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Nutritional Aspects of Disability and Rehabilitation

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Nutrition as a Factor Contributing to Disability

Disability from systemic diseases such as stroke, brain injury, chronic heart failure, chronic obstructive pulmonary disease, cancer as well as from major surgery or skeletal trauma may be exalted by alterations in the patient's nutrition induced by or associated with the primary disease. Fundamentally, and for practical purposes, the effects of altered nutrition relevant to disability are reduced skeletal muscle mass and function and an increased incidence of infection. These are particularly evident in elderly subjects in whom nutritional reserves are already low because of ageing.

The knowledge of both the mechanisms by which a disease may bring about nutritional alterations and the impact exerted, in turn, by poor nutrition on body compartments is of paramount importance to prevent or correct total body deterioration during the acute phase of the disease and limit the functional sequelae during the chronic phase of the disease.

Figure 1 reports the main mechanisms by which the primary disease may lead to nutritional deterioration. Tables 1 and 2 describe the impact of reduced skeletal mass and function as well as that of impaired immunological defences on a patient's function (Alden et al. 1987; Ulicny and Hiratzka 1992; Arora and Rochester 1982; Newton et al. 1987; Coonrod et al. 1984; Sahebjami and MacGee 1982; McMurray 1984).

Figure 1 highlights that muscle wasting can be brought about by the patient's inadequate energy and nutrient intake/supply (=increased ratio of energy nutrient consumption/intake), abnormalities in substrate utilization within muscle cells (anabolic/catabolic hormone imbalance, metabolic effect of cytokine overproduction), reduced protein synthesis (immobility) and augmented myofibrillar protein degradation (for example, from cytokine overproduction, elevated plasma levels of catabolic hormones). Whatever the mechanism(s), the final result is a reduction in muscle mass and function. Impairment in muscle mass and muscle function are particularly relevant to disability being primarily involved in locomotion and respiratory effort.

The other potential major consequence of poor nutrition is increased susceptibility to infections and their recurrence because of the reduction of immunocompetence that may characterize undernourished patients. Infections, particularly when repeated over time, have a negative impact on a patient's functioning because they exalt all the aforementioned mechanisms of muscle wasting and dysfunction.

Overall, the consequences of infection on skeletal muscle are poorly understood and thus underestimated in daily clinical practice. Ignoring the strict links between altered nutrition and an impaired immunological response to pathogens as well as between infection and muscle wasting can lead both to the patient being exposed to recurrent episodes and low resolution of infection and to the physician treating the infection symptomatically i.e. without removing the underlying mechanisms favouring the infection.

Particularly relevant to patients with a cerebral insult (from a vascular accident or trauma) is that an inadequate nutritional intake, especially in proteins/amino acids, may impede the retrieval from or even aggravate cognitive-motor defects as well as depressive symptoms.

Modifications in nutrition may occur subtly overtime so health professionals and caregivers should actively assess the patient's nutrition during the acute phase of a disease and monitor the nutrition periodically over time even when the patient's disability is in a stabilised phase. It is implicit in the foregoing that patients themselves, when possible, health professionals and care-givers have adequate information on the importance of nutrition to limit or even reduce the degree of disease-induced disability.

Nutritional Aspects of Rehabilitation in People with Disabilities

In clinical practice, nutritional interventions serve to prevent nutritional deterioration of the body or to provide energy and nutrients to subjects with a temporary or permanent loss of feeding autonomy. An important but almost unknown aspect of nutritional intervention is that nutrition can be used to enhance a patient's recovery from a functional impairment or disability, i.e. nutrients can improve metabolic abnormalities in injured organs/tissues (metabolic rehabilitation). For this purpose, however, it is essential that a patient's energy and protein intake/supply is sufficient for the body's requirements, otherwise body deterioration will continue to worsen and the results of metabolic rehabilitation will be disappointing. Body energy and protein requirements depend on a multitude of factors including type and severity of disease, possible presence of complications and comorbidities, the patient's actual nutritional status, drugs being taken, and physical activity. It is, therefore, necessary to estimate energy and protein needs in every patient with disability.

