AMERICAN UNIVERSITY OF BEIRUT

OPTIMIZING THE MANAGEMENT OF PATIENTS ADMITTED WITH ACUTE HEART FAILURE

by

SAMER KAMAL EL CHAMI

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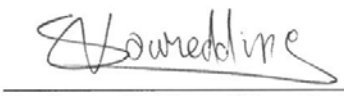
Approved by:



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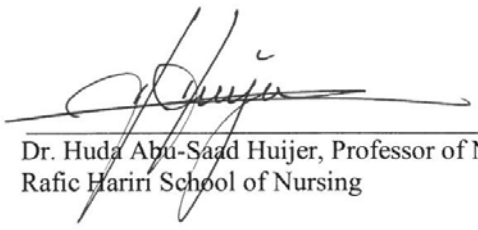

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AN ABSTRACT OF THE PROJECT OF

Samer Kamal El Chami for Master of Science
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Title: Optimizing the Management of Patients Admitted with Acute Heart Failure

Heart failure has been recognized as the most prevailing burden on the health care sectors worldwide. Recently, several countries, especially the high-income countries, have tried to establish health care programs in order to capture a comprehensive management of heart failure. This is being done through the emphasis on considering that heart failure management needs a specialized holistic care through the foundation of multi-disciplinary disease management program including a heart failure physician specialist and a heart failure nurse specialist. The European Society of Cardiology (ESC) through the Heart Failure Association (HFA) in addition to the American Heart Association and the Heart Failure Society of America (HFSA) established evidence-based guidelines for proper management of the heart failure. Based on these guidelines, the quality of practice can be measured, standardized and developed.

CONTENTS

LIST OF ABBREVIATIONS

ACC:	American College of Cardiology
ACEI:	Angiotensin Converting Enzyme Inhibitor
ACS:	Acute Coronary Syndrome
ADHERE:	Acute Decompensated Heart Failure National Registry
AHA:	American Heart Association
AHF:	Acute Heat Failure
AF:	Atrial Fibrillation
AP:	Arterial Pressure
APN:	Advanced Practice Nurse
ARB:	Angiotensin Receptor Blocker
ARDS:	Acute Respiratory Distress Syndrome
AV:	Atrioventricular
BEAUTIFUL:	MorBidity-mortality EvAlUaTion of the I_f inhibitor ivabradine coronary disease and left ventricULar dysfunction in patients with coronary disease and left ventricULar dysfunction
BiPAP:	Bi-level Positive Airway Pressure
BNP:	B-Type Natriuretic Peptide
BP:	Blood Pressure
BUN:	Blood Urea Nitrogen
CABG:	Coronary Artery Bypass Graft
CAD:	Coronary Artery Disease
CBC:	Complete Blood Count
CBR:	Complete Bed Rest
CCU:	Coronary Care Unit
CHD:	Coronary Heart Disease
CI:	Cardiac Index
CIBIS:	Cardiac Insufficiency Bisoprolol Study
CK-MB:	Creatine Phosphokinase MB Isoenzyme
CO:	Cardiac Output
CONSENSUS:	Cooperative North Scandinavian Enalapril Survival Study
COPD:	Chronic Obstructive Pulmonary Disease
COPERNICUS:	Carvedilol Prospective Randomized Cumulative Survival
COR:	Class of Recommendation
CPAP:	Continuous Positive Airway Pressure
CPK:	Creatine Phosphokinase
CRT:	Cardiac Resynchronization Therapy
CVD:	Cardiovascular Disease

CVP:	Central Venous Pressure
CXR:	Chest X-Ray
DIG:	Digitalis Investigation Group
DOSE:	Diuretic Optimization Strategies Evaluation
ECG:	Electrocardiogram
ECMO:	Extracorporeal Membrane Oxygenator
ED:	Emergency Department
EF:	Ejection Fraction
EPHESUS:	Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study
ESC:	The European Society of Cardiology
ETT:	Endotracheal Tube
HF:	Heart Failure
HFA:	Heart Failure Association
HF-PEF:	Heart Failure with Preserved Ejection Fraction
HF-REF:	Heart Failure with Reduced Ejection Fraction
HICU:	Heart Intensive Care Unit
HR:	Heart Rate
IABP:	Intra-Aortic Balloon Pump
INR:	International Normalized Ratio
IV:	Intravenous
ICD:	Implantable Cardioverter Defibrillator
$I_{f;}$	Funny Channels
JVP:	Jugular Venous Pressure
LAD:	Left Anterior Descending
LBBB:	Left Bundle Branch Block
LMC:	Labib Medical Center
LMWH:	Low Molecular Weight Heparin
LOE:	Level of Evidence
LOS:	Length of Stay
LV:	Left Ventricular
LVEF:	Left Ventricular Ejection Fraction
MCS:	Mechanical Circulatory Support
MD	Medical Doctor
MERIT-HF:	Metoprolol CR/XL Randomized Intervention Trial in Congestive Heart Failure
MI:	Myocardial Infarction
MOH:	Ministry of Health
MRA:	Mineralocorticoid Receptor Antagonist

NIV:	Non-Invasive Ventilation
NPO:	Nothing Per Os
NSAID:	Non-Steroidal Anti-Inflammatory Drug
NSS:	Normal Saline Solution
NT- proBNP:	N-Terminal Pro B-Type Natriuretic Peptide
NYHA:	New York Heart Association
OD:	Once Daily
OOB:	Out of Bed
OPTIMIZE-HF:	Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure
PAP:	Pulmonary Artery Pressure
PCI:	Percutaneous Coronary Intervention
PCWP:	Pulmonary Capillary Wedge Pressure
PEEP:	Positive End Expiratory Pressure
PRIDE:	Pro-BNP Investigation of Dyspnea in the Emergency Department
PSV:	Pressure Support Ventilation
PT:	Prothrombin Time
PTT:	Partial Thromboplastin Time
RAAS:	Renin-Angiotensin-Aldosterone- System
RALES:	Randomized Aldactone Evaluation Study
RBR:	Relative Bed Rest
RCT:	Randomized Controlled Trial
RN:	Registered Nurse
RR:	Respiratory Rate
$SpO_{2:}$	Peripheral Capillary Oxygen Saturation
SVRI:	Systemic Vascular Resistance Index
TIBC:	Total Iron Binding Capacity
TV:	Tidal Volume
US:	United States
VAD:	Ventricular Assist Device
VTE:	Venous Thromboembolism
WHO:	World Health Organization

CHAPTER 1

INTRODUCTION

Heart failure (HF) is a global medical problem affecting 26 million people worldwide. Hospitalizations due to HF are markedly increasing each year, the fact that led to ranking HF presentations as the leading cause of admissions in the United States (US) and Europe (Ambrosy et al., 2014). Consequently, the burden of HF on the healthcare system is translating into high expenditures that are difficult to cover, making this epidemic a significant clinical and public health concern (Ambrosy et al., 2014). Moreover, among the different types of cardiovascular diseases (CVDs), HF is presented as a complex clinical syndrome that results mostly from coronary artery disease and hypertension, with a distinguished impact on mortality. In 2010, the American Heart Association (AHA) reported that the leading cause of death in the US was attributed to CVD, with an estimation of 1 in every 3 deaths; HF alone was mentioned in 1 of every 9 death certificates in the US (Mozaffarian et al., 2014).

Several risk factors contribute to the development of HF in varying frequencies; these include coronary heart disease (CHD), hypertension, arrhythmias, cardiomyopathies, valvular heart disease, and congenital heart disease. Among those, AHA reported that hypertension was evident in 75% of HF cases. Although the global evolution of CHD management had contributed to enhancing the survival rate of CVD patients, the prevalence of HF has increased, particularly in the aging population. Furthermore, AHA is expecting that by 2030, HF prevalence will be increased by 46%, consequently increasing the cost of care by 127% (Go et al., 2014).

1.1. Background

HF is recognized as a clinical syndrome and not as solitary disease (Roger, 2013; Yancy et al., 2013). There is no single test to diagnose HF, as this syndrome could be manifested by diverse clinical features (Lindenfeld et al., 2010). To identify the syndrome of HF, multiple diagnostic criteria have been proposed based on various epidemiological studies such as the Framingham criteria, the Boston criteria, and Gothenburg criteria. These criteria depend on the presenting symptoms and imaging results, along with proper physical examination and health history data (Roger, 2013). The term "Heart Failure" does not literally mean failure of the heart, but rather reflects a syndrome where the heart is not capable to pump the blood fast enough to meet the body's needs (Casey, 2013). According to the American College of Cardiology (ACC) and AHA, HF is defined as "A complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood" (Yancy et al., 2013, p.153). Usually HF is a progressive disease, and the resultant impairment in systolic (pumping), diastolic (filling) function, or both will end with abnormal neurohormonal and circulatory symptoms such as fluid retention, shortness of breath, and fatigue especially on exertion (Lindenfeld et al., 2010). Clinically HF progresses through stages A through D, denoting progression from being at risk for HF (stage A) to having severe HF (stage D) (Go et al., 2014).

The lifetime risk for developing HF has been documented in various age, gender and ethnic groups (Muzaffarian et al., 2014). The European Society of Cardiology (ESC) has documented that 1-2% of the adult population in the developed countries has HF, with an increased prevalence by ≥ 10% among persons 70 years of age or older (McMurray et al., 2012). Several studies in the US have shown that the

lifetime risk for developing HF is 20% for both men and women regardless of age (Muzaffarian et al., 2014). Other investigators have reported that people at age 45 years have lifetime risks for HF when they reach 75 to 95 years of age that are estimated at 30% to 42% in white men, 20% to 29% in black men, 32% to 39% in white women, and 24% to 46% in black women. In the Rotterdam Heart Study, at age 55 years, the lifetime risk for HF is 33% for men and 29% for women (Roger, 2013). Furthermore, in those aged 65 to 74 years the reported incidence of new HF events among white men and women was 15.2 and 8.2 per 1000 population, respectively; in those aged 75 to 84 years, the incidence is 31.7 and 19.8, and in those ≥ 85 years the incidence is 65.2 and 45.6 (Go et al., 2014; Muzaffarian et al., 2014).

Hospitalizations after the diagnosis with HF are common. In one large population study in the US, 83% of HF patients were hospitalized at least once a year and in another 43% were hospitalized at least four times (Yancy et al., 2013), with a median length of stay (LOS) ranging from 4 to 20 days and in-hospital mortality range of 4% to 30%. In the OPTIMIZE- HF (Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients With Heart Failure) cohort study, 30% of the HF patients were re-admitted within 60 to 90 days post-discharge. Similarly, the ESC-HF (European Society of Cardiology–Heart Failure) pilot survey reported data from representative centers of 12 European countries showing that 31.9% of hospitalized HF patients were re-admitted in one year (Ambrosy et al., 2014).

Although HF is a progressive and a complex syndrome that could arise from multiple underlying problems or comorbidities (Lindenfeld et al., 2010; Ponikowski et al., 2014), the current implementation of HF evidence-based guidelines has contributed to improved outcomes and enhanced survival rates among HF patients (McDonagh et

al., 2011; Ponikowski et al., 2014). A recent systematic review of 29 trials showed that specialized multi-professional care in different settings reduced mortality in HF patients by 25%, HF hospitalizations by 26%, and all-cause hospitalizations by 19% (McDonagh et al., 2011). Furthermore, evidence-based clinical guidelines facilitated the establishment of HF care pathways, clinical decision tools and quality indicators to provide better performance criteria and outcomes measures (Ponikowski et al., 2014; Yancy et al., 2013).

Although the discovery of numerous evidence-based drug and device therapies has contributed to enhancing the survival among patients with HF, the mortality rates remain high, at approximately 50% within 5 years of diagnosis (Mozaffarian et al., 2014). In the ARIC (The Atherosclerosis Risk in Communities) study, the mortality rates were 10.4%, 22%, and 42.3% respectively at 30-day, 1-year, and 5-years after hospitalization for HF. The 5-year survival rates reported in another cohort study for HF patients in stages A, B, C, and D were 97%, 96%, 75%, and 20%, respectively (Yancy et al., 2013).

1.2. Significance

In Lebanon, CVDs dominate the highest rates of mortality and morbidity among the Lebanese population (Noureddine, Froelicher, Sibai & Dakik, 2010) accounting for 47% of total deaths at all ages (World Health Organization [WHO], 2014). This percentage reflects a significant increase from 2011, when deaths in Lebanon caused by CHD reached a level of 5,857 deaths, representing 27.44% of total deaths (WHO, 2011).

