We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 6

Malnutrition in Paraplegia

Yannis Dionyssiotis

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/58382

1. Introduction

Despite the advances in medical and nutritional science surveys show that 40-50% of patients admitted to hospitals are at risk of nutritional deficiency; one in three hospitalized patients are malnourished upon admission and up to 12% are severely malnourished [1, 2]. Malnutrition is a state in which a deficiency, excess or imbalance of energy, protein and other nutrients causes adverse effects on body form, function and clinical outcome [3, 4]. Studies report a variable prevalence of obesity from 40 to 66% in persons with SCI completing the spectrum of newly introduced concept in nutritional deficiency [5, 6, 7].

After the lesion paralysis and loss of function that usually occur and well documented hypercatabolic responses may lead to deleterious effects such as loss of lean body mass, obesity, increased susceptibility to infections, and reduced wound healing [5, 8, 9]. Unwanted weight gain should be prevented because induces the risk for diseases such as diabetes, coronary heart disease and dyslipidaimias in this population [8]. Mortality and pathogenesis of critically ill patients are affected by nutritional status. Body fat has been identified as a significant predictor of mortality. Moreover, some disorders such as carbohydrate intolerance, insulin resistance, lipid abnormalities, and heart disease occur prematurely and at a higher prevalence in disabled populations may be related to immobilization and skeletal muscle denervation [10]. According to the above the term malnutrition should include not only undernourishment but also obesity [11]. Therefore, the objective should be either the maintenance of optimal nutritional status of the patient, either to supplement the deficiencies in nutrients. Nutrition support therapy should be tailored to each patient. An optimal nutritional assessment and management of the disabled subject can minimize the complications associated with acute traumatic injury and long-term rehabilitation [12].

This chapter reviews methods of nutritional assessment and describes the physiopathological mechanisms of malnutrition, reviews specific nutritional studies, and the supplemental support which can be used in paraplegic subjects.

2. Nutritional assessment

For an initial assessment of nutritional status serial measurements to assess trends over time and then monitor the response to a dietary intervention may be useful. The proposed assessments should be interpreted collectively including the examination of possible factors that contribute to the nutritional status, such as age, sex, over-or under-hydration, interactions between drugs-food, metabolic stress, infection, and the existence of other diseases[13].

2.1. Diet history

During hospitalization adequate intake of nutrients is intercepted by many factors, and may be caused by anorexia, early satiety, immobility, depression. Moreover, gastrointestinal function is compromised: gastric dilatation and paralytic ileus occurs often, although the intestinal activity usually returns within the first week after injury.

2.2. Nutritional requirements

The provision of energy and nutritional requirements is a very important factor for patient management. Malnutrition, in this case undernourishment or over nutrition-obesity, can lead to muscle loss, atrophy of the lining of the intestine, immunochemical reduction, poor wound healing and fluid overload, hyperglycemia, high levels of urea nitrogen in blood, high triglycerides, elevated liver enzymes, respiratory exhaustion due to increased production of CO_2, and difficulty weaning from the oxygen, respectively. The assessment of nutritional requirements includes not only calculations but also the opinion of an expert clinician in order to assess the clinical and morphometric data before applying the equations that provide the energy and protein requirements [14].

There have been several methods for predicting energy expenditure (EE); the components and the methods for its determination and estimation, summarizing their main advantages and limitations have been recently reviewed [15]. However, because of various confusing factors such as infections and sepsis, hyper nutrition supportive nutritional diets, clinical procedures, postoperative medications, and changes in body weight such as sarcopenia, obesity, amputations and significant weight loss, the prediction equations can be complex and invalid [16].

A group of equations among these are Mifflin–St Jeor equation [17], the Harris-Benedict equation [18], the American College of Chest Physicians (ACCP) recommendation based on kcal/kg body weight [19], the Faisy equation [20], the Ireton-Jones equations [21, 22] and the Penn State equations [23, 24]. Because the Mifflin equation was designed for healthy people is not analyzed here.

The Harris-Benedict is calculated by sex with the following formula:

Men: Resting Metabolic Rate (RMR) (kcal/d)=67+Body Weight x 13.75+Height x 5 – Age x 6.8 [18, 24].

Women: RMR (kcal/d)=655+Body Weight x 9.6+Height x 1.85 – Age x 4.7 [25].

Ideal body weight is calculated by the Hamwi rule of thumb while metabolically active weight (MAW) is calculated as 25% of excess weight (actual weight − ideal weight) added to the ideal body weight [26]. The Ireton-Jones equations include one specifically for obese patients and one for general critical care populations:

Obesity: RMR (kcal/d)=Wt x 9+Gender x 606 – Age x 12+1844 [27].

Nonobese: RMR (kcal/d)=Wt x 5 – Age x 10+Gender x 281+Trauma x 292+1925 (for gender: male=1, female=0) [28].

To determine accurately the early energy expenditure after spinal cord injury, studies compared measurements of real resting energy expenditure (REE) with the Harris-Benedict equation (basic energy expenditure, BEE) [18]. During the first two weeks after the injury, the exact measurements of REE are similar to the estimated calorie needs, when used with BEE stressor/injury factor of 1.6. To avoid overestimation of calorie needs, the deletion of factor activity of 1.2 (rest in bed) is proposed. Kearns et al. reported that in 10 patients, the mean REE after acute injury was only 67% of BEE predicted by Harris-Benedict formula. They hypothesized that non-specific changes in neurogenic stimuli and reduced oxygen consumption by relaxing muscles contributed to their findings. Also, an interesting feature observed is that the REE was raised by 5% with the return of muscle tone [29]. Jeejeboy and Cerra proposed an alternative approach that uses body weight (kg) alone as a determining factor, and omits the variables of age, sex and height as used in HB equation. This type of assessment has proven to be accurate and efficient over time [30, 31]. Ireton-Jones and Owen et al. have developed specific formulas for the obese patient, which is common in SCI subjects. The predefined types may overestimate their needs due to increased fat mass in this population [21, 22, 32].

2.3. Assessment of subjects in the clinical setting

Patients admitted in the hospital should be examined for actual or potential occurrence of malnutrition because of an unintentional weight loss or gain. In the clinic this examination includes measurements of body weight depicting a loss of more than 10% of normal body weight within 6 months or loss of more than 5% of usual body weight within 1 month or 20% more or less than ideal body weight (IBW), calculation of body mass index (BMI) <18, depletion of visceral protein (serum albumin <3.5 g/dl, serum transferrin <200 mg/dl, serum cholesterol <160 mg/dl, serum pre – albumin <15 mg/ml, creatinine height index (CHI) <75% (measured by 24-hour creatinine excretion, which is typically associated with muscle mass of the patient as an indicator of malnutrition, especially in young men), and the presence of diet modifications (patient receives total parenteral nutrition (TPN) or enteral nutrition (EN), inadequate food intake due instructions for stopping any food by mouth (NPO), liquid diet, disorders of absorption, reduced swallowing capacity, increased metabolic needs, gastrointestinal disturbances (nausea, vomiting, diarrhea, constipa-

tion). Unintentional weight gain is an increase in body weight that occurs when a person takes in more calories than the body needs or uses [33, 34].

For able bodied persons the World Health Organization (WHO) advocates use of BMI as a population-level indicator of obesity which is not a direct measure of body fat, but a more accurate indicator of overweight and obesity than relying on weight alone. BMI is calculated using the equation weight (Kg)/height (m^2), which is a very practical and useful measure that allows the easy determination of categories of weight status. In able-bodied subjects overweight is defined as a BMI of 25–29.9 kg/m^2 and obesity as a BMI of ≥30.0 kg/m^2 and extreme obesity≥40 kg/m^2 (Table 1) [35, 36].

Classification	BMI (kg/m^2)	Obesity Category
Underweight	<18.5	-
Normal	18.5-24.9	-
Overweight	25.0-29.9	-
Obesity	30, 0-34, 9	I
Moderate obesity	35.0-39.9	II
Extreme obesity	> 40.0	III

Table 1. Classifications based on the weight for BMI and obesity category (published with permission from Dionyssiotis Y. [36])

In a chronic SCI population with paraplegia values of body mass index (BMI, kg/m^2) were not significant vs. controls, which is a finding in line with the literature [10, 37]. Nevertheless, Gupta et al demonstrated the usefulness of BMI as an indicator of obesity [38]. Whether the criteria of BMI may assess obesity in people with spinal cord injury the latest studies show the opposite [39]. The applicability of conventional BMI cut off values is into question [40, 41]. Another critical issue is that the relationship between BMI and disease is typically U-or J-shaped with those in the middle categories of BMI having the lowest risk compared to the lowest extreme and upper levels of BMI. It is under question if the cut-points for underweight, normal, overweight, and obese used in able-bodied populations can be applied to disabled subjects [42]. Not many studies investigated BMI in patients with MS. Nevertheless, BMI was found statistically less compared to age comparable controls [43].

