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# **New Modalities in Diagnosis and Management of Heart Failure**

*Essay*

**Submitted in partial fulfillment of Master  
Degree in Intensive Care**

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2017**

## **Abstract**

**Introduction:** Heart failure (HF) was classically viewed as synonymous with left ventricular pump dysfunction, usually progressive, resulting in a common end-stage cardiac phenotype of dilation, thinned walls, and poor contractility.

New modalities in management of are promising as Angiotensin receptor-neprilysin inhibition with LCZ696, new aldosterone receptor blockers, serelaxin, ularitide, etc

**Aims:** The aim of the essay is to throw a light on new modalities used for diagnosis and management of heart failure.

**Summary:** Heart failure (HF) is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood. The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to pulmonary and/ or splanchnic congestion and/or peripheral edema.

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**Keywords:** New Modalities, Diagnosis, Management, Heart Failure


بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

# قَالَ

سَبَّحَانَكَ لَا مَعْلَمَ لَنَا  
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ  
الْعَلِيمُ الْعَظِيمُ

صدق الله العظيم

سورة البقرة الآية: ٣٢



# ACKNOWLEDGEMENT

First of all, thanks to **Allah** whose magnificent help was the main factor in completing this work.

No words can express my deep sincere feelings towards **Prof. Dr. Alaa El-Din Abdel Wahab Korraa** Professor of Anesthesiology, Intensive Care and Pain, Faculty of Medicine-Ain Shams University for his continuous encouragement, guidance and support she gave me throughout the whole work. It has been a great honor for me to work under his generous supervision.

Special thanks are due to **Dr. Ehab Hamed Abdel Salam**, Assistant Professor of Anesthesiology, Intensive Care and Pain, Faculty of Medicine-Ain Shams University, for his sincere efforts, fruitful encouragement.

I would like to express my deepest gratitude for **Dr. Mohammed Sayed Shorbagy**, Lecturer of Anesthesiology, Intensive Care and Pain, Ain Shams University, for his great help and sincere advice in performing this thesis.

Last but not least, sincere gratitude to *My Family* for their continuous encouragement and spiritual support.

# Contents

| Subjects   | Page |
|--|------|
| List of abbreviations.....   | II   |
| List of figures.....   | VI   |
| List of tables.....  | VII  |
| • <b>Introduction</b> .....  | 1    |
| • <b>Aim of the work</b> .....   | 4    |
| • <b>Chapter (1): Pathophysiology of Heart Failure</b> .....                           | 5    |
| • <b>Chapter (2): Evaluation and Management of Heart Failure</b> .....                 | 23   |
| • <b>Chapter (3): Update in Diagnosis and Management of Heart Failure</b> .....        | 54   |
| • <b>Chapter (4): Importance and Management of Co-morbidity in Heart Failure</b> ..... | 76   |
| • <b>Summary</b> .....   | 93   |
| • <b>References</b> .....  | 96   |
| • <b>Arabic Summary</b>  |      |

## **List of Abbreviations**

|               |  |
|---------------|--|
| <b>ACC</b>    | : American College of Cardiology             |
| <b>ACE</b>    | : Angiotensin converting enzyme              |
| <b>ACE-I</b>  | : Angiotensin converting enzyme inhibitor    |
| <b>ACS</b>    | : Acute coronary syndrome                    |
| <b>ADHF</b>   | : Acute decompensated heart failure          |
| <b>AF</b>     | : Atrial fibrillation                        |
| <b>AHF</b>    | : Acute heart failure                        |
| <b>ANG</b>    | : Angiotensin                                |
| <b>ANP</b>    | : Atrial natriuretic peptide                 |
| <b>ARB-II</b> | : Angiotensin II receptors blockers          |
| <b>ARNI</b>   | : Angiotensin receptors-neprylisin inhibitor |
| <b>AT1</b>    | : Angiotensin type 1                         |
| <b>BMI</b>    | : Body mass index                            |
| <b>BNP</b>    | : Brain natriuretic peptide                  |
| <b>b.p.m</b>  | : Beat per minute                            |
| <b>BUN</b>    | : Blood urea nitrogen                        |
| <b>CABG</b>   | : Coronary artery bypass graft               |
| <b>CAD</b>    | : Coronary artery disease                    |
| <b>CBC</b>    | : Complete blood count                       |
| <b>CCB</b>    | : Calcium-channel blocker                    |
| <b>c GMP</b>  | : Cyclic guanosine monophosphate             |

