Malnutrition In Aging

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Citation

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Abstract

Malnutrition is a common, potentially serious, and frequently under-diagnosed condition among elderly individuals. Age-related physiologic changes in combination with organic and psychological disease processes contribute to the development of malnutrition in older adults. Profound malnutrition and serious illnesses often present concurrently, and each can accelerate the progression of the other. The importance of early detection and aggressive intervention is of utmost importance to arrest the downward spiral. Prompt diagnosis relies on a high index of suspicion and available screening tools. Once diagnosed, the underlying causes must be ascertained and treatment rapidly instituted. This article reviews the prevalence and effects of malnutrition in the elderly. Changes in food intake and body composition with aging are addressed, and the physiologic and pathologic causes of anorexia and weight loss in the elderly population are explored. The most commonly used screening tools for malnutrition in the elderly are discussed, along with non-pharmacologic and pharmacologic measures for treatment.

Protein energy malnutrition (PEM) is a common, potentially serious, and frequently under-diagnosed condition among elderly individuals.

Physiologic changes of aging interact with disease processes to place the elderly at increased risk for malnutrition.

Diagnosis of malnutrition is based on history taking, physical examinations and lab evaluation, and may be aided by nutritional screening tools.

Nutritional therapy is an important component in the management of malnourished individuals and when indicated, should not be delayed.

INTRODUCTION

Protein energy malnutrition (PEM) is a common, potentially serious and frequently under-diagnosed condition among elderly individuals. As humans age there is a physiological decrease in food intake that occurs to counterbalance the age-related decline in physical activity and resting metabolic rate. This has been referred to as the “anorexia of aging”. [1] This physiological anorexia increases the risk of developing pathological anorexia and weight loss when an older person develops a physical or psychological illness. Left untreated, anorexia and weight loss is associated with great morbidity and mortality in older adults. Community-dwelling elderly with PEM may exhibit reduced performance in the basic and instrumental activities of daily living as well as an increased incidence of hip fractures.[2] In the nursing home, PEM is associated with pressure ulcers, cognitive impairment, postural hypotension, infections, and anemia.[3] Its presence in hospitalized elderly patients has been linked to prolonged hospital length of stay, increased in-hospital mortality, and the development of various complications such as infections and pressure ulcers.[4,5,6,7]

The treatment of PEM has been shown to improve hospitalization outcomes in both surgical and medical elderly patients, with decreased mortality, morbidity and length of hospital stay. [8,9,10,11] These findings emphasize the importance of detecting and treating PEM in elderly patients. Many patients, however, never have their nutritional status appropriately evaluated while they are hospitalized. [12,13,14] In addition, hospitalized elderly patients are frequently allowed to remain on inadequate nutrient intake, [13,14] and even when nutritional problems are identified, adequate nutritional support is rarely provided. [15]

PREVALENCE OF MALNUTRITION

The prevalence of PEM varies greatly with the population studied and the criteria used to define malnutrition. It is estimated that up to 15% of community-dwelling and ambulatory elderly persons, [16] and 5% to 44% of homebound patients [17,18] are malnourished. The prevalence increases to 20% to 65% in hospitalized patients[19,20,21,22,23] and 23% to 85% in nursing home residents.[24,25,26] The point prevalence of PEM on admission to a long-term care facility was estimated at 54% in one study,[27] and 60% of residents showed a net weight loss following admission to an academic nursing home.[28] Blaum et al[29] report that approximately 10% of nursing home residents lost 5% of their weight within one month of admission, or 10% of their weight within six months. Nutritional compromise, however, need not be a necessary consequence of long-term care. Abbasi et al[30] report that nursing homes with aggressive evaluation and treatment policies for malnutrition had lower prevalence and fewer complications of PEM compared to nursing homes without such policies. Nutritional supplementation has been shown to improve the prevalence and outcome of PEM in long-term care facilities, with 50% of malnourished residents gaining weight and 63% showing improvement in PEM diagnostic criteria.[27]

This wide range of estimates for PEM reflects the plasticity and multitude of diagnostic criteria used to screen for and detect malnutrition, as well as the lack of a “gold standard” to establish the presence and degree of malnutrition. Over the past decade, efforts have been made to standardize and cross validate screening and diagnostic tools based on clinical outcomes and controlled prospective studies.

EFFECTS OF MALNUTRITION IN THE ELDERLY

Based on animal studies and human investigations, it is known that malnutrition can adversely affect virtually every organ system.[31] The extent of the clinical manifestation of malnutrition is related to the duration and the degree of nutritional compromise. The most striking clinical manifestations include delayed wound healing, development of pressure ulcers,[32,33,34] susceptibility to focal and systemic infections,[35,36,37] functional decline,[38,39] cognitive decline,[40] and delayed recovery from acute illness.[41,42] Hospitalized and malnourished elderly patients have nearly four times the risk of developing delirium as do those who are not malnourished.[43] Most manifestations are reversible with appropriate nutritional support, but with prolonged and profound malnutrition, clinical deterioration supervenes, culminating in irreversible organ damage and ultimately death. Profound malnutrition and serious illnesses often present concurrently, and each can accelerate the progression of the other. The importance of early detection and aggressive intervention is of utmost importance to arrest the downward spiral. Prompt diagnosis relies on a high index of suspicion and available screening tools.

CHANGES IN FOOD INTAKE AND BODY COMPOSITION WITH AGING

There is evidence from both cross-sectional[44,45,46] and longitudinal[47,48] studies that there is an increase in lean body mass and percentage of body fat in both men and women up to the age of 70 years. This is followed by a decline in both lean body mass and body fat. Data from the third National Health and Nutrition Examination Survey (NHANES III)[49] as well as other studies[50,51,52,53] clearly demonstrate a linear decline in food intake from the age of 20 to 80 years in both men and women. The average decrease in food intake was 1321 calories in males and 629 calories in females.[54] This decrease in food intake was predominantly due to a decrease in fat intake.[44] In the study by Wurtman et al,[52] older persons ate 55% less fat and 40% less carbohydrate than younger individuals. Protein consumption was equivalent in both age groups. Older persons were also less likely to snack between meals. Rolls et al[54] found that older persons consumed less calories over a single meal compared to younger persons. However, when given a yogurt preload, older subjects were likely to overeat compared to younger subjects, suggesting a failure of normal energy-setting mechanism that operate in younger persons. In another study by the same group, it was found that, in contrast to the young, older persons fail to develop sensory-specific satiety.[54]

Several investigators have demonstrated that older persons develop dysregulation of food intake based on internal cues.[55,56,57] Cook et al[58] found that following an overnight fast, older persons were less hungry and had less desire to eat than younger individuals. Clarkston et al[55] found that fasting older individuals were less hungry, and after a standardized meal they demonstrated a higher level of satiation than did younger persons. Utilizing two-day food diaries, de Castro[56] compared 3,007 healthy adults aged 20-80 years and reported that older subjects ate less but drank similar amounts of fluids compared to younger controls. They were likely to eat smaller meals and they tended to eat their meals at a slower pace. Hunger was an equal driving force for food intake in younger and older

subjects. Roberts et al[57] examined the effect of overfeeding or underfeeding in young and older subjects for 21 days. Following overfeeding, younger males decreased their food intake while older males failed to demonstrate this spontaneous hypophagia. This resulted in the younger men returning to their pre-experimental weight, while older men failed to lose weight. Following underfeeding, young men developed hyperphagia and regained the lost weight, while older persons failed to increase food intake and failed to regain the body mass lost during underfeeding.

