Original Article

Nutritional Repercussion in Advanced Heart Failure and its Value in Prognostic Assessment

Luís Guilherme Veloso, Mucio Tavares de Oliveira Junior, Robson Tadeu Munhoz, Paulo Cesar Morgado, José Antonio Franchini Ramires, Antonio Carlos Pereira Barretto

Instituto do Coração do Hospital das Clínicas - FMUSP - São Paulo, SP - Brazil

Objective

To analyze the nutritional repercussion in heart failure and its relations with left ventricular dysfunction and mortality.

Methods

A series of nutritional parameters in a group of 95 patients with advanced chronic heart failure, arising out of dilated cardiomyopathy and age < 65 years old, without concomitant diseases was studied. The duration of symptons, final diastolic diameter and left ventricular ejection fraction were verified. The nutritional assessment, included the ideal percentage of weight the triceps skin fold thickness, percentiles of circumference of muscular mass of the arm, the albumin serum levels and the lymphocytes global count.

Results

The nutritional situation was alterated in 45.3% to 94.7% of the patients in accordance to the assessment parameter used. There was neither correlation between the nutritional parameters and the length of symptoms, nor with the ventricular dysfunction level. That group of patients had a homogenous evolution, and 75.8% of them died in an average time of 21.86 weeks. The left ventricular diastolic diameter and ejection fraction did not allow for the prediction of survival. A diminished body mass identified a group with higher risk of death. The ideal percentage of the body mass was predictive of survival (p=0.0352), the patients with less than 80% of ideal weight had a higher relative risk of death of 1.99 (1.12–3.02) (p=0.0132).

Conclusion

Malnutrition is frequent in patients with advanced heart failure and dilated cardiomyopathy. The reduced body mass was a better predictor of survival than the left ventricular ejection fraction in patients under advanced stage of myocardial compromising.

Key words

malnutrition, dilated cardiomyopathy and advanced heart failure

Mailing address: Antonio Carlos Pereira Barretto - Rua Piave, 103 - 05620-010 - São Paulo, SP - Brazil
E-mail: pbarreto@cardiol.br, pereira.barretto@incor.usp.br
Sent for publishing on 04/19/2004
Accepted on 01/14/2005

Since the beginning of medicine, malnutrition is acknowledged as a manifestation associated to congestive heart failure (CHF), especially in its more advanced stages. Among the list of classical manifestations of cardiac diseases we find many levels of protein-caloric depletion, even the extreme clinical features, generically called cardiac cachexia[1,2].

The clinical and experimental data available indicate a cause-effect relation between CHF and malnutrition[3,4]. There are questions about which mechanisms lead to malnutrition and the importance of each mechanism in the maintenance and aggravation of the features[5,6].

Along with the questions concerning the physiopathology, there are others on the assessment way. The nutritional assessment, which is usually done in hospitalized patients is inaccurate and can use many parameters to achieve a general impression on the existence or not of protein-caloric malnutrition. The key point to determine the malnutrition level, as well as the best assessment parameters, has not been properly defined and standardized yet[7-10].

This study had as objective to assess the behavior of many indexes of malnutrition status in a group of chronic CHF carriers as a consequence of left ventricular dysfunction due to dilated cardiomyopathy, in an advanced stage of the disease. It also aimed at verifying whether the compromising level of nutritional parameters has any relation with the time of evolution of its symptoms, if the intensity of compromising of indicators of the nutritional status of the disease is related to the level of left ventricular systolic dysfunction, and if its changes have a prognostic value in advanced chronic CHF.

Methods

The nutritional status of 95 carriers of congestive heart failure due to dilated cardiomyopathy in functional class III/IV, hospitalized at Hospital Auxiliar de Cotoxó (HCFMUSP) for compensation, was assessed.

The patients were selected among those 1,176 hospitalized during the 12 months of sample selection for the study, from which 412 were carriers of congestive heart failure.

