ABSTRACT

The study was carried out to determine the impact of the inclusion of the β-blocker, carvedilol, a "third generation" β-blocker with α-vasodilating properties, to the standard treatment guidelines for heart failure, on mortality and length of hospital stay. The updated treatment guidelines were adopted and implemented at the medicine directorate of the Komfo Anokye Teaching Hospital in January, 2006 based on the updated treatment guidelines of the American College of Cardiologists, American Heart Association, Heart Failure Society of America and European Society of Cardiology. These guidelines recommend heart failure in-patients receiving an Angiotensin-Converting-Enzymes Inhibitor and/or Angiotensin-Receptor-Blocker, a Diuretic, a β-Blocker and an Aldosterone-Antagonist. The study took into consideration the age and sex distribution of the patients, their conditions on admission and the causes of heart failure presented at the medical wards.

It was retrospective, covering a four-year period. The period of study was divided into two i.e. January 2004 to December 2005 before the implementation of updated treatment guidelines and January 2006 to December 2007 after its implementation. All heart failure patients who fell within these periods were used. The setting was the medicine directorate of Komfo Anokye Teaching Hospital, Kumasi, Ghana, West Africa.

The study involved 411 heart failure patients consisting of 233 males and 178 females. The ages of the patients admitted with heart failure during the period under review ranged from 13 to 100 years. The median age of the patients was 57.6 years

with a standard deviation (SD) of 18.12. The male patients constituted 56.7% whiles 43.3% were female patients, the disease affecting more males than females during the period under review. The main causes of heart failure were hypertension (60.3% n=248), dilated cardiomyopathy (23.4% n=96), and valvular disease (4.6% n=19). Other conditions seen in patients were anaemia (0.49% n=2) and cardiogenic shock (1.9% n=8). Conditions of the patients on admission described as conscious/stable constituted 70.8%; conscious/unstable 27.7% and unconscious/unstable 1.5%.

The implementation of the updated treatment guidelines with the β-blocker, carvedilol included led to a drastic reduction in overall mortality from 35.27% to 17.16% representing an 18.11% decrease in mortality or 50% improvement. The patients' mean length of hospital stay was 5.1 days before its implementation and 3.2 days after its implementation representing 62.7% decrease. Almost all the patients received the loop diuretic, frusemide (97.8% n=402). The ACE-inhibitor, lisinopril, was used in 49.6% (n=204) of the patients, the aldosterone-antagonist, spironolactone was used in 45.8% of the patients (n=188) and the β-blocker, carvedilol was used in 22.4% (n=92) of the patients.

In conclusion, the implementation of the updated treatment guidelines for heart failure in January 2006 which included the β-blocker, carvedilol led to a significant reduction in mortality, length of hospital stay and overall improvement in the quality of life of heart failure patients. The loop diuretic, frusemide and the ACE-inhibitor, lisinopril and the aldosterone-antagonist, spironolactone were commonly used

whiles the β-blocker, carvedilol, was woefully underutilized.

CHAPTER ONE: INTRODUCTION

1.1 BACKGROUND OF STUDY

Chavey in 2008 reported that heart failure is a disease with considerable public health impact and the incidence increases with age, therefore its associated morbidity and mortality are expected to increase[1]. After the diagnosis of heart failure, the 1-year mortality rate is 20%, reported Barclay[2].

The prognosis of advanced heart failure is poor; with mortality rates reported in clinical trials and in community data indicating that within 3-5 years, around 50% of patients with a diagnosis of heart failure will have died[3].

Statistics at the medical wards of the Komfo Anokye Teaching Hospital indicate that heart failure was the 5th cause of death among the 10 top causes of death recorded in the year 2003 accounting for 118 deaths out of a total 394 heart failure admissions representing 30% mortality. In the year, 2004, heart failure was recorded as the 7th cause of death in the medical wards accounting for 22% mortality. The overall statistics for Ghana are unavailable.

In view of the above, the treatment guidelines for heart failure patients were updated in January 2006 at the medicine directorate based on the updated treatment guidelines of the Heart Failure Society of America and the American College of Cardiology/American Heart Association in 2005 and 2006 respectively [4].

The updated treatment guidelines addressed extensively the use of the β-adrenergic

blocker, carvedilol, in addition to the standard treatment with a diuretic and angiotensin-converting enzyme inhibitor and/or angiotensin receptor blocker.

Four large clinical trials, published between 1999 and 2001, showed that the addition of the β-blocker, carvedilol, to the standard treatment above, led to a significant increase in survival, reduction in hospital admission, length of hospital stay and improvement of symptoms and well-being irrespective of aetiology[6,7,8,9].

The use of the β-blocker, carvedilol, has been recognized as the most important advancement in the management of heart failure in the past decade[4].

1.2 STATEMENT OF PURPOSE

The purpose of this study was to determine the impact of the inclusion of the β-blocker, carvedilol, in the treatment guidelines of heart failure patients on mortality and length of hospital stay.

The study also took into consideration the age and sex distribution of the heart failure patients, their conditions on admission, causes and types of heart failure presented by the patients admitted at the medical wards of Komfo Anokye Teaching Hospital.

1.3 IMPORTANCE OF STUDY

These results will guide cardiologists, physicians and other clinicians who have been previously apprehensive with the use of the β-blocker, carvedilol, due to its negative inotropic effect to have their concerns allayed with the evidence induced in this study and consider providing β-blockers to the many patients with heart failure who are in need of this therapy.

It will provide motivation to ward pharmacists in the hospital to be involved in the management of heart failure patients both in the hospital and the community.

It will also provide data for public education on causes of heart failure and their prevention.

1.4 OBJECTIVES

1. To determine the age and sex distribution of heart failure patients, their conditions on admission, the causes and types of heart failure.
2. To document the use of the β-blocker, carvedilol, in the treatment protocol of heart failure patients.
3. To determine the outcome of patients i.e. discharged or died and mean length of hospital stay with carvedilol as part of the treatment protocol.
4. To make recommendations that would improve heart failure patients' clinical care.

CHAPTER TWO: LITERATURE REVIEW

2.0 HEART FAILURE

Heart failure has been described as a condition that occurs when the heart is unable to pump enough blood to meet the metabolic demands of the body. It is also defined as a clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricles to fill with or eject blood[10]. Heart failure can either be High Cardiac Output Failure or Low Cardiac Output Failure. In high cardiac output failure, the cardiac output is normal but inadequate to meet the needs of the body tissues such as in anaemia and thyrotoxicosis whiles low cardiac output occurs when the heart is unable to fill properly or eject blood efficiently[11]

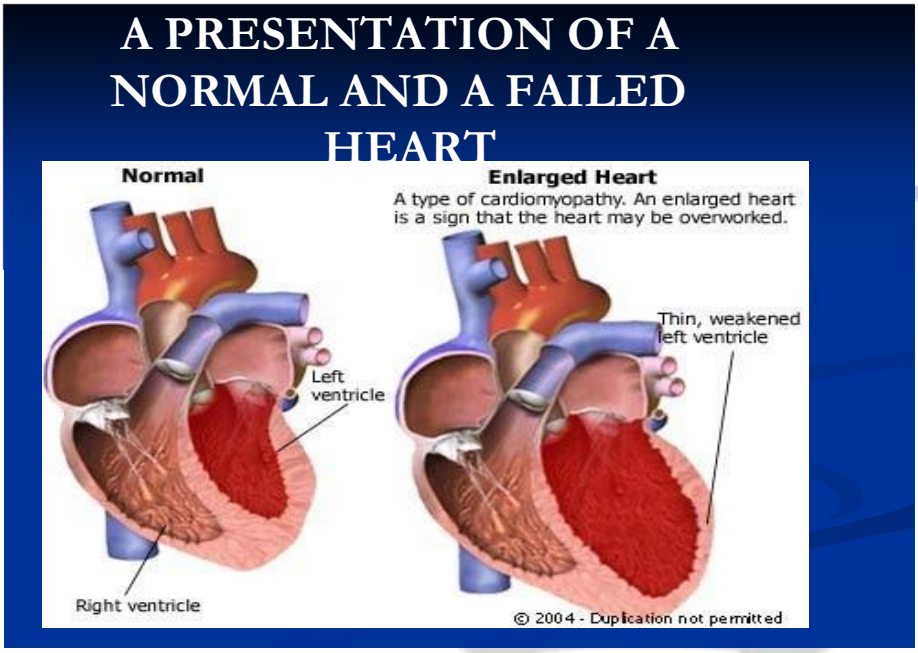


Figure 2.1: A normal and a failed heart (courtesy: Amanfo, cardiologist, children's hospital in philadelphia, U.S.A)

2.0.1 CLASSIFICATION OF HEART FAILURE

The New York Heart Association classified the condition of heart failure based on signs and symptoms of dyspnoea[3,4,5,] as follows.

CLASS 1:- no symptoms at any level of exertion/exercise

CLASS 11:-symptoms with severe exertion/exercise

CLASS111:-symptoms on moderate exertion/exercise

CLASS 1V:-symptoms with mild exertion or at rest.

These classifications have been adopted by both the Heart Failure Society of America and European Society of Cardiology as bases for their treatment guidelines. On the other hand, the American College of Cardiology and the American

Heart Association have identified four separate stages of heart failure and have classified heart failure[12,13,14,15] as follows

STAGE A:-High risk for heart failure with no structural damage or heart failure symptoms.

STAGE B:-Structural damage without heart failure symptoms

STAGE C:-Structural damage with previous or current heart failure symptoms.

STAGE D:-Refractory heart failure, specialized intervention.

To briefly compare the two classifications, patients with stage A are clearly those at high risk for developing heart failure such as patients with longstanding hypertension, coronary artery disease and diabetes mellitus requiring aggressive treatment. Patients with stage B have structural damage without any symptoms; therefore this stage is similar to the New York Heart Association class 1. Stage C is roughly similar to New York Heart Association classes II and III. Stage D can be likened to New York Heart Association class IV and includes special interventions with mechanical devices such as pace makers, and heart transplantation.

