International Journal of Medical Science and Clinical Research Studies

ISSN(print): 2767-8326, ISSN(online): 2767-8342

Volume 02 Issue 10 October 2022

Page No: 1186-1194

DOI: https://doi.org/10.47191/ijmscrs/v2-i10-33, Impact Factor: 5.365

Congestive Heart Failure for the Primary Care Physician

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ABSTRACT

Congestive heart failure is a clinical condition in which the heart cannot send enough blood to meet the body's metabolic needs due to pathological cardiac changes. In recent decades, a progressive increase in patients affected by this disease has been observed; this could be due to the increase in population age throughout the world, as it is a disease that mainly affects elderly individuals, a higher rate of general healing as a decrease in mortality results in a more significant number of patients affected by it, as well as more effective treatments for cardiac pathologies such as primary angioplasty in acute myocardial infarction, causing a more substantial number of patients with decreased cardiac function.

ARTICLE DETAILS

Published On:
31 October 2022

Available on:
https://ijmscr.org/

INTRODUCTION

Heart failure is the presence of signs and symptoms caused by functional and/or anatomical impairment of ventricular systole and/or blood ejection, clinical signs and symptoms in which the heart cannot satisfy the body's metabolic needs.[1]

Approximately 2% of the US population has congestive heart failure, most commonly systolic heart disease with reduced ejection fraction; the data show a higher prevalence in African Americans and Hispanics, which could indicate an etiological trend in certain ethnic groups; the incidence increases proportionally with age, an affectation of up to 10% is observed in individuals older than 60 years.[2. 3. 4]

Cardiac output, stroke volume multiplied by heart rate, is determined by three factors: preload, afterload, and ventricular contractility.[1]

Heart failure can be reduced ejection fraction (HFrEF, HF systolic) or preserved ejection fraction (HFpEF, HF diastolic). Heart failure with reduced ejection fraction is congestive heart failure with reduced volume, which reduces the ejection fraction; left ventricular ejection fraction is ≤35–40%, heart failure with preserved ejection fraction is congestive heart failure with reduced stroke volume, normal/reduced end-diastolic volume, and preserved ejection fraction (LVEF≥ 40–50%).[1, 2]

It can also be divided into right or left heart failure, depending on which ventricle has the dysfunction. Right heart failure is caused due to a dysfunction of the right ventricle that produces congestion in peripherin the vena cava and peripheral veins, which leads to an increase in venous hydrostatic pressure and makes peripheral edema, increased pressure jugular vein, ascites, and liver affectation. Left heart failure is caused by left ventricular dysfunction that results in tissue hypoperfusion and increased pulmonary capillary pressure. It can exist in both natures, right or left, which is known as biventricular (or global) heart failure [1,2]

Suppose a patient has echocardiographic signs obtained incidentally. If the patient is asymptomatic or stable symptomatic, a chronic compensated HF diagnosis is considered. At the same time, acute deterioration of HF or the onset of severe HF due to a critical heart condition such as myocardial infarction is acute decompensated heart failure.[1, 2]

ETIOLOGY

The leading causes of heart failure are mainly cardiometabolic diseases, including coronary arteries disease, systemic arterial hypertension, diabetes mellitus, valvular heart disease, kidney disease, and infiltrative diseases (amyloidosis). However, patients usually have more risk factors contributing to the disease's development, such as obesity, smoking, COPD, drugs, and alcoholism.[4]

Specific causes of heart failure of the systolic dysfunction type (reduced EF) can be caused by dilated cardiomyopathy (generally due to chronic alcohol consumption, idiopathic or Chagas disease), cardiac arrhythmias, and myocarditis. Moreover, specific causes for heart failure with diastolic dysfunction (preserved EF) can be restrictive

cardiomyopathy, hypertrophic cardiomyopathy, cardiac tamponade, or constrictive pericarditis.[4, 5]

CLINICAL FEATURES

Remembering that there are different modalities of heart failure, we can highlight general clinical characteristics such as nocturia, fatigue (see below NYHA classification), tachycardia, arrhythmias, auscultation of cardiac foci shows S3/S4 in gallop, as well as alternating pulse and the presence of cachexia in some patients. [6, 7]