Anthropometric standards such as the ideal body weight (IBW), the triceps skin fold thickness and the middle arm circumference which are common tools for assessment of nutrition may not be valid for disabled subjects due to water changes, atrophy of muscles because of immobility, increased body fat, and the inevitable weight loss beyond the normal. Patients' early weight loss is mainly due to loss of muscle rather than fat which bias the results of validity. In chronic paraplegics, the ideal weight has been estimated to be 4.5 to 6.5 kg below their respective controls finding which is in line with our recently published results [37]. Indeed, height and weight measurements are the key elements in nutritional assessment. The IBW is determined by the height. No matter which method of

calculation is used, the IBW should be adjusted for body type (frame sizes: small-IBW 10% reduction, middle size-no changes required, large size-IBW increased by 10%) and spinal cord injury (paraplegia-decrease IBW by 10-15%, tetraplegia-by 15-20%, respectively). The weight in admission is probably the most reliable measure of weight in determining the actual body weight (ABW) of the patient because is unreliable postoperatively or during an acute illness due to administration of fluids or due to edematous condition. As a chronic index, one can assume that the weight gain or loss is associated with an increase or decrease in lean body mass. To determine the weight which should be used on the nutritional calculations, first % IBW should be calculated through the equation: % IBW=actual body weight (actual body weight, ABW / ideal body weight (IBW) x 100. If the actual body weight (ABW) is less than IBW, use ABW, to define the nutritional requirements, if is greater than IBW, but less than 120%, it is necessary to determine nutritional needs using the adjusted relationship of body weight in the calculation needs: IBW+(ABW-IBW x 0.25). The nutritional status of patients can be categorized according to their ABW as a percentage of IBW as follows: over 200% of IBW (pathologic obesity), over 150% of IBW (obese), more than 120% IBW (overweight), 100% of IBW+/-10% (normal), 80-90% of IBW (mildly malnourished), 70-80% of IBW (moderately malnourished), less than 70% of IBW (severe nutritional deficiency–malnutrition), respectively [1].

3. Biochemical measurements

As with the visceral and somatic visceral proteins, non-dietary factors (i.e. blood loss, chronic infections, and fluid overload) should be considered as potential reasons for the reduction of serum concentrations [1]. Proteins are essential for tissue growth, maintenance and rebuilding their synthesis of hormones, enzymes, antibodies and cells transport molecules. In cases of protein excess protein is either metabolized for energy or stored as fat. The recommendations for protein intake in patients with spinal cord injury vary with respect to acute or chronic phase of the lesion and the presence of decubitus ulcers or not. Specific proteins (albumin, transferrin, and pre-albumin) are biochemical indicators used for assessing nutritional status [44].

The level of serum albumin is not a definitive measure of visceral protein status, but reflects the complex relationship between synthesis, degradation, and distribution. Given the long half-life of 21 days, serum albumin cannot be effectively used for monitoring the acute response to nutritional therapy. Therefore, albumin levels should be included in the initial profile for food control and monitoring purposes during hospitalization for measuring trends of visceral protein or as an indicator of chronic nutritional status. Beside this limitation there are many non-dietary factors that reduce the levels of albumin, regardless of nutritional status (inadequate composition: acute stress, hypoxia, impaired digestion, as in malabsorption, modified status as edematous fluid status and fluid overload, chronic loss of protein) (Table 2) [36].

Albumin (g/dl)	3.5-5	3-3.5	<3.5	<3.0	<2.5
nutritional status	normal	point that dietary intervention should be revised or adjusted	associated with poor outcome of surgery, rising costs of hospitalization and prolonged stay in ICU	severe malnutrition	increased morbidity and mortality

Table 2. Basic levels of albumin and nutritional status distribution (published with permission from Dionyssiotis Y. [36])

Due to the lower half-life (8-9 days) and the smaller size as a constituent body, transferrin is a better indicator of nutritional status of visceral protein from albumin. Normal levels of transferrin are between 200-400 mg/dl, and 150 mg/dl are considered nutritionally decision point or a point where nutritional support should be revised or adjusted. The transferrin levels are reduced in impaired synthesis as chronic infections, increased secretion, fluid overload, increased iron stores and increased in reduced iron stores as iron deficiency anemia and chronic blood loss, increased protein synthesis on estrogen therapy and oral contraception and dehydration. The serum concentration of transferrin is approximately 0.8 times the total iron binding capacity (TIBC). If direct measurement of transferrin is not possible due to the high cost and limited availability of equipment required, the level of transferrin can be easily calculated from TIBC, using the following formula: TIBC x 0.8-43=transferrin [45].

The third protein biochemical indicator is pre-albumin, which has very short half-life (2 days), making it an excellent nutritional index and due to this reason is increasingly used as an indicator of response to nutritional therapy. Reference values for pre-albumin are 16-35 mg/dl. A value of dietary intervention is 11 mg/dl because a value below this level means malnutrition. The failure of patients to increase pre-albumin above 11 mg/dl with dietary therapy is an indication that nutritional needs are not met. Concentrations should increase about 1 mg/dl per day or twice a week when the treatment is the appropriate. Non-dietary factors that reduce pre-albumin include stress, inflammation [46, 47, 48].

Physical measurements include protein nitrogen balance studies and measurement of creatinine / height index (CHI). Nitrogen balance studies measure the net change in total body protein. An assessment of nitrogen balance can be achieved by measurement of urinary urea (UUN) and compare it with the intake of nitrogen at the same time. The nitrogen balance is calculated as follows: N_2=balance intake N_2-N_2 elimination or=[protein (gr)]-(24 hour UUN+3) [6.25 gr nitrogen]. An "agent" of 3 is added to the equation for nitrogen losses in feces, skin, and the drainage of body fluids. When calculating the nitrogen balance a value of 0 meaning nitrogen balance (healthy adults), nitrogen balance> 0 (protein anabolism exceeds catabolism, usually consistent with pregnancy, growth, and recovery from disease or may indicate nutrient saturation, the goal in nutrition replenishment is a positive nitrogen balance of 4-6 grams per day and nitrogen balance <0 (the protein catabolism exceeds protein anabolism, occurs in situations of famine, increased catabolism due to trauma or surgery, and inadequate nutrition therapy), respectively. CHI measures the 24-hour creatinine excretion in urine and compares with an optimum value based on the ideal weight for height [49].

http://dx.doi.org/10.5772/58382

4. Malnutrition screening tools

Screening is important for the early detection of patients who are undernourished or at risk of developing malnutrition. Since January 2010, the Dutch Health Care Inspectorate (HCI) has defined under nutrition as a main care problem in rehabilitation centres, by establishing it as a Performance Indicator for Risk Steering Supervision. Dutch rehabilitation centres are now obligated to screen all rehabilitants for under nutrition on admission The Short Nutritional Assessment Questionnaire (SNAQ) is the recommended screening tool in this benchmark (Figure 1) [50]. However, various screening tools have been developed to detect a patient's nutritional status in many healthcare settings, but not in the rehabilitation setting. In the Netherlands, the SNAQ [51] and the Malnutrition Universal Screening Tool (MUST) are used for the hospital situation [52, 53]. The HCI advises the use of the SNAQ for under nutrition screening in rehabilitation centres [51]. Our results suggest the use of the SNAQ65+as a screening tool. This tool showed the best diagnostic accuracy of the quick and easy screening tools investigated (sensitivity 96%, specificity 77%) [52, 54].

