"A COMPARATIVE STUDY OF MYOCARDIAL PERFORMANCE INDEX WITH CONVENTIONAL ECHO CARDIOGRAPHIC PARAMETERS OF LEFT VENTRICULAR FUNCTION IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION PATIENTS & ITS ROLE IN PREDICTING INHOSPITAL MORBIDITY"

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CERTIFICATE

This is to certify that the dissertation entitled

" A COMPARATIVE STUDY OF MYOCARDIAL PERFORMANCE INDEX WITH CONVENTIONAL ECHOCARDIOGRAPHIC PARAMETERS OF LEFT VENTRICULAR FUNCTION IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION PATIENTS & ITS ROLE IN PREDICTING INHOSPITAL MORBIDITY" is the bonafide original work of Dr.P.M.NAGESWARAN, in partial fulfillment of the requirements for D.M branch-II (CARDIOLOGY) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in February 2006.

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CONTENTS

INTRODUCTION

Echocardiography is the most readily available and commonly used non invasive diagnostic tool in cardiology, especially for the assessment of LV function. Cardio vascular disease is the second most common cause of death after communicable diseases in India. Coronary heart diseases accounts for more than 50% of cardiovascular disease deaths. STEMI occurrence is a fatal event in approximately 20 to 30% of patients. Nearly one third of death occurs within one hour are mainly due to ventricular arrhythmias. But the late mortality is mainly depended on LV function. Hence LV function assessment is an integral part of any patient with acute STEMI.

LV function derangement can affect systolic function, diastolic function or both. Two – dimensional echocardiography is well suited for studies of systolic function, and Doppler echocardiography provides a noninvasive technique for the assessment of diastolic function. However these measurements are load-dependent and change with the location of the sample volume, rhythm, heart rate and quality of the image. Even though systolic and diastolic dysfunction often coexists, only a few Doppler echocardiography variables combine measurements of systolic and diastolic performance.

Recently, a new echocardiographic index combining the measurements of diastolic and systolic performance was defined. It was proposed first by Tei Chuwa et al in 1995. In its short life span, it has been shown to demonstrate powerful prognostic value in significant heart diseases such as dilated cardiomyopathy, idiopathic pulmonary hypertension, cardiac amyloidosis and recently in myocardial infarction. TEI index has also got various other advantages compared to classical 2D and Doppler parameters such as not being influenced by changes in blood pressure, heart rate, sex and age and not appearing to be affected significantly by loading condition.

AIMS

- To assess the role of myocardial performance index in Acute STEMI patients.
- To evaluate the relationship between myocardial performance index and area of infarct.
- To assess the relationship between myocardial performance index and systolic, diastolic dysfunction in Acute STEMI patients.
- To evaluate the relationship of Myocardial performance index with clinical parameters.
- To assess the Prognostic value of Myocardial performance index in In hospital morbidity.
- To find out, Is there any correlation between Ejection fraction derived from Myocardial performance index by Lax et al formula and that obtained by conventional Method (modified Simpson's method)?

REVIEW OF LITERATURE

MYOCARDIAL INFARCTION- AN OVERVIEW

Despite impressive improvement in diagnosis and management over the last four decades STEMI continues to be a major public health problem in the industrialized as well as developing economics like India. Our country is in the midst of epidemic of Diabetes and its resultant cardiovascular complications mainly due to improving socioeconomic status, westernization, changing lifestyles, obesity, stress and high incidence of insulin resistance seen in our population.

The diagnosis of Myocardial infarction requires presence of at least two of the following; characteristic symptoms, electro cardio graphic changes and a typical rise and fall in biochemical markers. The pathological hallmark of Myocardial infarction is coronary atherosclerosis with superimposed occlusive thrombus.

Identification and management of risk factors are essential for preventing coronary heart disease in a symptomatic individual and for preventing recurrent events in patients with established disease. The efficacy of secondary prevention of CHD has been well established and this generated enthusiasm for extending the same to primary prevention. The key parameter for risk assessment is defining the absolute risk, i.e., the probability of developing CHD over a time period.

Absolute risk can be divided in to high, intermediate, and low risk categories based on 10 year absolute risk of developing myocardial infarction as follows, high (> 20%), intermediate (10-20%), low (<10%) risk. high risk patients are considered as coronary artery disease equivalent and it includes patient with non coronary form of clinical atherosclerotic disease, type 2 diabetes mellitus, symptomatic patients with presence of multiple risk factors other than DM.

ASSESSMENT OF NORMAL CARDIAC FUNCTION

The study of cardiac function has progressed from a description of cardiac anatomy to quantifying physiology and to the unraveling of the molecular pathways. Assessment of cardiac function is necessary for determining the diagnosis, for prognostication, timing of intervention, assessment of therapy, detecting complication and for assessing clinical outcomes.

LEFTVENTRICULAR SYSTOLIC FUNCTION

The fundamental task of the cardiovascular system is to supply adequate quantities of oxygenated blood to the peripheral tissue. An important determinant of cardiac performance is left ventricular systolic function, which in turn is determined by preload, after load, myocardial contractility and heart rate.

The surrogate markers for the preload are ventricular end diastolic volume, end diastolic diameter and end diastolic pressure. Quantifying afterload in the intact circulation is more challenging one. Two approaches are used, one focuses on the vascular load, which is measured by peripheral vascular resistance. The next approach focuses on the development of tension in the ventricular wall. Contractility manifest in the intact circulation at the rate of pressure development and of shortening from any given preload. Measures of systolic function and contractility are often considered together and include stroke volume, ejection fraction, the maximum rate of pressure increases during isovolumic contraction, and a variety of more sophisticated measurements such as end systolic volume relationship, velocity of circumferential fiber shortening(VCF),after load corrected(VCF).

There are several indices of global left ventricular systolic function and contractility. Each index is variably dependent on preload and after load and can be modified by ventricular volume and myocardial mass. The ease of application to the clinical setting is

an important feature.

EJECTION FRACTION

The ejection fraction is defined as the ratio of stroke volume to end-diastolic volume. It is computed as follows:

EF=EDV-ESV/EDV*100(%)

Normal values of LVEF are 0.55 to 0.75 when determined by angiocardiography and echocardiography, but may be lower when determined by radionuclide angiography (0.50 to 0.65). There are no gender differences, but ejection fraction normally declines with age. An acute increase in after load, such as occurs during acute pressure loading, may decrease ejection fraction to 0.45 or 0.50 in normal subjects. However a reduction of LVEF below 0.45 indicates impaired myocardial function, independent of loading conditions.

The widespread utility of the ejection fraction in clinical practice is a result of multiple factors including the conceptual simplicity of its derivation, the ability to determine it easily and reproducibly, using a variety of different imaging techniques, and an extensive documentation of its clinical utility. This parameter has been shown to be of great prognostic value both short-term and long-term in patients with a variety of heart diseases. However, its limitation is its dependency not only on myocardial contractility but also on preload and after load, as well as heart rate and synchronicity of contraction. Therefore, it measures much more than contractility.

END-SYSTOLIC VENTRICULAR VOLUME OR DIMENSION

The clinical utility of end-systolic volume or dimension is its relative independence of preload. Although strongly after load dependent, it is of particular value in assessing left ventricular function in patients with valular regurgitation.

VELOCITY OF CIRCUMFERENTIAL FIBER SHORTENING (VCF)

This ejection phase index of systolic function has been used primarily in research studies. Typically, the changes in the left ventricular volume or circumference during systole (corrected to the end-diastolic value) are divided by ejection time to yield the mean velocity of ejection or fiber shortening.

AFTERLOAD -CORRECTED VCF

Since ejection phase indices such as VCF are highly after load dependent, they become more accurate indicators of ventricular function by correcting with some measure of after load. Using stress instead of pressure makes further correction for left ventricular geometry. There are several methods for calculating wall stress; the most common model in use is that of Sandler and Dodge. Patients with reduced left ventricular contractility have a downward shift of the systolic shortening-mean systolic wall stress relationship.

SLOPE OF END-SYSTOLIC PRESSURE-VOLUME RELATIONSHIP (END SYSTOLIC ELASTANCE)

The most reliable index for assessing myocardial contractility in the intact circulation is the ESPVR, which is almost insensitive to changes in preload, after load, and heart rate. This is widely used in animal studies and occasionally clinically. This relationship can be determined from instantaneous end-systolic pressure-volume coordinates from different cardiac contractions at varying preload and afterload conditions. The slope of this relationship represents the end-systolic elastance, which is a sensitive parameter for assessing myocardial contractility.

Assessment of elastance is difficult under clinical conditions, because it requires simultaneous pressure-volume relations, as well as changes in preload or afterload for construction of the ESPVR. However, this measurement is facilitated by the use of techniques

such as radionuclide angiographies or the use of conductance catheters, which allow continuous measurements of left ventricular volume while left ventricular volume is changed, e.g., by caval occlusion.

END- SYSTLIC STIFFNESS

This index of contractile function is derived from the exponential constant of the end systolic relation between wall stress and the natural logarithm of the reciprocal of wall thickness.

PRELOAD RECRUITABLE STROKE WORK

The relation between left ventricular stroke work and left ventricular end diastolic volume is a representation of systolic function. Like the end-systolic elastance, it is difficult to assess this parameter in patients because preload needs to be varied to generate this parameter.

MAXIMUM RATE OF PRESSURE RISE

The maximal rate of ventricular pressure rise (maximum dP/dt) is analogous to the maximal rate of tension development of isolated cardiac muscle, a well-established index of myocardial contractility. Maximum dP/dt is dependent not only on left ventricular contractility but also on heart rate, preload, after load, synchronicity of contraction, and myocardial hypertrophy. Since this isovolumic index of systolic function is preload dependent, the relationship between ventricular end-diastolic volume and dP/dt is a more accurate index of contractility than dP/dt alone.

REGIONAL INDICES OF LEFT VENTRICULAR FUNCTION

Global measures of left ventricular function, such as those described earlier, loose accuracy when the disease process affects regions of the ventricle differentially; this occurs most notably in coronary artery disease. Chronic myocardial ischemia, i.e., stunning or hibernation, and acute or old myocardial infarction may be associated with regional wall motion abnormalities with hypokinesis (reduced shortening) and akinesis (absent shortening)

or dyskinesis (systolic elongation or bulging). Often the nonischemic portion of the ventricle is hypercontractile, leading to a normal global left ventricular function in the presence of impairment of regional function.

The most widely used technique for assessing regional wall motion is the centerline method, which demonstrates that shortening at the apex normally is less than at the base. For normalization, systolic shortening may be divided by ejection time. These measures of endocardial motion and changes in wall thickness are after load dependent but do allow comparisons of different regions of the ventricle.

Diastolic Function

Diastolic function has been found to play an important role in cardiac morbidity and mortality and to influence both preload and after load. Diastole begins with the isovolumic relaxation period that starts after aortic valve closure. However, from a clinical standpoint, four separate phases of diastole need to be distinguished:

1) Iso volumic relaxation 2) early diastolic filling 3) slow ventricular filling (diastasis)

4) Atrial filling.

Diastolic function is influenced by several factors e.g. myocardial relaxation, ventricular filling, and the ventricle's passive elastic properties, but one of the major determinants is heart rate, which determines how much time is available for ventricular filling. An increase in heart rate shortens the diastolic filling time interval disproportionately. This reduction must be compensated by an increase in the rate of relaxation and an augmentation of elastic recoil with enhanced diastolic suction.

In the normal left ventricle, the end-systolic volume is smaller than its elastic equilibrium and it thus generates elastic recoil, which varies inversely with the end-systolic volume .The elastic recoil causes diastolic suction that fills the ventricle at a low pressure and induces a potential for negative left ventricular pressure in early diastole. This filling mechanism is important during exercise and allows the normal ventricle to reduce minimal diastolic pressure and to maintain end diastolic pressure constant despite a 3 fold to 5 fold

increase in cardiac output. A loss of elastic recoil occurs during acute ischemia with reduction in early diastolic filling accompanied by an increase in left atrial filling pressure and heart rate.

Another important determinant of diastolic function is the atrioventricular pressure gradient, which is dependent on atrial pressure, relaxation rate, viscous forces in the myocardium, and ventricular filling rates.

Abnormalities of Diastolic function

Diastolic abnormalities are characterized by abnormal filling indices, are commonly identified by echocardiography, and known to have reduced early diastolic filling rate or a prolonged isovolumic relaxation period, but without clinical symptoms. In this situation the ventricle is able to maintain a normal level of left ventricular filling pressure. Diastolic dysfunction is characterized by an increase in diastolic filling pressure, which may be responsible for the occurrence of dyspnea. This symptom may occur during exercise ("latent" diastolic dysfunction) or may be present also at rest ("manifest" diastolic function). Many patients with moderate to severe left ventricular hypertrophy may suffer from diastolic dysfunction. Diastolic heart failure is associated with the clinical signs of heart failure, such as paroxysmal nocturnal dyspnoea, orthopnea, and edema. The clinical differentiation between systolic and diastolic dysfunction is important, because prognosis and therapeutic interventions are different in these two forms of heart failure.

