Malnutrition in the Institutionalized Elderly: The Effects on Wound Healing

– Connie L. Harris, RN, ET; and Chris Fraser, HBSc, RD

Under-nutrition and protein-energy malnutrition are seen at alarmingly high rates in institutionalized elderly and in patients admitted to hospitals. A combination of immobility and loss of lean body mass — which comprises muscle and skin — and immune system challenges increases the risk of pressure ulcers by 74%. The development of pressure ulcers in the hospital affects 10% of admissions, with the elderly at the highest risk. Common causes of malnutrition in the elderly involve: decreased appetite, dependency on help for eating, impaired cognition and/or communication, poor positioning, frequent acute illnesses with gastrointestinal losses, medications that decrease appetite or increase nutrient losses, polypharmacy, decreased thirst response, decreased ability to concentrate urine, intentional fluid restriction because of fear of incontinence or choking if dysphagic, psychosocial factors such as isolation and depression, monotony of diet, and higher nutrient density requirements along with the demands of age, illness, and disease on the body. All have been found to delay healing and increase the risk of pressure ulcer development.

In addition, what is ingested should contain nutrients to support health and healing. The financial impact of malnutrition is high and the consequences for patient morbidity and mortality are severe. Practical suggestions to improve the nutritional status of long-term care residents include liberalizing previous diet restrictions where safe and appropriate, addressing impairments to dentition and swallowing, addressing physical and/or cognitive deficits, encouraging family and friends to provide favorite foods, auditing/addressing specific food underconsumption, and providing prudent nutrient supplementation. Clinicians must be aware of the numerous factors in play with regard to nutrition and its impact on not only general well-being but also on wound care. Nutritional intervention in pressure ulcer management is truly "healing from the inside out."

KEYWORDS: malnutrition, elderly, macro-nutrients, micro-nutrients, wound healing

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Under-nutrition and protein-energy malnutrition are seen in alarmingly high rates among institutionalized elderly and are estimated to affect 23% to 85% of the population.[1] The rate among patients admitted to the hospital is estimated to range from 20% to 50%.[2] A combination of immobility, loss of lean body mass (LBM) — consisting of muscle, skin — and immune system challenges increases the risk of pressure ulcers by 74%.[3]

Ms. Harris is an Enterostomal Therapist/Partner with E.T. NOW, Kitchener, Ontario. Ms. Fraser is a dietitian with Parkwood Hospital Site, St. Joseph's Health Care, London, Ontario, Canada. Please address correspondence to: Connie L. Harris, RN, ET, E.T. NOW, 120 Ottawa Street North, Suite 207, Kitchener, Ontario N2M 2S3, Canada; email: connieharris@rogers.com.

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A 1998 Swiss study showed a 10% incidence of pressure ulcer development in acute hospitals; in 2001, a German prevalence study showed an incidence of between 24% and 39% in patients over the age of 65 years. In patients with a fractured hip, many of whom are already malnourished at the time of fracture, the incidence is between 55% and 66%,[5,6] Pressure ulcers cause pain, slow rehabilitation, delayed hospital discharge, and increased costs for hospital care, long-term care facilities, and the community. This was the case with Ms. S, a frail 93-year-old with osteoarthritis and a poor appetite. Despite nutritional supplements, her serum album was 22. She was totally dependent for positioning and nourishment and during a brief illness she developed a 10 cm x 7 cm x 0.3 cm Stage III pressure ulcer on her right hip over the trochanter (see Figure 1).

This article explores the causes of malnutrition in the elderly, the role of nutrients, and the effects of malnutrition on wound healing at the cellular level and in clinical studies and offers suggestions for practical interventions to improve nutrition in this at-risk population.

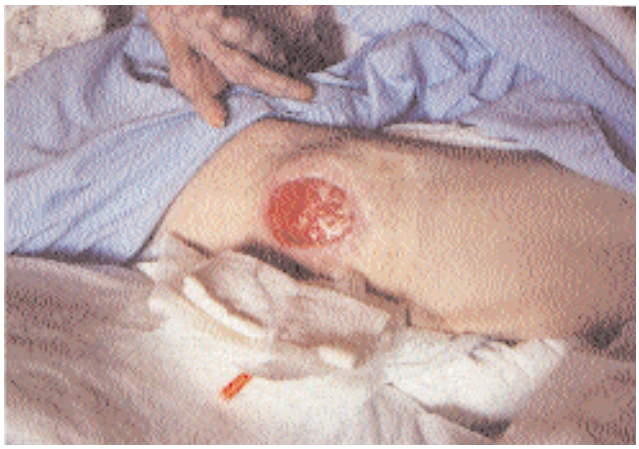

Figure 1
A frail elderly woman with a Stage III pressure ulcer.

