CLINICAL, ECHOCARDIOGRAPHIC, ELECTROCARDIOGRAPHIC AND CHEST RADIOGRAPHIC CHARACTERISTICS OF PATIENTS ADMITTED AT MUHIMBILI NATIONAL HOSPITAL MEDICAL WARDS WITH NYHA III/IV HEART FAILURE

By

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A Dissertation Submitted in Partial Fulfilment of the Requirements for the Degree of Master of Science (Cardiology) of the Muhimbili University of Health and Allied Sciences (MUHAS)

October, 2009.

CERTIFICATION

The undersigned certify that they have read and hereby recommend for the examination of a dissertation entitled *Clinical, Echocardiographic,Electrocardiographic and Chest Radiographic characteristics of patients admitted at Muhimbili National Hospital Medical wards with NYHA class III/IV heart failure,* in partial fulfilment of the Requirements for the degree of Master of Science (Cardiology) of the Muhimbili University of Health and Allied Sciences.

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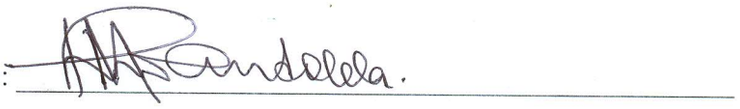
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DEDICATION

I dedicate this dissertation to my second born son, Daniel, who was born during the course of this work.

ABSTRACT

Background: Heart failure is a major public health problem in developed countries, imposing a huge burden on their health care systems. In Africa, improvements in the control of communicable diseases and the migration to the cities with changes in life styles, cardiovascular diseases such as hypertension with consequence heart failure are increasingly recognized as significant causes of morbidity and mortality.

Most of the published data on heart failure is based on work in Caucasian populations within the developed world, and data on its incidence, prevalence, etiology, treatment, and outcome in Africa are lacking.

Objective: The aim of this study was to describe the clinical,echocardiographic,electrocardiographic and chest radiographic characteristics of patients admitted at Muhimbili National Hospital medical wards with NYHA III/IV heart failure.

Materials and Methods: A cross section study was done for three months in 2008. During this period, a total of 100 patients with severe heart failure were evaluated clinically. All had thorough clinical history, standard resting transthoracic echocardiograms, resting 12 lead electrocardiograms, and standard postero anterior chest radiograms taken and data analysed.

Results: PND was a symptom which occurred to all the patients. Other most frequently occurring features included positive hepatojugular reflux, Elevated Jugular Venous pressure, tender hepatomegally and nocturnal cough. Dilated Cardiomyopathy was the most frequent echocardiographic diagnosis (28%), chronic rheumatic valvular disease was the second most frequent diagnosis (23%), left ventricular hypertrophy without LV dilatation (19%) and Isolated LV dilatation (18%). Fifty four (54%) patients had significant LV systolic dysfunction (LVEF<30%) and 9% had grade one LV diastolic dysfunction. Pulmonary Hypertension was found in 39% of the patients. The most frequently dilated chambers were the LA (58%) and LV (56%) respectively.

Conclusions:

1. PND was a common symptom to all the patients. Other most frequently occurring features included positive hepatojugular reflux, Elevated Jugular Venous pressure, tender hepatomegaly and nocturnal cough.
2. The most common echocardiographic finding of patients in this study was dilated cardiomyopathy followed by chronic rheumatic valvular disease. Left ventricular systolic dysfunction was the most common form of heart failure (54%).Left atrial dilatation was the most frequently dilated cardiac chamber, confirmed by both the ECG and Echo.
3. Sinus tachycardia was the most frequent Arrhythmias (47%), left atrial and right atrial abnormalities accounted for 42% and 7% respectively, 22% of patients had BBB, and 46% had LVH with secondary ST-T changes. Majority (76%) of patients had more than one ECG abnormalities.
4. Cardiomegaly was the most common radiographic finding accounting for 87%.Seventy six (76%) patients had interstitial oedema of varying severity and 12% had pleural effusion.

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ABBREVIATIONS

AV-Aortic Valve

ASD-Atrial Septal Defect

CHD-Congenital Heart Disease

CXR-Chest X rays

ECHO-Echocardiogram

ECG-Electrocardiogram

MV-Mitral Valve

LVOT-Left Ventricular Outflow Tract

LV-Left Ventricle

LVEF-Left Ventricular Ejection Fraction

LA-Left Atrium

MUHAS-Muhimbili University of Health and Allied Sciences

NYHA-New York Heart Association

PA-Postero anterior

PV-Pulmonic Valve

PND-Paroxysmal Nocturnal Dyspnoea

RVOT-Right Ventricular Outflow Tract

RA-Right Atrium

RV-Right Ventricle

SVT-Supra Ventricular Tachycardia

TV-Tricuspid Valve

VT-Ventricular Tachycardia

VES-Ventricular Extra Systole

VSD-Ventricular Septal Defect

1. INTRODUCTION.

In the past few decades, heart failure has emerged as a major public health problem in developed countries, imposing a significant burden on their health care systems.

Most of the published data on heart failure is based on work in Caucasian populations within the developed world, and data on its incidence, prevalence, etiology, treatment, and outcome in Africa are lacking.[1]

As a result of improvements in the control of communicable diseases on the one hand and the migration to the cities with changes in life styles on the other hand, cardiovascular and metabolic diseases such as hypertension,stroke and diabeitis mellitus are increasingly recognized as significant causes of morbidity and mortality in most African countries.[2] Increasing industrialization and urbanization, while raising living standards and increasing lifespan for many, accompanied by a change of diet (unfavorable changes in blood lipid composition resulting from diet changes), cigarette smoking, lack of exercise, and obesity contribute to risk factors for atherosclerotic cardiovascular disease [3].Diabetes Mellitus, hypertension, and coronary artery disease are the initial consequences and heart failure follows, which is the end product of myocardial damage caused by these disease processes alone or more usually in combination. Cardiovascular diseases account for 7% to 10% of all medical admissions to hospitals, for example,in Northern Savannah, Nigeria and Nairobi, Kenya, with heart failure contributing to 3% to7 %.[4, 5]

The problem of heart failure has been recognized in sub-Saharan Africa for over 60 years[6]

There are 3 major trends that are evident. Firstly, the vast majority of heart failure cases in sub-Saharan Africa are due to non ischemic causes, with rheumatic heart disease, hypertensive heart disease, and cardiomyopathy accounting for over 75% of cases in most series. Secondly, ischemic heart disease remains an uncommon cause of heart failure with no significant increase in its contribution to the cases of heart failure over the past 60 years. This observation supports the proposal that non ischemic heart disease is the priority for research and clinical intervention in Africa.[7] and thirdly, the contribution of cor pulmonale and pericarditis of about 20% to cases of heart failure reflects the continuing impact of tuberculosis on heart disease on

the continent. Cor pulmonale is mainly related to post-tuberculosis lung damage, and pericarditis is overwhelmingly due to tuberculous involvement.[8]

The majority of the clinical studies of heart failure in sub-Saharan Africa were conducted in the pre-echocardiographic era or without the application of echocardiography in the majority of cases.[9] Oyoo and Ogola[9] conducted what was probably the first echocardiography-based study of the etiology of heart failure in 91 consecutive patients who were admitted to the Kenyatta National Hospital in Nairobi, Kenya. In this study, the case definition was based on the Framingham study, and the clinical assessment included echocardiography. The main etiologies of heart failure were rheumatic heart disease (32%), followed by dilated cardiomyopathy (25%) and hypertensive heart disease (17%).The relative importance of these etiologies was similar for both genders. Ischemic heart disease accounted for only 2% of cases, a proportion that is similar to other parts of Africa.[9] Africa is challenged by more than 30 million people infected with the Human Immunodeficiency Virus (HIV).As a consequence, HIV cardiac related pathologies, which are mainly viral myocarditis, dilated cardiomyopathy and pericarditis are important causes of heart failure in most African countries, this brings another heavy burden of patients with heart failure.[10]

Several papers have been published recently showing that a rapid epidemiologic transition is on the way in sub-Saharan Africa. The urbanization process has increased the prevalence of cardiovascular risk factors, mainly hypertension, diabetes, and obesity, paving the way for the substrate to a new epidemic in the future,namely the ischemic heart disease.[11,12] A study evaluating causes and clinical presentation of 97 patients with heart failure was conducted in South Africa.[12] The main causes of heart failure were hypertension (54%), cardiomyopathy (26%), and valvular heart disease (24%)

In a study of 167 consecutive patients admitted to the Yaoundé General Hospital in Cameroon who were evaluated by echocardiography, heart failure accounted for 5.8% of all hospital admissions, rehospitalization rate was 8.3%, and the overall in-hospital mortality was 9 %[13]

Forty-four percent of patients were in New York Heart Association functional class III, and 7% were in class IV. Cardiac cavities were dilated, and left ventricular ejection fraction was low in patients with systolic (70%) and combined systolic and diastolic (20%) dysfunction.

The main aetiologies of heart failure were: Hypertension (49%), cardiomyopathies (34%) and valvular heart diseases (55%). Ischaemic heart disease was the fifth aetiology (39%).

Chronic rheumatic valvular disease is still an important cause of heart failure in Africa. The estimated prevalence of RHD in sub-Saharan Africa is 5.7 per 1000 and in North Africa 1.8 per 1000 derived from all relevant population-based data from the 1980s and 1990s.[15] It is a result of acute rheumatic fever (RF) which is an autoimmune disorder which ends up in characteristic changes of the mitral valve with echocardiographic diagnostic features being thickening at the leaflet edges, fusion of the commissures, and chordal shortening and fusion.[14] These anatomical changes lead to a typical functional appearance of the rheumatic mitral valve.Valvular heart diseases in Africa affect mainly children and young adults. Determinants of the persistence of RF and RHD in Africa are poverty, overcrowding, malnutrition, low level of disease awareness in the communities, shortage of resources for providing quality care and inadequate expertise of healthcare providers[16],potentially unidentifiable or more virulent group A streptococci, and possible yet unexplained underlying genetic susceptibility.[17]

2. LITERATURE REVIEW

Heart failure is a complex clinical syndrome that arises secondary to abnormalities of cardiac structure and/or function (inherited or acquired) that impair the ability of the left or right ventricle to fill or eject blood. These abnormalities may impair the functional capacity and quality of life of affected individuals; however, they do not necessarily dominate the clinical picture concurrently. Heart failure was once thought to arise primarily in the setting of depressed left ventricular (LV) ejection fraction (EF), epidemiological studies have shown that approximately 50 percent of patients who develop heart failure have a normal or preserved EF.[18]

PATHOPHYSIOLOGY OF HEART FAILURE

In recent years, much has been learned about the pathophysiology of heart failure, particularly in the area of fluid and electrolyte metabolism.

2.1. Regulation of body-fluid volume.

Evidence exists in support of the understanding of the regulation of body-fluid volume that is applicable to patients with edematous disorders such as cardiac failure.