## *List of Abbreviations*

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|              |  |
|--------------|--|
| <b>CHF</b>   | : Chronic heart failure                          |
| <b>CLI</b>   | : Critical limb ischemia                         |
| <b>CNP</b>   | : c-type natriuretic peptide                     |
| <b>CO</b>    | : Cardiac output                                 |
| <b>COMET</b> | : Carvedilol or Metoprolol European Trial        |
| <b>COPD</b>  | : Chronic obstructive pulmonary disease          |
| <b>CRP</b>   | : C-reactive protein                             |
| <b>CRT</b>   | : Cardiac resynchronization therapy              |
| <b>CV</b>    | : Cardiovascular                                 |
| <b>DC</b>    | : Dendritic cells                                |
| <b>ECG</b>   | : Electrocardiogram                              |
| <b>EF</b>    | : Ejection fraction                              |
| <b>ESC</b>   | : European Society of Cardiology                 |
| <b>GFR</b>   | : Glomerular filtration rate                     |
| <b>GTP</b>   | : Guanosine triphosphate                         |
| <b>HF</b>    | : Heart failure                                  |
| <b>HFNEF</b> | : Heart failure with normal ejection fraction    |
| <b>HFpEF</b> | : Heart failure with preserved ejection fraction |
| <b>HFrEF</b> | : Heart failure with reduced ejection fraction   |
| <b>HFSA</b>  | : Heart Failure Society of America               |
| <b>HIV</b>   | : Human immunodeficiency virus                   |

## *List of Abbreviations*

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**HMG CoA** : Hydroxymethylglutaryl-CoA

**HR** : Heart rate

**hs-CRP** : High-sensitivity C-reactive protein

**ICD** : Implantable cardioverter-defibrillator

**IV** : Intravenous

**JVD** : Jugular venous distension

**LFTs** : Liver function tests

**LV** : Left ventricular

**LVEDP** : LV End-Diastolic Pressure

**LVEF** : Left ventricular ejection fraction

**MAP** : Mean arterial pressure

**MI** : Myocardial infarction

**MRA** : Mineralocorticoid receptor antagonist

**NO** : Nitric oxide

**NP** : Natriuretic peptide

**NPR-A** : Neprylisin receptor-A

**NSAIDs** : Non steroidal anti inflammatory drugs

**NT-pro BNP**: N-terminal pro-B-type natriuretic peptide

**NYHA** : New York Heart Association

**PTX3** : Pentraxins

**PUFA** : Polyunsaturated fatty acids



## *List of Abbreviations*

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|                                 |   |
|---------------------------------|---|
| <b>RAAS</b>                     | : Renin–angiotensin– aldosterone system       |
| <b>RALES</b>                    | : Randomized aldactone evaluation study       |
| <b>RV</b>                       | : Right ventricular                           |
| <b>SAP</b>                      | : Serum amyloid P component                   |
| <b>SCD-HeFT</b>                 | : Sudden Cardiac Death in Heart Failure Trial |
| <b>SDF-1</b>                    | : Stromal cell-derived factor-1               |
| <b>SNS</b>                      | : Sympathetic nervous system                  |
| <b>SV</b>                       | : Stroke volume                               |
| <b>TNF- <math>\alpha</math></b> | : Tumor necrosis factor $\alpha$              |
| <b>TPR</b>                      | : Total peripheral resistance                 |
| <b>TSH</b>                      | : Thyroid stimulating hormone                 |
| <b>UA</b>                       | : Urine analysis                              |
| <b>U.S.</b>                     | : United States                               |
| <b>VEGF</b>                     | : Vascular endothelial growth factor          |
| <b>VT</b>                       | : Ventricular tachycardia                     |