Intrinsic Factors Risk Factors for Malnutrition in the Elderly

Both micronutrients (vitamins and minerals) and macronutrients (carbohydrates, fats, and protein) are intrinsic or essential factors in the wound healing cascade of inflammation, proliferation, and remodeling.[7] Pressure ulcer development or the presence of a chronic nonhealing ulcer places increased metabolic demands on the individual and initiates a cascade of catabolic events that includes an increase in the release of catabolic hormones (eg, cortisol and catecholamines) and a simultaneous down-regulation of the anabolic hormones (eg, testosterone and growth hormone).[8,9] This stress response results in the breakdown of the body's tissues, including stored fat and protein, in order to generate the raw materials needed for gluconeogenesis, a process that produces glucose to meet the increased energy needs associated with the metabolic stress of having a pressure ulcer. Without immediate and assertive nutritional intervention to provide the raw materials to meet this increased demand for energy, initiate wound closure, and replace potential losses during the wound healing process, healing will be delayed.[10,11] Catabolism also can be a response to stress, malnourishment, trauma, or infection. These factors can occur simultaneously, each contributing to the catabolic state, and also with inter-related cause and effect, causing a spiral of malnutrition that impacts the wound healing process.

Normal physiologic changes that occur in the aging human body contribute to undernourishment. Morley and Silver[12] described "an anorexia of aging" where a decrease in appetite results in decreased nutritional intake, weight loss, and deterioration in health in response to reduced physical activity and a decreased metabolic rate. This may lead to further inactivity and deconditioning with a loss of metabolic cell mass and physical frailty. Other factors that put the elderly at risk for malnutrition and dehydration include dependency on help for eating, impaired

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KEY POINTS

- Malnutrition in the elderly has many causes and a wide variety of serious consequences.
- In this in-depth review, the authors describe the role of nutrients in wound healing as well as common risk factors for, and signs of, malnutrition.
- Practical suggestions to improve the nutritional status of the elderly, particularly nursing home residents, are provided.

cognition and/or communication, poor positioning, frequent acute illnesses with gastrointestinal losses, medications that decrease appetite or increase nutrient losses (eg, diuretics and laxatives), polypharmacy, decreased thirst response, decreased ability to concentrate urine, and intentional fluid restriction because of fear of incontinence or choking if dysphagic.[13,14] A variety of psychosocial factors, such as social isolation and depression, also may impact negatively on an elderly individual's nutrition and hydration status.

Decreased taste thresholds[15] and a loss of olfactory distinction, more common in elderly than in younger individuals, leads to a loss of hedonistic pleasure in eating. Using a small study sample of 23 elderly and 32 young adults, De Graaf et al[16] demonstrated that, on average, the elderly preferred foods with higher flavor concentrations. In addition, poor dentition causes an inability to chew food properly and the presence of dysphagia (documented in 40% to 60% of long-term care residents) frequently contributes to undernourishment.[17,18]

Gastrointestinal changes — ie, a reduction in the mucosal surface of the small bowel diminishing the ability of the villi to absorb nutrients — were thought to impair absorption of nutrients in the elderly. Although still reported in the literature, more recent work by Corazzi et al[19] failed to confirm this theory in the absence of underlying disease. Decreased hydrochloric acid production in the stomach (hypochlorhydria) occurs in 50% of the elderly due to atrophic gastritis, in which the initiation of protein digestion is reduced and the assimilation of vitamin B_{12} is impaired.[15] The reduced stimulation of pancreatic enzymes and decreased bile released into the small intestine impairs the digestion and absorption of carbohydrates, proteins, fats, vitamins A and E and folate,[17] calcium carbonate, and ferric iron.[15] As a result, under-nutrition can occur even when nutritional intake is adequate. Constipation, fecal impaction, diarrhea, indigestion, nausea, and vomiting also inhibit appetite.[17]