Estimating a patient's energy requirement

The amount of energy a single patient needs can be calculated using the formula:
E_{TOT} *(kcal/day) = BMR + IF + PA + TEF*

where:

- **BMR** (kcal/day) is the basal metabolic rate (= the amount of energy necessary for metabolic activity of organs and tissues in resting conditions);
- **IF** is an injury (or disease) factor = 20% BMR (in case of fever add 0.07 BMR x each degree of fever above 98.6 °F);
- **PA** is physical activity (bed rest = 10% BMR, light activity = 30% BMR, moderate activity = 50% BMR)
- **TEF** is the thermal effect of food (a correction factor calculated as 10% of the BMR).

In rehabilitation settings, we adopt BMR values measured for some specific diseases by our group by indirect calorimetry ("gold standard") (Table 3) (Aquilani et al. 2000; Aquilani et al. 2001; Aquilani et al. 1999; Aquilani et al. 2003; Aquilani et al. 1990; Aquilani et al. 1997; Gallì et al. 1994).

Estimating a patient's body protein requirement

Together with adequate energy intake, sufficient daily protein ingestion plays a major role not only in nitrogen balance but also in metabolic rehabilitation.

The amount of protein ingested is considered to be adequate when it is 0.8-1 g/kg body weight/day in healthy adult subjects; the value rises to 1.5 g/kg in elderly individuals (>65 years) (Wolfe 2008).

Protein needs in chronic disabling disease are largely unknown. For some specific diseases we adopt, as for energy, the values derived from our investigations (Table 4) (Aquilani et al. 2001; Aquilani et al. 1999; Aquilani et al. 2003).

To understand the impact of exogenous proteins on injured organs and tissues, we have to refer sometimes to the elementary components of a protein, the amino acids (AAs). As shown in Table 5, the AAs are classified into non-essential AAs (NEAAs) that can be ingested in food but can also be derived from endogenous production, essential AAs (EAAs) that must be provided in food or as an exogenous supplement because they are not formed endogenously, and semi-essential AAs, i.e. NEAAs that in conditions of metabolic stress (trauma, wound repair) should be augmented through the use of specific foods or supplements. For clarity of the discussion, the terms proteins and AAs are interchangeably in the following.

Here we report some examples of how adjustments in AA intake/supply may induce metabolic rehabilitation.

Metabolic rehabilitation

To increase muscle mass and strength

In order to obtain an increase in a reduced skeletal mass, a balanced diet should be integrated with EAAs (Volpi et al. 2003). In fact, it has been documented, particularly in elderly, that: 1) high-protein diets or commercial nutritional supplements are unsuccessful at increasing protein synthesis and, therefore, muscle mass and strength (Welle and Thornton 1998); 2) the presence of carbohydrates in a nutritional supplement is not beneficial (Volpi et al. 2000); 3) EAAs but not NEAAs are mostly responsible for amino acid stimulation of muscle protein synthesis (Smith et al. 1998).

Two investigations by our group confirmed the importance of giving supplementary EAAs to patients.

In subjects with moderate/severe chronic heart failure (CHF), 2-month supplementation with 8 g/day oral EAAs increased body weight and muscle mass (Aquilani et al. 2008d) and improved physical autonomy (walking test), whereas increases in body weight, muscle mass, and physical functioning did not occur in the control CHF patients not given such supplementation. Interestingly, both supplemented and control patients had similar energy and protein intakes which were adequate for their body requirements.

In a very recent, preliminary investigation in severely undernourished, cachectic patients with severe chronic obstructive pulmonary disease, 3-month supplementation with 8 g/day oral EAAs (the same commercial product as used in the CHF study) increased body weight (>2 kg in 80% patients), fat free mass (measured by DEXA), and the walking test and improved quality of life (Baldi et al. 2008).