ETIOLOGY OF THE PHYSIOLOGICAL ANOREXIA OF AGING

The physiological decrease in food intake with aging, referred to as anorexia of aging, may be attributed to several factors. Older persons show multiple changes in taste sensation. Gustatory papillae atrophy has been demonstrated to occur in humans from middle age onward.[59] Taste threshold tends to increase with advancing age, with sweet taste being less affected than other modalities.[60] Average taste-detection threshold for older persons varies across the different taste modalities, with salt showing the greatest increase. There is no connection, however, between taste detection threshold and food consumption.[61,62] Schiffman et al[63] found that while in some instances the decline in enjoyment of food seen in older persons was reversed by the use of flavor enhancers, there was no net increase in caloric intake.

There is a marked decline in the ability to smell with aging.[64] Older subjects, when blindfolded, have approximately half the ability of younger subjects to recognize blended foods.[65] This olfactory dysfunction is more marked in patients with Alzheimer's disease[66] as well as those with Parkinson's disease.[67] The changes in smell sensation with aging were related to less interest in food-related activities and to a greater intake of sweets.[68]

In addition to changes in taste and smell, aging is associated with several changes in both the central feeding system and the peripheral satiety system. Animal studies have demonstrated a decrease in the endogenous opioid feeding drive with aging,[69,70] which is attributed to the reduction in opioid receptors that occurs with aging.[71] As endogenous opioid predominantly drives fat intake,[71] this finding is in keeping with the fact that the decrease in food intake with aging is predominantly due to a decrease in fat intake.[49] In addition, animal studies have shown an increased satiating effect of cholecystokinin in older animals compared to younger ones.[73] In humans, cholecystokinin levels were found to be elevated in older malnourished persons, suggesting that this compound may play a role in the maintenance of anorexia in older persons.[74] Neuropeptide Y has been demonstrated to stimulate food intake less in older rats compared to younger rats[75] but not mice.[76] The effectiveness of other neurotransmitters on modulating food intake does not appear to change with age.[77]

Leptin concentrations do not appear to be dramatically altered with aging and are more closely related to body fat levels.[78] In humans, increases in body fat were generally accompanied by an increase in leptin levels.[79] Perry et al[79] found an increase in leptin levels at middle age in women, followed by a decline in older women. A fifteen year longitudinal study in aging individuals found that in males, but not in females, leptin concentrations declined at a slower rate compared to the rate of body fat decline.[80] Testosterone administration in older males decreases leptin concentrations,[81] and testosterone concentrations decline longitudinally with aging.[82] These findings suggest a possible role for leptin in producing the psychological anorexia of aging in males, but not females.

With aging, a decline in gastric emptying of large meals has been associated with satiation.[83] It appears that there is a decreased adaptive relaxation of the fundus of the stomach, resulting in more rapid antral filling. Nitric oxide release causes adaptive relaxation. There is a decrease in gastric nitric oxide synthase in older animals with aging.[55] In humans, infusion of glyceryl trinitrate resulted in dilation of the fundus of the stomach and thus longer retention of the food in the fundus.[84] These findings support the concept that decreased adaptive relaxation of the gastric fundus may significantly contribute to early satiation in older persons.

CAUSES OF PATHOLOGICAL ANOREXIA AND WEIGHT LOSS

The causes of anorexia and weight loss in the elderly are numerous. These causes can generally be classified into three major groups: social, psychological, and physical.

Social causes--Poverty is by far the major social cause of anorexia and weight loss in the elderly.[85]A common, but often unrecognized, cause of poverty is iatrogenic. When physicians prescribe expensive medications, older persons with limited resources often reduce their food budget to afford their medications. Social isolation due to death of family and friends is another important cause of anorexia

and weight loss in the elderly.[86]de Castro et al[87] found that socialization results in increased food intake over a meal. Elder abuse, occurring in up to 5% of older persons, can result in anorexia secondary to distress or by deliberate food deprivation by the caregiver.[88]

Other social causes include inability to shop, cook, or feed oneself. Two percent of persons aged 65-84 years require assistance with feeding. This figure rises to 7% for persons aged 85 years or older. In the 75 to 84 years age group, 12% need help managing their finances, 16% need help with food preparation, and 29% need assistance with shopping.[89] In nursing homes, failure to pay attention to ethnic food preferences may result in food refusal and anorexia.[90]

Psychological causes--Depression is one of the most important treatable causes of weight loss in older persons. In the outpatient setting, depression has been shown to be one of the most common causes of weight loss in older adults.[91] In nursing home settings, depression was consistently recognized as a major cause of weight loss and failure to thrive among residents.[92,93,94] Depression is associated with increased corticotropin releasing factor, a potent anorectic agent, in the cerebrospinal fluid.[95,96] In addition, depressed patients have many symptoms that can lead to weight loss, including weakness (61%), stomach pains (37%), nausea (27%), anorexia (22%), and diarrhea. (20%)[97] Successful treatment of depression has been shown to reverse weight loss in nursing home residents.[98]

Dementia is commonly associated with weight loss.[99,100] Older persons with dementia often forget or refuse to eat, and feeding can become a time-consuming process. Excessive wandering, psychotropic medications, paranoid ideations, and associated depression are other implicated factors. Some patients with dementia develop apraxia of swallowing and must be reminded to swallow after each mouthful of food.[85] Late-life paranoia, later-life mania, and anorexia nervosa are other psychological conditions that may contribute to malnutrition in older persons.[101]

Physical causes--Numerous medical conditions can result in anorexia and weight loss in older persons. Most of these conditions cause weight loss by one or more of the following mechanisms: hypermetabolism, anorexia, swallowing difficulty, or malabsorption. Oral disease can lead to a decrease in energy intake of about 100 kcal/day.[102] Swallowing disorders are associated with increased risk of aspiration and food aversion, which may be conscious or subconscious. Diseases that interfere with the ability of the person to eat or to prepare food, such as stroke, tremors, or arthritis, can all lead to decreased food intake.

Infections are an important cause of weight loss in older persons, especially those residing in a nursing home. It is estimated that the average nursing home resident acquires a new acute infection every three months.[103] Infection may result in confusion, anorexia, and negative nitrogen balance, all of which may contribute to anorexia and weight loss.[104] Persons with chronic obstructive pulmonary disease experience a decrease in arterial oxygen tension when eating due to the thermic energy of eating and the brief interruption of respiration with swallowing. They frequently are unable to complete their meals due to dyspnea.[105] Their weight loss is further aggravated by hyperventilation and use of accessory muscles, leading to increased metabolism.[106] Acquired immunodeficiency syndrome (AIDS) and rheumatoid arthritis result in increased levels of circulating cytokines which in turn lead to increased resting energy expenditure and decreased serum albumin levels.[107] Hyperthyroidism,[108] Parkinson's disease,[109] and pheochromocytoma[110] cause hypermetabolism and may result in weight loss. Pancreatic insufficiency and gluten enteropathy may cause weight loss through malabsorbtion.

Numerous medications are associated with weight loss. Those most frequently implicated include digoxin, theophylline, nonsteroidal anti-inflammatory drugs, and psychotropic drugs such as fluoxetine, lithium and phenothiazines. Therapeutic diets are often unpalatable and poorly tolerated by older persons, leading to weight loss. In nursing homes, diabetic diets appear to have no effect on diabetic control.[111] Therapeutic diets, including low salt, low fat, and sugar-restricted diets should be avoided in the elderly.