The renal excretion of sodium and water normally parallels sodium and water intake, so that an increase in plasma and blood volume is associated with increased renal sodium and water excretion. In patients with heart failure, sodium and water are paradoxically retained, despite an increase in intravascular fluid volume. Renal sodium and water retention in these patients may be regulated not by the total blood volume, but by the degree of filling of another compartment, the so-called effective blood volume. The intrinsic renal mechanisms however, are not the primary cause of the sodium and water retention that results in pulmonary and peripheral edema in patients with heart failure, because renal sodium and water retention no longer occurs after successful heart transplantation.[19,20]

2.2. The role of arterial underfilling.

The integrity of the arterial circulation, as determined by cardiac output and peripheral vascular resistance or compliance, is the predominant determinant of renal sodium and water excretion. [19,20]

2.5. Non osmotic release of arginine vasopressin. .

Water retention in excess of sodium retention may occur in patients with heart failure and lead to hyponatremia.Hyponatremia is poor prognostic indicator in patients with heart failure[26].In patients with heart failure and hyponatremia, hypo-osmolality,which inhibits the release of arginine vasopressin in normal subjects, is associated with persistently high plasma concentrations of arginine vasopressin.[27]Arginine vasopressin causes anti diuresis by activating vasopressin V_2 receptors on the basolateral surface of the principal cells in the collecting duct, activation of these receptors initiates a cascade of intracellular signaling events by means of the adenylyl cyclase pathway, leading to translocation of aquaporin-2 water channels from cytoplasmic vesicles to the apical surface of the collecting duct, the result of which is fluid overload and further contributes to cardiac dysfunction in patients with severe heart failure.[28]

2.6. Role of natriuretic peptides.

In patients with heart failure, plasma atrial natriuretic peptide concentrations rise as atrial pressures increase. Brain or B-type natriuretic peptide is also increased in patients with heart failure.[29] Because plasma concentrations of brain natriuretic peptide are increased in patients with early heart failure or left ventricular dysfunction, plasma brain natriuretic peptide may be a sensitive diagnostic marker of heart failure.[25]

Atrial natriuretic peptide exerts its effects on the kidney at the levels of the glomerulus and collecting duct.In the glomerulus, it causes efferent arteriolar constriction and afferent arteriolar dilation, there by increasing the glomerular filtration rate. In the collecting duct, it decreases sodium reabsorption, thereby increasing sodium excretion. Atrial natriuretic peptide also inhibits the secretion of renin and aldosterone.The effects of brain natriuretic peptide on the kidney and on plasma renin activity and the plasma aldosterone concentration appear to be similar to those of atrial natriuretic peptide.[30]

Patients with heart failure are resistant to the natriuretic effect of exogenously administered natriuretic peptides. The resistance may be due to down-regulation of renal natriuretic peptide receptors, increased degradation of natriuretic peptides by neutral endopeptidase in the

proximal tubule, or decreased sodium delivery to the collecting duct as a result of a decrease in the glomerular filtration rate or increased sodium reabsorption in the proximal tubule.[31]

2.7. Role of endothelial hormones.

Prostacyclin and prostaglandin E are vasodilating hormones,they are primarily autocrine hormones, and depending on the species, one or both are found in the glomerular afferent arterioles. Angiotensin II,norepinephrine, and renal nerve stimulation increase the synthesis of these vasodilating prostaglandins, which then attenuate the vasoconstrictor effects of these three stimuli.[28]These vasodilatory prostaglandins may thus counterbalance the neurohormone induced renal vasoconstriction that occurs in heart failure. [28]

Nitric oxide is an even more potent vasodilator than prostacyclin and prostaglandin E. Endothelial cells contain a constitutive nitric oxide synthase the activity of which may be blunted in heart failure.[33] Thus, the constrictor action of the endogenous vasoconstrictors whose concentrations are elevated in heart failure, including angiotensin II, norepinephrine, and arginine vasopressin, may be increased by decreased nitric oxide synthesis in endothelial cells.Endothelin is one of the most potent vasoconstrictors,and plasma endothelin concentrations are increased in some patients with heart failure. High plasma endothelin concentrations are associated with a poor prognosis in patients with New York Heart Association class III or IV heart failure.[32]

2.8. CLINICAL FEATURES OF HEART FAILURE

2.8.1. Symptoms

History and physical examination remain the cornerstone of the clinical assessment and diagnosis of heart failure. The cardinal symptoms of heart failure are dyspnea, exercise intolerance, and fatigue. Symptoms are categorized into Major and minor, with dyspnea, orthopnea, paroxysmal nocturnal dyspnea, ankle edema, pulmonary edema, fatigue, exercise intolerance and cachexia comprising the major symptoms while weight loss, cough, nocturia, peripheral cyanosis, palpitations and depression comprising the minor symptoms.[34]

The purpose of the examination is to help determine the cause of heart failure, as well as to assess the severity of the syndrome.[35] Obtaining additional information about the hemodynamic profile, the response to therapy, and determination of the prognosis are important additional goals of the physical examination. The physical findings are also grouped into major and minor. The major include, tachycardia, elevated venous pressure, positive hepatojugular reflux, pulmonary rales, tachypnea, third heart sound, hepatomegaly,ankle edema, ascites and pleural effusion. The minor signs are mitral regurgitation, cardiomegaly, splenomegaly, hypotension, pulsus alternans, extra systoles, atrial fibrillation and weight loss.[36]

Table1: Sensitivities, specificities and predictive accuracy of different clinical features.[37]

Symptoms or Signs	Sensitivity (%)	Specificity (%)	Predictive accuracy (%)
Exertional dyspnoea	66	52	23
Orthopnoea	21	81	2
Paroxysmal Nocturnal Dyspnoea	33	76	26
History of Oedema	23	80	22
Tachycardia at rest	7	99	6
Rales	13	91	21
Third heart sound	31	95	61
Jugular venous distension	10	97	2
Oedema on examination	10	93	3

2.9. FRAMINGHAM CRITERIA FOR CONGESTIVE HEART FAILURE

This is widely used in clinical studies of congestive Heart failure. It has been accepted since 1948 and has a sensitivity of 100% and specificity of 78%.[38] Diagnosis of CHF requires the simultaneous presence of at least 2 major criteria or 1 major criterion in conjunction with 2 minor criteria.

Major criteria:

- Paroxysmal nocturnal dyspnea
- Neck vein distention
- Rales
- Radiographic cardiomegaly (increasing heart size on chest radiography)
- Acute pulmonary edema
- S3 gallop
- Increased central venous pressure (>16 cm H2O at right atrium)
- Hepatojugular reflux
- Weight loss >4.5 kg in 5 days in response to treatment

Minor criteria:

- Bilateral ankle edema
- Nocturnal cough
- Dyspnea on ordinary exertion
- Hepatomegaly
- Pleural effusion
- Decrease in vital capacity by one third from maximum recorded
- Tachycardia (heart rate>120 beats/min.)

Minor criteria are acceptable only if they can not be attributed to another medical condition (such as pulmonary hypertension, chronic lung disease, cirrhosis, ascites, or the nephrotic syndrome).

2.10. NYHA FUNCTIONAL CLASSIFICATION FOR CONGESTIVE HEART FAILURE.

The New York Heart Association (NYHA) Functional Classification was established in 1928, it provides a simple way of classifying heart disease severity. It places patients in one of four categories, based on how much they are limited during physical activity:

Class I: Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.

Class II: Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea, or anginal pain.

Class III: Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.

Class IV: Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.[39]

The other more objective method for clinical assessment of cardiac functional status is the measurement of peak oxygen uptake (MVO_2) exercise test. Cardiac and peripheral compensatory mechanisms are activated in patients with chronic congestive heart failure to restore impaired left ventricular performance partially or completely. Abnormal baroreflex function and increased norepinephrine spillover, sympathetic discharge, downregulation of beta-adrenergic receptors, depletion of myocardial sympathetic stores, and increased peripheral and central chemosensitivity with abnormally increased ventilatory response to exercise characterize the disease process resulting in the cardiopulmonary response to exercise[40].The magnitude of exercise capacity impairment is a function of the relative inability

to augment stroke volume and abnormalities in skeletal muscle metabolism, which may be the predominant cause of functional limitation in a significant proportion of patients with heart failure. Peak oxgen uptake (MVo_2) measurements in patients with compensated congestive heart failure are useful in risk-stratifying patients with congestive heart failure to determine the subsequent incidence of cardiac events. The ability to achieve a peak Vo_2 higher than 18 ml $O_2 \times kg^{-1} \times min^{-1}$ and anaerobic threshold higher than 14 ml $O_2 \times kg^{-1} \times min^{-1}$ is associated with a relatively good long-term prognosis and maximal cardiac output higher than 8 liters/min/m^2. Patients who are unable to achieve a peak$V\ o_2$ of 10 ml $O_2 \times kg^{-1} \times min^{-1}$ and anaerobic threshold of 8 ml $O_2 \times kg^{-1} \times min^{-1}$ have a poor prognosis, and their maximal exercise cardiac output is usually less than 4 liters/min/m^2.When the peak $V\ o_2$ ranges from 10 to 18 ml/kg/min, prognostic risk is moderate.Values higher than 30 are common in patients with mild to moderate heart failure and values higher than 40 are often observed in those with more severe heart failure.[41]

2.11. ECHOCARDIOGRAPHY

Echocardiography provides a comprehensive and reliable hemodynamic assessment of patients with valvular heart disease including determination of pressure gradients, stenotic and regurgitant valve orifice area, regurgitant volume, and pulmonary artery pressures. In heart failure patients, echocardiography has emerged as the preferred diagnostic method for assessing the anatomy and function of the heart valves, studying the myocardium, pericardium, and evaluating regional wall motion at rest and during pharmacological stress in heart failure. It is the single most useful test in the evaluation of patients with heart failure, it is excellent for serial studies, and allows an assessment of both global and regional LV function. In the assessment of heart failure, echocardiography is a reliable, reproducible, and safe technique with many important features, such as doppler echocardiography,doppler tissue imaging, and strain rate imaging, as well as cardiac motion analysis.[42] The most important features in the evaluation of heart failure patients are the assessment of the LV ejection fraction (LVEF), severity of LV remodeling, and changes in diastolic inflow properties.

Two-dimensional echocardiography is invaluable in the assessment of structural abnormalities of the valves, systolic and diastolic function in patients with heart failure. It is important to demonstrate objective evidence of structural or functional abnormalities to explain a patient's symptoms of heart failure because the symptoms are not specific and more than one third of patients with a clinical diagnosis of heart failure may not actually have heart failure.[43] An echocardiogram may be the single most useful test in evaluating the etiology of the patient with acute heart failure because of its ability to assess global systolic and diastolic function, regional wall motion abnormalities, valvular function, homodynamic, and pericardial disease.[44,45, 46] It has been well established that about 50 percent of patients with new onset of heart failure have a normal LVEF.[47]

2.11.1. Echocardiographic Findings in Heart Failure

General Findings: Size and shape of the ventricle, left ventricular ejection fraction, regional wall motion, synchronicity of ventricular contraction, left ventricular remodeling,(concentric versus eccentric),left or right ventricular hypertrophy. Morphology and severity of valve lesions, mitral inflow and aortic outflow properties; right ventricular pressure gradient

Systolic dysfunction: Reduced LVEF (<45%), dilated left ventricle, thin left ventricular wall, eccentric left ventricular remodeling, mild or moderate mitral regurgitation, pulmonary hypertension, signs of increased filling pressure, reduced mitral filling

Diastolic Dysfunction: Normal LVEF (≥45%-50%), normal left ventricular size, thick LV wall, dilated atria, concentric LV remodeling, No or minimal mitral regurgitation, pulmonary hypertension, abnormal mitral filling pattern, signs of increased filling pressure, cardiac chamber dimensions, valvular structure, haemodymamic measurements and left ventricular systolic and diastolic functions are estimated in accordance with the American society of echocardiography guidelines.