The classic features of heart failure with systolic dysfunction are symptoms of pulmonary congestion such as breathing difficulty, orthopnea, pulmonary edema, paroxysmal nocturnal dyspnea (characterized by acute nocturnal attacks of cough and dyspnea, caused by reabsorption of peripheral edema during the night, resulting in increased venous return), as well as cardiac asthma with asthma-like symptoms, with dyspnea, wheezing and cough, this as a result of increased pressure in the bronchial arteries, which increase airway compression and bronchospasm. On physical examination, bilateral basilar rales may be found during field auscultation, laterally displaced apical heartbeat, and evidence of peripheral hypoperfusion (coldness, pallor, and decreased pulses) [6,7,8]

Diastolic heart failure's clinical features are symptoms of fluid retention and increased central venous pressure, such as peripheral fovea-positive edema resulting from fluid transudation due to increased venous pressure. Appreciate symptoms of hepatic venous congestion such as abdominal pain and jaundice, nausea, and hyporexia/anorexia. During the physical examination, it is possible to see jugular venous distention, Kussmaul's sign, hepatosplenomegaly that can cause cirrhosis, and hepatojugular reflux. [6, 8]

maintain the glomerular filtration rate. The compensatory mechanism for aldosterone involves renal reabsorption of Na+ and H2O, which increases preload. Brain natriuretic peptide secretion is a hormone secreted by the ventricular myocyte at the time of increased filling and ventricular stretching, which helps to decrease the wedge pressure of the pulmonary capillary.[6, 7, 8, 9]

Correct classification of heart failure is essential because its medical treatment is based on it, and a higher success rate depends on it. Although there are different classifications, the most used are the AHA classification (Table 1), which is based on physical and structural changes of the heart, and the NYHA (Table 2), which classifies heart failure depending on the patient's symptom stage. [1]

AHA Classification [1]

Table 1. AHA classification. Congestive Heart Failure.

Stage	Description
Stage A	High risk of developing heart failure (pre-existing high blood pressure, coronary artery disease, diabetes mellitus) and no structural cardiac changes or symptoms.
Stage B	There is a structural damage to the heart (heart attack scars, dilation, hypertrophy) and no signs or symptoms of heart failure.
Stage C	Structural damage to the heart and signs or symptoms of heart failure.
Stage D	End-stage heart failure.

NYHA Classification[1]

Table 2. NYHA classification. Congestive Heart Failure.

Class	Signs and symptoms
Class I	No physical activity limitations and no symptoms of heart failure.
Class II	Slight limitations of moderate physical activity and comfortable at rest.
Class III	Marked limitations in physical activity (symptoms during daily activities such as dressing and walking around rooms) and comfortable only at rest.
Class IV	Discomfort during any form of physical activity and symptoms at rest.

High-output heart failure or heart failure secondary to organic dysfuction that generate a high-output state, in which cardiac output rises to meet the oxygen-metabolic demands of the tissues. It is caused due to peripheral vasodilation or arteriovenous shunt, causing a decrease in systemic vascular resistance and, thus, an increase in heart rate and stroke volume, which increases cardiac output. [10]

The conditions that lead to an increase in cardiac demand and, therefore, a state of high output is varied and can range from physiological such as exercise, pregnancy, and even fever, to secondary to an underlying pathology, such as morbid obesity, cirrhosis in advanced stages, severe anemia, systemic arteriovenous fistulas, Paget's disease, hypothyroidism, vitamin B1 deficiency, sepsis, myeloma, glomerulonephritis, carcinoid heart disease and/or other types of cancer. [10]

Symptoms are almost entirely shared with low-output heart failures, such as dyspnea, tachypnea, tachycardia, peripheral edema, fatigue, low blood pressure, and high-output symptoms such as mid-systolic murmur, S3 gallop, jugular distention with audible buzz, pulsatile tinnitus and binding peripheral pulses. The diagnosis is clinical, although an X-ray

or echocardiogram showing cardiomegaly suggests the disease. Treatment is symptom management and hemodynamic stabilization, as well as treatment of the underlying cause.[10]

DIAGNOSIS

Although many times the diagnosis can be made clinically, the most common differential diagnoses, such as COPD or pneumonia, can cause diagnostic confusion, so laboratory studies must be performed (CBC, blood chemistry with creatinine, sodium, glucose, liver chemistry, CRP, lipid profile, thyroid profile), cardiac biomarkers, electrocardiogram, chest x-ray, and echocardiogram, to establish a precise diagnosis, once the diagnosis of heart failure is confirmed, the underlying cause should be investigated (consider coronary angiography, imaging chest, and advanced cardiac imaging), and identification of modifiable risks (hypertension, coronary artery disease). [1, 12]