2.0.2 EPIDEMIOLOGY AND IMPACT OF HEART FAILURE

Heart failure is a leading cause of death and hospitalization in both developing and developed countries and its prevalence is expected to increase as the population ages. It affects 1% to 2% of persons aged 40—59 years. In the elderly above 65 years, the prevalence is 3% to 5% and it is 8% to 16% in those over 75 years[16]. The risk of sudden cardiac death is increased 6-to-9-fold in patients with heart failure and the disease affects more men than women[16]. It accounts for between 5-10% of

adult admission to hospital and accounts for 50% hospital readmission within 6 months. In the United States of America, annual hospital admissions for heart failure have increased 90% over a period of 10 years. The disease affects over 5 million Americans with 550,000 new cases every year. The annual hospital and nursing cost of heart failure in the United States is approximately 15 billion U.S dollars[17].

In Sub-Saharan Africa, heart failure accounts for 25% of all deaths equalling to deaths from all infectious and parasitic diseases. By 2020, heart failure deaths are expected to exceed deaths from infectious diseases according to World Health Organization report in 2000[18]

2.0.3 AETIOLOGY

Many clinical studies have been conducted to determine the aetiology of heart failure in different population settings. With the epidemiologic transition phenomenon[19], there has been an increased incidence of cardiovascular disease in West Africa, which is already dominated by infectious diseases[20,21]. This has posed the problem of "double-burden" of disease in the West African Sub-region.

Owusu in 2007, reported the main causes of heart failure in Kumasi, Ghana, as hypertension (42.5%; n=71), rheumatic heart disease (21.6%; n=36) and cardiomyopathy (17.4%;n=29). Other conditions he reported were electrocardiographic left ventricular hypertrophy (66.5%) and anaemia (47.0%)[3]

Amoah and Kallen in 2000 found the main causes of heart failure in Accra, Ghana, to be hypertension, rheumatic heart disease and cardiomyopathy[22].

Ladipo et al, in 1997, studying the pattern of heart disease in Nigeria, found

hypertension, peripatum cardiac disease, dilated cardiomyopathy and rheumatic heart disase as the major causes of heart failure[23].

Antony (1980) studied 315 cases of heart failure admitted to the General Hospital at Katsina in the Northern Savanna region of Nigeria. He found that cardiomyopathy was commonest cause (47%) of heart failure, peripatum heart failure formed the commonest cause of heart failure among female patients whiles anaemia and hypertension caused 13% and 12% of heart failure respectively[24].

Recent studies in West Africa provide important revelation in both epidemiology and clinical outcomes of heart failure. The studies revealed that the survival among sub-Saharan Africans with hypertensive heart failure is one year because the management and control of hypertension in the sub-region is poor[25,26,27,28]. Hypertension has been reported to account for up to 30% of hospital admissions for heart failure in West Africa and the prognosis of hypertensive heart failure among Black Africans have also been found to be poor[19].

The review of the ABC of Heart Failure categorizes the following as the causes of heart failure; coronary artery disease i.e. myocardial infarction and ischaemia, hypertension and dilated cardiomyopathy; valvular and congenital heart disease such as mitral valve disease, aortic valve disease and atrial septic defect; arrhythmias i.e. tachycardia, bradycardia and loss of atrial transport i.e. atrial fibrillation; alcohol and drugs i.e. cardiac depressant drugs like blockers and calcium antagonists[29].

High output heart failure is due to anaemia, thyrotoxicosis and pericardial disease i.e. constrictive pericarditis and pericardial infusion. Primary right heart failure is due to

pulmonary hypertension and tricuspid incompetence[29].

Various risk factors for heart failure have been described and clinical factors that are strongly and consistently associated with heart failure include; age, electrocardiographic left ventricular hypertrophy, overweight or obesity and diabetes mellitus.
Excessive alcohol consumption, cigarette smoking, dyslipidaemia, renal insufficiency, physical inactivity, low socioeconomic status, and increased heart rate are clinical factors less consistent with heart failure[30,31].

2.0.4 TYPES OF HEART FAILURE

Heart failure is usually described by the site of failure; Left-sided or Left Ventricular Heart Failure, Right-sided or Right Ventricular Heart Failure and Biventricular Heart Failure when both sides of the heart are affected. It can also be described as Systolic Heart Failure or Diastolic Heart Failure. These descriptions represent different clinical aspects of heart failure and not distinct diseases[32]. Left Ventricular Heart Failure primarily produces pulmonary signs and symptoms whiles Right Ventricular Heart Failure primarily produces systemic signs and symptoms[33].

2.0.5 SIGNS AND SYMPTOMS

A number of symptoms are associated with heart failure, but none is specific for the condition. Perhaps the best known symptom is dyspnoea. Clinical signs and symptoms of Left Ventricular Heart Failure are due to decreased left ventricular output, which results in fluid accumulation in the lungs. Symptoms include fatigue, dyspnoea, arthopnoea, crackles, wheezing, hypoxia, respiratory acidosis, nocturnal cough, cyanosis, palpitation, arrhythmias, elevated blood pressure, pulsus alternans, cool extremities at rest and in the advanced stage, confusion and memory

impairment[32]

Right-sided heart failure is often caused by disorders that increase vascular resistance such as pulmonary embolism or stenosis or hypertension. Clinical signs include dependant peripheral oedema, hepatomegaly, splenomegaly, jugular vein distinction, ascites, weight gain, arrhythmias, hepatojugular reflux, nausea, vomiting, anorexia, dizziness, syncope and weakness[32,33].

Complications of heart failure typically include pulmonary oedema, venostasis with a predisposition to thromboembolism associated primarily with prolonged bed rest, cerebral insufficiency and renal insufficiency with severe electrolyte inbalance[32,33]

2.0.6 DIAGNOSIS

Despite the high prevalence of acutely decompensated heart failure, diagnostic accuracy is 75% at best in the emergency unit and in clinical trials, and is as low as 50% in other settings. This is because signs and symptoms have a broad differential diagnosis; chest X-rays have limited utility, and 20% to 40% of heart failure patients have normal systolic function[17].

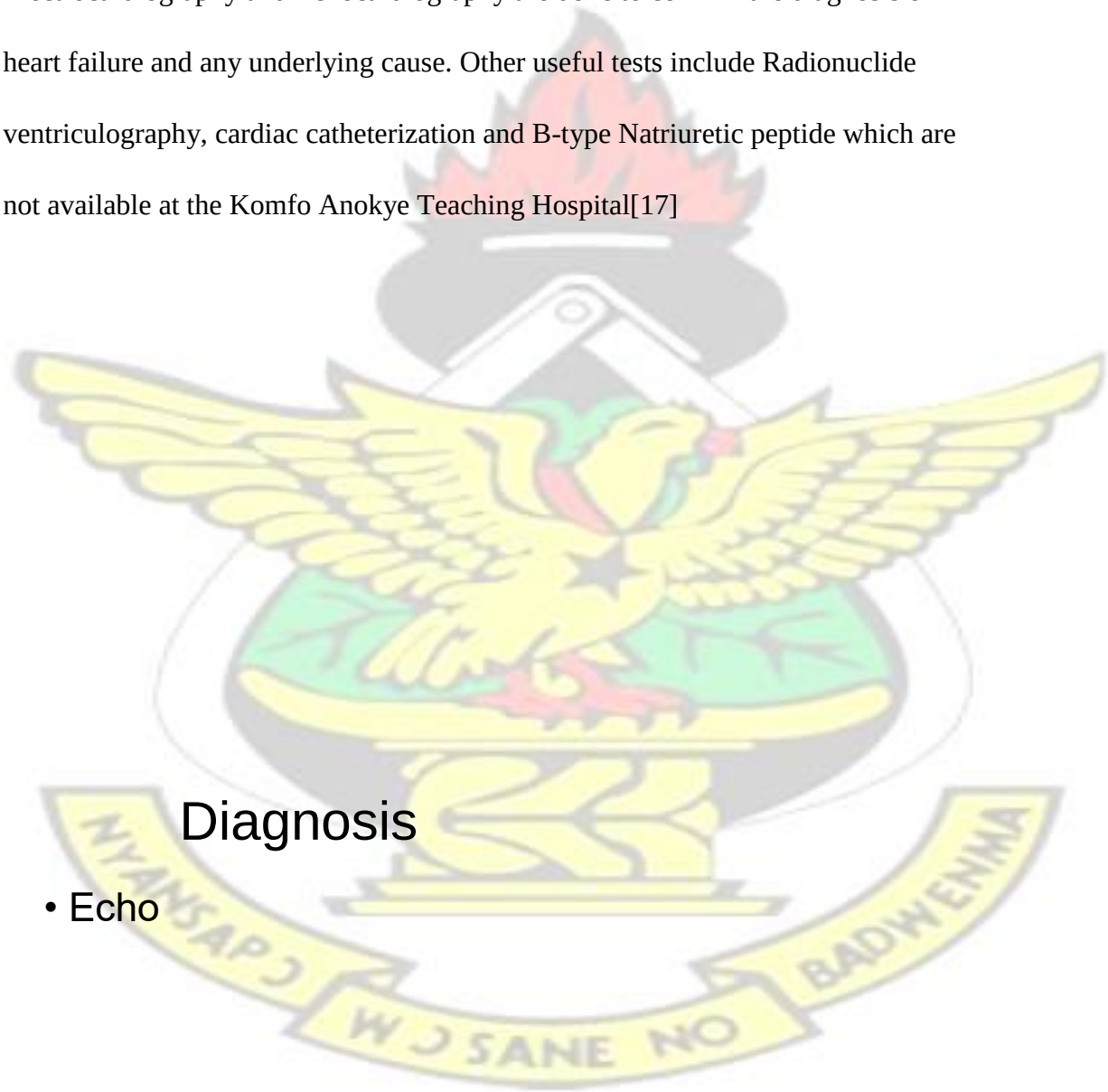
Physical examinations include general assessment to look for dyspnoea at rest, pedal oedema or generalized swelling, cyanosis, fever and pallor of mucous membrane. The pulse rate, rhythm volume and character are noted. Jugular venous pressure, the blood pressure, the apex beat, the heart sounds S1, S2, S3 and S4 and murmurs are also examined. The chest is auscultated for crackles and the presence of hepatomegaly and ascites are also investigated.[10,3].

Routine blood tests to check for anaemia, cholesterol, blood sugar, kidney, liver and

thyroid function tests are done to aid diagnosis[10].

Chest X-rays are obtained from patients to examine for increased cardiac size as judged by a cardiothoracic ratio and the presence of pulmonary upper lobe blood diversion and/or alveolar oedema and/or pleural effusion.