Left Ventricular Dimensions;LV dimensions are measured from 2D-guided M-mode echocardiograms of the LV at the level of mitral leaflet tips or the papillary muscle using the

parasternal view. If no significant regional wall motion abnormalities are present, the LV dimensions measured at the mid ventricular level can be used to calculate global LVEF.
The thicknesses of the ventricular posterior wall and the ventricular septum (from the leading edge to the trailing edge) are measured from the same M-mode echocardiogram. The long-axis and short-axis dimensions of the ventricle can also be obtained directly from systolic and diastolic frames of the 2D parasternal long-axis view and apical view. The LV end-diastolic and end-systolic dimensions are measured at the level of tips of the mitral leaflets as the largest and the smallest LV dimensions, respectively.

Right ventricular Dimensions: RV dilatation may be the first clue to RV pressure or volume overload. Chronic RV pressure overload is accompanied by an increase in RV wall thickness in addition to dilatation.The thickness of the RV wall is normally less than 5 mm and is measured best from the subcostal view at the peak of the R wave.[48] The size of the RV is measured best from the apical four-chamber view. The dimension of the LVOT is measured from the parasternal long-axis view, and the dimension of the RV outflow tract (RVOT) is measured from the parasternal short-axis view.

Left Atrial Size and Volume: LA dimension is determined from the parasternal long-axis view at end-systole. However, the size of the LA may be underestimated from the parasternal view because the chamber may enlarge longitudinally. Therefore LA size should also be measured from apical views (from the tip of the mitral valve to the posterior wall of the LA). However, LA volume is a better measure of LA size and provides better prognostic value. Four different methods are available for determining LA volume: (a) prolate ellipse, (b) biplane area-length, (c) biplane Simpson, and (d) 3D echocardiography.[49]

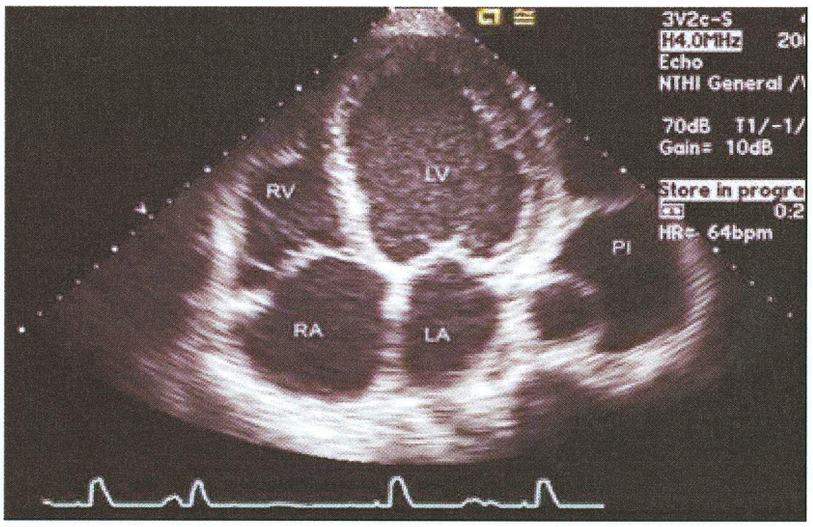


Figure1.Apical four chamber view of patient with dilated cardiomyopathy.There is dilatation of all four chambers and relative spherical geometry of the left ventricular geometry

Table2: The reference values for cardiac parameters in centimetres.[49]

Chamber	**WOMEN**				**MEN**			
	Normal	**Mild**	**Moderate**	**Severe**	**Normal**	**Mild**	**Moderate**	**Severe**
LVID(cm)	3.9-5.3	5.4-5.7	5.8-6.1	≥6.2	4.2-5.9	6.0-63	6.4-6.8	≥6.9
RV (cm)	2.7-3.3	3.4-3.7	3.8-4.1	≥4.2	2.7-3.3	3.4-3.7	3.8-4.1	≥4.2
LA (cm)	2.7-3.8	3.9-4.2	4.3-4.6	≥ 4.7	3.0-4.0	4.1-4.6	4.7-5.2	≥5.3
RA(cm)	2.9-4.5	4.6-4.9	5.0-5.4	≥5.5	2.9-4.5	4.6-4.9	5.0-5.4	≥5.5
LVEF%	≥55	45-54	30-44	<30	≥55	44-54	30-44	<30

Left Ventricular Functional parameters

Echocardiography can measure several parameters as an expression of systolic function of the heart. These parameters are LVEF, fractional shortening, stroke volume and cardiac index, systolic tissue velocity of the mitral annulus and myocardium, strain, and regional wall motion analysis.

Left Ventricular Ejection Fraction: This is a well-accepted expression of global LV function. It is a simple measure of how much end-diastolic volume is ejected from the LV with each contraction. LVEF has been found to be a strong predictor of clinical outcome in almost all major cardiac conditions, and it is used to select optimal management strategies. Objectively the LVEF is obtained using volumetric measurements as described by the following equation:

LVEF= (LVEDV-LVESV)/ (LVEDV)

Where, LVEDV and LVESV are LV end-diastolic volume and end-systolic volume, respectively.

LVEF can also be calculated from LV dimensions measured with M-mode or 2D echocardiography. M-mode or 2D echocardiographic measurement of LV dimensions from the mid ventricular level. The following formula is used to calculate LVEF:

$LVEF = LVEDD^2\text{-}LVESD^2/LVEDD^2$

Where, LVEDD and LVESD are end-diastolic dimension and end-systolic dimension, respectively. This equation is a percentage change in LV area, or fractional shortening of the LV short axis, which equals LVEF if the apical long-axis dimension remains the same from diastolic phase to systolic contraction.

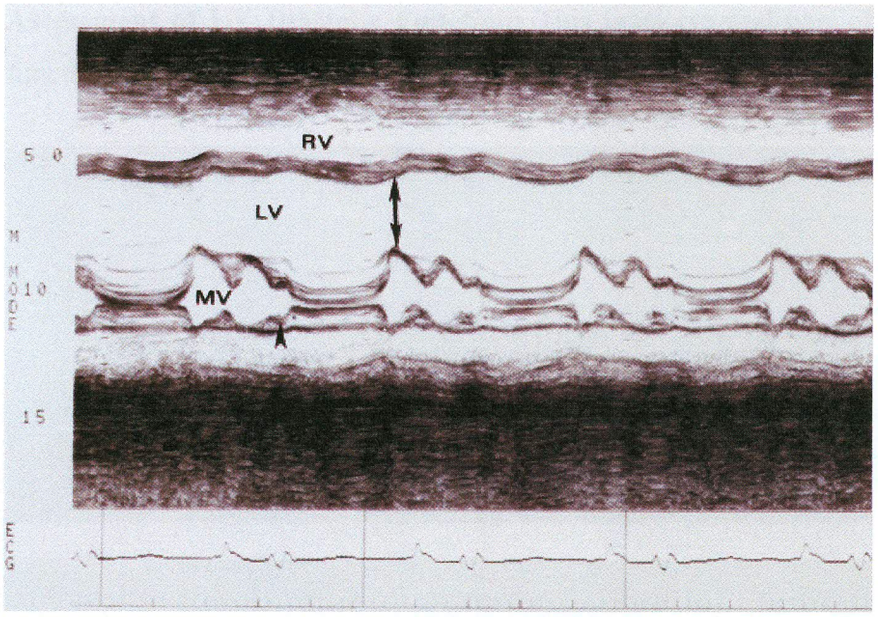


Figure2 (above): M-mode echocardiogram recorded in a patient with severe left ventricular systolic dysfunction.

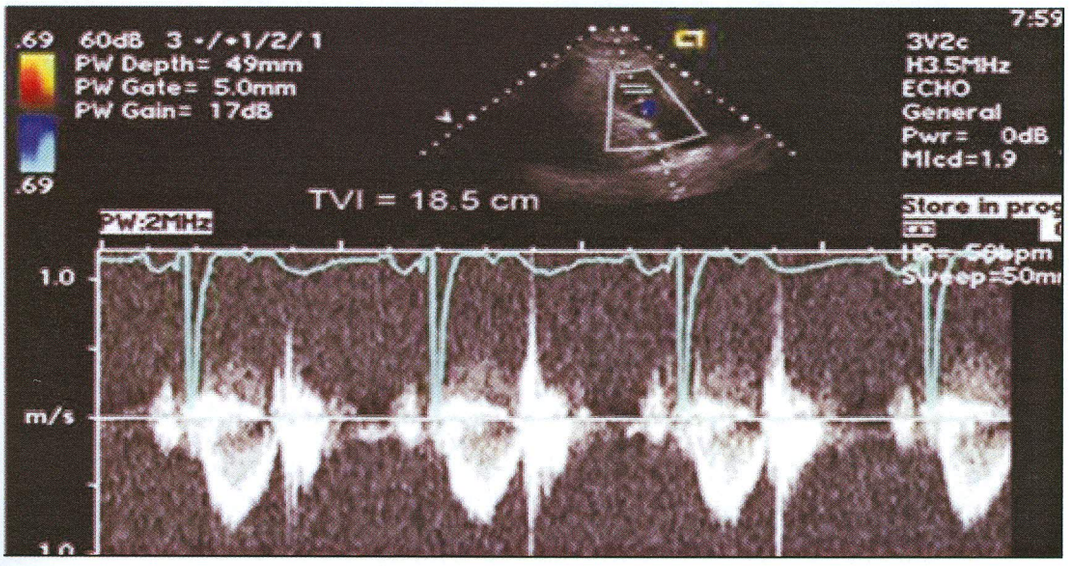


Figure3: Pulsed-wave Doppler in the right ventricular outflow tract demonstrating the impact of pulmonary hypertension of the contour of the outflow tract signal.

Assessment of LV Diastolic Function: This is an integral part of an evaluation of cardiac function because about 50 percent of patients with heart failure have a preserved LVEF.

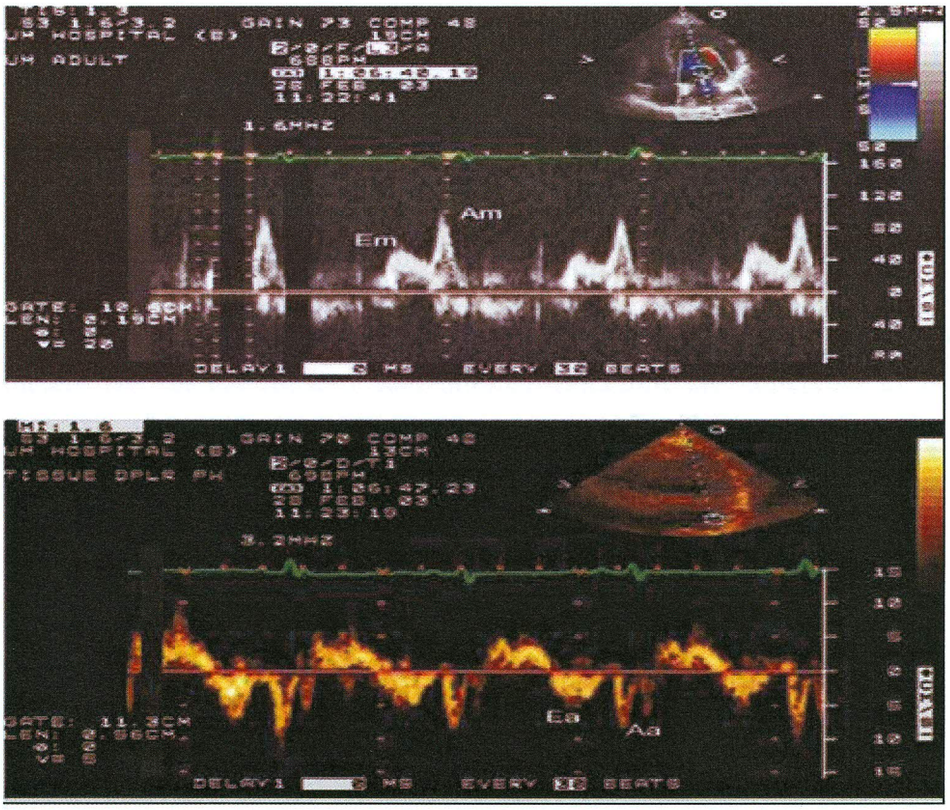


Figure4: Mitral inflow velocity and Doppler tissue annular recording in a patient with diastolic dysfunction and impaired relaxation. Note the reversed E/A ratio, which is paralleled by a reduced annular E_A/A_A ratio.

