Usefulness Of Tissue Doppler Imaging For the Diagnosis of Coronary Artery Disease in Patients With Left Bundle Branch Block

Dissertation submitted to
The Tamil Nadu Dr M.G.R Medical University, Chennai
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Certificate

This is to certify that **Dr N.Ganesan**, Post graduate student [2003-2006] in the Department of Cardiology, Government General Hospital Chennai & Madras Medical College, Chennai -03, has done this Dissertation on **"Usefulness Of Tissue Doppler Imaging For the Diagnosis of Coronary Artery Disease in Patients With Left Bundle Branch Block**" under my guidance and supervision in partial fulfillment of the regulations laid down by The Tamil Nadu Dr M.G.R Medical University, Chennai, for DM Cardiology – Branch II examination to be held in February 2006.

Dean

Madras Medical College

Chennai

Prof. Dr.V. Jaganathan., MD.,DM,.

Prof and Head

Department of Cardiology

Government General Hospital &

Madras Medical College

Chennai-03

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Dedicated to

my

Parents

&

Teachers

Abstract

Back ground

Non-invasive diagnosis of LAD stenosis in patients with LBBB is particularly difficult because of the well known ECG limitations, echocardiographic abnormal septal wall motion and possible myocardial scintigraphic antero-septal perfusion defect artifacts. Recent reports show that Tissue Doppler derived Post-systolic motion, an asynchronous contraction occurring during an isovolumetric relaxation period is a sensitive and specific marker for LAD occlusion.

Aim

The aim of this study was to analyze the usefulness of TDI and to characterize PSM in LBBB with or without stenosis of the left anterior descending coronary artery (LAD).

Methods and Materials

28 patients with chest pain with out prior myocardial infarction and complete LBBB were included for the study. Standard Doppler echocardiography and tissue Doppler of both the middle posterior septum and lateral mitral annulus were performed in the apical 4-chamber view. Tissue Doppler-derived septal PSM were measured and all patients underwent coronary angiography within 1 month.

Results:

Angiographically the study populations were divided into two groups. 13 without LAD stenosis and 15 with LAD stenosis.[LAD stenosis > 50%]. The Clinical, ECG and standard doppler diastolic indexes were comparable between the two groups. Septal tissue Doppler showed lower myocardial systolic (Sm) and atrial peak velocities (both $p < 0.05$), a higher PSM (PSM > 2cm/sec; $p < 0.005$), in patients with LAD stenosis. A Sm/PSM ratio < 1 was detected in 93.3 % of patients [14/15] with LAD stenosis (sensitivity 93.3%, specificity 100%). 30 %[4/13] patients without LAD stenosis also had PSM but with a Sm/PSM ratio <1**.**

Conclusion:

Pulsed Tissue Doppler derived high amplitude **Post Systolic Motion and reduced systolic myocardial velocity are** simple, non invasive markers with high sensitivity and specificity to diagnose LAD disease in the presence of LBBB.

The present dissertation is based on the following peer reviewed publications and conference records.

Skulstad H, Edvardsen T, Urheim S, et al. Post systolic shortening in ischemic myocardium: active contraction or passive recoil?

Circulation 2002; 106: 718-24.

Hosokawa H, Sheean FH, Suzuky T. Measurement of post systolic shortening to assess viability and predict recovery of left ventricular function after acute myocardial infarction.

J Am Coll Cardiol 2000; 35: 1842-9.

Jamal F, Kukulski T, D'hooge J, De Scheerder I, Sutherland G. Abnormal post systolic thickening in acutely ischemic myocardium during coronary angioplasty: a velocity, strain, and strain rate Doppler myocardial imaging study.

J Am Soc Echocardiogr 1999; 12: 994-6.

Jelena Celutkienea,*, George R. Sutherlandb, Aleksandras Lauceviciusa, Diana Zakarkaitea, Alfredas Rudysa, Virginija Grabauskienea. Is post-systolic motion the optimal ultrasound parameter to detect induced ischemia during dobutamine stress echocardiography?