Diseases such as chronic obstructive pulmonary disease (COPD), cancer, AIDS, and rheumatoid arthritis cause inflammation and increased cytokine production, which suppress appetite.[20] Acute and chronic infections, wounds, and hypermetabolism cause anorexia, micronutrient deficiencies, and increased need for energy and protein.[21] Drug side effects also cause diminished appetite and malabsorption. Depression and dementia can cause an inability to recognize the need to eat.[22] Other compounding factors that put the elderly at increased risk for malnutrition and which may or may not be associated with the factors previously noted include existing wounds, infection, neuromuscular disorders, diabetes, and renal impairment.[13]

Extrinsic Risk Factors for Malnutrition

Monotony of diet in institutional care and the timing of meals also impact nutritional wellness. Meals are often served between 9 a.m. and 5 p.m. with little or no opportunity for any flexibility or for a nutritious snack in the evening to break up the 16-hour stretch between dinner one day and breakfast the next.[23] Eating alone can reduce appetite by as much as 30% to 44%.[24,25]

Increased nutritional requirements with aging and wounds. Elderly adults with intact skin require a higher intake of dietary protein (1 to 1.5 g/kg of body weight) than younger adults (0.8 g/ kg of body weight).[26] In the older adult, energy (calorie) needs decrease; protein, fluid, fiber, vitamin, and mineral needs remain generally unchanged or increase.[27,] As a result, the nutrient density of the older adult's diet must increase, concentrating a relatively greater amount of nutrients within fewer calories.

When a wound develops, protein may need to be increased as much as 2.5 times and calories increased by 50% [11] (see Table 1). When a wound is present, the carbohydrate and total calorie intake must be adequate to meet the increased metabolic demands to spare protein and prevent its catabolism.

With protein-energy malnutrition, loss of lean body mass, which accounts for 75% of the body weight, occurs. A loss of 10% leads to impaired immunity, but the body will prioritize the need for protein substrate to allow healing. With a 20% loss, healing of existing wounds decreases and new wounds develop from loss of collagen, decreased zinc absorption, edema, weakness, and infection. Nutritional intake is directed to restoring lean body

mass, which competes with the demands of the wound.[28] Further loss of proteins occurs with heavily exudating or chronic exudating wounds and with edema.[10] In an already malnourished individual, a downward spiral of complications may commence.

Taking frequent serial weights in an elderly individual with a wound is important. Assessment of weight is inexpensive and noninvasive. Weight loss negatively impacts on wound healing. Clinicians should not assume that overweight individuals are well- or over-nourished. Weight loss in the presence of a wound implies catabolism and is not recommended. Facilities must have systems in place to monitor weight on a regular basis.[13]

The Role of Nutrients in Wound Healing

Nutrients involved in wound healing function in cellular, structural, and immune processes and in all three phases of wound healing. For example, zinc and iron play a role at the cellular level such as DNA synthesis, cell division, and proliferation. Protein, iron, zinc, and vitamins A and C are important in structural processes such as collagen synthesis and strength and re-epithelialization. These nutrients also are involved in immune processes such as antibody response, leukocyte migration to the wound, and disposal of waste products produced by leukocytes and contribute to the size and number of lymphocytes and killer T-cells.[29,30]

More than 50 nutrients, including water (dehydration is one of the most common nutrition-related problems in long term care[31]) are essential to sustain life and health, many of which play roles in skin integrity and the healing of pressure ulcers.

Carbohydrates. Adequate intake of carbohydrates is required to provide glucose to support normal cellular activities such as fibroblastic production and movement, leukocyte activity, mitosis, protein synthesis, and secretion of hormones and growth factors.[10,32] When a wound develops, need for calories increases to support the inflammatory process and cellular activity, angiogenesis (new blood vessel formation), and collagen deposition in the proliferative phase and to prevent destruction of proteins.[11]