The exclusion criteria of the study were situations that could cause changes in the nutritional situation of the patients or modify the natural history of the disease: age < 15 or > 65 years old; valvar dysfunctions subjected to surgical corrections; coronary failure; symptomatic cardiac arrhythmias; diastolic blood pressure at admission > 105 mmHg, diabetes mellitus; creatinine levels

> 2.0 mg/dl; chronic pneumopathy; degenerative chronic diseases; neoplasias; AIDS diagnosis; previous alcoholic ingesta > 100 ml of distilled spirits or 600 ml/day of beer and use of illegal drugs. Based on those criteria 312 patients were excluded. Other five patients later excluded for not having returned to the ambulatory.

The data from the clinical history and the physical exam were valued in the 95 patients for the assessment of the functional situation and the duration of the disease. All of them were submitted to a cardiologic assessment through an echocardiographic study, from which the measurement of left ventricular diastolic diameter (LVDD) was valued and the left ventricular ejection fraction (LVEF) assessed through the cube method.

All patients, in the absence of contraindications, were discharged with prescription of digital, diuretic and ACE inhibitors. In patients with renal insufficiency or in those who showed side effect with ACE inhibitor, hydralazina and nitrate were prescribed.

For the assessment of the nutritional status, anthropometric and laboratorial parameters, and determination of immune competence were considered[11]. The adopted parameters were chosen due to their better standardization, better reproducibility, feasibility and cost.

Besides those objective indicators, adopted for statistic analysis, the patients were classified in malnourished, normal or obese, based on their physical aspect, also taking into consideration the skin turgor and elasticity, the thickness and color of the hair and *facies*.

All patients were measured and weighed. The percentage of ideal weight was estimated as percentage in relation to the *standard* weight expected for the population of same sex and height, based on the tables of *American Society of Actuaries*, which are modified for median build and measurements without clothes[12]. The body weight obtained was compared through percentage to the ideal body weight from the tables, by adopting the criteria proposed by Blackburn et al.[13] for classification of results: normal (over 90% of ideal weight), mild nutritional compromising (between 80 and 90% of ideal weight), moderate nutritional compromising (between 70 and 79% of ideal weight) and important nutritional compromising (less than 70% of ideal weight).

The thickness in millimeters of the triceps skin fold (TSF) was obtained in accordance to the technique described by Jelliffe[14], by means of a Lange (*Cambridge Scientific Industries, Cambridge, Maryland*) plicometer or *caliper*. In the middle point of non-dominant arm (between the acromial process and the olecranon), in sitting position, with the arm freely stretched along the body, a fold of skin is pinched with the fingers where the *caliper* is applied, at a depth equal to the thickness of the skin fold, being verified the measurement 2 seconds after the total pressure is exerted by the *caliper* on the skin; the measurement is repeated twice, and the mean of the results is taken. The value obtained is compared with standardized tables[15], by estimating the level of compromising of adipose reserves in terms of the percentile in which the obtained value is situated in relation to population measurements: normal (over percentile 50), mild compromising (percentiles 25 to 50), moderate compromising (percentiles 10 to 24) and important compromising (below percentile 10).

The muscular circumference of the arm, in centimeters, was calculated from the measurements of the arm circumference and the thickness of the triceps skin fold, using the formula proposed by Jelliffe[14]: c2 = cl – 3.14s, in which **c2** is the muscular circumference, **c1** the arm circumference and **s** is the thickness of the triceps skin fold, in centimeters. The arm circumference is measured in the middle point between the acromial process and the olecranon.

Similarly to the thickness of the triceps skin fold, the values obtained were classified in percentiles, through the standardized tables for sex and age elaborated by a Frisancho[16] from population data of HANES I (*United States Health and Nutritional Examination Survey,* 1971-74) study. From the percentile obtained, the level of compromising observed was defined in relation to that parameter: normal (over percentile 50), mild compromising (percentiles 25 to 50), moderate compromising (percentiles 10 to 24) and important compromising (below percentile 10).

The serum level of albumin at admission was determined through bromocresol green method. In terms of albumin, the nutritional compromising level was classified within the following intervals[11,12]: normal (over 3.5 g%), mild compromising (2.8 to 3.5 g%), moderate compromising (2.1 to 2.7 g%) and important compromising (below 2.1 g%).