European Heart Journal (2004) 25, 932–942

Stig Urheim, MD; Thor Edvardsen, MD; Kjetil Steine, PhD, MD; Helge Skulstad, MD; Erik Lyseggen, MD; Olaf Rodevand, PhD, MD; Otto A. Smiseth, FACC, FESC, PhD, MD. Post-systolic shortening of ischemic myocardium – a mechanism of abnormal intraventricular filling

Am J Physiol Heart Circ Physiol (February 6, 2003).

Rodolfo Citro, M.D.*, and Maurizio Galderisi, M.D., F.E.S.C. Myocardial Post systolic Motion in Ischemic and Not Ischemic Myocardium: The Clinical Value of Tissue Doppler

Echocardiography Volume 22 Issue 6 Page 525 - July 2005

Saha S k et al Differential diagnoses of left bundle-branch block using tissue Doppler echocardiography: importance of both right and left ventricular systolic velocities and iso-volumic relaxation times. ;

Euro Echo 2004

Barletta G, Del Bene R, Lo Sapio P, et al. Post-ejection thickening as a marker of viable myocardium. An echocardiographic study in patients with chronic coronary artery disease.

Basic Res Cardiol 1998; 93: 313-24.

“The papers included in this dissertation have had minor changes compared
to the published versions. They have been formatted to provide a common
layout for all material and a few clerical errors have been corrected”

LIST OF ABBREVIATIONS

2-D	Two-dimensional
Am	Late diastolic velocity
ECG	Electrocardiogram
ECHO	Echocardiography
EF	Ejection fraction
Em	Early diastolic velocity
E-wave	Early diastolic wave
IVC	Isovolumic contraction
IVR	Isovolumic relaxation
LA	Left atrium / atrial
LBBB	Left Bundle Branch Block
LV	Left ventricle / ventricular
LAD	Left anterior descending coronary artery
M-mode	Motion mode
MPI	Myocardial Perfusion imaging
PCT	Precontraction time

PSM	Post systolic Motion
PSS	Peak systolic strain
PSV	Peak systolic velocity
Sm	Systolic Myocardial Velocity
TDI	Tissue Doppler imaging
RTm	Relaxation Time

Introduction

The non-invasive detection of CAD in patients with resting LBBB poses a particular problem. Although patients with LBBB have a high prevalence of CAD, LBBB also frequently occurs in non-ischemic conditions. Left bundle branch block (LBBB) is often associated with systemic arterial hypertension, aortic valve disease, and dilated cardiomyopathy[1].

Long term prognosis of patients with LBBB depends on underlying CAD[1]. ST segment changes on exercise ECG are non-diagnostic in these patients, and abnormal septal motion associated with the conduction abnormality may reduce the diagnostic accuracy of stress/rest regional wall motion comparisons using radionuclide ventriculography or echocardiography.[2, 16-25]

A number of authors have proposed that the development of a user-friendly quantitative approach should overcome the limitations of currently available noninvasive techniques. Several recent reports have shown that Tissue Doppler myocardial velocity imaging to be a sensitive alternative to the present echocardiographic and scintigraphic imaging techniques to evaluate CAD in patients with LBBB[8-16].

Prior investigations have shown that changes in tissue doppler derived parameters like post-systolic deformation and tissue velocities are sensitive and early markers of acute ischaemia.[8–12].However, there is a lack of data concerning these parameters (*post-systolic motion)* and its diagnostic value. This prospective observational study explored both Post systolic motion and other systolic, diastolic parameters as a marker of ischemia in patients with LBBB.

Background

Coronary artery disease remains the leading cause of mortality and morbidity. Earlier thought to be a disease of the modern world, it has been found to be equally or even more prevalent in underdeveloped and developing countries. This disease is, unfortunately, being witnessed in the younger population also. It remains the most common single cause of mortality and morbidity in men below 65 years of age. For early diagnosis of CAD, before the occurrence of major mishap like myocardial infarction, surface electrocardiogram remains a cheap, cost effective, and widely available and applicable approach. However the presence of Left bundle branch block significantly reduces the diagnostic power of ECG [3-5].