To enhance motor and cognitive recovery of patients with ischaemic stroke

Enhancement of neurological recovery

In patients with subacute ischaemic stroke (>14 days from index event), 21 days of protein supplementation (+20 g/day) improved neurological alterations, assessed by the National Institute of Health Stroke Scale, by an average of 4.4 points whereas in controls the improvement was an average of 3.0 points. This difference was statistically significant (Aquilani et al. 2008c).

This result may be explained by the fact that: 1) brain neurons process more amino acids than do tissues outside the brain (Clarke and Sokoloff 1998); 2) alimentary proteins/amino acids influence brain protein/amino acid content (Bourre 2004; Choi et al. 2000); 3) proteins may contribute to post-stroke cortical plasticity and recovery of motor activity of paretic limbs through the following mechanism: (i) by inducing axonal sprouting and formation of new neuronal network both in perilesional zones and in territories remote from the site of a lesion (Nudo et al. 1997); (ii) increased brain amino acid utilization may improve motor recovery by acting synergistically with rehabilitation procedures (Lindberg et al. 2007); and (iii) an increase in availability of amino acids in the brain may induce an increase in neurotransmitter synthesis (Dobkin 2005).

Interestingly, we found that increased ingestion of carbohydrates in relation to protein ingestion was positively correlated with higher deficit in neurological test. However, an adequate intake of carbohydrates (2.5-4 g/kg) should always be assured. Thus, the way for reducing carbohydrate/protein ratio is to augment protein intake/supplementation.

Enhancement of cognitive recovery

In patients with subacute stroke, 21 days of calorie-protein supplementation (+250 kcal, +20 g proteins) with an oral commercial formula enhanced the recovery of cognitive function, assessed by the Mini-Mental State Examination (Aquilani et al. 2008b), by an average of 4 points whereas cognitive dysfunction remained practically unchanged in the control group.

Plausible mechanisms that could explain this finding include: 1) energy can enhance memory by activating gut peptides and the vagus nerve (Kaplan et al. 2001), 2) proteins, hence amino acids, may influence brain structures: (i) directly, by restoring protein synthesis in ischaemic regions (protein synthesis may reduce the expansion of brain infarction) (Hata et al. 2000), by affecting the formation of neurotrophins (Mattson 1997), and by increasing neurotransmitter synthesis; and (ii) indirectly, by inducing an increase in the cerebral content of insulin (Baskin et al. 1988), a hormone involved in brain cognitive function and dysfunction (Zhao and Alkon 2001).

To reduce the Disability Rating Scale (DRS) in patients with Traumatic Brain Injury (TBI) in or not in a vegetative/minimally conscious state

In patients with severe TBI not in a vegetative/minimally conscious state at 2 months from the index event, a 15-day venous infusion of EAAs as branched chain AAs (BCAAs) improved the DRS score by an average of 6.3 points, whereas in control group the mean improvement in the DRS score was 2.8 points (Aquilani et al. 2005).

Plausible mechanisms for the BCAA-induced improvement in the DRS score include: 1) increased neuronal energy formation, since the shortage in such energy is responsible for alterations in ion pumps, which contribute to the death of neurons in TBI; 2) increased protein synthesis, which is essential for brain tissue repair, sprouting and circuitry remodelling; and 3) insulin-mediated actions, given that insulin may modulate cognitive activity in the central nervous system (Park 2001).

In patients with TBI in a vegetative/minimally conscious state at < 90 days from index event, a 15-day venous infusion of BCAAs permitted 68.2% of the patients to emerge from the vegetative/minimally conscious state (Aquilani et al. 2008a). The DRS score improved significantly only in patients who had received BCAAs.