GRADING OF DIASTOLIC DYSFUNCTION (DIASTOLIC FILLING PATTERN)

Grade 1 (mild dysfunction) -impaired relaxation with normal filling pressure

Grade 2 (moderate dysfunction)-pseudo normalized mitral inflow pattern

Grade 3 (severe reversible dysfunction) -reversible restrictive (high filling pressure)

Grade 4 (severe irreversible dysfunction)-irreversible restrictive (high filling pressure)

Haemodynamic measurements: Doppler echocardiography records blood flow velocities in meters per second (m/sec), and doppler velocities are converted to pressure gradients in millimeters of mercury (mmHg) according to the simplified Bernoulli equation:

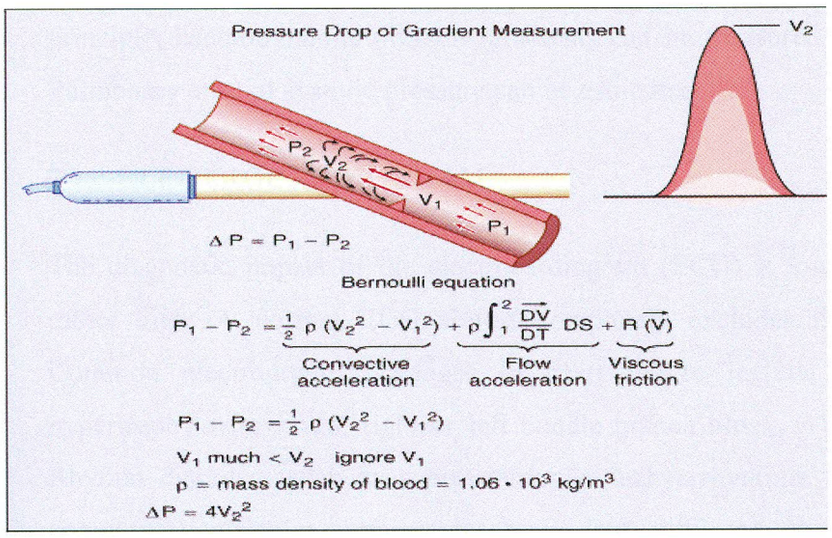


Figure5: Bernoulli equation for calculation of pressure gradient. (From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006)

The Bernoulli equation can be used to calculate a pressure gradient across any restrictive orifice. The components of the Bernoulli equation are convective acceleration, flow acceleration, and viscous friction. The full equation can be modified to $P_1-P_2 = (V_2^2-V_1^2) \times 4$ and further simplified in the absence of a significant V_1 velocity to $\Delta P=4V^2$. P_1 = pressure

proximal to an obstruction; P_2 = pressure distal to an obstruction; V_1 = velocity proximal to an obstruction; V_2 = Doppler velocity distal to an obstruction.[51]

Therefore: Pressure Gradient=$4(V)^2$

Where V is Blood flow velocity in m/sec.

When maximal Doppler velocity is converted to pressure gradient using the simplified Bernoulli equation, it represents the maximal instantaneous gradient. Mean gradient is an average of pressure gradients during the entire flow period. Maximal instantaneous or mean gradient obtained with doppler echocardiography has been shown to correlate well with corresponding gradients simultaneously measured by cardiac catheterization. Using this principle, haemodynamic pressure gradients can be measured across MV, TV, AV, PV and Pulmonary arterial systolic pressure can be estimated.[51]

2.12. ELECTROCARDIOGRAM

The diagnostic impact of the electrocardiogram (ECG) is low but the therapeutic impact is rather high. A normal ECG almost completely excludes the diagnosis of heart failure. Common electrographic changes in heart failure include, pathological Q waves, LV hypertrophy with strain, right or left bundle branch block, AV block, or T wave changes.[52] Rhythm disorders such as supraventricular tachyarrhythmia and atrial fibrillation are also common. Ventricular extrasystoles occur frequently and do not portend a poor prognosis, whereas nonsustained and sustained ventricular tachycardia may be considered as potentially harmful[53].The electrocardiogram (ECG) is an essential diagnostic tool in the evaluation of the patient with acute heart failure. In a study, Etude Française de l'Insuffisance Cardiaque Aiguë (EFICA)[54],which evaluated acute heart failure patients hospitalized in a hospital in France, it was shown that only 13 percent of ECGs were normal, with ischemic changes evident in 29 percent and atrial fibrillation in 25 percent. Of note, non cardiogenic pulmonary edema has been associated with giant, diffuse, symmetrical inverted T waves with QT prolongation, a finding also reported in pulmonary embolism and hypercatecholamine states.

proximal to an obstruction; P_2 = pressure distal to an obstruction; V_1 = velocity proximal to an obstruction; V_2 = Doppler velocity distal to an obstruction.[51]

Therefore: Pressure Gradient=$4(V)^2$

Where V is Blood flow velocity in m/sec.

When maximal Doppler velocity is converted to pressure gradient using the simplified Bernoulli equation, it represents the maximal instantaneous gradient. Mean gradient is an average of pressure gradients during the entire flow period. Maximal instantaneous or mean gradient obtained with doppler echocardiography has been shown to correlate well with corresponding gradients simultaneously measured by cardiac catheterization. Using this principle, haemodynamic pressure gradients can be measured across MV, TV, AV, PV and Pulmonary arterial systolic pressure can be estimated.[51]

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2.12.2. Left Ventricular Hypertrophy and Enlargement: Left ventricular hypertrophy (LVH) or enlargement produces changes in the QRS complex, the ST segment, and the T wave. The most characteristic finding is increased amplitude of the QRS complex. R waves in leads facing the left ventricle (i.e., leads I, aVL, V_5, and V_6) are taller than normal, whereas S waves in leads overlying the right ventricle (i.e., V_1 and V_2) are deeper than normal.

ST-T wave patterns vary widely in patients with left ventricular enlargement and hypertrophy. ST segment and T wave amplitudes can be normal or increased in leads with tall R waves. In many patients, however, the ST segment is depressed and followed by an inverted T wave.

These LVH-related repolarization changes usually occur in patients with QRS changes but can appear alone.

Other QRS changes seen in cases of LVH include widening of the QRS complex beyond 110 milliseconds, a delay in the intrinsicoid deflection, and notching of the QRS complex. Other abnormalities may include prolongation of the QT interval and evidence of left atrial abnormality[54].These electrocardiographic features are most typical of LVH induced by pressure overload of the left ventricle. Volume overload can produce a somewhat different pattern, including tall upright T waves and sometimes narrow (less than 30 milliseconds) but deep (0.2 mV or greater) Q waves in leads facing the left side of the septum. The diagnostic value of these changes in predicting the underlying hemodynamics is, however, very limited.

Diagnostic criteria: Many sets of diagnostic criteria for LVH have been developed on the basis of these abnormalities. Most methods assess the presence or absence of LVH, indicating whether LVH exists or not based on determined set of criteria. For example, the Sokolow-Lyon and Cornell voltage criteria require that voltages in specific leads exceed certain values. The Cornell voltage-duration method includes measurement of QRS duration as well as amplitudes.

Sokolow-Lyon index- $SV_1 + (RV_5 \text{ or } RV_6) > 3.5$ mV

or RaVL > 1.1 mV

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Cornell voltage criteria $SV_3 + RaVL \geq 2.8$ mV (for men)

$SV_3 + RaVL \geq 2.0$ mV (for women)

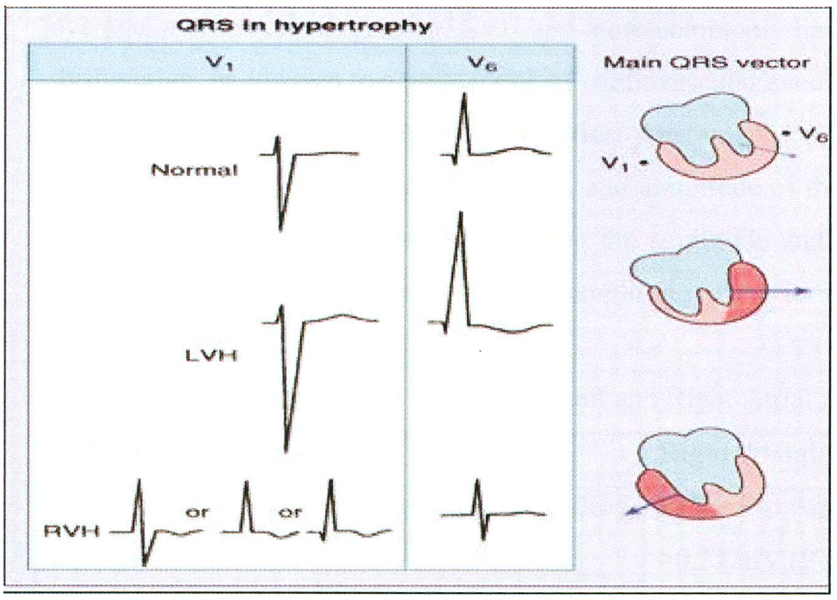


Figure6: Left ventricular hypertrophy and enlargement. (From Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 7th ed. St. Louis, CV Mosby, 2006, p 64)

Diagnostic accuracy: The relative diagnostic accuracy of these methods has been tested using autopsy, radiographic, echocardiographic and, most recently, magnetic resonance imaging[56]measurements of left ventricular size as standards. In general, these studies have reported low sensitivity and high specificity, sensitivities of 10-30% for Sokolow Lynon and 35-50% for Cornel Regression method Specificities for all measures varies from 85 percent to 95 percent for all criteria making these methods unsuitable as screening tests. But they still remain reliable diagnostic tests due to their high specificities.[55]

Clinical significance: An accurate electrocardiographic diagnosis of LVH is important to detect hypertrophy in association with overt cardiovascular disease (e.g., hypertension) or as an initial manifestation of various diseases, to assess prognosis, and to monitor progression or regression of hypertrophy during treatment. The presence of electrocardiographic criteria for

LVH identifies a subset of the general population with a significantly increased risk for cardiovascular morbidity and mortality.[57] Patients with repolarization abnormalities have, on average, more severe degrees of LVH and more commonly have symptoms of left ventricular dysfunction, in addition to a greater risk of cardiovascular events.[58,59]

2.12.3. Right and left atrial abnormalities: Anatomical or functional abnormalities of the left atrium alter the morphology, duration, and amplitude of the P waves in the clinical ECG. Specific abnormalities include increases in the amplitude and duration of the P wave in the limb leads, as well as an increase in the amplitude of the terminal negative portion of the P wave in lead V_1.