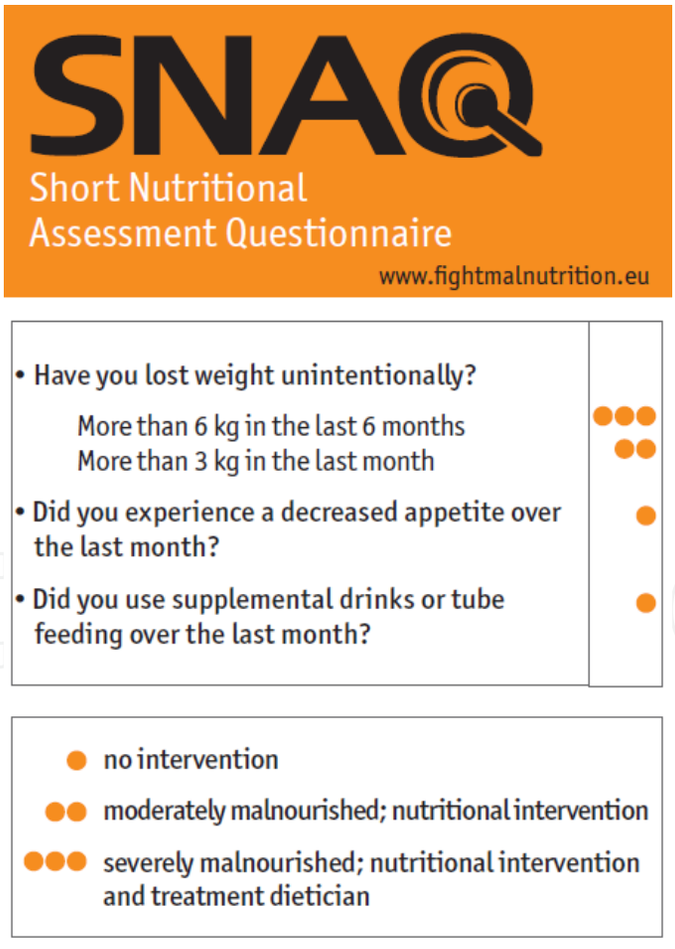

Figure 1. The Short Nutritional Assessment Questionnaire (SNAQ). Published with permission from: http://www.fight-malnutrition.eu/fight-malnutrition/screening-tools/

5. Monitoring

Healthcare professionals with relevant skills and training should review the indications, route, risks, benefits and goals of nutrition support at regular intervals. The time between reviews depends on the patient, care setting and duration of nutrition support. Intervals may increase as the patient is stabilised on nutrition support [55]. (NICE Clinical Guideline 32 Feb.2006 Nutrition Support in Adults: Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition, the whole guideline can be downloaded from: http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf)

6. Physiopathological mechanisms of malnutrition

6.1. Malnutrition in the acute phase of paraplegia

Pathophysiological mechanisms of malnutrition in paraplegia are multifactorial. There is a dramatic increase in energy expenditure, endogenous protein catabolism and nitrogen excretion after lesion-injury. Extensive multiple organ trauma, soft tissue injuries and fractures, may further increase hyper catabolic reactions. Also, the body temperature and energy expenditure increases due to pulmonary infections or urinary tract infractions, and pancreatitis. The metabolic rate does not seem to be affected by the small reductions in thyroxin levels in plasma observed after the injury [56, 57].

Metabolic changes are also present with the elevated catabolic hormonal and cytokine responses including increased blood levels of counter regulatory hormones (e.g., cortisol, catecholamines, and glucagon), increased blood and tissue levels of proinflammatory cytokines (i.e., interleukin-1, interleukin-6, interleukin-8, and tumor necrosis factor α), and peripheral-tissue resistance to endogenous anabolic hormones (i.e., insulin and insulin-like growth factor 1) to be primarily responsible for the initial changes in metabolism [58-61].

Glucose intolerance, which cannot be readily apparent during the acute phase, but may be caused by complications and physiological processes of acute care such as the initial hyper metabolic-catabolic stress response, administration of steroids, the parenteral / enteral nutrition, and atrophy as a consequence of aponeurosis which results in gluconeogenesis [62]. Glucose and lipid metabolism disrupt in acute post-traumatic phase. Increased hepatic gluconeogenesis and regional response to insulin result in hyperglycemia. The metabolism of glucose in combination with acute nerve injury has been studied extensively, especially as related to ischemia. These studies suggest that hyperglycemia which follows immediately after head injury or spinal cord may worsen the outcome. High serum glucose levels increase the availability of substrate for anaerobic glycolysis, and thus the production of lactic acid, which may have the reverse effect on neurological recovery from injury. The prevention of hyperglycemia, particularly during the first 2 to 8 hours after injury, seems to be very critical for optimal recovery. After 2 to 8 hours after injury, elevated glucose levels may be beneficial, allowing the beginning of intestinal or parenteral feedings in a short time after the injury. It is also likely the serum triglyceride levels to be found elevated due to the accelerated lipogenesis,

decreased lipoprotein lipase activity, and impaired clearance of triglycerides [63]. Glucose is the preferred energy molecule for the central nervous system, red blood cells, the cellular tissue, etc. A minimum quantity of 100-150 gr glucose per day is required for these functions and prevents the consumption of endogenous protein. The normal rate at which the body metabolizes carbohydrates or glucose is approximately 2-4 mg/ Kg/min. In times of severe stress, glucose metabolism may be increased to 3-5 mg/Kg/min. In most patients, administration of more than 400-500 gr glucose per day, exceeding the body's ability to metabolize and stored as energy. Sources of glucose include not only the liquid diet and peritoneal fluid filtration. Excess glucose is converted into fat (lipogenesis) and leads to an increased ratio of VCO_2/VO_2 (or RQ) [64].

The provision of lipids as a source of increased calories can facilitate protein maintenance, reduce the risk of excessive carbohydrates and reduce the total volume of liquid. Lipids are required to account for 30% of total calories supplied. In the acute phase after injury, large amounts of fat, especially as linoleic or omega-6 fatty acids have an immunosuppressive effect by triggering the release of arachidonic acid. This leads to synthesis of prostaglandins and then compresses the delayed hypersensitivity cell-regulated, proliferation of lymphocytes. In the presence of sepsis, high levels of serum triglycerides (250 gr/ml) indicate limited tolerance and decreased need for intravenous fluid delivery. A minimum of 4% of total energy requirements is necessary for the essential fatty acids to avoid deficiencies [65]. Unfortunately, although the hormonal cataract through increases in glycogenolysis and gluconeogenesis, is enhancing lipolysis, which provides endogenous glucose, amino acids, and free fatty acids that are required for cellular and organ function and wound healing and certain plasma levels of substrates are increased (i.e., glutamine) they could be insufficient to meet metabolic needs due to limited availability for use by peripheral tissues (because of factors such as insulin resistance and inhibition of lipoprotein lipase) [60, 61].

Acute post-traumatic nitrogen requirements are much higher than in normal state. Another serious metabolic issue is negative nitrogen balance, due to excessive secretion of nitrogen because of protein use by the body to meet energy needs in the first week, with a peak at 3 weeks and can last for a period of 7 weeks. This imbalance will respond only slightly increased protein intake and may be non-modifiable as a process during the acute phase. The more severe the injury the greater the amount of nitrogen excreted. The accelerated catabolism of muscle mass results in a supply of amino acids for the acute-phase of protein synthesis, gluconeogenesis, and the healing of wounds. Moreover, administration of glucocorticosteroids after injury may increase the catabolism of protein. The losses of nitrogen in the urine, mainly due to muscle atrophy because of paralysis, are increasing with the severity of the injury. On the other side, Cooper and Hoen stated that the secretion of more than 25 gr/day of nitrogen in the urine during the first two weeks after the injury is insufficient prognostic indicator for functional recovery of paralyzed muscles. The nitrogen losses after an injury are always present and last at least 7 weeks. In cases of acute injury, despite the provision of sufficient quantities of calories and protein usually occurs a negative nitrogen balance (NB), which peaks during the third week after injury. The same phenomenon has been observed in cases of severe poisoning with botulinum toxin (botulism) which resulted in paralysis of muscles. Negative

nitrogen balance following injury, has been associated with further findings. During the first weeks after injury, many patients experience a transient positive nitrogen balance, possibly due to initial delays in the loss of nitrogen [66]. Four conscientious objectors were immobilized on pelvic corset and leg casts for 6 to 7 weeks in a metabolic chamber. All 4 subjects showed an increase in nitrogen excretion and negative nitrogen balance. However, it took 4 to 5 days to develop. In conclusion, acute immobilization of paralyzed patients contributes to increased excretion of nitrogen which starts about a week after the injury [67].

Deficiencies in zinc and vitamin C have been associated with poor wound healing. The provision of these micronutrients supplementation in patients with these deficits enhances the healing. Adequate quantities of salts and vitamins are usually provided in a balanced diet. The supplemental micro-nutrient dietary substances are necessary if we suspect shortcomings intake or increased requirements because of circumstances specific diseases. Zinc is often prescribed to improve stress ulcers, is known to be involved in structural integrity of collagen. However, zinc levels in serum is similar in patients taking supplements that contain sulfur (220 mg daily) and do not affect the healing process of ulcers sprawling over a period of 2-3 months. Opposite physiological effects, such as metabolism of copper, copper deficiency and anemia may be caused by long-term supplementation of large amounts of zinc [68]. The role of vitamin C in collagen synthesis is crucial. Although the supplementation with vitamin C did not accelerate the healing of decubitus ulcers in patients, dietary intake of vitamin C has not been associated with the development of decubitus ulcers. Moreover, given that the subclinical deficiencies are difficult to show up, the minimum recommended dietary intake is proposed to 60mg [69]. Excessive excretion of potassium and abnormal hyponatremia; hypercalcemia, due to immobilization, particularly in young men and hypercalciuria exceed the normal range in 4 weeks, with higher values at 16 weeks, which can persist for a long time. Hypercalcemia occurs with anorexia, abdominal cramps, nausea, vomiting, constipation, polydipsia, polyuria, dehydration and did not respond to diets which restrict the intake of calcium and need to be treated with medication, hydration, and mobilization [70].

