

Nourishing Aspects in Heart Failure

P. Claude¹, S. Jermy²

Department of Psychology, University of Houston-Clear Lake, USA^{1,2}.

Abstract— Heart disappointment (HF) is a complex clinical disorder, of a dynamic character, that constrained prognostic factor and showed by different extracardiac angles. It speaks to a genuine and developing general medical issue around the world, both for its high predominance and the seriousness of its clinical indications, being the last basic pathway of generally maladies. Undernutrition is frequently connected with HF, particularly in the later phases of the malady, and may constantly achieve heart cachexia, an extreme indication identified with poor clinical forecast. Nourishing consideration is acknowledged as a vital and key piece of the treatment of HF and tries to improve the healthful status of the patient, intending to renew vitality holds, expanding skeletal muscle tissue and improving activity limit. Micronutrient insufficiency is basic in patients with HF and its source is by all accounts multifactorial, among which delayed utilization of diuretics, low dietary admission and expanded supplement misfortunes are connected. In this survey the healthful viewpoints will be tended to for HF, with accentuation on the suggestions and dietary proposals.



Keywords— Heart failure, hypertension, coronary illness.

1. Introduction

Heart disappointment (HF) can be characterized as an anomaly of the cardiovascular structure or capacity that prompts diminished ventricular filling and launch limit prompting disappointment of the heart to furnish oxygen at a rate steady with the prerequisites of processing tissues rate [1]. It is a complex clinical disorder, of a dynamic character, that has a constrained prognostic factor and showed by different extracardiac angles, including neuroendocrine initiation and cytokine discharge [2-5].

The HF is a multifactorial issue that includes different organ frameworks in its pathogenesis and unequivocally influences the personal satisfaction of influenced patients [6], causing useful restriction and requiring quick remedial mediation [5]. It speaks to a genuine and developing general medical issue around the world, both for its high commonness and the seriousness of its clinical signs, being the last regular pathway of most maladies [7-9]. Clinical appearances in patients with HF incorporate exhaustion, dyspnea during day by day exercises and paroxysmal nighttime dyspnea, hepatomegaly, prejudice to physical effort, night hack and water maintenance, which can cause pneumonic blockage and fringe edema [9,10]. In ongoing decades, with a superior comprehension of the infection procedure, it turned out to be certain that the neurotic changes include the cardiovascular framework as well as the neuroendocrine, invulnerable, musculoskeletal, hematologic, renal and gastrointestinal frameworks just as the healthful status [11].

Undernutrition is normally found in patients with HF, fundamentally in the most progressive phases of the malady, being connected with an improved danger of difficulties and mortality [12]. The execution of compelling wholesome techniques in HF remains a remedial test in clinical practice. In this audit the dietary viewpoints will be tended to for HF, with accentuation on the suggestions and wholesome proposals.

2. Undernutrition and HF

Undernutrition is an exemplary appearance and is regularly connected with HF. Inside the rundown of basic indications of the illness, shifting degrees of protein-calorie consumption can be found, just as extraordinary cases, conventionally called heart cachexia [13].

Undernutrition can happen because of lacking admission, the changed digestion, the professional fiery state, expanded oxidative pressure and expanded supplement misfortune [6,7], bringing about fit weight consumption (counting indispensable organs, for example, the myocardium itself), with negative ramifications on useful limit, and expanded post-usable inconveniences and mortality [11]. Concentrates with little examples show that absence of hunger may disclose up to half of instances of undernutrition in patients with any constant sickness [14]. Anorexia in HF is a result of diminished admission of supplements or mix of absorptive and metabolic changes (hypermetabolism, hypoxia, expanded vitality use, aggravation) [6,11,15]. In addition, changes in the stomach related tract, for example, gastric pressure and hepatic blockage, cause postprandial sentiment of completion [16]. Unpalatable weight control plans and depletion during encouraging further add to lacking supplement consumption [11].

The loss of supplements is because of the inside brokenness in HF, clarified by hypoperfusion and edema of the entrail. An investigation exhibited an expansion of the ileum divider and rising, slipping, transverse and sigmoid colon of patients with the illness contrasted with their controls [7,14,17]. The edema of the inside circle would be in charge of malabsorption of lipids, protein misfortune and different side effects, for example, queasiness and anorexia [6,15,17]. Moreover, the interminable utilization of diuretics may add to the expanded misfortunes of supplements [10,11].

HF patients had adjustments in the anabolism/catabolism parity coming about because of neurohormonal changes set apart by expanded degrees of catabolic variables (norepinephrine, epinephrine, angiotensin II, cortisol, free radicals and incendiary cytokines) and protection from anabolic hormones, for example, the development hormone (GH) and insulin. These progressions add to an expanded vitality consumption in resting [6,18]. Patients in utilitarian class III and IV demonstrate an expanded basal metabolic rate around 18%, contrasted and sound subjects [19]. The expanded vitality requests for crafted by the respiratory muscles, the hypertrophied myocardium and the hematopoietic framework adds to this expansion in the basal metabolic rate [6, 20].

Weight reduction in patients with HF is set apart by an unbalanced assembly of muscle and fat tissue, protein corruption being a noteworthy change in ailment movement [21, 22]. The decrease of bulk is related with narrow mindedness to early exercise, weariness, less quality, and henceforth crumbling in personal satisfaction [23]. The nearness of undernutrition is a significant prescient factor for decreased survival of patients, paying little respect to significant factors, for example, age, practical class and launch part [6,24]. The occurrence of undernutrition in HF is high and builds emergency clinic mortality. Albeit identified with complex components, undernutrition in patients with HF is a reversible condition with genuine healthful appraisal and checking, early location and appropriate eating regimen [25].

3. Cardiovascular cachexia

Ceaseless maladies by and large influence the healthful status of the patient and undernutrition influences the course of constant sickness, and this relationship is evaluated by the two components. Nourishing decay forced by HF is traditionally known as heart cachexia, whose nearness has significant prognostic ramifications [24,26,27].

Heart cachexia shows up following quite a while of dynamic weakening of the myocardial capacity [28]. The pathophysiological components engaged with the etiology of cachexia related with HF have not been completely explained. It is proposed this is a multifactorial neuroendocrine and immunologic sickness, wherein a mind-boggling balance among anabolic and catabolic procedures can cause weight reduction in patients with HF [29].

It is essential to separate cachexia from undernutrition or anorexia, which are reversible conditions with satisfactory nourishment consumption, which barely happens in cachexia [30,31]. Undernutrition and anorexia are frequently considered as the fundamental driver of cardiovascular cachexia. Notwithstanding, these conditions prevalently produce the loss of fat tissue while cachectic patients experience loss of bulk, fat and bone mass, demonstrating summed up weight reduction [3]. It was discovered that 68 % of HF patients have extreme muscle decay and when the malady is in a propelled stage various them create osteoporosis [28].