Table3: Common Diagnostic Criteria for Left and Right Atrial Abnormalities

Left Atrial Abnormality	**Right Atrial Abnormality**
Prolonged P wave duration >120 msec in lead II	Peaked P waves with amplitudes in lead II >0.25 mV (P pulmonale)
Prominent notching of the P wave, usually most obvious in lead II, with an interval between the notches of >40 (P mitrale)	Rightward shift of the mean P wave axis to above +75 degrees
Ratio between the duration of the P wave in lead II and the duration of the PR segment >1.6	Increased area under the initial positive portion of the P wave in lead V_1 to >0.06 mm-sec
Increased duration and depth of the terminal negative portion of the P wave in lead V_1 (the P terminal force) so that the area subtended by it exceeds 0.04 mm-sec	
Leftward shift of the mean P wave axis to between -30 and -45 degrees	

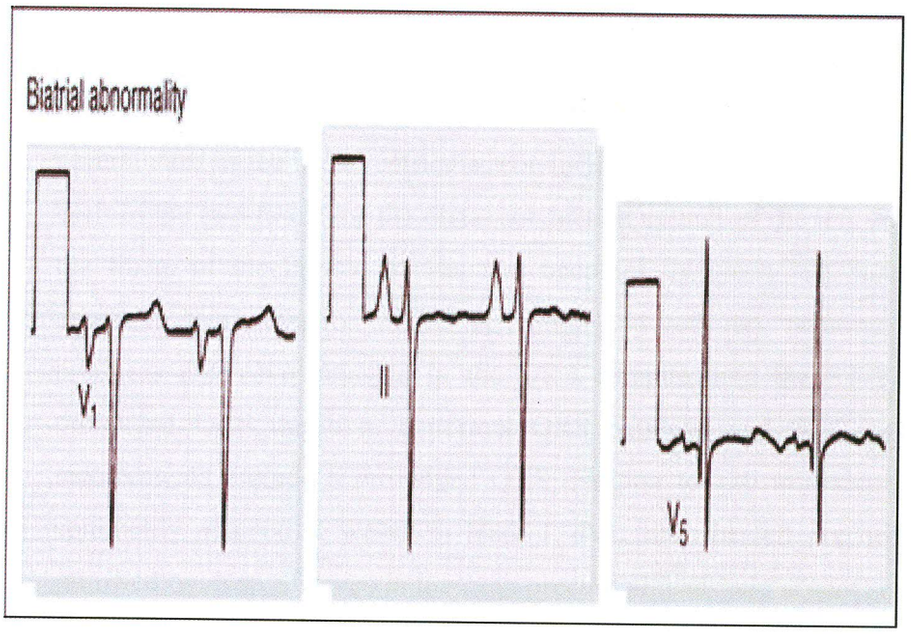


Figure7: Biatrial abnormality, with tall P waves in lead II (right atrial abnormality) and an abnormally large terminal negative component of the P wave in lead V_1 (left atrial abnormality). The P wave is also notched in lead V_5.(From: Libby: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed.Page 160)

Comparison of the various electrocardiographic abnormalities with echocardiographic criteria for left atrial enlargement demonstrates the limited sensitivity but high specificity for standard electrocardiographic criteria. For example, the presence of classic wide and notched P wave patterns has a sensitivity of only 20 percent but a specificity of over 90 percent for detecting echocardiographically enlarged left atria.[60]

Diagnostic accuracy of right atrial abnormalities: Echocardiographic correlations have shown that the electrocardiographic findings of right atrial abnormality have limited sensitivity but high specificity for detecting right atrial enlargement.

2.12.4. Left Bundle Branch Block: LBBB results from conduction delay or block in any of several sites in the intra ventricular conduction system, including the main left bundle branch, each of the two fascicles, the distal conduction system of the left ventricle or, less commonly, the fibers of the bundle of His that becomes the main left bundle branch. The result is extensive reorganization of the activation and recovery patterns of the left ventricle that produces extensive changes in the QRS complex and the ST-T wave.LBBB produces a prolonged QRS duration, abnormal QRS patterns, and ST-T wave abnormalities. Basic requirements include a prolonged QRS duration to 120 milliseconds or beyond, broad and commonly notched R waves in leads I, aVL, and the left precordial leads,narrow r waves followed by deep S waves in the right precordial leads and absent septal q waves. R waves are typically tall and S waves are deep. The mean QRS axis with LBBB is highly variable; it can be normal, deviated to the left or, less often, deviated to the right.

Common Diagnostic Criteria for Bundle Branch Blocks: Complete Left Bundle Branch Block requires that, the QRS duration be $\geq$ 120 msec, the R wave be broad and notched in lateral precordial leads (V_5 and V_6) and usually leads I and aVL.There should also be small or absent initial r waves in right precordial leads (V_1 and V_2) followed by deep S waves Absent septal q waves in left-sided leads. Prolonged intrinsicoid deflection (>60 msec) in V_5 and V_6.[61]

CLINICAL SIGNIFICANCE OF LBBB: LBBB usually appears in patients with underlying heart disease, although as many as 12 percent of patients with LBBB have no demonstrable heart disease.[62]Even among persons without overt heart disease, LBBB is associated with a higher than normal risk of cardiovascular and all-cause mortality.[63] It is associated with substantially higher than expected risks of high-grade atrioventricular block and cardiac death, mostly as a result of sudden death outside the hospital setting.[64]Among patients with coronary artery disease, the presence of LBBB correlates with more extensive disease, more severe left ventricular dysfunction, and reduced survival rates. Patients with associated left or right axis deviation have more severe clinical manifestations. Left axis deviation is associated with more severe conduction system disease that includes the fascicles as well as the main left bundle,

whereas right axis deviation suggests dilated cardiomyopathy with biventricular enlargement. In addition to the hemodynamic abnormalities produced by the underlying cardiovascular conditions, the abnormal ventricular activation pattern of LBBB itself induces hemodynamic changes. These include abnormal systolic function with dysfunctional contraction patterns, reduced ejection fraction and lower stroke volumes, and abnormal diastolic function.[65]

Right Bundle Branch Block: Right bundle branch block is a result of conduction delay in any portion of the right-sided intraventricular conduction system. The delay can occur in the main right bundle branch itself, in the bundle of His, or in the distal right ventricular conduction system. The QRS duration $\geq$ 120 msec
Broad, notched R waves (rsr′, rsR′, or rSR′ patterns) in right precordial leads (V_1 and V_2)
Wide and deep S waves in left precordial leads (V_5 and V_6)

Clinical significance of RBBB: RBBB is a common finding in the general population, and many persons with RBBB have no clinical evidence of structural heart disease. The high prevalence of RBBB corresponds to the relative fragility of the right bundle branch, as suggested by the development of RBBB after the minor trauma produced by right ventricular catheterization.In the group without overt heart disease; RBBB is not an independent predictor of cardiovascular mortality.[65] However, the new onset of RBBB does predict a higher rate of coronary artery disease, congestive heart failure, and cardiovascular mortality. When cardiac disease is present, the coexistence of RBBB suggests advanced disease with, for example, more extensive multi vessel disease and reduced long-term survival in patients with ischemic heart disease.

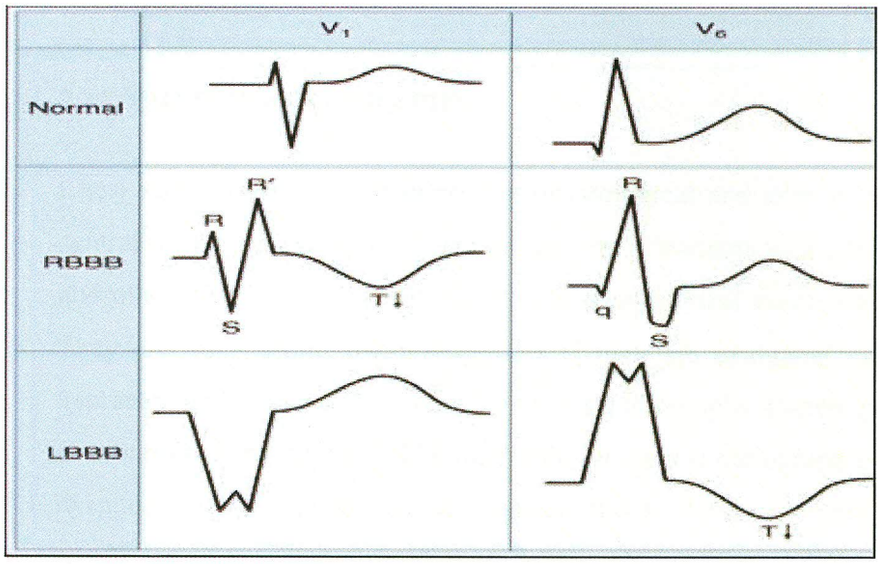


Figure8: Comparison of typical QRS-T patterns in right bundle branch block (RBBB) and left bundle branch block (LBBB) with the normal pattern in leads V_1 and V_6.(From Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 7th ed. St. Louis, CV Mosby, 2006, p 80.)

2.13. CHEST RADIOGRAPHY

Chest radiographs provide important physiological and anatomical information; they play a central role in the evaluation and management of patients with a wide variety of cardiovascular and other disorders. Standard 6-foot frontal and lateral chest films are almost always useful. They are very useful diagnostic tools if they are evaluated carefully, using a systematic approach and compared with prior chest radiographs (when possible), whether recorded conventionally or digitally. The main role for a chest radiograph is to exclude other causes for dyspnoea such as pleural effusion, pneumothorax, lung carcinoma, or pneumonia. Pulmonary edema supports a diagnosis of heart failure, although the reliability of identifying upper lobe venous blood diversion is poor. Cardiothoracic ratio is only of moderate value in identifying heart failure as the cause of breathlessness.[66]

In Acute Decompensated Heart Failure National Registry (ADHERE), a multicenter study involving 54639 patients in various regions in the United states of America, Chest radiography was performed in more than 90 percent of patients and there was evidence of congestion in more than 80 percent of these patients.[67]Chest radiographs are considered a standard component of the initial assessment of a patient with presumed HF. The presence of pulmonary venous congestion, interstitial edema, alveolar edema, and cardiomegaly increase the likelihood of HF.[68] Although most patients have cardiomegaly, the absence of pulmonary venous congestion or edema should not exclude the diagnosis of HF.[69] Data from the Acute Decompensated Heart Failure National Registry revealed that 18.7% of patients with HF had no signs of pulmonary congestion on the emergency department chest radiograph.[70]

In another study, radiographic evidence of pulmonary congestion was absent in 53% of patients with moderately elevated pulmonary capillary wedge pressure (PCWP) and 39% of patients with significantly elevated PCWP.[73] Studies have also shown that agreement among clinicians in chest x-ray interpretation is moderate to almost perfect for interstitial edema, but only moderate for cardiomegaly and vascular redistribution.[71] Chest radiographs should be

part of the initial evaluation of a patient with suspected HF, but absence of findings should not exclude the diagnosis.

