients who are at risk for or who have pressure ulcers. For patients who are underweight or who continue to lose weight, additional calories are recommended to prevent further weight loss or to promote weight maintenance.1 Caution, however, must be used to avoid overfeeding critically ill patients. Overfeeding in the critically ill has been associated with immunosuppression and exacerbation of hyperglycemia associated with the catabolic stress response.27,38

Macronutrients are found in oral, enteral, and parenteral formulas in various forms of carbohydrates, fats, and proteins. Carbohydrates account for approximately 35% to 55% of most standard enteral feeding formulations and are both the primary macronutrient and the main energy source.36 Carbohydrates also add to the osmolarity, aid in digestibility, and add sweetness or palatability to the formula.

Protein in enteral formulations can be in the form of intact proteins, hydrolyzed proteins, or free amino acids. Protein is essential to wound healing and plays an important role in fibroblast proliferation and collagen synthesis.28 Overall protein requirements recommended by the NPUAP/EUPAP for pressure ulcer healing are 1.25 to 1.5 g/kg of body weight daily.1 For patients with stage III/IV pressure ulcers, the recommended level is 1.5 to 2.0 g/kg, depending on the size of the pressure ulcer and the amount of protein loss from draining wounds.32 Caution, however, must be exercised in the calculation of protein needs, especially for critically ill patients. Excessive amounts of protein can overburden the kidneys or liver.
especially in patients with preexisting renal or hepatic disease. Excess protein may also be a risk factor for dehydration in patients with inadequate fluid intake.38

Fats provide a concentrated source of energy and provide essential fatty acids. The fat component of enteral formulas is usually a combination of medium- and long-chain triglycerides. Overall, the role of fatty acids in wound healing has not been established; however, the results of a study23 of 40 adult critical care patients did support the relationship between administration of fish oil and healing of pressure ulcers. Currently, however, no conclusive evidence indicates that routine supplementation with omega-3 fatty acids enhances healing of pressure ulcers.32 Macronutrient recommendations are summarized in Table 6.

### Micronutrients

Micronutrients are essential dietary elements and include vitamins, minerals, and other chemical components. For a patient who is at increased risk for pressure ulcers or who has a pressure ulcer, vitamin supplementation should be started if the patient has a known or suspected vitamin deficiency.17 Supplementation of any vitamin or mineral should be based on the patient’s clinical manifestations, laboratory data, stage of the pressure ulcer, and the clinical judgment of the registered dietitian and critical care team. Vitamin A, vitamin C, and zinc play a role in wound healing, and deficiencies in any of these may alter functions needed for optimal wound healing.32

Vitamin A maintains skin integrity and stimulates fibroblast and collagen synthesis.32 In patients with vitamin A deficiency and a pressure ulcer (any stage), 10 000 to 50 000 IU of vitamin A daily for 10 days is recommended.32 Vitamin C is required for the maturation of fibroblasts needed for collagen synthesis and also plays a role in immune function. Supplementation is based on the stage of the pressure ulcer. For a patient with a stage III or IV pressure ulcer, 1000 to 2000 mg/d of vitamin C is recommended if the patient is at risk for vitamin C deficiency or is nutritionally stressed.32 Zinc is a component of enzyme systems necessary for fibroblast synthesis. For patients with a known zinc deficiency who have a pressure ulcer of any stage, zinc sulfate is recommended at a dose of 220 mg twice daily for 10 to 14 days.32 Table 7 summarizes information on micronutrient supplementation.

---

### Table 6  Energy and macronutrient requirements in pressure ulcer management a,b

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total intake</th>
<th>Carbohydrates</th>
<th>Proteins</th>
<th>Fats</th>
<th>Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Functions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Promotion of anabolism</td>
<td></td>
<td>Primary energy source</td>
<td>Cell structure and function</td>
<td>Contribute to healthy cellular function</td>
<td>Skin turgor maintenance</td>
</tr>
<tr>
<td>Nitrogen retention</td>
<td></td>
<td>Major source of glucose</td>
<td>Fibroblast proliferation</td>
<td>15%-50% of total daily calories, depending on disease state</td>
<td>Perfusion and oxygenation of tissues</td>
</tr>
<tr>
<td>Collagen synthesis</td>
<td></td>
<td>Needed for collagen synthesis</td>
<td>Collagen synthesis</td>
<td></td>
<td>Solvent for vitamins, minerals, glucose, and other nutrients</td>
</tr>
<tr>
<td>Angiogenesis</td>
<td></td>
<td>Provide approximately 45%-65% daily energy</td>
<td>15%-20% of total calories in critically ill patients</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>General guideline: 30-35 Cal/kg body weight</th>
<th>No specific recommendations; based on pressure ulcer stage</th>
<th>Stage I/II: 1-1.4 g/kg</th>
<th>Stage III/IV: 1.5-2.0 g/kg</th>
<th>Maximum: 2.2 g/kg</th>
<th>No specific recommendations; based on pressure ulcer stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>≥25 Cal/kg</td>
<td>Stage I/II: 1-1.4 g/kg</td>
<td>30-40 mL/kg per day to maintain balance in adults</td>
<td>Stage I/II: ≥30 mL/kg per day (minimum of 1 mL/Cal)</td>
<td>Stage III/IV: 30-40 mL/kg per day (minimum of 1 mL/Cal)</td>
<td>Fluid intake must be adjusted for fluid losses</td>
</tr>
<tr>
<td>Stage II</td>
<td>28-30 Cal/kg</td>
<td>Stage III/IV: 1.5-2.0 g/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage III</td>
<td>30 Cal/kg</td>
<td>Maximum: 2.2 g/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage IV</td>
<td>33-35 Cal/kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum: 40 Cal/kg</td>
<td></td>
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</tr>
</tbody>
</table>