The cardiovascular cachexia is described by the power of fit weight misfortune, being brought about by numerous elements, for example the malabsorption of fat, the expansion of the catabolic catecholamines (norepinephrine, epinephrine, cortisol) and phases of alpha tumor putrefaction factor (TNF- α), just as the decrease in the creation of numerous anabolic segments [32]. Countless instruments to repay the inadequate myocardial capacity is initiated to ensure the heart and blood stream, prompting a mind boggling and ceaseless provocative state. The middle people engaged with this procedure incorporate proinflammatory cytokines, catecholamines, cortisol proteins and natriuretic peptides [3,33]. The TNF- α initiation prompts the apoptosis through explicit receptors in the cells surface and enacts protein corruption [34]. The TNF- α is mostly in charge of lessening in blood stream to skeleton muscle tissue, disturbing the endothelial brokenness [35]. The proinflammatory cytokine Interleukin-6 (IL-6) causes the intense stage reaction [36], which upkeep requires an abundance of basic amino acids gave to the detriment of living being protein misfortune [37].

Tentatively, it was discovered that the advancement of cachexia may prompt extra heart changes and fuel of a previous system of HF [3,38,39]. Besides, the decrease of intestinal retention is increasingly clear in patients with cachexia, particularly fat ingestion, and it is recommended that these progressions are associated with its etiology [40].

Because of cachexia changes happen in for all intents and purposes all organs and frameworks. Among the repercussions are cardiovascular variations from the norm, irregularities of the respiratory capacity, stamped decline in muscle and bone mass, diminished capacity to focus and urinary fermentation, diminished injury mending, powerlessness to weight ulcers in out of commission patients, changes in the gastrointestinal tract, with decreased resistance and expanded danger of contaminations [11].

It can regularly be hard to determine cachexia in patients to have HF, as the nearness of an edema influences the appraisal of body weight and other anthropometric estimations. Moreover, the elucidation of research center dietary appraisal is likewise constrained because of the impacts of inordinate extracellular liquid [41].

Without a consensual, all around acknowledged definition, a few definitions are proposed for its determination. Anker et al [24] proposed a definition dependent on the appropriateness of basic, non-edematous, inadvertent weight reduction without other wasteful maladies, 7.5% over the typical load for longer than a half year. Stamped weight reduction in a shorter period could be identified with the compounding of the malady itself and not to invulnerable and neuroendocrine changes that describe cachexia.

Accordingly, it was demonstrated that decrease of 6% of the weight is identified with poor guess, with this standard embraced for finding [42]. At the Consensus Conference on Cachexia, in 2006, a gathering of specialists proposed a definition dependent on the loss of more noteworthy than 5% in under a year during a perpetual sickness, or BMI < 20kg/m², related in any event with three of the accompanying criteria:

1) decline in muscle quality, 2) exhaustion, 3) anorexia, 4) diminished rate of without fat mass and 5) biochemical variations from the norm, for example, irritation, frailty or decreased serum egg white's fixation [30,31]. The commonness of cachexia may change contingent upon the demonstrative criteria utilized and the utilitarian class of HF. Studies have shown rates extending from 16% to 34% [24,42].

Heart cachexia can prompt an overwhelming visualization. After investigation of a survival bend 18 patients with HF in outpatient treatment, an expected death rate of half in cachectic people and 17% for those without this condition was determined [14,24].

4. Obesity

The expansion in BMI raises the danger of creating HF in both genders and paying little respect to other hazard factors [43,44]. This expanded hazard presented by stoutness is credited to hemodynamic adjustments to withstand extreme fat tissue digestion and oxygen utilization is expanded [45]. Cardiovascular yield ascends to the detriment of an expansion in flowing blood volume, prompting a constantly high condition of preload, which supports the increment in ventricular measurements [46].

Moreover, there are confirmations that a greater unsaturated fats aggregation, as long as its heart muscle cells oxidation decline, process known as lipotoxicity, can happen all the more as often as possible in fat people. Lipotoxicity can prompt heart cells apoptosis, other than utilitarian difference in the organ by influencing its contractility [47]. The unsaturated fats increment additionally adds to the presence of insulin obstruction [48], that just as stoutness, was recognized as an indicator of heart disappointment [49].

The Framingham consider that assessed 5,881 patients demonstrated a positive connection among BMI and the frequency of HF, showing the expanded danger of building up the malady in 5% to 7% for people for each one-unit increment in the estimation of typical BMI. Another examination found that corpulent people had a twofold danger of heart decompensation when contrasted with eutrophic ones [43]. Evaluations demonstrate that 15 to 35% of HF patients are large and 30-60% are overweight [50].

Be that as it may, despite the fact that it is viewed as a hazard factor and precipitant of HF, incomprehensibly, heftiness has been distinguished as an indicator of longer survival in patients with the illness, when contrasted with well-fed or malnourished people [51-53]. This "weight mystery" or "invert the study of disease transmission" has been accounted for in other interminable conditions (nephropathy on dialysis, propelled age, endless obstructive aspiratory sickness, liver cirrhosis, propelled malignancy and

AIDS [54,55] and in a few investigations that assessed patients with HF, however this remaining parts dubious [51,55-58].

Patients who are overweight have less entanglements during hospitalizations, lower danger of abrupt passing and better short-and long-haul guess than typical and underweight people [59]. Oreopoulos et al. [60], in a meta-investigation to evaluate the connection between expanding BMI and mortality in patients with HF, announced that in nine observational examinations with 28.209 people overweight and stoutness were related with lower mortality.

This defensive impact given by stoutness in HF is identified with a more noteworthy vitality hold for patients who are powerlessly presented to catabolic changes and hormonal initiation of stress frameworks. In this way, being overweight or corpulent in HF may mirror the metabolic stores have not been utilized, giving more and better resistance to metabolic pressure brought about by the infection [55,61]. Besides, the fat tissue produces receptors for tumor corruption factor (TNF- α), bringing about an expansion of these receptors in fat people. This expansion may have a cardioprotective job, killing and/or lessening the catabolic impacts of TNF- α [61].

Accordingly, to control abundance weight is gainful to anticipate the HF, yet once this is introduced, keeping away from weight reduction diminishes mortality [62]. There is proof that weight reduction in patients with HF is related with lopsided assembly of muscle and fat tissue, with expanded protein debasement [13,22]. There are still questions about the advantage of keeping up a BMI better than average cutoff points, with no formal proposals in regards to the sign of weight reduction or support of high BMI for people with HF overweight.

5. Nourishing Assessment

Taking into account that the nourishing status speaks to a hazard component for the HF event while being a condition related with the malady advancement and movement, the healthful appraisal of those patients must be object of observing so as to forestall the connected dangers.

In spite of the recorded and common snare among HF and undernutrition, isn't yet characterized what might be the best parameter to express the dietary status of patients with the infection. Nourishing analysis in these patients is especially hampered by the nearness of water maintenance, which can veil the assessment result. Anyway, the wholesome status of patients with HF ought to be deliberately evaluated with regards to multidisciplinary care, the advantages of which are settled [10].

The BMI, a parameter routinely used to analyze the dietary status, has diminished affectability to confirm the state of extreme undernutrition among patients with HF [63]. The Mini Nutritional Assessment (MNA) has been recognized as a valuable device for distinguishing patients with HF at high dietary hazard [26].