Relaxation

Isovolumic relaxation begins with aortic valve closure and ends with mitral valve opening. The important determinants of relaxation are maximal systolic pressure, end-systolic fiber stretch, coronary flow (erectile effect) and stored energy (elastic recoil). A delayed or

incomplete relaxation (>3.5 time constants) can retard and / or delay the onset of diastolic filling. In most patients with myocardial ischemia or systolic pump failure, both the rate of relaxation and elastic recoil are decreased in parallel, thereby elevating ventricular filling pressure.

The most commonly used parameter to evaluate left ventricular relaxation is the time constant of isovolumic pressure decay (τ (tau), expressed in milliseconds) In normal subjects, τ averages 48 milliseconds, with a range from 40 to 60 milliseconds. Relaxation is defined as being completed at 3.5 times the time constant τ after aortic valve closure.

Filling

In the normal ventricle there are two rapid filling phases: 1.the early diastolic phase from mitral valve opening to diastasis and 2.the atrial filling phase. Rapid diastolic filling is dependent on four mechanisms; 1.rate of relaxation 2.elastic ventricular recoil 3. Atrioventricular pressure gradient and 4. Passive elastic properties of the atrium and the ventricle. Assessment of diastolic filling can be obtained most conveniently from Doppler echocardiography, which allows an assessment of maximal filling velocities as well as the ratio between early (E wave) and late (A wave) filling velocity (E/A ratio). Late diastolic filling is dependent on the strength of left atrial contraction and the diastolic stiffness of the left ventricle.

Several indices of diastolic filling can be calculated, including the instantaneous filling rate, time to peak filling (PFR), the fraction of filling that occurs during the rapid filling phase, as well as acceralation and deceleration of early diastolic filling .The most rapid rate of filling occurs during the first half of diastole and is termed the early PFR.This index and the ratio of early to late diastolic filling, (PFR1/PFR2 or E/A ratio) are the most

commonly used parameters to describe left ventricular filling .

Passive Elastic Properties

It is necessary to distinguish between ventricular (chamber) and myocardial (muscle) properties. Ventricular stiffness is determined and defined by the pressure-volume relationship of the left ventricle and is directly related to clinical symptoms, whereas myocardial stiffness is determined by the stress-strain relationship of the structural composition of the myocardium. Ventricular stiffness reaches its nadir at the lower diastolic pressure but increases progressively during diastolic filling and is maximal at end- diastole. Myocardial stiffness impedes myocardial lengthening. Since wall stress and myocardial fiber length increase during diastolic filling, they reach maximal levels at end-diastole (i.e.; the preload), which according to the Frank-Starling principle is an important determinant of the extent of systolic myocardial fiber shortening.

Calculations of left ventricular chamber stiffness are carried out by plotting left ventricular diastolic filing pressure against left ventricular diastolic volume from the minimal diastolic pressure to the end diastolic pressure

ASSESSMENT OF SYSTOLIC FUNCTION - M MODE ECHOCARDIOGRAPHY

Measurement of the LV cavity dimension and wall thickness can be readily derived from M mode recordings and are usually made according to the recommendation of the American society of Echocardiography at end diastole and end systole. These measurements should be made from leading edge to leading edge to avoid incorporating artifacts and reverberation. They are accurate if the beam is orthogonal to the long axis of the ventricle.

The M-mode LV cavity dimension can be used to estimate **ventricular volumes** and

ejection fraction if desired, most simply by merely cubing (**teicholtz**) the value. But this calculation involves several assumptions regarding LV geometry that are not uniformly valid. In addition, M mode dimension may not be representative of the entire ventricle. The **fractional shortening** can also be measured. This value is often helpful in assessing systolic function, but it reflects the function of LV in one chord and in one plane and can be misleading with asynchronous contraction (for example LBBB) or segmental dyssynergy. An additional M-mode marker of systolic dysfunction is E point septal separation (**EPSS**), or the distance between the anterior MV leaflet at its most anterior opening excursion (E POINT), and the interventricular septum. A value of 8 mm or greater is abnormal.

ASSESSMENT OF SYSTOLIC FUNCTION BY 2D ECHOCARDIOGRAPHY

Since 2D-echocardiography enables visualization of the entire LV perimeter in multiple planes, it is significantly superior to M- mode approaches for the measurement of cardiac chamber volumes and EF. Numerous algorithms have been applied to calculate LV volumes by echocardiography. Most such algorithms have assumed that the LV conforms to the shape of a prolate ellipsoid and calculated volume by **diameter - length** or **area - length** formulas. Multiple studies comparing LV volume calculated by area length method to those obtained by other techniques have yielded good correlation, with best results obtained utilizing biplane apical views. Other algorithms have assumed an LV cavity configuration that is a combination of geometric shapes, such as cylinder - cone or a cylinder - hemi ellipse. Currently the most commonly used algorithms to calculate LV volume is based upon the **SIMPSON** rule, which derives measurements by dividing the LV by parallel planes into a number of small segments and then summating the area of the individual disks. This approach makes less assumption about the geometry of the ventricle. Several modifications of the basic Simpson rule method have been applied to calculate LV volumes. The optimal

correlation has been achieved with a modification that separately quantifies the volume of the apex as an ellipsoid.

Accurate calculation of LV volumes by Echocardiography requires high quality images to delineate the endocardium and image the entire LV perimeter. End systolic measurements are more accurate than those made at end diastole, probably owing to the superior endocardial definition. Nevertheless, Echocardiography calculations of LV volumes have generally yielded correlation coefficients in excess of 0.75 as compared with radionuclide angiography, cineangiography and autopsy studies. Hence calculations of LV volumes by these measurements are suitable for clinical decision making in the care of most patients.

DOPPLER ASSESSMENT OF SYSTOLIC FUNCTION

Although measurements of LV volume and ejection fraction can be obtained by 2D Echocardiography, Doppler interrogation provides the unique and complimentary Non-invasive assessment of systolic function. Thus LV systolic dysfunction often results in decreased aortic velocity and acceleration time. In the presence of mitral regurgitation, the acceleration of the MR jet can provide information regarding contractile function.

One of the most important applications of Doppler is in the calculation of the stroke volume. The volume of flow through any orifice or tube can be calculated as the product of cross - sectional area through which flow occurs and the velocity of that flow. Measurement of cross - sectional area can be derived from Echocardiography images, while velocity can be determined by Doppler. The cardiac output can be derived from the product of stroke volume and heart rate. Although the calculation of stroke volume by the Doppler method involves number of assumption, it has been shown to correspond well with thermo dilution, Fick, and the angiography calculations.

REGIONALFUNCTION

LV regional wall motion analysis is usually based on grading of contractility of individual segments. There are various LV segmental models depending on how the LV is subdivided. For the purpose of standardized analysis, the LV is divided into three levels (basal, mid, and apical) and 16 segments. The basal and mid (papillary muscle) levels are each subdivided into 6 segments, and the apical level is subdivided into 4 segments. All 16 segments can be visualized from multiple tomographic planes of surface echocardiography as well as Trans esophageal echocardiography(TEE). Based on the contractility of the individual segments, a numerical scoring system has been adopted in which higher scores indicate more severe wall motion abnormality (1 = normal; 2 = hypo kinesis; 3 = a kinesis; 4= dyskinesis 5 = aneurysmal). A wall motion score index (wmsi) is derived by dividing the sum of wall motion scores by the no. of visualized segments, and represents the extent of regional wall motion abnormalities

$$\text{WMSI} = \frac{\text{SUM OF WALL MOTION SCORES}}{\text{NO. OF VISUALIZED SEGMENTS}}$$

DIASTOLIC FILLING PROFILES

It is now well recognized that CHF Syndrome could be seen in the presence of an entirely normal LV systolic function. In these patients (approx. 30% of patients with CHF), the CHF is secondary to LV diastolic dysfunction. It is, mandatory to assess both systolic as well as diastolic LV function in all patients with CHF. There has been a great deal of interest in using mitral inflow velocity patterns to evaluate LV diastolic properties.Trans mitral filling reflects the pressure gradient between LA and LV during diastole. Diastole begins with myocardial relaxation at the end of systolic contraction (A2, aortic valve closure) and ventricular pressure starts to decline (isovolumic relaxation). When the left atrial pressure exceeds the LV pressure, the mitral valve opens to allow an early (E) rapid filling phase of the LV. The LV pressure continues to decline after the onset of

E, but soon increases again with continuous ventricular filling equalizing with left AP, resulting in a period of diastasis. Another bolus of ventricular filling occurs with left atrial contraction (A) whose contribution to the total C.O. depends on LV filling pressure and atrial contractility.

Pulse-wave Doppler mitral flow velocities reflect the Trans mitral pressure changes during diastole. Mitral flow velocities are measured by pulse-wave Doppler, with the sample volume placed between the leaflet tips, and the following diastolic filling parameters are derived: IVRT, early filling velocity (E), late filling velocity (A), and Deceleration Time(DT) of E. When the ventricular filling pattern is evaluated, it is important to keep the sample volume location. Both E and A velocities are smaller with the sample volume at the mitral annulus compared to the leaflet tip position. The DT is also shorter at the annulus position. Based on the Doppler velocity patterns, the diastolic filling abnormalities can be classified into three broad categories: relaxation abnormality, restrictive physiology, and pseudo normalization.

ABNORMAL RELAXATION

When myocardial relaxation is the predominant diastolic abnormality, IVRT is prolonged and the initial decline in LV pressure is slow. So, early filling is reduced, and there is a large compensatory filling with atrial contraction. The ventricle continues to relax even after the opening of the mitral valve, and it takes longer to equalize ventricular pressure with atrial pressure, resulting in a longer DT.Therefore, abnormal myocardial relaxation is characterized by a constellation of abnormalities consisting of :

1. Prolonged IVRT (≥110msec)
2. Low E velocity and high A velocity.
3. Reversed E/A ratio (≤1.0)
4. Prolonged DT (≥230msec)

RESTRICTIVE PHYSIOLOGY

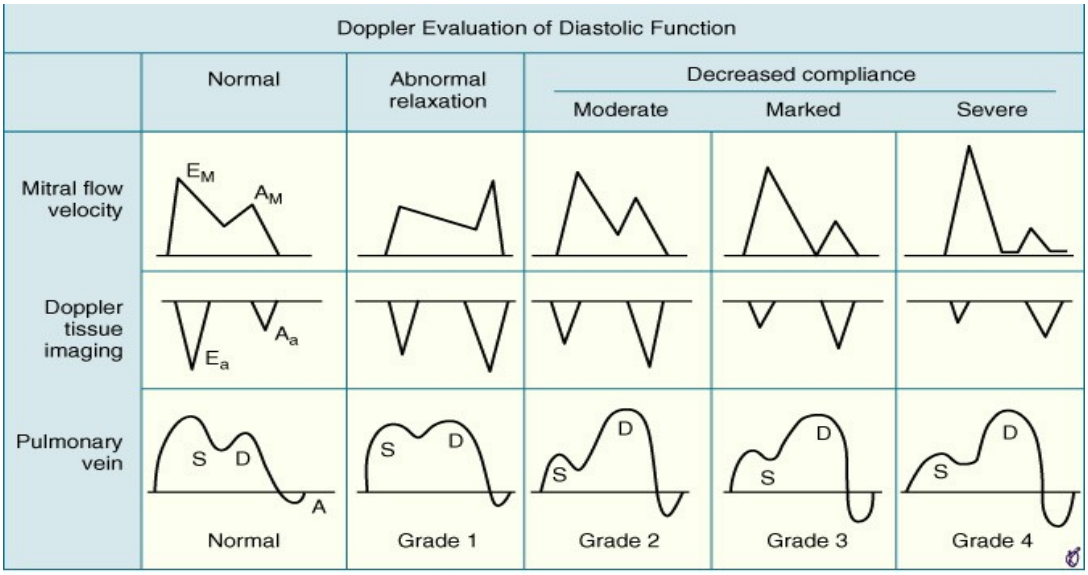
When ventricular compliance is decreased, the rise in ventricular diastolic pressure is very rapid during the early filling phase (short DT) and the elevated LV end –diastolic pressure minimizes ventricular filling due to atrial contraction (decreased A). With the resultant high left atrial pressure, the IVRT becomes shortened and the velocity is high. This particular diastolic filling pattern is indicative of “restrictive” physiology characterized by the following diastolic parameters:

1. Shortened IVRT (< 60 msec)
2. High E velocity and low A velocity.
3. Increased E/A ratio (≥2.0)
4. Shortened DT (≤ 150 msec)

restrictive physiology pattern is seen whenever LV diastolic pressure rises rapidly and end diastolic pressure is high, as in LV failure, restrictive cardiomyopathy, volume overload, and severe acute aortic regurgitation.

