# Echocardiographic assessment of left ventricular diastolic dysfunction in ischemic heart disease

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#### Summary:

Background: The most common underlying cause of Left Ventricular (LV) diastolic dysfunction is myocardial ischemia. Diastolic dysfunction has been reported in up to 90% of patients with coronary artery disease. Transient and sustained ischemia causes profound alterations in LV diastolic function that may result in severe heart failure symptoms in patients who have otherwise well-preserved LV systolic function. For patients with chronic ischemic heart disease, echocardiography is supported as a valuable test for assessing global left ventricular function.

Fac Med Baghdad 2011; Vol. 53, No. 4 Received June. 2010 Accepted Dec. 2010

Subjects & Methods: One hundred sixty five(165) subjects with a history of ischemic heart disease (85 males, and 80 females :patient group) with a mean age of 57± 10 years, in addition to sixty (60) healthy subjects of either sex served as controls(28 males, and 32 females) with a mean age  $56 \pm 10$  years were studied. This study was performed during the period from December 2006 until February 2008, at the echo unit of the Iraqi centre for heart diseases and the echo unit of the cardiac care unit at Baghdad / medical city teaching hospital. The plan of the study consisted of the following steps; Case history, Clinical & physical examination, Electrocardiography (ECG). Chest x-ray and Echocardiographic examination.

Results: Higher percent of IHD patients involved in this study developed Diastolic dysfunction (73%), while (27%) discovered to have neither systolic nor diastolic dysfunction by echocardiography. Patients who found to have diastolic dysfunction were those with nonSTEMI (36%), while (31%) were those with chronic stable angina as compared to STEMI (19%) and unstable angina (14%).

Conclusion: the results of this study, offer evidence that; higher percent of patients with IHD involved in this study developed diastolic dysfunction and they were assessed using echocardiographic parameters (M-mode and doppler).

Key words: Diastolic dysfunction, echocardiography, ischemic heart disease.

## Introduction:

Diastolic dysfunction refers to a condition in which abnormalities in mechanical function are present during diastole. Abnormalities in diastolic function can occur in the presence or absence of a clinical syndrome of heart failure and with normal or abnormal systolic function. Therefore, whereas diastolic dysfunction describes an abnormal mechanical property, diastolic heart failure describes a clinical syndrome. The mechanisms that cause abnormalities in diastolic function that lead to the development of diastolic heart failure can be divided into factors intrinsic to the myocardium itself (myocardial) and factors that are extrinsic to the myocardium (extramyocardial;), cellular and extracellular mechanisms, and neurohumoral activation each play a role in the development of diastolic heart failure caused by ischemia, pressure-overload hypertrophy, and restrictive and hypertrophic cardiomyopathy(1).

The most common underlying cause of LV diastolic

been reported in up to 90% of patients with coronary artery disease. Transient and sustained ischemia causes profound alterations in LV diastolic function that may result in severe heart failure symptoms in patients who have otherwise wellpreserved LV systolic function(2).

Poulsen (3), stated that Diastolic dysfunction has been recognized during the early as well during the post-MI phase with or without left ventricular systolic dysfunction. In the acute phase of MI both an abnormal relaxation pattern and restrictive left ventricular filling pattern are present. In addition, myocardial ischemia, cell necrosis, microvascular dysfunction, and regional wall motion abnormalities will influence the rate of active relaxation of the ventricles. Abnormal relaxation filling is the most pronounced filling pattern after one year post MI which might be related to the remodeling process including compensatory hypertrophy. scarring of the infarct zone leading to a non-uniform relaxation of the left ventricle (4). Left ventricular remodeling process following the very early phase post MI includes the scarring

dysfunction is myocardial ischemia. Diastolic dysfunction has

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process with collagen deposition in the infarcted and non-infarcted myocardium. The extent and quality of the repair process involving collagen deposition are believed to influence the remodeling process (5). As a consequence myocardial stiffness increases and diastolic filling decreases. This study was aimed to assess and evaluate the left ventricular diastolic dysfunction in ischemic heart disease among Iraqi population using echocardiographic parameters.

