



NUTRITIONAL MANAGEMENT OF HEART FAILURE

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AFFILIATIONS AND DISCLOSURES

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I have nothing to disclose.

LEARNING OBJECTIVES

1. State the limitations of current dietary sodium restriction recommendations in heart failure management.
2. Explain the relevance of addressing malnutrition within existing heart failure treatment algorithms.
3. Describe the impact of pharmacotherapy on nutritional status in heart failure patients.
4. Discuss existing evidence regarding the Dietary Approaches to Stop Hypertension (DASH) diet on heart failure outcomes.



HEART FAILURE (HF): ICD-9 428; ICD-10 I50

- ▶ 6.5 million Americans 20+ years of age have HF (NHANES 2011-2014)
- ▶ Prevalence projected to increase by 46% by the year 2030
- ▶ In 2012, cost for HF was \$30.7 billion
- ▶ Largest problem: HF hospitalizations
 - ▶ HF leading cause of hospitalization in adults 65+ years
 - ▶ 50+% of patients readmitted within 6 months
 - ▶ Policy focus: Hospital Readmissions Reduction Program (HRRP) - CMS established financial penalties for hospitals with higher than average 30-day readmission rates





“ Sodium restriction is the cornerstone of nutritional heart failure management, because increased sodium intake leads to increased fluid retention.

SODIUM RECOMMENDATIONS IN HF

Table. Guideline Recommendations for Dietary Sodium and Fluid Restriction in Heart Failure

Guideline	Year	Recommendation Sodium Restriction Recommendation Fluid Restriction Recommendation	Level of Evidence
National Heart Foundation of Australia/Cardiac Society of Australia and New Zealand	2006 ⁷	<3 g/d for NYHA class II without peripheral edema/ <2 g/d for NYHA class III and IV <2 L/d for all patients and <1.5 L/d during fluid retention episodes	C
Heart Failure Society, India	2007 ⁴	<2 g/d <2 L/d	Not Stated
European Society of Cardiology	2008 ⁵	Moderate restriction 1.5–2 L/d in patients with severe symptoms and especially with hyponatremia	C
Canadian Cardiovascular Society	2008 ⁶	<2 g/d 2 L/d	Not Stated
American College of Cardiology/American Heart Association	2009 ²	Moderate restriction (≤2 g/d, if volume overload, followed by fluid intake restriction to 2 L/d if fluid retention persists)	C
Royal College of Physicians	2010 ³	<i>Salt reduction</i> <i>Fluid restriction</i>	Limited; further research required
Heart Failure Society of America	2010 ⁸	2–3 g/d; <2 g/d may be considered in moderate to severe heart failure <2 L/d, if fluid retention persists and if severe hyponatremia (serum Na <130 mEq/L) is present	C
Scottish Intercollegiate Guidelines Network	2010 ⁹	<2.4 g/d tailored fluid restriction	1+
American Dietetic Association	2011 ¹⁰	<2 g/d 1.4–1.9 L/d depending on clinical symptoms	Fair

Level of Evidence: C=Limited populations evaluated. Only consensus opinion of experts, case studies, or standard of care; Fair=Benefits exceed the harms but quality of evidence is not as strong; 1+ = well-conducted meta-analysis, systemic reviews, or randomized controlled trials with low risk of bias. NYHA indicates New York Heart Association.

SODIUM RECOMMENDATIONS IN HF MANAGEMENT

Guideline [Ref]	Country	Statement
2013 ACCF/AHA Guideline for the Management of Heart Failure: Executive Summary ¹	USA	<p>“Sodium restriction is reasonable for patients with symptomatic HF to reduce congestive symptoms. (Level of Evidence: C)”</p> <p>“Even the widely embraced dictum of sodium restriction in HF is not well supported by current evidence”</p>
2017 Comprehensive Update of the Canadian Cardiovascular Society Guidelines for the Management of Heart Failure ²	Canada	<p>“Dietary sodium consumption for patients with HF remains controversial.”</p> <p>“We suggest that patients with HF should restrict their dietary salt intake to between 2 g/d and 3 g/d (Weak Recommendation; Low-Quality Evidence).”</p> <p>“The optimal quantity of salt in the diet is still a subject of debate. The amount should be adapted to the clinical situation, the severity of symptoms, and baseline consumption without interfering with other nutritional content.”</p>