The triceps skinfold (TSF) likewise gives off an impression of being satisfactory to express the fat hold strategy in these patients. An examination that assessed the prognostic estimation of different anthropometric parameters (weight list, emotional worldwide appraisal, TSF, midsection outline and arm periphery) uncovered that the TSF was the main parameter ready to separate among survivors and non-survivors and an imminent accomplice of patients with HF [64]. The emotional worldwide evaluation is generally depicted as solid and increasingly touchy in patients with HF [65].

The bioelectrical impedance investigation (BIA) has showed up in certain articles as an approach to evaluate weight in patients with HF, yet we question its legitimacy in blocked patients, though the extracellular liquid maintenance may overestimate without fat mass [66]. The hydration status of these patients is variable and there might be a subclinical edema to bargain the body structure investigation. Be that as it may, the BIA could be helpful for deciding the stage point (AF) acquired from estimations of obstruction (R) and reactance (Xc) that are free of the hydration status of the patient. In spite of the fact that their organic criticalness isn't completely comprehended, the AF mirrors the body cell mass, and thus it has been utilized as a nourishing pointer in grown-ups and youngsters [67-69]. Besides, it is perhaps the best marker of cell layer capacity and lower esteems are related with higher morbidity [68,69].

In this way, there are no well-characterized criteria to decide the dietary status in patients with HF, as there is a sure scale to review the various degrees of natural disability [13]. The writing shows that no single strategy can precisely express the healthful status of patients with the malady, and the data acquired from numerous parameters is progressively integrative and corresponding [70,71].

6. Nourishing Recommendations

Nourishing consideration is as of now acknowledged as a fundamental and key piece of the treatment of HF and looks to improve the healthful status of the patient, expecting to renew vitality holds, expanding skeletal muscle tissue and improving activity limit [29,72]. Taking into account that the ailment might be related with healthful deficiencies, anticipation and early mediation can improve the patient's condition and visualization of the ailment [2,3,6,11]. Be that as it may, these restorative estimates should be individualized. The HF is a disorder with a high commonness of different interminable comorbidities, yet most rules are produced for patients with a solitary infection. Nonetheless, the conjunction of different infections, for example, joint inflammation, kidney disappointment, diabetes mellitus or endless lung illness, with the HF disorder ought to require treatment adjustments [73]. Also, one ought to consider the utilization of prescriptions, for example, diuretics and anticoagulants that meddle with the dietary status of certain supplements. The utilization of circle diuretics may prompt potassium, sodium and magnesium consumption notwithstanding hyperglycemia, hyperlipidemia and hyperuricemia [11,72]. The anticoagulants, then again, can cause both mucosal damage in a few pieces of the gastrointestinal tract and encourage blood misfortune through the stomach related tract [74].

Notwithstanding loss of supplements, it has been found in certain examinations that patients with HF have deficient admission of vitality and supplements [75,76]. In this way, the direction and dietary solution ought to be gone before by nourishing determination [65] by the accessible strategies, including sustenance history, and ought not be limited uniquely to hydro - salt confinement [6].

On the off chance that important, liquid limitation ought to associate with 1000-1500 ml, and ought to consistently be made with patients with hyponatremia or symptomatic ones, and for the last mentioned, the observing of body weight ought to be done once a day [5,11]. It is imperative to take note of the presence of liquid maintenance, which might be shown by the unexpected increment of at least two kilos in three days [72].

Energy and macronutrient distribution	Recommendation	Authors
Energy	28 to 32 kcal/kg weight	Bocchi, 2009 [72]; Aquilani et al., 2003 [22]

Carbohydrates	50 to 55% of VCT*	Bocchi, 2009 [72]; Berry e Clarck, 2000 [2]; Heart Failure Society of America, 2006 [80]
Lipids	30 to 35% of VCT	Bocchi, 2009 [72]; Heymsfield et al., 1981 [81]
Proteins	15 to 20% of VCT (1,1g to 2,0g/kg/day)	Bocchi, 2009 [72]; Quinn et al.& Askanazi, 1987 [82] Hernandez 2012 [14]; Aquilani et al., 2003 [22]
Relation cal/g of N**	120 a 160: 1	Quinn & Askanazi, 1987 [82]

* VCT = total caloric value; **calorie/gram of nitrogen

Table 1: Distribution of energy and macronutrients in the diet for individuals with HF

7. Calories and Macronutrients

For the figuring of vitality needs, the handy technique is suggested (calorie/kg dry weight/day), thinking about the wholesome status of the patient, and ought to be utilized around 25-30 kcal/kg/day [6,14,77]. Past examinations show 28kcal/kg weight for patients with sufficient dietary status and 32kcal/kg weight for healthfully exhausted patients [22,72]. Vitality necessities may likewise change as per the practical class of HF. Patients in useful class III and IV show expanded basal metabolic rate around 18%, contrasted and solid subjects [19,72].

The conveyance of macronutrients in the eating routine does not contrast much from the all-inclusive community (Table 1). Be that as it may, in connection to protein consumption, patients with HF have higher necessities than the all-inclusive community, going from 1.1g/kg dry weight/day for patients with typical to 1.5-2.0g/kg dry/day for malnourished patients with cardiovascular cachexia or demonstrating misfortunes in view of nephropathy and/or intestinal malabsorption [10,11,14,22].

For patients with cardiovascular cachexia a vitality admission of 40 to 50 calories/m² body/surface time (around 30 to 40 calories/kg/day) has been proposed, including 1.5 - 2g protein/kg/day [3,29].

It is essential to be careful of hypo-or hyperalimentation. Over the top admission of vitality is identified with expanded physiological pressure and with raised plasma groupings of catecholamines and insulin, bringing about expanded reabsorption of sodium and water, and liver brokenness, exacerbating HF [11,29,78]. The lively substrates abundance acquired by hypercaloric or uneven eating regimens can contribute in specific events to the HF advancement and movement, through systems identified with glucotoxicity and lipotoxicity [79].

We prescribe constraining the admission of immersed fats, trans fats, dietary cholesterol (<200mg day by day) and straightforward sugars [14,72]. Moreover, it ought to be situated the expanded admission of mono-and poly-unsaturated fats, proteins of high natural worth and complex starches with low glycemic reaction [14,72]. Hyperglycemia prompts changes in the redox framework, builds oxidative pressure and diminishes the accessibility of nitric oxide, bringing about endothelial brokenness; these impacts are amplified in people with HF [83].

7.1 Omega 3

Supplementation with omega 3 comes as an adjunctive treatment for HF and is prescribed by rules for the treatment of the infection [1,72,73]. Studies propose that supplementation of polyunsaturated omega-3 unsaturated fats may improve ventricular systolic capacity, increment practical limit, decline the quantity of hospitalizations, decrease provocative markers and the rate and mortality of HF [79,84,85].