Finally the effect of drugs such as analgesics and barbiturates is crucial. Drugs that are frequently administered to acute paraplegic patients may themselves increase skeletal-muscle breakdown (corticosteroids), decrease splanchnic blood flow (pressor agents), or increase urinary loss of electrolytes, minerals, and water-soluble vitamins (diuretics). Infection, operative trauma, and other stresses may increase energy expenditure and protein and micronutrient needs [71-74]. The average daily dietary needs are modified because of the altered physiology of each body system and psychological integrity of a patient susceptible to an injury, potentially at any age, which cannot exclude the possibility of a pre-existing disease causing nutritional problems [75].

Moreover, the frequent coexistence of injuries from other systems, such as brain injury, maxillofacial injuries, fractures, etc., disturbs the normal physiology further. Studies in malnourished patients stated that malnutrition before a spine stabilization surgery is leading to postoperative complications, hyperthermia, which increases the caloric needs of the patient, and denervation, leading to atrophy and paralysis, which supply amino acids for gluconeogenesis, which, in turn, supplies glucose to meet caloric needs [1].

Serum hemoglobin and hematocrit may reflect a general state of malnutrition. Anemia, defined by low hemoglobin levels (<14 mg/dl) and hematocrit (<36%) reduces the oxygen in the blood and impedes the wound healing. Anemia may be due to a preexisting condition or as the result of unbalanced production and distribution of blood cells as a result of reaction to stress, gastrointestinal bleeding or obvious bleeding due to other trauma [76]. Low levels of total serum protein (<6.4 mg/dl) and protein (<3.5 mg/dl), accelerate the development of edema, which causes a decrease in skin elasticity and prevent the transfer of oxygen and nutrients from the blood to the skin. Also, the swelling may increase local tissue pressure, causing loss of regional blood flow and tissue damage. The loss of protein and protein secretion in pressure ulcers increases the deficiencies in proteins. The paralytic ileus occurs as a result of disturbance of the autonomic and simultaneous or ischemia as a complication of hypokalemia, abdominal trauma or sepsis, generally persists for 72 hours-1 week and may restrict the movement of the diaphragm [77]. Parenteral nutrition is indicated if paralytic ileus persists for more than 3-5 days. Ulcers and bleeding, which occur as a result of paralytic vasodilatation with ischemia, steroids, nasogastric tube irritation, and other causes should be treated with oral or enteral feeding as soon as possible but may require parenteral nutrition [78].

6.2. Malnutrition in the chronic phase of paraplegia and during aging

During aging with paraplegia other complications are added in the physiopathological context of "malnourished paraplegics".

A neglected factor is muscle tonus: hypotonia (low muscle tone, floppiness) results in a lower resistance to muscle movement. The lower the resistance, the fewer calories burned during movement. Furthermore, hypertonia (high muscle tone, spasticity) is limiting muscle movement and reduces caloric needs. Lack of movement results in muscle atrophy and a lower lean body mass, which in turn reduces the number of calories burned even at rest [79].

In spinal cord injured subjects is mainly central or abdominal obesity leading to metabolic, cardiovascular issues etc. There is conflicting evidence about the contribution of visceral and subcutaneous adipose tissue to different metabolic disorders after SCI. Moreover subjects with longstanding disabilities (i.e. spinal cord injury) are at increased risk for cardiovascular disease and cardiopulmonary disease because of extensive fat intake and limiting activities. In generally, subjects with disabilities are prone to developing vitamin D deficiency. Earlier work by Bauman et al suggested that approximately 32% of veterans with spinal cord injury (SCI) were absolutely deficient in vitamin D (25 hydroxyvitamin D [25(OH)D]). Most subjects have a high incidence of vitamin D deficiency as defined by levels of 25(OH)D<20 ng/mL. The reasons might be due to a combination of low dietary vitamin D intake and avoiding sun exposure because of depression or sensitivity in drugs i.e. dantrolene [80]. The low intake of vitamin D, which is supplied by food either in vitamin D2 (ergocalciferol, activated ergosterol), found in yeast, or vitamin D3 (cholecalciferol), found in fish, can be bypassed through supplements [81].

Moreover, reduced mobility and immobilization for long period cause pressure ulcers of the skin and the wound but can be prevented by adequate intake of quantity of protein, vitamin E, zinc, and fluids to maintain skin integrity [82].

Pneumonia and paralysis of respiratory muscles through malnutrition may further weaken the respiratory muscles. On the other site excessive feeding may lead to increased oxidation of glucose and production of carbon dioxide to be eliminated and further stress on the respiratory system. The fluid overload or aggressive implementation of parenteral support can lead to pulmonary edema. The reduced hydration can lead to reduced drainage of secretions, atelectasis, and pneumonia. Abdominal distension due to unabsorbed food by mouth or enteral feeding or swallowing air during feeding can lead to compromise the functioning of the diaphragm and predisposes to hypoventilation or aspiration [83]. Neurogenic bowel requires the right amount of food, fiber and fluids in order to be successful retraining of the bowel, and prevent constipation, diarrhea, incontinence, and autonomic dysreflexia as a result of fecal impaction. Bowel function may be compromised by hyperosmolar feeding through a tube, lactose intolerance or pseudomembranous colitis, prolonged treatment with antibiotics, which can cause diarrhea and require parenteral nutritional support. For neurogenic bladder vitamin C and other supplements are necessary for the acidification of urine and prevention of infection of the urinary tract.

7. The nutritional support

The provision of a nutritional supplement is definitely not a frontline management technique for poor oral intake. Supplements when administered correctly to patients can easily optimize nutrition and should be an adjunctive to nutrition.

Per os feeding is recommended for patients who are weaned from tracheal tubes, which are awakened, may follow commands and have good swallowing and intestinal function. Patients with central nervous system (CNS) acute diseases are frequently in coma or have their swallowing reflexes impaired and need parenteral nutrition or enteral tube feeding [84, 85]. Enteral nutrition (EN) is recommended for patients who are tubed, not able to swallow or to receive adequate diet orally but have good bowel function.

Early nutrition support through the enteral route has been shown to blunt catabolism, reduce complications and reduce length of stay in a number of patient populations, including both surgical and non-surgical neuro patients [86, 87]. However, nutrition support must be initiated within the 48-to 72-hour period immediately following injury or surgical insult to achieve these benefits. Clinicians are often hesitant to feed critically ill neuro patients too soon. However, studies indicate patients with severe neurological deficits and clinically silent abdomens can tolerate low-rate jejunal feedings within 36 hours of injury with a gradual increase in feeding rate to meet initial caloric goals within two to four days [88, 89]. If jejunal feedings are initiated prior to induction of pentobarbital infusion, even patients in pentobarbital coma can be fed enterally [90].