2016 European Society of Cardiology HF Guidelines omits sodium intake recommendations entirely due to the weak evidence.

2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure



A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America

Developed in Collaboration with the American Academy of Family Physicians, American College of Chest Physicians, and International Society for Heart and Lung Transplantation

Find: “Sodium” (0)

Find: “Nutrition” (1)

(continued)

Nitrate therapy can reduce pulmonary congestion and improve exercise tolerance in patients with HFrEF. However, the NEAT-HFpEF (Nitrate’s Effect on Activity Tolerance in Heart Failure With Preserved Ejection Fraction) trial (171) randomized 110 patients with EF \geq 50% on stable HF therapy, not including nitrates, and with activity limited by dyspnea, fatigue, or chest pain, to either isosorbide mononitrate or placebo and found no beneficial effects on activity levels, QoL, exercise tolerance, or NT-proBNP levels. On the basis of this trial, routine use of nitrates in patients with HFpEF is not recommended. This recommendation does not apply to patients with HFpEF and symptomatic CAD for whom nitrates may provide symptomatic relief. Phosphodiesterase-5 inhibition augments the nitric oxide system by upregulating cGMP activity. The RELAX (Phosphodiesterase-5 Inhibition to Improve Clinical Status and Exercise Capacity in Heart Failure with Preserved Ejection Fraction) trial (172) randomized 216 patients with EF \geq 50% on stable HF therapy and with reduced exercise tolerance (peak observed V_{O_2} <60% of predicted) to phosphodiesterase-5 inhibition with sildenafil or placebo. This study did not show improvement in oxygen consumption or exercise tolerance.

III: No Benefit

C

Routine use of nutritional supplements is not recommended for patients with HFpEF.

2013 recommendation remains current.

SODIUM INTAKE RECOMMENDATIONS IN HF – THE EVIDENCE.....

1. **Observational studies:** 2 studies

- NYHA class I and II: >2,800 mg/d sodium associated with increased HF hospitalizations
- NYHA class I and II: <3,000 mg/d associated with increased hospitalizations and mortality
- NYHA class III and IV: <3,000 mg/d associated with longer event-free survival

2. **Randomized clinical studies:** 9 short-term (< 100 participants)

- Outpatient: 2/6 studies found an improvement of clinical signs and symptoms (clinical congestion score, time needed for compensation of HF symptoms, NYHA functional class change, KCCQ summary score change)
- Inpatient: 0/2 studies found improvement

3. **Randomized clinical studies (low or questionable quality):**

- Indicate that low-sodium in comparison to moderate-sodium intake leads to higher readmission and mortality rates



SODIUM INTAKE IN HF DEBATE: OPEN QUESTIONS.....

- ▶ Are sodium intake recommendations for the healthy population applicable to HF patients?

(Paradoxically, the AHA now recommends 1,500mg/day for all Americans...)

- ▶ Should there be individualized recommendations for different ACC/AHA stages or NYHA functional classes in HF patients?
- ▶ What is the effect of sodium restriction in the context of GDMT (RAAS suppression) and fluid restriction?
- ▶ Should there be individualized sodium restriction levels depending on sodium sensitivity?
- ▶ What about HFrEF vs. HFpEF?





***Why are we so focused on sodium intake
alone?***

Emphasis on low-sodium intake in HF is commonly done with little consideration of nutritional status.