Once the laboratory studies have been requested, the findings are variable. Anemia or infection should be sought in the blood count based on hemoglobin levels or white formula levels; elevated creatinine would indicate renal changes, being important for the cardiorenal syndrome, hyponatremia is an indicator of poor prognosis, and fasting glucose as an indicator of risk factors such as diabetes. Altered liver chemistry could indicate hepatic venous congestion. Inflammatory markers such as elevated CRP indicate an infection or acute inflammation. The patient's lipid profile should be investigated and subsequently corrected as necessary. The thyroid profile is important due to the nature of its alterations.[1, 12, 13, 14]

Cardiac biomarkers should be requested in patients with suspected heart failure, brain natriuretic peptide (BNP) or brain natriuretic peptide N-terminal prohormone (NT-proBNP), it is indicated to request to establish the diagnosis of heart failure together with echocardiography, in addition to the evaluation of the prognosis, probability of diagnosis (Table 3), and the severity of the disease (taking the difference between their admission levels and their levels before discharge). [14. 15, 16, 17, 18]

Peptide biomarkers, the diagnostic probability for heart failure.18

Table 3.

peptide	Values			
BNP	<100pg/mL	Unlikely	>500pg/mL	Probable
NT-proBNP	<300pg/mL	Unlikely	>1000pg/mL	Probable

Measurement of BNP (or NT-proBNP) is instrumental in patients with unclear diagnoses. When combined with a physical exam and imaging, BNPg, has a high value at the diagnostic. A low BNP (or NT-proBNP) makes diagnosing acute heart failure highly unlikely in a patient with acute dyspnea. [1, 15, 16, 18]

Cardiac troponin T or I can show high levels, which suggests ischemia. However, they can also be elevated in heart failure without acute myocardial ischemia, and their values are potentially useful for risk stratification.[1, 15, 16, 17, 18, 19, 20]

Transthoracic echocardiography is indicated in all patients with suspected new-onset heart failure. It should also be performed in patients undergoing treatment with changes in the clinical characteristics of their disease. And for evaluation for device therapy. [18, 19, 20]

The classic findings that can be observed range from the characteristics of ventricular dysfunction when evaluated by LVEF (Table 4). [21]

Table 4. Classification by Ejection Fraction. [21]

Heart failure	Left ventricular ejection fraction	Classification
Normal	50-70%	Preserved/Normal
HFpFE	41-49%	preserved
HFrFE	<40%	reduced

As well as pericardial and pleural effusion, evidence of complications such as cardiac asynchrony, functional mitral regurgitation, or left atrial enlargement, and underlying causes such as local coronary artery wall motion abnormalities left ventricular hypertrophy due to systemic hypertension, or abnormalities of flow through the heart valves.[21, 22, 23]

Chest X-ray is indicated in case of new-onset heart failure or due to high suspicion, changes in the cardiac silhouette can be observed, from a cardiothoracic width ratio >0.5, boot-shaped heart seen in PA and left Ventricular enlargement, as well as signs of pericardial effusion. Findings of pulmonary congestion are not uncommon to observe, and valvular or pericardial cardiac calcifications are often recurrent.[1]

Electrocardiogram abnormalities are common but nonspecific, ranging from left ventricular hypertrophy, left axis deviation, ST-T, or P wave abnormalities to prolonged QT interval or complete or left bundle branch block incomplete can be observed. Recurrent changes due to pre-existing cardiac disease are ischemic changes from

myocardial infarction, arrhythmias, or pericardial effusion.[24, 25]

The diagnostic tests for heart failure have already been exposed. However, without limiting ourselves to the first instance tests, additional studies can be helpful when there is diagnostic uncertainty and as a diagnosis of underlying causes. There are advanced cardiac images, such as cardiac magnetic resonance imaging, which is the gold standard for evaluating ventricular volume, mass, and ejection fraction. Indications include diagnostic not being clear after echocardiography, investigating congenital heart disease in adults, or determining myocardial scar burden—alternatively, PET myocardial perfusion imaging as an alternative non-invasive study for suspected coronary artery disease. Right heart catheterization assesses proper heart function and pulmonary vascular resistance in patients considered for transplant mechanical circulatory advanced support. Blood pressure monitoring and electrocardiographic monitoring in patients with suspected systemic arterial hypertension or Holter monitoring in suspected paroxysmal atrial fibrillation or other arrhythmias.[1]

