

Prevalence of malnutrition and inadequate food intake in self-feeding rehabilitation patients with stroke

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Background. Malnutrition is known to impair functional outcome and prolong the hospital stay of rehabilitation patients with stroke. The aim of this study was to establish the prevalence of malnutrition in self-feeding patients with stroke and to investigate the adequacy of their food intake.

Methods. One hundred and fifty self-feeding patients with stroke were considered (85 M + 65 W; 60 ± 11 yrs, 30 ± 10 days from the acute event; 45.3% with right and 54.7% with left hemiplegia). Each patient underwent the following evaluations: a) anthropometry to diagnose the malnutrition; b) resting energy expenditure (REE by indirect calorimetry, standard protocol); c) calorie and macronutrient intakes by using 3 — day food diaries; d) 24 hr urinary nitrogen excretion to measure nitrogen balance (NB).

Results. Forty-five patients (=30%) were diagnosed as malnourished. Inadequacies in calorie ($\text{Kcal-I/REE} \leq 110\%$), protein ($<0.8 \text{ g/kg/day}$) and in carbohydrate ($<140 \text{ g/d}$) intake were found, respectively in 60, 50, 38.6% of all the patients with stroke. A negative NB was observed in more than 50% of patients. Food intake inadequacy was significantly greater in malnourished than in normally nourished patients.

Conclusions. The prevalence of malnutrition in self feeding rehabilitation patients with stroke is high and a self-feeding status is not synonymous with normal food intake. Nutritional evaluations should be routinely made in patients with the sequelae of cerebrovascular accidents.

Key words: Cerebrovascular disorders rehabilitation - Self-feeding - Nutrition disorders.

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Malnutrition is a condition that may induce a number of complications in surgical and medical patients.^{1,2} Among these complications, impairment of immunocompetence³⁻⁵ and respiratory and gastrointestinal functions⁶ as well as increased risk of pressure sore formation⁶ have been described. For these reasons, the rehabilitation of patients with stroke is negatively influenced by malnutrition. Indeed, it has been demonstrated that malnourished patients stay in hospital longer and have a poorer functional outcome than normally nourished ones.^{7,8} The prevalence of malnutrition in patients with stroke, as reported in literature, varies from 16% in acute settings⁹⁻¹² to 60% in rehabilitation wards.¹³ Among the factors impairing the nutritional status of patients with stroke, reduced motor ability to self-feed, cognitive dysfunction, depression and, when present, dysphagia may all play a role. All these conditions may act to cause a decrease in the amount of food intake. However, unless food intake is quantified and body calorie need is established in every rehabilitation

TABLE I.—*Clinical characteristics of the patients in this study.*

Parameters	Values
Age (years)	60±11
Sex (No)	
Male	85
Female	65
Lesion (%)	
Ischemic	64.6
Hemorrhagic	35.4
Hemiplegia (%)	
Right	45.3
Left	54.7
Dysphagia (%)	28
Co-morbidity (%)	
Systemic hypertension	27.3
Coronary artery disease	11.3
Diabetes	18.0
Infections (urinary, respiratory)	21.3

patient with stroke, it is impossible to judge whether spontaneous nutritional intake by patients with stroke is adequate for the body's requirements. This issue should not be underestimated in clinical practice as inadequate food intake may pass unsuspected simply because patients are found to be independent in their choice of food and intakes. The aims of this investigation were, therefore, twofold: to determine the prevalence of malnutrition and to establish the inadequacy of nutritional intake, when present, of self feeding rehabilitation patients at the time of admission to our institutes.

Materials and methods

Study population

One hundred and fifty self-feeding patients with stroke, consecutively admitted to our rehabilitation institutes were considered for the study. Table I reports their clinical characteristics. All patients had clinical findings consistent with a stroke and a positive Computed Tomography or Magnetic Resonance Imaging score. Within a week of admission each patient underwent the following nutritional evaluations.