PSEUDO NORMALIZATION

When relaxation abnormality and restrictive hemodynamics coexist, Doppler features of the latter predominate. The pseudo normalization pattern is present when the left atrial pressure rises moderately in the setting of abnormal myocardial relaxation, producing a diastolic filling pattern similar to the normal pattern. Pulmonary venous systolic forward flow velocity is decreased in pseudo normalization, whereas it is higher than diastolic forward flow in the true normal filling pattern. Pulmonary venous flow velocity helps in separating pseudo normal from true normal diastolic filling. The elevated left atrial pressure in the patient with pseudo normal mitral inflow produces a longer duration and higher velocity of pulmonary vein atrial flow reversal.



VENOUS FLOW PATTERN IN DIASTOLIC FILLING ASSESMENT

Venous flow (pulmonary and hepatic vein) velocity patterns are also useful in the evaluation of diastolic filling of respective ventricle. Analysis of the pulmonary venous filling patterns provides a 2nd window into LV diastolic function. The S wave, occurring during systole depends on atrial relaxation and mitral annular motion, the D wave occurring during diastole reflects left ventricular filling, and the A wave, which is opposite the other waves occur during atrial contraction, reflects left ventricular compliance. One indication for examining pulmonary venous flow pattern is to distinguish the truly normal filling from pseudo normalization. In the presence of pseudo normalization pattern atrium contracts against an increase in after load due to an

elevated filling pressure or a stiff ventricle, hence blood is preferentially ejected in to pulmonary veins, resulting in a high and prolonged pulmonary venous A wave.

COLOUR M MODE DOPPLER ECHO CARDIOGRAPHY

A limitation to conventional pulse wave Doppler is that it only provides the velocity of blood flow at a single point within the heart. Colour M mode recordings overcome this limitation by providing the spatial and temporal velocity characteristics of flow along an entire echocardiographic scan line. Color M mode echo is a useful technique for examining the dynamics of blood flow across the mitral valve. The velocity of blood flow is increased with rapid relaxation and LV suction. Combined evaluation of flow propagation velocity and early diastolic annular velocity can be used for estimation of filling pressure.

TISSUE DOPPLER IMAGING

Tissue Doppler imaging refers to the technique of determining directional velocities of tissue structures rather than the moving blood pool. Tissue Doppler imaging yields information on intra myocardial velocity, providing a unique inside into LV mechanics during isovolumic contraction and relaxation. It has been shown that relaxation velocities in the myocardium are universally correlated with τ, so that the calculation of the time constant of relaxation may be possible. Doppler evaluation of annular motion has shown tremendous promise for the evaluation of diastolic function. When evaluated from an apical transducer position, annular motion is opposite in direction to the mitral inflow signals. The early annular velocity (Ea) exceeds late annular velocity (Aa) in a manner similar to mitral valve E/A. In patients with pseudo normal or restrictive filling pattern annular motion is abnormally low implying that it is relatively independent of pre-load. E/Ea ratio has been shown to have good correlation with LV end diastolic pressure in several studies. Doppler tissue imaging can be used to calculate direction and velocity of motion in two adjacent myocardial segments of

known distance of separation. From this strain, reflecting the relative velocity of either separation or closure between these two points can be calculated. This can further be developed into “strain rate imaging”, which integrates the rate of distance change between 2 adjacent points over time, which in turn is a parameter that can be color encoded over a segment of the myocardium. strain rate imaging can provide an increased level of resolution and accuracy for identification of subtle wall motion abnormalities in patients with ischemic and those with nonischemic heart disease. Through the integrated use of Doppler echo and tissue Doppler imaging, it is possible to obtain a fairly precise picture of left ventricular systolic and diastolic functions. However atrial fibrillation or frequent ectopic beats introduce major limitations of these techniques.

LV FUNCTION IN MYOCARDIAL INFARCTION

Systolic function

When ante grade flow in an epicardial coronary artery is interrupted, the zone of myocardium supplied by that vessel immediately loses its ability to shorten and perform contractile work. Four abnormal contraction patterns develop in sequence; 1.dyssynchrony-dissociation in the time course of contraction of adjacent segments, 2) hypo kinesis-reduction in the extent of shortening; 3) a kinesis-cessation of shortening and4) dyskinesis-paradoxical expansion and systolic bulging. Accompanying dysfunction of the infarcting segment initially is hyper kinesis of the remaining normal myocardium. The early hyper kinesis of the non-infarcted zones is thought to be the result of acute compensatory mechanisms, including increased activity of the sympathetic nervous system and the Frank-starling mechanism. Increased motion of the noninfarcted region subsides within 2 weeks of infarction, during which time some degree of recovery can be seen in the infarct region as well,

particularly if reperfusion of the infarcted area occurs and myocardial stunning diminishes. If a sufficient quantity of myocardium undergoes ischemic injury, left ventricular pump function become depressed, cardiac output, stroke volume, BP, and peak dP/dt are reduced and end systolic volume is increased. The degree to which end systolic volume increases is perhaps the powerful predictor of mortality following STEMI.As the ventricle dilates during the first few hours to days after infarction, regional and global wall stress increase according to Laplace's law. The degree of ventricular dilatation, which depends closely on infarct size, patency of the infarcted related artery, and activation of the local renin- angiotensin system in the noninfarcted portion of the ventricle can be favourably modified by angiotensin converting enzyme (ACE) inhibition therapy, even in the absence of symptomatic left ventricular dysfunction.

The earliest abnormality is a reduction in diastolic compliance, which can be observed with infarcts that involves only 8 % of the total left ventricle on angiographic examination, when the abnormally contracting segment exceeds 15%, the ejection fraction may be reduced and elevations of left ventricular end diastolic pressure and volume occur. The risk of developing physical signs and symptoms of left ventricular failure also increase proportionally to increasing areas of abnormal Left Ventricular wall motion. Clinical heart failure accompanies areas of abnormal contraction exceeding 25% and cardiogenic shock accompanies loss of more than 40% of the left ventricular myocardium. Unless infarct extension occurs, some improvement in wall motion takes place during the healing phase, as recovery of function occurs in initially reversibly injured (stunned) myocardium. Regardless of the age of the infarct, patient who continues to demonstrate abnormal motion of 20 to

25% of the left ventricle are likely to manifest hemodynamic signs of left ventricular failure.

DIASTOLIC FUNCTION

The diastolic properties of the left ventricle are altered in infarcted and ischemic myocardium. These changes are associated with a decrease in the peak rate of decline in left ventricular pressure [peak (-) dP/dt], an increase in the time constant of the fall in left ventricular pressure, and an initial rise in left ventricular end–diastolic pressure. Over a period of several weeks, end-diastolic volume increases and diastolic pressure begins to fall towards normal. As with impairment of systolic function, the magnitude of the diastolic abnormality appears to be related to the size of the infarct.

If the infarct is of sufficient size, it depresses overall left ventricular function so that the left ventricular stroke volume falls and filling pressure rise. A marked depression of left ventricular stroke volume ultimately lowers aortic pressure and reduces coronary perfusion pressure, this condition may intensify myocardial ischemia and thereby initiate a vicious circle. The inability of the left ventricle to empty normally, also leads to an increased preload, i.e. it dilates the well perfused, normally functioning portion of the left ventricle. This compensatory mechanism tends to restore stroke volume to normal levels, but at the expense of a reduced ejection fraction. The dilatation of the left ventricle also elevates ventricular after load. This increase in after load not only depresses LV stroke volume but also elevates myocardial consumption which in turns intensifies myocardial ischemia. When regional myocardial dysfunction is limited and the function of the remainder of the left ventricle is normal, compensatory mechanism sustain overall left ventricle function. If

a large portion of left ventricle becomes necrotic, pump failure occurs i.e. overall left ventricular function becomes so depressed that the circulation cannot be sustained despite the dilation of the remaining viable portion of the ventricle.

The Myocardial Performance Index (Tei Index)

There are many limitations to the use of classical echocardiographic indexes for the estimation of systolic and diastolic left ventricular (LV) function. The ejection fraction and LV volumes are subject to large errors when the ellipsoid shape of the heart becomes spherical. Age, rhythm and conduction disturbances, and changes in loading all affect the Doppler signal of transmitral flow, which is the most commonly used method for studying diastolic function. Hence Tei Chuwa devised and published in 1995 an index of myocardial performance (the Tei index) that evaluates the LV systolic and diastolic function in combination.[5] The Tei index has proved to be a reliable method for the evaluation of LV systolic and diastolic performance, with clear advantages over older established indexes and prognostic value in many kinds of heart disease

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Calculation of the Tei index

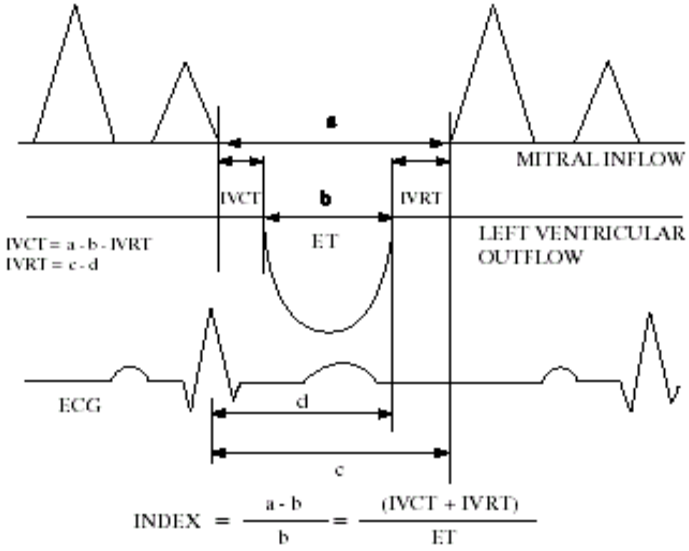


Fig. schematic representation of the measurement of TEI index

The Tei index is a pure number and is calculated from the ratio of time intervals (**a-b/b**) derived with the aid of pulsed Doppler echocardiography. Locating the sample volume at the tips of the mitral valve leaflets, in the apical 4-chamber view, enables the measurement of a, which is the time interval between the end and the start of transmitral flow. The sample volume is then located in the LV outflow tract, just below the aortic valve (apical 5-chamber view) for the measurement of b, the LV ejection time. The interval **a** includes the isovolumic contraction time (IVCT), the ejection time (ET) and the isovolumic relaxation time (IVRT), and the Tei index may also be expressed by the formula IVCT+IVRT/ET.For the evaluation of the right ventricular (RV) Tei index, the a interval, from the end to the start of trans-tricuspid flow (the interval from the end of the A wave to the start of the E wave), is obtained from the apical 4-chamber view with the Doppler sample volume located between the tips of the tricuspid valve leaflets. The b interval (RVET) is measured from the parasternal long-axis view, with the sample volume located just below the pulmonary valve. Normal range of LV

TEI index in an adult is 0.39± 0.05 and for RV is 0.28 ± 0.04.[3]

MODIFIED TEI INDEX

The major limitation of TEI index is that it is not measured in a single cycle, rather the measurements are obtained sequentially, and hence in the presence of marked heart rate fluctuations reliability of this method is restricted. Hence a modification is proposed for measuring TEI index by using tissue Doppler.[6] This modification enables to measure contraction and relaxation velocities in a single cycle simultaneously. Modified TEI index is measured as follows: The TDI program is set to pulse wave Doppler mode, filters were set to exclude high frequency signals & gains were minimized to allow a clear tissue signal. The spectral Doppler signal is adjusted to a nyquistic limit of 15- 20 cm/sec. TDI is obtained from apical 4 chamber view & a 2mm of sample volume is placed either at medial or lateral corner of mitral annulas. Velocities are recorded at sweep speed of 100 mm/sec. The time duration from end to onset of mitral annular velocity pattern during diastole is measured (measurement a'). The duration of S wave is measured from its onset to the end & it is marked as measurement b'. IVRT is calculated from subtracting the interval d'(R wave & cessation of systolic velocity), from the interval c' (R wave to onset of diastolic velocity). IVRT; c'- d'. IVCT is calculated by subtracting IVRT from (a'-b'). Modified TEI INDEX by TDI is calculated as a'-b'/b'

Tei index and age

In a study conducted in 161 children with no cardiovascular disease, aged from 30 days to 18 years, for determining the range of normal values for the Tei index and the effect of age.[7] It was found that Tei index was affected by age during the first 3 years of life,

showing a progressive reduction until the age of 3, but then it showed no further changes. The Tei index for children aged <3 years was significantly greater (0.40 ± 0.09) than for those aged between 3 and 18 (0.33 ± 0.02). The age-dependent changes in the index may reflect changes during the maturation of the myocardial characteristics of the LV in neonates and children. During development, the relation between total collagen and total protein reaches normal levels in 5 months and the relation between type I collagen (which mainly provides rigidity) to type III collagen (which provides elasticity) stabilizes after 3 years. The RV Tei index in 150 healthy children, mean age 5.1 ± 5.5 years, was 0.24 ± 0.04, irrespective of age.[7]

Tei index and preload

The effect of preload changes on the Tei index was investigated in 50 healthy volunteers and 25 patients with a previous infarction.[8] Three procedures were performed successively, the Valsalva manoeuvre(preload reduction), passive raising of the lower limb(preload increase) and administration of sublingual nitroglycerine (preload reduction). In the controls, the index increased significantly during the Valsalva manoeuvre (mainly as a result of a reduction in ET), after passive raising of the lower limb (primarily as a consequence of an increase in IVCT) and after nitroglycerine administration (as a result of a reduction in ET and a prolongation of IVCT). In contrast, no significant changes were seen in the index in the infarction patients during the above preload variations.