Potential mechanisms of the effect of BCAAs on the recovery from a vegetative state include: 1) increased brain energy formation (Clarke and Sokoloff 1998) leading to better restoration of ionic homeostasis, preservation of neuron viability by stopping cellular damage from free radical overproduction (Ahmed et al. 2000), and synthesis, axonal transport and secretion of neurotransmitters (Stenoiden and Brady 1998), in particular GABA (Wu et al. 2004); and 2) insulin-mediated restoration of neuronal activity in hippocampal, pyramidal neurons (Kern et al. 2002).

To improve muscle energy metabolism and physical activity in subjects with Chronic Renal Failure (CRF)

In patients with CRF (Aquilani et al. 1997), 1 year of a low protein diet (0.5 g/kg/day) with natural foods and a calorie intake equal to 1.4 x Resting Energy Expenditure, measured by indirect calorimetry, reduced muscle energy metabolism, increased physical capacity, and reduced symptoms of muscle weakness, particularly when climbing stairs or walking.

Possible mechanisms of the patients' improvement include: 1) the observed increase in muscle ATP concentration (muscle biopsy) possibly induced by a reduction in ATP consumption; 2) the observed slowing down of the velocity of the aerobic cycle; and 3) a hypothesized increase in stroke volume and in oxygen arterio-venous difference enabling greater exercise tolerance.

Conclusion

Nutrition may constitute an added value to rehabilitation in order to enhance the recovery of disability. Future studies probably will highlight the effect of nutrition to other frequent disabling conditions including skeletal trauma and body wasting after major surgery, especially in elderly patients.

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Figures and Tables

Figure 1. Main mechanisms leading to disease-induced nutritional deterioration.