# Subjects and methods:

One hundred sixty five(165) subjects with a history of ischemic heart disease (85 males, and 80 females ;patient group) with a mean age of  $57\pm10$  years, in addition to sixty (60) healthy subjects of either sex served as controls(28 males, and 32 females) with a mean age  $56\pm10$  years were studied. This study was performed during the period from December 2006 until February 2008, at the echo unit of the Iraqi centre for heart diseases and the echo unit of the cardiac care unit at Baghdad / medical city teaching hospital. The patients involved in this study were:

Patients with Ischemic heart disease (IHD) admitted in the cardiac care unit of Baghdad teaching hospital, the Iraqi centre for heart diseases at the specialist surgical hospital and at the medical ward in both hospitals. Also outpatients referred from specialist cardiologist for echocardiographic examination at the echo unit in both hospitals, all patients involved had no history of hypertension or diabetes mellitus. The plan of the study consisted of the following steps:

Case history. Clinical & physical examination, Electrocardiography (ECG), Chest x-ray and Echocardiographic examination.

The echocardiography was performed for each subject using Phillips (Envisor C) system (USA) echocardiographic device, with a transducer operating at 2.0 to 3.5 MHz. Left ventricular internal dimensions at diastole (LVIDd) and systole (LVIDs). interventricular septum thickness, posterior wall thickness. ejection fraction (EF %) and fractional shortening (FS%) were measured, using M-mode echocardiography to assess left ventricular systolic function. While, the left ventricular diastolic function was determined by pulsed Doppler echocardiography that involved recordings of transmitral inflow velocities from a 2x2mm sample volume positioned at mitral valve leaflet tips traced along the black-white interface. to measure peak E and A wave velocities and their ratio (E/A ratio), E-wave deceleration time (DT) and isovolumic relaxation time (IVRT), patients who discovered to have systolic dysfunction were excluded from the study.

**Statistical analysis:** All calculations and analyses were performed using Statistical Package for the Social Sciences, (SPSS version 11.5 for windows, SPSS Inc., Chicago, Illinois) computerized Program. The level of statistical significance

was defined as P value <0.05, which was obtained by comparing the calculated t-value to the tabulated t-value at 95% confidence interval. The student (t) test was used to compare variables of patient groups with that of the control.

#### **Results:**

The demographic characteristics of the 225 subjects involved in this study are shown in table (1). The characteristics of (165 patients) regarding their age and gender were compared with that of the control group (60 subjects). It is clear from this table that there was no significant differences in age and gender between the patient and control groups (P = 0.496, P = 0.235) respectively

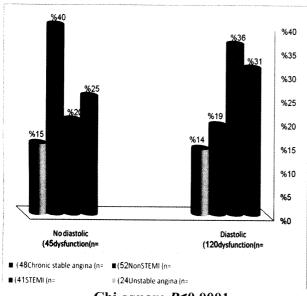
Table 1: The Demographic characteristics of patient and control groups.

	Normal San	1 mg (mg mg)	Gender		
	Number (%)	Age (years) Mean± SD	Males No. (%)	Females No. (%)	
Group1 (DD)	120 (73)	57 ± 9 55 (46)		65(54)	
Group2 (ND)	45 (27)	57 ± 12	30(67)	15 (33)	
Control	60	56 ± 10	28 ( 47)	32 (53)	
Total	225	56 ± 10	113(53)	112(47)	
P value		0.496	0.235		

DD: Diastolic Dysfunction.

ND: No Diastolic Dysfunction.

Higher percent of patients in group1 who found to have diastolic dysfunction were those with nonSTEMI (36%), while 31% were those with chronic stable angina as compared to those with STEMI (19%) and unstable angina (14%). Patients in group2 who found to have no diastolic dysfunction higher percent of them were those with STEMI (40%) as compared to those with chronic stable angina (25%), non STEMI (20%) and unstable angina (15%). As shown in figure (1) below:



From the comparison of M-mode parameters between patient groups with diastolic dysfunction (DD) and control subjects (table 2), there were a significant statistical increases in the left ventricular internal diameter in diastole (LVIDd), left ventricular internal diameter in systole (LVIDs), interventricular septum thickness (IVS) and posterior wall thickness, while there were a significant statistical decreases in fractional shortening (FS%), and in ejection fraction (EF%), (p< 0.05). Also the Comparison between Doppler transmitral inflow parameters showed that there were a significant decreases in peak E velocity and E/A ratio (p< 0.05). While, there were a significant increases in peak A velocity, Deceleration time (D time) of E wave and in isovolumic relaxation time (IVRT), (p< 0.05).

Chi-square P<0.0001

Figure 1: The different types of ischemia among patient groups.

Table (2): Comparsion of Echocardiographic parameters between patient groups with diastolic dysfunction(DD) and control subjects.