## **List of Figures**

| <b><u>No.</u></b> | <b><u>Figure</u></b>  | <b><u>Page</u></b> |
|-------------------|---|--------------------|
| <b><u>1</u></b>   | Factors affecting Cardiac output.   | <b>11</b>          |
| <b><u>2</u></b>   | Left ventricular dysfunction.   | <b>13</b>          |
| <b><u>3</u></b>   | Compensatory mechanism in LV dysfunction.   | <b>20</b>          |
| <b><u>4</u></b>   | Hemodynamic profiles of patients presenting with advanced heart failure, as described by a $2 \times 2$ table.  | <b>28</b>          |
| <b><u>5</u></b>   | A man with congestive heart failure and marked jugular venous distension. External jugular vein marked by an arrow.   | <b>30</b>          |
| <b><u>6</u></b>   | Pedal edema during and after the application of pressure to the skin.   | <b>31</b>          |
| <b><u>7</u></b>   | Angiotensin receptor neprilysin inhibitors have the potential to modulate two counter-regulatory neurohormonal systems in HF: the renin– angiotensin–aldosterone system and natriuretic peptide system. | <b>69</b>          |

## **List of Tables**

| <b><u>No.</u></b> | <b><u>Table</u></b>  | <b><u>Page</u></b> |
|-------------------|--|--------------------|
| <b><u>1</u></b>   | Signs and symptoms of LV failure.  | <b>14</b>          |
| <b><u>2</u></b>   | Signs and symptoms of RV failure.  | <b>15</b>          |
| <b><u>3</u></b>   | HF is commonly classified using the New York Heart Association (NYHA) classification system.       | <b>22</b>          |
| <b><u>4</u></b>   | Essential topics in patient education with associated skills and appropriate self-care behaviours. | <b>37</b>          |

## Introduction

Heart failure (HF) was classically viewed as synonymous with left ventricular pump dysfunction, usually progressive, resulting in a common end-stage cardiac phenotype of dilation, thinned walls, and poor contractility (**Mann and Bristow, 2005**).

The prevalence of HF follows an exponential pattern, and it rises with age. Heart failure affects 6% to 10% of people over the age of 65 years. Although the relative incidence is lower in women than in men, women constitute at least half of the cases of HF because of their longer life expectancy. In the United States ( U.S.), the treatment of HF has a direct cost of over \$34 billion per year, most of which results from hospitalization (**Fang et al., 2008**).

Heart failure may be caused by myocardial failure but may also occur in the presence of near-normal cardiac function under conditions of high demand. Heart failure always causes circulatory failure, but the converse is not necessarily the case, because various noncardiac conditions (eg, hypovolemic shock, septic shock) can produce circulatory failure in the presence of normal, modestly

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impaired, or even supranormal cardiac function. To maintain the pumping function of the heart, compensatory mechanisms increase blood volume, cardiac filling pressure, heart rate, and cardiac muscle mass. However, despite these mechanisms, there is progressive decline in the ability of the heart to contract and relax, resulting in worsening heart failure (**O'Riordan, 2014**).

Most patients admitted to the hospital with HF have a worsening of chronic HF, although 15% to 20% of HF hospitalizations represent new diagnoses of HF. Patients with a new diagnosis of HF are much more likely to present with pulmonary edema or cardiogenic shock, while decompensation of chronic HF usually presents with other signs of congestion and fluid retention, such as weight gain, exertional dyspnea, or orthopnea. These symptoms can begin days or weeks before presentation. A history of coronary artery disease (CAD) is present in 60% of patients, hypertension in 70%, diabetes in 40%, and renal impairment in 20% to 30%. At presentation, most HF patients are relatively normotensive. Patients admitted with HF having a relatively preserved left ventricular ejection fraction (LVEF) tend to be older, female, and more likely to present with severe hypertension (**Fonarow et al., 2007**).

Evidence-based treatment of heart failure includes Angiotensin-Converting Enzyme Inhibitors (ACE-I), Angiotensin II Receptor Blockers (ARB-II's),  $\beta$ -blockers, diuretics, aldosterone antagonists, and digitalis. They have shown their efficacy in improving the symptoms and natural history of heart failure independently of its cause. Diuretic therapy is certainly effective in improving symptoms in heart failure patients (**Flather et al., 2000**).

New modalities in management of are promising as Angiotensin receptor-neprilysin inhibition with LCZ696, new aldosterone receptor blockers, serelaxin, ularitide, etc (**McMurray, 2013**).

## **Aim of the Work**

The aim of the essay is to throw a light on new modalities used for diagnosis and management of heart failure.