Left bundle branch block

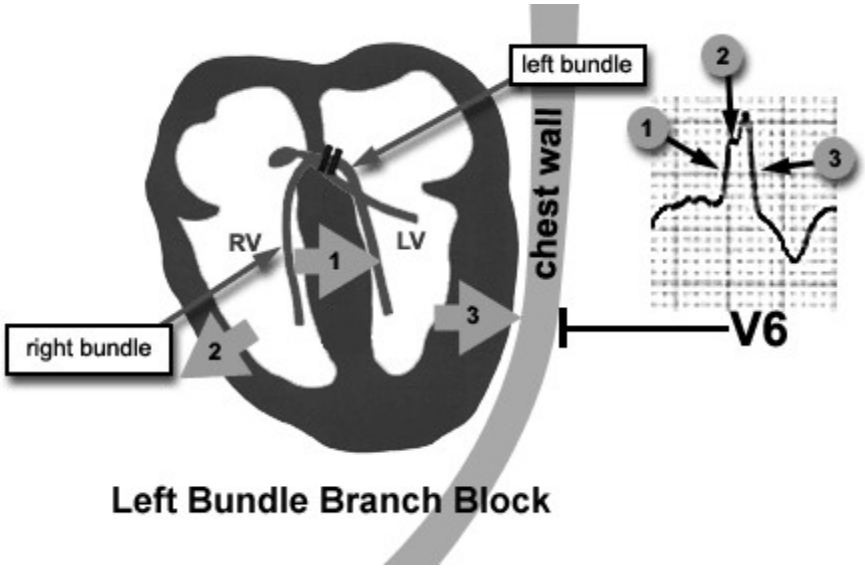
Left bundle branch block is most commonly associated with atherosclerotic coronary artery disease[1.] Other aetiological disorders are idiopathic dilated cardiomyopathy, hypertensive heart disease, aortic valvular disease or non-specific fibrosis of the cardiac conduction system[2]. In a few patients, left bundle branch block may be rate related or idiopathic. Although the presence of left bundle branch block was associated with a three- to fourfold increase in cardiovascular mortality in the Framingham study, [1,] patients without clinically overt heart disease have an excellent short- and long-term prognosis. [2,3.] Thus, it is important to determine whether left bundle branch block is associated with coronary artery disease or other underlying abnormalities.

LBBB Relationship with Coronary Heart Disease and Mortality

In the Framingham Heart Study, univariate analysis showed an increased mortality from cardiovascular disease was seen in people with left bundle-branch block. This is due to high prevalence of coronary heart disease in patients with left bundle-branch block. In studies of myocardial infarction/ischemic heart disease, Left bundle-branch block has been shown to be a strong predictor of high mortality at follow-up.[1-3]

Studies by Froelicher et al[1] and Coronary Artery Surgery Study also clearly indicates that the left bundle-branch block was the result of infarction of the proximal conduction system due to LAD disease.

However, the onset of chronic left bundle-branch block is only rarely accompanied by clinically recognized myocardial infarction or other signs of ischemia. Most often, left bundle-branch block is discovered as an incidental accompaniment to chronic coronary artery disease.



Diagnosis of CAD in the presence of LBBB

The prognosis in patients with left bundle-branch block (LBBB) is related primarily to the presence or absence of underlying cardiac disease. Hence, it would be desirable, in the presence of LBBB, to have a noninvasive method of differentiating between patients with and without CAD. Efforts have been on to invent and design procedures, right from Master's Two-Step-Test[1] to scintigraphy, which may diagnose this disease in large populations and yet be non-invasive, easy to perform and cost effective.