2.13.1. Techniques: Different systems exist for reading chest radiographs, but the system should be able to cover for the thoracic spine, neck (for masses and tracheal position), costophrenic angles, lung apices, retro cardiac space, and retrosternal space. Looking at these areas allows definition of the mediastinal position and cardiac and aortic situs and the presence of pleural effusions, scarring, or diaphragmatic elevation. The lung field evaluation should involve a careful search for infiltrates or masses, even when examining a chest radiograph for cardiovascular abnormalities. The overall size of the cardiac silhouette, its position, and the location of the ascending and descending aorta must be specifically evaluated[71].Overall cardiomegaly can be judged with reasonable accuracy on the PA view by noting whether or not the diameter of the heart exceeds half the diameter of the thorax. Cardiomegaly is a common but nonspecific finding.[71, 72]

2.13.2. Lungs and Pulmonary vasculature: Pulmonary vascularity can best be defined by looking at the middle zone of the lungs (i.e., the third of the lungs between the hilar region and the peripheral region laterally) and comparing a region in the upper lung field with a region in the lower lung field at equal distances from the hilum[73] .Vessels should be larger in the lower lung but sharply marginated in both the upper and lower zones. In normal persons, the vessels taper and bifurcate and are difficult to define in the outer third of the lung. They normally cannot be seen near the pleura. Two distinct patterns of abnormality are recognizable. When pulmonary arterial flow is increased, as in patients with a high-output state (e.g., pregnancy, severe anemia, and hyperthyroidism) or left-to-right shunt, the pulmonary vessels are seen more prominently than usual in the periphery of the lung. They are uniformly enlarged and can be traced almost to the pleura, but their margins remain clear. With elevated pulmonary artery pressure, in contrast, the vessel borders become hazy, the lower zone vessels constrict, and the upper zone vessels enlarge, and vessels become visible farther toward the pleura, in the outer third of the lungs

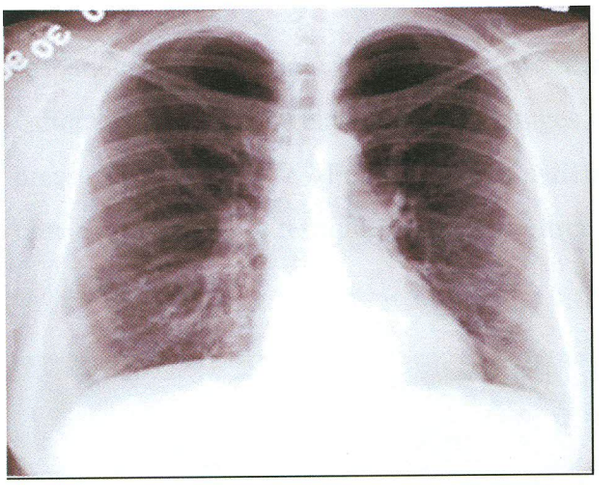


Figure9(above): PA Chest radiograph of a 47-year-old woman with a small atrial septal defect showing a normal cardiac contour, with mild pulmonary vascular plethora at the periphery of the lung fields. From: Libby: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed.Page 335)

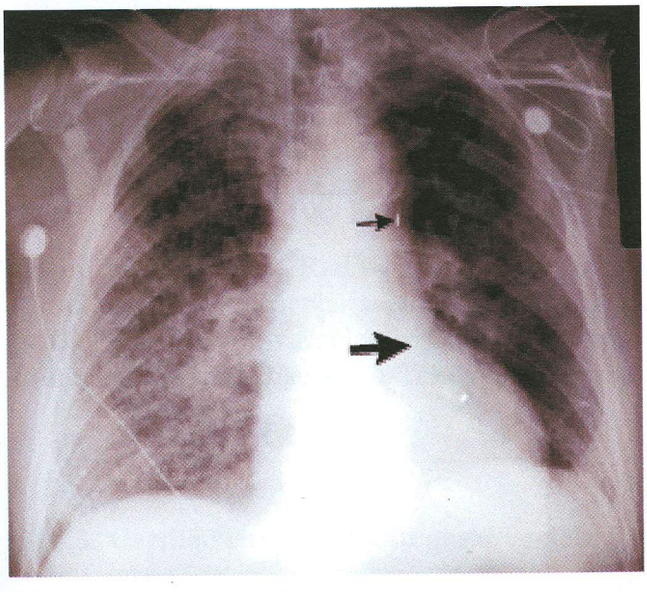


Figure10: PA view Chest radiography of a patient with acute pulmonary edema(From: Libby: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed.Page 335)

In patients with chronic heart failure, there are chronic changes in the pulmonary vascular pattern that do not correlate with the changes that occur in patients with normal left ventricular pressure at baseline.[73,74] For example, a patient with a chronic elevation of LVEDP to 25 to 30 mm Hg may have a normal pulmonary vascular pattern or only moderate rather than marked redistribution.

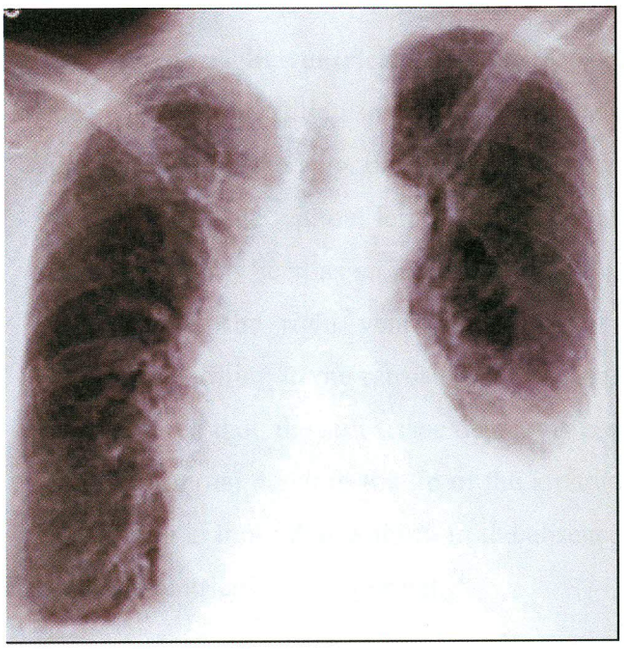


Figure11: PA view Radiograph of an elderly man after acute myocardial infarction complicated by ventricular septal defect (VSD) and heart failure demonstrating cardiomegaly, marked pulmonary vascular redistribution, and a large left pleural effusion.From Libby: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th ed.Page336)

2.13.3. Cardiac chambers and great vessels: Definition of individual chambers clearly in a normal chest radiograph is impossible, when the cardiac silhouette is enlarged, it is often related to biventricular failure, and individual chamber enlargement is not discernible[75] In acquired valvular disease and in many types of congenital heart disease, however, individual chamber enlargement develops and is crucial to plain film (and often clinical) diagnosis[70]. In

one study it was found that, CXR alone is not diagnostic of specific cardiac lesions, with a low accuracy of only 71%. It was therefore suggested that less emphasis should be placed on the use of radiographs alone in diagnosing specific congenital cardiac lesions[76].Plain films remain important, because they allow fairly straightforward and inexpensive assessment of current status as well as changes over time and are routinely and readily available.

2.13.4. The right ventricle: The classic signs of right ventricular enlargement are a boot-shaped heart and filling in of the retrosternal airspace.[77] The former is caused by transverse displacement of the apex of the right ventricle as it dilates. In adults, it is unusual for the right ventricle to dilate without left ventricular dilation, so this boot shape is not often obvious. It is most commonly seen as an isolated finding in congenital heart disease, classically in tetralogy of Fallot.As the right ventricle dilates, it expands superiorly as well as laterally and posteriorly, filling in the retrosternal airspace. For the right ventricle to be considered dilated, it is required that, the soft tissue density is confined to less than one third of the distance from the suprasternal notch to the tip of the xiphoid in a normal lateral chest radiograph. If the soft tissue fills in more than a third, in the absence of other explanations, it is a reliable indication of right ventricular enlargement.[71]

2.13.5. The left atrium: Signs which define left atrial enlargement include dilation of the left atrial appendage, which appears as a focal convexity where there is normally a concavity between the left main pulmonary artery and the left border of the left ventricle on the frontal view. Because of its location, as the left atrium enlarges, it elevates the left main stem bronchus. In so doing, it widens the angle of the carina. Third, as the left atrium enlarges posteriorly, it may cause focal bowing of the middle to low thoracic aorta toward the left. This bowing is distinguishable from the tortuosity seen with progressive atherosclerosis, which involves the descending thoracic aorta either in its upper portion or diffusely. Fourth, with marked left atrial enlargement, a double density can be seen on the frontal view, because the left atrium projects laterally toward the right as well as posteriorly, and the discrete outline of the blood-filled left atrium is surrounded by air-filled lung. Finally, on the lateral film, left atrial enlargement appears as a focal, posteriorly directed bulge.[78]

2.13.6. The left ventricle: Left ventricular enlargement is characterized by a prominent, downwardly directed contour of the apex, as distinguished from the transverse displacement seen with right ventricular enlargement. On the PA film, the overall cardiac contour is also usually enlarged, although this is a nonspecific finding. On the lateral film, left ventricular enlargement appears as a posterior bulge below the level of the mitral anulus.

2.14. OTHER INVESTIGATIONS USED FOR DIAGNOSIS OF HEART FAILURE

2.14.1. Plasma B-type natriuretic peptide: The natriuretic peptide system consists of five structurally similar peptides, termed ANP, urodilantin (an isoform of ANP), BNP, C-type natriuretic peptide (CNP), and Dendroaspis natriuretic peptide (DNP).ANP, a 28–amino acid peptide hormone, is produced principally in the cardiac atria, whereas BNP, a 32–amino acid peptide originally isolated from porcine brain, was later identified as a hormone that was primarily produced in the cardiac ventricles.Both ANP and BNP are secreted in response to increasing cardiac wall tension; however, other factors such as neurohormones (e.g., angiotensin II, ET-1) or physiological factors (e.g., age, gender, renal function) may also play a role in their regulation. The biosynthesis, secretion, and clearance of BNP differ from ANP, suggesting that these two natriuretic peptides have discrete physiological and pathophysiological roles. Whereas ANP is secreted in short bursts in response to acute changes in atrial pressure, the activation of BNP is regulated transcriptionally in response to chronic increases in atrial/ventricular pressure. ANP and BNP are initially synthesized as prohormones that are subsequently proteolytically cleaved, respectively, by corin and furin, to yield large biologically inactive N-terminal fragments (NT-ANP or NT-BNP) and smaller biologically active peptides (ANP or BNP).

B-type natriuretic peptide (BNP) is secreted by the heart, and the plasma concentration is elevated in left ventricular hypertrophy or dysfunction (systolic or diastolic), and particularly in those with heart failure. The biological importance of the natriuretic peptides in renal sodium handling has been demonstrated in multiple studies employing natriuretic peptide receptor antagonists, as well as overexpression of ANP or BNP.Several studies have confirmed its value as a rule-out test for heart failure in patients presenting with new symptoms in either primary or secondary care settings[79]. Raised plasma concentration can occur in other conditions, such as acute myocardial infarction, pulmonary embolism, and renal failure; and normal values are higher in the elderly and in women. The current European and North American heart failure guidelines suggest that the measurement of the plasma concentration of BNPs can be useful in confirming or refuting a diagnosis of heart failure, particularly at the time of first presentation in the acute setting.[80]

2.14.2. Magnetic Resonance Imaging: It provides high-resolution images of cardiac structure and ventricular function. Contrast agents such as gadolinium can provide information on inflammation, fibrosis and myocardial perfusion. Valve function can also be assessed, although with less reliability than myocardial structure and function. Although becoming main

stream in large hospitals, the expense of the equipment and its lack of portability make it of limited use in population-based studies.