Electrocardiography and Echocardiography are done to confirm the diagnosis of heart failure and any underlying cause. Other useful tests include Radionuclide ventriculography, cardiac catheterization and B-type Natriuretic peptide which are not available at the Komfo Anokye Teaching Hospital[17]



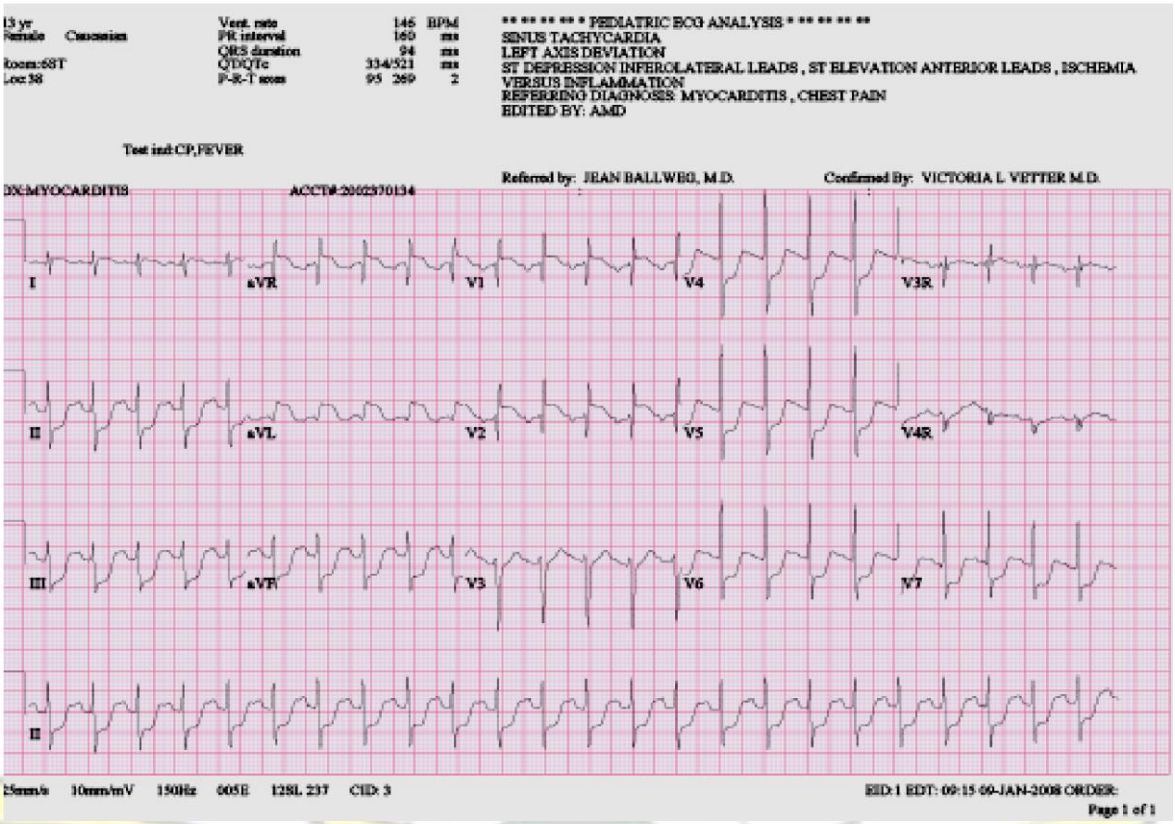


Figure 2.3: **ECG of a failing heart (courtesy: Amanfo, cardiologist, children's hospital in Philadelphia, U.S.A). This is a method of recording the electrical currents that pass through the heart muscle during each heartbeat to detect abnormal heart rhythm.**

Diagnosis

- CXR

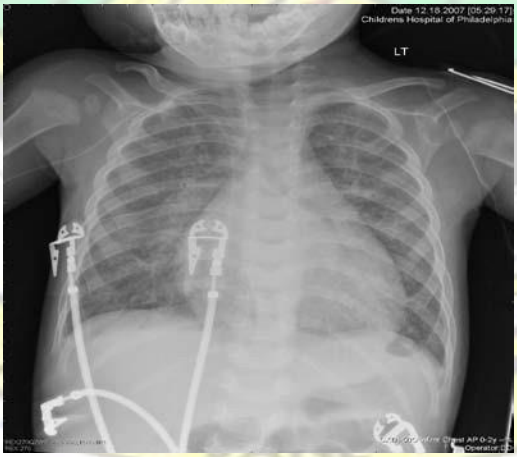


Figure 2.4: Chest x-ray of a heart failure patient (courtesy: Amanfo, cardiologist, children's hospital in Philadelphia, U.S.A). Chest x-ray determines the size of the heart whether enlarged or otherwise.

2.0.7 GOALS OF THERAPY

The goals of therapy in the treatment of heart failure are to relieve patients of signs and symptoms of the disease, to prevent worsening of the disease leading to hospitalization, to improve the quality of life of the patient and to increase survival i.e. reduce mortality. Symptoms improve within a few weeks to a few months of starting therapy[34].

2.1 THERAPY

There are two types of treatments of heart failure i.e. Pharmacological and Non-Pharmacological interventions.

2.1.1 NON-PHARMACOLOGICAL THERAPY

This includes patient education, counselling on life style modification and regular self-monitoring. Life-style modification involves patients quitting smoking, limiting alcohol-intake and water restriction i.e.2L/day, eating of diet low in calories, saturated fat and limiting salt intake i.e. 2gm/day. Counselling involves admonitioning patients to quit smoking, to undergo routine blood pressure, cholesterol and blood sugar checks.

Self-monitoring will involve patients weighing themselves daily to watch for fluid build-up and starting mild to moderate aerobic exercise programme approved by the physician[2].

2.1.2 PHARMACOLOGICAL THERAPY

Several landmark randomized clinical trials over the past two decades have given an insight into the pharmacotherapeutic management of heart failure[4,35].

Based on data from these trials, the updated treatment guidelines include broader, stronger recommendations for Beta-blocker therapy and strong recommendations for

angiotensin II receptor blockers[4] The aldosterone antagonist, spironolactone is also included in the guidelines. A meta-analysis of randomized trials also support the role of diuretics in the management of heart failure[39]

2.1.2.1 DIURETICS

Strong clinical consensus[36,37,38,39,40] indicates that diuretics should be used to treat volume overload. A meta-analysis of several small trials also suggests that the use of diuretics reduces the risk for death and worsening heart failure. In the hospital, frusemide is the loop diuretic commonly used to treat heart failure patients. However, the use of non-potassium-sparing diuretics was independently associated with worse outcomes, possibly due to electrolyte depletion[40]. Diuretics should not be used alone to manage heart failure[36-40]. The dose should be carefully tailored to the individual patient to control fluid retention but avoid hypotension and renal failure.

2.1.2.2 ANGIOTENSIN-CONVERTING ENZYME (ACE) INHIBITORS

Yan *et al* in 2005 reported that overwhelming evidence supports the use of ACE inhibitors in treating and preventing heart failure[35]. The ACE inhibitors have long been recognized as the cornerstone in heart failure management and as a result, these agents have typically been started before any other drugs and Uddin *et al* strongly recommend that clinicians make aggressive attempts to prescribe ACE inhibitors for all patients with heart failure. An initial low dose of lisinopril (2.5mg) daily has routinely been prescribed to patients in the hospital titrating to a high dose (5mg) once daily till a target dose of 20-35mg once daily has been achieved. In case of ACE inhibitors intolerance i.e. angioedema and severe cough, angiotensin II receptor blockers e.g. candesartan 4 or 8mg once daily has been prescribed titrating to the

target dose of 32mg once daily[4]

2.1.2.3 β-ADRENERGIC RECEPTOR BLOCKERS

The updated guidelines address extensively the use of β-blockade in heart failure, as the use of β-blockers has been recognized as the most important advancement in heart failure in the past decade. Specifically, the guidelines recommend the routine administration of a β-blocker in patients with mild-to-severe heart failure and left ventricular ejection fraction of 40% or less[12].

2.1.2.4 ALDOSTERONE ANTAGONISTS

Plasma aldosterone levels maybe elevated by 20-fold in patients with heart failure, writes Weber [42]. This effect is due to increased production of adrenal glands which are stimulated by high plasma angiotensin concentrations, and to decreased hepatic aldosterone clearance secondary to hepatic hypoperfusion. Aldosterone is also produced in the heart and blood vessels and may have important paracrine effects. Aldosterone regulates urinary retention of sodium, excretion of potassium and tissue inflammatory response and stimulates cytokine secretion, fibroblast growth and collagen turnover[43]. These phenomena in turn lead to myocardial fibrosis and adverse ventricular modelling. These observation suggest that direct blockade of aldosterone may have a salutory effects in patients with heart failure[44].

Patients should, however, be carefully selected, based not only on New York Heart Association class, but also on the ability to monitor renal function and plasma potassium concentration.

For situations in which this monitoring is anticipated to be not feasible, the risks may outweigh the benefits of aldosterone antagonists. As a measure of quantitative

guidance, aldosterone antagonists are not recommended when serum creatine level is greater than 221μmol/L (or creatine clearance <30ml/min), if serum potassium concentration is greater than 5.0mEq/L or while in the presence of other potassium-sparing diuretics reported Adams et al[45]

Randomized Aldactone Evaluation Study (RALES) in 1999 demonstrated a dramatic reduction in overall mortality (~30%) with the use of spironolactone 25mg/day in patients with moderate-to-severe heart failure. This reduction in mortality rate was not limited to mortality due to progressive heart failure, but included mortality due to sudden cardiac death as well. Based on the result of RALES, the guidelines recommended the use of low-dose spironolactone 25mg/day in patients with severe heart failure despite standard therapy[4] reaching a target dose of 50mg once daily.

2.1.2.5 DIGITALIS GLYCOSIDES

A recent systematic review indicates that therapy with digoxin reduces the incidence of hospitalization and improves the clinical status of symptomatic patients[47]. However, given the lack of mortality benefit, therapy with digoxin is recommended only in patients who continue to experience symptoms while receiving optimal medical treatment, including ACE inhibitors and β-blockers[41,42]. Serum digoxin concentrations do not correlate with clinical efficacy[43]. Routine repeated measurement of serum digoxin levels is therefore not necessary unless nonadherence or toxicity is suspected. The usual maintenance dosage of digoxin is 0.125mg/day; smaller doses that achieve a low serum concentration <0.9mg/ml is recommended[46].