HF PATIENTS ARE AT RISK FOR MALNUTRITION

57% of HF patients at least mildly malnourished

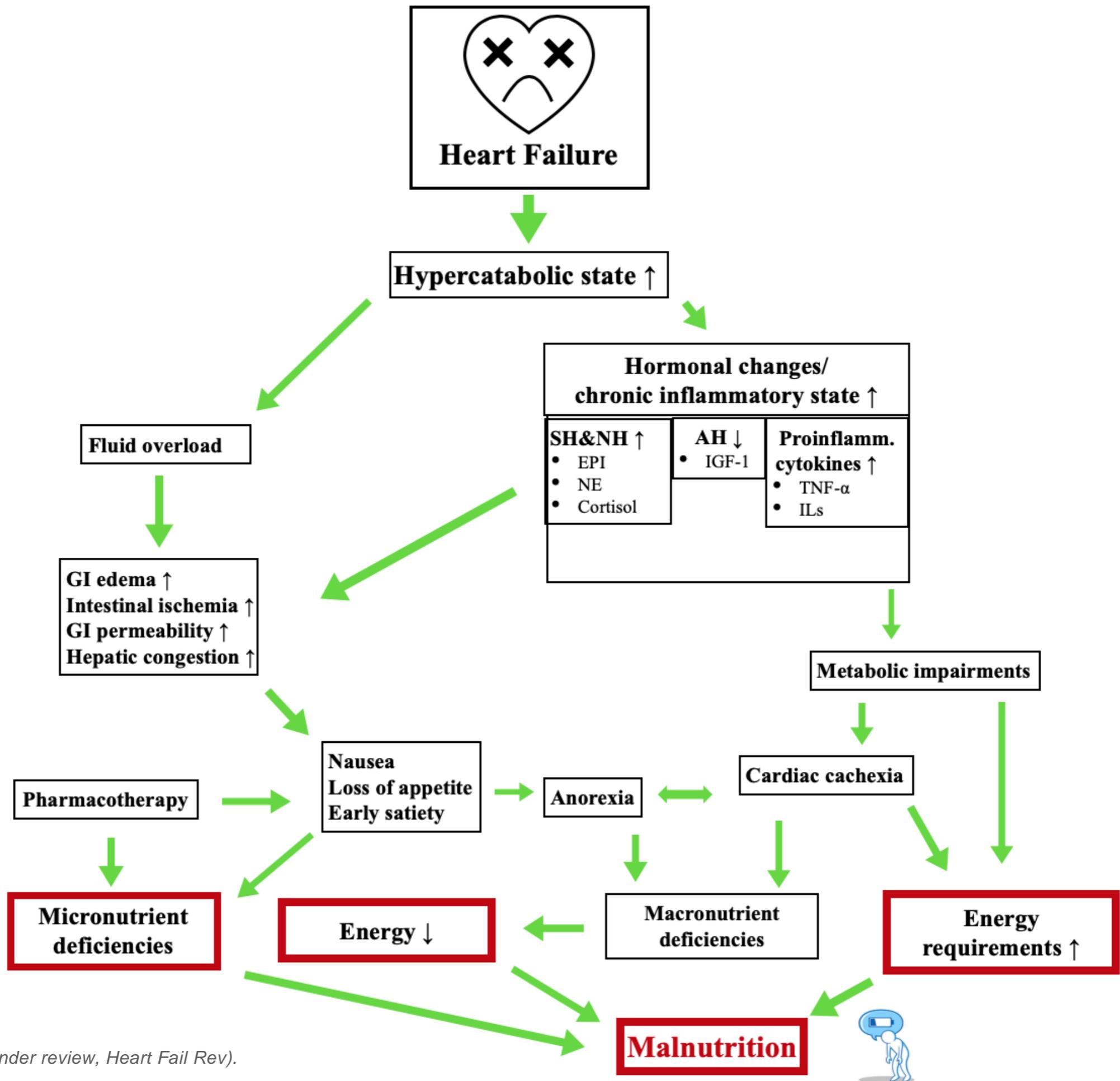
3 reasons:

1. HF pathophysiology
2. Inadequate energy and nutrient intake
3. Medication therapy (adverse) effects

Consequences:

- ▶ Cardiac cachexia
 - Increased functional decline
- ▶ Increased mortality

It may be important to consider malnutrition as a *potential prognostic value* in HF.