Briefly, in those patients when the preload decreased the IVCT/ET ratio showed a reduction while the IVRT/ET ratio increased, leaving the index unchanged. Although these result show a change in the Tei index under different preload conditions, the extent of the

changes was small (<10%), a fact that explains the preservation of the prognostic value of the Tei index despite variations in preload.

Tei index and haemodynamic indexes

In a prospective study [9] 17 patients with idiopathic dilated cardio myopathy (EF: 24% ± 11%) and 19 patients with ischaemic heart disease (EF: 49% ± 13%) underwent catheterization and a Doppler echo examination. In all cases simultaneous recordings were made of LV pressures and Doppler velocity curves and the following were calculated: maximum rate of pressure increase during isovolumic systole (peak+dP/dt), maximum rate of pressure decrease (peak -dP/dt) and the time constant of pressure reduction during isovolumic relaxation (tau). The Tei index was found to be significantly correlated with all three variables, providing confirmation that it is a reliable measure of total LV function. The index was also found to be more sensitive in the evaluation of diastolic relaxation than parameters such as the deceleration time of the E wave (DT) and the E/A ratio, which showed a weaker correlation with peak -dP/dt and tau.

Tei index and coronary artery disease

In patients with acute myocardial infarction, the Tei index was found to be significantly more pathological (greater) than in healthy controls.[10] IVCT and IVRT were prolonged and ET was significantly shorter in the patients with acute myocardial infarction. The Tei index also showed predictive value in relation to the severity of coronary artery disease. It was more pathological in the group of infarction patients who had severe coronary artery disease than in those with 1- or 2-vessel disease, for both anterior and inferior

infarctions. In 21 patients who had a complicated course after a first myocardial infarction (death, heart failure, arrhythmias, post-infarction angina) the mean value of the Tei index was significantly higher than in 75 patients with an uncomplicated course (0.65 ± 0.20 versus 0.43 ± 0.16, p: 0.0001).[11] The higher value of the Tei index was due to prolongation of the IVCT ,and shortening of the ET. Tei index values ≥ 0.47 showed 90% sensitivity and 68% specificity in identifying patients with events, while in a multivariate, model the index on admission continued to be an independent prognostic factor for in-hospital cardiac events. Ling et al performed dobutamine stress echo testing in 27 individuals.[12] The Tei index was significantly higher at peak dobutamine stress in the group with ischaemia (13 patients) than in the 14 subjects who had a negative test. More generally, the Tei index in the ischaemic subgroup showed an increase at peak stress, while in the remaining subjects the index showed no significant change as the test progressed. At the onset of ischaemia IVCT and IVRT were prolonged and the ET shortened, resulting in an increased, pathological Tei index. The index appears to be useful in the recognition of myocardial ischaemia and the development of LV dysfunction during a stress echo examination.

Tei index and heart failure

In patients with dilated cardiomyopathy the index was found to reflect the severity of LV dysfunction and was proved to be an independent prognostic factor for mortality, similar to the EF.[13]The higher values of the Tei index in patients than in healthy individuals were attributable to prolongation of the isovolumic intervals and a shortening of ET. The Tei index was significantly correlated with NYHA class, EF and ventricular volumes, while values >0.77 were associated with higher 1-, 3- and 5-year mortality. The Tei index was significantly

greater in patients with heart failure than in controls and was correlated with LV end-diastolic pressures. Values >0.47 identified heart failure patients with a sensitivity of 86% and a specificity of 82%. [14]

Harjai et al [15] investigated the prognostic value of the Tei index in 60 patients with severe, symptomatic heart failure (EF <30%) of ischaemic aetiology or not. The endpoints were death from any cause and heart transplantation. A strong correlation was found between a high Tei index (>1.14) and the long term outcome, independently of other clinical and echo indexes that have been proved to have prognostic value, such as age, sex, EF, coronary artery disease, NYHA class, mitral regurgitation, RV systolic dysfunction and the deceleration time of early diastolic filling (DT). A Tei index >1.4 was an independent prognostic factor for death or emergency heart transplant during two years' follow up and had more predictive power than EF or NYHA class. In another study the Tei index was evaluated at rest and after the administration of a low dose of dobutamine in patients with idiopathic or ischaemic dilated cardiomyopathy and was correlated with parameters from cardiopulmonary exercise testing. An advanced NYHA class and a restrictive filling pattern were associated with higher values of the index, while a negative correlation was found between the Tei index and systolic indexes (stroke volume, cardiac output), diastolic indexes (E/A, A) and cardiopulmonary exercise testing parameters (peak oxygen consumption, anaerobic threshold). Dobutamine administration caused a shortening of IVRT and IVCT, prolongation of ET and improvement (reduction) of the Tei index. Multivariate analysis revealed that the index was an independent prognostic factor for exercise tolerance. In patients with isolated diastolic dysfunction, assessed in terms of the E/A ratio of transmitral flow or the S/D relation of the pulmonary veins, the index was found to be significantly elevated mainly as a result of a prolongation of

IVRT.[16]

Tei index and heart transplantation

Since systolic and diastolic dysfunctions are often both present during episodes of cardiac rejection, the Tei index was investigated as a possible harbinger of acute rejection.[17] In a small sample of post-transplant patients the values of the index ranged from 0.2 to 0.45 during periods of nonrejection and from 0.2 to 0.8 during periods of rejection, a difference that approached statistical significance. It seems that the Tei index might be a useful prognostic factor for cardiac rejection in post-transplant paediatric patients. Similarly, in 13 post-transplant men a Doppler study was performed and the index was calculated during the same 24-hour period in which a myocardial biopsy was taken. The isovolumic systole and isovolumic relaxation times showed statistically insignificant prolongation and the ET shortened significantly with progressively increasing biopsy scores (stage I, II and III), while the increase in the Tei index was more significant. Multivariate stepwise regression analysis showed that the Tei index was the sole independent factor to be correlated with the biopsy score of the transplanted heart.

Tei Index and valvular disease

Haque et al [18] investigated the effect of valve dysfunction on the Tei index, calculating the index in 76 patients with aortic or mitral valve disease before and after surgical valve replacement or repair. The authors found that the index may underestimate the presence of aortic stenosis, aortic regurgitation and mitral stenosis, while it may overestimate the presence of mitral regurgitation. The values of the Tei index increased postoperatively, to a statistically

significant degree, in patients with aortic stenosis, aortic regurgitation and mitral stenosis, whereas it decreased in mitral regurgitation, though not significantly. The differences were most evident in aortic stenosis and were positively correlated with the preoperative values of peak aortic flow velocity. In patients with severe aortic stenosis, symptoms of heart failure can be attributed to systolic, diastolic or combined LV dysfunction. In 10 symptomatic patients[19] with severe aortic stenosis (orifice 0.6 ± 0.2 cm2), compromised systolic function (EF = 45%) and increased LV end-diastolic pressure determined invasively (32 ± 8 mmHg), the IVCT was prolonged and the ET shortened, resulting in a significantly elevated Tei index compared to healthy controls. In 22 patients with severe, symptomatic aortic stenosis (orifice 0.7 ± 0.2 cm2), physiological systolic function and increased filling pressures (22 ± 7 mmHg), there was shortening of IVRT and IVCT, a prolongation of ET and a consequent decrease in the value of the Tei index. Thus, the index was able to discriminate between those patients with severe aortic stenosis who had depressed systolic function and those whose systolic function was preserved. The index was significantly higher when there was combined systolic and diastolic dysfunction and significantly lower in the case of primarily diastolic dysfunction.

Tei index and pulmonary hypertension

The RV Tei index was found to be the most powerful Doppler parameter for distinguishing patients with primary pulmonary hypertension from healthy individuals.[20] Furthermore, there was a significant correlation between the index and the patients' functional condition as well as with total survival. An increase in the index by 0.1 increased the risk of death by 1.3 times. The index was found to be independent of heart rate or loading conditions (RV systolic and diastolic pressure or diastolic pulmonary pressure or presence and severity

of tricuspid regurgitation). The IVCT was prolonged in the patients, probably because of an earlier start of isovolumic systole due to increased RV end-diastolic pressures and an earlier intersection of the right atrial and RV pressure curves. The finding of a significant prolongation of IVRT showed the coexistence of RV diastolic dysfunction, while the shortened ET was attributed to an increase in pulmonary vascular resistance, to a reduction in RV filling with the reduction in stroke volume and to the presence of tricuspid regurgitation.

Measurement of the Tei index is non-invasive and easily obtained, it does not require the presence of an echo cardiographer with great experience and it does not materially prolong the time required for the examination. The calculation of the index is not based on a geometric model or on volume measurements; it is first and foremost a ratio of time intervals, independent of ventricular geometry. It is also independent of blood pressure, heart rate and age and it appears to be of great prognostic value in many different clinical settings. Of course, the Tei index has its disadvantages and its use may present difficulties. For example, its precise measurement is infeasible in patients with atrial fibrillation, frequent ventricular ectopic stimuli, disturbances of intraventricular or atrioventricular conduction, a permanent pacemaker, or when Doppler images of sufficient quality cannot be acquired. Furthermore, it is affected to some degree by loading conditions.

The Tei index is not a gold standard method, However, it appears to be reliable for the evaluation of the severity of myocardial dysfunction in an appreciable number of diseases and can help determine which patients need early intervention.

ESTIMATION OF LV EF FROM TEI INDEX

Lax et al [36] have proposed EF estimation from tei index as an alternative to 2D measurement of EF. He proposed a formula **EFMPI= 0.60 – (0.34X MPI).** They tested EF derived from this formula against EF measurement obtained by radionuclide angio & EF obtained from 2D echo measurements. They found that there is a good agreement between EF obtained from MPI and other methods. While Torstein Hole et al [35] tested this method in acute MI settings and find that there is significant under estimation of EF derived from MPI when compared to that from radio nuclide angio, they also found in their study that there was only a moderate correlation between EF derived from MPI and that from 2D measurements. Although the concept of deriving a measurement like EF from another calculated measurement should usually be avoided in clinical practice, but in situations where EF is important for making clinical decision and the image quality is not satisfactory the ability of MPI to indicate EF can be utilized.

REVIEW OF RELATED ARTICLES

- **Index of myocardial performance after early phase of myocardial infarction in relation to its location.** Cacciapuoti F, [21] : J Am Soc Echocardiogr. 2004 Apr; 17(4):345-9.

To define the degree of heart derangement in recent myocardial infarction (MI) occurring in different wall locations of myocardium, Cacciapuoti F et al echocardiographically evaluated left ventricular volumes, ejection fraction, wall-motion score index, isovolumetric contraction and relaxation time, ejection time, and the index of myocardial performance in 74 patients with MI. Mean values of left ventricular end-diastolic and end-systolic volumes and ejection fraction were nearly alike in all patients, whereas wall-motion score index and index of myocardial performance were clearly prolonged in those with anterior MI in comparison with the values recorded in patients with lateral or inferior MI. The mean values of isovolumetric contraction and relaxation time suggest that a prevalent systolic dysfunction is present in the early phase of MI. In addition, the different index of

myocardial performance prolongation in MI otherwise located suggests evidence that MI located in the anterior wall has more serious effect than lateral or inferior MI.