At admission the lymphocytes count in peripheral blood was elaborated, used was a nutritional parameter, expressing compromising of the immuno system[11]. That parameter was also subdivided in four intervals: normal (over 2,000 lymphocites/mm^3), mild compromising (1,200 to 2,000 lymphocytes/mm^3), moderate compromising (800 to 1,199 lymphocytes/mm^3) and important compromising (below 800 lymphocytes/mm^3)[11].

In table I is shown the main characteristics of the studied population.

It was tried to verify whether there was a relation between the evolution time of CHF and the level of left ventricular dysfunction with the nutritional parameters, by determining the linear correlation coefficient of Pearson among the variables analyzed.

For the analysis of survival two methods were used: estimate of Kaplan-Meier: The estimate of survival observed in the sample studied, which does not depend on any parameter. Analyzed along with the non-parametric test of Log-Rank and the Semi-Parametric model of proportional risks of Cox: suitable for the study of continuous variables, by means of presupposition that the survival curves do not cross, which means that the risk is proportional during the whole time interval studied. Besides making possible to indicate which variables are important in the survival prediction of patients, it allows for obtaining the risk rate between two levels of a given variable. The significance level of 5% was adopted in this study.

Results

The subjective analysis of nutritional status classified 23/95 (24.2%) patients as malnourished, 62/95 (65.3%) as normal and 10/95 (10.5%) as obese. The main results from the nutritional assessment are shown in table II.

Except the arm muscular circumference, most of variables analyzed assessed the nutritional status as normal or with mild intensity changes.

Only the percentile of the arm muscular circumference showed a correlation with the evolution time of CHF (r=-0.2105, p=0.0355). There was no correlation among the other variables of assessment of nutritional status and the evolution time of CHF symptoms prior to the inclusion in the study: Percentage of ideal weight (r=0.0797 p=0.4306); Percentile of triceps skin fold

Table I - Main characteristics of the patients (prevalence and percentage in the casuistry on of numeric variables, mean and standard deviation)

Variable	Values
Age (years old)	47.35±12.96
Sex (male)	68 (71.6%)
History time (months)	37.75±41.58
Idiopathic dilated cardiomyopathy	58 (61.05%)
Chagas' Disease	34 (35.78%)
Peripartum Cardiomyopathy	3 (3.15%)
Functional Class III/IV	60 (63.15%)/ 35 (36.84%)
LVDD (mm)	72.65±8.56
LVEF (%)	34.84±6.65%
Deaths (1 year)	72 (75.78%)
Inotropic suport	27 (28.43%)
Re-hospitalizations	33 (34.73%)

LVDD - left ventricular diastolic diameter; LVEF - left ventricular ejection fraction.

Table II - Nutritional assessment (incidence in percentage)

	Nutritional changes			
	Normal	Mild	Moderate	Intense
Percentage of ideal weight	54.73	25.26	11.57	8.42
Triceps skin fold	11.57	36.84	18.94	32.63
Arm muscular circumference	5.26	28.42	4.21	62.10
Albuminemia	50.52	34.73	11.57	3.15
Lymphocytes count	42.10	33.68	16.84	7.36

(r=-0.0148 p=0.8842); Percentile of arm muscular circumference (r=-0.2105 p=0.0355); albumin (r=0.0152 p=0.8807) and lymphocytes (r=-0.1171 p=0.2458).

Only the left ventricular final diastolic diameter had a correlation coefficient of 0.2768 with the percentage of ideal weight (p=0.0058). Except for that finding, there was no correlation among any of the nutritional status indicators analyzed and the myocardial compromising level (tab. III).

In the survival analysis only the percentage of ideal weight was a predictor of mortality, both through the test of Log-Rank (p=0.0132) and by the model of Cox (p=0.0168). For the other variables studied, the statistic analysis did not identify them as prognostic: Thickness of triceps skin fold (p=0.7233 and 0.0801), arm muscular circumference (p=0.9723 and 0.3313), albuminemia (p=0.4897 and 0.4246) and lymphocytes count (p=0.4529 and 0.5303) respectively for the test of Log–Rank and the model of Cox.