In a planned companion consider with a follow-up of 12.7 years, 52,972 Japanese individuals of both genders matured 40-79, there was a lower death rate from every single cardiovascular reason among people with higher utilization of sustenances wealthy in omega 3, even lower for patients with just HF [85]. In the GISSI - HF contemplate [86], which included 6,975 patients, 1g of omega 3 added to streamlined treatment brought about a 9% decrease in mortality. An admission of omega-3-rich nourishments at any rate two times every week, for example, fish would be suggested [14,77].

7.2 Micronutrients

The insufficiency of micronutrients, for example, selenium, thiamine, calcium, magnesium, zinc and nutrient D [6,14,72,87-89], is basic in patients with HF and its causes is by all accounts multifactorial, among which delayed utilization of diuretics, low dietary admission and expanded supplement misfortunes are connected [14,72,73]. Among the advantages saw in studies assessing the effect of the supplementation of micronutrients' edifices in these patients, there is a more prominent exercise resilience and diminished side effects [14].

Witte et al [90], in a controlled twofold visually impaired clinical preliminary to evaluate the effect of nutrient and mineral enhancements in patients with HF with a level of discharge part of the left ventricle (%LVEF) $\leq 35\%$, found that when getting a case with high dosages of calcium, magnesium, copper, selenium, riboflavin, folate, nutrients A, B6, B12, C, D and E, and coenzyme Q10, patients demonstrated an abatement in ventricular chambers, improved by $5.3 \pm 1.4\%$ in %LVEF and personal satisfaction scores contrasted with the control bunch that got fake treatment ($p < 0.05$).

Consequently, supplementation of certain micronutrients would be reasonable, especially for patients with interminable utilization of diuretics, who become inadequate in water-solvent nutrients, particularly thiamine, and a few minerals, including potassium and magnesium, calcium, zinc e selenium [11,15,18]. In spite of the fact that the consequences of a few examinations propose that supplementation of certain micronutrients is likely [87,90], there is no agreement on explicit proposals on nourishing HF, uncovering the requirement for increasingly trial considers here.

7.3 Sodium

Confining sodium is usually prescribed for patients with HF by various creators and rules [5,91,92]. In any case, disputable outcomes on sodium limitation don't permit a further meaning of the ideal measure of this supplement in the eating regimen of patients with the sickness. It appears to be steady to consider its decrease just for patients in stages III and IV for side effect improvement [5,11,72,73].

Diets containing 2g of sodium were related with diminished admission of calories and supplements. Confinement of salt admission to 3g just profited patients with cutting edge HF [93]. The utilization of salts of potassium chloride might be considered, with checking of serum potassium in patients with renal illness and attending utilization of potassium-saving diuretics [72].

7.4 Thiamine

Concentrates in Brazil demonstrated that thiamine lack is seen in 30-33% of patients with coronary illness [76,88]. In patients with HF, it happens essentially in individuals who are malnourished, old, and with a serious ailment [15,94,95]. Nutrient B1 is an impetus in numerous synthetic procedures including the heart, the sensory system and the muscles. Its lack is very much reported in patients with HF because of delayed

utilization of diuretics [75,76,96]. It is in charge of beriberi and high-yield HF, reversible by rebuilding of serum levels of the nutrient [97].

For quite a long time it has been demonstrated that constant utilization of high dosages of circle diuretics in patients with HF is related with the consumption of the thiamine saves [15,98]. In one of these examinations [88] a constructive outcome of spironolactone as saving the spoliation of thiamine instigated by furosemide was watched. Another examination [89] found that thiamine substitution caused a normal 13% expansion in left ventricular discharge part, certified by different creators. Cunha et al [88] found in their investigation that thiamine insufficiency was not related with protein-vitality undernutrition, proposing that we ought to think about nutrient inadequacy even in eutrophic people.

Thiamine assumes a significant job in myocyte constriction, as exhibited in test creature models [88]. It goes about as a coenzyme in vitality digestion [88,94], and its inadequacy meddles with oxidative digestion, supporting a gathering of pyruvate and lactate, which can bother HF [87,99]. Subsequently, despite the fact that the dosages for thiamine supplementation in patients with HF are not set up [88], and there are no approved polls for evaluation of admission of this nutrient [95], supplementation in these patients can be legitimized by the advantages referenced above and must be considered, particularly in those with alcoholic etiology of IC. Being conceivable to propose that qualities between the Recommended Dietary Allowance (RDA) and the Tolerable Upper Intake Level (UL) be considered to the patients in long haul utilization of high diuretics portions.

7.5 Nutrient D

Studies recommend that cardiovascular ailments are related with hypovitaminosis D [100-102], particularly those with HF, thinking about its constrained action, inclination to stay in the home condition, in addition to the most reduced retentiveness that normally goes with the illness [75]. An insufficiency of this nutrient can prompt hypertension and expanded cardiomyopathy [100]. An exploratory examination demonstrated weakening of myocardial withdrawal when patients are offered an eating routine low in nutrient D. Moreover, it was discovered that these creatures came back to an ordinary capacity when there was a supplementation of this nutrient [15].

In India, the insufficiency of this nutrient was pervasive, with detailed rates of somewhere in the range of 70 and 100% [103-106]. It is ordinarily acknowledged that the master incendiary resistant reaction just as nutrient D inadequacy go before the advancement of HF [107]. Support of ordinary serum nutrient D is significant for the counteractive action and treatment of HF, advocating the requirement for supplementation on the event or danger of this [108,109]. By and by, controlled investigations are important to set up the perfect measurement which advantages the HF understanding.

7.6 Iron

In a meta-examination that included 153,000 patients with HF there was a 37.2% pervasiveness of iron deficiency [110]. A few elements are identified with the etiology in this gathering of patients, for example, low nourishment admission, diminished intestinal assimilation, blood misfortune from the gastrointestinal tract, variations from the norm in the generation of red platelets, declining renal capacity, perpetual aggravation, hemodilution and utilization of meds [75].

Iron deficiency diminishes the oxygen supply to the tissues, causing diminished renal perfusion and stamped neurohormonal incitement, and compounds the signs and indications of HF, for example, loss of hunger, exhaustion, edema and ischemia [9,111].

There is a pathophysiological interrelationship between iron deficiency, renal brokenness and HF, highlighting the cardio-renal-frailty disorder, in which the three parts structure an endless loop in which everyone is fit for causing or overstimulating the other [112,115]. The remedy of frailty in HF converts into an improvement of the cardiovascular capacity (expanded heart yield, diminished left ventricular mass, avoidance of left ventricular dilatation, improvement of NYHA useful class and myocardial ischemia) and renal capacity [115-117].

Albeit questionable, the particular treatment of iron deficiency includes the rectification of healthful insufficiencies and the utilization of erythropoiesis invigorating specialists [75]. The substitution of intravenous iron may improve manifestations. The suggested portion is one ampoule of iron for each week for ten weeks, or two ampoules each other week for five weeks, by observing month to month rates of hemoglobin/hematocrit and quarterly ferritin levels. The down to earth recipe to ascertain the all out iron portion (in mL) is: $N \text{ (ml)} = [(weight \text{ in kg} \times 2.4 \times Dhb) + 500 \text{ mg}] / 20$, where N=amount of iron in mL to be regulated intravenously; Dhb=difference between the ideal and discovered hemoglobin; 500 mg=required hold of iron [9,117].