2.1.2.6 ANTITHROMBOTIC AGENTS

Patients with heart failure are at increased risk for stroke, probably because of blood stasis in the hypokinetic ventricle; haemostatic abnormalities; and established risk factors, such as hypertension and atrial fibrillation[49,50]. Warfarin and Antiplatelet Therapy in Heart Failure Trial (WATCH) which recruited 1587 patients with heart failure randomly assigned to receive aspirin, warfarin (target international normalized ratio 2.0 to 3.0) or clopidegrel[50]. Rates of the composite endpoint of death, myocardial infarction, or stroke did not differ among the three groups. However, it was reported that patients receiving warfarin were less likely than those receiving aspirin or clopidegrel to require hospitalization for heart failure, suggesting that inhibition of prostaglandin by aspirin may worsen heart failure[6]. At present, no strong evidence supports the routine use of antiplatelet or anticoagulant therapies in patients who have heart failure of non-ischaemic cause and normal sinus rhythm[5]. If indicated, aspirin should be administed in the lowest effective dose.

2.2 MONITORING

2.2.1 FULL BLOOD COUNT

Patients with heart failure undergoing treatment must have their full blood count investigated before therapy and two weeks after initiation of therapy. Low haemoglobin level can precipitate heart failure i.e.high cardiac output.

2.2.2 SERUM ELECTROLYTES

Patients potassium and sodium levels must be monitored. High serum sodium will cause fluid retention and thus aggravate generalized oedema in heart failure patients. Potassium serum level >5.0 mmol/L is likely to cause significant hyperkalaemia.

Most of the drugs commonly used in heart failure therapy i.e. ACE inhibitors, angiotensin-receptor blockers, spironolactone (and patients with renal dysfunction)

are likely to increase serum potassium levels and therefore must be monitored. Nonsteroidal anti inflammatory drugs have been reported to cause hyperkalaemia in up to 46% of hospitalized patients[49] and must be avoided in heart failure patients.

2.2.3 BLOOD PRESSURE

Yancy in 2004 reported that aggressive treatment strategies for heart failure must begin with prevention, and hypertension represents a major contributing factor. It has been estimated that as many as 90% of persons with heart failure have an antecedent history of hypertension. The American Medical Association at its 107th annual convention in July, 2004 reported the following benefits of lowering blood pressure: a 50% average reduction in the incidence of heart failure; a 35% to 40% average reduction in the incidence of stroke; and a 20% to 25% average reduction in the incidence of myocardial infarction. It, therefore, becomes imperative that all patients who present with heart failure have their blood pressure monitored daily to assess the efficacy of medication and quality of life of patients[17]

2.2.4 RENAL FUNCTION TEST

Shlipak in 2003 reported that approximately one third to one half of patients with heart failure have renal insufficiency which is among the strongest predictors of mortality in patients with heart failure[50] It is, therefore, important to determine the renal function of each heart failure patient. Blood chemistry and creatine are monitored before and during therapy.

2.2.5 LIVER FUNCTION TEST

Since hepatomegaly is a complication of heart failure, it is important to determine the liver function before and during therapy.

2.3 MORBIDITY/MORTALITY

Owusu in 2007 reported that heart failure is a major health problem in the West African-Sub region accounting for 29% of deaths at Komfo Anokye Teaching Hospital, Kumasi, Ghana and was responsible for 10% of deaths in hospitalized adult medical patients at Korle-Bu Teaching Hospital, Accra [3].

It is also reported that the 1-year mortality rate after the diagnosis of heart failure is 20% reports Barclay[2]

Yancy, also reported that 5 million patients in the United States have heart failure, with a 50% hospital readmission rate within 6 months and 50% mortality at 5 years. The mortality rate is as much as 12% at 30 days after hospitalization and 33% at 12 months[17]

2.4 LENGTH OF HOSPITAL STAY

Heart failure is among the leading causes of hospitalization in most developed countries reports Yancy and in the past 10 years, annual hospital admissions for heart failure have increased 90% with 6.5 million hospital days per year and the median hospital length of stay is 6 days in U.S[17].

Kossovsky M.P *et al* reported a mean length of stay of 13.2 days in an observational study conducted in the medical wards of the Geneva University hospitals, Geneva, Switzerland. The length of stay of 13.2 days observed in the study exceeds that reported by Yancy from the U.S.A and is similar to length of stay in hospitals in the Scotland, the Netherlands and Italy[51].

The data suggested that careful reorganisation of the process of care should accompany any attempt to reduce length of stay to avoid detrimental effects on

quality of care.

It is also well noted that length of stay between days 1 and 10 at the Komfo Anokye Teaching Hospital is sufficiently associated with the following criteria; improvement in dyspnoea; improvement in neck vein distinction; improvement in pulmonary auscultation; stable or decreased body weight; and no change in medications during 24 hours before discharge.

CHAPTER THREE: METHODOLOGY

3.0 MATERIALS AND METHODS

3.1 STUDY TYPE

The study was a hospital-based retrospective descriptive carried out at the medicine directorate, Komfo Anokye Teaching Hospital in Kumasi from January 2004 to December 2005 before the implementation of the updated treatment guidelines and January 2006 to December 2007 after the implementation of the treatment guidelines in January 2006.

3.2 SAMPLING METHOD

Heart failure patients aged between 13 and 100 years who were admitted at the medical wards of the hospital were used. Documentation of patients in the nurses' admission and discharge books at the wards was poor and hence only patients who were properly documented within the said periods of the study were used.

3.3 PILOT STUDY

Four house-officer ward-pharmacists were trained on the study and the data collection. The questionnaire was pre-tested using 20 patients each year under review i.e. 2004, 2005, 2006 and 2007. Each house-officer was assigned to a particular year. The actual data collection was carried out after a month of the pilot exercise.

3.4 DATA COLLECTION TOOLS

The updated treatment guidelines for heart failure were implemented in January 2006. Data collection Forms (Appendix 1) were used to collect data on all patients admitted at the medical wards from January 2004 to December 2005 before the implementation of the updated treatment guidelines.

Data collection Forms (Appendix 2) were also used to collect data on all patients

admitted from January 2006 to December 2007, after the implementation of the updated treatment guidelines.

The folder numbers and names of all patients admitted during the periods under review were extracted from the admission and discharge books on the wards. These folders were subsequently retrieved from the Medical Records Department for the data collection.

The information on data collection Forms were grouped into 4 main sections namely;

SECTION A—Patient Details

SECTION B—Heart Failure Treatment on Admission

SECTION C—Monitoring Parameters

SECTION D—Outcome

SECTION A—PATIENT'S DETAILS

It consisted of patient's folder number i.e. patient's identity number found at the back of the folder, age and sex which provided the demographic characteristics of the population i.e. sex and age distribution of the patients were found in the history sheet of the patient in the folder. These form questions 1, 2, 3 and 4 i.e. name of the patient, identity or folder number, sex and age.

Question numbers 5 and 6 consisted of the date of admission and the date of discharge or death which gave the length of hospital stay were found in the history sheet of the patient.

Question numbers 7 and 8 indicated the patient's systolic and diastolic blood pressure values which determined stable or unstable condition of the patients on admission.

Question number 9 indicated the consciousness or unconsciousness of the patient on admission all found in the clerking notes.

Question numbers 10 and 11 indicated the type and cause of the patient's heart failure.

Questions 1-11 were used to achieve objective number one.

SECTION B:-HEART FAILURE TREATMENT ON ADMISSION

Question number 12 indicated the medications the patients received whiles on admission as found in the treatment sheet in the folder. The drug was labelled YES when prescribed and NO when not prescribed. Question 12 was used to achieve objective number two.

SECTION C:-MONITORING

Question numbers 13, 14, 15, and 16 consisted of the various biochemical investigations carried out i.e. renal function, liver function, full blood count and the investigation parameters such as chest X-ray, electrocardiography (ECG), and echocardiography. These were obtained from the laboratory filings or medical notes in the folder.

When it was done, it was labelled YES and NO when it was not done.

SECTION D: - OUTCOME

Question numbers 17 and 18 denoted the fate of the patient i.e. discharged or died. These were obtained from the discharge and admission notes in the patient's folder. These were used to achieve objective 3.

3.5 DATA PROCESSING AND ANALYSIS

The data collected using the Data Collection Forms were entered separately into

STATA Version 7.0 statistical software for analysis. Descriptive analysis of baseline parameters was provided and numbers and percentages were calculated.

3.6 LIMITATION OF STUDY

As a result of the retrospective nature of the study, some vital information on some patients could not be obtained due to poor documentations in the admission and discharge books at the wards. Some names and folder numbers retrieved from the admission and discharge books could not be traced from the medical records department. These had the potential of affecting the observed results.

CHAPTER FOUR: RESULTS

411 patients were involved in the study comprising 233 males and 178 females who were admitted at the medical wards of the medicine directorate from January 2004 to December 2007.

207 patients were involved in the study before the implementation of updated treatment guidelines in 2004 and 2005 and 204 patients were involved in the study after the implementation on the updated treatment guidelines in 2006 and 2007.

4.1 DEMOGRAPHIC CHARACTERISTICS

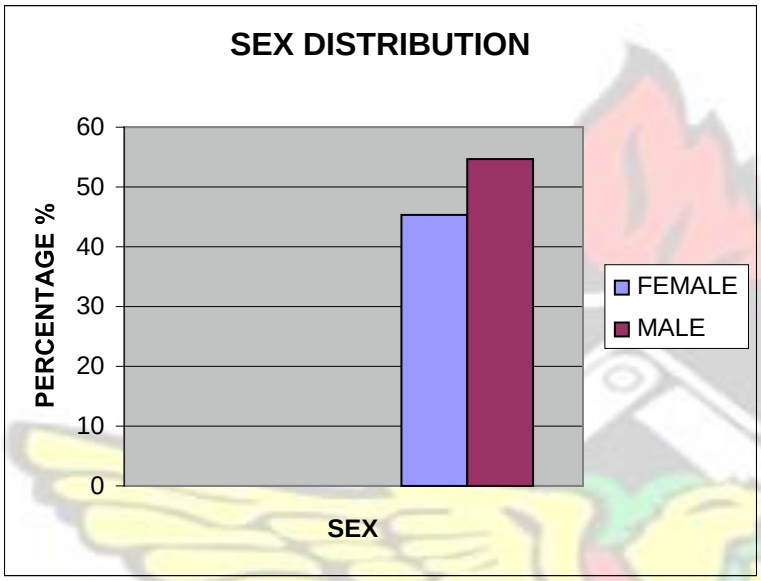


Figure 4.5: male and female percentage frequency of heart failure patients.