Nasogastric or nasoenteric feeding tubes should not be used for periods longer than 4 weeks because of discomfort and the risk of nasal injury and sinusitis. Placement of a percutaneous endoscopic gastrostomy (PEG) tube should be considered for patients who continue to require enteral feeding beyond 4 weeks[90]. PeG is also indicated as firstline intervention in conditions

http://dx.doi.org/10.5772/58382

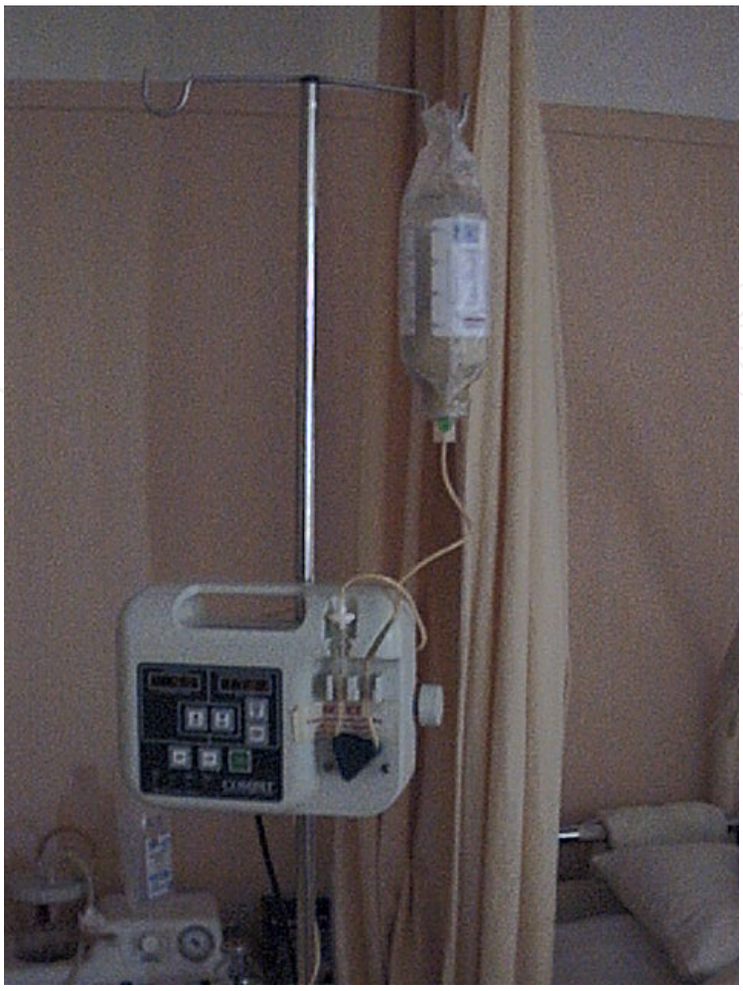

Figure 2. The enteral feeding pump type COMPAT (unpublished photo courtesy of Dionyssiotis Y). The system is a relatively simple, lightweight, easy to use for managing all types of enteral feeding. Have an audible and visual alarm that alerts you when each of the following conditions: empty container feeding, low battery, change the dose, or the existence of j free flow out of the system (waste). The memory of the pump retains infusion rate, volume delivered, dose limit even after turning it off. It is designed to provide precision dosing. Start enteral feeding schedule and progress.

where enteral feeding is expected to be required for longer than 2–4 weeks, for example in patients with acute stroke [91]. Although complications of PEG tube feeding are rare in stable patients, they become increasingly common in critically ill and debilitated patients. One of most feared complication of enteral feeding: aspiration hypoxia/ pneumonia. Clinically, gastric residual volume (GRV) measurement was frequently used as marker to predict aspiration & pneumonia. Elevated GRV: associated with comorbidities such as vasopressor use, sedation sepsis, vomiting. GRV: no significance between GRV> 200ml and GRV> 400ml, low sensitivity as a marker of aspiration [92].

With increasing frequency, nasogastric feeding tubes are replaced by PEG to provide semi-long-term enteral nutrition because of various advantages of a PEG in daily use [93-95]. In contrast to a nasogastric feeding tube, PEG does not interfere with the swallowing mechanism, which reduces the possibility of choking, especially when oral feeding is initiated during

neurological recovery. The cosmetic advantage of a PEG, which can be worn invisibly underneath the patients' clothes, may play a psychological role during recovery. PEG placement is associated with a mortality rate of 1-3 per cent, major complication rates of 3-9 per cent and minor complication rates of 5-45 per cent. The risk of aspiration, frequently associated with nasogastric feeding tubes, has not been eliminated with PEG placement [96-100].

Another interesting issue is early compared with late introduction of the feed. With data limited to ICU patients there was no overall difference in mortality rates with either EN or PN with no apparent difference in mortality rates across groups receiving EN or PN (RR 1.08; 95% CI 0.70 to 1.65). As suggested compared with PN, EN was associated with a significant reduction in infectious complications (RR 0.61; 95% CI 0.44 to 0.84; p=0.003). The early compared with late introduction of enteral feed only suggested that early EN was associated with a trend toward a reduction in mortality (RR 0.52; 95% CI 0.25 to 1.08; p=0.08) when compared with delayed nutrient intake and infection risk was not different [101]. The compilation of 11 high quality studies comparing enteral and parenteral nutrition revealed a significant effect in favor of parenteral nutrition [odds ratio (OR) 0.51, 95% confidence interval (CI) 0.27–0.97]. A subgroup analysis of trials comparing parenteral nutrition with early or late enteral feeding showed that there was no survival benefit in parenteral nutrition when enteral nutrition was provided early. The benefit of parenteral nutrition was confined to trials comparing it with late enteral nutrition. Therefore, this metaanalysis confirms at least a finding already reported in earlier metaanalyses: there is no increased mortality risk with parenteral nutrition! [102].

A major concern with EN is the discrepancy between prescribed and delivered amount of nutrient, the major causes of which are diarrhea, vomiting or gastric stasis. Furthermore, enteral nutrient delivery is gradually increased in critically ill patients in order to avoid the possibility of gastrointestinal intolerance, so that a few days are required to achieve the caloric target. Administering the total nutritional requirement of mechanically ventilated medical patients starting on day 1 was associated with greater infectious complications and prolonged length of hospital stay compared to patients in whom a gradual approach was implemented [103]. Despite the caloric deficiency, EN is still superior to PN so that non-energetic effects of EN, such as immune modulation or protection of the intestinal mucosal barrier, seem to be of greater value in the critically ill than the mere energetic supply. The issue of the better enteral access (gastric vs. post-pyloric route) is not yet settled. However, available evidence does not support the routine insertion of post-pyloric tubes as long as the gastric route is effective [104, 105, 106].

Aside from the potential problems associated with receiving in adequate or excessive nutrition or medication therapy, additional injury to the patient may result from using the gut that is at risk for bacterial or candidal translocation. Therefore, enteral nutrition should be started only if the potential benefits outweigh the risks [107, 108].

However, nutritional support is not without adverse effects and risks. Early EN may be associated with high gastric residuals, bacterial colonization of the stomach, and increased risk of aspiration pneumonia. PN has been associated with gut mucosal atrophy, overfeeding,

hyperglycemia, an increased risk of infectious complications and increased mortality rates in critically ill patients [104, 105].

8. Transition from parenteral to enteral feeding and vice versa

Malabsorption and maldigestion must be recognized early in the decision making process in the use of enteral nutrition. Weight loss, signs of macronutrient (i.e., decreased visceral protein status, hypoglycemia, and steatorrhea) and micronutrient (electrolytes, trace elements, and vitamins) abnormalities suggest that the intestine may not be optimally functioning [107].

The European Society for Clinical Nutrition and Metabolism guidelines recommends that: "All patients receiving less than their targeted enteral feeding after 2 days should be considered for supplementary parenteral nutrition" [108].

Despite considerable controversy in this field, physicians generally agree on two key aspects: firstly, the enteral route is preferable whenever possible, and secondly, if possible, enteral nutritional support should be started early (within 24–48 h after admission) [101, 108, 109].

The American Society for Parenteral and Enteral Nutrition (ASPEN) and Society of Critical Care Medicine (SCCM) guidelines recommend that parenteral nutrition be initiated after 1 week, unless the patient is severely malnourished. By contrast, the European Society of Enteral and Parenteral (ESPEN) guidelines recommend consideration of a combination of enteral and parenteral nutrition after only 2–3 days in the ICU if enteral nutrition alone is insufficient at that time [108, 110].

In the early phase of rehabilitation enteral feeding solutions with low osmolarity (<300 mOsm/l) to prevent hyperosmolar diarrhea (appeared in case of long time left unfeeded intestine, and low calorie <1Kcal/ml) are usually used (Table 3).

Products	Novartis Novasource start 500ml	Nutricia Pre-nutrison 500ml	Fresenius Intestamin 500ml	Abbott Osmolite HP
Energy Kcal/ml	0, 75	0, 5	0, 5	1
Protein g/100ml	5 27%	2 16%	8, 5 68%	5, 2 20, 8%
Gluamine g/100ml	1	N.A.	6	1, 4
Carbohydrates g/100ml	8 43%	6, 1 49%	17, 7 54%	16, 4 64, 9%
Fat g/100ml	2, 5 30%	1, 95 35%	0, 2 2%	1, 5 13, 3%
Fibers	0, 5	0	0	0
Osmolarity	250	140	N.A.	269

Table 3. Starters for enteral nutrition

The rate of early products' infusion is shown in the Table 4. Moreover, Table 5 depicts most used enteral products according to categories and their characteristics.