DIAGNOSING MALNUTRITION

Nutritional assessment in an older person should involve taking a thorough history and physical examination in addition to anthropometric and biochemical measures. A carefully obtained history is the most valuable tool for identifying persons at risk for malnutrition. Weight change over time is one of the most important aspects of the history. Unintentional weight loss of 10 pounds or more over a period of 6 months is a strong indicator of nutritional risk and morbidity.[112] A dietary history includes providing the patient with a simple questionnaire (food diary) that inquires into quantity as well as quality of food intake. The history should also ascertain the presence of risk factors for

deficient nutrition intake such as poverty, social isolation, and inability to shop, prepare food, or feed.[3] Additionally, any chronic medical condition that may potentially affect nutritional status must be documented, such as diabetes, cardiopulmonary disease, cerebrovascular disease, gastrointestinal conditions, depression, dementia, and rheumatological disease. Acute illnesses may demand increased nutritional requirements, and the frequency and severity of such events must be noted. Review of both prescription and over-the-counter medications is essential to avoid polypharmacy. Many commonly used drugs are anorexigenic, notably digoxin, theophylline, non-steroidal anti-inflammatory drugs (NSAIDs), and psychotropic drugs such as fluoxetine, lithium and phenothiazines.[113] A comprehensive list of potential drug offenders can be found at http://ltcnutrition.org.

The physical examination should determine general body habitus, present body weight and height, and the presence of any sign of nutritional deficiency in the skin, hair, nail, eyes, mouth, or muscles. The body mass index (BMI) is a useful measurement for assessing nutritional status and can be calculated using the formula (BMI= Weight (kg)/ [height (m)] ^{2}). The association between BMI and mortality in older adults follows a J-shaped curve, unlike the U-shaped curve relationship in younger adults.[114,115] Data from several studies of elderly aged 60-90 years indicate the lowest mortality occurred at progressively increasing body weight,[116] and higher mortality occurred with lower body weight.[117] The desired BMI for older people is 24 to 29, compared with 20 to 24 in younger persons, and a measure below 24 is an indicator of malnutrition in older persons.[118] BMI, however, may not be as informative in the elderly as it is at younger ages. There is little documentation relating BMI to direct measurements of body composition in the elderly, especially at very old ages or in non-caucasian ethnic groups. In addition, stature often cannot be measured accurately in the elderly individuals because of increased prevalence of spinal curvatures, which is reported to be as high as 30%.[119] For such individuals, the estimation of stature from knee height is probably the best method for providing this information.[120] Knee height has been measured in the Third National Health and Nutrition Examination Survey, or NHANES III, for persons 60 years of age or older.

Other anthropometric methods include measurements of arm circumference, mid-arm muscle area, calf circumference, triceps skin-fold, and subscapular skin-fold thickness. Calf circumference has been recommended as a more sensitive measure of the loss of muscle mass in the elderly than arm circumference and midarm muscle area.[119] Skin-fold thickness is frequently used to assess body fat stores. The accuracy of this technique in nutritional evaluation is hampered by the unpredictable response of subcutaneous fat to undernutrition and the absence of a definite correlation between skin-fold thickness and total body fat. In older men, skin-fold thickness over the scapula and iliac crest have been found to correlate most closely with total body fat, while the triceps and the thigh are the preferred measurement sites in older women.[121]

LABORATORY EVALUATION

Serum albumin level is the most frequently utilized biochemical marker for malnutrition. With a half-life of approximately three weeks, albumin is a good indicator of baseline nutritional status but is less useful in assessing effectiveness of acute nutritional intervention. Furthermore, cytokines and postural changes, commonly present in acute illness, may result in lower albumin levels. Cytokines such as tumor necrosis factor, interleukin-2 and interleukin-6 inhibit synthesis of albumin at the gene level[122,123] and may facilitate extravasation of albumin from the intravascular to the interstitial space.[124,125] The dilution effect of chronic recumbency is associated with increased intravascular volume and may diminish measured albumin by as much as 0.5 mg/dL.[126] These factors, in part, explain the rapid decline in serum albumin levels (beyond what is expected from diminished nutritional intake) shortly after an elderly person is hospitalized.[127] Nonetheless, albumin levels less than 3.5 mg/dL are strongly suggestive of PEM,[128] and levels less than 3.2 mg/dL are excellent predictors of mortality and morbidity in the elderly. There is a 24% to 56% increase in the likelihood of dying for every decline of .35mg/dL in serum albumin.[129]

Serum cholesterol level is another biochemical marker of malnutrition. Serum levels less than 160 mg/dL suggest low lipoprotein levels, and thus low visceral protein. Rudman et al1[130] showed cholesterol levels below 160 mg/dL to be highly predictive of subsequent mortality in a cohort of nursing home residents, and even lower levels were correlated with a ten-fold increase in mortality. A decrease in cholesterol levels from above 160 mg/dL to less than 120 mg/dL during acute hospitalization has been associated with increased length of stay, complications, and mortality.[131] Hypocholesterolemia, however, develops later in the progression of malnutrition, so its value as a screening tool

is limited.

Total lymphocyte count (TLC) is another useful screening test. Selzer et al[132] reported a four-fold increase in mortality with a TLC of less than 1500 cells/mm. Levels less than 800 cells/mm indicate severe malnutrition.[133] Clinically, this is reflected in suppressed cellular immunity, as evidenced by delayed cutaneous hypersensitivity testing with common antigens such as Candida. Specifically, the CD4 cell count has been noted to drop with progressive malnutrition,[134] and the CD4:CD8 ratio declines, as occurs with HIV patients. The relative immune compromise is reversed with nutritional support, but, left untreated, may progress to sepsis and death. As with hemoglobin, a low white blood cell count may be related to specific nutrient deficiencies such as folate, vitamin B12, and iron.

Proteins with a shorter half-life than albumin are occasionally used to assess acute response to nutritional intervention. Prealbumin, with a half-life of two days, and retinol binding proteins, with a half-life of two hours, fit this criterion. Leptin, a protein produced by fat cells, is a good marker of total body fat. It is being studied and validated as a nutritional marker but is affected by testosterone levels, age, and gender. It is important to note that all these tests are affected by non-nutritional factors and none alone is adequately sensitive or specific to diagnose malnutrition.

SCREENING TOOLS

Since no single physical finding, historical fact, or biochemical test in itself is a sufficient predictor or determinant of malnutrition, several screening tools have been developed to better document and monitor PEM. The most common and best-evaluated are discussed below.

The Instant Nutritional Assessment (INA)-- The INA is one of the simplest and most practical nutrition screening tools. It was introduced by Seltzer and coworkers[135] in 1997, and is widely used. It combines three easily obtainable elements – lymphocyte count, albumin, and weight change. This test is often referred to as “LAW”, the first letter of each item. While each item alone may have low predictive value, used together the three identify individuals at risk for malnutrition with a high degree of accuracy.

The Subjective Global Assessment (SGA)-- The SGA differs from the INA in that it does not use laboratory criteria but relies heavily on functional capacity and physical signs of malnutrition. Specifically, it combines information from the patient's history (such as weight loss, dietary intake, functional status), physical examination (such as muscle and fat distribution, edema), and the clinician's judgement. As such, it is highly dependent on the screening clinician for accuracy. On initial validation, its ability to predict infection as a complication of malnutrition was compared to six other independent methods (including albumin and anthropometric measures). The SGA was found to be 82% sensitive and 72% specific,[136] and outperformed all six methods. However, when this study was repeated with less experienced clinicians, the results were much less promising.[137] Other studies have also led to the conclusion that the SGA is only reliable in the hands of well-trained clinicians.