In a survey of 401 persons conducted by Noureddine et al. (2013), a positive medical history of heart disease was noticed in 52.2% of Lebanese people who were not previously diagnosed with heart disease (Noureddine, Massouh, & Froelicher, 2013). In the absence of reliable prevalence data, this finding suggests a high prevalence of heart disease in Lebanon. Besides, the Lebanese ministry of public health (MOH) has mentioned in its 2011 statistical bulletin that heart disease was the leading cause of hospital admissions, namely 30,940 admissions (including ischemic heart disease, hypertension and other forms of heart disease) out of total 37,277 admissions due to circulatory disorders. At that time, those between 60 and 65 years of age accounted for the highest proportion of admissions among patients with heart disease (Harb, 2011).

In Lebanon, an estimate of 10% represents the older adults segment of the population with an age of 65 years and above. With the fast growing of this segment, it is expected to reach 12% by 2030 and 18% by 2050 (Hajjar, 2013). Moreover, Sibai et al. (2009) expected an increase in the burden of the CVD among Lebanese people, especially with the accelerated adoption of unhealthy lifestyles. The recent statistical data showed the prevalence of CVD risk factors among Lebanese population such as hypertension to be 28.8% (WHO, 2014), diabetes 27.4 % (WHO, 2014), obesity 27.4% (WHO, 2011), hypercholesterolemia with more than 1 in 500 (Fahed et al., 2011), and smoking 32% (WHO, 2014). With HF mostly a disease of older adults, it is reasonable to expect an increasing burden of HF in Lebanon, thus necessitating evidence-based care to this population in order to prevent and control this syndrome.

HF data in Lebanon is absolutely scarce. Only one recent Lebanese study conducted in 2010 by Deek, Skouri and Noureddine, has shown the readmission rates and related factors among HF patients. The surveyed sample included 187 HF patients

admitted to a well-known academic teaching hospital in Beirut with a mean age of 63.71 years. In terms of risk factors in the sample, the distribution was hypertension (61%), CHD (50.8%), atrial fibrillation (17.1%), diabetes (41.7%), and hyperlipidemia (15%). The hospital LOS ranged between 1 and 38 days. The overall readmission rates were 15%, 22.2%, and 27.8% at 30, 60 and 90 days following discharge. Out of 187 HF patients, 72 were readmitted within 90 days of their index discharge for all causes. Exacerbating HF was the major cause of readmissions, including multiple readmissions for some. Twenty-eight patients (15%) were readmitted within 30 days of the index discharge, 42 (22.5%) within 60 days, and 52 (27.8%) within 90 days (Deek et al., 2014). The authors of this study noted that the findings suggest improper adherence of physicians to the evidence-based guidelines when managing those HF patients, including improper prescriptions of discharge medications. Although for the past five years in Lebanon, multiple conferences and awareness campaigns were held to raise the awareness for proper management of HF, adherence to the full guidelines is still suboptimal in terms of practice for both physicians and nurses who are in need to update their knowledge regarding the in-hospital management of HF as well as providing discharge instructions (Deek, Skouri & Noureddine, 2014). Unfortunately, findings from US surveys on hospital discharges have shown similar problems. For example, in one study, more than a quarter of HF patients did not receive a prescription upon discharge. Also in Europe, the recommended medications were not prescribed at the recommended doses (Ponikowski et al., 2014).

The Labib Medical Center (LMC) in Saida is a tertiary center that provides acute coronary care services. Basically, the patients with critical cardiac disease are managed in the Heart Intensive Care Unit (HICU) that has a capacity of six beds for

both medical and surgical patients. The stabilized patients can be then transferred to the step down unit for further monitoring. The majority of the patients come with CHD (40%), HF (25%), acute pulmonary edema (15%), valvular heart disease (10%), cardiogenic shock (5%), hypertensive crisis (3%), and severe arrhythmias (2%). Interestingly, there is anecdotal evidence of high readmission rates among HF patients. The current practice of HF management at LMC is not yet standardized. Based on personal experience as nurse manager at LMC, I have noticed that the majority of the hospitalized HF patients are managed differently based on the attending's preferences and not on evidence-based guidelines. Therefore, the aim of this project is to propose a standardized plan for the management of inpatients with acute heart failure at LMC. Such a plan will include the management of HF patients admitted with exacerbating and acute HF. The project shall include the development of a clinical pathway for inpatients, and pre-printed orders for HF management starting from the emergency department going through the coronary care unit stay and discharge. Implementation and evaluation plans will also be presented. In summary, the purpose of the project is to develop a clinical pathway for acute HF patients that is evidence-based and at the same time fits the context of Labib Medical Center.

CHAPTER 2

LITERATURE REVIEW

2.1. Overview of Heart Failure

Heart failure, as mentioned in chapter I, is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood (Yancy et al., 2013). Accordingly, the ability of the heart to support the physiological needs of the body is compromised and the development of HF processes either in acute or chronic phases would be associated with a variety of clinical manifestations (Givertz et al., 2013). Although disorders of the pericardium, myocardium, endocardium, and heart valves may contribute to HF development, the majority of symptoms in patients with HF are mainly due to the impaired left ventricular (LV) function (McMurray et al., 2012; Yancy et al., 2013).

HF syndrome incorporates two main processes: Acute and chronic. The acute process of HF is characterized by the abrupt onset of deteriorating cardiac functions in either previously healthy persons due to a clear precipitant (such as acute coronary syndrome [ACS]), or even as a gradual decompensation in persons with pre-existing HF. On the other hand, chronic HF describes the established process of impaired cardiac function in persons with ongoing and variable physiological consequences who are managed on outpatient basis (Casey, 2013; McMurray et al., 2012; Yancy et al., 2013). The focus of this review will be mainly on the acute heart failure (AHF) process and its proposed management guidelines.

Most clinical trials use LV ejection fraction (EF), measured by Doppler

echocardiography, to classify HF into two distinct types: systolic and diastolic. Based on the EF cut-off points, new terms have been used: HF with reduced LV function (HF-REF) for systolic dysfunction and HF with preserved LV function (HF-PEF) for diastolic dysfunction (McMurray et al., 2012; Yancy et al., 2013). The AHA, ACC and ESC have proposed certain criteria for the definition of HF syndrome. HF-PEF criteria include: clinical signs and symptoms of typical HF, LVEF mildly reduced or normal and LV not dilated, and LV diastolic dysfunction evident by Doppler echocardiography or cardiac catheterization (McMurray et al., 2012; Yancy et al., 2013). On other hand, HF-REF criteria include: typical signs and symptoms of HF and reduced LVEF (McMurray et al., 2012). Different EF cut-off levels have been mentioned in the literature. The American Society of Echocardiography recommended the EF threshold of 55%, while the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry and the Acute Decompensated Heart Failure National Registry (ADHERE) database used 40% as the cut point (Roger, 2013). In addition, the 2013 AHA and ACC guidelines adopted the cut-off level of 40% to further classify HF-REF as a clinical diagnosis with EF $\leq$ 40% and HF-PEF with EF >40% (Yancy et al., 2013). Also, the 2012 ESC guidelines considered EF $\leq$ 35% for HF-REF and noted that level for effective therapy among those patients (McMurray et al., 2012).

HF can be challenging to diagnose especially in the early stages as the exhibited clinical features may arise from different factors, which are sometimes non-organ specific or advanced chronic illnesses such as anemia and chronic obstructive lung disease (Cowie et al., 2014; Roger, 2013). Although HF-REF and HF-PEF share common etiologies such as CAD, hypertension, diabetes mellitus, metabolic syndrome,

and atrial fibrillation (AF), HF-PEF diagnosis is largely one of exclusion among other differential diagnoses. On the other hand, HF-REF can be clearly associated with antecedent myocardial infarction and hypertension as major causes, in addition to viral infection, alcohol abuse, and chemotherapy (McMurray et al., 2012; Yancy et al., 2013). Moreover, HF-PEF and HF-REF may vary according to the epidemiological profile of patients whereby older, obese, and more often female patients are more likely to have HF-PEF (McMurray et al., 2012; Yancy et al., 2013).

2.1.1 . *Pathophysiological Changes*

The underlying mechanisms that lead to heart failure affect the adaptation capacity of the heart. Consequently, structural and functional changes will result in numerous physiological manifestations. The key compensatory mechanisms include the pathological remodeling of the LV in order to increase the work force of the heart, the activation of the neurohormonal renin-angiotensin-aldosterone system (RAAS) to increase the blood volume through the enhanced sodium and water retention, and the activation of sympathetic system to increase the pre-load and filling pressures of the heart through the constriction of veins. As HF progresses, the prolonged activation of the compensatory mechanisms will have detrimental effects on the heart, such as increased workload due to increased heart rate and afterload as side effects of the activation of the RAAS and sympathetic nervous systems. Then compensation fails, with a significant effect on multiple organs of the body (lungs, blood vessels, kidneys, liver, bone marrow, and muscles) and eventually will end up in a vicious cycle leading to advanced stages of HF (Casey, 2013; McMurray et al., 2012; Yancy et al., 2013).

Eventually myocardial maladaptive changes will result in impaired circulation

with back flow of blood from the heart to the venous system, which will enhance the build-up of fluid in the lungs (pulmonary edema) and tissues (peripheral edema). The symptoms may vary among patients and may present as breathlessness, dyspnea, paroxysmal nocturnal dyspnea, swollen legs and ankles, and increased body weight. Also changes to the skeletal muscles (muscle acidosis) will contribute to limited physical activity and fatigue. Moreover, the typical signs that could be found in HF patients are: Elevated jugular venous pressure, hepatojugular reflux, third heart sound (gallop rhythm), laterally displaced apical impulse, and cardiac murmur (Cowie et al., 2014; McMurray et al., 2012; Yancy et al., 2013).

2.1.2. *Clinical Evaluation*

Further evaluation of HF patients should be done once the diagnosis is confirmed. Certain measures should be taken to identify the underlying etiology and its correctable causes, assess the nature and severity of symptoms, and then establish an adequate management plan based on the patient's prognosis (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013).

Careful history and physical examination are an essential part to start with the assessment of patients with HF. Identification of cardiac and non-cardiac causes (i.e.: comorbidities) of HF is better done by history taking at this stage; this is complemented with vital signs, electrocardiogram (ECG), and a complete physical assessment. Echocardiography is a corner stone diagnostic measure that helps to evaluate cardiac structure and function and guide the appropriate treatment; it provides the measure of ejection fraction (EF). In addition, chest radiography (X-ray) and complete blood work (biochemical, hematological, and BNP/NT-proBNP testing) are often ordered, with

BNP/NT-proBNP used for diagnosis and to monitor the effectiveness of treatment.

Assessment of the severity of HF involves evaluation of the functional capacity and a staging system to guide treatment. Functional capacity of patients with HF can be assessed using the New York Heart Association (NYHA) classification tool (I through IV). It is used on a daily basis to assess the physical activity limitations caused by cardiac symptoms and as a prognostic measure to predict the impact of HF (Yancy et al., 2013).

The description of the classes is shown in Table 1.

Table 1. NYHA Classification System

Class	Characteristics
I	Ability to do regular physical activities with no symptoms
II	Slightly limited physical activity that may cause breathlessness and
III	Markedly limited physical activity and minimal symptoms even at rest
IV	Symptomatic even at rest, and unable to do any physical activity (even

For assessment of HF severity progression, the ACC and AHA have established the HF staging system (A through D). Stage A is designated for patients who are at risk for HF and have no structural heart disease and are asymptomatic. Stage B is designated for patients who are at risk for HF and have structural heart disease but are still asymptomatic of HF. Stage C patients have HF symptoms and evidence of structural heart disease. Stage D patients have HF that is refractory to usual medical management and thus are in need for specialized management such as mechanical assistive device therapy and cardiac transplantation (Yancy et al., 2013).