Day	Rate	Drops per minute (20 drops=1ml)	Total Volume
1	30ml/h	10	max 500ml
2	40ml/h	10	max 1000ml
3	60ml/h	20	max 1500ml
4	90ml/h	30	max 2000ml
5	100ml/h	30	max 2000ml

Table 4. Starting enteral nutrition rate

Categories	Products	Characteristics
Isotonic	Osmolite HN (Abbott)	1 kcal/ml
	Pediasure (Abbott)	
	Frebini (Fresenius)	
	Fresubin Original (Fresenius)	
	Isosource Standard (Novartis)	
	Nutrison Standard (Nutricia)	
	Nutrini Standard (Nutricia)	
	Tetrini Standard (Nutricia)	
Enriched with fibers	Jevity FOS (Abbott)	1 kcal/ml
	Fresubin Energy Fibre (Fresenius)	1, 5 kcal/ml
	Fresubin Original Fibre (Fresenius)	1 kcal/ml
	Novasource Forte (Novartis)	1, 5 kcal/ml
	Novasource Gl Control (Novartis)	1, 1 kcal/ml
	Cubison (Nutricia)	1 kcal/ml
	Nutrison Multifibre (Nutricia)	
	Stresson Multi Fibre (Nutricia)	1 kcal/ml
Hypercaloric	Ensure Plus (Abbott)	1, 5 kcal/ml
	Fresubin Energy (Fresenius)	
	Fresubin HP Energy (Fresenius)	

http://dx.doi.org/10.5772/58382

Categories	Products	Characteristics
	Novasource Forte (Novartis)	
	Nutrison Energy (Nutricia)	
	Nutridrink (Nutricia)	
Hyperprotein	Perative (Abbott)	1, 31 kcal/ml
	Fresubin HP Energy (Fresenius)	1, 5 kcal/ml
	Intestamin (Fresenius)	0.5 kcal/ml)
	Infatrini (Nutricia	1 kcal/ml

Table 5. Most used enteral solution products per company

The goal of rehabilitation is to return through a gradual transition from the feeding tube back in swallowing if possible. The steps for neurocognitive and neuromuscular patients are based in a clinical algorithm proposed by Buchholz [111].

The initial (preparatory) phase focuses on physiologic readiness for oral nutrition and incorporates medical and nutrition stability (normal swallowing function and nutrition values in normal range), and includes implementation of intermittent tube feeding, and swallowing assessment. The second (weaning) phase is described as a graduated increase in oral feeding, with corresponding decreases in tube feeding. In a patient able to consume more than 75% of his nutrition requirements consistently by mouth for 3 days, all tube feedings are discontinued. Subjects during weaning phase are being continuously evaluated for specific clinical parameters including weight, hydration, and swallowing ability, focusing on respiratory complications [111, 112].

Author details

Yannis Dionyssiotis[1,2*]

1 Rehabilitation Center "Aghios Loukas o Iatros", Trikala Thessaly, Greece

2 University of Athens, 1st Department of Orthopaedics, General University Hospital Attikon, Athens, Greece

References

[1] Dawodu TS, Scott DD, Chase M. Nutritional management in the rehabilitation setting. http://emedicine.medscape.com/article/318180-overview

[2] Leistra E, Neelemaat F, Evers AM, van Zandvoort MH, Weijs PJ, van Bokhorst-de van der Schueren MA, Visser M, et al. Prevalence of undernutrition in Dutch hospital outpatients. Eur J Intern Med 2009;20(5):509-13.

[3] Harris D, Haboubi N. Malnutrition screening in the elderly population. J R Soc Med 2005;98(9):411-4.

[4] Stratton RJ, Green CJ, Elia M. Disease Related Malnutrition: an Evidence Based Approach to Treatment. Oxford: CABI, 2003

[5] Anson CA, Shepherd C. Incidence of secondary complications in spinal cord injury. Int J Rehabil Res 1996;19(1):55-66.

[6] Chen YM, Ho SC, Lam SS, Chan SS. Validity of body mass index and waist circumference in the classification of obesity as compared to percent body fat in Chinese middle-aged women. Int J Obes (Lond) 2006;30(6):918-25.

[7] Liang H, Chen D, Wang Y, Rimmer JH, Braunschweig CL. Different risk factor patterns for metabolic syndrome in men with spinal cord injury compared with able-bodied men despite similar prevalence rates. Arch Phys Med Rehabil 2007;88(9): 1198-204.

[8] Dufoo M Jr., Oseguera AC, Dufoo-Olvera M, Lopez OG, Palacios JL, Trejo AA, Toledo GC, et al. Metabolic changes and nutritional status in the spinal cord injured patient ASIA A. Evaluation and monitoring with routine laboratories, a feasible option. Acta Ortop Mex 2007;21(6):313-7.

[9] Rodriguez D. Nutritional Assessment and Management in spinal cord injury patients. In Charles Tator and Edward Benzel (Eds). Contemporary Management of Spinal Cord Injury: From Impact to Rehabilitation.), 2nd edition, Publisher: Thieme / AANS; 2000.

[10] Dionyssiotis Y. Body Composition in Disabilities of Central Nervous System. In: El Maghraoui, Editor. Dual Energy X-Ray Absorptiometry, Rijeka: InTech; 2012.p 75-94.

[11] Shetty P. Malnutrition and Under nutrition. Medicine.2003;31(4):18-22.

[12] Peiffer SC, Blust P, Leyson JF. Nutritional assessment of the spinal cord injured patient. J Am Diet Assoc 1981;78(5):501-5.

[13] McClave SA, Martindale RG, Vanek VW, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). JPEN J Parenter Enteral Nutr 2009;33(3):277-316.

[14] Pinheiro Volp AC, Esteves de Oliveira FC, Duarte Moreira Alves R, Esteves EA, Bressan J. Energy expenditure: components and evaluation methods. Nutr Hosp 2011;26(3):430-40.

[15] Williams RR, Fuenning CR. Circulatory indirect calorimetry in the critically ill. JPEN J Parenter Enteral Nutr 1991;15(5):509-12.

[16] Long CL, Schaffel N, Geiger JW, Schiller WR, Blakemore WS. Metabolic response to injury and illness: estimation of energy and protein needs from indirect calorimetry and nitrogen balance. JPEN J Parenter Enteral Nutr 1979;3(6):452-6.

[17] Mifflin MD, St Jeor ST, Hill LA, et al. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr 1990;51(2):241-7.

[18] Harris JA, Benedict FG. A Biometric Study of Human Basal Metabolism. Proc Natl Acad Sci USA 1918;4(12):370-3.

[19] Cerra FB, Benitez MR, Blackburn GL, Irwin RS, Jeejeebhoy K, Katz DP, et al. Applied nutrition in ICU patients. A consensus statement of the American College of Chest Physicians. Chest 1997;111(3):769-78.

[20] Faisy C, Guerot E, Diehl JL, Labrousse J, Fagon JY. Assessment of resting energy expenditure in mechanically ventilated patients. Am J Clin Nutr 2003;78(2):241-9.

[21] Ireton-Jones CS, Turner WW, Liepa GU, Baxter CR. Equations for estimation of energy expenditures in patients with burns with special reference to ventilator status. J Burn Care Rehab 1992;13(3):330-3.

[22] Ireton-Jones CS, Turner WW. Actual or ideal body weight: which should be used to predict energy expenditure? J Am Diet Assoc 1991;91(2):193-5.

[23] Frankenfield DC, Coleman A, Alam S, Cooney RN. Analysis of estimation methods for resting metabolic rate in critically ill adults. JPEN J Parenter Enteral Nutr 2009;33(1):27-36.

[24] Frankenfield DC. Validation of an equation for resting metabolic rate in older obese, critically ill patients. JPEN J Parenter Enteral Nutr 2011;35:264-9.

[25] Frankenfield DC, Ashcraft CM, Galvan DA. Longitudinal prediction of metabolic rate in critically ill patients. JPEN J Parenter Enteral Nutr 2012;36(6):700-12.

[26] Hamwi GL. Therapy: changing dietary concepts. In: Danowski TS, ed. Diabetes Mellitus: Diagnosis and Treatment. New York, NY: American Diabetes Association; 1964.

[27] Frankenfield DC, Hise M, Malone A, Russell M, Gradwell E, Compher C.Prediction of resting metabolic rate in critically ill adult patients: results of a systematic review of the evidence. J Am Diet Assoc 2007;107(9):1552-61.

[28] Campbell CG, Zander E, Thorland W. Predicted vs. measured energy expenditure in critically ill, underweight patients. Nutr Clin Pract 2005;20(2):276-80.