The nutritional screening initiative and the DETERMINE checklist-- The DETERMINE checklist was developed by the Nutritional Screening Initiative (NSI), an interdisciplinary multiorganizational effort aimed at introducing nutritional screening into the American health care system.[138] The DETERMINE Your Nutritional Health Checklist is a self-administered list of ten questions covering different risk factors for malnutrition. Four questions cover dietary concerns, four questions cover general health assessment, and two questions cover social and economic issues. Each question is scored according to its importance as determined by the developer of the checklist. Patients with a total score of six or higher (highest score is 21, with higher being worse), are directed to various follow up and assessment.

The DETERMINE Checklist was developed as a screening, education, and public awareness tool. It is not a reliable diagnostic tool, as a high score was shown by Sahyoun et al[139] to be a weak predictor of mortality. It has shown somewhat better predictability as a screening tool in retrospective and prospective validation studies. This tool has found widespread use; one of its best attributes is in promoting public awareness of malnutrition.

The malnutrition risk scale (SCALES)--The malnutrition risk scale was developed by Morley et al[140] as an outpatient screening tool. The acronym SCALES represents the six elements in this screening tool (Sadness, Cholesterol, Albumin, Loss of weight, Eating problems, and Shopping) that cover common known risk factors for malnutrition, including depression, which has emerged as a major risk factor for malnutrition and death,[141] but is often overlooked. The SCALES screening does not include functional or physical assessments, and therefore the user

does not need to be a trained or experienced professional. A score of three or higher suggests high risk for malnutrition.

The Mini-Nutritional Assessment (MNA)-- The MNA[142] is a simple, rapid, and reliable tool for assessing nutrition in the elderly and has rapidly become the screening tool of choice for many geriatric clinicians. It is frequently used in the nursing home setting and is composed of 18 items that require a professional to administer. It requires 10-15 minutes to complete and does not require laboratory tests. It consists of four sections: anthropometric, general, dietary, and self-assessment. The MNA was shown to be 98% accurate when compared with a comprehensive nutritional assessment which included food records and laboratory tests.[143] In another study, there was no added benefit in adding biochemical measurements, as the test was highly accurate with or without these tests.[143]

MANAGEMENT OF ANOREXIA AND WEIGHT LOSS

The cornerstone in management of anorexia and weight loss is early detection and prompt treatment of reversible disorders. The major treatable causes of weight loss are summarized in table 1.[144,145] Special attention should be given to treating depression and eliminating anorexogenic medications. Dietary restrictions should be eliminated when possible, with the diet liberalized to patient preferences. Delivering hot meals through programs such as Meals on Wheels and providing feeding assistance to patients with disabilities or dementia often helps maintain food intake to adequate levels. Older patients with dysphagia can often be taught by a speech pathologist the correct swallowing techniques and positioning for swallowing safely. Dietary manipulation, such as thickened liquids, is an important component of dysphagia management.

Table 1: Commonly encountered reversible causes of anorexia and weight loss in older persons

- Polypharmacy
- Depression
- Alcoholism
- Swallowing disorders
- Poor dentition
- Poverty
- Hyperthyroidism
- Hyperparathyroidism
- Hypoadrenalism
- Malabsorption
- Inability to feed
- Therapeutic diets
- Dementia (excessive wandering, and paranoia)
- Infections

Nutritional therapy is an important component in the management of malnourished individuals. Nutrition therapy can take one of three forms: oral caloric supplementation, tube feeding, or parenteral feeding. Numerous oral and enteral feeding formulas are available and differ in source and percent proteins and fat, lactose content, osmolality, calorie per unit volume, and cost. There is emerging evidence that oral supplementation improves outcome in elderly persons with hip fractures,[146] pressure ulcers,[147] and pneumonia.[148] In some nursing homes, “medication pass” supplementation is practiced. This consists of providing liquid caloric supplementation at the time medications are passed, and in the malnourished elderly such a practice may increase the daily caloric intake by up to 15%. Most oral formulas provide 1 kcal/ml, and the total amount of calories consumed is simply the volume, in milliliters, consumed.

In cases where metabolic demand exceeds what can be met by oral intake alone (e.g. cancer cachexia) or with mechanical problems (e.g. esophageal obstruction, endotracheal intubation, neuromuscular disease, or orofacial surgery), a brief course of tube feeding may be indicated. Access to the gut is achieved by a nasogastric (NG) or nasointestinal tube, or a percutaneous gastric (PEG) or percutaneous intestinal (PEJ) tube. Aspiration of gastric content is the most serious complication of feeding tubes, and is very common, even with aspiration precautions. As much as 40% of deaths related to tube feeding are due to aspiration pneumonia, with aspiration occurring in 44% of patients with NG tubes, and 56% of patients with PEG tubes in a long-term care setting.[149] Duodenal tubes do not provide added protection against aspiration. In fact, a false sense of security with feeding tubes that traverse the pylorus may lead to relaxation of reflux precautions, increasing the risk of aspiration. Medications that decrease the lower esophageal sphincter or delay gastric emptying must be

avoided if possible.

In a study of 1386 nursing home residents with cognitive impairment and malnutrition, Mitchell et al[150] found no evidence that tube feeding prolonged survival. In addition, Weaver et al[151] found no improvement in the quality of life or morbidity in tube-fed patients. Tube feeding candidates must therefore be selected carefully, and a return to oral feeding must occur as soon as possible. Similar concerns surround the use of central or peripheral parenteral feeding. Parenteral feeding should be limited to relatively brief periods of time and is restricted to individuals with a nonfunctional gut. It is of benefit in acute management of pseudomembranous colitis, acute pancreatitis, or a severe flare-up of Crohns disease to rest the gut while recovery occurs. Metabolic parameters and electrolytes must be monitored closely. Parenteral nutrition has no place in the management of chronic age-related malnutrition.

Several medications have been suggested for the treatment of anorexia and weight loss in older persons (table 2). Megestrol acetate has been used successfully to stimulate appetite and promote weight gain in patients with AIDS[152] and cancer.[153] Megestrol acetate, dosed at 160 mg daily, was shown to prevent nutritional deterioration in older males with head and neck cancer receiving either radiation or chemotherapy.[154] Administration of megestrol acetate to elderly malnourished nursing home residents resulted in mild weight gain but was associated with a variety of side effects including delirium, megacolon, edema and congestive heart failure.[155]

Growth hormone has been shown to stimulate weight gain in severely malnourished older patients.[156,157] Growth hormone is extremely expensive, however, and treatment for more than six months has been associated with a variety of side effects including arthralgias, carpal tunnel syndrome, and gynecomastia.[158,159] The use of growth hormone to treat intensive care unit patients with severe malnutrition resulted in increased mortality.[160] Oxandrolone, an oral anabolic steroid with potent anabolic activity and minimal androgenic activity, was shown to have a positive impact on weight gain in patients with AIDS[161] and alcoholic hepatitis.[162] Nandrolone, another anabolic steroid, improved nutritional status in patients with renal failure.[163] Testosterone increased muscle strength and decreased leptin levels in older persons.[164] In another study, it decreased fat mass and increased muscle mass.[165]

Table 2: Drugs available for the treatment of anorexia and weight loss

- Megestrol acetate
- Oxandrolone
- Testosterone
- Cyproheptadine
- Dronabinol
- Metoclopramide

Metoclopramide, a prokinetic agent, has been used to treat early satiation and anorexia. It was also found to stimulate appetite in persons with anorexia caused by the cancer-associated dyspepsia syndrome.[166] Its use, however, may result in dystonic reactions and worsening of parkinsonian symptoms. Cyproheptadine, an antiserotonergic agent, has been shown to have a mild positive effect on appetite in malnourished adults.[167] Duranabinol (tetrahydrocannabinol) is an antiemetic that promotes food intake. It has been shown to promote mild weight gain in patients with cancer or AIDS[168] and stimulate weight gain when administered to patients with Alzheimer's disease.[169] Adverse effects include euphoria, somnolence, and fatigue.