2.1.3. *Acute Heart Failure*

Acute heart failure (AHF) is a complex syndrome characterized by the rapid onset of new HF (de novo) or acute worsening of chronic HF (decompensated HF). AHF is a life threatening condition that requires urgent medical attention and repeated hospital admissions (Cowie et al., 2014; Givertz et al., 2013; McMurray et al., 2012). The re-admission rates for AHF patients are estimated at 25% within one month (Cowie et al., 2014) and 50% within six months (Yancy et al., 2013). Moreover, AHF hospitalization is associated with significant short and long-term mortality and morbidity. Reported in-hospital death rates ranged between 4% and 10%, whereas the mortality rate reached 20% to 40% at one year for those AHF patients who survived their hospitalization. Moreover, the 5-year mortality rate was reported at 70% (Cowie et al., 2014).

In most cases, AHF may arise as a worsening condition of chronic HF (McMurray et al., 2012; Yancy et al., 2013), with a relatively equal distribution among patients with HF-PEF and HF-REF (Yancy et al., 2013). The data of the OPTIMIZE-HF registry have shown that 88% of AHF patients had history of chronic heart failure rather than a de novo (i.e. new onset of acute heart failure in a patient without previously known cardiac dysfunction) presentation (Givertz et al., 2013), while data of the Italian registry have shown that among 1,855 patients admitted with AHF, 43% of patients had de novo HF and the other 57% had worsening chronic HF (Oliva et al., 2012).

AHF patients may present with rapid or gradual episodes of acute symptoms. Symptoms may take a period of time (days to weeks) in patients with pre-existing HF, who may present with volume overload manifestations such as increased breathlessness and edema (NYHA III to IV). In these patients concurrent infections, COPD exacerbation, anemia, kidney dysfunction, non-adherence to diet/drug therapy,

uncontrolled hypertension, endocrine abnormalities, alcohol and drug abuse are common precipitant factors. On the other hand, new onset of acute presentations may occur over a very short period of time (minutes to hours) in association with precipitants like ACS and its possible mechanical complications, rapid arrhythmias or conduction disturbances, acute pulmonary embolism, hypertensive crisis, cardiac tamponade, aortic dissection, perioperative problems, and peripartum cardiomyopathy (McMurray et al., 2012; Yancy et al., 2013). AHF patients may also vary according to the cardiac and non-cardiac comorbidities. In the ADHERE study, data has shown the prevalence of most common comorbidities to include high blood pressure (72%), coronary artery disease (58%), diabetes (44%) and atrial fibrillation (30%) (Abraham et al., 2005).

2.2. Management Guidelines for Acute Heart Failure

The management guidelines of HF have been issued since many years by different international organizations. Guidelines were developed by experts in the field, based on different research designs and thorough review of evidence. With frequent updates, use of the evidence-based guidelines aims to provide an organized and structured approach to clinical management, improve the quality of care and optimize patient outcomes. Moreover, the clinical guidelines are used to monitor the performance measures at the level of individual patient care and system as well. The international organizations share many similarities in the approach of AHF management. However, at certain points of care, the organizations have slightly different level of evidence classification systems.

The most comprehensive and latest guidelines on the management of AHF were developed by the ESC in 2012, Heart Failure Society of America (HFSA) in 2010,

and ACC in collaboration with AHA in 2013. In addition, the National Heart Foundation of Australia updated their guidelines for the management of HF in 2011. The task forces of these cardiovascular societies have developed recommendations for AHF management based on the best evidence available. The term "Class of Recommendation (COR)" is used as an estimate measuring the benefit/risk ratio of the considered management strategy. Recommendations are classified from class I, where the evidence is very strong for benefit to class III, where the evidence is strong for harm or lack of benefit of the treatment (Yancy et al., 2013). The guidelines are also evaluated by the "Level of Evidence (LOE)" used as the basis for the recommendation (types of studies conducted to obtain the evidence). The levels of evidence range from level A where evidence is based on systematic review of randomized clinical trials (RCTs) to level C where evidence is based on opinions of experts. Both terms, COR and LOE, are used together (COR, LOE) to assign the certainty of the proposed management (Yancy et al., 2013).

2.2.1. *Initial Management of AHF*

Effective management of AHF patients requires prompt identification and diagnosis along with rapid treatment and evaluation (Cowie et al., 2014). Weintraub et al. (2010) noted that the vast majority of AHF patients are treated initially at emergency departments (ED) with nearly 80% of them admitted to the hospital for further management. The key aims should focus primarily to relieve symptoms, optimize volume status and stabilize blood pressure, maintain blood oxygen levels, and prevent organ damage. In addition, patients who might benefit from revascularization therapy, mechanical circulatory support, and those at risk of thromboembolism and in need for

anticoagulant therapy must be identified and managed accordingly (Cowie et al., 2014; Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013).

2.2.1.1. Assessment

Early diagnosis of AHF is hard since patients may have symptoms that are non-specific and difficult to differentiate from other conditions. Hence, careful history in combination with proper physical assessment is needed (Cowie et al., 2014; Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013). The patient's presenting characteristics would guide the targeted therapies and prognostic information. Assessment includes hemodynamic parameters, degree of congestion as well as the peripheral perfusion (wet or dry), the time course of symptoms, comorbidities, presence of chest pain, pulmonary status (rales), presence of dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, presence of cardiogenic shock, presence of precipitant factors such as ACS and arrhythmias, and previous hospitalizations. Patients with pre-existing HF should be carefully reviewed for their maintenance therapy. Adjustments of medications may be done during their hospitalization in light of worsening HF, such as the reduction of beta-blockers doses in patients with marked volume overload or low cardiac output, and the reduction or even withholding of angiotensin converting enzyme inhibitors (ACEIs) or angiotensin receptor blocker (ARB) doses in AHF patients with worsened renal function (Cowie et al., 2014; Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013). However, in some patients, the continuation of ACE inhibitors or ARBs and beta-blockers may be well tolerated and result in better outcomes *(Class I, B).*

In addition to history and physical assessment, a number of diagnostic aids are

recommended. Chest radiology (X-Ray) is used to assess the presence of interstitial or alveolar edema *(Class I, C)* but its sensitivity varies, as it may not detect early congestion in some patients with breathlessness. A 12-lead ECG is used to assess the characteristics of heart rhythm and conduction and detect underlying abnormalities (i.e.: QRS morphology, ST segments, LBBB, atrial and ventricular arrhythmias). It is more helpful to detect the ACS in AHF patients *(Class I, C).* Transthoracic echocardiogram is recommended to assess cardiac structure and function. Measurement of EF through echocardiography as mentioned before is required to guide the treatment and obtain prognostic information *(Class I, C).*

Laboratory evaluations represent an important tool to guide the management of AHF, such as determining the patients' suitability for certain medications (i.e.: diuretics, ACEIs, and cordarone), monitoring the adverse effects of current medications (i.e.: hyponatremia or hypokalemia), and detecting treatable causes and comorbidities (i.e.: thyroid dysfunction and anemia). Initial evaluation includes urinalysis, hematological blood tests (complete blood count), and biochemical blood tests (i.e.: blood sugar, electrolytes, BUN, serum creatinine, GFR, Calcium, Magnesium, Iron level, troponin I level, fasting lipid profile, liver and thyroid function tests) *(Class I, C).*

Moreover, measurements of natriuretic peptides whether B-type (BNP) or N-terminal pro B-type (NT- proBNP) are useful to support the clinical judgment and certainty for the diagnosis of AHF, especially in patients who present with non-specific symptoms *(Class I, A).* According to the ESC, multiple studies have examined the threshold concentration that confirms the diagnosis of AHF, with cut-off points of NT-proBNP at $\geq$ 300 pg/mL and of BNP at $\geq$ 100 pg/mL (McMurray et al., 2012), while the data from the N-terminal Pro-BNP Investigation of Dyspnea in the Emergency

Department (PRIDE) study has shown that NT-proBNP levels increased with age. Therefore, they recommended new cut-off points levels for NT-proBNP according to the age of patients as follows: > 450 pg/mL for patients younger than 50 years of age; > 900 pg/mL for patients age 50 years or older; and ≥1800 pg/mL for patients 75 years or older (Lindenfeld et al., 2010).

Invasive hemodynamic monitoring should be performed on AHF patients who have uncertain and persistent hemodynamic instability. Usually, a pulmonary artery catheter is used to measure the intra-cardiac pressures to guide therapy in AHF patients with respiratory distress and impaired perfusion *(Class I, C)*. Moreover, the insertion of an arterial line may be helpful for continuous monitoring of blood pressure in patients with persistent hypotension despite treatment (Lindenfeld et al., 2010; McMurray et al., 2012).

2.2.1.2. Pharmacological Management in The Acute Phase

This section describes the recommended oxygen therapy and various medications used in managing AHF. Oxygen therapy is used in patients with respiratory distress and evident hypoxemia (pulse oximeter oxygen saturation [SpO2]<90% or PaO2 <60mmHg) *(Class I, C).* Usually oxygen is started at 40%-60% titrating to keep SpO2 >90%. Non-invasive ventilation (NIV) using continuous positive airway pressure (CPAP) is considered for dyspneic cooperative patients and a respiratory rate >20 breaths/min to reduce breathlessness, hypercapnia and acidosis *(Class IIa, B),* whereas invasive ventilation (endotracheal tube) may be used for patients with severe respiratory distress associated with disturbed consciousness (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013).

AHF patients with evident fluid overload should be considered for loop diuretic therapy *(Class I, B).* Also, decompensated HF patients who were already on diuretic therapy at home should be considered for increased doses of diuretics *(Class I, B).* Although intravenous loop diuretics may rapidly relieve symptoms especially in patients with acute pulmonary edema, doses should be directed to achieve optimal volume status and relieve congestion, without significant adverse outcomes. Excessive use of diuretics may induce the reduction of intravascular volume and hence increase the risk of hypotension episodes and worsening renal dysfunction, especially in patients with previous kidney disease. Further evaluation of diuretic therapy can be done through the daily monitoring of patient's signs and symptoms, total fluid balance, body weight, and blood biochemistry tests (Potassium and Sodium).

The doses regimen of diuretic therapy, whether intermittent or continuous infusions, has been tested by the DOSE (Diuretic Optimization Strategies Evaluation) trial. The finding did not show any significant difference in symptoms relief and outcomes of patients when using alternative modes of infusion therapy (McMurray et al., 2012; Yancy et al., 2013).

When symptoms persist and diuresis is inadequate, it is reasonable to increase the diuretic doses accordingly or even add another type of diuretic therapy (thiazide) *(Class IIa, B).* Also low dose of dopamine (in small doses < 3mcg/kg/min) may be considered at this level *(Class IIb, B)* despite its uncertainty. Ultrafiltration, which is similar in principle to hemodialysis but using filtration at lower pressure and a slower rate for patients who are hemodynamically unstable, may be considered on patients who fail to get symptomatic relief with the diuretic therapy *(Class IIb, B)*. However, multiple studies have not shown significant weight reduction among patients who underwent

ultrafiltration as compared to standard diuretic therapy (Yancy et al., 2013).

Morphine may be used in AHF patients, particularly those with acute pulmonary edema. Morphine acts to relieve anxiety and distress associated with dyspnea. Moreover, as a venodilator, it is thought to produce mild venodilation, contributing to reduced preload and diminished sympathetic response. However, the use of morphine in AHF is not well supported by prospective data concerning in-hospital outcomes and so the use of morphine is suggested with caution, especially in those with severe respiratory distress and altered mental status (Lindenfeld et al., 2010; McMurray et al., 2012).

Vasodilators can act to reduce the preload and afterload of the heart and hence increase the stroke volume. They are used as an adjunctive therapy with diuretics to relieve symptoms of congestion. Intravenous nitroglycerine or nitroprusside is considered in patients in the absence of hypotension *(Class I, C).* AHF patients with hypertension, coronary ischemia, and significant mitral regurgitations are considered for intravenous nitroglycerin. Further evaluation of blood pressure (hypotension), heart rate (tachyphylaxis), and volume status should be done routinely. Cautious use of nitroglycerin is also considered in patients with significant mitral or aortic stenosis (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013).