TREATMENT

The initial treatment for uncomplicated congestive heart failure begins with modifying the patient's lifestyle; these changes reduce the risk factors associated with the progression of heart failure and other cardiometabolic comorbidities such as diabetes mellitus and hypertension. These lifestyle changes will be specific to each case. However, in general terms, we can mention that aerobic exercise, smoking cessation, alcoholism, drug use, and weight loss are essential to reduce the progression of the disease. Should consider states of immunosuppression in patients with comorbid conditions, requiring immunization with pneumococcal and seasonal influenza vaccines, adapting to each case depending on national epidemiological recommendations. [1, 15, 21, 26]

Initial pharmacological treatment of heart failure.[8, 15]

It is important to make the patient understand the pathophysiological bases of their disease, which makes the treatment efficacy and, therefore, their quality of life substantially improve; salt restriction ranges from <3g/day to <1.5g/day, Depending on the stage where the patient is, foods rich in potassium should be avoided during the administration of aldosterone antagonists, and fluids should be limited to 1.5-2 L per day in patients with stage D, who present with edema and/or hyponatremia. The patient must control and recognize the symptoms from daily weight control; if there is an increase of >2kg in less than three days, the patient must return to the doctor to evaluate the use of diuretics. The patient must be able to recognize symptoms of worsening heart failure, such as increased dyspnea at lower exertion than before, and must recognize new symptoms suggestive of medication side effects. It is recommended to carry a copy of the medical records and avoid destinations with limited medical attention. [1, 26, 27]

Furthermore, in the case of significant atherosclerosis, revascularization should be considered. In the case of anemia, specific treatment for each type of anemia should be given to all patients with NYHA class II and III symptoms. [1, 27, 28, 29, 30]

Most antiarrhythmic agents should be avoided, as well as potassium channel blockers, except amlodipine (simultaneous use of calcium channel blockers with beta-blockers can cause complete heart block), thiazolidinediones, and anesthetics. Inhaled antidepressants should also be avoided, and due to the increased incidence of depression in patients of all age groups in recent years, careful selection of antidepressants should be made. [1, 15, 26, 29, 31]

Pharmacological treatment for heart failure is based on the stage of the disease in which the patient is present (Table 5), adding additional therapies to the treatment as symptoms worsen. The recommendations call for gradual initiation of all medications, starting with the lowest recommended dose and slowly increasing the dose, with each visit, up to the target dose. [1, 8, 15, 26, 29, 32, 33]

Table 5. ACEs; angiotensin-converting enzyme inhibitors. BRA; Angiotensin receptor blockers. B-Blockers; Beta receptor blockers.

Stage A			
Treatment of cardiovascular and comorbid risk factors and medications for heart failure are not routinely indicated.			
Pharmacological Class	examples	Indications	Surveillance
Stage B			
ACEIs	Enalapril Captopril Lisinopril	All patients with HFrFE	Blood pressure Renal function, Potassium 2 weeks after initiation or dose change
BRA	losartan Valsartan	Patients who do not tolerate ACE inhibitors	Blood pressure Renal function Potassium 2 weeks after initiation or dose change

B-Blockers	metoprolol Bisoprolol	Add after patient stabilization with the antihypertensives above	Increase the dose from the minimum recommended to the target; if hypotension occurs, consider reducing the number of antihypertensives mentioned above to adapt to beta blockers.
Stage C			
aldosterone antagonist	Spironolactone Eplerenone	All patients with HFrEF with NYHA II-IV symptoms and an LVEF <35%. Evaluate its use in patients with HFpEF.	Regular monitoring of potassium, the tendency to hyperkalemia.
diuretics	Handle Furosemide Bumetanide Thiazides Hydrochlorothiazide	For all patients with fluid retention and volume overload, thiazides, as a synergistic effect, do not use as ba as treatment.	Evaluate the dose according to weight, volume, and hemodynamic stability with periodic review of serum electrolytes.
Isosorbide and Hydralazine	isosorbide Hydralazine	Patients who do not tolerate ACE inhibitors or ACEs used in Afro-descendants.	Separate dosage of both drugs and monitor volume depletion and hypotension.
Stage D			
Additional measures to the treatment above: For patients with Stage D Heart Failure, invasive intervention or a palliative care approach should be considered. Continuous intravenous inotropic support should be considered a bridge before heart transplantation or mechanical circulatory support.			