THE ROLE OF MICRONUTRIENT DEFICIENCIES IN HF PATHOPHYSIOLOGY

Micronutrients	Food Sources	Biological Action Relevant to HF	Pathophysiologic Effect of Deficiency in HF
Vitamin C	Fruits (e.g., citrus), dark green leafy vegetables, cruciferous vegetables, potatoes, tomatoes	Reduction in oxidative stress	Not known; supplementation has shown improvements in endothelial function, markers of oxidative stress, and inotropic response
Vitamin B1	Pork, fish, legumes, fortified grains and cereals	Catalyst in chemical processes of the heart, muscles, and nervous system; energy metabolism	“Wet beriberi” as a consequence of long-term diuretic use: increased HR, edema, shortness of breath
Vitamin D	Fatty fish, fortified products (e.g., cereal, milk, juice), mushrooms treated with UV light	Regulation of muscle function and blood pressure, anti-inflammatory response	Hypertension, cardiomyopathy
Calcium	Dairy products, beef, fish, dark green leafy vegetables, almonds, fortified products	Increased myocardial contractility; influence on bone health, blood clotting, muscle and nerve function	Related to vitamin D deficiency: hypertension, cardiomyopathy
Magnesium	Dark green leafy vegetables, chocolate, almonds, avocado, yoghurt, beef, chicken, fish, fruit (e.g., bananas)	Influence on nerve function, protein synthesis, bone health, and enzyme function	Arrhythmias, increased HR, impaired glucose tolerance, muscle fatigue; suppl. improves endothelial function in HF
Potassium	Meats, fish, cruciferous vegetables, tomatoes, fruit (e.g., citrus, cantaloupe, kiwi), dairy products, nuts	Regulation of acid-base balance, protein synthesis, carbohydrate metabolism, and electrical activity of heart	Arrhythmias, iatrogenic mortality
Selenium	Breads, grains, meat, poultry, fish, eggs	Reduction in oxidative stress, thyroid hormone metabolism	<u>Keshan disease</u> : cardiomyopathy
Zinc	Beef, chicken, oysters, fortified cereals, seeds, peanuts	Reduction in oxidative stress; catalyst in cellular metabolism: immune function, protein synthesis	Loss of taste, impaired appetite, diarrhea, hair loss, immunological abnormalities, delayed wound healing, decubitus ulcers

HF IS ASSOCIATED WITH MICRONUTRIENT DEFICIENCIES

Micronutrients	Observational studies assessing nutrient status in HF	Intervention studies assessing supplementation in HF	Comments
Vitamin A	No status difference HF vs. control	None	Evidence limited
Vitamin C	Mean plasma concentration lower in HF vs. control	Improvements in endothelial function, markers of oxidative stress, and inotropic response	No studies assessing effect of suppl. on HF symptoms, physical function, and HF prognosis
Vitamin E	No status difference HF vs. control	Mixed effects on oxidative stress; higher number of HF events; higher risk of developing HF if baseline LV <50%; no change in natriuretic peptides, inflammation, QoL	Potential risk of supplementation
Vitamin B1	Increased risk of B1 deficiency in HF	Increase in LVEF	Interventional evidence limited
Vitamin B2 & B6	Increased risk of B2 and B6 deficiency in HF	None	Evidence limited
Vitamin B12 & Folate	No status difference; B12 deficient HF patients more likely to have reduced LVEF	Decrease in BP, HR, and endothelial function with suppl. of B6, B12, and folate	Evidence limited
Vitamin D	Lower circulating vit D levels in HF vs. control; status associated with incidence and prevalence of HF; negative correlation between mean plasma vit D and NT-proBNP, NYHA class, physical function, mortality; positively correlated with ventricular function	Decrease in systemic inflammation, reduction in BNP	Most evidence; intoxication and hypercalcemia reported with megadose of 864,000 IU of vit d3
Selenium, Zinc, Copper	Selenium deficiency an accepted cause of reversible HF (Keshan Disease); lower levels of selenium and zinc in HF vs. control; higher levels of copper	None	Evidence limited
Multiple Micronutrients	None	Reduction in LV end diastolic volume; increase in LVEF;	Evidence limited

HF MEDICATION THERAPY AFFECTS

NUTRITIONAL STATUS

▶ Loop and thiazide diuretics:

Deplete serum potassium, magnesium, calcium, and thiamine through excessive renal excretion

Secondary increases in aldosterone contribute to hypokalemia

Lead to increased sodium appetite → RAAS activation

▶ ACE inhibitors and ARBs:

Lead to anorexia

Increase serum potassium and zinc

Dysgeusia

▶ Aldosterone antagonists and beta blockers:

Increase serum potassium

▶ Digoxin:

Leads to anorexia



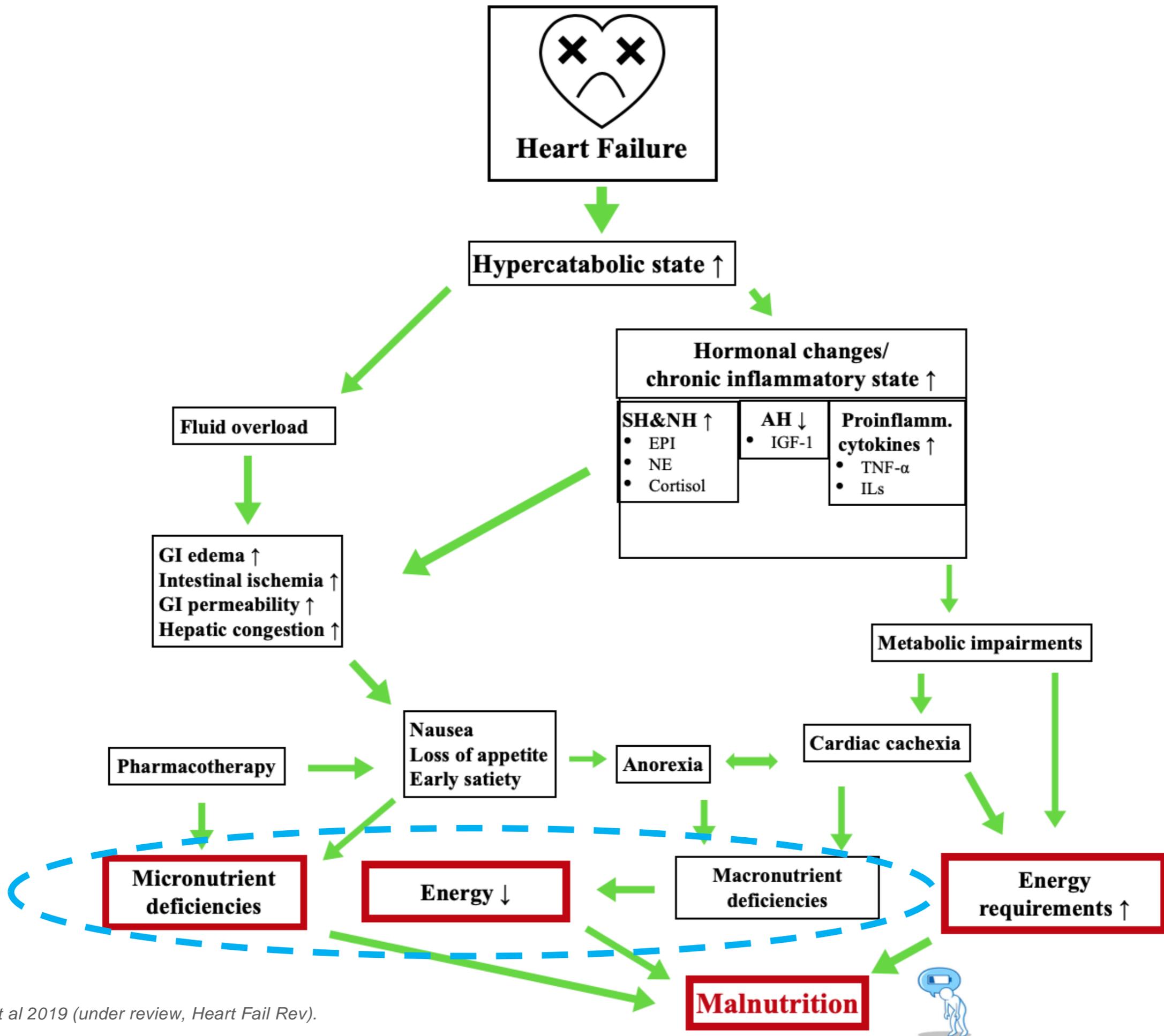
Still thinking about recommending a low-sodium diet to HF patients?

Think again.

SODIUM INTAKE IS NEGATIVELY ASSOCIATED WITH NUTRIENT INTAKE AMONG HF PATIENTS

- ▶ Individuals with high sodium intake are more likely to show:
 - High energy intake
 - High fast food consumption
- ▶ Individuals with low sodium intake are more likely to show:
 - Low energy intake
 - Low intakes of protein, fat, and carbohydrates
 - Consumption of less than 50% of the RDA of vitamins D and E, magnesium, and potassium
 - Low intakes of calcium, zinc, and thiamin (!)
 - Low intake of fruits, vegetables, and milk