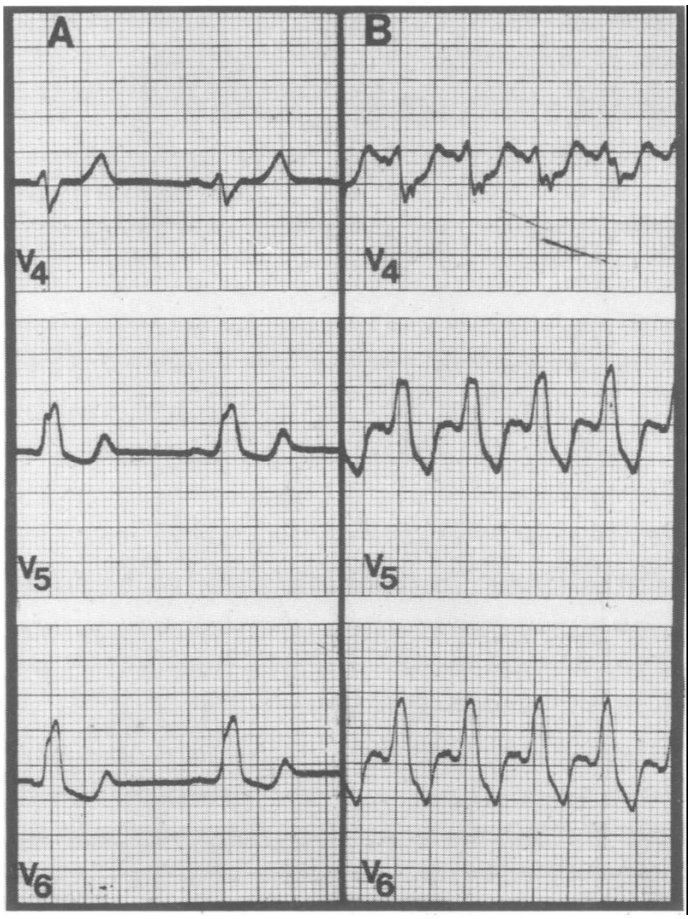
Unfortunately, most non-invasive stress tests have limited value for the detection of coronary artery disease in left bundle branch block patients. Exercise-induced ST segment changes are indeterminate for ischaemia[3-5] and myocardial perfusion studies, especially exercise perfusion studies, often suffer from false positive perfusion defects in the interventricular septum in the absence of left anterior descending coronary artery stenosis [7].

ECG stress testing and Left Bundle Branch Block

ECG stress testing is one of the most widely used investigative techniques in cardiology and is less expensive than other imaging techniques. Used correctly, this method can help confirm the diagnosis of CAD in symptomatic patients. It is also useful in assessing functional capacity and provides prognostic information about patients with known CAD[4-5]. Development of stress protocols and treadmill stress tests are major achievements in this direction. Conventional analysis of electrocardiogram (ECG) during anginal pain or after stress test is based on ST-segment and J-point shift, which has shown a low sensitivity and specificity [4-5].

A number of reports[4-5] have suggested that electrocardiographic stress testing

might be useful for evaluating ischemia in patients with LBBB, particularly if 2 mm of ST depression rather than the usual 1 mm of depression were required for a positive test. Unfortunately, all of these studies involved very small patient populations. Series involving larger groups of patients, have uniformly shown poor specificity for CAD even when the 2-mm criterion is used.



Interpretation of stress ECG on conventional ST-segment parameters still remains an accepted method for diagnosis of myocardial ischemia but it has a relatively low sensitivity (63%) and specificity (74%),[4-5.] especially in single and two vessel diseases if there is no associated left bundle branch block. Its sensitivity and specificity falls dramatically in the presence of LBBB. Hence exercise stress testing may not be helpful in

patients with left bundle branch block particularly when it is applied as a screening method.