CONCLUSION

Malnutrition in the elderly is a common problem albeit under diagnosed and when diagnosed is often undertreated. Early detection is the key to management of malnutrition. Older persons experience physiological anorexia in response to decreased metabolism and physical activity. This places them at increased risk for developing malnutrition when a disease process occurs. The majority of causes of anorexia and weight loss in older persons are treatable. The cornerstone to proper management of malnutrition in the elderly is early detection and prompt treatment of reversible causes. Nutritional assessment and screening should be an integral part of the overall care of the elderly. The diagnosis of malnutrition should not be based on a single marker. A thorough history and physical examination, in conjunction with anthropometric and biochemical measures should be utilized. The use of nutritional supplementation, when indicated, should not be delayed. Although several potential drugs have been suggested for the management of anorexia and weight loss, none of these appear to be sufficiently cost effective for routine use, or sufficiently tolerated for long term use.

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References

1. Morley JE, Silver AJ: Anorexia in the elderly. Neurobiol Aging 9:9-16, 1985.
2. Sullivan DH, Wallas RC: Impact of nutritional status on morbidity in a population of geriatric rehabilitation patients. J Am Geriatr Soc 42:471-477, 1994.
3. Kamel HK, Morley JE: Anorexia of Aging. "Oxford Text Book of Geriatric Medicine," 2nd ed. London: Oxford University Press pp 158-168, 2000.
4. Hickman DM, Miller RA, Rombeau JL: Serum albumin and body weight as predictors of postoperative course in colorectal cancer. J Parenter Enter Nutr 4:314-316, 1980.
5. Sullivan DH: Risk factors for early hospital readmission in a select population of geriatric rehabilitation patients; the significance of nutritional status. J Am Geriatr Soc 42(5):471-477, 1994.
6. Kamel HK, Karcic E, Karcic A, Barghouthi H: Nutritional Status of Hospitalized Elderly; differences between nursing home patients and community-dwelling patients. Annals of Long-Term Care 8(3):33-38, 2000.
7. Kamel HK, Azharrudin A, Thomas DR: Hypoalbuminemia and effect on outcomes in hospitalized patients. J Investing Med 46(70):293-295, 1998.
8. Sagar S, Harland P, Shields R: Early postoperative feeding with elemental diet. BMJ 1:293-295, 1979.
9. Askanazi J, Hensle TW, Starker PM: Effect of immediate postoperative nutritional support on length of hospitalization. Ann Surg 203:236-239, 1986.
10. The Veterans Affairs Total Parenteral Nutrition Cooperative Study Group: Preoperative total parenteral nutrition in surgical patients. N Engl J Med 325:525-532, 1991.
11. Bastow MD, Rawlings J, Allison SP: Benefits of supplementary tube feeding after fractured neck of femur: a randomized controlled trial. BMJ 287:1589-1592, 1983.
12. McNab H, Rstivo R, Ber L: Dietetic quality assurance practices in Chicago area hospitals. J Am Diet Assoc 85:635-637, 1987.
13. Prevast EA, Butterworth CE: Nutritional care of hospitalized patients. Am J Clin Nutr 27:432, 1974.
14. Riffer J: Malnourished patients feeding rising costs. Hospitals 60:86, 1986.
15. Mullen JL, Buzby GP, Mattews DC: Reduction of operative morbidity and mortality by combined preoperative and postoperative nutritional support. Ann Surg 192:604-613, 1980.
16. Sullivan DH: Undernutrition in older adults. Ann of Long-Term Care 8:41-46, 2000.
17. Cederholm C, Hellerstrom K: Nutritional status in recently hospitalized and free-living elderly subjects. Gerontology 38:105-110, 1992.
18. Peter D. Hart Research Associates Inc: Nutrition screening initiative: telephone survey of 750 doctors, nurses, and healthcare administrators. April 1-8, 1993.
19. Bienia R, Ratcliff S, Barbour GL: Malnutrition in the hospitalized geriatric patient. J Am Geriatr Soc 30:433-436, 1982.
20. Costans T, Bacq Y, Brechot JF: Protein-energy malnutrition in elderly medical patients. J Am Geriatr Soc 40:263-268, 1992.
21. Mowe M, Bohmer T: The prevalence of underdiagnosed protein-calorie undernutrition in a population of hospitalized elderly patients. J Am Geriatr Soc 39:1089-1092, 1991.
22. Willard MD, Gilsford RB, Price RA: Protein-calorie malnutrition in a community hospital. JAMA 243:1720-1722, 1980.
23. Bistrian BR, Blackburn GL, Vitale J: Prevalence of malnutrition in general medical patients. JAMA 235:1567-1570, 1976.
24. Silver AJ, Morley JE, Strome LS: Nutritional status in an academic nursing home. J Am Geriatr Soc 36:487-491,1988.
25. Shaver HJ, Loper JA, Lutes RA: Nutritional status of nursing home patients. J Parenter Enteral Nutr 4:367-370, 1980.
26. Muncie HJ, Carbonetto C: Prevalence of protein-calorie malnutrition in an extended care facility. J Fam Pract 14:1061-1064, 1982.
27. Thomas DR, Verdery RB, Gardner L: A prospective study of outcome from protein-energy malnutrition in nursing home residents. J Parenter Enteral Nutr 15:400-404, 1991.
28. Diongi R, Dominioni L, Jemos V: Diagnosing malnutrition. Gut 27:5-8, 1986.
29. Blaum CS, Fries BE, Fiatarone MA: Factors associated with low body mass index and weight loss in nursing home residents. J Gerontol A Biol Sci Med Sci 50:162-168, 1995.
30. Abbasi AA, Rudman D: Observations on the prevalence of protein-calorie undernutrition in VA nursing homes. J Am Geriatr Soc 41:117-121, 1993.
31. Silberman H: Consequences of malnutrition. In: Silberman H (ed): "Parenteral and Enteral Nutrition," Norwalk, CT: Appleton & Lang, pp 1-18,1989.
32. Breslow RA, Hallfrisch J, Guy DG: The importance of dietary protein in healing pressure ulcers. J Am Geriatr Soc 41:357-62, 1993.
33. Pinchcofsky-Devin GD, Kaminski MV: Correlation of pressure sores and nutritional status. J Am Geriatr Soc 34:87, 1986.
34. Allman RM, Laprade CA, Noel LB: Pressure sores among hospitalized patients. Ann Intern Med 105:337, 1986.
35. Kaiser FE, Morley JE: Idiopathic CD4+ T lymphopenia in older persons. J Am Geriatr Soc 42:1291-1294, 1994.
36. Thompson JS, Robbins J, Cooper JK: Nutrition and immune function in the geriatric population. Clin Geriatr Med 3:309, 1987.
37. Chandra RK: Nutrition-immunity-infection interactions in old age. In Chandra RK (ed): "Nutrition, immunity and illness in the elderly," New York: Pergamon Press, pp 173, 1985.
38. Sem SW, Nes M, Engedal K: An attempt to identify and describe a group of non-institutionalized elderly with the lowest nutritional score. Compr Gerontology 2:60, 1988.
39. Bianchetti A, Rozzini R, Carabellese C: Nutritional intake, socioeconomic conditions, and health status in a large elderly population. J Am Geriatr Soc 38:521,1990.
40. Goodwin JS, Goodwin JM, Garry PJ: Association between nutritional status and cognitive function in a healthy elderly population. JAMA 249:2917-2921, 1983.
41. Buzby GP, Mullen JL, Mathews DC: Prognostic nutritional index in gastrointestinal surgery. Am J Surg 139:160-167, 1980.
42. Miller DK, Morley JE: Nutritional Epidemiology. Ann Rev Gerontology 15:20-53, 1995.
43. Inouye SK, Charpentier PA: Precipitation factors for delirium in hospitalized elderly persons. Predictive model and interrelationship with baseline vulnerability. JAMA 275:852-857, 1996.