Inotropic use is reserved for patients with cardiogenic shock (severe reduction in cardiac output and compromised vital organ perfusion). Inotropes such as Dobutamine (2.5-20 mcg/kg/min) are used to maintain systemic perfusion (increased cardiac output) and preserve end-organ perfusion *(Class I, C).* Evaluation should be done through hemodynamic monitoring as well as the observation of potential side effects (arrhythmias and myocardial ischemia). Also in patients with refractory stage D

HF, inotropic use is considered as a bridge therapy for further definitive therapy (coronary revascularization, mechanical circulatory support [MCS] therapy, device therapy, and heart transplantation) *(Class IIa, B).* In addition, when Dopamine is used at doses of 3 to 5 mcg/kg/min, it acts on the beta-adrenergic receptors and boosts the inotropic activity of the heart. Also, at higher doses of more than 5 mcg/kg/min, dopamine can act on both alpha and beta adrenergic receptors to add a peripheral vasoconstrictor activity and increase blood pressure (Lindenfeld et al., 2010; McMurray et al., 2012).

The use of vasopressors such as norepinephrine (0.2–1.0 mcg/kg/min) and epinephrine (0.05–0.5 mcg/kg/min) should be considered in patients with persistent hypotension and hypoperfusion *(Class IIb, C).* Such medications have a prominent peripheral arterial vasoconstriction activity and hence increased blood pressure. Cautious use of these medications is considered in hypovolemic patients as they may induce organ damage (McMurray et al., 2012; Yancy et al., 2013).

Patients with AHF are at high risk of venous thromboembolism due to their decreased cardiac output, increased systemic venous pressure, and pathological changes that promote blood clotting. Venous thromboembolism prophylaxis with heparin or another anti-coagulant should be considered upon admission *(Class I, B).* For patients admitted with adequate renal function (serum creatinine <2.0 mg/ dL), evidence from randomized trials suggests that enoxaparin 40 mg should be administered subcutaneously once daily or unfractionated heparin 5,000 units subcutaneously every 8 hours (Yancy et al., 2013).

2.2.1.3. Non-Pharmacological Initial Interventions

The use of an intra-aortic balloon pump (IABP) is considered to support the circulation mainly in patients with cardiogenic shock. However, IABP may be used also before surgical correction of acute mechanical complications (i.e.: inter-ventricular septal rupture and acute mitral regurgitation), and also before procedural correction in selected patients with acute myocardial ischemia or infarction, such as before, during, and after percutaneous or surgical revascularization. In addition, short-term use of IABP may be helpful to bridge patients until implantation of ventricular assist device (VAD) or heart transplantation. Mizuno et al. (2014) have found in their study (The Acute Decompensated Heart Failure Syndromes [ATTEND]) that the in-hospital mortality rate among cardiogenic shock patients (without ACS) treated with the IABP was estimated at 23% compared to the 60%-70% of mortality rate among patients who were not treated with IABP.

Furthermore, the use of the extracorporeal membrane oxygenator (ECMO) can be used in specific patients with acute and rapidly deteriorating HF (bridge to definitive therapy decision). However, the proper benefit from initiating ECMO therapy is uncertain, particularly in patients where full evaluation has not been possible and therefore, death may occur with reasonable MCS (McMurray et al., 2012). These management guidelines apply to all adult age groups; considerations for older adults depend on risk stratification and co-morbidities. Moreover, the management approach does not differ substantially between HF-PEF and HF-REF patients, since the determining factor for acute HF management is the clinical presentation (fluid volume status and perfusion), and accompanying co-morbidities, as shown in the sections below.

2.2.2. *Special Considerations in AHF*

2.2.2.1. Acute Coronary Syndrome

ACS is a major life threatening precipitant for AHF, which should be identified early by ECG and serum biomarkers (such as Troponin), and treated optimally according to the ACS guidelines *(Class I, C).* Coronary angiography and revascularization should be undertaken as an urgent procedure in patients with hemodynamic instability and as an emergency procedure in those in cardiogenic shock (McMurray et al., 2012). In the presence of an ST elevation ACS or a new LBBB, immediate primary percutaneous coronary intervention (PCI) is recommended in order to reduce the risk of premature death *(Class I, A).* Moreover, intravenous thrombolytic therapy is recommended if PCI cannot be performed *(Class I, A).* In the presence of a non- ST elevation ACS, early PCI is recommended in order to reduce the risk of recurrent ACS *(Class I, A).*

The main indication for surgical revascularization in HF patients is significant CAD; however other considerations include the myocardial viability status, amenable coronary anatomy, and concomitant medical therapy (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013). Coronary artery bypass graft (CABG) surgery is recommended for suitable HF patients who are expected to survive more than one year and are on optimal medical therapy with angina and amenable coronary anatomy, especially significant left main stenosis (>50%) or left main equivalent *(Class I, C),* and those with angina and two- or three-vessel CAD, including a left anterior descending (LAD) stenosis, who are otherwise suitable for surgery and expected to survive more than one year with good functional status, and to reduce the risk of

consequent cardiovascular complications *(Class I, B).* CABG is reasonable in patients with mild to moderate LV systolic dysfunction and significant multi-vessel CAD or proximal LAD stenosis when viable myocardium is present in order to improve survival *(Class IIa, B),* and those with severe LV dysfunction (EF <35%) and significant CAD to improve morbidity and mortality for patients, HF, and significant CAD *(Class IIa, B).* CABG may be considered in patients with ischemic heart disease, severe LV systolic dysfunction, and operable coronary anatomy whether or not viable myocardium is present *(Class IIb, B).* However, CABG is not recommended in patients without angina and without viable myocardium *(Class III, C).*

2.2.2.2. Valvular Diseases

AHF patients with valvular problems are usually managed with surgery. Surgical aortic valve replacement is reasonable in symptomatic patients with confirmed critical aortic stenosis contributing to the worsening LV dysfunction *(Class IIa, B).* In patients who cannot tolerate the surgical intervention, transcatheter aortic valve replacement is more reasonable *(Class IIa, B).* Aortic valve repair or replacement is recommended in symptomatic and asymptomatic patients with severe aortic regurgitation and EF <50% (McMurray et al., 2012).

The assessment of mitral regurgitation in the presence of LV dysfunction is a bit complex. Thus differentiating between primary and secondary mitral regurgitation is crucial for the decision whether to repair or to replace, while considering some other factors such as EF measurements, presence of current comorbidities (i.e.: ACS and AF), and the ultimate effect of optimal medical therapy. In primary mitral regurgitation due to flail leaflets, surgical repair may improve symptoms especially in patients with

severe LV dysfunction (EF<30%); however its effect on survival is uncertain, whereas secondary mitral regurgitation may arise as a mechanical complication of significant ACS leading to ruptured papillary muscles or reduced leaflet closing. Combined valve and coronary surgery (confirmed CAD by angiography) should be considered in symptomatic LV systolic dysfunction patients who are suitable for surgical intervention. In non-operable patients, percutaneous edge-to-edge repair may be considered to improve symptoms (McMurray et al., 2012). According to the ACC/AHA (2013), studies showed that transcatheter mitral valve repair or mitral valve surgery for functional mitral insufficiency is of uncertain benefit.

2.2.2.3. Managing Arrhythmias

Atrial fibrillation (AF) is a significant factor for the development of acute HF symptoms, particularly in patients with rapid ventricular response. Initial management should be pursued with either rate control or rhythm control strategies, although emergency cardioversion *(Class IIb, C)* is considered in patients with severe hemodynamic instability. The management approach should also take into consideration the correctable causes, possible precipitating factors, and thromboembolism prophylaxis. For rate control, initial management with a beta-blocker therapy is preferred over digoxin (especially in HF-REF), whereas the need for later combination treatment might be needed for more effective therapy. Nondihydropyridine calcium channel blockers, such as Diltiazem, are preferred for rate control in patients with HF-PEF. Similarly, digoxin is considered as an adjunct therapy for more effective therapy. For rhythm control, amiodarone is still the preferred antiarrhythmic medication, especially in patients with HF-REF. Moreover, if the rate control strategy fails in HF-

REF patients, AV node ablation and cardiac resynchronization therapy (CRT) device placement may be required later.

Electrical cardioversion to return sinus rhythm is recommended when major ventricular arrhythmia is contributing to the patient's hemodynamic instability. Also, in acute situations with severe brady-arrhythmias or heart blocks, insertion of transvenous pacing wires is considered to improve symptoms while another treatment options of device therapy such as implantable cardioverter defibrillator (ICD), CRT-Pacemaker (CRT-P) and CRT-Defibrillator (CRT-D) could be decided later in light of optimal medical management, hemodynamic status of patients, and prognostic factors *(Class I, C)*. Moreover, pharmacological conversion of ventricular arrhythmias is better managed with the use of amiodarone.

2.2.3. *Pharmacological Management After Stabilization*

Treatment with ACEIs or ARB should be started in patients with reduced EF unless there is any contraindication, such as severe hypotension, bilateral renal artery stenosis, and severe kidney dysfunction *(Class I, A)*. Different RCTs used ACEIs in patients with severe HF such as the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS), which showed an absolute risk reduction of mortality by 14.6% (McMurray et al., 2012). Usually treatment with an ACEI or ARB should be initiated at low doses (i.e.: captopril 6.25mg 3 times/day or candesartan 4 to 8 mg once daily), followed by gradual dose increments if lower doses have been well tolerated until the predetermined target dose is reached. Further evaluation of ACEI/ARB therapy can be done through frequent monitoring of renal function and serum potassium. For patients who do not tolerate the side effects of ACEI (cough, rash, and taste disturbances), a

switch to ARB therapy may be more helpful (McMurray et al., 2012; Yancy et al., 2013).

Beta-blocker therapy should be initiated in patients with reduced EF early after stabilization, i.e. when reaching optimum volume hemodynamic status *(Class I, A)*. Small doses should be started (i.e.: Bisoprolol 1.25 mg once daily) and then up titrated as far as possible before discharge. Three RCTs have studied the use of beta-blockers in patients with decompensated HF: the Cardiac Insufficiency Bisoprolol Study II (CIBIS II), the Carvedilol Prospective Randomized Cumulative Survival (COPERNICUS), and the Metoprolol CR/XL Randomized Intervention Trial in Congestive Heart Failure (MERIT-HF) trials. All have shown reduction of mortality rate by 34%. Moreover, more than 90% of those studied patients were on ACE inhibitor therapy (McMurray et al., 2012). It was also noted that high doses of ACE inhibitors should not be started before initiation of beta-blocker therapy. Moreover, earlier low dose of an ACE inhibitor added to a beta-blocker may produce a better prognosis and reduction in mortality. Continuous evaluation is recommended for fluid retention and worsening symptoms, severe bradycardia or heart block, and hypotension (Yancy et al., 2013).

Ivabradine is used only in patients with sinus rhythm. It may be added to the beta-blocker therapy in HF patients particularly those with heart rate $\geq$ 70 beats/min and EF $\leq$35% *(Class IIa, B)*, and it may also be considered in HF patients with either beta-blocker contraindication or intolerance *(Class IIb, C)*. The pharmacological action on the "funny channels [I_f]" inhibition of the sinus node will produce its effect to slow the sinus node firing rate. The key evidence of Ivabradine was shown by The Systolic Heart failure treatment with the I_f inhibitor ivabradine Trial (SHIFT). Among HF patients (NYHA class II–IV) with sinus rhythm (rate $\geq$70 beat/min) and EF $\leq$35%, HF

hospitalization was decreased by 26% and mortality-morbidity by 4.2%. The maximal dose of Ivabradine was 7.5mg twice daily in addition to the concomitant therapy of a diuretic (in 84% of patients), digoxin (22%), an ACE inhibitor (79%), an ARB (14%), a beta-blocker (90%), and a mineralocorticoid receptor antagonist (MRA) (60%). Ivabradine also improved LV function and quality of life. Additional evidence for Ivabradine came from another RCT entitled the MorBidity-mortality EvAlUaTion of the I_f inhibitor ivabradine in patients with coronary disease and left ventricULar dysfunction (BEAUTIFUL) trial. Among HF patients with CHD and an EF <40%, ivabradine was given at 7.5 mg twice daily and was well tolerated during HF hospitalization, although it did not reduce cardiovascular mortality significantly (McMurray et al., 2012).

HF patients who are already on ACEIs or ARBs and beta-blockers with reduced EF should start with mineralocorticoid receptor antagonist (MRA) therapy at the earlier stages unless contraindicated *(Class I, A)*. Reduction of mortality risk by 30% in patients with severe HF taking spironolactone 25– 50 mg was noted in the Randomized Aldactone Evaluation Study (RALES) trial. Another RCT, the Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study (EPHESUS) have shown the reduction of mortality risk by 15%. Most of EPHESUS study patients were on ACEI or ARB (87%) and beta-blocker (75%), in addition to the MRA. Special precautions should be taken in patients on MRA therapy. Side effects may arise such as hyperkalemia and worsening renal function. Hence, serial monitoring of serum electrolytes and renal function is mandatory. Serum creatinine should be $\leq$ 2.5 mg/dL in men, or $\leq$ 2.0 mg/dL in women (or estimated glomerular filtration rate >30 mL/min/1.73 m^2), and potassium should be less than 5.0 mEq/L.