Protein. Lymphocytes, leukocytes, phagocytes, monocytes and macrophages — immune system cells — are mainly comprised of proteins and are necessary to initiate a healthy inflammatory response in the healing process.[33] Protein-energy malnutrition may result in immunodeficiency with increased frequency and severity of infections as well as cause atrophy of the thymus gland and wasting of peripheral lymphoid tissue, with impairment of cell-mediated immune responses.[34] Activation of macrophages (necessary for phagocytosis of dead cells and antimicrobial ability), production of antigen-specific cytotoxic T-lymphocytes, and release of cytokines such as growth factors that trigger responses in other cells all are necessary to wound healing.[35] An impaired inflammatory response prevents the wound from progressing to the proliferative phase.[33] Protein-energy malnutrition reduces fibroblastic (fiber-producing) cellular activity and delays angiogenesis in the proliferative stage and reduced collagen synthesis and maturation in the remodeling stage, leading to increased wound dehiscence.[10,36]

In addition to protein catabolism to meet the increased energy needs, proteins are lost in wound exudates, which can contain as much as 100 g of protein per day.[36] A deficiency in serum albumin, which accounts for more than 50% of total serum proteins, causes impaired wound perfusion, reducing the osmotic pressure in the intravascular space. This causes interstitial edema that accumulates in the posterior trunk and legs, reducing tissue oxygenation and tissue tolerance to the forces of pressure.[37] Edema increases the distance over which nutrients must travel to reach a wound; it also may be a factor in unidentified malnutrition by masking muscle and fat loss.[29] Existing wounds may deteriorate to a more serious state. In Breslow et al's[11] long-term care study of 48 malnourished patients or those assessed to be at nutritional risk with Stage II to Stage IV pressure ulcers, the most severe hypoalbuminemia was seen in residents who developed Stage IV pressure ulcers (full-thickness skin loss with extensive destruction, tissue necrosis or damage to muscle, tissue, bone, tendon, joint, or articular capsule).

Albumin is freely permeable across cell membranes from the intravascular and extravascular compartments.[36] Albumin provides a source of amino

TABLE 1
A GUIDELINE TO SUPPLEMENTATION IN PRESSURE ULCER MANAGEMENT[57-59]

Stage of pressure ulcer	*Calories (kcal/kg)	*Protein (g/kg)	*Fluid (cc/kg)	Multivitamin‡/ mineral supplement	Vitamin C (mg)	Zinc‡ (mg, elemental†)
I	30 - 35	1.2	min. 27-30	Assess intake, losses	Assess intake, losses	Assess intake, losses
II	min. 35	1.2 - 1.5	30 - 35	Yes	Assess intake, losses	Assess intake, losses
III	35-40	min. 1.5	35 - 40	Yes*	500 bid	25 bid X 8 weeks
IV	35-40+	1.5 - 2.0\	35-40	Yes*	500 bid	25 bid X 8 weeks

* *Use clinical judgment when managing patients/residents who are significantly over- or underweight when establishing ranges for energy, protein, and fluid provision*
† *Consider a multivitamin that contains 5,000 IU of vitamin A (many standard vitamin/mineral supplements contain 3,000 to 3,500 IU of vitamin A)*
‡ *175 mg zinc gluconate = 25 mg elemental zinc; 110 mg zinc sulfate = 25 mg elemental zinc*
min. = minimum

TABLE 2
PRECAUTIONS, CONTRAINDICATIONS, AND POTENTIAL RISKS OF COMMON NUTRIENT SUPPLEMENTATION FOR PRESSURE ULCER MANAGEMENT[30,44,60,61]

Protein	Impaired renal and hepatic function
Fluid	Impaired renal and hepatic function Congestive heart failure Syndrome of inappropriate antidiuretic hormone (SIADH)
Vitamin C	Risk for oxalate stone development
Vitamin A	Chronic renal failure
Iron	Anemia of chronic disease Intractable iron deficiency anemia Liver and pancreatic disease Dialysis Constipation/gastrointestinal distress
Zinc	Gastrointestinal distress Secondary copper deficiency Alterations in immune response Alterations in lipid profile

acids. In a catabolic state or during wound healing when the body cannot meet increased energy demands, albumin deficiency impairs the inflammatory and proliferative stages. Some potential symptoms of protein deficiency include dry flaky skin; peripheral edema; sparse, easily pluckable, dyspigmented hair; muscle wasting; and cramps.[29] The key sources of protein include meat, fish, poultry, eggs, dairy products, legumes, seeds, and grains.[38]

Fats. Fats provide a concentrated source of calories, are an essential part of cell membranes, precursors for prostaglandins (which regulate numerous activities in cellular inflammation and metabolism), and contribute to local and systemic cell signaling pathways.[39,40] Essential fatty acid deficiency impairs immunocompetence.[41]

Vitamins and minerals.