Table 6. Drugs for the treatment of chronic heart failure with decreased Ejection Fraction. Initial dose and maximum dose. [8]

Drug	Initial Dose	Maximum dose
diuretics		
Furosemide	20-40 mg every 24 hours every 12 hours	400mg/day
Bumetanide	0.5-1.0 mg every 24 hours/12h	10mg/day
Hydrochlorothiazide	25mg every 24h	100mg/day
Angiotensin-converting enzyme inhibitors		
Enalapril	2.5 mg every 12 hours	50mg q8h
Captopril	6.25 mg every 8 hours	10-20mg every 12h
Lisinopril	2.5-5.0 mg every 24 hours	20-40mg every 24h
Angiotensin receptor antagonists		
losartan	25-50mg every 24h	150mg every 24h
Valsartan	40 mg every 12 hours	160mg q12h
B receptor antagonists		
Bisoprolol	1.25mg every 24h	10mg every 24h
Metoprolol Succinate CR	12.5-25mg every 24h	200mg every 24h
Aldosterone antagonists		
Spironolactone	12.5-25mg every 24h	25-50mg every 24h
Eplerenone	25mg every 24h	50mg every 24h
Additional Drugs		
Combination of hydralazine and isosorbide dinitrate	10-25mg/10mg q8h	75mg/40mg q8h

The use of Digoxin should be considered in patients with HFrFE with persistent symptoms and refractory to first-line treatment; renal function should be closely monitored. [1, 8, 15, 26, 29, 30, 31]

To consider the use of cyclic nucleotide-modulated hyperpolarization-activated channel blockers (ivabradine), all of the following must be present:

- NYHA class II-III
- HFrEF <35%
- Sinus rhythm with HR >70bpm at rest with the maximum tolerated dose of B-Blockers.

Patients who present arrhythmias such as ventricular tachycardia or fibrillation, in combination with heart failure, can cause symptomatic worsening and increase the risk of sudden cardiac death; these patients are candidates for

invasive interventions, devices with a pacemaker, and/or defibrillator functions. [34, 35]

The implantable cardioversion defibrillator works by administering an electrical shock, restoring sinus rhythm if an arrhythmia such as ventricular fibrillation or ventricular tachycardia is detected. [34, 35, 36, 37, 38]

The indications for its placement are for patients who previously presented sustained ventricular tachycardia or cardiac arrest secondary to ventricular fibrillation or tachycardia, as well as patients with HFrEF with expected survival > one year if they receive medical treatment for 3-6 months and still comply with any of the following criteria:

- Stage B with ischemic cardiomyopathy if LVEF is >30%
- Stage C with dilated cardiomyopathy or ischemic heart disease with LVEF >35% and NYHA class II-III symptoms [34, 35, 36, 37, 38]

In patients with end-stage heart failure, heart transplantation is the only cure. Unfortunately, most patients are not candidates. Patients accepted for transplantation generally require bridge measures, such as inotropic and/or mechanical circulatory support. Any patient with end-stage heart failure who is not a transplant candidate should be referred for palliative care. [1, 8, 37, 38]

COMPLICATIONS

Decompensated heart failure is the worsening of heart failure symptoms. It is the most common cause of hospitalization for heart failure complications and one of the most common causes of hospitalization in older adults. Multiple causes can trigger pre-existing acute cardiac decompensation, but It can also occur in patients without a history of a heart condition. The diagnosis is based on typical clinical features as well as imaging findings. Management can be complicated because multiple comorbidities often accompany it. Most patients require treatment with diuretics, vasodilators, respiratory support, medications for underlying heart failure, and careful fluid management. [39, 40, 41]

Other common complications are:

- Cardiorenal syndrome
- Cardiac arrhythmias
- Cardiogenic shock
- Cerebrovascular accident (Usually due to thromboembolism)
- Cardiac cirrhosis (right CHF)
- venous stasis

CARDIORENAL SYNDROME

It is a complex syndrome in which renal function is progressively diminished as a result of significant cardiac dysfunction; it occurs in 30% of patients with acute decompensated heart failure; its pathophysiology depends on which side of the heart the dysfunction is, in the case of systolic dysfunction, cardiac output is decreased, causing renal hypoperfusion, which would cause prerenal renal failure; in the case of diastolic dysfunction, it causes systemic venous and renal venous congestion, which decreases the gradient of transglomerular pressure, a decrease in glomerular filtration rate and consequently a decrease in renal function. The AHA has classified it into five types: Type I: Acute cardiorenal síndrome is when the heart failure leads to acute kidney injury.