---

a Energy and macronutrient requirements can be adjusted on the basis of clinical condition, medical history, and clinical judgment of the dietitian or physician.
b Based on information from Stotts,17 Dorner et al,28 and Stechmiller.32
Table 7 Micronutrient supplementation in pressure ulcer management\textsuperscript{a,b}

<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>Benefits to wound healing</th>
<th>Normal daily requirements</th>
<th>Dosing for pressure ulcer management</th>
<th>Indications of deficiency in relation to wound healing</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc, trace mineral</td>
<td>Integral in cell replication and growth. Collagen cofactor. Protein synthesis.</td>
<td>Men: 11 mg/d, Women: 8 mg/d</td>
<td>Supplementation recommended only in serum deficiency. Standard for any stage pressure ulcer: 220 mg/d for 10-14 days because of insoluble tendencies of zinc and absorption difficulty. Additional supplementation due to excess small intestinal fluid losses, ileostomy, and stool output may be required as follows: Small intestinal fluid losses: 12.2 mg zinc per liter lost. Stool output: 17.1 mg of zinc per kilogram of stool. Ileostomy: 17.1 mg of zinc per kilogram of drainage. Severe deficiency: continuous intravenous infusion of 50-100 mg/d, if tolerated, with close monitoring.</td>
<td>Delayed wound healing. Decreased collagen and protein synthesis. Impaired immune function. Deficiencies associated with patients with diarrhea, malabsorption, hypermetabolic states, stress, sepsis, burns, ulcers. Deficiencies can lead to loss of appetite and abnormal taste, which can hinder nutritional intake.</td>
<td>Poor wound healing due to decreased immune function. Disruption of normal phagocytic activity. Impaired neutrophil and lymphocyte function. Copper- and calcium-binding interactions leading to copper and calcium deficiencies. Gastrointestinal tract irritation: nausea, vomiting, diarrhea.</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Energy and macronutrient requirements can be adjusted on the basis of clinical condition, medical history, and clinical judgment of the dietitian or physician.

\textsuperscript{b} Based on information from Stotts,\textsuperscript{17} Dorner et al,\textsuperscript{28} and Stechmiller.\textsuperscript{32}
Amino acid supplementation with arginine and glutamine may be considered in patients with existing pressure ulcers, but evidence of the role these agents play in pressure ulcer prevention or wound healing remains limited. Arginine plays a role in protein synthesis, but evidence to substantiate its efficacy in wound healing is minimal. Therefore dietary supplementation to augment wound healing is currently not recommended. Moreover, caution is advised with arginine supplementation in critically ill patients with sepsis because the amino acid may contribute to unstable hemodynamic status in these patients. Glutamine is one of the most abundant amino acids in the body. It functions as a fuel source for fibroblasts and epithelial cells for wound healing and helps preserve the functional integrity of the intestinal brush border. Little information is available on the role of glutamine supplementation in the treatment of pressure ulcers; thus its effect on healing of pressure ulcers has not been established.

Hydration

In addition to food requirements, adequate hydration plays a vital role in nutritional status and more specifically in the maintenance and repair of skin integrity via oxygenation of both healthy and wounded tissues. Water acts as a diluent for micronutrients, glucose, and the removal of waste products from the body. Current recommendations of ASPEN for water intake are 30 mL/kg of body weight or 1.0 to 1.5 mL/Cal consumed. In addition, fluid boluses, flushes, or supplemental intravenous hydration may be necessary in some patients to prevent dehydration. For pressure ulcer healing, 30 to 40 mL/kg or 1500 mL/d is recommended, depending on the amount of fluid losses experienced by the patient (eg, draining wounds, febrile states, gastrointestinal fluid losses) and the patient’s comorbid conditions, such as renal or cardiac disease.