Anthropometry

- a. Height, estimated from knee height;¹⁴
- b. Weight, measured by a mechanical weight-lifter (Partner, Pabish) and recorded both as a percentage of reference weight and of usual weight;

c. Triceps skinfold thickness (TST, in mm), using a Harpenden skinfold caliper;

d. Midarm circumference (MAC), to estimate mid-arm muscle circumference (MAMC) using the formula $MAMC = MAC \text{ (cm)} - 0.314 \times TST \text{ (mm)}$.¹⁵ From MAMC estimates, arm muscle area (AMA) was calculated as following:

$$AMA \text{ (cm}^2\text{)} = \frac{MAMC^2}{12.56}$$

The patient's non-paretic arm was used for measurements of TST and MAC.

Biochemistry

Serum albumin level and total lymphocyte count were registered from the routine biochemical laboratory tests.

Resting energy expenditure measure (REE; kcal/day)

This was performed by indirect calorimetry with a Horizon metabolic measurement chart (Beckman Instruments Anaheim, CA). A canopy was used to collect the expired air. REE was performed 12 to 14 hours after overnight fasting. Oxygen consumption and carbon dioxide production were measured at 1-minute intervals and the mean of the 60-minute measurement period was calculated.¹⁶ REE values were calculated as described in the literature^{17,18} and referred to predicted values from Harris-Benedict's equation (H-B).¹⁹ REEs between 90-110% of the predicted H-B were considered normal; a REE <90% H-B was taken to indicate a condition of hypometabolism; a REE >110% H-B a state of hypermetabolism

From oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$), the respiratory quotient (RQ) was calculated as follows: $RQ = \dot{V}CO_2 / \dot{V}O_2$ (normal values 0.7 — 1.0).

Evaluation of calorie and macronutrient intake

This evaluation was made by recording the type and weight of the patient's cooked and uncooked food for three days. The findings were converted into the raw equivalent when necessary, using appropriate Tables.²⁰ Nutritional analysis using a computed system designed by our group allowed us to calculate the actual calorie and macronutrient intake. In all patients, calorie intake (kcal — I) was expressed either

TABLE II.—*Distribution of altered nutritional indicators in malnourished patients with stroke (No=45).*

Loss of weight	No patients	%
Group A	35	77.8
With presence of:		
AMB <5th percentile	9	25.7
ALB <3.5 g/dl	9	25.7
Lymph <1500 n/mm ³	5	14.3
ALB <3.5+lymph<1500	2	5.7
Group B	10	22.2
With presence of:		
AMB <5th	0	
ALB <3.5 g/dl	7	70
Lymph <1500 n/mm ³	3	30
ALB <3.5+limph <1500	0	

Group A) Patients with a loss of usual body weight >10% but with actual body weight (ABW) lower than reference weight (RBW). Group B) Patients with a loss of usual body weight ≥5% plus one or more of other malnutrition indicators (see methods).

in absolute values or as a percentage of REE (kcal-I/REE). On the last day of food recording, 24-hour urine was collected to determine nitrogen excretion (Nu). It was thus possible to calculate nitrogen balance (NB), according to the following equation:

$$NB(g/day) = Ni - Nu + 2g$$

Where Ni is the nitrogen intake obtained from the protein intake (g) divided by 6.25 and 2 g is a constant of the number of grams of nitrogen to compensate for losses, mainly fecal.²¹

Criteria for malnutrition

The diagnosis of malnutrition was established by the presence of:

1. Loss of usual body weight ≥10% but with actual body weight (ABW) lower than reference weight (RBW) (GROUP A patients).
2. Loss of usual body weight ≥5% plus one or more of the following malnutrition indicators (GROUP B patients):^{21 22}
 - a. AMA <5th percentile (a marker of reduced muscle protein store);
 - b. serum albumin <3.5 g/dl (an indicator of reduced visceral protein synthesis);
 - c. total lymphocytes <1500 n/mm³ (an indicator of impaired immunocompetence).

Criteria for inadequate food intake

1. Inadequate calorie intake for body needs was established when the patient's daily calorie intake was ≤110%REE(kcal-I/REE).
2. Inadequate protein intake (Prot-I) was judged to occur when intake was <0.8 g/day.
3. Inadequate carbohydrate intake (CHO-I) was present when intake was <140 g/day, a value that represents the daily minimum amount of CHO necessary for the function of brain and other glucose-dependent tissues (predominantly blood cell elements).
4. Inadequate lipid intake was estimated when lipid intake was <0.5 g/kg/day.