[29] Kearns PJ, Thompson JD, Werner PC, Pipp TL, Wilmot CB. Nutritional and metabolic response to acute spinal-cord injury. JPEN J Parenter Enteral Nutr 1992;16(1):11-5.

[30] Jeejeebhoy KN. Total parenteral nutrition at home. Can J Surg 1976;19(6):477-8.

[31] Cerra FB, Shronts EP, Raup S, Konstantinides N. Enteral nutrition in hypermetabolic surgical patients. Crit Care Med 1989;17(7):619-22.

[32] Owen OE, Kavle E, Owen RS, et al. A reappraisal of caloric requirements in healthy women. Am J Clin Nutr 1986;44(1):1-19.

[33] Jeejeebhoy KN, Baker JP, Wolman SL, Wesson DE, Langer B, Harrison JE, et al. Critical evaluation of the role of clinical assessment and body composition studies in patients with malnutrition and after total parenteral nutrition. Am J Clin Nutr 1982 ; 35(5 Suppl):1117-27.

[34] Klein JD, Hey LA, Yu CS, Klein BB, Coufal FJ, Young EP, et al. Perioperative nutrition and postoperative complications in patients undergoing spinal surgery. Spine (Phila Pa 1976). 1996;21(22):2676-82.

[35] Alpers DH, Klein S. Approach to the patient requiring nutritional supplementation. In Yamada T, ed. Textbook of Gastroenterology, 4th edn. Baltimore: Lippincott Williams & Wilkins, 2003.

[36] Dionyssiotis Y. Malnutrition in spinal cord injury: more than nutritional deficiency. J Clin Med Res 2012;4(4):227-36.

[37] Dionyssiotis ϒ, Petropoulou K, Rapidi CA, Papagelopoulos PJ, Papaioannou N, Galanos A, Papadaki P, and Lyritis GP. Body Composition in Paraplegic Men. Journal of Clinical Densitometry 2008;11(3):437-43.

[38] Gupta N, White KT, Sandford PR. Body mass index in spinal cord injury – a retrospective study. Spinal Cord. 2006;44(2):92-4.

[39] McDonald CM, Abresch-Meyer AL, Nelson MD, Widman LM. Body mass index and body composition measures by dual x-ray absorptiometry in patients aged 10 to 21 years with spinal cord injury. J Spinal Cord Med. 2007;30:S97-104.

[40] Jones LM, Legge M, Goulding A Healthy body mass index values often underestimate body fat in men with spinal cord injury. Arch Phys Med Rehab 2003;84(7): 1068-71

[41] Buchholz AC, Bugaresti JM. A review of body mass index and waist circumference as markers of obesity and coronary heart disease risk in persons with chronic spinal cord injury. Spinal Cord. 2005;43(9):513-8.

[42] Laughton GE, Buchholz AC, Martin Ginis KA Lowering body mass index cutoffs better identifies obese persons with spinal cord injury. Spinal Cord 2009;47(10):757-62.

[43] Formica CA, Cosman F, Nieves J, Herbert J, Lindsay R. Reduced bone mass and fat-free mass in women with multiple sclerosis: effects of ambulatory status and glucocorticoid Use. Calcif Tissue Int 1997;61(2):129-33.

[44] Charney P. Nutrition assessment in the 1990s: where are we now? Nutr Clin Pract. 1995;10(4):131-9.

[45] Ingenbleek Y, Van Den Schrieck HG, De Nayer P, De Visscher M. Albumin, transferrin and the thyroxinebinding prealbumin/retinol-binding protein (TBPARBP) complex in assessment of malnutrition. Clin Chim Acta 1975;63(1):61-7.

[46] Devoto G, Gallo F, Marchello C, Racchi O, Garbarini R, Bonassi S, et al. Prealbumin serum concentrations as a useful tool in the assessment of malnutrition in hospitalized patients. Clin Chem 2006;52(12):2281-5.

[47] Mears E. Linking serum prealbumin measurements to managing a malnutrition clinical pathway. J Clin Ligand Assay 1999;22:296-303.

[48] Robinson MK, Trujillo EB, Mogensen KM, Rounds J, McManus K, Jacobs DO. Improving nutritional screening of hospitalized patients: the role of prealbumin. JPEN J Parenter Enteral Nutr 2003;27(6):389-95;

[49] Frankenfield D. Energy expenditure and protein requirements after traumatic injury. Nutr Clin Pract 2006;21(5):430-7.

[50] Hertroijs D, Wijnen C, Leistra E, Visser M, van der Heijden E, Kruizenga H. Rehabilitation patients: undernourished and obese? J Rehabil Med 2012;44(8):696-701.

[51] [Set Performance Indicator rehabilitation centers.] Commissie Prestatie-indicatoren, Revalidatie Nederland en Nederlands Vereniging van Revalidatieartsen 2011 (in Dutch).

[52] Kruizenga HM, Seidell JC, de Vet HCW, Wierdsma NJ, van Bokhorst-de van der Schueren. Development and validation of a hospital screening tool for malnutrition: the short nutritional assessment questionnaire (SNAQ). Clin Nutr 2005; 24(1):75–82.

[53] Stratton RJ, Hackston A, Longmore D, Dixon R, Price S, Stroud M. Malnutrition in hospital outpatients and inpatients: prevalence concurrent validity and ease of use of the 'Malnutrition Universal Screening Tool' ('MUST') for adults. Br J Nutr 2004; 92(5): 799–808.

[54] Kruizenga HM, de Vet HC, Van Marissing CM, Stassen EE, Strijk JE, Van Bokhorst-de Van der Schueren MA, et al. The SNAQ(RC), an easy traffic light system as a first step in the recognition of undernutrition in residential care. J Nutr Health Aging 2010;14(2):83-9.

[55] National Collaborating Centre for Acute Care (UK). Nutrition Support for Adults: Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition. London: National Collaborating Centre for Acute Care (UK); 2006 Feb.

[56] Kolpek JH, Ott LG, Record KE, Rapp RP, Dempsey R, Tibbs P, Young B. Comparison of urinary urea nitrogen excretion and measured energy expenditure in spinal cord injury and nonsteroid-treated severe head trauma patients. JPEN J Parenter Enteral Nutr 1989 ;13(3):277-80.

[57] Claus-Walker J, Halstead LS. Metabolic and endocrine changes in spinal cord injury: IV. Compounded neurologic dysfunctions. Arch Phys Med Rehabil 1982;63(12):632-8.

[58] Wilmore DW. Catabolic illness: strategies for enhancing recovery. N Engl J Med 1991;325(10):695-702.

[59] Burnham EL, Moss M, Ziegler TR. Myopathies in critical illness: characterization and nutritional aspects. J Nutr 2005;135(7):1818S-23S.

[60] Bongers T, Griffiths RD, McArdle A. Exogenous glutamine: the clinical evidence. Crit Care Med 2007;35(9 Suppl):S545-52.

[61] Cree MG, Wolfe RR. Postburn trauma insulin resistance and fat metabolism. Am J Physiol Endocrinol Metab 2008;294(1):E1-9.

[62] Thibault-Halman G, Casha S, Singer S, Christie S. Acute management of nutritional demands after spinal cord injury. J Neurotrauma 2011;28(8):1497-507.

[63] Robertson CS, Grossman RG. Protection against spinal cord ischemia with insulin-induced hypoglycaemia. J Neurosurg 1987;67(5):739-44.

[64] Burr RG, Clift-Peace L, Nuseibeh I. Haemoglobin and albumin as predictors of length of stay of spinal injured patients in a rehabilitation centre. Paraplegia 1993;31(7):473-8.

[65] Gottschlich MM, Matarese LE, Shronts EP. Nutrition Support Dietetics Core Curriculum. 2 ed. Silver Springs, MD: A.S.P.E.N., 1993.

[66] Cooper IS, Hoen TI. Metabolic disorders in paraplegics. Neurology 1952;2(4):332-40.

[67] Whedon GD, Dietrick JE, Shorr E. Modification of the effects of immobilization upon metabolic and physiologic functions of normal men by the use of an oscillating bed. Am J Med 1949;6(6):684-711.

[68] Eleazer GP, Bird L, Egbert J, Ryan C, Wei M, Guest K. Appropriate protocol for zinc therapy in long term care facilities. J Nutr Elder 1995;14(4):31-8.

[69] ter Riet G, Kessels AG, Knipschild PG. Randomized clinical trial of ascorbic acid in the treatment of pressure ulcers. J Clin Epidemiol 1995;48(12):1453-60.

[70] Peruzzi WT, Shapiro BA, Meyer PR Jr, Krumlovsky F, Seo BW. Hyponatremia in acute spinal cord injury. Crit Care Med 1994;22(2):252-8.