Table III - Correlation coefficients of Pearson (r) and significance (p) among parameters of nutritional assessment and left ventricular ejection fraction (LVEF) and diastolic diameter (LVDD)

		x LVDD	x LVEF
% ideal weight	r	0.2768	0.0366
	p	0.0058	0.7176
PERCTSF	r	0.1737	0.1197
	p	0.0872	0.2354
PERCAMC	r	-0.1310	0.1816
	p	0.1985	0.0706
Albumin	r	0.1694	-0.0488
	p	0.0954	0.6294
Lymphocytes	r	0.0432	-0.1003
	p	0.6726	0.3208

% ideal weight- percentage of ideal weight; PERCTSF- percentile of the thickness of triceps skin fold; PERCAMC- percentile of arm muscular circumference.

The carriers of moderate or important change of ideal weight, with the weight observed below percentage 80, had higher mortality, with a risk rate of 1.99 (CI 95%; 1.12 to 3.02) (fig. 1).

The non-parametric analysis by Kaplan-Meier and by the test of Log-Rank, and the semi-parametric analysis by the model of proportional risks of Cox showed that both the fraction of ejection and the LV diastolic diameter were not significant in the prediction of survival time of the patients studied.

Discussion

In a population of patients with advanced heart failure (severe), due to dilated cardiomyopathy (Idiopathic 61%, chagasic 36% and peripartum 3%) was detected a malnutrition level in 45.3% to 94.7% of the patients, according to the assessment parameter used. The presence of malnutrition did not have any relation with the duration of clinical manifestations or with the intensity of myocardial compromising. However, the malnourished patients, when assessed through the reduction of ideal weight, had a higher mortality than those without signs of malnutrition.

The association between the clinical features of cachexia and heart failure is a classic phenomenon and quite frequently observed in clinical practice. Its prevalence varies in accordance with the composition of the studied population, being described in percentage that varied from 13.7 to 61.5%, which seemed to have a relation with the cause of heart failure and a certain relation with the duration of symptoms and the intensity of heart failure[7-10,17].

Such differences in the incidences can also be related to the different ways of assessment of nutritional status and to the lack of a single definition on what malnutrition and cachexia is.

Among the most frequently used measures for the assessment of nutritional status, some are easy to obtain, such as weight and height, which allow us for assess the nutritional status, by comparing the obtained values with those from tables or assessing the body mass index. We used the tables from *American Society of Actuaries*[12]. Due to the simplicity of obtaining, we used the percentage of ideal weight, the measurement of triceps skin fold, the arm muscular circumference and laboratory exams, through the dosage of albuminemia and lymphocytes count[11-14], in our studies to assess the nutritional status.

All those variables have been used in many studies that proposed to assess the nutritional status in different diseases. No single measurement is ideal, and the use of many of them to achieve a

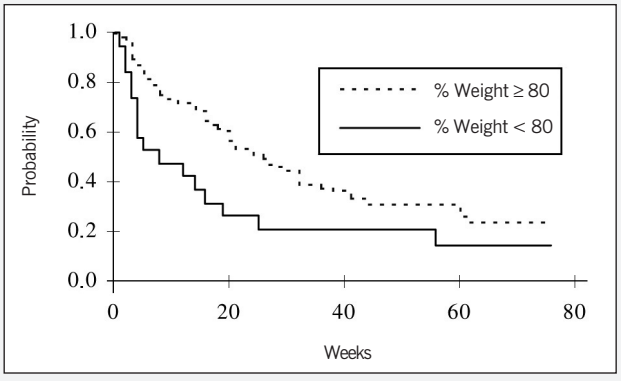

Fig. 1- Survival estimates (Kaplan-Meier) according to the percentage of ideal weight.

real assessment of the nutritional status of a patient is important.