Statistical analysis

The unpaired "t" test was used to compare nutritional parameters, REEs and RQs between malnourished and normally nourished patients. Values are data as x±sd. The level of statistical significance was set at p<0.05.

Results

This study shows that 45 patients with stroke were diagnosed as malnourished according to the above malnutrition criteria (Table II). Thus, the prevalence of malnutrition in self-feeding patients with stroke was 30%. Among the malnourished patients, 18 (=40%) were dysphagic. In 71 % of group A patients, alterations in muscle protein stores, visceral protein synthesis, immunocompetence or a combination of the last two functions were also observed.

Tables III and IV report resting energy expenditure, nutritional intake, nitrogen balance and respiratory quotient in patients with stroke. Malnourished patients had lower calorie intakes and resting energy expenditures than normally nourished ones (1036±487 vs 1434±375, p<0.02 and 1111±171 Kcal vs 1404±238, p<0.001 respectively). When normalised for body weight, REEs were similar between the malnourished and the normally nourished patients (19.6±2.2 Kcal/kg vs 18.4±2.3, respectively n.s.). When expressed in absolute values REE was found to be ≤1300 kcal/day in the malnourished patients and 1500 kcal/day in 80% of the normally nourished ones. Only in 20% of

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TABLE III.—*Resting Energy Expenditure (REE) in self-feeding patients with stroke.*

Parameters	Patients		
	All	Malnourished	Normally nourished
kcal/day	1291±210	1111±171*	1404±238
range	(823-1883)	(823-1326)	(989-1883)
kcal/kg/day	19.3±2.8	19.6±2.2	18.4±2.3
range	(13.8-21.9)	(13.8-21.8)	(14.8-21.9)
% predicted**			
90-110% (normometabolic)	58.6	57.5	64.7
<90% (hypometabolic)	32	42.2	24.8
>110% (hypermetabolic)	9.4	0	10.5

*) $p<0.001$ malnourished *vs* normally nourished patients. **) Figures are percentage of patients.

TABLE IV.—*Nutritional analysis of food intake and respiratory quotient in patients with stroke. Values are mean±sd; p=level of significance; ns=not significant. Statistical analysis conducted between malnourished and normally nourished patients.*

Parameters	Patients			
	All	Malnourished	Normonourished	p
A-Intakes				
Calories				
kcal-I	1292±463	1036±486	1434±374	<0.02
kcal-I/REE	99.4±32	88.5±37	106±27	—
Protein				
g/kg/day	0.78±0.33	0.66±0.39	0.93±0.33	<0.05
% kcal-I	16.4±3.2	15.6±3.4	16.8±3	—
Lipid				
g/kg/day	0.8±0.3	0.66±0.36	0.87±0.28	<0.01
% kcal-I	34.5±5.5	33.5±6.5	35.1±5	—
Carbohydrate				
g/kg/day	2.78±0.95	2.99±1.01	2.39±0.72	ns
g/day	195±77	141±50	224±73	<0.005
% kcal-I	48.5±6.4	49±8.5	48.4±5	—
B-Nitrogen balance				
g/day	-0.65±3.4	-1.84±2.99	0.08±3.5	ns
C-Respiratory Quotient	0.77±0.1	0.71±0.09	0.81±0.09	<0.01

TABELLA V.—*Percentage of patients with inadequate nutritional intakes.*

Patients	Nutritional intakes				
	CHO <140 g/day	Protein <0.8 g/kg	Lipid <0.5 g/kg	kcal-I/REE ≤110%	Negative nitrogen balance
All patients (No 150)	38.6	50	19.3	64.6	56.6
Malnourished patients (No 45)	77.6	77.7	37.7	82.2	75.5
Normonourished patients (No 105)	17.1	30.4	7.7	51.4	45.7

CHO=carbohydrates; kcal-I=calorie intake; REE=Resting Energy Expenditure

this latter group was REE >1500 kcal/day. The Harris-Benedict formula failed to predict energy requirements in more than 37% of patients with stroke. In comparison with the normally nourished patients, the malnourished ones had lower protein (0.6 ± 0.32 g/kg *vs* 0.89 ± 0.29 g/kg, $p<0.04$), lipid (0.51 ± 0.24 g/kg *vs* 0.88 ± 0.31 g/kg, $p<0.01$), and carbohydrate intakes (140 ± 49.7 g/day *vs* 224 ± 73.5 g/day, $p<0.005$). Nitrogen balance was similar in malnourished and normally nourished patients, while respiratory quotient (RQ) was lower in the former group (0.71 ± 0.09 *vs* 0.81 ± 0.09 $p<0.01$).