7.7 Antioxidants

Patients with HF have raised oxidative pressure coming about because of expanded generation of free radicals or exhaustion of endogenous cancer prevention agents [87,118]. Utilization of cell reinforcements as an adjuvant treatment for patients with HF is probably examined in rules for treatment [72,73].

HF patients have lower plasma selenium levels than solid people. Selenium is a basic mineral for the union of glutathione peroxidase and its lack has been recognized as a reason for non-ischemic HF [87]. As zinc, manganese, copper, and a few nutrients, for example, C and E and riboflavin, selenium is a cell reinforcement that can add to the decrease of oxidative pressure and harm brought about by it, while limiting the pernicious impacts of hypoxia [75,87].

Magnesium levels may likewise be diminished in patients with HF in 30% of cases [83], joined by muscle inadequacy of this mineral, which may add to the manifestations of weariness [15]. Hypomagnesemia can cause cardiovascular arrhythmias and diminished glucose resistance in patients with HF [119]. Likewise, it is related with poor forecast and can cause positive equalization of sodium and potassium [83].

HF has additionally been connected to zinc lack by urinary misfortune, gastrointestinal edema or low protein consumption [75]. This micronutrient is a piece of the copper-zinc superoxide dismutase perplexing, equipped for cleansing the cell from free radicals [83]. Its insufficiency can prompt diminished impression of taste, loss of craving, invulnerable variations from the norm, deferred wound recuperating and expanded helplessness to creating weight bruises [75].

Nutrients C and A can likewise moderate the harm by hypoxia and endothelial apoptosis in patients with HF [14,29,83].

7.8 Nourishing treatment in HF

The sign for enteral sustenance for patients with HF isn't explicit, and ought to be considered, similarly as with other neurotic conditions, when oral sustaining is unthinkable or when the individual can't eat enough to meet 65% of their dietary needs [29,78]. It must be prescribed as a procedure for counteractive action or treatment of heart cachexia [6,23]. The utilization of bolstering cylinders should begin with little volumes, progressively advancing and keeping away from liquid over-burden [11,28]. Na terapia nutricional do paciente com IC é importante evitar a sobrecarga hídrica [11,28]. Equations with a higher caloric thickness (1,5 a 2 cal/ml) ought to be utilized by enteral course so as to coordinate the necessities in a lower volume. Liquid parity ought to be checked and, when all is said in done, liquid limitation is demonstrated (1-1.5 liters/day) [28]. Found in these terms, the recipes ought to likewise contain a high convergence of proteins per liter. In patients that need an under 1 liter for each day water limitation, uncommon consideration must be given to the micronutrients need. On the off chance that they don't meet the RDA, they ought to be enhanced.

Parenteral nourishment ought to be initiated when the gastrointestinal tract isn't working or to enhance enteral healthful help [29]. One ought to be cautious with hyperalimentation, which can prompt heart decompensation [11,29,78]. Since patients with heart disappointment have low resilience to the organization of enormous volumes, parenteral nourishment through a focal venous access is utilized more frequently than fringe get to. It permits imbue of packed hyperosmolar arrangements in lower volumes [11].

In this specific circumstance, we feature the requirement for a multiprotection and interdisciplinary group in motivation behind acquiring viable outcomes.

8. Conclusion

Undernutrition is frequently connected with HF, particularly in the later phases of the sickness, and may incessantly achieve heart cachexia, an extreme indication identified with a more regrettable clinical forecast. The HF movement and its medication treatment at the finding, notwithstanding for eutrophic patients, leave them at potential danger of healthful debilitation and micronutrients insufficiency, featuring the requirement for methodically appraisal of the nourishing status of those people so as to build up individualized and fitting dietary direction. For the nourishing suggestion of proteins, starches and lipids there are as of now explicit rules, yet not for nutrients and minerals. Studies venture toward the requirement for micronutrients supplementation over the RDA to guarantee the micronutrients backing and address the improved misfortunes, anyway it has just been built up the amounts for iron supplementation.

Despite that is very much called attention to by the writing the importance of the HF dietary checking, numerous investigations will at present be expected to define explicit micronutrients measurement rules in the HF healthful treatment.

9. References

1. Perk J, Backer GD, Gohlke H, Graham I, Reiner Z, et al. (2012) European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). *Eur Heart J* 33: 1635-701.
2. Berry C, Clark AL (2000) Catabolism in chronic heart failure. *Eur Heart J* 21: 521-32.
3. Anker SD, Sharma R (2002) The syndrome of cardiac cachexia. *Int J Cardiol* 85: 51-66.

4. Sharon Ann Hunt, William T. Abraham, Marshall H. Chin, Arthur M. Feldman, Gary S. Francis, et al. (2005) Guideline update for the diagnosis and management of chronic heart failure in the adult. *J Am Coll Cardiol* 46: 1116-43.
5. Bocchi EA, Vilas-Boas F, Perrone S, Caamaño AG, Clausell N, et al. (2008) I Latin American Guideline to assessment and treatment in decompensated heart failure. *Arq Bras Cardiol* 85: 1-48.
6. Sahade V, Montera VSP (2009) Nutritional treatment for heart failure patients. *Rev Nutr* 22: 399-408.
7. Romeiro FG, Okoshi K, Zornoff LAM, Okoshi MP (2012) Gastrointestinal Changes associated to Heart Failure. *Arq Bras Cardiol* 98: 273-7.
8. Cabrera AJR, Zerquera JMC, Ortiz ABH (2007) Progresos en la insuficiencia cardíaca. *MedIntMex* 23: 321-9.
9. Barretto ACP, Cardoso MN, Cardoso JN (2010) Iron deficiency in heart failure patients. *Rev Bras Hematol Hemoter* 32: 89-94.
10. Latado AL (2009) Diet Prescription in Chronic Heart Failure: Why Don't We Do It?. *Arq Bras Cardiol* 93: 454-5.
11. Okoshi MP, Romeiro FG, Paiva SAR, Okoshi K (2013) Heart Failure-Induced Cachexia. *Arq Bras Cardiol* 100: 476-82.
12. Boagev RC (2010) Cost considerations in the treatment of heart failure. *Texas Heart Ins J* 37: 557-8.
13. Sahade V, Passos LCS (2005) Prevalence of malnutrition in heart failure patients. *Rev Bras Nutr Clin* 20: 65-70.
14. Hernández MA, Patinõ AF (2012) Consideraciones nutricionales en el paciente con falla cardíaca crónica. *Rev ColombCardiol* 19: 312-9.
15. Witte KA, Clark AL (2001) Chronic heart failure and micronutrients. *J Am CollCardiol* 37: 165-74.
16. Mustafa I, Leverage X (2001) Metabolic and nutritional disorders in cardiac cachexia. *Nutrition* 17: 756-60.
17. Sandek A, Bauditz J, Swidsinski A, Buhner S, Weber-Eibel J, et al. (2007) Altered intestinal function in patients with chronic heart failure. *J Am CollCardiol* 50: 1561-9.
18. Witte K, Clark A (2002) Nutritional abnormalities contributing to cachexia in chronic illness. *Int J Cardiol* 85: 23-31.
19. Obisesan TO, Toth MJ, Donaldson K, Gottlieb SS, Fisher ML, et al. (1996) Energy expenditure and symptom severity in men with heart failure. *Am J Cardiol* 77: 1250-2.
20. Poehlman ET, Scheffers J, Gottlieb SS, Fischer ML, Vaitekevicius P (1994) Increased resting metabolic rate in patients with congestive heart failure. *Ann Arch Intern Med* 121: 60-2.
21. vonHaehling S, Doehner W, Anker SD (2007) Nutrition, metabolism, and the complex pathophysiology of cachexia in chronic heart failure. *Cardiovasc Res* 73: 298-309.
22. Aquilani R, Opasich C, Verri M, Boschi F, Febo O, et al. (2003) Is nutritional intake adequate in chronic heart failure patients? *J Am CollCardiol* 42: 1218-23.
23. Anker SD, Chua TP, Ponikowski P, Harrington D, Swan JW, et al. (1997) Hormonal changes and catabolic/anabolic imbalance in chronic heart failure and their importance for cardiac cachexia. *Circulation* 96: 526-34.