Doses of Spironolactone should be initiated at 12.5 to 25 mg daily, while Eplerenone doses should be initiated at of 25 mg/d, increasing to 50 mg daily. For those patients with hyperkalemia or marginal renal function (estimated glomerular filtration rate 30 to 49 mL/min/ 1.73 m^2), an initial regimen of every-other-day dosing is advised. Moreover, other measures should be considered such as discontinuation of potassium supplements as well as the non-steroidal anti-inflammatory drugs (NSAID) after initiation of MRA (Yancy et al., 2013).

The addition of digoxin therapy may be considered in patients with reduced EF and persistent symptoms. Digoxin may be used in adjunct with the conventional therapy of neurohormonal antagonists to control the ventricular rate, especially in patients with atrial fibrillation (AF) when up-titration of beta-blocker doses is not possible. The findings from a single large RCT (Digitalis Investigation Group [DIG]) supported by a meta-analysis of other smaller trials have shown that the use of digoxin with diuretics and ACE inhibitors has successfully improved the symptoms of severe HF, although it had no effect on survival (Ambrosy et al., 2014). The use of digoxin can be associated with several side effects such as atrial and ventricular arrhythmias (atrioventricular blocks), particularly in the context of hypokalemia. Thus, careful monitoring of hemodynamics, serum electrolytes and renal function is advised. In addition, special precautions should be taken when digoxin is used with other drugs that could depress the sinus or atrioventricular nodal function (amiodarone or beta-blocker).

Digoxin therapy regimen is usually initiated and maintained at a dose of 0.125 to 0.25 mg daily. Plasma concentration of digoxin should be maintained in the range of 0.5 to 0.9 ng/mL, while obvious digoxin toxicity is associated with serum digoxin levels >2 ng/mL. Precautions are highly considered in patients with impaired renal function.

The concomitant use of digoxin with some other medications (such as clarithromycin, dronedarone, erythromycin, amiodarone, cyclosporine, verapamil) may induce increased serum digoxin concentration. Thus digoxin doses should be reduced accordingly if these drugs are initiated (Yancy et al., 2013).

2.2.4. *Non-Pharmacological Management After Stabilization*

Maintenance of adequate hemodynamic status is required after the stabilization phase till discharge. This could be done through proper and continuous monitoring of hemodynamic parameters such as heart rate, heart sounds and rhythm, blood pressure, cardiac output, breathing rates and patterns, and oxygen saturation (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013). Patients' physical examinations should be undertaken as per protocols to assess the effectiveness of the management and adequate relief of symptoms. Evaluation of the patients' fluid balance, peripheral perfusion, body weight, jugular venous pressure, extent of edema, and urine output are considered to monitor volume status. In addition, the daily monitoring of the laboratory tests helps also to monitor the effective doses and the potential side effects of current medications. This includes but not limited to blood urea nitrogen (BUN), creatinine, potassium, and sodium (Lindenfeld et al., 2010; Yancy et al., 2013).

Evaluation and management of the coexistent comorbidities is a key component of care (McMurray et al., 2012). Symptom management of AHF should consider other underlying causes such as COPD and anemia (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013). Moreover, optimal management of certain comorbidities such as CAD, hypertension, and DM is associated with enhanced clinical outcomes among discharged HF patients (Yancy et al., 2013).

Appropriate educational sessions should be provided to the patient and family (or caregivers) by a multidisciplinary team. Education should be initiated earlier after the stabilization phase (taking into consideration the suitability of timing and the patient's willingness and cooperation) and continued across the hospital course to include the pre-discharge and post-discharge plan. The in-hospital educational sessions or materials should ensure the patient's proper understanding of the disease etiology, underlying causes of the symptoms, and proposed management. This could help to enhance the patient's self-care and medication adherence. Also, intensive delivery of discharge instructions coupled with a well-coordinated follow-up of care, have shown improved clinical outcomes, reduced cost of care, and decreased risk of re-hospitalization or death among hospitalized HF patients (Yancy et al., 2013). Discharge instructions should emphasize the following: ensuring adherence to the prescribed discharge medications, worsening signs and symptoms and when to refer for medical counseling, monitoring of daily weight and flexible diuretic therapy, activity level including sexual activity, exercises and training, diet (i.e.: Sodium intake precautions <3g/day) and fluid restrictions, smoking cessation, highlighting alcohol consumption amounts, immunization, and assigning follow-up appointments within 7 to 14 days post discharge (Lindenfeld et al., 2010; McMurray et al., 2012; Yancy et al., 2013).

Finally, the evidence-based guidelines have been developed to improve and standardize the care provided to patients. Consequently the quality of care could be measured using two distinct types of performance measures: process and outcome measures *(Class I, B).* A document that includes performance measures for inpatients HF care was recently published by the ACCF/AHA/American Medical Association–Physician Consortium for Performance Improvement (Yancy et al., 2013).

The process performance measures mainly reflect the adherence to the management guidelines in the context of delivered care, such as the prescription of recommended medications (i.e.: beta-blocker) or undertaking the appropriate procedure or diagnostic measure such as LVEF assessment during hospitalization and on discharge, while the outcome measures include the 30-day mortality and 30-day readmission rates (Muzaffarian et al., 2014).

ESC guidelines highlight the significant role of the management programs in achieving holistic care for HF patients. The integrated role of the nurse specialist was shown by several studies as an essential component of chronic HF disease management. The great impact was on the awareness of self-care, optimized chronic therapy, reduced readmission rates, and reduced exacerbation episodes (McDonagh et al., 2011; McMurray et al., 2012). On the other hand, there is insufficient literature about the significant role of advanced practice nursing in AHF management. Usually at the earlier stages of AHF, medical therapy constitutes the major part of the management where the key nursing role is contributing to improve outcomes through astute patient assessment, well structured and coordinated care, patient education, optimization of medical treatment, and psychosocial support. Moreover, the HF nurse specialist can ensure the application of evidence-based practice through the monitoring of HF quality indicators. Furthermore the extended role of nurses includes optimal discharge plan according to proposed discharge criteria of HF patients, in addition to gathering the follow-up plan after patient discharge.

The following chapter proposes a clinical pathway and pre-printed orders for patients admitted with AHF.

CHAPTER 3

PROPOSED IN-HOSPITAL HEART FAILURE PROGRAM

3.1. Clinical Pathway for Acute Heart Failure

The clinical pathway (Appendix A1) is proposed to standardize practice and offer a structured approach to AHF management. The aim is to enhance the quality of care by improving patient outcomes, promoting patient safety, increasing patient satisfaction, and optimizing the use of resources. The pathway includes the steps noted in the evidence-based guidelines to facilitate rapid assessment and accelerate the treatment of AHF, taking into consideration the management of precipitants contributing to AHF.

When a patient presents to the ED with HF suggestive symptoms such as shortness of breath, fatigue, and lower extremities edema, the ED team is prompted to initiate rapid triage including the RN initial assessment of vital signs to recognize the hemodynamic status of the patient. Moreover the MD shall collect a careful history of comorbidities and physical assessment to recognize the acuity of illness as well as identify the causes of AHF. Accordingly, continuous monitoring of cardiac function (ECG and NYHA class) should be done along with the ordered investigations such as CXR, blood works, and echocardiography (if not done within the last 6 -12months). The ED triage duration depends on the acuteness of the clinical symptoms such that evaluation must occur with rapid treatment to relieve symptoms particularly in patients with severe respiratory distress and evident impaired tissue oxygenation (cyanosis). Oxygen therapy should be started to maintain SPO2 >94%.

Based on the clinical and diagnostic evaluations, differential diagnoses have to be ruled out including cardiac and non-cardiac causes of symptoms. Cardiac causes include acute myocardial infarction, acute pulmonary edema, cardiogenic shock, decompensated HF, and right HF. Non-cardiac causes include COPD exacerbation, pulmonary embolus or fibrosis, pneumonia/sepsis, acute respiratory distress syndrome (ARDS) and anemia. BNP tests may not be done at LMC because of its high cost and lack of coverage by the insurers, so it will not be included and this is a limitation in our clinical pathway.

The rapid management of HF symptoms should be based on the hemodynamic status and the degree of congestion and systemic perfusion. Patients are clinically classified as: 1) wet and cool if they are congested yet have inadequate perfusion (cardiogenic shock), 2) dry and cool if they are not congested but have inadequate perfusion (hypovolemic shock), or 3) wet and warm if their organ perfusion is adequate but they have congestion. Signs of congestion include shortness of breath, paroxysmal nocturnal dyspnea, jugular vein distention, rales, hepatomegaly, ascites, weight gain and peripheral edema. Signs of low perfusion are: Low blood pressure (SBP < 90 mm Hg), peripheral and nail bed cyanosis, cool extremities, altered mental status, fatigue, pre-renal azotemia and low urine output (Joseph et al., 2009). The AHF order set should be initiated once the diagnosis is confirmed and the consequent management should be held appropriately.

For dry and cool patients, intubation is indicated in case of desaturation and altered mental status. A trial of 500 mL NSS is given; if the patient responds then functional assessment is made and medications are reviewed. If there is no response to fluid bolus, then inotropes and/or vasopressors (Dobutamine and dopamine) are given

and doses titrated to SBP > 90 mmHg. Invasive hemodynamic monitoring is attempted in case of persistent hemodynamic instability. If the patient is wet and cool, then intubation, inotropes and/or vasopressors are started. When SBP > 90 mm Hg, then diuretics are given. Invasive hemodynamic monitoring may be attempted as needed. For the wet and dry patient who is mentally alert but with low saturation, non-invasive oxygen therapy is given and morphine sulfate for severe dyspnea. Loop diuretics (Furosemide) and vasodilators (nitroglycerin) are given to relieve the congestion.

In parallel to patient stabilization, other morbidities are attended to. The pathway indicates management strategies for AHF precipitant factors. For patients presenting with acute coronary syndrome, the ACS protocol should be initiated rapidly, taking into consideration the onset time of acute symptoms. Patients who present with cardiogenic shock (SBP < 90 mm Hg, CI < 2.2 L/min/m^2 with PCWP > 15 mm Hg and manifestations of end-organ hypoperfusion including oliguria, abnormal mental status, cold clammy skin, and evidence of metabolic acidosis on laboratory testing) may be in need for circulatory support such as IABP. For AHF patients with hypertensive crisis, diuretics and vasodilators in addition to morphine sulfate are recommended. Patients presenting with acute valvular disease are managed percutaneously or by surgery once stable. Furthermore, severe arrhythmias such as ventricular or atrial arrhythmias may contribute to AHF symptoms. The management should be directed according to the hemodynamic status; for those with hemodynamic compromise, electrical cardioversion is indicated, while chemical cardioversion through medications (amiodarone and diltiazem) is indicated in hemodynamically stable patients. Moreover, assessment for thromboembolism prophylaxis is highly considered in AHF patients. Prophylactic doses

of heparin should be started or even low molecular weight heparin medications unless contraindicated.

The clinical outcome of acute symptoms of HF should be evaluated and managed appropriately before the disposition of patients from the ED (see transfer criteria on page 2 of the pathway), then the care of the patient shall be communicated to the concerned team, whether to the cathlab team for PCI or operating room team for surgical decision or the CCU team for intensive care management.

At the CCU, the non-pharmacological care process should be continued with further management and observation of the clinical progress. The multidisciplinary team should continue hemodynamic monitoring, physical assessment, and optimum fluid balance. Further blood works and diagnostic procedures are indicated in addition to the circulatory and respiratory management. The pharmacological care process is continued with consideration of comorbidities and ongoing titration of medications. Certain medications are highly indicated for AHF once the acute phase has been resolved such as ACE inhibitors, beta-blockers, MRAs, ivabradine, and digoxin (see page 3 of the pathway). These medications should be considered according to the current clinical evaluation. The key-nursing role is evident through complementary assessment and management activities (see page 2 of the pathway). The CCU RNs shall initiate a therapeutic relationship with the AHF patients and assess the proper time and willingness of patients to conduct the educational sessions. Raising awareness of the patients and their significant others about the underlying disease etiology and proposed management is aimed to enhance the patients 'involvement for better self-care'. The APN role is demonstrated with the coordination of care among members of the

multidisciplinary team as well as through the monitoring of best practice and quality indicators.