The normal weight (ideal weight) table, adopted as a reference, was the one recommended by Jelliffe[14]. The main concern about the use of such table is the fact that it comes from a population study carried out in the USA some decades ago. However, in the country itself where the table was elaborated by the American Society of Actuaries, authors already warned about the fact that the studied population had an income below the average and mixed ethnical composition, which does not properly reflect the majority Anglo-Saxon biotype of the nation[18]. Despite the absence of a population study with a similar dimension in our milieu, comparative analyses with national more limited numerically series, validate the use of that table in Brazil. Other American tables more recently elaborated, as those by Frisancho et al., seem to have a better correlation with the biotype of small Brazilian populations studied[16,19,20].

Although our sample had showed a wide dispersion of values in relation to the duration of CHF symptoms, a correlation between that variable and the different indicators of nutritional status analyzed was not observed. Although the percentile of arm muscular circumference had shown some significance in the analysis by the coefficient of Pearson, with p=0.0355, we considered the obtained correlation coefficient (r=-0.21) as very low, allowing for, at most, perceiving a tendency that the patients with a longer period of congestive manifestations show a greater compromising of the skeletal muscular mass. However, the low correlation coefficients observed in the analysis of other variables suggest that, at the pre-end-stage of CHF, the duration of congestive symptoms is not a determining factor of the nutritional compromising observed.

The analyzed indicators of nutritional status had low correlation coefficients with LVEF determined by the echocardiogram. Only the percentile of arm muscular circumference reached values close to significance, with p=0.0706. However, the low value of correlation coefficient (0.18) and the disagreement on that was observed in all remaining variables reinforces the impression that the ejection fraction, analyzed through this method, has no correlation with the nutritional status compromising. That result is compatible with previous observations[7] in another sample with similar methodology, and also with the results from the study by Carr et al.[10], which analyzed nutritional indicators by utilizing the hemodynamics and the two-dimensional echocardiogram, estimating the ejection fraction through the Simpson's planar formula, as methods for the study of myocardial function.

Left ventricular dimension, echocardiographically assessed by LVDD, showed a correlation coefficient of 0.2768 with a significant percentage of ideal weight (p=0.0058). We also noted values of p close to 0.05, although non-significant, in the correlation of LVDD with the percentile of triceps skin fold and with albuminemia, which could indicate a tendency. However, the correlation coefficients were lower than 0.20. Besides, such data seem to differ from a previous observation[8]. The final impression provided by that set of data is that there was no clear relation between the left ventricular dimension and the nutritional compromising in the analyzed sample.

Those results suggest that the nutritional repercussion in advanced heart failure, despite resulting from left ventricular contractile dysfunction as common etiologic factor, has in each individual factor other than those that transcend the simple compromising of the cardiac pump as main determinants. The nutritional compromising could maybe be better understood at the extent of the complex peripheral adaptation and the neurohormonal response to myocardial failure.

The increase of cytokine level, such as the tumor necrosis factor-alpha and interleukin-6 can partly explain those findings. The cytokines are very high in the advanced forms of the disease and studies showed a relation between the increase of its levels and the loss of weight[16, 21,22].

Our results do not differ from those shown in the literatures that also do not demonstrate having a correlation between the level of malnutrition and the level of ventricular dysfunction[23,24]. Even when analyzed in large populations, such as the big clinical essays, no correlation was verified between the malnutrition level and the cardiac dysfunction level[25,26].

In this severe chronic CHF and dilated cardiomyopathy group, already in the pre-end-stage of the disease, the LVEF and LVDD parameters determined through echocardiography did not have any predictive value in relation to survival time. However, the great homogeneity of the group in relation to those parameters and the seriousness of clinical manifestations can be also included in that observation. It is possible that in this sample uniformly situated at the end of the clinical spectrum of CHF, the left ventricular function indicators have a reduced prognostic value, to the detriment of other clinical and laboratorial variables.

Although in patients with less severe forms of the disease, the ventricular ejection fraction and diameters are identified as prognostic, it is important to emphasize that in the advanced forms of the disease they seem to lose that capacity, as in studies from our Institution, in populations with similar characteristics to those in this analysis, the level of heart compromising assessed through echocardiography did not allow for identify which patients would have worse evolution[27,28].

If the ventricular compromising was not a good predictor of evolution, our study showed that the presence of malnutrition identified a group of patients with a higher potential for bad evolution.

