

**Relation between severity of left ventricular  
systolic dysfunction and repolarisation**

**abnormalities on the surface ECG**

**Thesis**

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# List of Abbreviations

ABMMNCs.....	Autologous Bone marrow derived mononuclear cells.
ACC.....	American College of cardiology.
ACCP .....	American College of Chest Physicians.
ACE.....	Angiotensin converting enzyme.
ADHERE.....	Acute Decompensated Heart Failure National Registry.
AF.....	Atrial fibrillation.
AHA.....	American Heart Association.
AHF.....	Acute heart failure.
ARB .....	Angiotensin receptor blocker.
ATP.....	Adenosine tri-phosphate.
AV .....	Atrio-ventricular.
BNP .....	Beta type Natriuretic peptides.
Ca .....	Calcium.
CAD.....	Coronary artery disease.
CARE-HF.....	Cardiac Resynchronization in Heart failure.



cGMP.....Cyclic guanine monophosphate.

CHF..... Chronic heart failure.

COMPANION..... Comparison of Medical Therapy, Pacing, and  
Defibrillation in Heart Failure.

CRT.....Cardiac resynchronization therapy.

CSCs.....Cardiac stem cells.

CT.....Computed tomography.

DCM.....Dilated cardiomyopathy.

DHF.....Decompensated heart failure.

DM .....Diabetes mellitus.

DTI .....Tissue Doppler imaging.

ECG ..... Electrocardiogram.

ED.....Erectile dysfunction.

EF.....Ejection fraction.

EMMs..... Electromechanical maps.

ES cells.....Embryonic stem cells .

ESC..... European Society of Cardiology.

FDA .....Food and drug association

HF.....Heart failure.

HFSA .....Heart Failure Society of America.

HMG-CoA..... $\gamma$ -hydroxy- $\gamma$ -methylglutaryl coenzyme A.

HsCRP.....High sensitivity C-reactive protein.  
 HT .....Heart transplant.  
 HZ.....Hertz.  
 ICD .....Implantable cardioverter defibrillator.  
 INR .....International normalization ratio.  
 IV ..... Intravenous.  
 K.....Kalium(Potassium).  
 LQT.....Long QT.  
 LQTS .....Long QT Syndrome  
 LV .....Left ventricle.  
 LVEF.....Left ventricular ejection fraction.  
 LVH .....Left ventricular hypertrophy.  
 LVOT.....Left ventricle outflow tract.  
 LVSD.....Left ventricular systolic dysfunction.  
 MgSO<sub>4</sub> ..... Magnesium sulphate.  
 MI .....Myocardial infarction.  
 MIRACLE .....Multicenter InSync Randomized Clinical Evaluation .  
 MRI.....Magnetic resonance imaging .  
 MSCT.....Multi-slice computed tomography.

Na .....Natrium(Sodium).

NYHA.....New York heart association.

PDE- $\alpha$  .....Phosphodiesterase type –  $\alpha$ .

PH.....Pulmonary hypertension.

QRSd.....QRS complex duration.

QTc.....The heart rate– corrected QT.

RAPID-CHF..... Randomized Controlled Trial of Ultrafiltration for  
Decompensated Congestive Heart Failure.

RV ..... Right ventricle

SAVE .....Survival and ventricular enlargement.

SBP.....Systolic blood pressure .

SCD.....Sudden cardiac death.

SR.....Sarcoplasmic reticulum.

SR.....Sinus rhythm.

Tp–Te .....Peak to the end of the T wave.

TVI.....Time velocity integral.

WASH.....Warfarin/Aspirin Study in Heart Failure.

# Introduction

Heart failure is primarily a condition of the elderly, and thus the widely recognized “aging of the population” also contributes to the increasing incidence of HF. The incidence of HF approaches 10 per 1000 population after age 60, and approximately 80% of patients hospitalized with HF are more than 60 years old. (*Masoudi FA, et al. 2002*)

The development of HF can be appropriately characterized by considering 4 stages of the disease, This staging system recognizes that HF, like coronary artery disease, has established risk factors and structural prerequisites; that the development of HF has asymptomatic and symptomatic phases; and that specific treatments targeted at each stage can reduce the morbidity and mortality of HF. (*Hunt SA ,et al. 2005*)

Transthoracic two-dimensional and Doppler echocardiography is one of the most important and frequently performed diagnostic procedures for patients with cardiovascular disease. It provides highly accurate diagnostic information regarding the anatomy and physiology of the cardiac chambers, valves, major vessels,

and pericardium in a non-invasive and instantaneous manner. This information can immediately affect the further diagnostic work-up for the patient, dictate therapeutic decisions, determine response to therapy, and predict patient outcome. Because transthoracic two-dimensional/ Doppler echocardiography plays such a major role in the care of patients with suspected or known cardiovascular diseases. (Mark A, et al. ۲۰۰۲)

The long QT syndrome (LQTS) is a genetic cardiac channelopathy in which most affected individuals have delayed ventricular repolarization manifest with prolongation of the corrected QT (QTc) interval on the electrocardiogram. (Andrew J, et al. ۲۰۰۷)

As for the QT interval measurement from the surface ECG, there are two parts that need to be detected, i.e. QRS onset and T wave offset. The former is usually a less difficult task due to relative sharp deflection change in QRS onset in most cases, which also corresponds to a sharp rise of the action potential in the cardiac muscle cells at the beginning of the depolarization. Whereas, the T wave offset measurement is much more difficult in most cases. (Willemis JL, ۱۹۸۷)

## **Aim of the work**

The aim of the present study is to evaluate the relation between severity of left ventricular systolic dysfunction assessed by echocardiography and repolarisation abnormalities (duration of QTc interval , duration of JTC interval and prolongation of QRS complex) on the surface ECG.

## **Pathophysiology**

Progression of heart failure is viewed as resulting from the interplay of haemodynamic and neurohormonal mechanisms. Both mechanisms support the inotropic state of the heart following an injury to the myocardium, but when sustained for long periods, their ability to augment cardiac contractility wanes, and, instead, these same mechanisms act to enhance ventricular wall stress, thereby impairing ventricular performance. As the heart-failure state evolves, endogenous mechanisms that are normally activated to control wall stress become exhausted, and peripheral vasoconstriction and sodium retention develop. Unopposed activation of haemodynamic stresses and neurohormonal systems leads to further destruction of the myocardium and progression of the underlying disease.

*(Packer M. 1992)*