Administration of Enteral Nutrition and Pressure Ulcers

Nursing Care Considerations

Delays in starting and continuing nutritional support lead to marked deficits in nutritional stores to meet a patient’s energy needs. Underfeeding results in adverse clinical outcomes, including a possible increase in the risk for pressure ulcers, increased risk for nosocomial infection, loss of lean body mass, prolonged duration of weaning from mechanical ventilation, and delayed wound healing in patients with existing pressure ulcers. Although the nutritional guidelines of ASPEN and the Society for Critical Care Medicine state that enteral feeding be started early, ideally within the first 24 to 48 hours after ICU admission, a systematic review of the nursing, dietary, and medical literature revealed variations in the actual time of initiation of nutritional support, from 2 to 8 days after admission, and patients received only a mean of 63% of their estimated energy requirements.

Underfeeding can be due to many clinical situations. These include interruptions in enteral feeding during routine nursing care, such as repositioning or administering wound or incontinence care. Prolonged periods of no oral intake related to procedures, unstable hemodynamic status, administration of medications, and cessation of enteral feeding because of perceived gastrointestinal complications such as high gastric residual volumes (GRVs) and diarrhea can also result in underfeeding.

In 2 studies in critical care patients, the mean length of feeding interruptions was 6 h/d. Common causes of interruptions in these studies included problems with small-bore feeding tubes, increases in GRV or feeding intolerance, weaning, shock states, and interruptions related to procedures or routine patient care, such as bathing, repositioning, and skin care. Prolonged requirements for nothing by mouth before procedures affects delivery of enteral feedings and can result in underfeeding. Currently, no consensus exists on the appropriate fasting times required before a procedure in patients receiving enteral feedings. Although the guidelines of the American Society of Anesthesiologists indicate that avoidance of clear liquids for 2 to 4 hours before surgery is sufficient, the intake of milk-based products before an operative procedure requires longer fasting times, up to 6 hours. For patients receiving enteral feedings, the guidelines state that the amount of time feedings are withheld may need to be modified on the basis of the clinical judgment of the practitioner. In a recent study of critical care patients in Korea, interruptions in feedings for procedures accounted for 17.5% of the total interruption time. An evaluation of the length of time feedings are withheld in patients undergoing bedside or
operative procedures might be considered in an effort to determine if the times match current guidelines.

High GRVs can result in interruptions in enteral feeding, adversely affecting the overall volume of nutritional formula administered. The GRV value is considered the proxy for gastric motility and is the most common parameter for assessing feeding tolerance in critically ill patients. However, measurement of GRV can be subjective, and little evidence supports or indicates an acceptable residual volume. Some investigators have suggested that the clinical utility of GRV measurements is limited. In a study in 205 patients treated with mechanical ventilation who were receiving enteral nutrition, adverse events such as ventilator-associated pneumonia and vomiting did not differ between patients in whom GRV purposely was not measured and patients in whom GRV was measured. Current nutritional guidelines suggest that enteral feedings should not be withheld if a patient has a GRV less than 500 mL and no other indications of feeding intolerance. In addition, in an effort to maximize feeding tolerance, the administration of agents that promote motility, such as metoclopramide, should be given to these patients. Conversion to a duodenal or jejunal feeding tube may also be necessary to promote feeding tolerance.

Diarrhea, a commonly reported side effect of enteral feeding, can result in interruptions in enteral feedings and can adversely affect skin integrity. Although no universal definition is currently accepted, diarrhea can be described as an abnormal volume or consistency of stool, with stool water content greater than 500 mL, every 8 hours. Common causes of diarrhea in patients receiving enteral feedings include medications with a sorbitol base, antibiotics, infections such as those caused by Clostridium difficile, bacterial overgrowth in the gastrointestinal tract, and intolerance to the formula used for feeding. During the evaluation for causes of diarrhea, a workup for all sources of diarrhea, including infectious processes and medication-induced diarrhea, should be undertaken. Enteral feeding should be discontinued only if the diarrhea is refractory to all other treatment options. Once other causes of the diarrhea are ruled out, changes to the enteral feeding formula or the brand of formula might be necessary, including introduction of an enteral formula with fiber.

Moisture, from sources such as diarrheal stools, has long been recognized as a risk factor for pressure ulcers and is included as a subscale of the Braden Scale. Prolonged exposure of the skin to diarrheal stools, which contain caustic enzymes and bacteria, can make the skin more vulnerable to the mechanical forces of pressure, shear, and friction, thus increasing the risk for pressure ulcers. In patients with existing pressure ulcers, stool is a contaminant that can have deleterious effects on wound healing and can lead to wound infection. Nursing care of patients with diarrhea may include the use of skin barrier creams and pastes for skin protection. For incontinent patients, use of highly absorbent disposable underpads may also be beneficial to wick excess moisture and liquid stool from the skin. In critical care patients with diarrheal incontinence, bowel management systems, also known as fecal containment devices, should also be considered to contain and divert liquid stool from the skin.