Once the clinical condition is stabilized, the MD may decide to transfer the patient to the telemetry floor for further evaluation, or the patient may be discharged from the CCU if the discharge criteria are met (Appendix A3). Upon discharge, the MD shall ensure the readiness of patients for transitional care, provide documented discharge medications, and assign follow-up visits as soon as possible. Certain measures such as device therapy may be indicated later for some patients and hence proper instructions should be provided. The nursing role should be focused on education of patients about the proposed management and maintenance of their self-care following discharge. The APN role is demonstrated through the verification of adequate discharge plan and the monitoring of performance process measures, documentation of discharge instructions and follow-up appointments.

3.2. Preprinted Orders

The following (Appendix A2) is the proposed pre-set orders for AHF management at LMC

CHAPTER IV

PROGRAM IMPLEMENTATION AND EVALUATION

4.1. Piloting Phase

The proposed clinical pathway and AHF pre-printed order set will be presented to the multidisciplinary team including cardiologists, nurses, pharmacists, and dieticians at LMC for review and approval. Then, the medical record committee should approve it before the piloting and implementation phase. Once the approval is secured, communication with the concerned staff in the ED and CCU should be initiated as well as awareness sessions to ensure their proper understanding of and adherence to the pathway. Consequently, a piloting phase can be started over a period of three months to check the applicability within the available resources at LMC, and to check the feedback evaluation from members of the health care team about the feasibility of its implementation.

4.2. Evaluation Phase

During a 6-months intervention phase of the AHF proposed pathway and pre-set orders, a process evaluation should be noted as during the pilot phase. Some issues may need to be clarified to the concerned staff taking into consideration their beneficial feedback as end users at the application level.

Moreover, in order to measure the quality of care, process and outcome performance measures should be noted (see Appendix A4) before and after the implementation phase. A retrospective medical record review over 1 year prior to

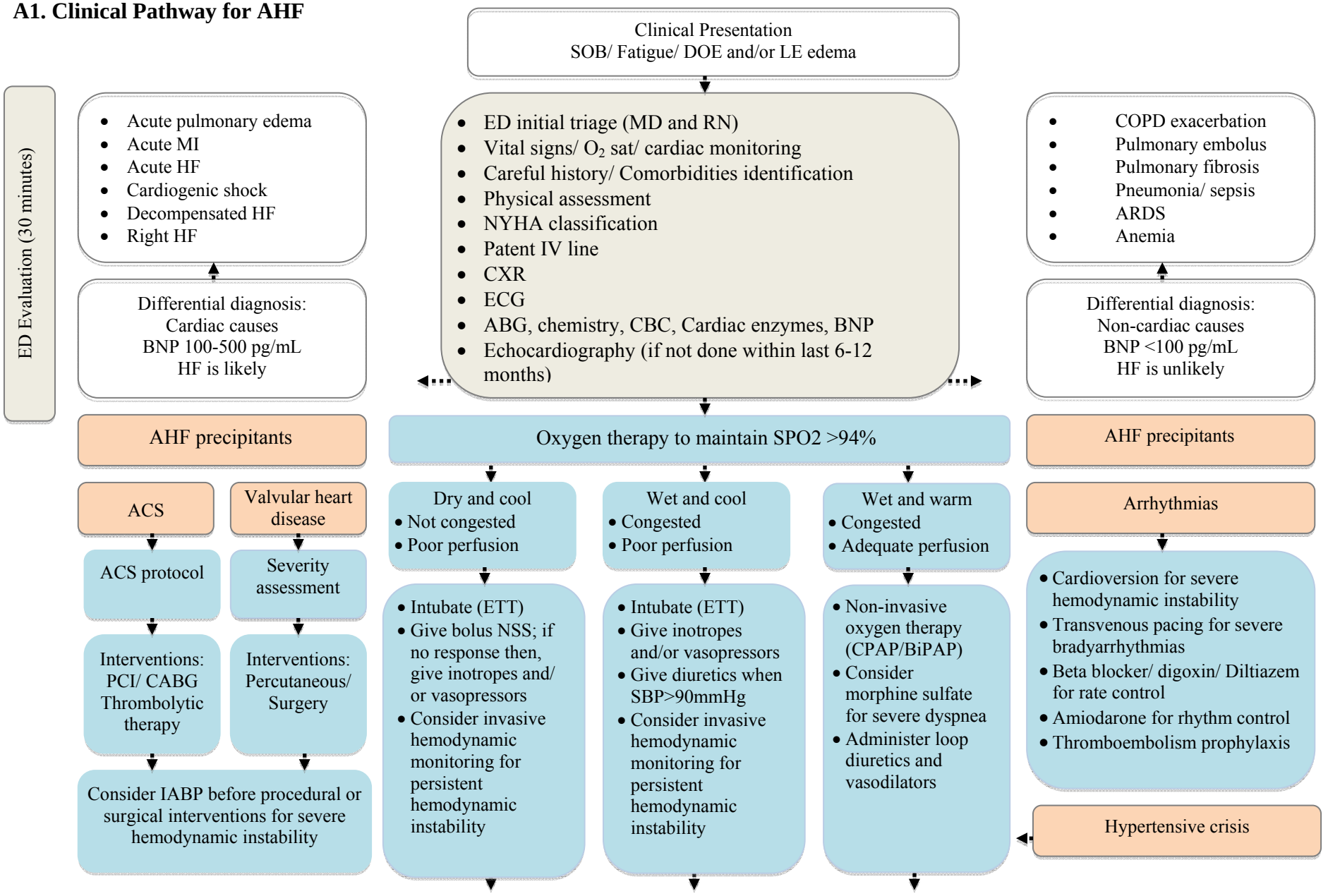
implementation of the pathway will be conducted and the following noted: percent of patients who underwent echocardiography for LVEF assessment, discharge medications prescribed such as ACEI/ARB and beta-blocker (process performance measures), mortality and readmission rates at 30, 60 and 90 days following hospital discharge (outcomes measures). The same evaluations will be performed over 1 year after the first 6 months of implementation of the pathway, in addition to documentation of discharge instructions.

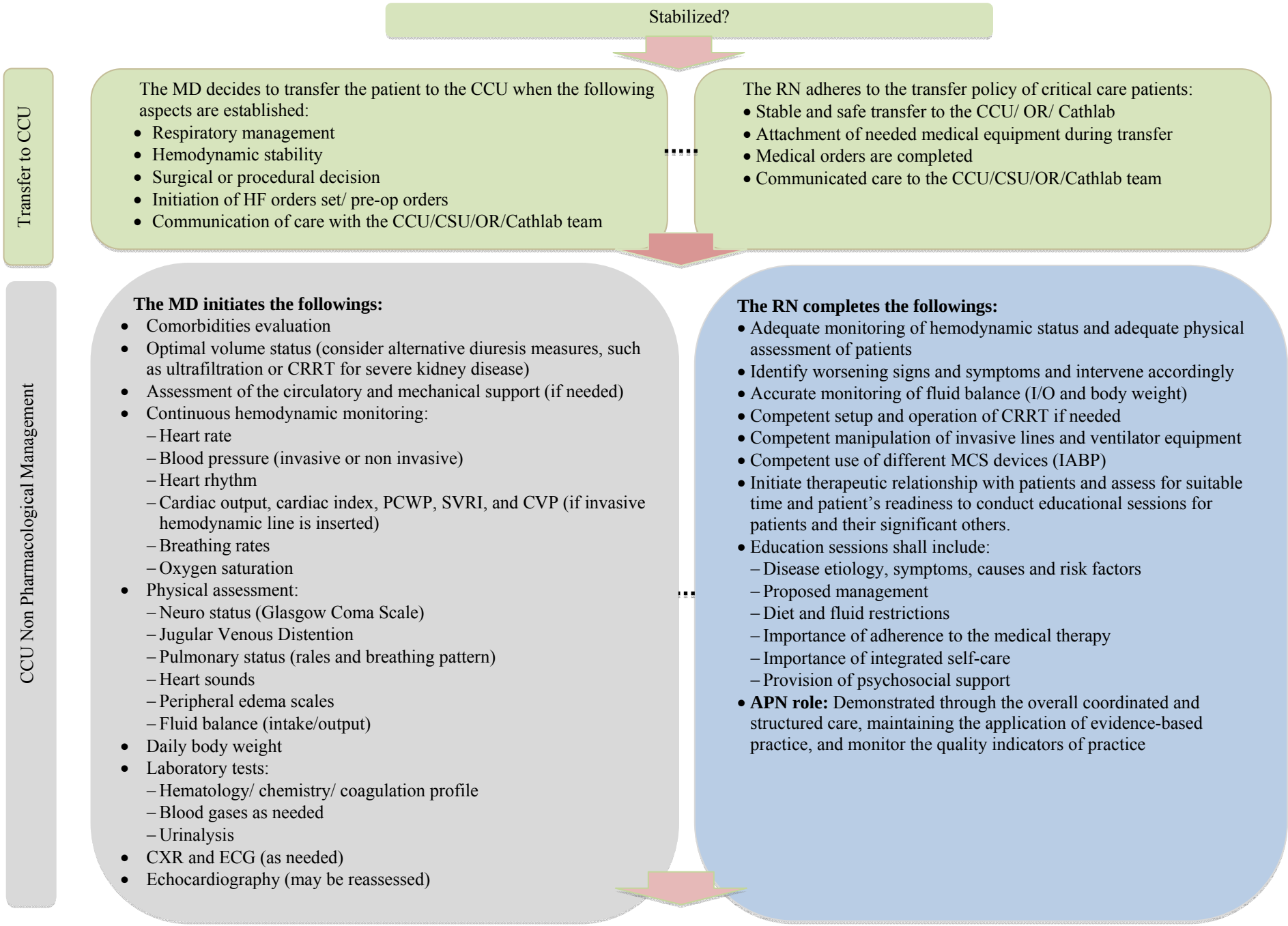
4.3. Conclusion

The successful application of the proposed pathway and pre-set orders could be done through the professional and mutual communication among multidisciplinary team members. The literature has shown that improved outcomes of AHF patients were achieved through the adoption of evidence-based practice; however integrated clinical expertise is also recommended at the level of the individual patient, depending on the comorbidities present. BNP measurement, which is used to guide treatment, is not a part of the pathway. This is a limitation that recommends the negotiation with insurers to cover the BNP test.