-Age distribution of heart failure patients

The ages of the patients admitted with heart failure during the period under review ranged from 13 to 100 years with the range 68-76 years forming 18.02%.

The median age of the patients was 57.6 years with a standard deviation (SD) of 18.12. Males appeared to develop heart failure at a younger age with the minimum age of 13 years. The minimum age of females was 17 years.
The difference in age between the males and the females was not statistically significant (p=0.20).

The age distribution is shown in figure 4.6 below

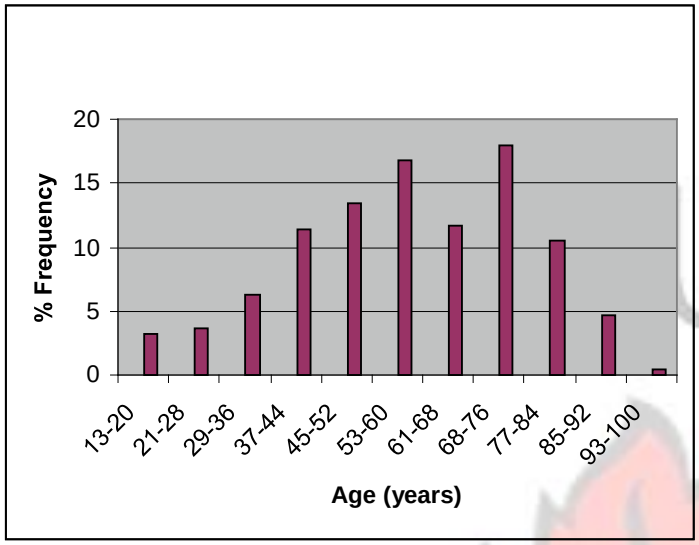


Figure 4.6: the percentage frequency of ages of heart failure patients

-Causes of heart failure

The main causes of heart failure were hypertension (60.3% n=248), dilated cardiomyopathy (23.4% n=96), valvular disease (4.6% n=19). Other conditions seen in patients were anaemia (0.49% n=2) and cardiogenic shock (1.9% n=8). Patients diagnosed of heart failure of unknown or queried cause represented 8.52% as shown in table 4.1 and figure 4.7 below

Type of heart failure	Frequency	%Frequency	% Cum
Biventricular failure of unknown cause	35	8.52	8.52
Biventricular failure due to cardiogenic shock	7	1.70	10.22
Biventricular failure due to cardiomyopathy	87	21.17	31.39

Biventricular failure due to hypertension	188	45.74	77.13
Biventricular failure due to valve disease	7	1.70	78.83
Biventricular failure due to anaemia	2	0.49	79.32
Biventricular failure due to mitrial incompetence	12	2.92	82.24
Left ventricular failure of unknown cause	2	0.49	82.73
Left ventricular failure due to cardiomyopathy	9	2.19	84.92
Left ventricular failure due to hypertension	60	14.60	99.52
Left ventricular failure due to cardiogenic shock	1	0.24	99.76
Right ventricular failure of unknown cause	1	0.24	100.00
TOTAL	411	100.00	

Table 4.1: causes of heart failure

-Condition of patients on admission

Conditions of the patients on admission described as conscious/stable constituted 70.8%; conscious/unstable 27.7% and unconscious/unstable 1.5%. Consciousness was assessed as patients being alert and oriented in time, person and place. Stability

was based on patient's systolic pressure ≥85mmHg and heart rate of ≥68beats per minute denoting adequate cardiac output. This is shown in figure 4.8 below. Most of the patients were in the New York Heart Failure Association Classes I and II forming about 71% whiles those with severe heart failure i.e. New York Heart Failure Association III and 1V forming the 28% and 1% respectively.

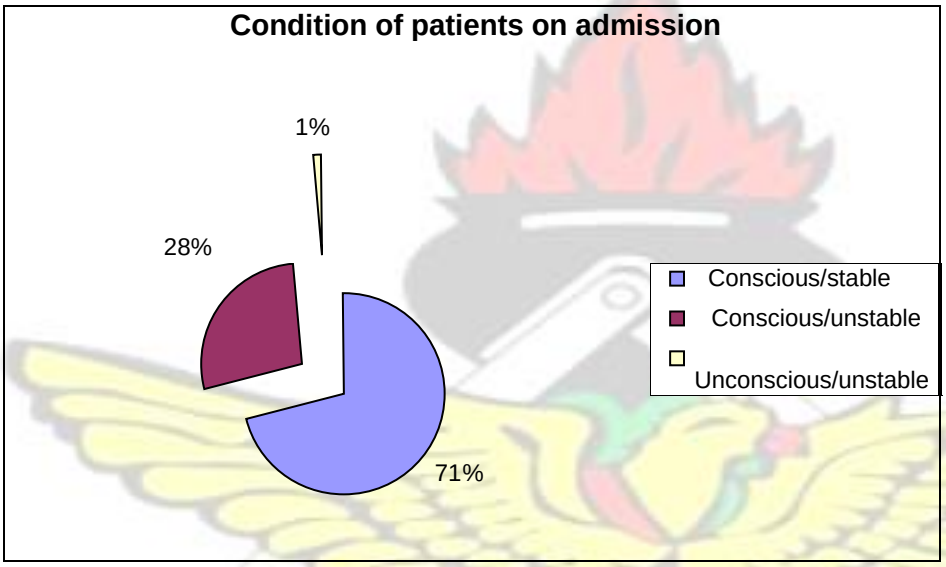


Figure 4.8: severity of conditions of patients on admission

-Pharmacological Treatment of Heart Failure Patients

Almost all the patients received the loop diuretic, frusemide (97.8% n=402).The ACE-inhibitor, lisinopril was administered to 49.6% (n=204) patients, the aldosterone-antagonist, spironolactone administered to 45.8% of the patients (n=188) and the β-blocker, carvedilol administered to 22.4% (n=92) of the patients. This is shown in figure 4.9 below

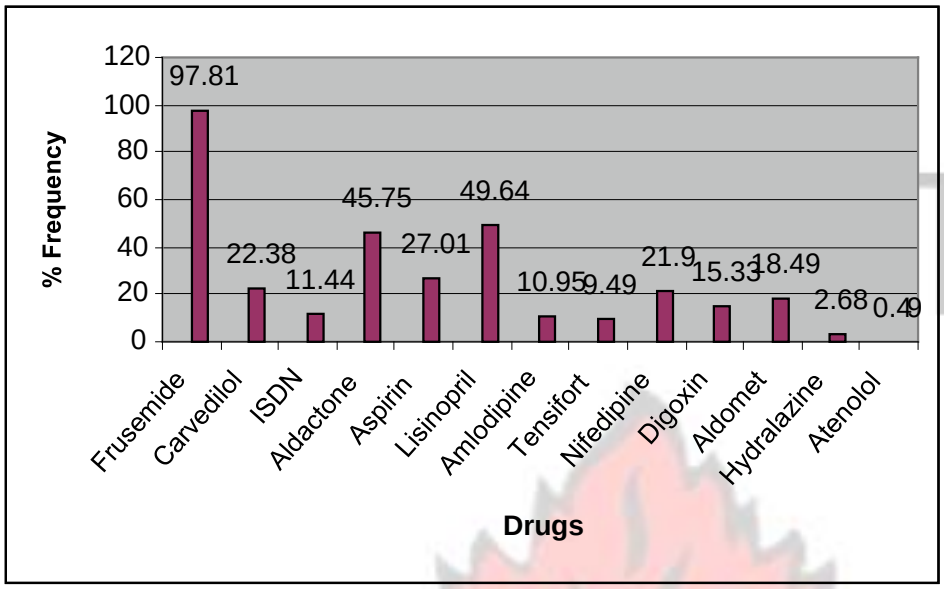


Figure 4.9: the overall frequency of drugs administration

-Drugs administration before and after implementation of updated treatment guidelines constituting the 4-year period as shown in figure 4.10 below

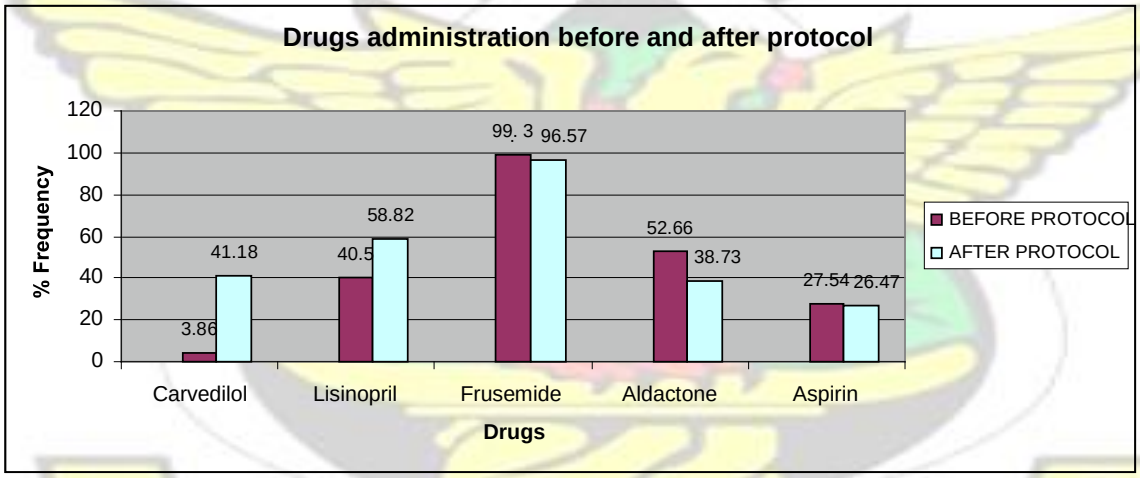


Figure 4.10: Drugs administration before and after the implementation of updated treatment guidelines.