Table V reports the percentage of patients with stroke who had inadequate macronutrient and calorie intakes and negative nitrogen balance. More than 75% of the patients with diagnosed malnutrition met the criteria for inadequate carbohydrate, protein, and calorie intakes while in the normally nourished patients calorie intake was inadequate and negative nitrogen balance was present in more than 50% and in about 46% respectively.

Discussion

This study conducted in rehabilitation patients with stroke yielded two main results: the prevalence of malnutrition was 30% and an inadequate nutritional intake was present in the majority of patients, mainly in those with a diagnosis of malnutrition.

The prevalence of malnutrition in this study was lower than the previously reported prevalences of 49 and 60%.^{11 13} This discrepancy is probably due to our different study plan which considered only self-feeding patients. Indeed, feeding-dependent patients were excluded as artificial nutrition in acute services is known to be associated with malnutrition.¹¹ Another factor that contributed to the lower prevalence of malnutrition in our patients than that found by the other authors is that we adopted a more restrictive

indicator of malnutrition, as a loss of weight >10% usual body weight but with the actual body weight lower than reference weight.

Diabetes, previous episodes of stroke and dysphagia are well-recognised causes of malnutrition in patients with cerebrovascular accidents.¹¹ However, in this study, malnutrition was also diagnosed in patients without a history of the above conditions meaning that other factors may play a role in worsening the nutritional status of patients with cerebrovascular accidents. Although we can not exclude that some patients may be undernourished before the acute event of stroke^{9 12} it is likely that nutritional wasting is associated with hospitalisation in acute wards, since the proportion of malnutrition has been found to increase during hospital stay.^{9 12}

It is of interest that nutritional parameters were found to have deteriorated in acute stroke patients with swallowing disorders, despite aggressive early enteral nutrition providing a total number of calories greater than that calculated as necessary by calorimetry.¹² Thus, it can be anticipated that a lack of/or insufficient calorie intake in acute ill patients, particularly those with clinical complications, may lead to malnutrition. Unless the causes of malnutrition and the role of therapeutic nutritional intervention in acute stroke patients is well understood, a high prevalence of malnutrition among rehabilitation patients will not be surprising. Fortunately, during hospitalization in rehabilitation wards, malnutrition has been found to be reversible if adequate nutritional therapy is provided.⁷

This investigation demonstrates that self-feeding is not automatically associated with normal food ingestion as an inadequate food intake can be present in a large population of self-feeding rehabilitation patients with stroke. The inadequacy of food intake is evident in more than 75% of malnourished patients and also in many of normally nourished ones. In malnourished patients the inadequacy of alimentation is nearly global because it regards calorie, protein and carbohydrate intakes. The observed inadequate food intake was not due to an excess in body energy requirements because the majority of patients with stroke in this study were normo- or hypometabolic, but to a real low food assumption. It is possible that dysphagia, paralysis of the extremities, depression, apraxia, agnosia or neglect contributed to reducing food intake by the patients in this study.²³

The clinical consequences of food inadequacy are that malnourished patients become more and more

malnourished and normally nourished patients are at risk of developing malnutrition.