The relative risk of death for the 19 patients with less than 80% of the forecast ideal weight was 1.99, practically twice as expected for the remaining of the sample. The analysis of the survival curves of Kaplan-Meier evinces a greater mortality in patients with a greater reduction of body mass (fig. 1). That group, regarded as carrier of moderate or important change of the nutritional parameter studied, also had a tendency to a greater early mortality. When analyzed was a continuous variable, in the presence of other nutritional indicators, the percentage of ideal weight exerted a significant effect on survival.

Analyzing the results of literature we can generally notice that the punctual assessment of nutritional status has not been identified as a good predictor of evolution of the patients[16, 23-26]. Although the number of publications demonstrating that cachexia would be a predictor of evolution of patients has been increasing, in all of them it was characterized as a weight change (reduction) during the follow-up. The loss of weight of 6% and 7.5%[23-26] was a predictor of mortality. Those studies documented that patients who lost weight after the beginning of the follow-up had worse evolution, regardless of initial nutritional situation. None of the studies, even employing a great number of patients, identified the basal nutritional situation as predictor of prognostic[25,26].

Despite the evidences that the total body mass is a prognostic indicator of CHF in advanced stage, the compromising of caloric reserves (estimated through the percentile of TSF) or skeletal muscular mass (assessed through the percentile of AMC), singly considered, did not show a predictive value for survival in the sample studied. AS in CHF there is a prevailing nutritional compromising of marasmus type – in which the decrease of body mass essentially occurs at the expense of muscular mass and adipose tissue –, it would be expected that those indicators showed a prognostic value, similarly to the analysis of percentage of ideal weight.

The changes in plasmatic albumin or lymphocytes count were not prognostic either.

Those results must be analyzed carefully, as they refer to an extremely severe population, a fact that can have made difficult the identification of mortality predictors. We cannot either exclude the possibility that the lack of significance for the variables studied has been arising out of the small population studied.

It is always important to emphasize that those results cannot be applied to populations with less severe clinical records.

The nutritional situation of the patients is also influenced by socioeconomic factors, and that is another limitation of the study, as those data were not analyzed in that group of patients.

In conclusion, the changes of anthropometric, biochemical, and/or immunologic parameters compatible with moderate or important nutritional compromising are frequent findings in advanced CHF. The subjective assessment of nutritional status shows lower sensitivity for the detection of moderate or important changes of nutritional indicators. The changes of nutritional indicators studied were generally not related to the evolution time of CHF symptoms or to the level of left ventricular compromising. A moderate or important diminishing of body mass, determined through the percentage of ideal weight, was associated to a shorter survival and showed an independent prognostic value. Other forms of assessment did not show correlation with the prognostic.