2.14.3. Cardiac catheterization: Allows measurement of intracardiac pressures, estimation of cardiac output, detection of valve abnormalities, quantification of left ventricular ejection fraction, and the detection of epicardial coronary artery disease.

3. PROBLEM STATEMENT AND RATIONALE:

Although cardiovascular disease, and its major component heart disease, exists in epidemic proportions in western developed countries, it is also an increasing problem in the developing world contributing to significant morbidity and premature mortality in vulnerable population.[3] The overall burden of cardiovascular disease is predicted to rise by approximately 150% in the developing world within the next 20 years. In Africa alone, it is predicted that it will affect 1.3 million people per annum during this period.[3] With increased affluency and changes in life style, the major causes of death have shifted from infectious diseases and nutritional deficiencies to more chronic disorders: a phenomenon with distinct stages known as "epidemiologic transition.[3]Most of the published data on heart failure however is based on work in Caucasian populations within the developed world, and data on its incidence, prevalence, etiology, treatment, and outcome in Africa are lacking.[1]

Early and timely diagnosis of heart failure is of paramount importance for timely treatment and therefore reducing morbidity and mortality associated with heart failure. Good clinical assessment and appropriate diagnostic tests (Echo, ECG and chest radiography) are important in achieving this goal. These tools are easily available and affordable by the majority of patients in developing countries.

It is therefore relevant to describe patients with heart failure using parameters and important diagnostic tools available at MNH, Tanzania.

4. RESEARCH QUESTIONS:

1. What are the clinical and demographic characteristics of patients with severe Heart failure?
2. What are the Echocardiographic characteristics of patients with severe heart failure?
3. What are the Electrocardiographic characteristics of patients with severe heart failure?
4. What are the Chest radiographic characteristics of patients with severe heart failure?

5. BROAD OBJECTIVE:

To describe the clinical, echocardiographic, electrocardiographic and chest radiographic characteristics of heart failure patients at Muhimbili National Hospital

Specific objectives

1. To describe the demographic characteristics of heart failure patients at MNH.
2. To describe the clinical characteristics of heart failure patients at MNH
3. To describe echocardiographic characteristics of heart failure patients at MNH
4. To describe electrocardiographic findings of heart failure patients at MNH
5. To describe chest radiographic findings of heart failure patients at MNH

6. MATERIALS AND METHODS

6.1. Study Design: Descriptive cross-sectional study.

6.2. Study Setting: Medical wards, Muhimbili National Hospital, Dar es Salaam.

Dar es Salaam is a city with population of 3,500,000 estimated 2003. It has three districts, each with a municipal hospital. Muhimbili National Hospital (MNH) is a tertiary referral and University teaching hospital.

It also serves as a regional hospital for Ilala,Temeke and Kinondoni municipality hospitals and serves about 1000 outpatients per day and admission bed occupancy of about 1500 patients a week. On average about 200 patients are admitted on medical wards per week.MNH admits patients above 7years onto medical wards.

6.3. Study Population: All patients admitted in the medical wards with NYHAIII/IV heart failure

6.4. Study Period: The study was carried out for the period three months, from December, 2008 to February, 2009.

6.5. Study Subjects: All patients who were admitted in the medical wards with a primary diagnosis of heart failure NYHAIII/IV due to any cause.

Clinical Diagnosis of congestive heart failure was based on Framingham Criteria for Congestive Heart Failure and functionally assessed by NYHA Functional Classification for Congestive Heart Failure. Eligible patients in NYHA class III and IV were recruited for this study.

6.5. Inclusion Criteria: All patients with age above seven years admitted in the medical wards.

6.6. Exclusion criteria:

Patients who did not give consent to participate in the study

Patient known to have history of asthma or chronic obstructive pulmonary Disease (COPD)

6.7. Enrolment of subjects:

Consecutive enrolment of all eligible study subjects during the study period.

6.8. Sample size:

One hundred (100) was an estimated number of eligible study subjects based on availability of patients and time given for the study. At the MNH echo room, three days are available for adult echocardiograms; three patients were investigated per day, making 9 patients in one week, and 36 patients per month. Therefore 100patients were recruited at the end of three months.

6.9. Study Procedures: The investigator visited each admitting medical ward everyday to identify those suspected to be in Heart Failure. All the files of patients admitted overnight were reviewed by the author, detailed history was obtained, complete cardiovascular examination done and patients with features of heart failure in NYHAIII and IV were considered eligible for recruitment into study. All patients had ECHO, ECG and chest radiography as part of their diagnostic work up. All the information on history, physical findings and investigations of patients found to have NYHAIII and IV was entered in structured questionnaire.

Complete standard cardiovascular physical examination was done. Blood pressure was taken with a mercury sphygmomanometer and blood pressure of $\geq$140/90mm of mercury was taken

as hypertension, Normal value for blood pressure was taken as 120-139/80-89mmHg.The investigator supervised by a senior cardiologist at Muhimbili National Hospital performed Echoes on all recruited participants. Echoes were done using a PHILIPS, HP 7550 Echocardiography machine with a 3.0MHz transducer.

Standard Transthoracic Echocardiography was performed with the patient at rest.

Two dimensional (2D), MMode and Doppler modalities were used to study cardiac anatomy, and to evaluate haemodynamic status. The cardiac chamber sizes, LVEF were measured using MMode,and compared to the standard reference as per American College of Echocardiography reference Values.

Resting, standard12 lead Electrocardiograms were obtained in all patients with patients in supine position, in a quite room, using PHILIPS machine (Pager, Trim III).Recording was done with standard ECG paper at a speed of 25mm/sec. and calibration 10mm/1mV standardization. The ECG findings were interpreted by the author and reviewed and approved by senior Cardiologist. Sokolow- Lynon and Cornell Voltage criteria were used for diagnosis of LVH (specificity 10-30% and specificity 85-95%).

The standard Postero anterior (PA) view of chest radiography was taken with patient standing and holding breath at maximum inspiratory effort. The films were interpreted by discussion between the author and senior diagnostic radiologist, who approved the findings. Cardiothoracic ratio (CTR) of more than 50% was defined as cardiomegaly.Other findings pertinent to cardiac failure included interstitial oedema, alveolar oedema and pleural effusion.

6.10. Data entry, cleaning and analysis: Information obtained was collected using questionnaires. All questionnaires were daily checked for completeness and consistencies. Then data was entered into computer using SPSS version 10 for further cleaning, categorizing of continuous variables and eventually analysed.

7. ETHICAL CONSIDERATION: The study received ethical clearance from the Research and Publication committee of MUHAS and the Ethics department of MNH. Patients were included after informed verbal and written consent.

For patients under 18years, consent was obtained from their parents or guardians. All patients who participated in this study were regularly reviewed by the author, given treatment in the wards and later followed in cardiac clinic. In case more expertise was needed on treatment of these patients, a senior cardiologist was consulted. Patients therefore had the benefit of having best medical care, however, there was no any financial gain obtained by participating in this study.

8. RESULTS:

One hundred consecutive patients with NYHA class III and IV were studied over a period of three months. There were 54(54%) males, and 46(46%) females, their mean age 42±20.73years.Females were younger than males. The mean age for females was35±16.7years and males had mean age of 43±19.6years.

The clinical, echocardiographic, electrocardiographic and chest radiographic characteristics of these patients were evaluated and analyzed. Many patients (58%) were not on any medication and 95% had been admitted at most three times for heart failure (Table4)

Table4: Baseline demographic Data (n=100)

Patients Characteristics	Numbers (%)
Age (years)	
Range	9-87
Mean (Total)	42±20.73
Mean (Males)	43±19.6
Mean (Females)	35±16.7
Sex	
Males	54(54%)
Females	46(46%)
NYHA class	
III	54(54%)
IV	46(46%)
Treatment Category	
On Treatment	42(42%)
Not on Treatment	58(58%)
Blood pressure	
Normotensive	57(57%)
Hypertensive	37(37%)
Hypotensive	9(9%)
Number of admissions for Heart Failure	
Up to three times	95(95%)
More than three times	5(5%)

All patients in this study had PND and Positive hepatojugular reflux, elevated jugular venous pressure, tender hepatomegaly and nocturnal cough accounted for most frequent clinical features in these patients. (Table5)

Table5: Clinical Characteristics of patients at admission (n=100)

Clinical entity	Numbers (%)
Paroxysmal Nocturnal Dyspnoea	100(100)
Hepatojugular Reflux	98(98)
Elevated JVP	95(95)
Pulmonary rales	52(52)
Third (S3) gallop	32(32)
Tender Hepatomegally	98(98)
Nocturnal Cough	93(93)
Bilateral ankle oedema	68(68)
Displaced Apex	67(67)
Tachycardia (HR≥100/min)	59(59)
Cardiac Murmurs	36(36)
Ascitis	25(25)
Cyanosis	1(1)

Dilated Cardiomyopathy was the most frequent echocardiographic diagnosis (28%), chronic rheumatic valvular disease was the second most frequent diagnosis (23%), left ventricular hypertrophy without chamber LV dilatation found in 19% of patients and Hypertensive dilated cardiomyopathy was in 18% of these patients.

Fifty four patients (54%) had significant LV systolic dysfunction (LVEF<44%), including severe and moderate LV systolic dysfunction and 9% had LV diastolic dysfunction. Pulmonary Arterial Hypertension was found in 39% of the patients; Out of them, 32(82%) had mild and 7(18%) had moderate hypertension. None of them had severe pulmonary arterial hypertension

The most frequently dilated chambers were the LV and LA. (Table6)

Table6: Echocardiographic Findings of patients with heart failure (n=100)

Echo Findings	Numbers (%)
Echocardiographic diagnosis	
Dilated cardiomyopathy	28(28)
Chronic Rheumatic Valvular Disease	23(23)
Left Ventricular Hypertrophy	19(19)
Hypertensive Dilated Cardiomyopathy	18(18)
CHD (VSD, ASD)	5(5)
Pericardial Effusion (Pericarditis)	4(4)
Hypertrophic Cardiomyopathy	2(2)
Ebstein's anomaly of Tricuspid Valve	1(1)
LV ejection Fraction (%)	
<30(Severely abnormal)	16(16)
30-44(Moderately abnormal)	38(38)
44-54(Mildly abnormal)	37(37)
>55(Normal)	9(9)
Diastolic Dysfunction (grade I)	9(9)
Pulmonary Arterial Hypertension	39(39)
Mild Pulmonary arterial Hypertension	32(82)
Moderate Pulmonary arterial Hypertension	7(18)
Cardiac Chamber Dilatation	
LA	86(86)
LV	65(65)
RA	52(52)
RV	40(40)

Sinus tachycardia was the most frequent Arrhythmias (47%), of patients with atrial abnormalities,left atrial abnormalities accounted for (42%),22% of patients had BBB,and 46%

had LVH with ST-T changes. Majority (76%) of patients had more than one ECG abnormalities (Table7)

Table7: Electrocardiographic findings of heart failure patients (n=100)

ECG findings	Number (%)
Rhythm abnormalities	
Sinus tachycardia	47(47%)
Ventricular extrasystole	16(16%)
Atrial fibrillation	7(7%)
Atrial Flutter	2(2%)
Supraventricular Tachycardia	1(1%)
Atrial Abnormalities	
Left Atrial abnormality	42(42%)
Right atrial abnormality	5(5%)
Bi atrial Abnormalities	5(5%)
Conduction abnormalities	
LLLB	11(11%)
RBBB	11(11%)
First degree AV Block	7(7%)
Ventricular Changes	
LVH with ST-T changes (Strain)	46(46%)
Left axis deviation	11(11%)
Right Ventricular hypertrophy	2(2%)
Right axis deviation	3 (3%)
Other findings	
More than one ECG findings	76(76%)
Single ECG abnormality	23(23%)
T changes	7(7%)
Normal ECG findings	1(1%)
Ischemic changes (Q wave infarction)	1(1%)

Chest radiographic films were available for 86 patients and among them, cardiomegaly was the most common finding accounting for 87%.Of all patients, 76.5% had interstitial oedema of varying severity and 12% had pleural effusion (Table8)

Table8: Chest radiographic findings of heart failure patients (n=86)

Radiographic finding	Numbers (%)
Cardiomegaly	75(87)
Interstitial oedema	
Normal (no congestion)	21(24.4)
Mild	42(48.8)
Moderate	21(24.4)
Severe	2(2.3)
Pleural Effusion	
Right sided	10(11.6)
Left sided	2(2.3)
Mild	9(75)
Moderate	2(17)
Severe	1(8)

9. DISCUSSION

Heart failure is a major public health problem in developed countries imposing a huge burden in their health systems. Unlike developing countries the major cause of HF in developed countries is due to ischaemic heart diseases and its complications.