One practical correlate of this study is that resting energy needs can be initially set at 1300 kcal/day in malnourished patients and at 1500 kcal/day in normally nourished ones. These values may be very useful in a clinical setting for planning nutritional therapy when it is not possible to weigh the patients or to measure their resting energy consumption directly, in the absence of a reliable theoretical formula to predict energy consumption. This study shows that three quarters of malnourished patients and about half of normally nourished ones have a negative nitrogen balance, indicating the presence of increased endogenous protein catabolism. It is likely that reduced protein intake is the main factor inducing the excess of endogenous protein breakdown since 83% of the negative nitrogen balances occurred in patients with a protein intake lower than 0.8 g/kg/daily. Two other factors may also contribute to nitrogen imbalance, mainly in patients with a diagnosis of malnutrition. One factor is the observed reduced carbohydrate ingestion that is known to spare protein utilization. The second factor may be tissue peripheral insulin resistance induced by immobility.²⁴ It is particularly noteworthy that insulin resistance has been demonstrated to be proportional to immobility duration.²⁵ The main clinical consequences of the negative nitrogen balance may be: a) increased muscle wasting because of increased gluconeogenesis, b) impaired immunocompetence,²⁶ c) asthenia with a reduction in the patients ability to participate in rehabilitation therapy.

An interesting result of our study is that carbohydrate intake in malnourished patients was lower than the minimum amount necessary for brain and other glucose dependent tissue functions.

Low carbohydrate intake may explain the significant lower respiratory quotient (RQ) in the malnourished than in the normally nourished patients, meaning that organ and tissue metabolism in the former group is oriented towards the preferential utilisation of lipids with consequent reduced biochemical and mechanical cellular efficiency.

The contribution of ingested carbohydrates to daily energy intake was lower than recommended (48% instead of $\geq 55\%$)²⁰ meaning that the spontaneous alimentary regimen of patients with stroke may not be qualitatively well-balanced. This is particularly evident when considering that the lipid calorie contribution to daily energy intake was high (33 — 35% instead of recommended value < 30%).²⁰ The dietary analysis

conducted on food diaries showed that low carbohydrate intake by patients was due to a low amount of carbohydrate-rich foods such as pasta and bread, while the high fat intake was attributable to dairy products and cured meat (mainly ham) that patients preferred to meat/fish. It is unlikely that the hospital diet contributed to the above unbalanced diet of the patients since, in our institutes, the carbohydrate content of meals is planned to offer at least 55% of the daily energy intake and a maximum of 30% of lipids are provided. We are not, at present, able to understand why patients with stroke reduce their intakes in carbohydrates, while we suspect that, as regards the lipids, dairy products and ham are chosen because they are soft and, consequently, they require less co-ordination in mastication and deglutition.

In clinical practice, low carbohydrate ingestion is a dangerous practice for at least three reasons. First, it may deteriorate brain dysfunction in patients with cognitive and motor impairments; second it causes an increase of gluconeogenesis, the biochemical process that may induce alterations in both muscle and plasma aminograms. Aminoacids normally pass the blood — brain barrier and compete with each other for entry into the brain ²⁷ to be partly transformed into neurotransmitters. Third, reduced glucose availability may induce a reduction in acetylcholine synthesis, as glucose normally provides the acetyl moiety of acetylcholine. There is considerable evidence to suggest that the positive effects of glucose on learning, memory, and other cognitive functions are mediated via acetylcholine.²⁸

We therefore speculate that reduced carbohydrate intake in patients with stroke may interfere with brain neurotransmission directly and/or indirectly by inducing alterations in tissue/plasma aminograms.

In conclusion, the prevalence of malnutrition is high among self-feeding rehabilitation patients with stroke and, more importantly, patients' actual food intakes can be greatly inadequate for their body needs.

Clinical implications

A number of clinical and ethical practices may be derived from this study.

1. Every patient with stroke should be routinely evaluated both for his/her nutritional status and adequacy of food intake.
2. It is necessary that physicians/physiatrists and

care-givers become aware of the nutritional problems induced by stroke. A nutritional survey of stroke should be routinely begun in the acute services. Nutritional parameters to diagnose malnutrition are very easy to use at the bed side.

3. Hospital managers should plan the purchase of a weighing-chair and skinfold calipers, the costs of which are very low compared to the cost of a single patient's prolonged stay in hospital (with or without complications).

Riassunto

Prevalenza della malnutrizione ed inadeguata assunzione alimentare in pazienti con stroke in fase riabilitativa.