Vitamin C. Vitamin C is necessary for optimal immune response, cell mitosis, and monocyte migration into the wound tissue that transforms into macrophages during the inflammatory phase of wound healing.[10] Vitamin C uptake increases during phagocytosis of debris and bacteria by macrophages; thereby, controlling infection. An antioxidant, vitamin C destroys free radicals that are by-products of oxidation and can damage healthy cells.[42] It forms bonds between the collagen fiber strands, providing extra stability and strength. If these bonds are not formed within the cell, the collagen will be rapidly broken down by collagenase, an enzyme found in the extracellular fluid. In addition, vitamin C is essential for angiogenesis; vitamin C deficiencies cause increased

capillary fragility, decreased wound strength, wound dehiscence,[32] and impaired collagen production.[33] The metabolic stress of a pressure ulcer increases the requirement for vitamin C, and studies have suggested that the concentration of vitamin C rises in healing tissue.[29] Some potential physical signs of vitamin C deficiency include bruising, purpura, petechiae around hair follicles, and swollen, spongy and/or bleeding gums.[29] The main sources of vitamin C include citrus fruits and juices, strawberries, tomatoes, sweet peppers (especially red), potatoes, broccoli, cauliflower, brussels sprouts, and cantaloupe.[38]

Vitamin A. Vitamin A improves cell-mediated immunity; is necessary for wound debridement, fibroplasia and epithelialization; and improves collagen synthesis and cross-linking. It plays a unique role in counteracting the delay in wound healing caused by steroids, diabetes, and radiation damage.[43] Vitamin A deficiency causes increased susceptibility to infections and decreases epithelialization and collagen and granulation tissue development in the proliferative stages of wound healing.[33,40]

Some potential symptoms of vitamin A deficiency include dry skin, taste and smell impairment, follicular hyperkeratosis, and corneal or conjunctival dryness.[44] Key food sources of vitamin A include liver, milk/dairy products, egg yolk, and fish oils.[29]

B vitamins. Thiamine, riboflavin, pyridoxine (vitamin B_6), folic acid, and pantothenate are B vitamins that assist in leukocyte formation, participate in metabolic processes necessary to provide energy required for the anabolic process of wound healing, and are essential cofactors in enzyme activity. Thiamine, riboflavin, vitamin B_{12}, and pyridoxine are necessary for collagen matrix synthesis.[36] Deficiencies have an indirect effect on wound healing by decreasing host resistance because of impaired antibody formation and white blood cell function, which also increases susceptibility to infections.[43] Symptoms of B vitamin deficiency include moodiness, restlessness, irritability, insomnia, sore or dry mouth and tongue, and muscular weakness. Key food sources containing many of the B vitamins include meat, liver, fish, chicken, whole and enriched grain products, beans (legumes), nuts, dairy products and leafy greens.

Vitamins E and K. Oral vitamin E is contraindicated during pressure ulcer management because it interferes with collagen synthesis, scavenges oxygen in the vicinity of the wound, prolongs the inflammatory phase of wound healing, and may interfere with the beneficial roles of vitamin A in wound management.[29,30,45]

Vitamin K is essential in the clotting process but contributes little to wound healing. However, vitamin K deficiency contributes to decreased coagulation, which impairs the inflammatory phases of wound healing.

Iron. Iron is necessary for optimizing tissue perfusion by transporting oxygen to the tissues and is necessary for collagen synthesis. A deficiency may cause increased tissue ischemia, impaired collagen cross-linking, and decreased wound strength.[39] Possible symptoms of iron deficiency include loss of energy (mild fatigue to exhaustion), pallor, sore tongue, digestive tract disturbances, appetite disorders, and brittle spoon-shaped nails.[29] Food sources of iron include meat, poultry, fish, liver, organ meats, eggs, legumes, nuts, dried fruit, fortified breads, cereals, and pastas. The iron found in animal sources is more bio-available than that provided by plant foods. Iron absorption from foods is enhanced if iron is consumed in a meal or snack that contains a source of vitamin C.[38]