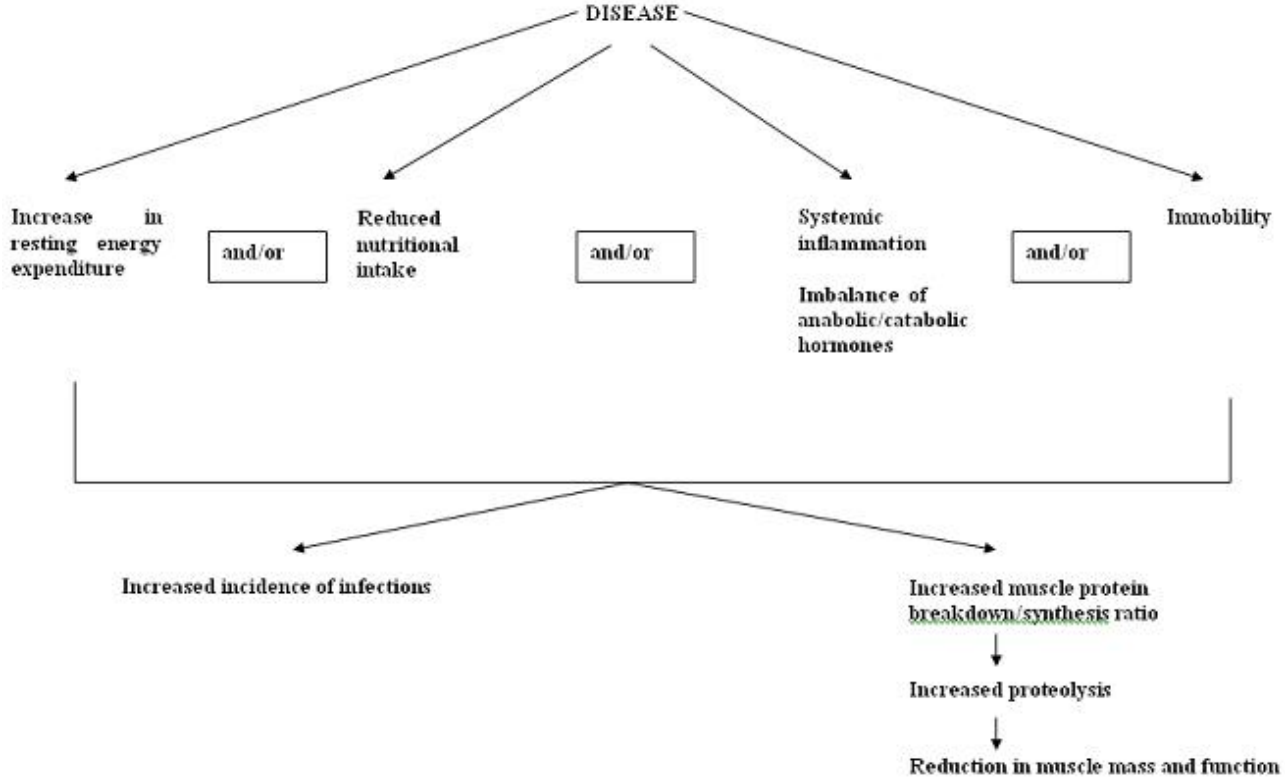

Table 1. Summary of the functional effects of reduced muscle mass in various body districts

DISTRICT	IMPAIRMENT
SKELETAL MUSCLES	• Weakness
MYOCARDIUM	• Loss of myocardial mass proportional to the loss of skeletal mass • Decrease in global ventricular contractility • Decrease in stroke volume and cardiac output proportional to myocardial mass
RESPIRATORY MUSCLES	• Impaired muscle efficiency ↓ muscle fatigue
MUSCLES OF MASTICATION	• Weakness ↓ early fatigue ↙ ↘ dysphagia low nutritional intake

Table 2. Summary of the effects of malnutrition on the immune system

DISTRICT	IMPAIRMENT
SYSTEMIC IMMUNITY	• Decrease in cell-mediated immuno-competence: reduction in circulating T-lymphocytes, in helper T-cells and suppressor cells, in helper/suppressor T ratio • Decrease in secretory immunoglobulin A
AIRWAY DEFENCES	• Increased bacterial adherence to oral and airway cells • Alteration in mucociliary function • Alteration in alveolar macrophage function • Impaired recruitment of leucocytes

Table 3. Resting Energy Expenditure (measured by indirect calorimetry) in some conditions requiring rehabilitation

CONDITION	DAILY RESTING ENERGY EXPENDITURE
Traumatic Brain Injury	23.8±5 kcal/kg
Spinal Cord Injury - Paraplegic (with infection and pressure sores)	23.7±3.1 kcal/kg
Spinal Cord Injury - Tetraplegic (with infection)	21.4±4 kcal/kg
Ischaemic Stroke	19±2.1 kcal/kg
Chronic Heart Failure	22.9±2.4 kcal/kg
Chronic Obstructive Pulmonary Disease	18±3.6 kcal/kg
Chronic Renal Failure	19.5±0.84 kcal/kg
Following Hip Replacement Surgery	15.4±3.4 kcal/kg

Table 4. Estimated protein needs in some specific conditions. Values may vary greatly in relation to comorbidities and should, therefore, be considered as starting points.

CONDITION	DAILY ESTIMATED PROTEIN NEED
Chronic Heart Failure	>1.1 g/kg
Spinal Cord Injury - Paraplegic	>1 g/kg
Spinal Cord Injury - Tetraplegic	>1 g/kg
Stroke *(>14 days from index event)*	>1 g/kg
Chronic Obstructive Pulmonary Disease	>1.2 g/kg

Table 5. Amino acid classification

ESSENTIAL AMINO ACIDS	NON-ESSENTIAL AMINO ACIDS
Leucine (*Branched chain amino acids*)	Alanine
Isoleucine (*Branched chain amino acids*)	Aspartic Acid
Valine (*Branched chain amino acids*)	Cysteine
Phenylalanine	Glutamic Acid
Lysine	Histidine
Methionine	Serine
Threonine	Arginine
Tryptophan	Asparagine
	Glycine
	Glutamine
	Proline
	Tyrosine
	Citrulline