-IMPACT ON MORTALITY

The implementation of the updated treatment guidelines with the β-blocker,

carvedilol included led to a drastic reduction in overall mortality from 35.27% before

the implementation of updated guidelines to 17.16% after the implementation of updated guidelines representing an 18.11% decrease in mortality. This is shown in figure 4.11 below

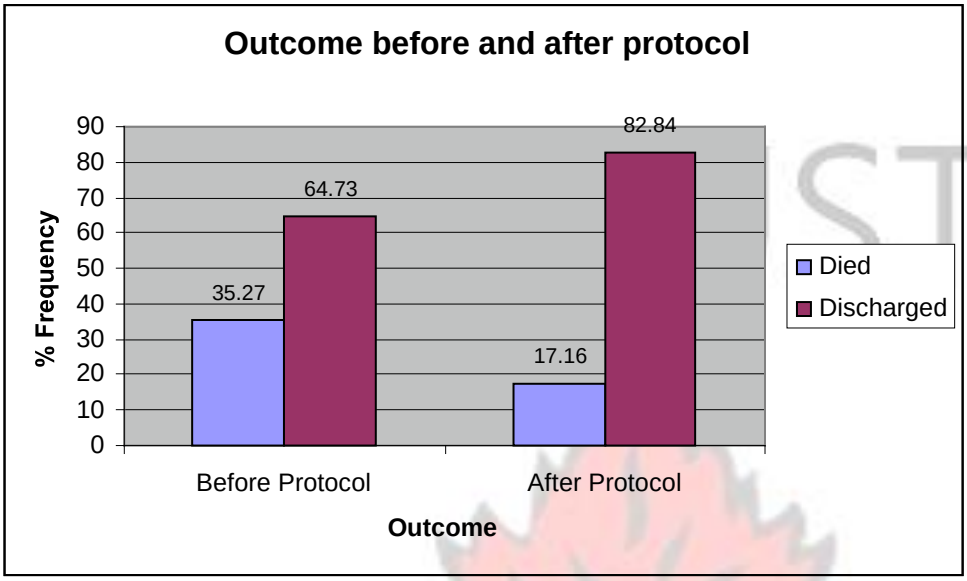


Figure 4.11: Outcome before and after the implementation of updated treatment guidelines.

-IMPACT ON LENGTH OF HOSPITAL STAY

The median length of hospital stay of the patients was 5.1 days in 2004 and 2005 before the implementation of updated treatment guidelines and 3.2 days after the implementation of the treatment guidelines representing 62.7% decrease.

Days	Frequency	% Frequency	% cum
1	9	4.35	4.35
2	36	17.39	21.74
3	26	12.56	34.30
4	35	16.91	51.21
5	23	11.11	62.32
6	21	10.14	72.46
7	13	6.28	78.74
8	12	5.80	84.54
9	7	3.38	87.92
10	14	6.76	94.69
11	6	2.90	97.58
12	2	0.97	98.55
13	1	0.48	99.03
14	2	0.97	100.00
TOTAL	207	100.00	

Table 4.2: Length of hospital stay before implementation of updated treatment guidelines.

Length of stay after implementation of updated treatment guidelines

Days	Frequency	% Frequency	% Cum
1	4	1.96	1.96
2	58	28.43	30.39
3	67	32.84	63.24
4	50	24.51	87.75
5	21	10.29	98.04
6	3	1.47	99.51
7	1	0.49	100.00
Total	204	100.00	

Table 4.3: Length of hospital stay after the implementation of updated treatment guidelines.

	Before implementation	After implementation
PARAMETER	**NUMBER OF PATIENTS**	**NUMBER OF PATIENTS**
HYPERTENSIVE EMERGENCY MONITORING CHART (B.P)	**402**	**411**
DIABETIC EMERGENCY MONITORING CHART (RBS)	**303**	**360**
DIABETIC EMERGENCY MONITORING CHART (FBS)	**310**	**411**
ELECTROCARDIOGRAPHY (ECG)	**391**	**400**
RENAL FUNCTION TESTS	**401**	**403**
LIVER FUNCTION TESTS	**399**	**411**
ECHOCARDIOGRAPHY (ECHO)	**206**	**381**
TEMPERATURE CHART	**411**	**411**
DRUG ADMINISTRATION CHART	**396**	**402**

Table 4.4: Monitoring Parameters of the patients documented before and after the implementation of the updated treatment guidelines.

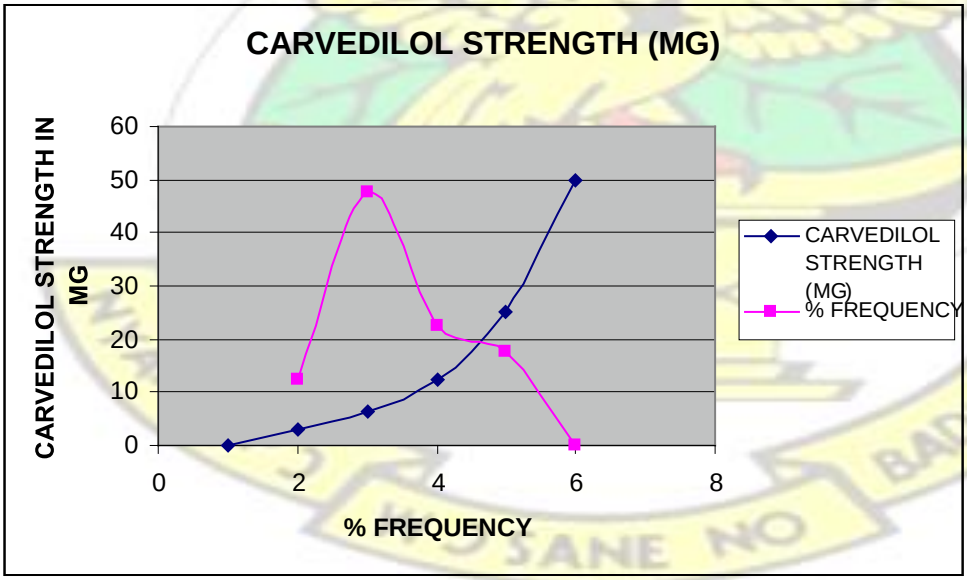


Figure 4.14: Strength of carvedilol used in the treatment of heart failure patients.

CHAPTER FIVE: DISCUSSION

The ages of the patients admitted with heart failure during the period under review ranged from 13 to 100 years with the range 68-76 years forming 18%.

The median age of the patients was 57.6 years with a standard deviation (SD) of 18.12. This correlates with median age of 56.15 (18.5) reported by Owusu in his study of the causes of heart failure in Kumasi[3]. A similar mean age of 53 (12.1) years was reported by Isezuo *et al* among Gambians and Nigerians with heart failure[26]. However, Amoah and Kallen reported a low mean age of 42.3 (0.9) years in their study at the National Cardiothoracic Centre in Accra[22]. This may be due to the many congenital heart cases reported at the centre.

The mean age of the patients in this study is also lower than the 74 years and the 70(10.8) years reported in the general European population[24] and in the Framingham Heart Study[25] respectively. This could be explained by the differences in the aetiology of heart failure in the developed and developing worlds. In the developed world, the main cause of heart failure is ischaemic heart disease whiles in the developing world rheumatic heart diseases are the common cause. Many people with ischaemic heart diseases are surviving because of advances in treatment in the developed world. Again in the developed world, the proportion of the population that is elderly is increasing[24,25] resulting in high life expectancy in the developed than in the developing world.

Males constituted 56.7% and females made up 43.3% of heart failure patients in the study representing a ratio of 1.3:1.0. Amoah and Kallen also reported a similar ratio

of 1.2:1.0 of male to female affected with the disease at the National Cardiothoracic Centre in Accra[22]. This confirms available data[17, 18] that heart failure affects more males than females. This is probably due to the fact that the incidence of hypertension is more prevalent in males than females and hypertension is a major cause of heart failure in the sub-region [27].

The main causes of heart failure in this study were hypertension (60.3%), valvular disease (4.6%) and dilated cardiomyopathy (23.4%). Data available shows that the main causes of heart failure in Africa are hypertension, rheumatic heart disease, cardiomyopathy and pericardial disease[22,23,24]. Hypertension was seen as a common cause of heart failure in this study and was reported as such by Owusu, in Kumasi[3] and Amoah and Kallen in Accra[22].

Given the importance of hypertension, it is alarming that the awareness, treatment, and control rates of hypertension in Africa are as low as 20%, 10% and 1%, respectively [26, 27]. Owusu reported that the detection and control of hypertension remains a challenge even in developed countries, with as many as 70% of hypertensive patients with uncontrolled high blood pressure[3].

23.4% of the patients in this study had dilated cardiomyopathy as the cause of heart failure. This correlates with several studies extensively conducted in Africa[3,22,23,24, 85]. It accounted for 25.2% of the cases of heart failure seen in Nairobi[85], 16.8% of the cases seen in Accra[86], and 47% of the cases seen in Katsina in northern savanna region of Nigeria[23] and Owusu reported 15.2% of cases in Kumasi[3]. Likely causes of dilated cardiomyopathy are viral myocarditis,

chronic excessive alcohol intake and malnutrition[29].

46% of the patients in this study had valvular disease as cause of heart failure. In Accra, Pobee reported that valvular heart disease presumably rheumatic heart disease, occurred in 20.4% of all cardiovascular diseases[86] and was responsible for 11% of heart failure deaths. Owusu reported 17.4% cases of valvular disease as a cause of heart failure in Kumasi[3]. Mitral regurgitation and aortic stenosis are common causes of heart failure, secondary to valvular disease. Mitral regurgitation leads to volume overload in contrast with aortic stenosis, which leads to pressure overload[29] The poor socioeconomic conditions, overcrowding and inadequate medical services in Africa are said to favour the high incidence rates of rheumatic heart disease. Poor housing and overcrowding promote the rapid spread of group A streptococcal infection. Inadequate medical resources and expertise of health-care providers result in inadequate diagnosis and treatment of streptococcal pharyngitis which eventually destroys the valves necessitating high cost valve replacement surgery beyond the means of the poor[86].