- **The Doppler echocardiographic myocardial performance index predicts left-ventricular dilation and cardiac death after myocardial infarction.**

MÃ¸ller JE, et al[22] Cardiology 2001; 95:105-11.

Maller et al carried out a study to investigate the value of the Doppler-derived myocardial performance index to predict early left-ventricular (LV) dilation and cardiac death after a first acute myocardial infarction (AMI), Doppler echocardiography was performed within 24 h of hospital admission, on day 5, 1 and 3 months of MI in 125 consecutive patients. The index measured on day 1 correlated well with the change in end-diastolic volume index observed from day 1 to 3 months following AMI ($r = 0.66$, $p < 0.0001$). One-year survival in patients with Doppler index of 0.63 was 89%, and 37% in patients with index ≥ 0.63, ($p < 0.0001$). Multivariate analysis identified myocardial performance index ≥ 0.63 (relative risk 5.6, $p < 0.0001$), E-wave deceleration time <140 ms (relative risk 2.7, $p = 0.008$) and Killip class $\geq$ II (relative risk 4.0, $p = 0.04$) to be independent predictors of cardiac death. Hence it was concluded that the Doppler echocardiographic myocardial performance index is a predictor of LV dilation and cardiac death after a first AMI.

- **Tei index as a method of evaluating Left ventricular dysfunction in acute myocardial infarction**.

Nearchou NS, et al [23] Hell J Cardiol; 46:35-42.

In a study conducted to assess the relationship of LV diastolic dysfunction with MPI in acute myocardial infarction in 105 patients it was found that MPI index was high in patients with impaired relaxation and decreased peak filling rate, but in patients with pseudo normalization MPI was not found to be significantly elevated. Hence the role of MPI in diastolic dysfunction group needed further studies for establishing its relationship.

- **Serial changes and prognostic implications of a Doppler-derived index of Combined left ventricular systolic and diastolic myocardial performance in acute myocardial infarction.**

  Poulsen SH, et al [24] Am J Cardiol 2000; 85:19-25.

This study was done to investigate the serial changes and prognostic value of a nongeometric Doppler-derived index of myocardial function that combines systolic and diastolic time intervals of the left ventricle in acute myocardial infarction (AMI). The Doppler index was measured in 60 consecutive patients with AMI and in 30 patients admitted to hospital with suspected but disproved AMI who served as controls. The patients were studied at days 1, 5, 90, and 360 after arrival in the coronary care unit.

The index was significantly higher i n patients with AMI than in control subjects at days 1 and 360.

The index was significantly higher in Patients who developed congestive heart failure or died Compared with survivors who were free of congestive heart

Failure.

Univariate analysis demonstrated that the Doppler index ≥ 0.60, deceleration time ≤ 140 ms, ejection fraction ≤ 0.40%, anterior wall MI, and age were significant predictors of outcome. Multivariate stepwise analysis showed that the index ≤ 0.60, deceleration time ≤ 140 ms, and age were independent predictors of outcome. Hence it can be concluded MPI reflects severity of left ventricular dysfunction and has incremental prognostic value in patients with AMI.

- **Evaluation of a Doppler-derived index combining systolic and diastolic**

Left ventricular function in acute myocardial infarction.

Karvounis HI, [25] Angiology. 2004 Jan-Feb; 55(1):21-8

In this study the applicability of the Doppler-derived myocardial performance index (MPI), in patients with acute myocardial infarction (AMI) was evaluated to indicate whether this index reflects the severity of LV dysfunction in this subgroup of patients. Post-AMI patients were compared with age- and sex-matched healthy subjects. Patients were evaluated within 24 hours of the AMI and 1 month thereafter, Patients were divided into group A (Killip Class I), and group B (Killip Class II-III), The LV ejection fraction (EF), transmitral E and A waves, E/A ratio, deceleration time [DT], isovolumic contraction time [IVCT], isovolumic relaxation time [IVRT], MPI, LV end-systolic and end-diastolic volume indices [ESVi and EDVi] and wall motion score index [WMSi]) were evaluated. It was found that EF, ESVi, MPI, and WMSi were significantly elevated at day 1 in patients with killip class II, III, it was found to persist even after 1 month. One-year mortality was significantly lower in group A patients. This study shows that the MPI, reliably indicated LV dysfunction post-AMI, significantly correlated with clinically determined functional class, and possibly has some prognostic implication.

- **Long-term prognostic value of an index of myocardial performance in Patients with myocardial infarction.**

Szymanski P, et al[26] Clin Cardiol. 2002 Aug; 25(8):378-83.

This study was done to assess the long-term prognostic value of MPI in 90 patients discharged from hospital after acute myocardial infarction (AMI).All the patient were followed for an average of 58 months. After multivariate Cox analysis, Tei index > 0.55 LV end-systolic volume > 65 ml, and mitral E wave deceleration time ≤ 0.145 s were the only independent predictors of cardiac events during the follow-up period. In a subgroup of patients with Preserved LV systolic function (ejection fraction > 0.40), MPI was the only predictor of cardiac events. Hence this study concluded that MPI is a useful tool for risk assessment in patients following myocardial infarction, and in a subgroup of patients with normal or only mildly impaired systolic function.

- **Myocardial performance index for assessment of left ventricular outcome in successfully recanalyzed anterior myocardial infarction.** M Kato,[27] 2005; 91; 583-588 Heart

This study was done to investigate whether the myocardial performance index (MPI) can predict left ventricular functional outcome in patients with early recanalisation after anterior acute myocardial infarction (MI) and to determine when the index should be measured.32 consecutive patients with their first anterior acute MI who had complete occlusion of left anterior descending coronary artery who underwent successful PCI within six hours of symptom onset, in whom Left ventricular anterior wall motion score index (A-WMSI), left ventricular end diastolic pressure (LVEDP), left ventricular ejection fraction (LVEF), and left ventricular end diastolic volume(LVEDV) were measured .It was found that there was a significant negative correlation between MPI on day 2 and the coronary diastolic deceleration

time as well as a significant positive correlation with the coronary diastolic deceleration rate. MPI on day 2 was significantly correlated with the short and long term changes of A-WMSI and with the short term changes of LVEDP. Furthermore, MPI on day 2 was significantly correlated with the short and long term changes of LVEF and of LVEDV.From this it can be concluded that Doppler derived MPI on day 2, representative of the early coronary micro vascular state, can predict the left ventricular functional outcome after early successful recanalisation of a patient with first anterior acute MI.

- **Prognostic importance of systolic and diastolic function after acute myocardial infarction** Jacob E. et al,[28] Am Heart J 2003; 145:147-53.)

This study was done to determine the prognostic importance of LV systolic, diastolic, and overall LV function in a large consecutive population with AMI. LV systolic, diastolic, and global function was assessed by means of wall motion score index (WMSI), mitral flow pattern, and Tei index. The primary end point was all-cause death. In a multivariate model including and clinical parameters, WMSI had important prognostic information. When mitral filling pattern and quartiles of Tei index were added to the model, restrictive filling (mitral deceleration time $\leq$ 140 ms) , higher Tei index values were associated with risks, Hence it can be concluded that Mitral deceleration time and the Tei index have independent and important prognostic value after AMI.

- **Doppler index of myocardial performance and its relationship with Mitral E wave deceleration time in acute Q-wave myocardial infarction**

Sekuri C, et al [29]Anadolu Kardiyol Derg. 2004 Jun; 4(2):108-13.

The study was done to assess myocardial systolic and diastolic functions by myocardial performance index (MPI) and its relationship with E - wave deceleration time (DT) in early phase of acute Q-wave myocardial infarction (MI). This study was carried out in 50 patients with

acute MI, (25 pts with

Anterior MI and 25 pts with inferior MI). The index and DT was found to be higher in anterior than in inferior MI. Myocardial performance index was positively correlated

with DT in inferior MI and negatively correlated with

Anterior MI, hence it can be concluded that

Doppler-derived MPI reflects severity of global left ventricular d

ysfunction in early phase of acute MI and may be a useful parameter in

these patients.

- **Tei-Index in coronary artery disease—validation in patients with overall cardiac and isolated diastolic dysfunction.** Bruch C,[30] Z Kardiol. 2002 Jun; 91(6):472-80.

  This study is carried out to validate t

he Tei-Index in CAD patients with overall cardiac or isolated diastolic

dysfunction. Sixty subjects were included who

underwent left heart catheterization for invasive measurement of left

Ventricular end-diastolic pressure (LVEDP): 20 symptomatic CAD patients had

Overall cardiac dysfunction (defined by a LV ejection fraction (EF)

< 45% and a LVEDP ≥ 16 mmHg,

NYHA class 2.7 +/- 0.4, OCD group), 29 symptomatic CAD patients had

Isolated diastolic dysfunction (defined by an EF > 45%,

A normal end-diastolic diameter index and a

LVEDP ≥ 16 mmHg, NYHA class 2.3 +/- 0.4, IDD

Group) and 11 asymptomatic control subjects (EF 65 +/- 9%, LVEDP 11 +/-

4 mmHg, CON group) were studied. The Tei-Index

was easily and reproducibly measured in all study subjects. In the OCD g

roup, isovolumic contraction time was prolonged and ejection time was shortened in comparison to the CON group, resulting in a significantly increased Tei-Index. In the IDD group, isovolumic relaxation time was prolonged and isovolumic Contraction time was shortened in comparison to controls, resulting in a largely unchanged Tei-Index.

Using a Tei-Index > 0.49 as a cut-off, OCD patients were identified with a sensitivity of 96% and a specificity of 86%. This study concludes that The Tei-Index is a valid and readily derived indicator of global cardiac dysfunction in CAD patients with impaired systolic and diastolic LV performance. But the use of this index seems to be limited in CAD patients with primary diastolic dysfunction.

- **Prognostic value of the Tei index combining systolic and diastolic Myocardial performance in patients with acute myocardial infarction Treated by successful primary angioplasty.** Sasao H, et al [31] Heart Vessels. 2004 Mar; 19(2):68-74.

This study was done to assess the usefulness of the Tei index by echocardiography for evaluation of infarct size and clinical outcome in patients with AMI treated by successful primary angioplasty. 10 age-matched control subjects and 43 consecutive patients with first AMI treated by successful primary angioplasty were analyzed. The Tei index of the AMI patients was significantly greater than that of the control subjects. Also, the Tei index has showed a significant positive correlation with peak creatine kinase values and (99m) Tc-tetrofosmin scores. Moreover, multiple logistic regression analysis showed that the Tei index >0.70 was the only significant explanatory factor for cardiac death or developed congestive heart failure. The Tei index combining systolic and diastolic myocardial performance reflects infarct size and might be a predictor of clinical outcome in patients with AMI treated by successful primary angioplasty.

- **Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements.** Tei C, [9] J Am Soc Echocardiogr. 1997 Mar; 10(2):169-78.

This study was done with the purpose to correlate the Doppler index with accepted indexes of cardiac catheterization of left ventricular performance. Thirty-four patients with ischemic heart disease or idiopathic dilated cardiomyopathy prospectively underwent a simultaneous cardiac catheterization and Doppler echocardiographic study. Invasive measurements of peak +dP/dt, peak -dP/dt, and tau were obtained from the high-fidelity left ventricular pressures. A Doppler index of myocardial performance was defined as the summation of isovolumetric contraction and relaxation time divided by ejection time. There was a correlation between Doppler measurement of isovolumetric contraction time and peak+dP/dt and Doppler measurement of isovolumetric relaxation time and peak -dP/dt. Left ventricular ejection time correlated with peak +dP/dt and peak -dP/dt. The Doppler index correlated with simultaneously recorded systolic peak +dP/dt and diastolic peak -dP/dt and tau. This study documents that a simple, easily recordable, noninvasive Doppler index of myocardial performance correlates with invasive measurement of left ventricular systolic and diastolic function and appears to be a promising noninvasive measurement of overall cardiac function.

- **Value of the Doppler index of myocardial performance in the early phase of acute myocardial infarction.** Poulsen SH : [32]J Am Soc Echocardiogr. 2000 Aug; 13(8):723-30.

This study was conducted in 64 patients with acute myocardial infarction (MI) within 1 hour after their arrival to the hospital and in 39 age-matched healthy subjects. The MPI was significantly higher in patients with MI compared with healthy subjects. In patients with MI and in-hospital congestive heart failure (CHF), the index was significantly higher compared with patients without CHF. In a multivariate regression analysis, the index >0.45 was the strongest independent predictor of the development of CHF. This simply obtained non-geometric Doppler index, assessed in the early phase of MI, detected and graded left ventricular dysfunction and identified patients at risk for the development of CHF.

- **Doppler echocardiographic index for assessment of global right ventricular function.** Tei C, et al [20]J Am Soc Echocardiogr. 2000 Sep; 13(9):882-3.