Health professionals must distinguish between iron deficiency anemia and anemia of chronic disease, which may be present concurrently with many disease and inflammatory states (eg, chronic non-healing pressure ulcers). Inappropriate iron supplementation or supplementation without vigilant monitoring in anemia of chronic disease can lead to iatrogenic hemochromatosis. To detect iron deficiency, a serum ferritin assay should be ordered alone. Its utility is increased if a marker of inflammatory disease such as C-reactive protein (CRP) is assessed concurrently because ferritin is an acute phase reactant and may be elevated in the absence of iron overload in the presence of an inflammatory process. Serum iron and total iron binding capacity (TIBC), as well as ferritin, remain a valid combination of tests for the investigation of iron overload.[46,47]

Zinc. Zinc is an essential mineral found in almost every cell and important in the inflammatory phase

of wound healing, producing antibodies and activating lymphocytes. It is necessary for DNA replication and essential for cells that have a rapid cellular apoptosis/proliferation rate, including inflammatory cells, epithelial cells, and fibroblasts. Zinc stimulates the activity of more than 100 enzymes and is necessary for membrane stability and the maturation of collagen in the proliferative and remodeling phases of wound healing.[48] Deficiency may decrease rates of fibroplasia, epithelialization, and collagen synthesis as well as wound strength and impair immune response, increasing susceptibility to recurrent infections.[39] Some potential consequences of zinc deficiency include impaired sense of taste and smell, increased risk for respiratory infections, and seborrhea-like dryness and redness of the nasolabial fold and eyebrows. Food sources of zinc include meat, fish, seafood (especially oysters), poultry, liver, eggs, milk, legumes, whole wheat products, and wheat germ.[50]

The efficacy and risk of zinc supplementation for pressure ulcer management is a subject of much discussion in the literature. The general belief is that zinc supplementation is beneficial when a patient/resident is zinc-deficient but not in the absence of deficiency.[30,50] The most commonly utilized test to assess zinc status is a plasma zinc test but given the strong homeostatic control of zinc in the body and the many factors that may influence the determination of zinc status (eg, metabolic stress, sepsis, aging, exercise, time of sample, and fasting versus non-fasting sample), a significant number of authors acknowledge that assessing zinc status in patients is difficult.[30,49]

Health professionals must use clinical judgment and fully assess a patient's zinc intake, potential losses, and conditions that may negatively affect zinc status. Although a plasma or serum zinc concentration does not necessarily correlate with whole body zinc status, hospitalized patients are at particularly high risk for overt or subclinical zinc deficiency for many reasons.[51] The elderly may have a subclinical zinc deficiency; if they are injured or experience chronic ulcers, the requirement for zinc will be increased and unmet. The effects of previous deficiency will be compounded by any physical stress, such as a wound.[52] The elderly with zinc deficiency may benefit most from zinc supplementation.[29]

Zinc is absorbed primarily in the small intestine; however, only 20% to 40% of ingested zinc is absorbed. Zinc absorption is impaired by gastrointestinal disorders and by high fiber, phytate, and calcium intake. Zinc absorption is also decreased in older adults. Ninety percent (90%) of zinc loss occurs via the fecal route, including biliary losses. Gastrointestinal and pancreatic disorders, emesis, and diarrhea contribute to zinc losses. Re-absorption of endogenous zinc occurs more distally in the small bowel; therefore, disorders affecting this portion of the gastrointestinal tract will result in greater zinc excretion. Two percent (2%) to 10% of losses occur through the urinary tract. Conditions with increased urine production or increased muscle breakdown (eg, acute catabolism, diuretic use, and diabetes) and the reduced ability of kidneys to concentrate urine are factors in zinc loss.[53] Zinc is also lost through wound exudate. Large wounds, chest tubes, and subcutaneous wound drains contribute further to zinc losses. Infection, trauma, and acute stress have been associated with sequestration of zinc in the liver, resulting in depressed serum zinc levels.[30] Low serum albumin, which results in increased urinary zinc excretion, has a direct impact on zinc levels because zinc is primarily bound to albumin.[30] These and other factors such as potential drug-nutrient interactions must be taken into account when considering zinc supplementation for wound management.