References

1. Katz AM, Katz PB. Diseases of the heart in works of Hyppocrates. Br Heart J 1962; 24: 256-64, apud Pittman JG, Cohen P, 1964, p.403.
2. Pittman JG, Cohen P. The pathogenesis of cardiac cachexia. N Engl J Med. 1964; 271: 403-9.
3. Gottdiener JS, Gross HA, Henry WL, Borer JS, Ebert MH. Effects of self-induced starvation on cardiac size and function in anorexia nervosa. Circulation. 1978; 58: 425-33.
4. Alden PB, Madoff RD, Stahl TJ, Lakatua DJ, Ring WS, Cerra FB. Left ventricular function in malnutrition. Am J Physiol. 1987; 253 (suppl H): H380-7.
5. Ansari A. Syndromes of cardiac cachexia and the cachetic heart: Current perspective. Prog Cardiovasc Dis. 1987; 30: 45-60.
6. Morrison WL, Edwards RHT. Cardiac cachexia - We know about the mechanisms but not how to reverse it. B Med J. 1991; 302: 301-2.
7. Velloso LGC, Csengeri LF, Alonso RR et al. Desnutrição na miocardiopatia dilatada: correlação com índices ecocardiográficos da função ventricular esquerda. Arq Bras Cardiol. 1992; 58: 189-92.
8. McMurray J, Abdullah I, Dargie HJ, Shapiro D. Increased concentrations of tumor necrosis factor in "cachetic" patients with severe chronic heart failure. Br Heart J. 1991; 66: 356-8.
9. Bollet AJ, Owens S. Evaluation of nutritional status of selected hospitalized patients. Am J Clin Nutr. 1973; 26: 931-8.
10. Carr JG, Stevenson LW, Walden JA, Heber D. Prevalence and hemodynamic correlates of malnutrition in severe congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol. 1989; 63: 709-13.
11. Bernard MA, Jacobs DO, Rombeau JL. Suporte nutricional e metabólico de pacientes hospitalizados. Rio de Janeiro, Guanabara, 1988. Cap.3, p.24-42: Avaliação nutricional.
12. Jelliffe DB, Jelliffe EFP. Community nutritional assessment - with special reference to less technically developed countries. New York, Oxford University Press, 1989, p.56-550.
13. Blackburn GL, Bistrian BR, Mains BS. Nutritional and metabolic assessment of the hospitalized patients. J Parent Ent Nutr. 1977; 1: 11-22.
14. Jelliffe DB. The Assessment of the nutritional status of the community - with special reference to field surveys in developing regions of the world. Geneve, WHO, 1966 - WHO Monograph Series, n.53, p.50-242.
15. Torosian MH, Mullen JL. Nutritional assessment. In: Kaminski, M.V., ed. Hyperalimentation: a Guide for Clinicians. New York, Marcel Dekker Inc, 1985, p.47-75.
16. Frisancho AR. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr.1981; 34: 2540-5.
17. Anker SD, Rauchhaus M. Insights into the pathogenesis of chronic heart failure: immune activation and cachexia. Curr Opin Cardiol. 1999; 14: 211-16.
18. Gray GE, Gray LK. Anthropometric measurements and their interpretation: Principles, practices, and problems. J Am Dietetic Association. 1980; 77: 534-9.
19. Costa OL, Santos DM, Nespoli CA et al. Padrões de normalidade para medidas antropométricas - estudo sistemático em uma população adulta brasileira. Rev Hosp Clin Fac Med S Paulo. 1987; 42: 49-54.
20. Faintuch J, Pereira M L, Faintuch BL, Machado MCC, Pinotti HW. Novos parâmetros para avaliação nutricional: investigação preliminar. Rev Hosp Clin Fac Med S Paulo. 1983; 38: 164-66.
21. Brink M, Anwar A, Delafontaine P. Neurohormonal factors in the development of catabolic/anabolic imbalance and cachexia. Int J Cardiol. 2002; 85: 111-21.
22. Conraads VM, Bosmans JM, Vrints CJ. Chronic heart failure: an example of a systemic chronic inflammatory disease resulting in cachexia. Int J Cardiol. 2002; 85: 33-49.
23. Horwich TB, Fonarow GC, Hamilton MA et al. The relationship between obesity and mortality in patients with heart failure. J Am Coll Cardiol 2001; 38: 789-95.
24. Anker SD, Coats AJS. Cardiac cachexia. A syndrome with impaired survival and immune and neuroendocrine activation. Chest. 1999; 115: 836-47.
25. Davos CH, Doehner W, Rauchhaus M et al. Body mass and survival in patients with chronic heart failure without cachexia: the importance of obesity. J Cardiac Fail. 2003; 96: 29-35.
26. Anker SD, Negassa A, Coats AJS et al. Prognostic importance of weight loss in chronic heart failure and the effect of treatment with angiotensina-converting enzyme inhibitors: an observational study. Lancet. 2003; 361: 1077-83.
27. Canesin MF, Giorgi D, Oliveira Jr MT et al. Ambulatory blood pressure monitoring of patients with heart failure. A new prognosis marker. Arq Bras Cardiol. 2002; 78: 83-9.
28. Del Carlo CH, Pereira-Barretto AC, Cassaro-Strunz C, Latorre MRDO, Ramires JAF. Serial measure of cardiac troponin T levels for prediction of clinical events in decompensated heart failure. J Cardiac Fail 2004; 10: 43-48.