- Type II: Chronic cardiorenal syndrome. Chronic heart failure leads to chronic kidney disease.
- Type III: Acute renocardiac syndrome. Acute kidney injury leads to acute heart failure.
- Type IV: Chronic renocardiac syndrome. Chronic kidney disease leads to chronic heart failure.
- Type V: Secondary SRC. Systemic disease leads to kidney and heart failure.

Suspicion of cardiorenal syndrome is classically made when a patient with heart failure presents with decreased glomerular filtration rate and increased creatinine that cannot be explained by underlying kidney disease. The treatment is to treat heart failure and nephroprotective measures. The prognosis depends on laboratory levels; in the case of creatinine >3mg/dl, it is associated with a poor prognosis.[1, 15, 39, 40, 41]

PROGNOSIS

The prognosis in patients with heart failure is poor unless the cause is corrected. However, it depends mainly on the patient, the type and severity of heart disease, medication regimens, and lifestyle changes. The of patients with preserved EF is better than those with decreased EF, and worse prognostic factors are elevated BNP, hyponatremia, systolic BP <120mmHg, diabetes, anemia, weight loss or low weight, S3, use implantable cardioverter-defibrillator and frequent hospitalizations for heart failure. [1, 42]

1-year survival according to NYHA stage:

- Class I: 95%
- Class II: 85%
- Class III: 85%
- Class IV: 35%

CONCLUSION

Heart Failure is an important public health problem, so the therapeutic approach must be multidisciplinary to impact all phases of the syndrome positively; that is why currently, the world trend is toward creating Heart Failure Clinics that are defined as specialized services for timely diagnosis and treatment of HF but also develop advanced research and educational programs to treat patients comprehensively.

BIBLIOGRAPHY

I. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, et al. (2013). ACCF/AHA guideline for managing heart failure: a report of the American College of Cardiology

Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol [Internet]. 2013;62(16):e147-239. Availablefrom: http://dx.doi.org/10.1016/j.jacc.2013.05.019

II. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart disease and stroke statistics-2020 update: A report from the American Heart Association: A report from the American Heart Association. Circulation [Internet]. 2020;141(9):e139–596. Available from: http://dx.doi.org/10.1161/CIR.0000000000000757

III. Bahrami H, Kronmal R, Bluemke DA, Olson J, Shea S, Liu K, et al. Differences in the incidence of congestive heart failure by ethnicity: the multi-ethnic study of atherosclerosis. Arch Intern Med [Internet]. 2008;168(19):2138–45. Available from: http://dx.doi.org/10.1001/archinte.168.19.2138

IV. cdc.gov. [cited 2022 Jul 31]. Available from: https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_heart_failure.htm

V. Jia G, Hill MA, Sowers JR. Diabetic cardiomyopathy: An update of mechanisms contributing to this clinical entity. Circ Res [Internet]. 2018;122(4):624–38. Available from: http://dx.doi.org/10.1161/CIRCRESAHA.117.311586

VI. Barker JC, Mitteness LS. Shedding light on nocturia. Geriatr Nurs [Internet]. 1989 [cited 2022 Jul 31];10(5):239–40. Available from: https://www.ncbi.nlm.nih.gov/books/NBK293/

VII. Okoshi MP, Capalbo RV, Romeiro FG, Okoshi K. Cardiac cachexia: Perspectives for prevention and treatment. Arch Bras Cardiol [Internet]. 2016; Available from: http://dx.doi.org/10.5935/abc.20160142

VIII. Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. Harrison's principles of internal medicine vol 1 20/E (book). 20th ed. McGraw-Hill Education/Medical; 2018.

IX. Colucci WS. Evaluation of the patient with suspected heart failure. Post TW, editor.

X. Givertz MM, Haghighat A. High-output heart failure. Post TW, editor.

XI. Filippatos TD, Elisaf MS. Hyponatremia in patients with heart failure. World J Cardiol [Internet]. 2013;5(9):317–28. Available from: http://dx.doi.org/10.4330/wjc.v5.i9.317

XII. Rosano GM, Vitale C, Seferovic P. Heart failure in patients with diabetes mellitus. Card Fail Rev [Internet]. 2017;3(1):52–5. Available from: http://dx.doi.org/10.15420/cfr.2016:20:2

XIII. Hilscher M, Sanchez W. Congestive hepatopathy: Congestive hepatopathy. Clin Liver Dis (Hoboken) [Internet]. 2016;8(3):68–71. Available from: http://dx.doi.org/10.1002/cld.573