Published Data on incidence, prevalence, aetiology, treatment and outcome of heart failure is scanty in Africa; however the underlying aetiology in Africa is rarely due to IHD.[1]

This study describes the clinical, echocardiographic, electrocardiographic and

Chest radiographic characteristics of patients admitted with NYHA class III and IV heart failure at a tertiary care centre in Tanzania. In this three months study, 100 patients who met Framingham clinical criteria for heart failure were recruited, clinically evaluated, standard transthoracic echocardiography perfomed, standard postero anterior chest radiography taken and standard 12 lead ECG recorded and analysed.

All patients had PND and Positive hepatojugular reflux accounted for 98%, elevated jugular venous pressure 95%, tender hepatomegaly 98%and nocturnal cough 93%.Other clinical features included bilateral ankle oedema which accounted for 68%, and displaced apex 67%, tachycardia 59%, pulmonary rales 52%, cardiac murmur 36%, Third (S3) gallop in32% and ascitis in 25% of patients. Only 1% had central cynosis.

In a study done by Desai et al in South Africa[12],Ninenty seven(97) patients with severe heart failure were followed over 4 years, the findings revealed a displaced hypodynamic apical impulse in 72% and gallop rhythm in 92% of the patients.

Functional mitral regurgitation was present in 43%. Additional symptoms and signs include dependent oedema, dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, persistent cough, abdominal discomfort secondary to passive congestion of the liver and other organs, precordial pain, and palpitations. There was only mention of other clinical findings like dependent oedema, dypnoea, and PND without indicating the percentages.

In this study, the precordial findings were almost similar to findings by Desai et al. However, other clinical findings were different from those by Desai et al.In his study; there were a very high percentage of patients with Third sound (S3) gallop while there were 32% with this finding in this study.

The differences in the findings in this study and that done by Desai could be explained by the difference in recruitment criteria used for recruitment of patients. While Framingham criteria was used to recruit patients of both genders regardless of the cause of heart failure,Desai selectively recruited women with heart failure due to Peripartum Cardiomyopathy.

These general clinical features (Exertional dyspnoea, Orthopnoea, Paroxysmal Nocturnal dyspnoea,history of oedema, Resting Heart rate, Rales, Third heart sound ,Jugular venous distension ,Oedema on examination)are useful for clinical evaluation of patients with heart failure; however suffer low sensitivities of below 30%, though their specificities are well above 70 %[37].The low sensitivities means that, using clinical symptoms and signs only leads to missing patients in heart failure, hence this calls for the importance of combining clinical features and investigations to increase the accuracy of diagnosing patients with heart failure.

Strictly, the use of Framingham criteria for heart failure improves the diagnostic accuracy with specificity of 100% and specificity of 78%.[38]

In this study, echocardiographic findings revealed that, (28%) of all patients had dilated cardiomyopathy, followed by chronic rheumatic valvular disease (23%), left ventricular hypertrophy without LV dilatation was found in 19% of patients and Hypertensive dilated cardiomyopathy was found in 18% of these patients.

The most frequently dilated chambers were the LA and LV (86% and 65% respectively).Ishaemic heart disease was not revealed in this group of patients.

Fifty four (54%) patients had significant LV systolic dysfunction (LVEF<44%) and 9% had LV diastolic dysfunction. Pulmonary Hypertension was found in 39% of the patients.

Oyoo and Ogola[9] conducted an echocardiography-based study of the etiology of heart failure in 91 consecutive patients who were admitted to the Kenyatta National Hospital in Nairobi, Kenya. The main etiology of heart failure was rheumatic heart disease (32%), followed by dilated cardiomyopathy (25%) and hypertensive heart Disease (17%).

The relative importance of these etiologies was similar for both genders.Ischemic heart diseases accounted for only 2% of cases, a proportion that is similar to other parts of Africa.[9]

In a study of 167 consecutive patients admitted to the Yaoundé General Hospital in Cameroon and evaluated by echocardiography, heart failure accounted for 5.8% of all hospital admissions, rehospitalization rate was 8.3%, and the overall in-hospital mortality was 9 %
Forty-four percent of patients were in New York Heart Association functional class III, and 7% were in class IV. Cardiac cavities were dilated, and left ventricular ejection fraction was low in patients with systolic (70%) and combined systolic and diastolic (20%) dysfunction.The Main aetiologies of heart failure were: Hypertension (49%), cardiomyopathies (34%) and valvular heart diseases (55%). Ischaemic heart disease was the fifth aetiology (39%)
The causes of heart failure were similar in frequency in this study and that done by Kingue, S. et al in Cameroon[14], with changes due to remodeling accounting for the highest frequency followed by chronic rheumatic valvular disease in both cases.
Ischaemic heart disease had insignificant contribution to the causes of heart failure in all these three studies.
The difference in causes of heart failure found by Ogola,E et al in Kenya[9]differ from findings in this study and that done by Kingue S et al could be explained by the time difference between these studies.The study by Ogola et al was done 10years ago, while that by Kingue was done 4years ago, and this study was done recently(one year ago). This could be due to a changing pattern in the causes of heart failure as Africa is experiencing an epidemiological transition from Rheumatic Valvular heart disease to diseases of change in life style and increased cardiovascular risk factors like systemic hypertension, diabetis and obesity, but it could also be due to Myocardiditis or HIV related cardiomyopathy.
The urbanization process, which is happening at a rapid pace in Africa has enormously increased the prevalence of cardiovascular risk factors, mainly hypertension, diabetes, and obesity, paving the way for the substrate to a new epidemic in the future, the ischemic heart disease.[11,12]
In this study, 73% of all patients had Rhythm abnormalities with sinus tachycardia the most frequent Arrhythmias (47%) and atrial fibrillation accounted for 7% of all patients.
Fifty two (52%) percent had atrial abnormalities, with left atrial abnormalities accounting for (42%), and bundle branch block accounted for 22% of all patients with LLB and RBB

accounting for equal proportions.And of all patients, 46%had LVH with ST-T changes, 1% of patients had normal ECG findings and another 1% had ischaemic changes. Majority (76%) of patients had more than one abnormal ECG findings.In Epidémiologie Francaise de Insuffisance Cardiaque Aiguë (EFICA)[53]only 13 percent of ECGs were normal, with ischemic changes evident in 29 percent and atrial fibrillation in 25 percent.

Another study by Desai et al [12] found Left ventricular hypertrophy by electrocardiographic voltage criteria was present in 66% and ST-T wave abnormalities in 96% of the patients. In both cases, the proportion of patients with normal ECG findings is low, confirming the fact that a normal ECG almost completely excludes the diagnosis of heart failure[51]. LVH by Voltage criteria contributed to singnificant proportion in this study and that by Desai.

The proportion of patients with atrial fibrillation was different between this study and that found in EFICA, the explanation for these differences could be due to differences in the selection criteria used in recruiting study participants, differences in type and number of heart failure medication used by these patients and NYHA classes of these patients.

In this study, 86% of all patients had chest radiography taken, cardiomegaly was the most common chest radiographic finding accounting for 87%.Almost 76% of all patients had pulmonary congestion, (2% severe, 24% moderate, and 49% mild) and 12 had pleural effusion.

In Acute Decompensated Heart Failure National Registry (ADHERE) Chest radiography was performed in more than 90 percent of patients in and there was evidence of congestion in more than 80 percent of these patients.[66] Studies have also shown that agreement among clinicians in chest x-ray interpretation is moderate to almost perfect for interstitial edema, but only moderate for cardiomegaly and vascular redistribution.[70]Both these studies show significantly high number of patients with pulmonary congestion although the ADHERE study did not show the proportion of patients with cardiomegaly and pleural effusion .How ever, pulmonary oedema supports a diagnosis of heart failure, although the reliability of identifying upper lobe venous blood diversion is poor and Cardiothoracic ratio is only of moderate value in identifying heart failure as the cause of breathlessness.[65] The main role for a chest radiograph therefore is to exclude other causes for dyspnoea such as pleural effusion, pneumothorax, lung

carcinoma, or pneumonia and should be considered a standard component of the initial assessment of a patient with presumed HF. The presence of pulmonary venous congestion, interstitial edema, alveolar edema, and cardiomegaly increase the likelihood of HF[67].Although most patients have cardiomegaly, the absence of pulmonary venous congestion or edema should not exclude the diagnosis of HF.[68]Chest radiographs should be part of the initial evaluation of a patient with suspected HF, but absence of findings should not exclude the diagnosis. Each of the four diagnostic modalities, clinical evaluation, Echo, ECG and Chest suffer varying levels of sensitivities and specificities. To improve the diagnostic accuracy therefore, they should be used in combination to supplement each other.

10. Conclusions:

1. PND was a common symptom to all the patients. Other most frequently occurring features included positive hepatojugular reflux, Elevated Jugular Venous pressure, tender hepatomegaly and nocturnal cough.
2. The most common echocardiographic finding of patients in this study was dilated cardiomyopathy followed by chronic rheumatic valvular disease, left ventricular hypertrophy and hypertensive dilated cardiomyopathy.
3. Left atrial dilatation was the most frequently dilated cardiac chamber, confirmed by both the ECG and Echo
4. Males were older (mean age 43±19.6years) than females (mean age35±16.7years)

11. Limitations of the study:

1. In this study, biochemical markers such as Beta type Natriuretic Peptide (BNP) were not used for diagnosis of heart failure due to financial constraints. This marker is currently suggested by the European and North American heart failure guidelines in confirming or refuting a diagnosis of heart failure, particularly at the time of first presentation in the acute setting.
2. Chest radiography was taken only in PA view, which had limitations in defining individual cardiac chamber enlargement and other findings.
3. Tissue Doppler imaging was not done in this study to classify different stages of diastolic dysfunction. Only grade one diastolic dysfunction was studied by continuous wave Doppler.

12. Recommendations:

1. A much larger study which uses biochemical marker to improve the diagnostic accuracy of heart failure.
2. Treatment outcome study would be advisable. Longer time is necessary to follow up these patients.

Appendix1

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