44. Silver AJ, Guillen CP, Kahl MJ: Effect of aging on body fat. J Am Geriatr Soc 41:174-180, 1993.
45. Aloia JF, Vaswani A, Ma R, Flaster E: Aging in women-the four-compartment model of body composition. Metabolism 45:43-48, 1996.
46. Kuskowska-Wolk A, Rossner S: Prevalence of obesity in Sweden: cross-sectional study of a representative adult population. J Intern Med 227:241-246, 1990.
47. Steen B, Isaksson B, Svanborg A: Body composition at 70 and 75 years of age: a longitudinal population study. J Clin Exp Gerontol 1:185-192, 1979.
48. Chumlea WC, Garry PJ, Hunt WC, Rhyne RL: Distributions of serial changes in stature and weight in healthy elderly population. Hum Biol 60:917-925, 1988.
49. Third National Health and Nutritional Examination Survey, phase III: Daily dietary fat and total food energy intake. MMWR Morb Mortal Wkly Rep 43:116-125, 1994.
50. Subar AF, Harlan LC, Mattson ME: Food and nutrient intake differences between smokers and nonsmokers in the US. Am J Publ Health 80:1323-1329, 1990.
51. Third National Health and Nutritional Examination Survey, phase III: Daily dietary fat and total food energy intake. MMWR Morb Mortal Wkly Rep 43:116-125, 1994.
52. Wurtman JJ, Leiberman H, Tsay R, Nader T, Chew B: Calorie and nutrient intakes of elderly and young subjects measured under identical conditions. J Gerontol 43:174, 1988.
53. Rolls BJ, Dimeo KA, Shide DJ: Age-related impairments in the regulation of food intake. Am J Clin Nutr 62:923, 1995.
54. Rolls BJ, Mcdermott TM: Effect of age on sensory-specific satiety. Am J Clin Nutr 54:99,1991.
55. Clarkston WK, Pantano MM, Morley JE: Evidence for the anorexia of aging: gastrointestinal transit and hunger in healthy elderly vs. young adults. AM J physiol 273:755, 1997.
56. de Casto JM: Age-related changes in spontaneous food intake and hunger in humans. Appetite 21:255, 1993.
57. Roberts SB, Fuss P, Heyman MB: Control of food intake in older men. JAMA 27:216, 1994.
58. Cook CG, Andrews JM, Jones KL: Effects of small intestinal nutrient infusion on appetite and pyloric motility are modified by age. Am J Physiol 273:755, 1997.
59. Aliara E: Investigations on the human taste organs: the structure of taste papillae at various ages. Arch Anat Embriol 42:506-514,1939.
60. Weiffenbach JM: Chemical senses in aging. In Getchell TV (ed): " Smell and taste in health and disease," New York: Raven, pp 69-92, 1991.
61. Drewnoski A, Henderson SA, Driscoll A, Rolls BJ: Salt taste perceptions and preferences are unrelated to sodium consumption in healthy older adults. J Am Diet Assoc 96:471-474, 1996.
62. Stevens JC, Cain WS, Demaque A, Ruthruff AM: On the discrimination of missing ingredients: aging and salt flavor. Appetite 16:129-140, 1991.
63. Schiffman SS, Warwick ZS: Effect of flavor enhancement of foods for the elderly on nutritional status: food intake, biochemical indices, and anthropometric measures. Pysiol Behav 53:395-402, 1993.
64. Doty RL, Shaman P, Applebaum SL, Giberson R, Siksoski L, Rosenberg L: Smell identification ability: changes with age. Science 226:1441-1443, 1984.
65. Schiffman S: Food recognition by the elderly. J Gerontol 32:586-592, 1997.
66. Ter Laak HJ, Renkawek K, Van Workun FP: The olfactory bulb in Alzheimer's disease: a morphologic study of neuron loss, tangles and senile plaques in relation to olfaction. Alz Dis Assoc Disord 8:38-40, 1994.
67. Doty RL, Golbe LI, McKeown DA: Olfactory testing differentiates between progressive supranuclear palsy and idiopathic Parkinson's disease. Neurology 43:962-965, 1993.
68. Duffy VB, Backstrand JR, Ferris AM: Olfactory dysfunction and related nutritional risk in free-living, elderly women. J Am Diet Assoc 95:879-886, 1995.
69. Gosnell BA, Levine AS, Morley JE: Effects on aging on opioid modulation of feeding in rats. Life Sci 32:2793-279, 1983.
70. Kavaliers M, Hirst M: The influence of opiate agonists on day-night feeding rhythms in young and old mice. Brain Res 326:160-167, 1985.
71. Morley JE, Kumar VB, Mattammal M: Measurement of nitric oxide synthase and its mRNA in genetically obese (ob/ob) mice. Life Sci 57:1327-1331 1995.
72. Morley JE, Flood JF, Silver AJ: Opioid peptides and aging. An NY Acad Sci 579:123-32, 1990.
73. Silver AJ, Flood JF, Morley JE: Effect of aging on fluid ingestion in mice. J Gerontol 46:117-121, 1991.
74. Bertelemy P, Bousisson M, Vellas B: Postprandial cholecystokinin secretion in elderly with protein-energy undernutrition. J Am Geriatr Soc 40:365-369, 1992.
75. Pich EM, Messori B, Zoli M: Feeding and drinking responses to neuropeptide Y injections in the paraventricular hypothalamic aged rats. Brain Res 575:267-271, 1992.
76. Morley JE, Hernandez EN, Flood JF: Neuropeptide Y increases food intake in mice. Am J Physiol 253:516-522, 1987.
77. Rosenthal MJ, Morley JE: CRF and age-related differences in behavior of mice. Neurobiol Aging 10:167-171, 1989.
78. Nicklas BJ, Toth MJ, Goldberg AP, Poehlman ET: Racial differences in plasma leptin concentrations in obese postmenopausal women. J Clin Endocrinal Metab 82:315-317, 1997.
79. Perry HM, Morley JE, Horowitz M, Kaiser FE, Miller DK, Witter G: Body composition and age in African American and Caucasian women; relationship to plasma leptin levels and gonadal steroids. Metabolism 46(12):1399-405, 1997.
80. Baumgartner RN, Walters DL, Morley JE, Patrick P, Montoya GD, Garry PJ: Age-related changes in sex hormones affect the sex difference in serum leptin independently of changes in body fat. Metabolism 48(3):378-84, 1999.
81. Sih R, Morley JE, Kaiser FE, Perry HM: Testosterone replacement in older hypogonadal men: a 12-month randomized controlled trial. J Clin Endocrinol Metabol 82:1661-1667, 1997.
82. Morley JE, Perry HM, Kaiser FE: Longitudinal changes in testosterone, luteinizing hormone and follicle stimulating hormone in healthy older males. Metabolism 46:410-413, 1996.
83. Morley JE, Kumar VB, Mattammal MB: Inhibition of feeding by a nitric oxide synthase inhibitor: effect of aging. Euro J Pharmacol 311-315, 1996.
84. Morley JE, Thomas DR: Anorexia and aging: Pathophysiology. Nutrition 15(6):499-503, 1999.
85. Morley JE, Morley PMK: Psychological and social factors in pathogenesis of weight loss. Ann Rev Gerontal Geriatr 15:83-109, 1995.
86. McIntosh WA, Shifflet PA, Picou JS: "Social" support stressful events, strain, dietary intake and elderly. Med Care 27:140-53, 1989.
87. De Castro JM, Brewer EM, Elmore DK, Orazco S: Social facilitation of the spontaneous meal size of humans occurs regardless of time, place, alcohol or snacks. Appetite