If a patient receives both iron and zinc supplements, zinc should be given with meals and iron provided between meals because of the interaction and competition for absorption between iron and zinc.[54]

Practical Suggestions to Improve Nutrition Status in the Elderly

It is important to note the overlap in some of the predominant sources of protein, iron, and zinc. If a patient/resident consistently avoids high-quality protein sources provided at meals, his/her intake of protein as well as iron, zinc, and potentially other nutrients likely will be inadequate. This and other observed patterns of intake will affect nutritional status and ability to heal and must be investigated because they hold clues to underlying dental, dysphagia, gastrointestinal, food preference, and other

issues. Menus at healthcare facilities should be scrutinized to identify potential barriers to optimal nutrient provision, given the clinical challenges faced by the patients/residents.

Practical considerations to optimize oral intake in the institutionalized elderly include the liberalization of previous diet restrictions if safe and appropriate; addressing existing impairments in dentition and swallowing; and addressing physical and/or cognitive deficits related to the individual's condition or disability. Encouraging family or friends to bring favorite foods, beverages, and sauces that contain flavorings and spices that may be preferred for their cultural or taste appeal and incorporating snacks/supplements/fluids into therapy and recreation sessions may improve nutritional intake. Snack/supplement audits can be conducted to determine which items are not consumed. Addressing the reasons why snacks are not consumed and providing appropriate alternatives may help. If adequate oral intake of nutrients from foods and supplements cannot be sustained in order to meet the increased requirements of a pressure ulcer, the option of tube feeding support should be discussed with the patient/resident and his/her decision maker. Quality of life issues should be paramount in an individual's care plan.

Med pass. A practical and valuable strategy to enhance the nutritional status of institutionalized older adults is the "med pass" program.[55,56] The essence of this nutrition intervention is the provision of medications using a nutrient-dense liquid nutritional supplement rather than water, juice, or ginger ale. A small serving — (ie, 2 oz (60 mL) of a 2-calorie per mL product – provided with medications four times daily between meals is less likely to result in fullness that affects food intake than a full can provided with or between meals, and results in the consumption of an additional 500 calories and approximately 40 g of protein (depending on the product), vitamins, and minerals. Suggestions to enhance the success of this program include obtaining physician orders to support the med-pass program and recording the program onto the medication administration record. If dysphagia to regular fluids is an issue and the facility does not provide pre-thickened supplements, these supplements can be thickened at the bedside at the time of administration.

A Guideline to Supplementation in Pressure Ulcer Management

Table 1 summarizes guidelines for nutrient supplementation based on pressure ulcer stage. These guidelines were developed by summarizing the literature and outcomes in clinical practice. However, it is important to note that nutrition intervention for pressure ulcer management must be based on thorough individual assessment and take into account potential risks for supplementation, precautions, and contraindications.

Precautions, Contraindications, and Potential Risk of Nutrient Supplementation

Nutrition intervention for pressure ulcer management is not a "cookie cutter" approach — ie, recommendations for all Stage III pressure ulcers, for example — will not be identical and must be based on thorough nutrition assessment and full awareness of a patient's/resident's complete clinical picture. Table 2 highlights some of the clinical conditions in which the prescription of elevated levels of protein, fluid, vitamins C and A, iron and zinc, which require particular caution when recommending supplementation and potential risks of inappropriate supplementation are contraindicated. This list of precautions and contraindications is not exhaustive. Patients/residents who receive nutrition and hydration partially or exclusively from tube feeding may not require additional nutrient supplementation depending on assessed needs, the nutrient content of the volume of formula provided, the extent of depletion, adequacy of oral intake if any, and extent of nutrient losses. As with any nutrition intervention, thorough assessment, individualization of intervention, and sound clinical judgment are imperative.

Conclusion

Malnutrition has deleterious consequences on the body's ability to prevent and heal pressure ulcers. Wound care clinicians must identify patient/resident populations at risk for malnutrition, such as the

institutionalized elderly, and take immediate and effective preventive and corrective measures.

Ms. S — with interventions to improve her nutrition and fluid intake, meticulous moist wound healing, and pressure relief — healed her pressure ulcer and survived for 2 more years in the LTC facility. Nutritional intervention in pressure ulcer management is truly "healing from the inside out." - OWM

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