The purpose of this study was to assess the clinical value of a Doppler-derived index, in assessing global right ventricular function in patients with primary pulmonary hypertension. The study population comprised 26 consecutive patients with primary pulmonary hypertension and 37 age-matched normal subjects. The index was compared with

available parameters of systolic or diastolic function, clinical symptoms, and survival. Right ventricular isovolumetric contraction time and isovolumetric relaxation time were prolonged significantly in patients with primary pulmonary hypertension compared with normal subjects. Ejection time was shortened significantly in patients with pulmonary hypertension. However, the index was the single most powerful variable to discriminate patients with primary pulmonary hypertension from normal subjects and was the strongest predictor of clinical status and survival. The index was not significantly affected by heart rate, right ventricular pressure, right ventricular dilation, or tricuspid regurgitation. It is well known that right ventricular systolic and diastolic dysfunction coexist in patients with primary pulmonary hypertension. This index appears to be a useful noninvasive means that correlates with symptoms and survival in-patient with primary pulmonary hypertension.

- **Prediction of Heart failure in Post Myocardial Infarction: Comparison of Ejection fraction, trans mitral filling parameters, and the Index of Myocardial performance.**

Steven j. lavine, M.D.[33] **ECHOCARDIOGRAPHY: vol.20, no. 8, 2003 691-701**

This study was conducted to determine the comparative predictive value of EF, trans mitral filling parameters, and MPI for the development of congestive heart failure after first MI. 109 patients were studied and it was found that those who developed CHF had greater LV dilatation, lower EF, higher E/A ratio, shorter DT, and increased MPI. Even in patients who developed late LV failure also showed similar finding. Hence the authors concluded that the early and ultimate development of CHF following first MI was associated with a moderately reduced EF, pseudo normalization, and an increased MPI.

- **Importance of the Index of Myocardial performance in evaluation of left**

ventricular function. Sinan Dagdelen, M.D., [34]Echocardiography vol.19, no.4, 2002, 273-278

This study was done in 82 patients to assess the importance of MPI in-patient with critical coronary artery disease. This study showed that there were significant difference in IVRT, MPI, DT, in patients with critical coronary stenosis when compared with those with out critical stenosis. Hence this study concludes that MPI might be a useful parameter and an early indicator of LV dysfunction in patients with critical coronary artery disease.

- **Estimation of LV EF from Doppler derived Index of Myocardial Performance in patients with Acute Myocardial Infarction:TorsteinHole, M.D.,[35] Echocardiography: vol.20.3, 2003 231-236**

This study was done to evaluate the Lax et al formula to calculate EF from MPI in acute MI with established parameters. 62 patients were studied; the EF derived by MPI was compared with radio nuclide angiography, 2D methods. The results showed that there was only a modest agreement between the three methods.

MATERIALS AND METHODS

This study was conducted in 60 patients who were admitted to CCU, Department of Cardiology, Stanley medical college with acute myocardial infarction (STEMI). 30 age matched & sex matched control were taken for comparison.

SELECTION CRITERIA

Patients who were admitted to CCU, with first episode of acute myocardial infarction were included. The diagnosis of myocardial infarction was based on presence of any 2 of the following 3 criteria,

1. Typical precordial pain
2. ECG changes suggestive of MI (ST segment elevation of >0.1 mv in limb leads or > 0.2 mv in precordial leads)
3. Elevated cardiac enzymes.

The diagnosis of I episode of MI was determined if the previous ECG was normal or there was no history or symptoms suggestive of coronary disease.

EXCLUSION CRITERIA

The following groups of patients were excluded from the study. Patients with

1. Significant Valvular heart disease
2. Pericardial disease
3. Cardiomyopathies
4. Unstable angina
5. Significant tachy or bradyarrythmias.
6. On pace maker therapy

The entire patients who were included in the study were evaluated on the basis of proforma, detailed history with special focus on chest pain duration and risk factors were obtained. Patients were evaluated with a thorough echo cardio graphic analysis with in 48 hrs of admission. Their treatment history and in hospital complications were noted.

TWO –dimensional and M-mode measurements were obtained with patients in left lateral position using an ALOKA SSD4000 phased array system equipped with tissue Doppler and harmonic imaging technology. Para sternal long and a short axis as well as apical four- and two chamber views were used for the evaluation of the functions of the left ventricle and the heart valves. LV dimension and fractional shortening (FS) of the left ventricle were calculated by using teicholtz formula. Ejection fraction was obtained by modified Simpson's method.

Pulse-wave Doppler measurements of mitral inflow were obtained with the transducer on the four-chamber view with a 1-2 mm Doppler sample volume was placed between the tips of the mitral leaflets during diastole. The left ventricular outflow velocity curve was recorded from the apical long axis view/apical five chamber view with the sample volume positioned just below the aortic valve.

Doppler velocities and time intervals were measured from mitral inflow and left ventricular outflow recordings. Isovolumetric relaxation time (IVRT) was the time interval from cessation of left ventricular outflow to onset of mitral inflow, ejection time (ET or MPI Measurement B) was the time interval from the onset and cessation of left ventricular outflow, and mitral early diastolic (E) flow deceleration time (DT) was the time interval between the peak E velocity and the end of the early diastolic flow. Total systolic time interval was measured from the cessation of one mitral flow to the beginning of the following mitral inflow (MPI Measurement A). Isovolumetric contracting time (ICT) was calculated by subtracting ET and IVRT from the total systolic time interval. MPI was calculated by using the formula MPI= MPI A- MPI B/MPI B or (IVRT+ICT)/ET.

Tissue Doppler echo was performed by activating the tissue Doppler function in the same machine. Images were obtained in the apical four chamber view with the filter setting were kept low and gains were adjusted at the minimal optimal level to minimize noise. 1.7 mm sample volumes were placed at both septal and lateral mitral annular site and systolic velocity (Sm), early and late diastolic velocities (Em or E', Am or A') were obtained and average values were taken . E/E' ratio was calculated

Statistical Analysis

Datas are expressed as mean value ± standard deviation (SD). Comparisons between parameters of the groups were performed by using an unpaired Student's t-test. Statistical significance was defined as $P < 0.05$.Statistical analysis was done using SPSS software system.

RESULTS AND ANALYSIS

Our study population included 60 patients and 30 age, sex matched controls. Among the 60 patients 43 (71.7%) were males and 17(28.3%) were females. While out of 30 controls 20 were males and 10 were females. Out of 43 male patients 14 had anterior, 15 had antero septal, 3 had inferior, and 11 had inferior/lateral or posterior or RV myocardial infarction. Among the 17 females, 4 had anterior, 4 had anteroseptal, 3 had inferior and 6 had inferior/lateral or posterior or RV myocardial infarction.

They were thoroughly evaluated by 2D, M-Mode, Doppler and Tissue Doppler echocardiography. The results of the study were as follows,

TABLE.1

.ECHOPARAMETERS IN PATIENTS & CONTROLS

(*) Denotes statistically significant

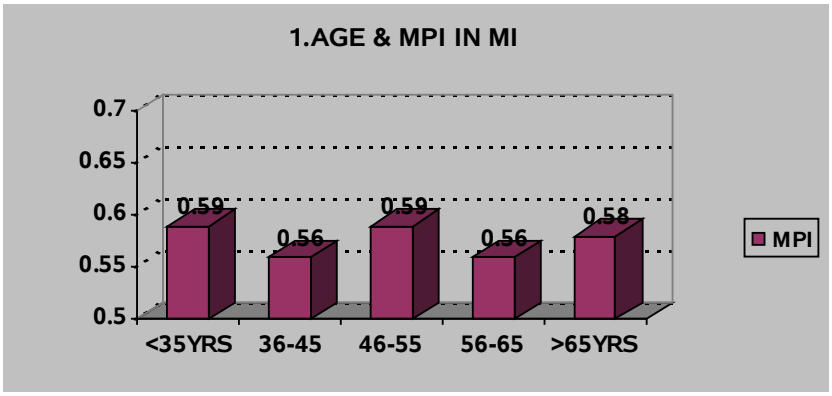
TABLE.2. INFARCT AREA AND PARAMETERS

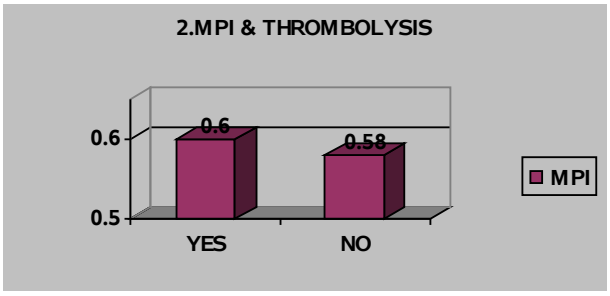
(*) Denotes statistically significant

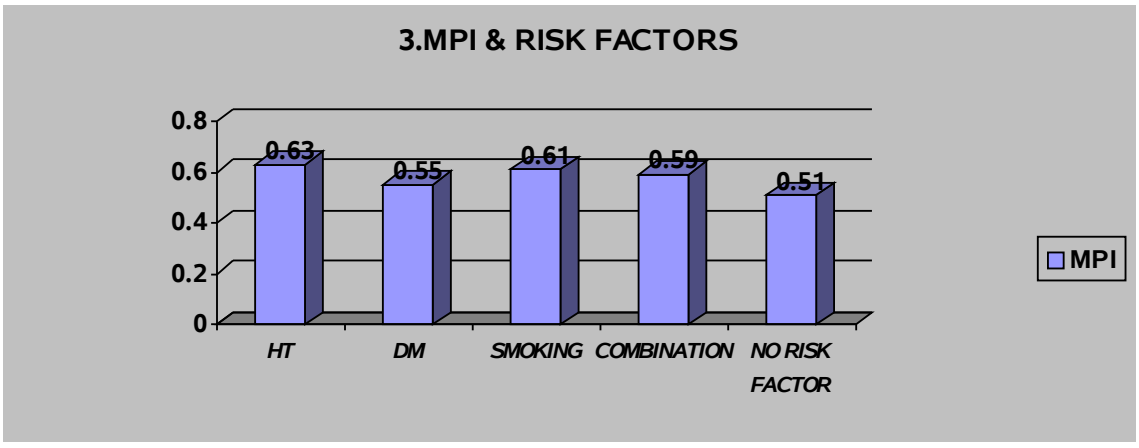
TABLE.3 SEX DISTRIBUTION OF DIFFERENT PARAMETERS.

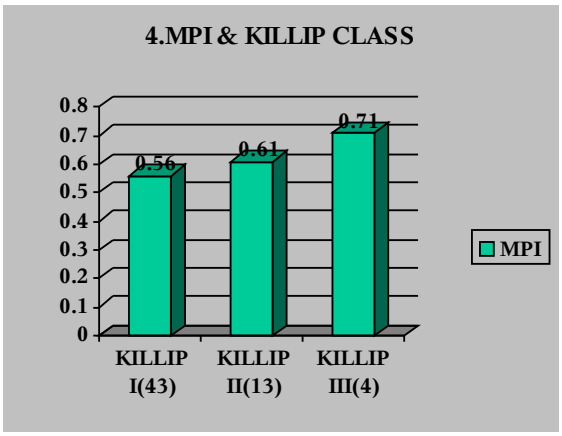
Statistically no difference seen in sex distribution of various parameters.