[71] De Jonghe B, Appere-De-Vechi C, Fournier M, Tran B, Merrer J, Melchior JC, et al. A prospective survey of nutritional support practices in intensive care unit patients: what is prescribed? What is delivered? Crit Care Med 2001;29(1):8-12.

[72] Nardo P, Dupertuis YM, Jetzer J, Kossovsky MP, Darmon P, Pichard C. Clinical relevance of parenteral nutrition prescription and administration in 200 hospitalized patients: a quality control study. Clin Nutr 2008;27(6):858-64.

[73] Shaw JH, Wildbore M, Wolfe RR. Whole body protein kinetics in severely septic patients: the response to glucose infusion and total parenteral nutrition. Ann Surg 1987;205(3):288-94.

[74] Streat SJ, Beddoe AH, Hill GL. Aggressive nutritional support does not prevent protein loss despite fat gain in septic intensive care patients. J Trauma 1987 ;27(3): 262-6.

[75] Monroe MB, Tataranni PA, Pratley R, Manore MM, Skinner JS, Ravussin E. Lower daily energy expenditures as measured by a respiratory chamber in subjects with spinal cord injury compared with control subjects. American Journal of Clinical Nutrition 1998; 68(6):1223-7.

[76] Perkash A, Brown M. Anaemia in patients with traumatic spinal cord injury. Paraplegia 1982;20(4):235-6.

[77] Blissitt PA. Nutrition in acute spinal cord injury. Crit Care Nurs Clin North Am 1990;2(3):375-84.

[78] Braunschweig C, Levy P. Sheean P, Wang X. Enteral compared to parenteral nutrition: a meta analysis American Journal of Clinical Nutrition 2001;74(4):534-42.

[79] Yin L, McLennan M, Bellou TF. Overweight in children with intellectual disabilities: No Simple Matter. ICAN: Infant, Child, & Adolescent Nutrition April 2013 5: 92-6.

[80] Bauman WA, Zhong YG, Schwartz E. Vitamin D deficiency in veterans with chronic spinal cord injury Metabolism 1995; 44(12):1612–6.

[81] Dionyssiotis Y. Bone loss and fractures in multiple sclerosis: focus on epidemiologic and physiopathological features. Int J Gen Med 2011;4:505-9.

[82] Maklebust J, Magnan MA. Risk factors associated with having a pressure ulcer: a secondary data analysis. Adv Wound Care 1994;7(6):25, 27-8, 31-4 passim.

[83] Fishburn MJ, Marino RJ, Ditunno JF Jr. Atelectasis and pneumonia in acute spinal cord injury. Arch Phys Med Rehabil 1990;71(3):197-200.

[84] Endersbe LA. Nutrition Support in Neurologic Impairment. In: Shronts, Eva P, editors. Nutrition support dietetics. Maryland, Aspen: Silver Spring, p.107-18, 1989.

[85] Jacksic T, Blakburn GL. Nutrition and CNS disease, the unconscious patient. In: Jeejeebhoy KN, editor. Current therapy in nutrition. Toronto, Philadelphia: B C Decker Inc., p.269-78, 1988.

[86] Minard G, Kudsk K A. Is early feeding beneficial? How early is early? New horizons (Baltimore, Md.), 2(2), p. 156-63, 1994.

[87] Nyswonger GD, Helmchen RH. Early enteral nutrition and length of stay in stroke patients. J Neurosci Nurs 1992; 24(4):220-3.

[88] Kirby DF, Clifton GL, Turner H, Marion DW, Barrett J, Gruemer HD. Early enteral nutrition after brain injury by percutaneous endoscopic gastrojejunostomy. JPEN J Parenter Enteral Nutr 1991;15(3):298-302.

[89] Grahm TW, Zadrozny DB, Harrington T: The benefits of early jejunal hyperalimentation in the head-injured patient. Neurosurgery 1989; 25(5):729-35.

[90] Magnuson B, Hatton J, Zweng TN, Young B. Pentobarbital coma in neurosurgical patients: nutrition considerations. Nutr Clin Pract 1994;9(4):146-50.

[91] O'Keefe SJ. A guide to enteral access procedures and enteral nutrition. Nat Rev Gastroenterol Hepatol 2009;6(4):207-15.

[92] Zaloga GP. The myth of the gastric residual volume. Crit Care Med 2005;33(2):449-50.

[93] Park RH, Allison MC, Lang J, Spence E, Morris AJ, Danesh BJ, et al.Randomised comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding in patients with persisting neurological dysphagia. BMJ 1992;30:(304):1406-9.

[94] Wicks C, Gimson A, Vlavianos P, Lombard M, Panos M, Macmathuna P, et al. Assessment of the percutaneous endoscopic gastrostomy feeding tube as part of an integrated approach to enteral feeding. Gut 1992;33(5):613-6.

[95] Allison MC, Morris AJ, Park RH, Mills PR. Percutaneous endoscopic gastrostomy tube feeding may improve outcome of late rehabilitation following stroke. J R Soc Med 1992;85(3):147-9.

[96] Larson DE, Burton DD, Schroeder KW, DiMagno EP. Percutaneous endoscopic gastrostomy. Gastroenterology, 1987; 93(1), 48-52.

[97] Miller RE, Castlemain B, Lacqua FJ, Kotler DP. Percutaneous endoscopic gastrostomy. Results in 316 patients and review of literature. Surg Endosc 1989;3(4):186-90.

[98] Burtch GD, Shatney CH. Feeding gastrostomy. Assistant or assassin? Am Surg 1985;51(4):204-7.

[99] Fay DE, Poplausky M, Gruber M, Lance P. Long-term enteral feeding: a retrospective comparison of delivery via percutaneous endoscopic gastrostomy and nasoenteric tubes. Am J Gastroenterol 1991;86(11):1604-9.

[100] Marik PE, Zaloga GP. Early enteral nutrition in acutely ill patients: a systematic review. Crit Care Med 2001;29(12):2264-70. Erratum in: Crit Care Med 2002;30(3):725.

[101] Heyland DK, Dhaliwal R, Drover JW, Gramlich L, Dodek P; Canadian Critical Care Clinical Practice Guidelines Committee. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. JPEN J Parenter Enteral Nutr 2003;27(5):355-73.

[102] Simpson F, Doig GS. Parenteral vs. enteral nutrition in the critically ill patient: a meta-analysis of trials using the intention to treat principle. Intensive Care Med 2005;31(1):12-23.

[103] Kudsk KA, Croce MA, Fabian TC, Minard G, Tolley EA, Poret HA, et al. Enteral versus parenteral feeding. Effects on septic morbidity after blunt and penetrating abdominal trauma. Ann Surg 1992;215(5):503-11; discussion 511-3.

[104] Heyland DK, Konopad E, Alberda C, Keefe L, et al. How well do critically ill patients tolerate early, intragastric enteral feeding? Results of a prospective, multicenter trial. Nutr Clin Pract 1999;14(1):23-8.

[105] Rello J, Quintana E, Ausina V, Castella J, Luquin M, Net A, et al. Incidence, etiology, and outcome of nosocomial pneumonia in mechanically ventilated patients. Chest 1991;100(2):439-44.

[106] Heyland DK, Macdonald S, Keefe L, Drover JW. Total parenteral nutrition in the critically ill patient: a meta analysis. JAMA 1998; 280(23):2013-9.

[107] Baumgartner TG, Cerda JJ, Somogyi L, Baumgartner SL. Enteral Nutrition in Clinical Practice Croatian Med J 1999;40(4):515-27.

[108] Singer P, Berger MM, Van den Berghe G, Biolo G, Calder P, Forbes A, Griffiths R, Kreyman G, Leverve X, Pichard C, ESPEN. ESPEN Guidelines on Parenteral Nutrition: intensive care. Clin Nutr 2009;28(4):387-400.

[109] Martindale RG, McClave SA, Vanek VW, McCarthy M, Roberts P, Taylor B, et al. American College of Critical Care Medicine; A.S.P.E.N. Board of Directors. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition: Executive Summary. Crit Care Med 2009;37(5): 1757-61.

[110] Vincent JL, Preiser JC. When should we add parenteral to enteral nutrition? Lancet 2013;381(9864):354-5.

[111] Buchholz AC. Weaning patients with dysphagia from tube feeding to oral nutrition: a proposed algorithm. Can J Diet Pract Res 1998;59(4):208–14.

[112] Crary MA, Groher ME. Reinstituting oral feeding in tube-fed adult patients with dysphagia. Nutr Clin Pract 2006;21(6):576-86.

IntechOpen