APPENDIX

A1. Clinical Pathway for AHF




Stabilized?
Transfer to CCU
The MD decides to transfer the patient to the CCU when the following aspects are established:
• Respiratory management
• Hemodynamic stability
• Surgical or procedural decision
• Initiation of HF orders set/ pre-op orders
• Communication of care with the CCU/CSU/OR/Cathlab team
The RN adheres to the transfer policy of critical care patients:
• Stable and safe transfer to the CCU/ OR/ Cathlab
• Attachment of needed medical equipment during transfer
• Medical orders are completed
• Communicated care to the CCU/CSU/OR/Cathlab team
CCU Non Pharmacological Management
The MD initiates the followings:
• Comorbidities evaluation
• Optimal volume status (consider alternative diuresis measures, such as ultrafiltration or CRRT for severe kidney disease)
• Assessment of the circulatory and mechanical support (if needed)
• Continuous hemodynamic monitoring:
– Heart rate
– Blood pressure (invasive or non invasive)
– Heart rhythm
– Cardiac output, cardiac index, PCWP, SVRI, and CVP (if invasive hemodynamic line is inserted)
– Breathing rates
– Oxygen saturation
• Physical assessment:
– Neuro status (Glasgow Coma Scale)
– Jugular Venous Distention
– Pulmonary status (rales and breathing pattern)
– Heart sounds
– Peripheral edema scales
– Fluid balance (intake/output)
• Daily body weight
• Laboratory tests:
– Hematology/ chemistry/ coagulation profile
– Blood gases as needed
– Urinalysis
• CXR and ECG (as needed)
• Echocardiography (may be reassessed)
The RN completes the followings:
• Adequate monitoring of hemodynamic status and adequate physical assessment of patients
• Identify worsening signs and symptoms and intervene accordingly
• Accurate monitoring of fluid balance (I/O and body weight)
• Competent setup and operation of CRRT if needed
• Competent manipulation of invasive lines and ventilator equipment
• Competent use of different MCS devices (IABP)
• Initiate therapeutic relationship with patients and assess for suitable time and patient's readiness to conduct educational sessions for patients and their significant others.
• Education sessions shall include:
– Disease etiology, symptoms, causes and risk factors
– Proposed management
– Diet and fluid restrictions
– Importance of adherence to the medical therapy
– Importance of integrated self-care
– Provision of psychosocial support
• APN role: Demonstrated through the overall coordinated and structured care, maintaining the application of evidence-based practice, and monitor the quality indicators of practice

CCU Pharmacological Management

The MD completes:

- Comorbidities management (Metabolic syndrome, AF, hypertension, chronic kidney disease, etc.…)
- Medication reconciliation
- Titration of concomitant medication doses including inotropes and vasopressors
- Assessment of the risk for Venous Thromboembolism and gastric ulcers
- ACS treatment protocol (if initiated)

ACEI/ ARB	• Start if reduced EF unless contraindicated • Start at low doses then gradual increase if lower doses well tolerated • Captopril 6.25mg TID/ Enalapril 2.5mg OD/ candesartan 4-8 mg OD • Patients who cannot tolerate ACEI should be started with ARB • Monitor serum creatinine and K level
Beta- Blocker	• Initiate with reduced EF early after stabilization when volume status optimum • Start with small doses (i.e.: Bisoprolol 1.25 mg once daily), then up titrate as far as possible before discharge • Monitor bradyarrhythmias and hypotension
MRA	• Use with ACEI/ ARB and beta-blockers with reduced EF at the earlier stages unless contraindicated • Start with Spironolactone 12.5 to 25 mg daily, or Eplerenone doses of 25 mg/d, increasing to 50 mg OD • Monitor serum K and Cr
Ivabradine	• Use with ACEI, beta blockers and MRA, only in patients with sinus rhythm and heart rate ≥□□0□□□□t/min • Consider in patients with either beta-blocker contraindication or intolerance, but with current ACEI doses • Can be given at 7.5 mg BID as tolerated
Digoxin	• May be considered with reduced EF. • May be used to control the rate, especially in AF when up-titration of beta-blocker doses is not possible • Doses should be 0.125 to 0.25 mg OD. • Monitor serum digoxin level, serum electrolytes, and heart rhythm

Stabilized?

Discharge plan

Before discharge, the MD may transfer the patient to the telemetry unit for further monitoring and/or discharge the patient when the following criteria is met:

- **Discharge criteria:**
  - Improved respiratory rate since admission
  - Weight decreased since admission
  - Congestion absent (no dyspnea, peripheral edema) for at least 48 hours
  - BP stable off inotropes for at least 48 hours
  - Optimal titration of oral therapy for 24 hours
  - Patient verbalizes understanding of discharge medications, diet, salt and fluid restrictions, and symptoms to be reported after discharge
  - Creatinine reduced; electrolytes within normal
- **On discharge, the MD shall:**
  - Assess the readiness of patients for transitional care
  - Ensure patients' understanding of their therapy
  - Consider device therapy in appropriate patients when needed after optimal medical management (CRT-P and CRT-D)
  - Assess the criteria for long-term mechanical support management in patients with advanced HF (LVAD)
  - Schedule follow-up visits within 7-14 days post discharge
  - Document discharge plan including prescribed medications
  - Consider palliative care for appropriate advanced HF cases

The RN completes the followings:

- Intensive educational sessions on discharge to include
  - Self care maintenance
  - Adherence to the prescribed medications
  - Identified worsening signs and symptoms and when to refer to medical counseling
  - Monitoring of daily weight and flexible diuretic therapy as indicated
  - Activity level including sexual activity
  - Exercise and trainings
  - Diet (i.e.: Sodium intake precautions <2 g/day) and fluid restrictions
  - Smoking cessation
  - Restricted alcohol consumption
  - Immunization

- **APN role:** verification of adequate discharge plan, monitoring of process performance measures through prescribed discharge medications and documented procedures such as echocardiography results, discharge instruction and follow up appointment.

A2. Preprinted Orders for AHF

LABIB MEDICAL CENTER s.a.l Acute Heart Failure – Order Set PH-FO-00 | Edition: 1 | *Page 1 of 3*	Patient Identification

☑ **Check the Applicable Order**	**Nurse's Signature**	**Date &Time**
Acute Heart Failure Status: ☐Acute Decompensation of Chronic HF ☐New Onset HF **Etiology of HF:** ☐CHD ☐Hypertension ☐Valvular ☐Idiopathic ☐Other_____ **Patient has:** ☐HF-REF ☐HF-PEF EF: _____ % **NYHA Class (select one):** ☐I ☐II ☐III ☐IV **ACC HF Stage (select one):** ☐A ☐B ☐C ☐D		
Allergies/ reactions: ☐ No Known Allergies ☐ Other: _____		
Vital Signs: ☐Continuous monitoring ☐ Every _____ hrs Non-Invasive Hemodynamics: ☐BP, HR, RR, SPO2 Invasive Hemodynamics: ☐ AP ☐CI, PAP, PCWP, SVRI		
Activity: ☐ CBR ☐ RBR ☐OOB ☐Assist/ encourage ambulation every shift		
IV lines: ☐Peripheral ☐Central IV Fluids: _____ IV at _____ mL/hr IV Fluids: _____ IV at _____ mL/hr IV Fluids: _____ IV at _____ mL/hr		

Lab tests:	**Now**	**AM**	**Daily**	**Repeat in**
☐ CBC	☐	☐	☐	_____
☐ TSH	☐	☐		
☐ Digoxin level	☐	☐		_____
Cardiac enzymes				
☐ roponin I	☐	☐	☐	_____
☐ CPK	☐	☐	☐	_____
☐ CK-MB	☐	☐	☐	_____
☐ Blood gases	☐	☐	☐	_____
☐ Urinalysis	☐	☐	☐	_____
Cultures				
☐ _____	☐			_____
☐ _____	☐			_____

	Now	**AM**	**Daily**	**Repeat in**
☐ Blood sugar	☐	☐	☐	_____
☐ BUN	☐	☐	☐	
☐ Creatinine	☐	☐	☐	_____
☐ Electrolytes	☐	☐	☐	_____
☐ Calcium	☐	☐	☐	_____
☐ Magnesium	☐	☐	☐	_____
☐ Phosphorus	☐	☐	☐	_____
☐ Liver enzymes	☐	☐	☐	_____
☐ PT	☐	☐	☐	_____
☐ PTT	☐	☐	☐	_____
☐ INR	☐	☐	☐	_____
☐ Iron/ TIBC	☐	☐	☐	_____
☐ Lipid profile	☐	☐	☐	_____

Diagnostic Procedures:
☐ ECG
☐ CXR
☐ Echocardiography ☐ Not needed, done before: date _____ EF ___ % ☐ Re-assess in _____

Oxygen Therapy: Titrate oxygen therapy to maintain SPO2 > 94%
Non-invasive therapy:
☐ Supplemental O2 at ____ L/min by _____
☐ CPAP _____
☐ BiPAP: IPAP _____ EPAP _____ PSV _____
Invasive therapy:
☐ ETT _____
☐ Ventilator settings: Mode ____TV ____ mL RR ___ breath/min FiO2 ___% PEEP ___ PSV ____cm

Physician Name _____Signature _____ Date &Time: _____

☑ **Check the Applicable Order**	**Nurse's Signature**	**Date &Time**
Services Consultation: ☐ Cardiac catheterization ☐ Cardiac-surgery ☐ Nephrology ☐ Pulmonary ☐ Dietary ☐ Other _____		
General Care: ☐ Insert foley catheter ☐ Insert nasogastric (NG) tube ☐ Weight daily (at same time and using same scale) ☐ Monitor Intake/ output every _____ hrs and assess daily cumulative fluid balance Restrict fluids to: ☐ 2000 mL/24 hrs (recommended standard) ☐ 1500 mL/24 (for those with hyponatremia)		
Diet/ Nutrition: ☐ NPO ☐ NG feeding _____ /24hrs ☐ PO ☐ Regular diet (sodium > 2g) ☐ Sodium restricted diet (< 2g) ☐ Diabetic diet ☐ Protein restricted diet ☐ Low animal fat diet ☐ Other _____		
Medications: **Diuretics** ☐ Furosemide (Lasix) _____ mg IV now, and _____ mg IV in _____ hrs ☐ If urine output < _____ mL in 3 hours, give another _____ mg and repeat in _____ hrs later ☐ Furosemide _____ mg/hr continuous drip ☐ Furosemide _____ mg every _____ hrs ☐ IV ☐ PO ☐ Hydrochlorothiazide _____ mg PO every _____ hrs ☐ Other: Name _____ Dose _____ Route _____ Frequency _____ hrs **Nitrates** ☐ Isosorbide dinitrate: Name _____ Dose _____ Route _____ every _____ hrs ☐ Isosorbide dinitrate: Name _____ Dose _____ Route _____ every _____ hrs **Opiates** ☐ Morphine sulfate _____ mg IV push (for severe dyspnea or pain) ☐ now ☐ every _____ hrs **Inotropes/ vasopressors** ☐ Dobutamine (250mg /250mL). Start at _____ mcg/ kg/ min IV continuous infusion and titrate as per protocol (0.5 – 20 mcg/kg/min). Notify MD and hold if SBP> ____ mmHg; HR > _____ beat/min ☐ Dopamine (200mg /250mL). Start at _____ mcg/ kg/ min IV continuous infusion and titrate as per protocol (1 – 20 mcg/kg/min). Notify MD and hold if SBP> ____ mmHg; HR > _____ beat/min ☐ Norepinephrine (8mg /250mL). Start at _____ mcg/ min IV continuous infusion and titrate as per protocol (2 – 20 mcg/min). Notify MD and hold if SBP> ____ mmHg; HR > _____ beat/min ☐ Other: Name _____ Concentration _____. Start at _____ IV continuous infusion and titrate as _____ Notify MD and hold if SBP> ____ mmHg; HR > _____ beat/min **Anticoagulants** ☐ Heparin _____ units IV bolus ☐ Heparin (25000 units/ 250 mL) _____ units IV continuous infusion. Titrate as per heparin protocol ☐ Low molecular weight heparin (LMWH). Name _____ dose _____ mg subcutaneously every _____ hrs ☐ Other: Name _____ dose _____ mg route _____ frequency _____ hrs **Potassium Supplement** KCL _____ mEq every _____ hrs ☐ IV ☐ PO/ NG		

Physician Name _____Signature _____ Date &Time: _____

☑ **Check the Applicable Order**	**Nurse's Signature**	**Date &Time**

Other Medications:

☐ **Aspirin** _____ mg PO/NG OD

☐ **Amiodarone** _____ mg IV loading dose, then _____ mg/ 24 hrs IV continuous infusion maintenance dose. Notify MD and hold if SBP < _____ mmHg; HR < _____ beat/min

☐ **Digoxin** _____ mg PO OD (presently on digoxin). Monitor digoxin serum level and potassium serum level, and HR

☐ **Digoxin** _____ mg (0.125-0.25 mg) PO OD. Notify MD if HR < _____ beat/ min and Potassium level < _____ mEq/ L

☐ **Statin/ lipid medication** _____ mg PO OD every evening

☐ Statin is contraindicated because _____

Angiotensin Converting Enzyme Inhibitor (ACEI):

☐ Cannot tolerate ACEI/ or ACEI is contraindicated because:

☐ Severe cough ☐ Renal insufficiency (Cr is > 2.0 and GFR is < 30) ☐ Angioedema

☐ Significant renal artery stenosis ☐ Other _____

☐ Name _____ dose _____ mg PO every _____ hrs

Angiotensin Receptor Blocker (ARB) (used when ACEI cannot be tolerated or contraindicated):

☐ Cannot tolerate ARB because of:

☐ Severe cough ☐ Renal insufficiency (Cr is > 2.0 and GFR is < 30) ☐ Angioedema

☐ Significant renal artery stenosis ☐ Other _____

☐ Name _____ dose _____ mg PO every _____ hrs

Beta-Blocker (Do not begin until volume status is stabilized):