24. Anker SD, Ponikowski P, Varney S, Chua TP, Clark AL, et al. (1997) Wasting as independent risk factor for mortality in chronic heart failure. *Lancet* 349: 1050-3.
25. Gomez MJP, Gomez PC (2008) Evaluation of the nutritional status em com heart failure patients (II). *Enferm Cardiol* 2008: 46-50.
26. Palomas JLB et al. (2011) Ladesnutricio'nenlamortalidad Influence of long-term patients hospitalized for heart failure. *Rev Esp Cardiol* 64: 725-8.
27. Anker SD, Coats AJ (1999) Cardiac cachexia: a syndrome with impaired survival and immune and neuroendocrine activation. *Chest* 115: 836-47.
28. Mijan A, Martin E, Mateo B (2006) cardiac cachexia. *Nutr Hosp* 21: 84-93.
29. Vieira LP, Caçapava CR, Nakasato M (2004) Cardiac cachexia: a challenge to the dietician. *Rev Bras Nutr Clin* 19: 138-42.
30. Evans WJ, Morley JE, Argiles J, Bales C, Baracos V, et al. (2008) Cachexia: a new definition. *ClinNutr.* 27: 793-9.
31. vonHaehling S, Stepney R, Anker SD (2010) Advances in understanding and treating cardiac cachexia: highlights from the 5th Cachexia Conference. *Int J Cardiol* 144: 347-9.
32. Brink M, Anwar A, Delafontaine P (2002) Neurohormonal factors in the development of catabolic/anabolic imbalance and cachexia. *Int J Cardiol* 85: 111-21.
33. Genth-Zotz S, Bolger AP, Kalra PR, Coats AJ, Volk HD et al. (2004) Heatschockprotein 70 in patientswithchronicheartfailure: relation to disease severity and survival. *Int J Cardiol* 96: 397-401.
34. von Haehling S, Genth-Zotz S, Anker SD, Volk HD (2002) Cachexia: a therapeutic approach beyondcitokine antagonismo. *Int J Cardiol* 85: 173-83.
35. Anker SD, von Haehling S (2004) Inflammatorymediators in chronicheartfailure: an overview. *Heart* 90: 464-70.
36. Baumann H, Gauldie J (1994) The acutephase response. *Immunol Today* 15: 74-80.
37. Kotler DP (2000) Cachexia. *Ann Intern Med* 133: 622-34.
38. Gut AL, Sugizaki MM, Okoshi MP, Carvalho RF, Pai-Silva MD, et al. (2008) Food restriction impairs myocardial inotropic response to calcium and beta- adrenergic stimulation in spontaneously hypertensive rats. *Nutr Res* 28: 722-7.
39. Okoshi MP, Okoshi K, Matsubara LS, Dal Pai-Silva M, Gut AL, et al. (2006) Myocardial remodeling and dysfunction are induced by chronic food restriction in spontaneously hypertensive rats. *Nutr Res* 26: 567-72.
40. King D, Smith ML, Chapman TJ, Stockdale HR, Lye M (1996) Fat malabsorption in elderly patients with cardiac cachexia. *Age Ageing* 25: 144-9.
41. Araújo JP, Lourenço P, Rocha-Gonçalves F, Ferreira A, Bettencourt P (2011) Nutritional markers and prognosis in cardiac cachexia. *Int J Cardiol* 146: 359-63.
42. Anker SD, Negassa A, Coats AJ, Afzal R, Poole-Wilson PA, et al. (2003) Prognostic importance of weight loss in chronic heart failure and the effect of treatment with angiotensin-converting-enzyme inhibitors: an observational study. *Lancet* 361: 1077-83.
43. Kenchaiah S, Evans JC, Levy D, Wilson PWF, Benjamin EJ, et al. (2002) Obesity and the risk of heart failure. *N Engl J Med* 347: 305-13.