XIV. Poelzl G, Ess M, Mussner-Seeber C, Pachinger O, Frick M, Ulmer H. Liver dysfunction in chronic heart failure: prevalence, characteristics, and prognostic significance: LIVER DYSFUNCTION IN CHRONIC HEART FAILURE. Eur J Clin Invest [Internet]. 2012;42(2):153–63. Available from: http://dx.doi.org/10.1111/j.1365-2362.2011.02573.x

XV. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Colvin MM, et al. (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. Circulation [Internet]. 2017;136(6):e137–61. Available from: http://dx.doi.org/10.1161/CIR.0000000000000509

XVI. Suzuki S, Sugiyama S. The molar ratio of N-terminal pro-B-type natriuretic peptide/B-type natriuretic peptide for heart failure-related events in stable outpatients with cardiovascular risk factors. Intern Med [Internet]. 2018; Available from: http://dx.doi.org/10.2169/internalmedicine.0471-17

XVII. Chen Y, Wen Z, Peng L, Liu X, Luo Y, Wu B, et al. Diagnostic value of MR-proANP for heart failure in patients with acute dyspnea : a meta-analysis. Acta Cardiol [Internet]. 2020;75(1):68–74. Available from: http://dx.doi.org/10.1080/00015385.2018.1550887

XVIII. Chow SL, Maisel AS, Anand I, Bozkurt B, de Boer RA, Felker GM, et al. Role of biomarkers for the prevention, assessment, and management of heart failure: A scientific statement from the American Heart Association. Circulation [Internet]. 2017;135(22):e1054–91. Available from: http://dx.doi.org/10.1161/CIR.0000000000000490

XIX. Hudson MP, O'Connor CM, Gattis WA, Tasissa G, Hasselblad V, Holleman CM, et al. Implications of elevated cardiac troponin T in ambulatory patients with heart failure: a prospective analysis. Am Heart J [Internet]. 2004;147(3):546–52. Available from: http://dx.doi.org/10.1016/j.ahj.2003.10.014

XX. Wettersten N, Maisel A. Role of cardiac troponin levels in acute heart failure. Card Fail Rev [Internet]. 2015;1(2):102–6. Available from: http://dx.doi.org/10.15420/cfr.2015.1.2.102

XXI. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. (2016). ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the exceptional contribution of the Heart Failure Association (HFA)) of

the ESC. Eur Heart J [Internet]. 2016;37(27):2129–200. Available from: http://dx.doi.org/10.1093/eurheartj/ehw128

XXII. Salerno M, Sharif B, Arheden H, Kumar A, Axel L, Li D, et al. Recent advances in cardiovascular magnetic resonance: Techniques and Applications: Techniques and applications. Circ Cardiovascular Imaging [Internet]. 2017;10(6). Available from: http://dx.doi.org/10.1161/CIRCIMAGING.116.003951

XXIII. Omar AMS, Bansal M, Sengupta PP. Advances in echocardiographic imaging in heart failure with reduced and preserved ejection fraction. Circ Res [Internet]. 2016;119(2):357–74. Available from: http://dx.doi.org/10.1161/CIRCRESAHA.116.309128

XXIV. Natanzon A, Kronzon I. Pericardial and pleural effusions in congestive heart failure-anatomical, pathophysiologic, and clinical considerations. Am J Med Sci [Internet]. 2009;338(3):211–6. Available from: http://dx.doi.org/10.1097/MAJ.0b013e3181a3936f

XXV. McDowell K, Petrie MC, Raihan NA, Logue J. Effects of intentional weight loss in patients with obesity and heart failure: a systematic review. Obes Rev [Internet]. 2018;19(9):1189–204. Available from: http://dx.doi.org/10.1111/obr.12707

XXVI. Department of Health and Human Services and US Department of Agriculture. Dietary guidelines for Americans. 2015.

XXVII. Izadi M, Alemzadeh-Ansari MJ, Kazemisaleh D, Moshkani-Farahani M. Air travel considerations for the patients with heart failure. Iran Red Crescent Med J [Internet]. 2014;16(6):e17213. Available from: http://dx.doi.org/10.5812/ircmj.17213

XXVIII. Travel and heart disease [Internet]. www.heart.org. [cited 2022 Jul 31]. Available from: https://www.heart.org/en/health-topics/consumer-healthcare/what-is-cardiovascular-disease/travel-and-heart-disease