TREATMENT

Almost all the patients (97.8%) in this study received frusemide, a loop diuretic in accordance with the updated treatment guidelines since all the patients were symptomatic. The frusemide was to manage fluid overload and to relieve the patients of symptoms like breathlessness. Strong clinical consensus[36-40] indicates that diuretics should be used to treat volume overload. A meta-analysis of several small trials also suggests that the use of diuretics reduce the risk of death and worsening

heart failure[39]. However, diuretics are not supposed to be used alone to manage heart failure. The dose should be carefully tailored to the individual patient to control fluid retention but avoid hypotension and renal failure. All the patients had fluid overload either as pulmonary oedema, peripheral oedema or both.

It is not clear why only 49.64% (n=204) of the patients in this study received the ACE-inhibitor, lisinopril, since 70.8% of the patients on admission were in stable condition. At least all these patients would have qualified to be administered lisinopril. The ACE-inhibitors have been described as the cornerstone in heart failure treatment. The updated treatment guidelines state that all patients with symptomatic heart failure due to systolic left ventricular dysfunction should receive an ACE-inhibitor[69]. Overwhelming evidence supports the use of ACE-inhibitors in treating and preventing heart failure[35]. A meta-analysis of 32 trials completed before 1995 that involved 7105 patients with heart failure concluded that ACE-inhibitors reduced the total mortality rate by 23% and the combined end point of death from or hospitalization for heart failure by 35%[35]. Furthermore, long-term follow-up of these studies suggests that these benefits are sustained over many years.

Despite their proven efficiency, there was no reason for its underprescription or usage as shown in this study. Again, it was observed that low doses of the drug were mostly used. The assessment of treatment with lisinopril and survival randomized trial showed that high doses of lisinopril significantly reduced the risk of death from or hospitalization for heart failure[70].

Cough, a well known side-effect of ACE-inhibitors may also be caused by uncontrolled heart failure. This may require intensification of therapy rather than

withdrawal.

Inspite of the implementation of the updated treatment guidelines which state categorically that all patients admitted for heart failure should receive a diuretic, an ACE-inhibitor, and a β-blocker, only 22.38% of the patients in this study received the β-blocker, carvedilol. Owusu, also found that only 17% of heart failure patients in his study received the β-blocker, carvedilol[3].

Despite its underprescription, its inclusion in the standard therapy after the implementation of the updated treatment guidelines in 2006 reduced mortality rate by 18.11% in this study. This correlates with the outcome of many multiple clinical trials that have evaluated the effects of β-blockers in more than 10,000 patients with heart failure[35]. Meta-analyses of early small studies showed that the use of β-blockers reduced the all-cause mortality rate by approximately 32%, regardless of the cause of heart failure[72-74]. A carvedilol-post-infarction survival control in left ventricular dysfunction, which included patients without clinical heart failure who had had myocardial infarction showed that carvedilol reduced the all-cause mortality rate by 23%[75]. Therefore, the updated guidelines recommend that both symptomatic and asymptomatic patients with left ventricular dysfunction must be treated with β-blockers and ACE-inhibitors[64]. Together with ACE-Inhibitors, β-blockers result in reversing cardiac remodeling, improve morbidity, reduce hospitalization and mortality. Because, carvedilol is predominately cleared by the liver the agent may be safer in patients with renal insufficiency

reported by Michael[50]. Atenolol, a first generation β-blocker was unfortunately used in 0.49% of heart failure patients in this study. Although, atenolol is a β-blocker, it is not recommended in heart failure treatment. Its use in cardiology is restricted to atrial fibrillation following acute myocardial infarction to slow rapid ventricular response.

In addition, no recent clinical studies have established its effectiveness in heart failure. Other β-blockers that are recommended in heart failure are bisoprolol and metoprolol. Both bisoprolol and metoprolol are β-selective adrenergic blocking agents without additional α-vasodilating properties. The COMET trial showed that carvedilol was substantially more effective than the short-acting metoprolol and bisoprolol in reducing the overall mortality rate due to the effect of additional α-blockade and non-selective β-blockade. Bisoprolol and metoprolol are the preferred cardio-selective β-blockers for heart failure patients with asthma[87, 88]

The aldosterone-antagonist, spironolactone was given to 45.75% of heart failure patients in this study. Surprisingly most of the patients received spironolactone in higher doses between 50-100mg daily. The Randomized Aldactone Evaluation Study (RALES) showed that low dose spironolactone i.e. 25mg daily or on alternate days increased survival, reduced hospital admissions and improved New York Heart Association (NYHA) class when added to standard therapy of a loop diuretic, an ACE-inhibitor and in a minority of cases, a β-blocker in patients with severe (NYHA class III or IV) heart failure. It is the recommendation of this study that all patients with symptomatically moderately severe or severe (NYHA class III

or IV) heart failure should receive spironolactone as a second line therapy after an ACE-inhibitor and a β-blocker. There is no evidence of benefit in patients with mild heart failure[76].

The safety and efficacy of spironolactone used with an ACE-inhibitor and an angiotensin receptor blocker as well as β-blocker is uncertain and the use of all 3 inhibitors of the renin-angiotensin-aldosterone system together is not recommended[76].

It should also be used with caution or avoided in patients with serum plasma potassium levels >5.0mmole/L and a serum creatine level>221mmole/L.

11.4% of the patients received isosorbide dinitrate whiles 2.7% received hydralazine separately. Shlipak reported that hydralazine-dinitrate combination improves survival in patients who cannot tolerate an ACE-inhibitor or Angiotensin receptor blocker because of renal impairment[50]. This is supported by the Veteran Administration Cooperative Vasodilator-Heart Failure Trial which showed that the hydralazine and isosorbide dinitrate combination improved survival and left ventricular ejection fraction. Daily doses of hydralazine, up to 100mg or intravenous infusion up to 300 micrograms in combination with isosorbide dinitrate 40-160mg up to a maximum of 240mg in the presence of cardiac glycosides and diuretics have been found to reduce mortality of patients with chronic heart failure[77,78]. No patient received this combination in this study. There is no evidence of proven benefit when either nitrates or hydralazine are used alone but nitrates are often prescribed without hydralazine as showed in this study.

Digoxin was prescribed to 15.3% of patients in this study, a majority of whom had

atrial fibrillation and unrecordable blood pressures. Digitalis is the oldest therapy for heart failure, and also remains one of the most commonly prescribed medicines to treat heart failure mainly due to its positive inotropic action through inhibition of the sodium-potassium-adenosine triphosphatase. Short-term studies showed that therapy with digitalis alleviated symptoms of heart failure and improved exercise tolerance and left ventricular ejection fraction[79]. Overall, digoxin did not affect survival but led to a 28% reduction in heart failure hospitalization[79]

Therefore, in heart failure, digitalis probably acts primarily as a neurohormonal moderator rather than a weak positive inotropic agent[80]. It is, therefore, recommended that the use of digoxin in heart failure must not be restricted to atrial fibrillation but should be considered for symptomatic treatment of heart failure caused by left ventricular dysfunction.

As many as 22% of the patients in this study received the calcium-channel blocker, nifedipine. The reason for this was to treat arterial hypertension in heart failure. However, the antihypertensive and lipid-lowering treatment to prevent heart attack trial (ALLHAT)[81] and a recent meta-analyses[82] suggested that hypertensive patients treated with calcium-channel blockers are at risk for heart failure. The updated guidelines recommend the use of the newer calcium antagonists i.e. felodipine and amlodipine to treat arterial hypertension or angina in heart failure. These newer calcium-channel blockers do not have effect on survival. However, maximizing the dose of β-blockers and ACE-inhibitors, which extend survival in heart failure, is thus preferred over the addition of calcium-channel blockers to control hypertension and angina[81,82].

Tensifort, a combination therapy, made up of lisinopril 5mg and amlodipine 5mg, was administered to 9.5% of the patients in this study. Although the drug components in tensifort are all indicated for treatment in heart failure, the updated treatment guidelines do not recommend such practice as upwards or downwards titration of the individual drugs to suit the individual heart failure patient is unachievable. It is, therefore, the recommendation of this study that such combinations are avoided in heart failure therapy.

Low dose aspirin was given to given to 27% of the patients especially in the NYHA Class II and IV. Indications for its frequent use are not clear. A meta-analysis documented a trend toward reduced benefit of treatment with an ACE-inhibitor when it is combined with aspirin therapy[83]

At present, no strong evidence supports the random use of antiplatelet or anticoagulant therapies in patients who have heart failure of non-ischaemic cause and normal sinus rhythm[84].

The patients' mean length of hospital stay was 5.1 days between 2004 and 2005 before the implementation of updated treatment guidelines and 3.2 days between 2006 and 2007 after the implementation of the treatment guidelines representing 62.7% decrease. The overall mean length of hospital stay was 4.2 days with a standard deviation of 2.43.

Kossovsky *et al* reported a mean length of stay of 13.2 days in an observational study conducted in the medical wards of the Geneva University hospitals, Geneva, Switzerland[51]. The length of stay of 13.2 days observed in the study exceeds the mean length of stay of 6 days reported by Yancy[17] in the United States of

America but is similar to that of other countries such as the Scotland, the Netherlands and Italy[51].

Several studies have shown no detrimental effects of shorter length of hospital stay on various patient outcomes such as hospital readmission or death in the weeks following discharge and that in principle, tests and treatments can be organised effectively to allow a short length of hospital stay without compromising quality of care[3,21,22]. 70.8% of the patients on admission were in stable condition and therefore it was possible, after the implementation of the updated treatment guidelines, to achieve shorter length of hospital stay to reduce hospital cost and congestion at the wards.

The inclusion the β-blocker, carvedilol led to a reduction in overall mortality from 35.27% between 2004 and 2005 before the implementation of updated guidelines to 17.16% in 2006 and 2007 after the implementation of updated guidelines representing an 18.11% decrease in mortality. This correlates with the result of several clinical trials[12,13,14,15,16] which concluded that the inclusion of βblocker, carvedilol, to standard therapy of a loop diuretic and an ACE-inhibitor increased left ventricular ejection fraction in both ischaemic and non-ischaemic cardiomyopathies. Gilbert *et al* in a Multicentre Oral Carvedilol Heart Failure Assessment (MOCHA) trial designed to establish the efficacy and safety of carvedilol in chronic heart failure reported in a study of 345 patients that carvedilol in addition to the standard therapy lowered mortality by 73% and lowered hospitalization rate from 58% to

64%[62]. As in the combined carvedilol groups' trial in MOCHA, the relative reduction in mortality of 73% supports the fact that the observed reduction in mortality rate of 18.11% in this study was not due to chance. In addition, results in the combined U.S carvedilol heart failure trials programme indicated a similar, highly statistically significant reduction in mortality by 65% which was consistent across all trials[63].