15:89-101, 1990.
88. Lachs MS, Fulmer T: Recognizing elder abuse and neglect. Clin Geriatr Med 48:3-10, 1993.
89. Guralnik JM, Simonsick E:. Physical disability in older Americans. J Gerontol 48:3-10, 1993.
90. Morley JE: Anorexia in older patients: its meaning and management. Geriatrics 45:59-66, 1990.
91. Thompson MP, Morris LK: Unexplained weight loss in the ambulatory elderly. J Am Geriatr Soc 35:927-932, 1987.
92. Morley JE, Kraenzle D: Causes of weight loss in a community nursing home. J Am Geriatr Soc 42:583-585, 1994.
93. Katz IR, Beaston-Wimmer p, Parmelee P, Friedman E, Lawton MP: Failure to thrive in the elderly: exploration of the concept of delineation of psychiatric components. J Geriatr Psychiatry Neurol 6:161-169, 1994.
94. Silver AJ, Morley JE, Strome LS, Jones D, Vickers L: Nutritional status in academic nursing home. J Am Geriatr Soc 36:487-491, 1988.
95. Krahn DD, Gosnell BA, Levine AS, Morley JE: Behavioral effects of corticotropin-releasing- factor: localization and characterization of central effects. Brain Res 443:63-69, 1988.
96. Nemeroff CB, Bissetti G, Widerov E: Elevated concentration of corticotropin-releasing-factor-like immunoreactivity in depressed patients. Science 226:1342-1343, 1984.
97. Kivela SL, Nissinen A, Tuomiiehto J: Prevalence of depressive and other symptoms in elderly Finnish men. Acta Psychiatr Scand 73:93-100, 1986.
98. Morley JE, Kraenzle D: Weight Loss. J Am Geriatr Soc 43:82-83, 1995.
99. Sandman PO, Adolfsson R, Nygren C. Halimans G, Umbiac B: Nutritional status and dietary intake in institutionalized patients with Alzheimer's disease and multi-infarct dementia. J Am Geriatr Soc 35:31-38, 1987.
100. Chenoweth B, Spencer B. Dementia: the experience of family caregivers: Gerontologist 26:267-274, 1986.
101. Morley JE, Anorexia in older persons: Drugs & Aging 8(2)134-155, 1996.
102. Sullivan DH, Martin W, Flaxman, Hagen JE: Oral health problems and involuntary weight loss in a population of frail elderly. J Am Geriatr Soc 41:725-31, 1993.
103. Farber BF, Brennen C, Puntereri AJ: A prospective study of nosocomial infections in chronic care facility. J Am Geriatr Soc 32:499-502, 1943.
104. Beisel WR: Magnitude of the host nutritional responses to infection. Am J Clin Nutr 30:1236-1247, 1977.
105. Schols A, Mostert R, Cobbin: Transcutaneous oxygen saturation and carbon dioxide tension during meals in patients with chronic obstructive pulmonary disease. Chest 100:1287-1292, 1991.
106. Donahue M, Rogers RM: Mechanisms of weight loss in chronic obstructive pulmonary disease. Monaldi Arch Chest Dis 48:522-529, 1993.
107. Roubenoff R, Grimm LW, Roubenoff RA: Albumin, body composition and dietary intake in chronic inflammation. In Rosenberg IM (ed): "Nutritional Assessment of Elderly Population," New York: Raven, pp 30-39, 1999.
108. Morley JE: The aging endocrine system. Postgraduate Med 73:107-120, 1983.
109. Darrieu G, Llau ME, Rascol O: Parkinson's disease and weight loss: a study with anthropometric and nutritional assessment. Clin Auton Res 2:153-157, 1991.
110. Mooradian AD, Morley JE, Korenman SG: Endocrinology in aging. Dis Mon 34:395-461, 1988.
111. Coulston AM, Mandelbaum D, Reaven GM: Dietary management of nursing home residents with non-insulin dependent diabetes mellitus. Am J Clin Nutr 51:67-71, 1990.
112. Rosenberg IH. Nutrition and aging. In Hazzard WR (ed): "Principles of geriatric medicine and gerontology," New York: McGraw-Hill, pp 49-59, 1990.
113. Morley JE, Thomas DR, Kamel HK: Nutritional deficiencies in long-term care: part 1 detection and diagnosis. The Annals of Long-Term Care 5:128, 1998.
114. Kamel HK, Iqbal MA: Body mass index and mortality among Hospitalized elderly patients. Archives Int Med 161(11):1459, 2001.
115. Landi F, Onder G, Gamassi G:Body mass index and mortality among hospitalized patients. Arch Intern Med 160:2641-2644, 2000.
116. Andres R, Elahi D, Tobin JD:Impact of age on weight goals. Ann Intern Med 103:1030, 1985.
117. Andres R: Mortality and obesity. In Andres R, Bierman EL, Hazzard WR (ed): "Principles of geriatric medicine," New York: McGraw-Hill, pp 99, 1985.
118. Dimaria RA, Amella E. Malnutrition in the elderly: In Maddox GL (ed): "The encyclopedia of aging," 2nd ed. New York: Springer, pp.599-601.
119. Pearson MB, Bassey EJ, Bendall MJ: The effects of age on muscle strength and anthropometric indicies within a group of eldlery men and women. Age Ageing 14:230-234, 1985.
120. Chumlea WC, Guo S: Equations for predicting stature in white and black elderly individuals. J Gerontol Med Sci 47:197-203, 1993.
121. Steen B, Bruce A, Saksoon B: Body composition in 70 year old males and females in Gothenburg, Sweden. A population study. Acta Medica Scand 61:87-112, 1997.
122. Rothschild MA, Oratz M, Schreiber SS: Serum albumin. Hepatology 8:385, 1988.
123. Doweiko JP, Nompleggi DJ: The role of albumin in human physiology and pathophysiology, part III: albumin and disease states. JPEN 15:476, 1991.
124. Ballmer-Weber BK, Dummer R, Kung E: Interlukin 2-induced increase of vascular permeability without decrease of the intravascular albumin pool. Br J Cancer 71:78,1995.
125. Ling PR, Schwartz JH, Bistrian BR: Mechanisms of host wasting induced by administration of cytokines in rats. Am J Physiol 272:333-339, 1997.
126. Dixon M, Paterson CR: Posture and the composition of plasma. Clin Chem 24:824-826, 1978.
127. Courtney ME, Greene HL, Folk CC: Rapidly declining serum albumin values in newly hospitalized patients: prevalence, severity, and contributory factors. JPEN 6:143, 1982.
128. Reilly JJ, Hull SF, Albert N, et al. Economic impact of malnutrition: A model system for hospitalized patients. JPEN 12:371-376, 1988.
129. Miller DK, Morley JE: Nutritional epidemiology. Ann Rev Gerontol Geriatr 15:20-53, 1995.
130. Rudman D, Mattson DE, Nagraj HS: Prognostic significance of serum cholesterol in nursing home men. JPEN 12:155, 1988.
131. Noel MA, Smith TK, Ettinger WH: Characteristics and outcome of hospitalized older patients who develop hypocholesterolemia. J Am Geriatr Soc 39:455, 1991.
132. Seltzer MH, Bastidas JA, Cooper DM: Instant nutritional assessment. JPEN 3:157, 1979.
133. Blackburn GL, Bistrian BR, Maini BS. Nutritional and metabolic assessment of the hospitalized patient. JPEN 1:11, 1977.
134. Kaiser FE, Morley JE: Idiopathic CD4+ lymphopenia in older persons. J Am Geriat Soc 42:1291-1294, 1994.
135. Seltzer MH, Bastidas JA, Cooper DM: Instant