XXIX. Zannad F, Ferreira JP, Pocock SJ, Anker SD, Butler J, Filippatos G, et al. SGLT2 inhibitors in patients with heart failure with reduced ejection fraction: a meta-analysis of the EMPEROR-Reduced and DAPA-HF trials. Lancet [Internet]. 2020;396(10254):819–29. Available from: http://dx.doi.org/10.1016/S0140-6736(20)31824-9

XXX. von Haehling S, Ebner N, Evertz R, Ponikowski P, Anker SD. Iron deficiency in heart failure: An overview. JACC Heart Fail [Internet]. 2019;7(1):36–46. Available from: http://dx.doi.org/10.1016/j.jchf.2018.07.015

XXXI. Guck TP, Elsasser GN, Kavan MG, Barone EJ. Depression and congestive heart failure. Congest Heart Fail [Internet]. 2003;9(3):163–9. Available from: http://dx.doi.org/10.1111/j.1527-5299.2003.01356.x

XXXII. Richards TR, Tobe SW. Combining other antihypertensive drugs with β-blockers in hypertension: a focus on safety and tolerability. Can J Cardiol [Internet]. 2014;30(5 Suppl): S42-6. Available from: http://dx.doi.org/10.1016/j.cjca.2013.08.012

XXXIII. Allen LA, Fang JC. Goldilocks dilemma of dose titration in heart failure with reduced ejection fraction: Too little, too much, or just right?: Too little, too much or just right? Circ Heart Fail [Internet]. (2017);10(8). Available from: http://dx.doi.org/10.1161/CIRCHEARTFAILURE.117.004406

XXXIV. Leyva F, Zegard A, Acquaye E, Gubran C, Taylor R, Foley PWX, et al. Outcomes of cardiac resynchronization therapy with or without defibrillation in patients with nonischemic cardiomyopathy. J Am Coll Cardiol [Internet]. 2017;70(10):1216–27. Available from: http://dx.doi.org/10.1016/j.jacc.2017.07.712

XXXV. Peal JE, Mathews IG, Runnett C, Thomas HE, Ripley DP. An update on cardiac implantable electronic devices for the general physician. JR Coll Physicians Edinb [Internet]. 2018;48(2):141–7. Available from: http://dx.doi.org/10.4997/JRCPE.2018.208

XXXVI. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NAM 3rd, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: A report of the American college of cardiology/American heart association task force on practice guidelines (writing committee to revise the ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices): Developed in collaboration with the American association for thoracic surgery and society of thoracic surgeons: A report of the American college of cardiology/American heart association task force on practice guidelines (writing committee to revise the ACC/AHA /NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices): developed in collaboration with the American association for thoracic surgery and society of thoracic surgeons. Circulation [Internet]. 2008;117(21):e350-408. Available from: http://dx.doi.org/10.1161/CIRCUALTIONAHA.108.189742

XXXVII. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, et al. (2013). ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA): The Task Force on cardiac

pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J [Internet]. 2013;34(29):2281–329. Available from: http://dx.doi.org/10.1093/eurheartj/eht150

XXXVIII. Mechanical circulatory support in a nutshell [Internet]. American College of Cardiology. [cited 2022 Jul 31]. Available from: https://www.acc.org/latestincardiology/articles/2015/11/12/09/28/mechanical-circulatory-support-in-a-nutshell

XXXIX. Aronson D. Cardiorenal syndrome in acute decompensated heart failure. Expert Rev Cardiovasc Ther [Internet]. 2012;10(2):177–89. Available from: http://dx.doi.org/10.1586/erc.11.193

XL. Rangaswami J, Bhalla V, Blair JEA, Chang TI, Costa S, Lentine KL, et al. Cardiorenal syndrome: Classification, pathophysiology, diagnosis, and treatment strategies: A scientific statement from the American heart association. Circulation [Internet]. 2019;139(16):e840–78. Available from: http://dx.doi.org/10.1161/CIR.0000000000000664

XLI. Mahon NG, Blackstone EH, Francis GS, Starling RC 3rd, Young JB, Lauer MS. The prognostic value of estimated creatinine clearance alongside functional capacity in ambulatory patients with chronic congestive heart failure. J Am Coll Cardiol [Internet]. 2002;40(6):1106–13. Available from: http://dx.doi.org/10.1016/s0735-1097(02)02125-3

XLII. UpToDate. ASAMonitor [Internet]. 2021 [cited 2022 Jul 31];85(10):23–23. Available from: https://www.uptodate.com/contents/prognosis-of-heart-failure