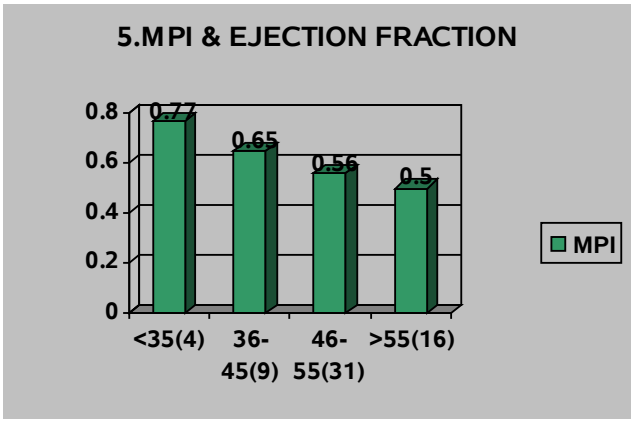
MYOCARDIAL PERFORMANCE INDEX (MPI) AND PARAMETERS IN PATIENTS

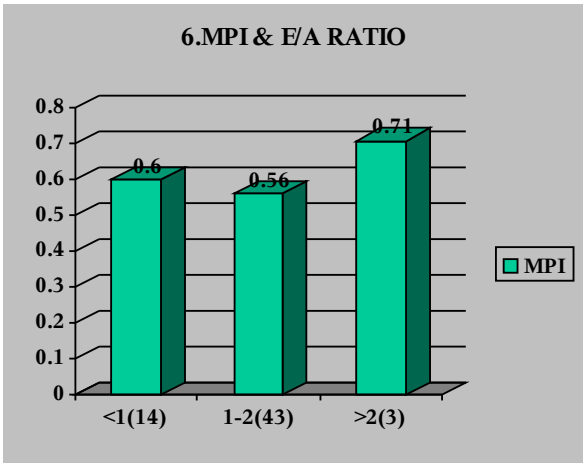


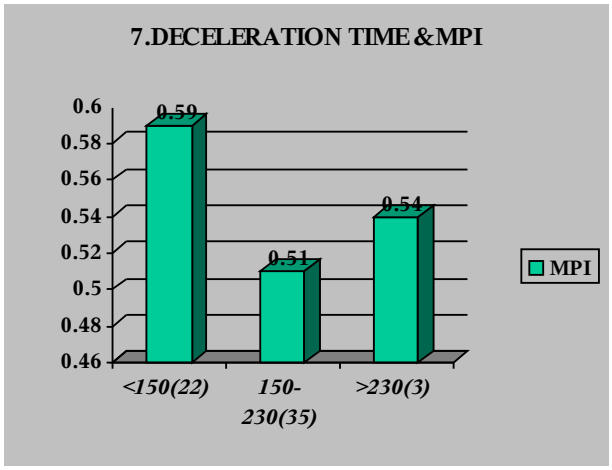


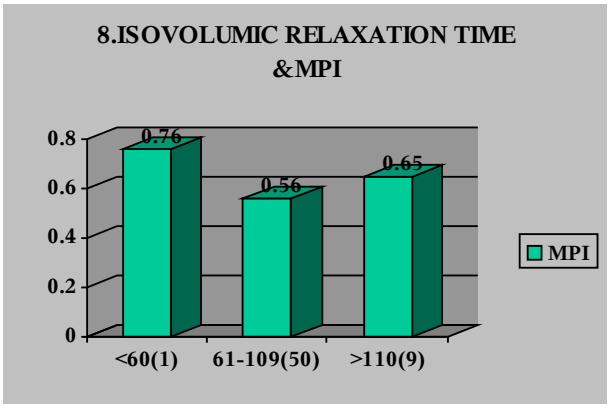


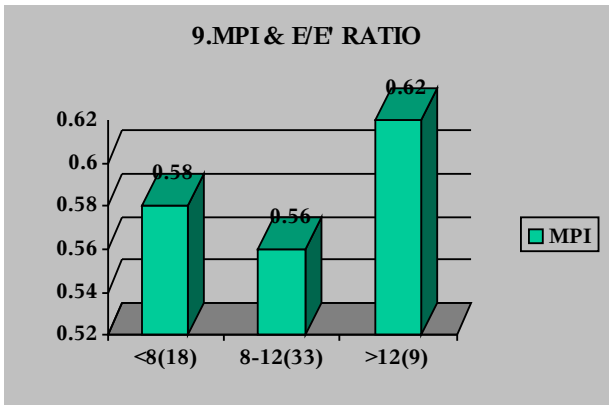

4.MPI & KILLIP CLASS
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.56
0.61
0.71
KILLIP I(43)
KILLIP II(13)
KILLIP III(4)
MPI

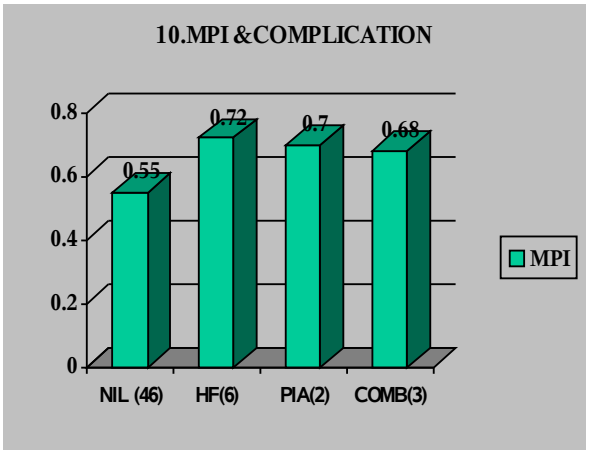

5.MPI & EJECTION FRACTION
0.8
0.6
0.4
0.2
0
0.77
0.65
0.56
0.5
<35(4)
36-45(9)
46-55(31)
>55(16)
MPI

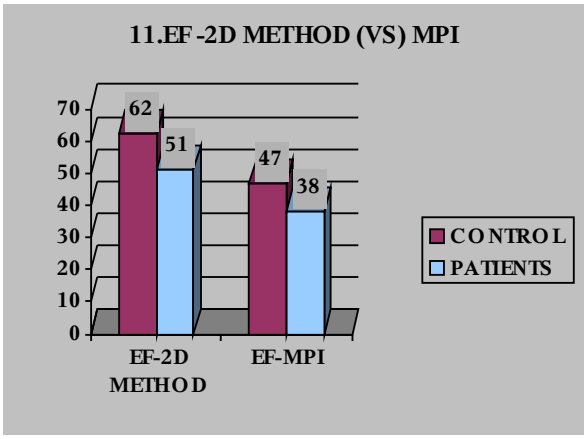

6.MPI & E/A RATIO
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.6
0.56
0.71
<1(14)
1-2(43)
>2(3)
MPI


7.DECELERATION TIME &MPI
0.6
0.58
0.56
0.54
0.52
0.5
0.48
0.46
0.59
0.51
0.54
<150(22)
150-230(35)
>230(3)
MPI


8.ISOVOLUMIC RELAXATION TIME &MPI
0.8
0.6
0.4
0.2
0
0.76
0.56
0.65
<60(1)
61-109(50)
>110(9)
MPI


9.MPI & E/E' RATIO
0.62
0.6
0.58
0.56
0.54
0.52
0.58
0.56
0.62
<8(18)
8-12(33)
>12(9)
MPI


10.MPI &COMPLICATION
0.8
0.6
0.4
0.2
0
0.55
0.72
0.7
0.68
NIL (46)
HF(6)
PIA(2)
COMB(3)
MPI


11.EF -2D METHOD (VS) MPI
70
60
50
40
30
20
10
0
62
51
47
38
EF-2D METHOD
EF-MPI
CONTROL
PATIENTS

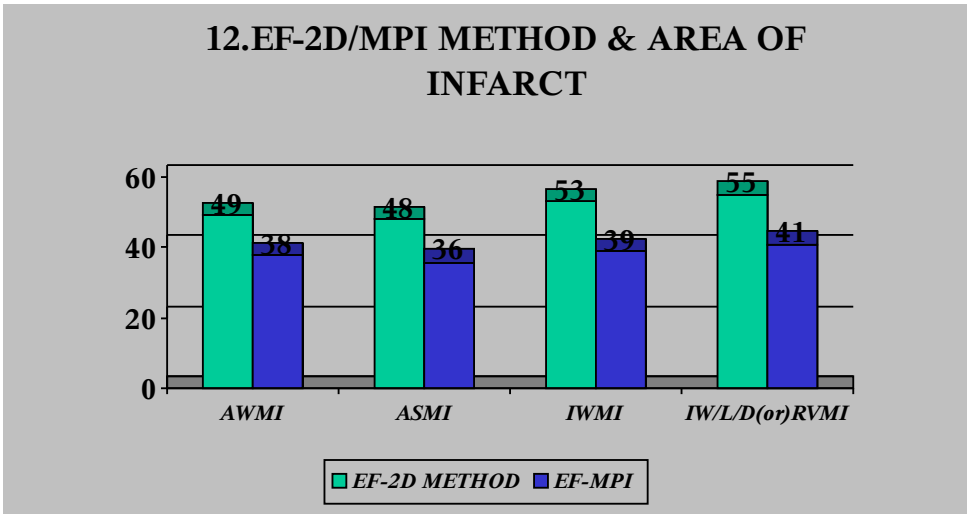

12.EF-2D/MPI METHOD & AREA OF INFARCT
60
40
20
0
49
38
48
36
53
39
55
41
AWMI
ASMI
IWMI
IW/L/D(or)RVMI
EF-2D METHOD
EF-MPI

TABLE 4 : STATISTICAL SIGNIFICANCE OF MPI VS PARAMETRS

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Discussion

We studied 60 patients and 30 controls. They were studied on the basis of detailed history with focus on risk factors and chest pain duration, detailed Echocardiographic analysis was done in all the patients and controls, in hospital complications such as heart failure and post infarct angina were noted.

Clinical and Echo parameters in Patients versus controls:

Among the 60 patients, 43 were male patients and 17 were female patients. Out of 30 controls 20 were males and 10 were females. When comparing patients with controls, the mean age of patient population was 50±12years while that of controls was 49±13 years. While the mean age of male patients were 48± 11 as against 56±12 in female patients, males in our study were younger than female patients, which are in consistent with the general pattern of coronary artery disease. Mean systolic BP was 128±24 in patients as against 116±8 in controls. While Diastolic Blood pressure in patients was 86±14 as against 77±5 in controls, although systolic and Diastolic BP was slightly higher in patients than in controls statistically it was not significant.

We found that LV dimension and volumes in diastole and systole were significantly increased in patients, when compared to controls, while LVDd was 4.9 ±0.5 as against 4.5 ±0.4 in controls, while that of LVDs was 3.5 ±0.6 as against 3 ±0.3 in controls. Similarly EDV was 109.9 ±27.3 in patients as against 94.8 ±15.0 in controls. ESV was 54.67±20.5 as against 35.1±7.1 in controls.

Ejection Fraction in patients was significantly lower than in controls, while it was 50.9 ± 8.3 in patients as against 62.2 ± 3.9 in controls. Similarly Fractional shortening(FS) was also significantly lower in infarction patients when compared with controls, while it was 28.1±6 versus 33.7±4.6 in patients and controls respectively. Significant reductions in both parameters were in consistent with the depressed systolic function occurring in MI patients. Ejection time was found to be significantly reduced in patients than in controls while it was 238.0±24.9 in patients as against 268.7±24.9 in controls again reflecting depressed systolic function.

There was not much significant difference in E, A, E/A ratio in Myocardial infarction patients when compared with controls. Deceleration time was found to be significantly lower in patients with MI than in controls. While DT was 162 ±3.6 in patients when compared to controls with 188.1±29.4.This reductions in DT reflects elevated filling pressure found in MI patients' secondary to diastolic function abnormalities.

IVRT and ICT were found to be significantly elevated in MI patients than in controls. While IVRT was 92.17±17.1 as against 81.2±15.3 and ICT was 48.8±22.2 and 24.3±10 in patients and controls respectively.

Similarly E/E'ratio, another Echo parameter which has got correlation with filling pressure found to be elevated significantly in MI patients than in controls. While it was 10±3.7 in MI patients as against 7.8±1.9 in controls.

While MPI, an index of combined systolic and diastolic functions was found to be elevated significantly in MI patients than in controls, while it was 0.60+/-0.13 in patients as against 0.39±0.68 in controls.

Infarct area and parameters

When we analyzed clinical and echo parameters by area of the infarct, we found that pulse rate was significantly lower in inferior infarct when compared to anterior infarcts, while it was 66±9,78±12 in inferior and inferior/posterior or lateral or RVMI, as against 91±15, 92±16 in anterior wall and anterior septal MI respectively. This is in consistent with the established finding of increased vagal tone with bradycardia seen in inferior infarcts. while

analyzing Blood pressure we found that there was not much difference between the infarct area and BP statistically.

We have also found that there was not much statistical difference in thrombolysis treatment among different infarct areas. Out of 60 patients, 45 patients (75%) were thrombolysed in our study, while 15 patients were not thrombolysed mainly because of late presentation. When analyzing chest pain duration prior to admission we found that inferior wall MI patients were admitted late when compared to other infarcts.

When analyzing the killip class of patients we found that patients with anterior infarcts had higher killip class than inferior infarcts, while the mean killip class is 1.44±0.62 and 1.58±0.77 in anterior wall and antero septal MI respectively. It was 1 &1.10±0.3 in inferior &inferior / lateral or posterior or RVMI respectively.

When analyzing the LV dimension and volume there was not much difference between infarcts in different areas. End systolic volume was found to be significantly higher in anterior infarcts than in inferior infarcts. But it did not attain statistical significance

Ejection Fraction was found to be significantly lower in anterior infarcts than in inferior infarcts while it was 48.9±8.8 & 48.34±7.8 in anterior and anteroseptal MI respectively, when compared to 53.13±8.03 &55.3±6.87 in inferior and inferior /lateral or posterior or RVMI respectively.