nutritional assessment. JPEN 3:157, 1979.
136. Detsky AS, Baker JP, Mandelson RA: Evaluating the accuracy of nutritional assessment techniques applied to hospitalized patients: methodology and comparisons. JPEN 8:153, 1984.
137. Pettigrew RA, Charlesworth PM, Farmillo RW:Assessment of nutritional depletion and immune competence: a comparison of clinical examination and objective measurements. JPEN8:21, 1984.
138. White JV, Dwyer JT, Posner BM:Nutrition Screening Initiative: development and implementation of the public awareness checklist and screening tools. J Am Diet Assoc 92:163, 1992.
139. Sahyoun NR, Jaques RF, Dallal GE: Nutrition Screening Initiative checklist may be a better awareness/education tool than a screening one. J Am Diet Assoc 97:760, 1997.
140. Morley JE: Death by starvation. A modern American problem? J Am Geriatr Soc 37:184, 1989.
141. Blazer O, Williams CD: Epidemiology of dysphoria and depression in an elderly population. Am J Psychiatry 137:439, 1980.
142. Guigoz Y, Vellas B, Garry PJ: Mini Nutritional Assessment: a practical assessment tool for grading the nutritional status of elderly patients. Facts Res Gerontol 4(suppl 2):15, 1994.
143. Katz S, Ford AB, Moskowitz RW: Studies of illness in the aged. The index of ADL: a standardized measure of biological and psychosocial function. JAMA 185:914, 1963.
144. Morley JE, Silver AJ: Nutritional issues in nursing home care. Ann Intern Med 123: 850-859, 1995.
145. Morley JE: Why do physicians fail to recognize and treat malnutrition in older persons? J Am Geriatr Soc 39:1139-1140, 1991.
146. Delmi M, Rapin CH, Bengoa JM: Dietary supplementation in elderly patients with fractured neck of the femur. Lancet 335:1013-1016, 1990.
147. Breslow RA, Hallfrisch J, Guy DG: The importance of dietary protein in healing pressure ulcer. J Am Geroatroc Soc 41:357-362, 1993.
148. Woo J, Ho SC, Mak YT. Nutritional status of elderly patients during recovery from chest infection and the role of nutritional supplementation assessed by a prospective randomized single blind trial. Age Ageing 23:40-48, 1994.
149. Kamel HK, Thomas DR, Morley JE: Nutritional deficiencies in long-term care: Part II - Management of protein energy malnutrition and dehydration. Ann of Long-Term care 6:250, 1998.
150. Mitchell SI, Keely DL, Lipitz LA: The risk factors and impact on survival of feeding tube placement in nursing home residents with severe cognitive impairmaent. Arch Intern Med 157:327-332, 1997.
151. Weaver JP, Odell P, Nelson C:Evaluation of the benefits of gastric tube feeding in an elderly population. Arch Fam Med 2:953-956, 1993.
152. Roenn JH: Randomized trials of megestrol acetate for AIDS-associated anorexia and cachexia. Oncology 51:19-24, 1994.
153. Simons JP, Aaronson NK, Vansteenkiste JF: Effects of medroxyprogesterone acetate on appetite, weight and quality of life in advanced-state non-hormone-sensitive cancer: A placebo-controlled multicenter study. J Clin Oncol 14:1077-1084, 1996.
154. Fietau R, Riepl M, Kettner H: Supportive use of megestrol acetate in patients with head and neck cancer during radio (chemo) therapy. Eur J Cancer 33:75-79, 1997.
155. Castle S, Nguen C, Joaquine A: Megestrol acetate suspension therapy in the treatment of geriatric anorexia/cachexia in nursing home patients. J Am Geriatric Soc 43:83583-6, 1995.
156. Mainguet P: Effect of cyproheptadine on anorexia and loss of weight in adults. Practitioner 208:797-800, 1972.
157. Kaiser FE, Silver AJ, Morley JE: The effect of recombinant human growth hormone on malnourished older individuals. J Am Geriatr Soc 39:235-40, 1991.
158. Holloway L, Butterfield G, Hentz RL: Effects of recombinant human growth hormone on metabolic indices, body composition and bone turnover in healthy women. J Clin Endocrinol Metabol 79:470-479, 1994.
159. Cohn L, Feller AG, Draper MW: Carpal tunnel syndrome and gynecomastia during growth hormone treatment of elderly men with low circulating IGF-1 concentrations. Clin Endocrinol 39:417-425, 1993.
160. Takala J, Ruokonen E, Webster NR: Increased mortality associated with growth hormone treatment in critically ill adults. NEJM 341:785-792, 1999.
161. Berger JR, Pall L, Hall CD: Oxandrolone in AIDS-wasting myopathy. AIDS:1657-1662, 1996.
162. Mendenhall CL, Mortiz TE, Rosell GA: A study or oral nutritional support with oxandrolone in malnourished patients with alcoholic hepatitis: Results of a Department of Veterans Affairs cooperative study. Hematology 17:564-576, 1993.
163. Johansen KL, Mulligan K, Schambelan M: Anabolic effects of nandrolone decanoate in patients receiving dialysis. JAMA 281:1275-1281, 1999.
164. Sih R, Morley JE, Kaiser FE, Perry HM III, Patrick P, Ross C: Testosterone replacement in older hypogonadal men: A 12-month randomized controlled trial. J Clin Endoc Metabol 82:1661-1667, 1997.
165. Nyder PJ, Peachey H, Hannoush P: Effect of testosterone treatment on body composition and muscle strength in men over 65 years of age. J Clin End Metabol 84:2647-2653, 1999.
166. Nelson KA, Walsch TD: Metoclopramide in anorexia caused by cancer-associated dyspepsia syndrome. J Palliative Care 9:14-18, 1993.
167. Kardinal CG, Loprinzi CL, Shaid DJ: Controlled trial of cyproheptadine in cancer patients with anorexia and/or cachexia. Cancer 65:2657-2662, 1990.
168. Beal JE, Olson R, Laubenstein L: Dronabinol as a treatment for anorexia associated with weight loss in patients with AIDS. J Pain Symptom Manage 10:89-97, 1995.
169. Volicer L, Stelly M, Morris J: Effects of dronabinol on anorexia and disturbed behavior in patients with Alzheimer's disease. Int J Geriatr Psychiatry 12:913-919, 1997.

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