M-mode &Doppler Parameters	STEMI (N=YY)	NonSTEMI (N=٤٣)	Unstable angina (N=VV)	Chronic stable A (N=TV)	Control (N=60)	P Value
LVIÐd (cm)	4.81±0.76*	4.97±0.60÷	4.87±0.40*	4.89 ± 0.53°	4.77 ± 0.35	0.047
LVIDs (cm)	3.03±0.70°	3.20±0.58÷	3.04±0.62*	3.16 ± 0.45°	2.79 ± 0.34	0.00018*
IVS (cm)	1.23±0.32*	1.20±0.31÷	1.05±0.15*	1.06±0.20°	$0.89 \pm 0.13$	0.0002*
Posterior wall thickness(cm)	1.24±0.31*	1.14±0.25÷	1.04±0.25*	1.07±0.21°	$0.88 \pm 0.11$	0.0003*
Fractional shortening(FS½)	36±8.07*	35±7.35+	36±5.56*	36 ± 5.76°	39± 5.33	0.0003*
Ejection Fraction (EF%)	62±7.96*	67±6.49÷	65.60±7.70*	66 ± 7.04°	69± 6.08	0.0004*
Peak E velocity (cm/s)	68±20°	61±14†	63±5*	62 ± 10°	79 ± 4	0.0006*
Peak A velocity (cm/s)	102±21°	91±15†	97±17*	101 ± 10°	64 ± 6	0,0002*
E / A ratio	0.65±0.11*	0.67±0.11+	0.65±0.12*	0.61 ± 0.13°	1.23 ± 0.13	0.0005*
D time of E wave( msec.)	247±27°	237±25†	207±28*	245 ± 23°	180 ±11	0.0001*
IVRT (msec.)	145±23*	134±15†	123±28*	142 ± 11°	100 ± 6.8	0.0001*

Values were expressed as Mean ±SD.

Table (3) showed that there were no significant statistical differences in echocardiographic parameters of patient groups who found to have no diastolic dysfunction when compared with those of the control subjects.

<sup>&#</sup>x27;.†.♦. significant P value ≤ 0.05.

Table (3): Comparsion of Echocardiographic parameters between patient groups with no diastolic dysfunction (ND) and control subjects.

M-mode & Doppler Parameters	STEMI (N=18)	NonSTEMI (N=9)	Unstable angina (N=7)	Chronic stable A (N=H)	Control (N=60)	P Value
LVIDd (cm)	4.79± 0.34	4.78± 0.23	4.81± 0.42	4.79± 0.6	$4.77 \pm 0.35$	0.72
LVIDs (cm)	2.88 ±0.25	2.81 ± 0.2	2.8 ± 0.5	2.82 ± 0.4	2.79 ± 0.34	0.64
IVS (cm)	0.91 ±0.16	0.9 ± 0.21	$0.92 \pm 0.6$	$0.9 \pm 0.8$	$0.89 \pm 0.13$	0.09
Posterior wall thickness(cm)	0.9 ± 0.15	$0.89 \pm 0.2$	$0.89 \pm 0.8$	$0.89 \pm 0.6$	$0.88 \pm 0.11$	0.18
Fractional shortening(FS%)	39 ± 4.24	38 ± 3.4	38 ±0.4	38 ±5.2	39± 5.33	0.54
Ejection Fraction (EF%)	68 ± 6.97	68 ± 6	68 ± 5.4	68 ± 7.4	69± 6.08	0.22
Peak E velocity (cm/s)	80 ± 4.86	80 ± 3	79 ± 3	79 ±6	79 ± 4	0.225
Peak A velocity (cm/s)	65 ± 5.79	66 ± 4.6	65 ± 5.6	65 ± 5	64 ± 6	0.18
E / A ratio	$1.24 \pm 0.1$	$1.25 \pm 0.20$	1.25 ± 0.26	$1.26 \pm 0.3$	$1.23 \pm 0.13$	0.118
D time of E wave ( msec.)	182 ± 16	181 ± 10	182 ± 12	182 ± 14	180 ±11	0.07
IVRT (msec.)	103 ± 10	102 ±9.26	101 ±9.4	101 ±8.2	100 ± 6.8	0.072

Values were expressed as Mean ±SD.