Obiettivo. La malnutrizione causa una riduzione del recupero funzionale ed un allungamento dei tempi di degenza dei pazienti con stroke in fase di riabilitazione.

Scopo. Determinare la prevalenza della malnutrizione e l'adeguatezza degli introiti alimentari in pazienti con stroke, autonomi sotto il profilo alimentare.

Metodi. Sono stati considerati 150 pazienti (85 maschi e 65 femmine; 60±11 anni; 30±10 giorni dall'evento acuto; 45,3% con emiplegia destra e 54,7% con emiplegia sinistra. Ogni paziente è stato sottoposto al seguente protocollo di indagine: a) valutazione dello stato nutrizionale mediante misure antropometriche (peso, altezza, spessore della plica tricipitale, circonferenza muscolare del braccio) e misure biochimiche (albumina sierica e conta dei linfociti totali nel sangue); b) misura del consumo energetico a riposo (CER con calorimetria indiretta); c) quantificazione degli introiti calorici e dei macronutrienti (carboidrati=CHO; proteine=Prot, lipidi=Lip) mediante diario alimentare di 3 giorni; d) misura dell'azoto escreto nelle urine delle 24 ore per calcolare il bilancio azotato (BA).

Criteri di malnutrizione: 1) riduzione del peso corporeo abituale preictus ≥10%, ma con peso corporeo attuale inferiore al peso corporeo desiderabile; 2) riduzione del peso corporeo abituale preictus ≥5% più uno o più dei seguenti indicatori di malnutrizione: area muscolare del braccio <5° percentile e/o albumina sierica <3,5 g/dl e/o linfociti totali <1500 nmm³. Criteri di inadeguatezza degli introiti calorici: 1) calorica=kcal-I≤110% CER; 2) proteica=Prot<0,8 g/kg/die; 3) carboidrati= CHO<140 g/die; 4) lipidica=Lip<0,5 g/kg/die.

Risultati. In base ai criteri adottati 45 pazienti sono risultati malnutriti (=30% della popolazione studiata). Sono stati osservati: kcal-I ≤110% CER in più del 60% di tutti i pazienti; Prot<0,8 g/kg/die nel 50%; CHO<140 g/die nel 38,6%; Lip<0,5 g/kg/die nel 19%. Il bilancio azotato è risultato negativo in più del 56% dei pazienti. Queste inadeguatezze sono risultate distribuite e più marcatamente evidenti nella popolazione dei malnutriti rispetto ai normonutriti.

Conclusioni. La prevalenza di malnutrizione nei pazienti con stroke è alta. Gli introiti calorici e di macronutrienti possono essere insufficienti anche in pazienti con stroke normonutriti. Lo studio dimostra che la presenza di un'osservabile autonomia ali-

mentare in questi pazienti può non essere sinonimo di un normale apporto calorico. Gli Autori concludono auspicando l'introduzione di un'indagine nutrizionale in pazienti con stroke in fase riabilitativa.

Parole chiave: Emiplegia, riabilitazione - Autonomia alimentare - Malnutrizione.