☐ Cannot tolerate beta-blocker because of:

☐ Bradycardia ☐ Significant asthma or COPD ☐ Other _____

☐ Name _____ dose _____ mg PO every _____ hrs (start with small doses)

Notify MD and hold if SBP< _____ mmHg; HR < _____ beat/ min

Mineralocorticoid Receptor Antagonist (MRA) (Used after the initiation of ACEI/ARB and beta-blocker)

☐ Spironolactone _____ mg PO every _____ hrs

☐ Other: Name _____ dose _____ mg PO every _____ hrs

Monitor potassium and creatinine serum levels

Ivabradine (Start in patients with sinus rhythm only and EF <35%)

Not indicated because of: ☐ HR< 70 beat/min ☐ Irregular heart rhythm

Ivabradine _____ mg PO every ____ hrs

Venous thromboembolism (VTE) prophylaxis (If anticoagulant therapy is not yet initiated)

☐ Heparin 5000 units subcutaneously every 8 hrs

☐ Sequential compression device (SCD)

Gastric Ulcer Prophylaxis

☐ Name _____ dose _____ every _____ hrs ☐ IV ☐ PO/ NG

Others:

☐ Name _____ dose _____ route _____ frequency _____

☐ Name _____ dose _____ route _____ frequency _____

☐ Name _____ dose _____ route _____ frequency _____

Physician Name _____Signature _____ Date &Time: _____

A3. AHF Discharge Criteria

			Patient Identification
LABIB MEDICAL CENTER s.a.l			
Acute Heart Failure – Discharge Checklist			
PH-FO-00	Edition: 1	*Page 1 of 1*	

☑ **Check the following discharge criteria**	**Comments**
Before Discharge ☐ Successful weaning of invasive respiratory and circulatory support ☐ Improved respiratory condition since admission (respiratory patterns and rate are adequate) ☐ Congestion absent (no dyspnea, peripheral edema) for at least 48 hours ☐ At least near optimal volume status achieved ☐ Hemodynamic stability (BP stable and off inotropes for at least 48 hours) ☐ Weight decreased since admission ☐ Optimal titration of oral therapy for 24 hours ☐ Careful assessment of renal function and serum electrolytes ☐ Ambulated to assess the functional capacity and symptomatic hypotension ☐ Readiness assessment for transitional care **On Discharge** Patient and family education completed (60 minutes session) including: ☐ Detailed discharge medication and the need for medication adherence ☐ Precipitating factors and self care maintenance ☐ Diet, salt and fluid restrictions ☐ Recommended activity level ☐ Symptoms to be reported after discharge (when and how often) ☐ Monitoring of daily weight ☐ Follow-up visits schedule ☐ Follow-up tests to be done ☐ Smoking cessation ☐ Restricted alcohol consumption ☐ Immunization ☐ Access to a formal heart failure disease management program or team	

A4. AHF Quality Performance Measures

	LABIB MEDICAL CENTER s.a.l
	Acute Heart Failure – In-Hospital Quality of Care Measures Sheet

Clinical Indicator	Performance	Measurement
ACE/ ARB therapy at discharge for patients with left ventricular systolic dysfunction	Process	Percentage of patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% who were prescribed ACEI or ARB at hospital discharge **Numerator:** All patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% who were prescribed ACEI or ARB therapy at hospital discharge **Denominator:** All patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% Denominator exceptions: Documentation of contraindication for ACEI/ ARB therapy, such as: • Hypersensitivity to ACEI and ARBs • Hypotension • Renal dysfunction • Bilateral renal artery stenosis • In-hospital mortality
Beta-Blocker therapy at discharge for patients with left ventricualr systolic dysfunction	Process	Percentage of patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% who were prescribed beta-blocker at hospital discharge **Numerator:** All patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% who were prescribed beta-blocker therapy at hospital discharge **Denominator:** All patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF <40% Denominator exceptions: Documentation of contraindication for beta-blocker therapy, such as: • Hypersensitivity to beta-blocker • Improper volume status (overload) • Inotropes/ vasopressors treatment • Bradyarrhythmias • Hypotension • In-hospital mortality
Left Ventricular Ejection Fraction Assessment	Process	Percentage of patients aged ≥18 years with a principal discharge diagnosis of HF with documentation in the hospital record of the results of an LVEF assessment performed either before arrival or during hospitalization, or documentation in the hospital record that LVEF assessment is planned for after discharge **Numerator:** HF patients with documentation in the hospital record that LVEF assessment was done in the last 6-12 months before arrival, during hospitalization, or is planned to be done after discharge **Denominator:** All patients aged ≥18 years with a diagnosis of HF with a current or prior LVEF assessment Denominator exceptions: • LVEF assessment done within the last 6-12 months • In-hospital mortality
Heart Failure In-hospital mortality	Outcome	Percentage of in-patient mortality for patients with a primary diagnosis of HF **Numerator:** All patients aged ≥18 with a primary diagnosis of HF who passed away during hospitalization **Denominator:** All patients aged ≥18 who was admitted to the hospital with a diagnosis of HF

BIBLIOGRAPHY

Abraham, W., Adams, K., Fonarow, G., Costanzo, M., Berkowitz, R., & LeJemtel, T. et al. (2005). In-Hospital Mortality in Patients With Acute Decompensated Heart Failure Requiring Intravenous Vasoactive Medications. *Journal Of The American College Of Cardiology*, *46*(1), 57-64.

Abyad, A. (2001). Health Care for Older Persons: A Country Profile-Lebanon. *Journal Of The American Geriatrics Society*, *49*(10), 1366-1370.

Ambrosy, A., Butler, J., Ahmed, A., Vaduganathan, M., van Veldhuisen, D., Colucci, W., & Gheorghiade, M. (2014). The Use of Digoxin in Patients With Worsening Chronic Heart Failure. *Journal Of The American College Of Cardiology*, *63*(18), 1823-1832.

Ambrosy, A. P., Fonarow, G. C., Butler, J., Chioncel, O., Greene, S. J., Vaduganathan, M., & ... Gheorghiade, M. (2014). The global health and economic burden of hospitalizations for heart failure: lessons learned from hospitalized heart failure registries. *Journal Of The American College Of Cardiology (JACC)*, *63*(12), 1123-1133.

Casey, G. (2013). Heart failure. *Kai Tiaki Nursing New Zealand*, *19*(2), 20-24

Cowie, M. R., Anker, S. D., Cleland, J. F. G., Filippatos, G., Jaarsma, T., Jourdain, P,…López-Sendón, J. (2014). Improving care for patients with acute heart failure. Before, during and after hospitalization. *ESC Heart Failure*. Published online at http://onlinelibrary.wiley.com/doi/10.1002/ehf2.12021/pdf. DOI: 10.1002/ehf2.12021

Deek, H., Skouri, H., & Noureddine, S. (2014). Readmission rates and related factors in heart failure patients: A study in Lebanon. *Collegian*. doi:10.1016/j.colegn.2014.11.001

Fahed, A. C., Safa, R. M., Haddad, F. F., Bitar, F. F., Andary, R. R., Arabi, M. T., et al. (2011). Homozygous familial hypercholesterolemia in Lebanon: A genotype/phenotype correlation. *Molecular Genetics and Metabolism*, *102*(2), 181-188.

Givertz, M., Teerlink, J., Albert, N., Westlake Canary, C., Collins, S., & Colvin-Adams, M. et al. (2013). Acute Decompensated Heart Failure: Update on New and Emerging Evidence and Directions for Future Research. *Journal Of Cardiac Failure*, *19*(6), 371-389.

Go, A. S., Mozaffarian, D., Roger, V. L., Benjamin, E. J., Berry, J. D., Blaha, M. J., & ... Turan, T. N. (2014). Heart disease and stroke statistics--2014 update: a report from the American Heart Association. *Circulation*, *129*(3), e28-e292.

Harb, H. (2011). *Statistical bulletin. Ministry of Public Health.www.moph.gov.lb*. Retrieved 2 December 2014, from http://www.moph.gov.lb/Publications/Documents/Statistical%20Bulletin%202011.pdf

Hajjar RR. (2013). *The Annual Meeting of the American Geriatric Society – Special interest Group on Ethno-geriatrics, Grapevine, Texas, USA.* Retrieved from http://www.syndicateofhospitals.org.lb/Content/uploads/SyndicateMagazinePdfs/1555_28-31eng.pdf

Joseph, S. M., Cedars, A. M., Ewald, G. A., Geltman, E. M., & Mann, D. L. (2009). Acute decompensated heart failure: Contemporary Medical Management. *Texas Heart Institute, 36*, 510-520.

Lindenfeld, J., Albert, N., Boehmer, J., Collins, S., Ezekowitz, J., Givertz, M., & ... Walsh, M. (2010). HFSA 2010 Comprehensive Heart Failure Practice Guideline. *Journal Of Cardiac Failure*, *16*(6), e1-194.

McDonagh, T., Blue, L., Clark, A., Dahlström, U., Ekman, I., &Lainscak, M. et al. (2011). European Society of Cardiology Heart Failure Association Standards for delivering heart failure care. *European Journal Of Heart Failure*, *13*(3), 235-241.

McMurray, J., Adamopoulos, S., Anker, S., Auricchio, A., Bohm, M., & Dickstein, K. et al. (2012). ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. *European Heart Journal*, *33*(14), 1787-1847.

Mizuno, M., Sato, N., Kajimoto, K., Sakata, Y., Minami, Y., & Munakata, R. et al. (2014). Intra-aortic balloon counterpulsation for acute decompensated heart failure. *International Journal Of Cardiology*, *176*(3), 1444-1446.

Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M.,…Turner, M. B. (2014). Heart and stroke statistics-2015 update: A report from the American Heart Association. *Circulation.* Advance online publication December 17, 2014. DOI: 10.1161/CIR.0000000000000152

Noureddine, S., Froelicher, E. S., Sibai, A. M., & Dakik, H. (2010). Response to a cardiac event in relation to cardiac knowledge and risk perception in a Lebanese sample: A cross sectional survey. *International Journal of Nursing Studies*, *47*(3), 332—341.

Noureddine, S., Massouh, A., & Froelicher, E. S. (2013). Perceptions of heart disease in community-dwelling Lebanese. *European Journal of Cardiovascular Nursing*, *12*(1), 56—63.

Oliva, F., Mortara, A., Cacciatore, G., Chinaglia, A., Di Lenarda, A., & Gorini, M. et al. (2012). Acute heart failure patient profiles, management and in-hospital outcome: results of the Italian Registry on Heart Failure Outcome. *European Journal Of Heart Failure*, *14*(11), 1208-1217.

Ponikowski, P., Anker, S., Al Habib, K., Cowie, M., Force, T., & Hu, S. et al. (2014). Heart failure: preventing disease and death worldwide. *ESC Heart Failure*, n/a-n/a.

Roger, V. (2013). Epidemiology of Heart Failure.*Circulation Research*, *113*(6), 646-659.

The World Health Organization. (2009). *Chronic Disease Risk Factor Surveillance Data Book for Lebanon*. Retrieved 2 December 2014, from http://www.who.int/chp/steps/2008_STEPS_Lebanon.pdf

The World Factbook. (2014). Retrieved 2 December 2014, from https://www.cia.gov/library/publications/the-world-factbook/geos/le.html

Weintraub, N., Collins, S., Pang, P., Levy, P., Anderson, A., & Arslanian-Engoren, C. et al. (2010). Acute Heart Failure Syndromes: Emergency Department Presentation, Treatment, and Disposition: Current Approaches and Future Aims:

A Scientific Statement From the American Heart Association. *Circulation*, *122*(19), 1975-1996.

World Health Organization. (2011). *Noncommunicable Diseases (NCD) Country Profiles, 2011*. Retrieved 2 December 2014, from http://www.who.int/nmh/countries/2011/lbn_en.pdf

World Health Organization. (2014). *Noncommunicable Diseases (NCD) Country Profiles , 2014.*. Retrieved 2 December 2014, fromhttp://www.who.int/nmh/countries/lbn_en.pdf

Yancy, C. W., Jessup, M., Bozkurt, B., Butler, J., Casey, D. E., Drazner, M. H., et al. (2013). 2013 ACCF/AHA guideline for the management of heart failure: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, *62*(16), e147—e239.