BODY MASS INDEX AND NUTRITIONAL

RISK IN HF PATIENTS

- ▶ “*Obesity paradox*.” HF patients with higher than normal BMIs have shown to have improved outcomes and rates of survival
 - Suggested explanations:
 1. Neutralization of TNF- α and lipopolysaccharides through excess adipose tissue
 2. Obesity among HF patients is associated with younger age, and increased albumin levels, lean muscle mass, strength, and exercise tolerance
- ▶ No significant relationship between dietary energy intake and BMI in HF patients → other pathophysiologic factors at play
- ▶ Malnutrition is prevalent among overweight and obese patients
 - BMI may mask malnutrition in presence of edema



BMI not recommended for nutritional risk stratification in HF



A CASE FOR THE DIETARY APPROACHES TO STOP HYPERTENSION (DASH) DIET

THE DIETARY APPROACHES TO STOP HYPERTENSION (DASH) DIET.....

- ▶ Prompted by the rising prevalence of HTN, the DASH diet was developed in the mid-1990s
- ▶ Dietary pattern with emphasis on high amounts of fruits and vegetables, protein and fiber, low-fat, dairy products, whole grains, poultry, fish, and nuts; nutrient-rich foods containing minerals: potassium, calcium, magnesium
- ▶ Shown to reduce BP irrespective of sodium intake
- ▶ Associated with reduced risk of CHD and HF
- ▶ Formally adopted into:
 1. 2010 Dietary Guidelines for Americans
 2. 2013 AHA/ACC Cardiovascular Disease Prevention Guidelines (strong recommendation: level 1 A)

STUDIES EVALUATING THE DASH DIET

IN LIFE PATIENTS

Study	Population	Key Results	Comments
Levitan et al. ²⁶	Retrospective n = 3215 Women Age 50–79 years Follow-up 4.6 years ≥1 HF hospitalization	<ul style="list-style-type: none"> • Compared to lower DASH diet score, higher scores were associated with lower hazard rate of death. • In patients who had HF hospitalization, 43.1% died during follow-up. • Non-significant but beneficial trend with Mediterranean diet 	HF etiology, subtypes, and functional classes were not recorded.
Hummel et al. ²⁷	Prospective n = 13 Women/men = 12/1 Age 72 ± 10 years Follow-up 21 days HF with preserved ejection fraction NYHA class II/III Treated hypertension Chronic kidney disease	<ul style="list-style-type: none"> • Significantly reduced ambulatory blood pressure and arterial stiffness • 24-h urinary sodium levels declined 	Dietary intervention — all food provided No significant functional improvement with six-minute walk test
Hummel et al. ²⁸	Prospective n = 14 Women/men = 13/1 Otherwise same as in Hummel et al. ²⁷	<ul style="list-style-type: none"> • Improved arterial elastance measured by a radial artery tonometer • Improved ventricular diastolic function assessed by echocardiography. • Improved ventricular–arterial elastance coupling 	
Rifai et al. ³⁰	Prospective, randomized-controlled n = 48 Women/men = 19/29 Average age 60 years Stage C, NYHA classes I–III Follow-up 3 months	<ul style="list-style-type: none"> • Improved arterial elasticity in HF patients following DASH diet compared to general dietary recommendations in HF • Increased six-minute walk test distance • Improved quality of life questionnaire results • Trend to improved DASH diet compliance over time 	

ADDITIONAL STUDIES

1. GOURMET-HF study:

- Randomized trial to examine the effects of home-delivered, low-sodium DASH meals on disease-specific quality of life, HF symptoms/physical limitations, 30-day hospital readmissions, and cardiac biomarkers among HF patients post-discharge
- N=66; aged 71 ± 8 years; 30% female; EF $39 \pm 18\%$
- DASH arm in comparison to control showed trends towards improved HF symptoms/physical limitations and 30-day readmissions

2. DASH-HF study:

- Ongoing: Clinical study examining the effect of DASH meals on cardiometabolic, hemodynamic and physical function markers in advanced HF patients with CardioMEMS devices.

TAKE HOME POINTS

- **Weak evidence for recommending sodium restriction** to HF patients
- **Malnutrition** is a **common** phenomenon among HF patients
 - Characterized by energy imbalance and micronutrient deficiencies
 - Associated with cardiac cachexia, functional decline, and mortality
 - Complicated by pharmacotherapy
- Recommending sodium restriction may contribute to malnutrition and worsen HF outcomes
- **Evidence supportive of DASH diet** pattern in controlling clinical HF endpoints, but larger trials are needed





THANK YOU.

Questions?