References

- Weinsier RL, Hunker EM, Krumdieck CL, Butterworth CE. Hospital malnutrition, a prospective evaluation of general medical patients during the course of hospitalization. *Am J Clin Nutr* 1979;32:418-26.
- Buzby GP, Mullin JL, Mathews DC, Hobbs CL, Rosato EF. Prognostic nutritional index in gastrointestinal surgery. *Am J Surg* 1980;139:160-7.
- Mullin TJ, Kirkpatrick JR. The effect of nutritional support in immune competency in patients suffering from trauma, sepsis or malignant disease. *Surgery* 1981;90:610-5.
- Ashkenazi J, Weissman C, Rosenbaum SH, Hyman AI, Milic-Emili J, Kinney JM. Nutrition and the respiratory system. *Crit Care Med* 1982;10:163-72.
- MacFadyen BY, Dvoric SJ, Ruberg RL. Management of gastrointestinal fistulas with parenteral hyperalimentation. *Surgery* 1972;74:100-5.
- Mullholland JH, Tvi C, Wright AH, Vini V, Shafiroff B. Protein metabolism and bedsores. *Ann Surg* 1943;118:1015-23.
- Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW. Prolonged length of stay and reduced functional improvement rate in malnourished stroke rehabilitation patients. *Arch Phys Med Rehabil* 1996;77:340-5.
- Gariballa SE, Parker SG, Taub N, Castleden CM. Influence of nutritional status on clinical outcome after acute stroke. *Am J Clin Nutr* 1998;68:275-81.
- Axelsson K, Asplund K, Norberg A, Alafuzoff I. Nutritional status in patients with acute stroke. *Acta Med Scand* 1988;224:217-24.
- Axelsson K, Asplund K, Norberg A, Eriksson S. Eating problems and nutritional status during hospital stay of patients with severe stroke. *J Am Diet Assoc* 1989;89:1092-6.
- Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW. Malnutrition in stroke patients on the rehabilitation service and at follow up: prevalence and predictors. *Arch Phys Med Rehabil* 1995;76:310-6.
- Davalos A, Ricart W, Gonzalez-Huix F, Soler S, Marrugat J, Molins A *et al.* Effect of malnutrition after acute stroke on clinical outcome. *Stroke* 1996;27:1028-32.
- Newmark SR, Sublett D, Block J, Geller R. Nutritional assessment in a rehabilitation unit. *Arch Phys Med Rehabil* 1981;62:279-82.
- Chumlea WC, Roche AF, Steinbaugh ML. Estimating stature from knee height for persons 65-90 years of age. *J Am Geriatr Soc* 1985;33:116-20.
- Hopkins B. Assessment of nutritional status. In: Shronts EP, editor. *Nutrition support dietetics-core curriculum*. Silver Springs, MD: Am Soc Parenteral Enteral Nutr;1989:15-62.
- Aquilani R, Bovio G, Segagni S. Il dispendio energetico a riposo in pazienti con insufficienza renale cronica. *Riv Ital Nutr Parent Ent* 1994;12:151-6.
- Ferranini E. The theoretical bases of indirect calorimetry: a review. *Metabolism* 1988;37:287-301.
- Frayn KN. Calculation of substrate oxidation rates *in vivo* from gaseous exchange. *J Appl Physiol* 1983;55:626-34.
- Harris JS, Benedict FG. A biometric study of basal metabolism in man. Washington, DC: Carnegie Institute, 1919 (Carnegie Institution of Washington, publication N 279).
- Tabelle di composizione degli alimenti. Istituto Nazionale della Nutrizione, 1989.
- Robin AP, Greig PD. Basic principles of intravenous nutritional support. In: *Clinics in chest medicine*. Philadelphia: WB Saunders Company 1986;7(1):29-39.
- Gasull MB, Cabré, Vilar L, Alastrue A, Montserrat A. Protein energy malnutrition: an integral approach and simple new classification. *Hum Clin Nutr* 1984;38:418-31.
- Teasell RW, Finestone HM, Greene-Finestone LS. Dysphagia and nutrition following stroke. In: Teasel RW, editor. *Physical medicine and rehabilitation: State of the art reviews, long-term consequences of stroke*. Philadelphia: Hanley and Belfus, 1993.
- Dolkas CB, Greeleaf JE. Insulin and glucose responses during bed rest with isotonic and isometric exercise. *J Appl Physiol* 1977;43:1033-8.
- Lipmann RL, Schnure JJ, Bradly EM. Impairment of peripheral glucose utilization in normal subjects by prolonged bed rest. *J Lab Clin Med* 1970;76:221-30.
- Newsholme EA, Parry-Billings M. Properties of glutamine release from muscle and its importance for the immune system. *J Parent Ent Nutr* 1990;4(5):635-75.
- Laterra J, Keep R, Betz AL, Goldstein GW. Blood-brain cerebrospinal fluid barriers. In: Siegel GJ, Agranoff BW, Albers RW, Fisher SK, Uhler MD, editors. *Basic neurochemistry- molecular, cellular and medical aspects*. 6th ed. Philadelphia: Lippincott-Raven 1998.
- Ragozzino ME, Unick KE, Gold PE. Hippocampal acetylcholine release during memory testing in rats: augmentation by glucose. *Proc Natl Acad Sci USA* 1996;93:4693-8.