44. Thrainsdottir IS, Aspelund T, Gudnason V, Malmberg K, Sigurdsson G, et al. (2007) Increasing glucose levels and BMI predict future heart failure: experience from the Reykjavík Study. *Eur J Heart Fail* 9: 1051-7.
45. Cano M et al. (2010) Assessment body in patients with renal insufficiency lacomposição crônica .*Nutr Hosp* 25: 682-7.
46. Rocha IE, Victor EG, Braga MC, Barbosa e Silva O, Becker Mde M (2007) Echocardiography Evaluation for Asymptomatic Patients with Severe Obesity. *Arq Bras Cardiol* 88: 52-8.
47. Sharma S, Adroque JV, Golfman L, Uray I, Lemm J, et al. (2004) Intramyocardial lipid accumulation in the failing human heart resembles the lipotoxic rat heart. *FASEB J* 18: 1692-700
48. Poornima IG, Parikh P, Shannon RP (2006) Diabetic cardiomyopathy:the search for a unifying hypothesis. *Circ Res* 98: 596-605.
49. Ingelsson E, Sundström J, Ärnlöv J, Zethelius B, Lind L (2005) Insulin Resistance and Risk of Congestive Heart Failure. *JAMA* 294: 334-41.
50. Gustafsson F, Kragelund CB, Torp-Pedersen C, Seibaek M, Burchardt H, et al. (2005) Effect of obesity and being overweight on long-term mortality in congestive heart failure: influence of left ventricular systolic function. *Eur Heart J* 26: 58-64.
51. Kalnatar-Zadeh K, Block G, Horwich TB, Fonarow GC (2004) Reverse epidemiology of conventional cardiovascular risk factors in patients with heart failure. *J Am Coll Cardiol* 43: 1439-44.
52. Horwich TB, Fonarow GC, Hamilton MA, MacLellan WR, Woo MA, et al. (2001) The relationship between obesity and mortality in patients with heart failure. *J Am Coll Cardiol* 38: 789-95.
53. Davos CH, Doehner W, Rauchhaus M, Ciccoira M, Francis DP, et al. (2003) Body mass and survival in patients with the chronic heart failure without cachexia: the importance of obesity. *J Card Fail* 9:29-35.
54. Lainscak M, Von Haehling S, Doehner W, Anker SD (2012) The obesity paradox in chronic disease: facts and numbers. *J Cachexia Sarcopenia Muscle* 3: 1-4.
55. Artham SM, Ventura HO (2007) Insuficienciardiaca and the “ paradox of obesidade: lahistoriacontinua. *RevEspCardiol* 60: 1113-7.
56. Banack HR, Kaufman JS (2013) The obesity paradox explained. *Epidemiology* 24: 461-2.
57. Dorner TE, Rieder A (2010) Obesity paradox or reverse epidemiology: is high body weight a protective factor for various chronic conditions. *Dtsch Med Wochenschr* 135: 413-8.
58. Lenz M, Richter T, Mühlhauser I (2009) The morbidity and mortality associated with overweight and obesity in adulthood: a systematic review. *Dtsch ArzteblInt* 106: 641-8.
59. Dyer A, Stamler J, Garside DB et al. (2004) Long-term consequences of body mass index for cardiovascular mortality: the Chicago Heart Association Detection Project in Industry study. *Ann Epidemiol* 14: 101-8.
60. Oreopoulos A, Padwal R, Kalantar-Zadeh K et al. (2008) Body mass index and mortality in heart failure: a meta-analysis. *Am Heart J* 156: 13-22.
61. Pinheiro AS et al. (2007) Obesity: protective factor in patients with heart failure? *Rev Bras Nutr* 22: 20-7.
62. BarretoACP (2003) Obesity and heart failure. *Rev Assoc Med Bras* 49: 2-3.

63. Campillo B, Paillaud E, Uzan I, Merlier I, Abdellaoui M, et al. (2004) Value of body mass index in the detection of severe malnutrition: influence of the pathology and changes in anthropometric parameters. *Clin Nutr* 23: 551-9.
64. Zuchinali P, Souza Gc, Alves Fd, Goldraich LA et al. (2013) Triceps Skinfold as a Prognostic Predictor in Outpatient Heart Failure. *Arq Bras Cardiol* 101: 434-41.
65. Yamauti AK, Ochiaia ME, Bifulo PS de Araujo MA, Alanoso RR et al. (2006) Subjective Global Assessment of Nutritional Status in Cardiac Patients. *Arq Bras Cardiol* 87: 772-7.
66. Britto EP, Mesquita ET (2008) Bioelectrical Impedance Analysis in Heart Failure *Rev Soc Cardiol RJ* 21: 178-83.
67. Llamas L, Baldomero V, Lelgsas ML, Rodota Me (2013) Phase values from the angle by bioelectrical impedance; nutritional status and prognostic value. *Nutric Hosp* 28: 286-95.
68. Barbosa-Silva MC, Barrows AJ, Heymsfield SB, Pierson RN (2005) Bioelectrical impedance analysis: population reference values for phase angle by age and sex. *American Journal Clinical Nutrition* 82: 49-52.
69. Silva RD, Dutra MPV, Ignêz M, Elsas G, Azevedo ZMA et al. (2007) Associação between phase angle and I Gravidade PRISM gives Sepse . *RevBras Ter Int* 19: 297-303.
70. Omran ML, Morley JE (2000) Assessment of protein energy malnutrition in older persons, part I: History, examination, body composition, and screening tools. *Nutrition* 16: 50–63.
71. Omran ML, Morley JE (2000) Assessment of protein energy malnutrition in older persons. Part II: Laboratory evaluation. *Nutrition* 16: 131–40.
72. Bocchi EA, Marcondes-Braga FG, Ayub-Ferreira SM, Rohde LE, Oliveira WA, et al. (2009) Brazilian Society of Cardiology. III Brazilian Guidelines on Chronic Heart Failure. *Arq Bras Cardiol* 93: 1-71.
73. Clyde W. Yancy, Mariell Jessup, BiykemBozkurt, Javed Butler, Donald E. Casey, et al. (2013) Guideline for the Management of Heart Failure: A Report of the American College of Cardiology Foundation/American Heart Association ACCF/AHA . *Circulation* 128: 240-327.
74. Ghali JK (2009) Anemia and heart failure. *Curr Opin Cardiol* 24: 172-8.
75. Frediani JK, Reilly CM, Higgins M, Clark PC, Gary RA, et al. (2013) Quality and Adequacy of Dietary Intake in a Southern Urban Heart Failure Population. *Cardiovasc Nurs* 28: 119–28.
76. Lourenço BH, Vieira LP, Macedo A, Nakasato M, Marucci Mde F, et al. (2009) Nutritional Status and Adequacy of Energy and Nutrient Intakes among Heart Failure Patients. *Arq Bras Cardiol* 93: 541-8
77. Rujinski MN (2007) Nutrición em La insuficiencia cardíaca um granes labón. *Ver Insuf Card* 2: 115-7.
78. Akner G, Cederholm T (2001) Treatment of protein-energy malnutrition in chronic nonmalignant disorders. *Am J Clin Nutr* 74: 6-24.
79. Nodari S, Triggiani M, Campia U, Manerba A, Milesi G, et al. (2011) Effects of n-3 polyunsaturated fatty acids on left ventricular function and functional capacity in patients with dilated cardiomyopathy. *J Am Coll Cardiol* 57: 870-9.
80. Heart Failure Society of America (2006) Executive summary: HFSA 2006 comprehensive heart failure practice guideline. *J Card Fail* 12:10-38.
81. Heymsfield SB, Smith J, Redd S, Whitworth HB (1981) Nutrition support in cardiac failure. *Surg Clin North Am* 61: 635-52.