Furthermore the results in this study are strongly supported by the hypothesis[72,73,74,75] that improvement in left ventricular function observed with inclusion of the β-blocker, carvedilol, to standard therapy of a loop diuretic and an ACE-inhibitor is associated with improved survival.

DISCHARGE

Before the implementation of the updated treatment guidelines between 2004 and 2005, 64.73% of patients were discharged whiles 35.27% died during admission. After the implementation of guidelines in 2006 and 2007, 82.84% of the patients went home alive whiles 17.16% died during admission. The highest in-hospital mortality occurred in patients who were admitted with NYHA Classes III and IV heart failure. This clearly indicates that with strict adherence to the updated treatment guidelines i.e. translating evidence-based therapy into clinical practice, more lives with heart failure could be saved.

MONITORING PARAMETERS

Hypertensive emergency monitoring charts i.e. blood pressure charts were found in the folders of all the patients in this study. It is significant that daily measurement of the blood pressure was done to monitor the progress of patients and

efficacy of the antihypertensive agents used. Most heart failure cases were caused by hypertension[27].

Diabetic emergency monitoring charts to monitor serum glucose found in the folders of some patients. It was not clear whether random blood sugar tests were not performed in the remaining 26% of the patients or they got lost in their folders.

Blood tests i.e. full blood count was investigated in all the patients in this study.

Biochemical investigations form a major component in the diagnosis and prognosis of heart failure. They help in the correct choice of drug therapy for each heart failure patient.

Electrocardiography and echocardiography were performed in all patients suspected of heart failure to confirm and also to know the cause of the disease. All the patients were booked for these investigations.

Chest-X-rays were done for all the patients which helped in the diagnosis of the disease to determine the size of the heart whether enlarged or otherwise.

Not all the patients had their drug administration and temperature charts well documented by the ward nurses. These are important parameters to confirm that patients were receiving their medicines at the right time interval and in correct doses.

CHAPTER SIX: CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

The study has revealed that the prescribing patterns in the medical wards have shown delays in translating the evidence from clinical trials of heart failure pharmacological agents into clinical practice, thereby denying patients with heart failure the benefits of drug treatments proven to improve well-being and prolong life.

The ACE-inhibitor, lisinopril, and the loop diuretic, frusemide were mostly prescribed. However, the β-blocker, carvedilol, was woefully underutilized.

The combination of these drugs as recommended in the updated treatment guidelines in 2006, reduced mortality and morbidity, length of hospital stay and improved patients' quality of life significantly.

The inclusion of the β-blocker, carvedilol, contributed to these achievements.

The role of β-blockers in the treatment of heart failure has made tremendous progress in recent times, as these agents are now proven as having the most impact on a number of lives saved and reduced rehospitalization.

Previous general concerns limiting the use of β-blockers in various patient subsets have been allayed by the evidence induced in this study and the time is now to overcome these barriers and to consider providing β-blockers to the many patients with heart failure who are in need of this therapy.

To conclude, strong evidence deduced in this study supports the use of an ACE-

inhibitor and a β-blocker as the first line therapies to prevent and treat heart failure. The aldosterone-antagonist, spironolactone offers additional symptomatic and mortality benefits in patients with advanced heart failure.

The challenge now lies in the ability of the medical profession and healthcare system to bridge the gap between evidence-based medicine and clinical practice in providing the most effective therapies and above all preventive strategies for heart failure. As data on pharmacologic and other treatment methods for this condition evolve, their optimal and effective utilization together are anticipated to ameliorate this serious cardiovascular disease.

Despite the limitations in study design, it is expected that this study provides baseline data for future comparisons and monitoring of the management of patients with heart failure seen at the Komfo Anokye Teaching Hospital in Kumasi.

6.2 RECOMMENDATION

1. All heart failure patients discharged from the hospital must receive the β-blocker, carvedilol or any other treatment guideline-recommended β-blockers like metoprolol or bisoprolol for asthmatic heart failure patients unless absolutely contraindicated.

   Diuretics and digoxin are used to relieve symptoms

   Hydralazine and nitrates are often reserved for patients with clinically significant renal dysfunction.

   The calcium-antagonists, amlodipine and felodipine should be used to treat

hypertension or angina in heart failure.

Aspirin in low doses should be used only when heart failure is of ischaemic nature.

Routine use of aspirin in all heart failure patients should be discouraged.

2. It is important to constantly monitor patient's heart rate, blood pressure and clinical status i.e. symptoms, signs-especially signs of congestion and body weights.
3. It is important to check blood chemistry 1-2 weeks after initiation of therapy and 1-2 weeks after final dose titration.
4. Patients must be counselled on the following

- to recognize and monitor symptoms of heart failure
- to comply with medications to prevent worsening of the conditions and rehospitalization
- to make proper food choices and other dietary changes
- manage stress and regularly check blood pressures and glucose
- to get enough rest
- to maintain a low-level exercise routine
- to reduce salt intake and alcohol consumption.

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APPENDIX 1 QUESTIONNAIRE

HEART FAILURE PATIENTS

ADMITTED AT MEDICAL WARDS OF KATH BEFORE PROTOCOL

SECTION A

1. INITIALS OF PATIENT……2. SEX: MALE( ) FAMALE ( )

3.PATIENT'S (FOLDER) NUMBER…….4. AGE:……

5.DATE OF ADMISSION…… 6. DATE OF DISCHARGE…….

7. SYSTOLIC B.P…… 8. DIASTOLIC B.P…….

9. CONDITION OF PATIENT ON ADMISSION

TICK

CONSCIOUS/STABLE…… ( )

CONSCIOUS/UNSTABLE…… ( )

UNCONSCIOUS/UNSTABLE… ( )

10.TYPE OF HEART FAILURE----BVF ( )…LVF ( )….RVF ( )

11. CAUSE OF HEART FAILURE…HYPERTENSION ( ) CARDIOMYOPATHY ( ) VALVULAR DISEASE ( ) ANAEMIA ( ) CARDIOGENIC SHOCK ( )

SECTION B

12. HEART FAILURE TREATMENT ON ADMISSION

TICK YES-GIVEN NO-NOT GIVEN

DRUG	YES	NO
FRUSEMIDE		
ALDACTONE		
ISOSORBIDE DINITRATE		
ASPIRIN		
LISINOPRIL		
AMLODIPINE		
CARVEDILOL		
TENSIFORT		
NIFEDIPINE		
DIGOXIN		
HYDRALAZINE		
ALDOMET		
ATENOLOL		

SECTION C

MONITORING/INVESTIGATION
TICK WHEN DONE

13. RENAL FUNCTION TEST ( )

14. LIVER FUNCTION TEST ( ) THYROID FUNCTION TEST ( )

15. FULL BLOOD COUNT ( ) FASTING BLOOD SUGAH ( )

16. CHEST X-RAY ( )

17. ELECTROCARDIOGRAPHY ( ) ECHOCARDIOGRAPHY ( )

SECTION D

OUTCOME

TICK WHERE APPROPRIATE

18. DISCHARGED ( )

19. DIED ( )

20. MONITORING PARAMETERS CHARTED AT THE WARD

PARAMETER	YES	NO
HYPERTENSIVE EMERGENCY MONITORING CHART		
DIABETIC EMERGENCY MONITORING CHART		
TEMPERATURE MONITORING CHART		
DRUG ADMINISTRATION CHART		
FLUID INPUT/OUTPUT CHART		

APPENDIX 2

QUESTIONNAIRE

HEART FAILURE PATIENTS

ADMITTED AT MEDICAL WARDS OF KATH AFTER PROTOCOL SECTION A

1. INITIALS OF PATIENT……2. SEX: MALE( ) FAMALE ( )

3. PATIENT'S (FOLDER) NUMBER…… 4. AGE:……

5. DATE OF ADMISSION…… 6. DATE OF DISCHARGE…….

7. SYSTOLIC B.P…… 8. DIASTOLIC B.P…….

9. CONDITION OF PATIENT ON ADMISSION

TICK

CONSCIOUS/STABLE…… ( )

CONSCIOUS/UNSTABLE…… ( )

UNCONSCIOUS/UNSTABLE... ( )

10. TYPE OF HEART FAILURE----BVF ( )…LVF ( )….RVF ( )

11. CAUSE OF HEART FAILURE…HYPERTENSION ( ), CARDIOMYOPATHY ( )
VALVULAR DISEASE ( ) ANAEMIA ( )
CARDIOGENIC SHOCK ( )

SECTION B

12. HEART FAILURE TREATMENT ON ADMISSION

TICK YES-GIVEN NO-NOT GIVEN

DRUG	YES	NO
FRUSEMIDE		
ALDACTONE		
ISOSORBIDE DINITRATE		
ASPIRIN		
LISINOPRIL		
AMLODIPINE		
CARVEDILOL		
TENSIFORT		
NIFEDIPINE		
DIGOXIN		
HYDRALAZINE		
ALDOMET		
ATENOLOL		

SECTION C

MONITORING/INVESTIGATION
TICK WHEN DONE

13. RENAL FUNCTION TEST ( )

14. LIVER FUNCTION TEST ( ) THYROID FUNCTION TEST ( )

15. FULL BLOOD COUNT ( ) FASTING BLOOD SUGAH ( )

16. CHEST X-RAY ( )

17. ELECTROCARDIOGRAPHY ( ) ECHOCARDIOGRAPHY ( )

SECTION D

OUTCOME

TICK WHERE APPROPRIATE

18. DISCHARGED ( )

19. DIED ( )

20. PARAMETERS CHARTED AT THE WARD

PARAMETER	YES	NO
HYPERTENSIVE EMERGENCY MONITORING CHART		
DIABETIC EMERGENCY MONITORING CHART		
TEMPERATURE MONITORING CHART		
DRUG ADMINISTRATION CHART		
FLUID INPUT/OUTPUT CHART		