#### Discussion:

Diastolic dysfunction is common in the community and is often unaccompanied by overt symptoms and signs of congestive heart failure. Despite the absence of symptoms, subjects with advanced diastolic dysfunction have accompanying structural abnormalities that reflect an increased risk for adverse cardiovascular outcomes and have a reduced quality of life (6). At the bedside, diastolic dysfunction is difficult to diagnose and to differentiate from systolic dysfunction on the basis of medical history, physical examination, electrocardiography and chest radiography. Two-dimensional and M-mode echocardiography are excellent for diagnosing systolic dysfunction, and Doppler echocardiography has become well accepted as a reliable, reproducible and practical noninvasive method for diagnosis and longitudinal follow-up of patients with diastolic dysfunction. Clinical cardiologists can now comprehend, interpret and prognosticate and treat their patients based on Doppler flow velocity curves (7). Higher percent of IHD patients involved in this study developed Diastolic dysfunction (73%), while (27%) discovered to have neither systolic nor diastolic dysfunction by echocardiography (table 1) Signs of diastolic dysfunction have also been reported in many patient groups with coronary artery disease (6), (7), (8) (5) (9). Niushui et alc 10), found that 66% of patients with stable coronary heart disease had normal diastoric function, 24% chad impaired relaxation (mild diastoric dysfunction) and 10% had pseudonormal or restrictive filling (moderate to severe LV diastolic dysfunction). Compared with patients with normal LV diastolic function, those with LV diastolic dysfunction were older; more likely to have experienced a previous myocardial infarction, stroke, or revascularization. The difference between results of this study and that of Xiushui et al., could be explained by the large number of patients (693) involved in their study as compared to us (165).

Alternatively, the normal values which were taken as standards were obtained from sixty healthy Iraqi individuals matched for age and gender with the patient groups. Thus, they served as a control group for comparison. However, the Echo and Doppler parameter values of our Iraqi control subjects were approximately near the values of (11), (12). It is clear that higher percentage of patients (36%) with nonSTEMI in group1 developed echocardiographic diastolic dysfunction, as compared to those with STEMI and those with unstable angina (figure 1), similar findings were noted by (13)(14)(15). According to Gerber: (14) patients with Acute coronary syndrome (ACS)without ST elevation generally have less extensive myocardial necrosis and are more likely to have an open infarct-related artery or collateral blood supply to the ischemic area. Therefore, it is likely that they also will have a higher incidence of potentially reversible causes of clinical heart failure caused by stunned or hibernating invocardium, diastolic dysfunction, or transient ischemic valvular dysfunction. Shamir et al., (15) stated that

even within the group of patients with non-ST elevation ACS, there was substantial variation in the risk of development of congestive heart failure (CHF). There was an approximate twofold increased risk of development of CHF in patients with NSTEMI compared with patients with unstable angina, suggesting that myocardial cell necrosis is an important determinant for development of CHF in patients with ACS. From the comparison of M-mode echocardiographic parameters (table 2) between patient groups with diastolic dysfunction and control subjects, there were a significant statistical increases in the left ventricular internal diameter in diastole (LVIDd). left ventricular internal diameter in systole (LVIDs), interventricular septum thickness (1VS) and posterior wall thickness. The same results were reported by (16) & (17). Actually, Michael et al (16), stated that the left ventricular hypertrophy plays a major role in the development of diastolic dysfunction in many if not most of patients. Once left ventricular hypertrophy has occurred and left ventricular diastolic pressure begins to rise, myocardial ischemia may play a role in worsening Diastolic dysfunction, even without significant coronary stenoses. Despite that there were a significant statistical decreases in ejection fraction (EF%) and fractional shortening (FS%)when compared with the control subjects (table2). Our patients with diastolic dysfunction had a normal ejection fraction (65%), This suggest that subjects with diastolic dysfunction and an ejection fraction > 50% classified as having an isolated diastolic dysfunction (3). Likewise, other studies stated that left ventricular systolic function remained normal in patients with left ventricular diastolic heart failure (18) (3).

From the results of the present study (table 2). The significant statistical decreases in peak E velocity. E/A ratio and the significant increases in peak A velocity. Deceleration time (D time) of E wave and in isovolumic relaxation time(IVRT) were in agreement with findings reported by(10),(19)&(20). However, patient groups having these findings were diagnosed to have mild diastolic dysfunction of the left ventricle which represents the earliest stage of diastolic dysfunction that involved impaired LV relaxation with initially normal LV filling pressures. Consequently, this impaired relaxation might lead to decreased early filling (E wave) and increased filling with atrial contraction (A wave) in which that the Mitral inflow patterns show an E/A ratio less than L the IVRT is prolonged (100 msec.), with prolongation of the DT 200 msec.(10),(19)(21).

The non significant changes in M-mode and Doppler mitral inflow echocardiographic parameters of patient groups in comparsion with control subjects (table 3) suggest that these patients developed no systolic neither diastolic dysfunction. However, these findings goes with what had been reported by (22) (23) (20).

#### Conclusions:

In conclusion, the results of this study, offer evidence that; higher percent of patients with IHD involved in this study developed diastolic dysfunction and they were assessed using echocardiographic parameters (M-mode and doppler).

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