82. Quinn T, Askanazi J (1987) Nutrition and cardiac disease. *Crit Care Clin* 3: 167-84.
83. Sandek A, Doehner W, Anker SD, von Haehling S (2009) Nutrition in heart failure: an update. *Curr Opin Clin Nutr Metab Care* 12: 384-91.
84. Zhao YT, Shao L, Teng LL, Hu B, Luo Y, et al. (2009) Effects of n-3 polyunsaturated fatty acid therapy on plasma inflammatory markers and N-terminal pro- brain natriuretic peptide in elderly patients with chronic heart failure. *J Int Med Res* 37: 1831-41.
85. Yamagishi K, Iso H, Date Ch, Fukui M, Wakai K, et al. (2008) Fish Ω -3 polyunsaturated fatty acids, and mortality from cardiovascular diseases in a nationwide community-based cohort Japanese men and women. *J Am Coll Cardiol* 52: 988-96.
86. GISSI-HF Investigators (2008) Effect of n-3 polyunsaturated fatty acids in patients with chronic heart failure (the GISSI-HF trial): a randomized double-blind, placebo-controlled trial. *Lancet* 372: 1223-30.
87. de Lorgeril M, Salen P, Accominotti M, Cadau M, Steghens JP, et al. (2001) Dietary and blood antioxidants in patients with chronic heart failure. Insights into the potential importance of selenium in heart failure. *Eur J Heart Fail* 3: 661-9.
88. Cunha S, Albanesi Filho FM, Bastos VLFC, Antelo DS, Souza MM (2002) Thiamin, selenium, and copper levels in patients with idiopathic dilated cardiomyopathy taking diuretics. *Arq Bras Cardiol* 79: 4540-65.
89. Seligmann H, Halkin H, Rauchfleisch S, Kaufmann N, Motro M, et al. (1991) Thiamine deficiency in patients with congestive heart failure receiving longterm furosemide therapy: a pilot study. *Am J Med* 91: 151-5.
90. Witte KK, Nikitin NP, Parker AC, von Haehling S, Volk HD, et al. (2005) The effect of micronutrient supplementation on quality-of-life and left ventricular function in elderly patients with chronic heart failure. *Eur heart J* 26: 2238-44.
91. Heart Failure Society of America¹, Lindenfeld J, Albert NM, Boehmer JP, Collins SP, et al. (2010) HFSA 2010 comprehensive heart failure practice guideline. *J Card Fail* 16: 1–194.
92. Dickstein K, Cohen-Solal A, Filippatos G, McMurray JJ, Ponikowski P, et al. (2008) ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology: developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *Eur Heart J* 29: 2388–442.
93. Lennie TA, Song EK, Wu JR, Chung ML, Dunbar SB, et al. (2001) Three gram sodium intake is associated with longer event-free survival only in patients with advanced heart failure. *J Card Fail* 17: 325-30.
94. Payne-Emerson H, Lennie TA (2008) Nutritional considerations in heart failure. *Nurs Clin North Am* 43: 117-32.
95. Rocha RM, Silva GV, de Albuquerque DC, Tura BR, Albanesi Filho FM (2008) Influence of Spironolactone Therapy on Thiamine Blood Levels in Patients with Heart Failure. *Arq Bras Cardiol* 90: 324-8.
96. Reyes A, Leary W (1993) Renal excretory responses to single and repeated administration of diuretics in healthy subjects: clinical connotations. *Cardiovasc Drugs Ther* 7: 29–44.
97. Lip GY, Gibbs CR, Beevers DG (2000) ABC of heart failure: aetiology. *BMJ* 320: 104-7.
98. Yui Y, Itokawa Y, Kawai C (1980) Furosemide induced thiamine deficiency. *Cardiovasc Res* 14: 537-40.

99. Sole MJ, Jeejeebhoy KN (2002) Conditioned nutritional requirements: therapeutic relevance to heart failure. *Herz* 2: 174-8.
100. Pilz S, Tomaschitz A, Marz W, Drechsler C, Ritz E, et al. (2011) Vitamin D, cardiovascular disease and mortality. *Clin Endocrinol* 75: 575–84.
101. Brewer LC, Michos E.D, Reis JP (2011) Vitamin D in atherosclerosis, vascular disease, and endothelial function. *Curr Drug Targets* 12: 54–60.
102. Soskić S, Stokić E, Isenović ER (2014) The relationship between vitamin D and obesity. *Curr Med Res Opin* 30: 1197-9.
103. Multani SK, Sarathi V, Shivane V, Bandgar TR, Menon PS, et al. (2010) Study of bone mineral density in resident doctors working at a teaching hospital. *J. Postgrad. Med* 56: 65–70.
104. Marwaha RK, Puri S, Tandon N, Dhir S, Agarwal N, et al. (2011) Effects of sports training & nutrition on bone mineral density in young Indian healthy females. *Indian J Med Res* 134: 307–13.
105. Baidya A, Chowdhury S, Mukhopadhyay S, Ghosh S (2012) Profile of vitamin D in a cohort of physicians and diabetologists in Kolkata. *Indian J Endocrinol Metab* 16: 416–7.
106. Ritu G, Ajay G (2014) Vitamin D deficiency in India: Prevalence, causalities and interventions. *Nutrients* 6: 725-75.
107. Mora C, Myers RA, Coll M, Libralato S, Pitcher TJ, et al. (2009) Management effectiveness of the world's marine fisheries. *PLoS Biol* 7: 1-11.
108. Ku YC, Lin ME, Ku Cs, Liu TY, Lin SL (2013) Relationship between vitamin D deficiency and cardiovascular disease. *World J Cardiol.* 5: 337-46.
109. Ellam T, Hameed A, ulHaque R, Muthana M, Wilkie M (2014) Vitamin D deficiency and exogenous vitamin D excess similarly increase diffuse atherosclerosis calcification in apolipoprotein E knockout mice. *PLoS One* 9: 887-67.
110. Groenveld HF, Januzzi JL, Damman K, van Wijngaarden J, Hillege HL, et al. (2008) Anemia and mortality in heart failure patients a systematic review and meta-analysis. *J Am CollCardiol* 52: 818-27.
111. Murphy CL, McMurray JJ (2003) Approaches to the treatment of anaemia in patients with chronic heart failure. *Heart Fail Rev* 13: 431-8.
112. Silverberg DS, Wexler D, Blum M, Wollman Y, Iaina A (2003) The cardio-renal syndrome: does it exist? *Nephrol Dial Transplant* 18: 7-12.
113. Gil P, Justo S, Caramelo C (2005) Cardio-renal failure: an emerging clinical entity. *Nephrol Dial Transplant* 20: 1780-3.
114. Villacorta H, Saenz-Tello BF, Santos EB, Steffen R, Wiefels C, et al. (2010) Renal Dysfunction and Anemia in Patients with Heart Failure with Reduced versus Normal Ejection Fraction. *Arq Bras Cardiol* 94: 378-84.
115. Reis FJFB, Fernandes AMS, Bitencourt AGV, Neves FBCS, Kuwano AY, et al. (2009) Prevalence of Anemia and Renal Insufficiency in Non-Hospitalized Patients with Heart Failure. *Arq Bras Cardiol* 93: 268-74.
116. Marecos C, Falcão LM (2010) Anemia and cardiorenal syndrome in heart failure: review article. *Rev Port Med Intern* 17: 263-73.
117. Pereira CA, Roscani MG, Zanati SG, Matsubara BB (2013) Anemia, Heart Failure and Evidence-Based Clinical Management. *Arq Bras Cardiol* 101: 87-92.

118. Keith ME, Jeejeebjoy KN, Langer A, Kurian R, Barr A, et al. (2001) A Controlled trial of vitamin E supplementation in patients with congestive heart failure. *Am J Clin Nutr* 73: 219-24.
119. Fuentes J, Salmon A, Silver M (2006) Acute and chronic oral magnesium supplementation: effects on endothelial function, exercise capacity, and quality of life in patients with symptomatic heart failure. *Congest Heart Fail* 12: 9–13.



This work is licensed under a Creative Commons Attribution Non-Commercial 4.0 International License.