On analysis of E, A, E/A, IVRT, E/E' ratio there was not much difference among the different areas of infarct. But Deceleration time (DT) was found to be decreased in anterior infarcts than in inferior infarcts, while DT was 149.9±30.8 &155.9±33.3 in anterior and anteroseptal MI respectively. It was 164.3 ± 21&180.82 ± 42.3 in inferior infarct and inferior/lateral or posterior or RVMI, respectively although the difference was statistically not significant.

When analyzing isovolumetric contraction time it was found to be significantly elevated in AnteroseptalMI when compared to other areas of infarcts.

On analysis of myocardial performance index (MPI) we found that MPI was significantly higher in anterior infarcts than in inferior infarcts while it was 0.62±0.07 in anterior and 0.66±0.18 in anteroseptalMI as against 0.58±0.11 in inferior and 0.52±0.10 in inferior /lateral or posterior or RVMI respectively.

To summarize we found that there was significant reduction in Ejection Fraction, elevation of MPI, higher killip class, higherICT in anterior infarcts when compared to inferior infarcts, and the pulse rate was significantly lower in inferior infarcts than in anterior infarcts. These finding indicates that impairment of the systolic function is much more in anterior infarcts than in inferior infarcts.

MYOCARDIAL PERFORMANCE INDEX AND CLINICAL, ECHO PARAMETER IN MI PATIENTS

When we analyzed MPI in relation to parameters like age, sex, chest pain duration, and Thrombolytic status we found that there was not much difference statistically.

MPI and Killip class

Among the 60patients 43 (71.7%) were in Killip class I, 13(21.7%) belongs to Killip class II, and 4 (6.6%) belongs to Killip class III.When we analyzed the killip class of the patients and its relation to MPI we found that MPI was significantly (P= 0.007) higher when the killip class of the patients increased, while it was 0.56±0.10 in class I, 0.61±0.17 in class II, 0.71±0.2 in class III.Our findings were similar to those observed by **Karvonish ht et al,**[25] in their study they found that MPI was higher in killip class II & III, compared to Killip class I(MPI of 0.68 in Killip class II & III as against 0.34 in killip class I). It is a well established fact that killip class correlates with possible MI outcomes, since MPI reflects the killip class it

can also be used for assessing post MI out come. Higher MPI is associated with poor outcomes.

Myocardial performance index and Systolic function parameters:

LV Dimension and volume

We found in our study that there was statistically significant correlation between MPI and LV dimension, volumes. With an increase in LV dimension & volumes there was increase in MPI. Our findings were similar to that of **Lavine et al** [33] finding, in their study they found that MPI was significantly higher in patients, who developed heart failure, with increased LV dimension & decreased Ejection fraction.

EJECTION FRACTION

When we analyzed the relationship of Ejection fraction with myocardial performance indices we found that with decreasing level of EF, there was an increase in MPI, while it was 0.50±0.07 in patients with EF >55,0.56±0.10 in patients with EF of 46-55,0.65±0.16 in patients with EF of 36-45, and it was 0.77±0.17 in patients with EF<35.This statistically significant (P= 0.000) inverse correlation of MPI with EF implies that MPI has got good correlation with reduction in LV systolic function, similarly MPI was found to be correlated well with other systolic function parameter like FS (P =0.006),ET (0.000) also.

MPI and Diastolic function parameter

When we analyzed the relationship of MPI with diastolic function parameter like E/A ratio, IVRT, DT, E/E'ratio, we found that MPI was significantly associated with abnormalities in all the above mentioned diastolic function parameters except E/A ratio.

When analyzing E/A ratio with MPI we found that MPI was higher when the E/A ratio was less than 1or more than 2. While MPI was 0.60±0.17 in patients with E/A ratio of <1, it was 0.56±0.11 when E/A was between 1 – 2, and it was 0.71± 0.02 when E/A ratio was >2,

Although MPI was high when there was abnormality associated with E/A ratio but it does not attained statistical significance.

When we analyzed Deceleration time with myocardial performance index, MPI was significantly (P= 0.001) increased when there was abnormality in Deceleration time. MPI was 0.59±0.18 when DT was lesser than 150, when compared to 0.51±0.10 when DT was between 150-230.Our findings were similar to **Poulsen et al's** [32]finding, they also found in their study that MPI was significantly higher in patient's with Deceleration time of < 140 msec.They had concluded in their study that decreased Deceleration time and elevated MPI were independent predictors of out come.

When we analyzed isovolumetric relaxation time with MPI, we found there was significant (P= 0.012) increase in MPI when IVRT was prolonged while MPI was 0.56±0.11 when IVRT was between 61-109, while it was 0.65±0.2 when IVRT was >110 hence MPI correlates significantly with prolonged IVRT.

On analyzing E/E' ratio with myocardial performance index, we found that the MPI was significantly (P=0.003) associated with elevated E/E' ratio. When the E/E' ratio was more than 12, MPI was found to be 0.62±0.11 as against 0.58± 0.08when E/E' ratio was less than 8.

Myocardial performance index and complication in patients

On analyzing the relationship of patients with complications such as heart failure, post infarction angina, we found that patients with complication following myocardial infarction had significantly elevated MPI, when compared with patients who did not have any complication. MPI was 0.55± 0.1 in patients with out any complication, while it was 0.72 ± 0.3 in patients with heart failure, 0.70 ± 0.1 in patients with post infarction angina and MPI was 0.68 ± 0.08 in patients with combination of heart failure and post infarction angina. Hence we found from our study that MPI has got good correlation with complication

following myocardial infarction, so it can be used for prognosis following myocardial infarction

Myocardial performance index and Risk factors

In our study out of 43 males 34 were smokers, While 6 patients had diabetes, 4 had Hypertension, 11 had combination of risk factors such as HT and DM, DM and smoking, HT and smoking or HT, DM, Smoking.

When analyzing the relationship of risk factors such as Hypertension, Diabetes, smoking with MPI, We found that the level of MPI was found to be higher in patients with risk factors than in patients with out any risk factors While MPI was found to be 0.63± 0.3 in patients with Hyper tension, while MPI was 0.55 ±0.1 in patients diabetes, it was 0.61 ± 0.13 in patients with smoking and MPI was 0.59 ± 0.1 in patients with combination of risk factors, When compared to patients with out risk factors in whom it was only 0.51 ±0.1.

To conclude, We found from our study that MPI was found to be higher in Myocardial infarction patients especially in Anterior Infarcts, and MPI also has got good correlation with Killip class , Systolic function parameters like LV dimension, volume, ejection fraction and fractional shortening as well as Diastolic function parameters like Deceleration time, IVRT,E/E' ratio. MPI was also found to be higher in patients who developed complication such as Heart failure and post infarction angina than in patients who did not have any complication. Hence MPI can be used for assessing LV function as well as for predicting In hospital out come in acute ST elevation myocardial infarction patients.

Ejection fraction derived from MPI versus EF by 2D method

When we analyzed the EF derived from Myocardial performance index by Lax et al formula (EF = 0.60 – (0.34 x MPI)) with that of EF by 2 D (modified Simpson's method). We found that EF by MPI method significantly under estimated the EF both in patients and in

controls. While the mean EF by MPI method was 47 as against 62 by 2D method in controls and the mean EF by MPI method were 38 as against 51 by 2D method in patients. Similarly when analyzing the EF in different areas of infarct, we found that EF derived by Lax et al formula under estimated the EF when compared to 2D method in all patients regardless of the area of infarct. While the EF by MPI method was 38 in anterior, 36 in antero septal, 39 in inferior and 41 in inferior / lateral or posterior or RV myocardial infarction as compared to 49 in anterior, 48 in antero septal, 53 in inferior and 55 in inferior with lateral or posterior or RV myocardial infarction by 2D method.

Hence although EF derived from MPI method reflected the downward trend of Ejection fraction in patients regardless of the area of infarct and also in controls, MPI method was found to under estimate the EF significantly when compared to that by 2D method. Our finding were similar to that of TORSTEIN HOLE et al,[35] they also found only moderate agreement between EF by MPI and 2D methods, hence EF estimation by MPI methods needs further large scale studies prior to be recommended as a method to estimate EF in patients in whom EF estimation by 2D method was difficult due to technical or other problems.

CONCLUSION

The following conclusions were derived from our study

- Myocardial performance index was found to be significantly elevated in myocardial infarction patients than in controls.
- Myocardial performance index elevation was found to be significantly more in anterior infarcts than in Inferior infarct patients.
- Myocardial performance index has got a good correlation with systolic as well as diastolic function parameters. While Myocardial performance index was found to have a significant inverse relationship with Ejection fraction, it was also found to have significant relationship with abnormalities in diastolic function parameters like Deceleration time, Isovolumic relaxation time and E/E' ratio.
- Myocardial performance index has got a significant positive correlation with Killip class and Risk factors such as Hypertension, diabetes and smoking.
- Myocardial performance index was found to be significantly elevated in patients who developed in hospital complications like Heart failure, Post infarction angina than in patients who did not have any complications. Hence myocardial performance index can be used as a prognostic indicator for predicting out comes.
- There was significant underestimation of the Ejection fraction by Lax et al formula using myocardial performance index when compared with the Ejection fraction by 2D method. Hence Ejection fraction estimation from myocardial performance index using Lax et al formula needs further large-scale studies before it can be used for clinical purpose.

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PROFORMA

MYOCARDIAL PERFORMANCE INDEX IN MI

NAME: AGE: SEX:

OCCUPATION: C.D.NO:

ADDRESS:

SOCIO ECONOMIC STATUS

RISK FACTOR PROFILE:

HYPERTENSION SMOKING OBESITY (BMI)

DIABETES HYPER LYPIDEMIA PREVIOUS CAD

FAMILY HISTORY OF CAD PERSONALITY

CLINICAL PROFILE:

CHEST PAIN DURATION

VITAL PARA METERS: P R: BP:

KILLIP CLASS

TIMI SCORE

INFARCT TERRITORY

THROMBOLYTIC STATUS

DRUGS USED

COMPLICATION

CHF ARRYTHMIAS PIA DEATH

INVESTIGATION:

BLOOD SUGAR: CARDIAC ENZYMES:

ECG:

CHEST PA:

ECHO CARDIO GRAPHY:

SYSTOLIC FUNCTION: LV DIMENSION; LVDd: LVDs:

EDV: ESV: EF:

FS: RWMA: WMSI:

DIASTOLIC FUNCTION:

TRANS MITRAL FLOW PATTERN: E A E/A RATIO:

DT IVRT

PULMONARY VENOUS FLOW PATTERN: S/D RATIO

PV 'a' VELOCITY

TISSUE DOPPLER E' A' E/E'RATIO

SYSTOLIC AND DIASTOLIC FUNCTION:

TEI INDEX: MEASURE MENT A (END TO BEGINNING OF MITRAL FLOW)

MEASURE MENT B (ET)

(A-B/B)

IVRT IVCT

MPI- EF = (0.60- (0.34 * MPI)

GLOSSARY

STEMI: ST ELEVATION MYOCARDIAL INFARCTION

LVDd :LEFT VENTRICULAR DIAMETER -DIASTOLE

LVDs: LEFT VENTRICULAR DIAMETER-SYSTOLE

CHD: CORONARY HEART DISEASE

DM: DIABETES MELLITUS

LVH:LEFT VENTRICULAR HYPERTROPHY

M.I: MYOCARDIAL INFARCTION

EF: EJECTION FRACTION

EDV: END –DIASTOLIC VOLUME

ESV: END- SYSTOLIC VOLUME

LVEF: LEFT VENTRICULAR EJECTION FRACTION

ESPVR: END SYSTOLIC PRESSURE VOLUME RELATIONSHIP

PFR: PEAK FILLING RATE

LBBB: LEFT BUNDLE BRANCH BLOCK

IVCT:ISOVOLUMETRIC CONTRACTION TIME

RVET: RIGHT VENTRICULAR EJECTION TIME

TDI: TISSUE DOPPLER IMAGING

DT: DECELARATION TIME

NYHA: NEWYORK HEART ASSOCIATION

AMI: ACUTE MYOCARDIAL INFARCTION

LVEDP: LEFT VENTRICULAR END DIASTOIC PRESSURE

LVEDV: LEFT VENTRICULAR END DIASTOLIC VOLUME

FS: FRACTIONAL SHORTENING

TEE: TRANS ESOPHAGEAL ECHOCARDIOGRAPHY

WMSI: WALL MOTION SCORE INDEX

IVRT:ISOVOLUMIC RELAXATION TIME

MR: MITRAL REGURGITATION

MPI : MYOCARDIAL PERFORMANCE INDEX