Enteral Feeding Protocols

Studies on the efficacy of standardized enteral feeding protocols have indicated marked improvements in the delivery of enteral feedings and improved caloric intake. Incorporation of a feeding protocol such as the
PEPuP protocol (enhanced protein-energy provision via the enteral route in critically ill patients) may be considered to optimize a patient’s nutrition. Elements of this protocol include daily volume-based feeding goals instead of hourly feeding rates, early initiation of both promotility agents and protein supplementation, liberalization of the GRV threshold, and the use of trophic (small-volume) feedings. Similarly, Miller et al proposed the mnemonic “CAN WE FEED?” as a means to improve enteral nutrition in ICU patients. This mnemonic—C, critical care severity; A, age; N, nutrition risk screening; W, wait for resuscitation; E, energy requirements; F, formula selection; E, enteral access; E, efficacy; and D, determination of tolerance—can be used by the critical care team to guide the early implementation of a nutritional therapy plan for critically ill patients.

Along with enteral feeding protocols is the incorporation of standardized nursing protocols into clinical practice as a means to lessen undernutrition in critical care patients. These protocols should include such elements as titration schedules to reach the goal infusion rate, protocols for flushing enteral tubes, and a defined list of clinical situations that would result in interruption of enteral feeding. Planning nursing care to optimize the volume of enteral feeding delivered by combining medications or combining routine nursing care such as bathing with local wound care or repositioning whenever possible will also minimize interruptions in enteral feeding. In a systematic review of the literature on enteral nutrition, Kim et al found that early initiation of feedings and rapid escalation to the goal feeding rate or starting feedings at the goal rate improved energy and protein intake in critical care patients more than did late initiation of feeding or progressing the rate or volume of feedings gradually.

Conclusion

Critical care nurses are frontline caregivers, vital to both appropriate delivery of enteral feedings and prevention and management of pressure ulcers in critical care patients. Early referral by a critical care nurse to a registered dietitian is the essential first step in improving nutritional outcomes for patients found to be at risk during the initial nutritional screening and pressure ulcer risk assessment. In patients with existing pressure ulcers, understanding the relationship between nutrition and pressure ulcer healing affords nurses the ability to advocate for appropriate nutritional supplementation.

In enterally fed patients, frequent skin inspections of the sacral and buttocks regions, coupled with the incorporation of a low air loss support surface, may be necessary to offset the damaging effects of shear induced by continuous elevation of the head of the bed. Diarrhea, though often hypothesized to be related to enteral feedings, is often due to other causes. Recognizing the multifactorial nature of diarrhea and continuing enteral feedings until other causes have been ruled out can result in improved delivery of the feedings. Providing adequate skin protection by using topical barrier creams or a bowel management system or both should also be part of the overall care plan for a patient experiencing diarrhea. Finally, incorporation of nursing protocols that address interventions to minimize interruptions in enteral feeding may help in providing a standardized and sustained approach to delivery of feedings in this population at risk for pressure ulcers.

Pressure ulcer prevention and healing in critically ill patients may be especially challenging because of the patients’ burden of critical illness and degree of physiological compromise. However, efforts to support the provision of adequate nutrition may be instrumental in halting the development or worsening of a pressure ulcer. Nutritional management in the ICU requires a team approach; registered dietitians, critical care nurses, and intensivists are all integral players in the process. Although opportunities for empirical investigation of the role that nutrition plays in the prevention and healing of pressure ulcers are plentiful, surely, optimization of nutrition can be considered an essential ingredient of interventions to prevent and treat pressure ulcers.

The combination of appropriate screening of nutritional status and risk for pressure ulcers, early collaboration with a registered dietitian, and administration of appropriate feeding formulas and micronutrient and macronutrient supplementation to promote wound healing are practical solutions to improve the nutritional status of critical care patients. The incorporation of standardized nutritional management protocols and the implementation of enteral feeding protocols may also provide the vital elements to augment nutrition, which
ultimately can result in improved clinical outcomes in critical care patients at risk for pressure ulcers. CCN

Financial Disclosures
None reported.

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To learn more about caring for patients with pressure ulcers, read “Predictors of Pressure Ulcers in Adult Critical Care Patients” by Cox in the American Journal of Critical Care, September 2011;20(6):364-375. Available at www.ajcconline.org.

References