Echocardiography

Echocardiographically, left bundle branch block is characterized by asynchronous contraction of the ventricles [10], resulting in the (M-mode) echocardiographic hallmark of left bundle branch block, the early systolic posteriorly directed septal notch, first described by McDonald in 1973 [14,22,26-27]. After the occurrence of this notch, several types of septal motion have been described. Classically, septal motion is anterior and described as paradoxical.[14] However, normal posterior motion and several intermediate types (flat) may also occur[14]. Patients with ischemic heart disease and septal infarction are more likely to have flat septal motion. Normal posterior motion occurring in LBBB is now thought to be due to conduction disturbances occurring in distal purkinje system.

Mechanism of paradoxical septal motion

In left bundle branch block patients ventricular activation starts in the right ventricle and the right side of the septum. Transseptal activation from right to left is transmyocardial and thus slow. Activation of the left ventricle proceeds also from right to left, with the basal and posterolateral wall activated last, although activation of the latter is relatively rapid because of impulse entrance in the distal Purkinje network. Thus, in normal subjects the onset of right and left ventricular contraction occurs nearly simultaneously, whereas in left bundle branch block patients there is asynchronous onset of right and left ventricular contraction. This mechanical asynchrony, resulting in dynamic changes in pressure and volume between the ventricles, continues throughout the cardiac cycle [10]. During early systole, right ventricular isovolumic contraction is

unopposed by left ventricular contraction, causing the septum to move passively posteriorly (explaining the early systolic septal notch). Abrupt anterior septal motion occurs at the time of normalization of the transseptal pressure gradient by a decrease in right ventricular volume with pulmonic ejection and a rise in left ventricular pressure with isovolumic contraction. During simultaneous right and left ventricular ejection several types of septal motion can be encountered. [anterior, flat or posterior]. Hence, echocardiography has limited value in diagnosing CAD in patients with LBBB[24,26-27].

Stress echocardiography

Stress echocardiography aids in assessment of chest pain, viable myocardium, and functional capacity; in evaluation of cardiovascular risk before noncardiac surgery; and in risk stratification after myocardial infarction (MI). It is now part of the armamentarium of clinical cardiologists for assessment and risk stratification of patients with CAD.

Although exercise is the traditional form of stress, pharmacologic stress echocardiography has emerged as a promising technique in the past few years. Dobutamine stress echocardiography (DSE) is widely used both for the detection of coronary artery disease (CAD) and the evaluation of the functional significance of angiographically-proven coronary lesions[28]. Considerable literature now indicates that dobutamine stress echocardiography is an established stress modality for the detection of coronary artery disease in patients without left bundle branch block[28]. However, dobutamine stress echocardiography is only moderately sensitive and highly specific test for the detection of coronary artery disease in left bundle branch block patients. It is capable of visualizing septal motion and assessing septal myocardial thickening which might be relatively preserved in left bundle branch block patients, especially in those

patients without coronary artery disease [14,22, 27].

However, the subjective interpretation of stress echocardiography remains the most significant disadvantage of this technique.[24] The lack of uniform diagnostic criteria remains a significant limitation in the agreement of even expert readers. This is especially true in situations where image quality is poor and wall motion abnormalities are subtle. Furthermore, recent studies have shown the physiological limitations of the human eye to resolve rapid, short-lived motion[24.]

Nuclear Perfusion Imaging / Scintigraphy

Even in the presence of angiographically normal coronary arteries, patients with LBBB often have reversible or fixed septal defects on nuclear perfusion imaging, particularly when combined with exercise or dobutamine stress.[6,24] The exact mechanism is unclear but it is possible that a delayed activation of the septum in the presence of LBBB may lead to a reduction in coronary flow and hence tracer delivery to the septum. Patients with LBBB have an impairment of early diastolic blood flow in the LAD due to an increase in early diastolic compressive resistance resulting from delayed ventricular relaxation. Furthermore, exercise scintigraphic perfusion defects in these patients are associated with a reduced coronary flow reserve, indicating abnormalities of microvascular function in the same vascular territory.

Dobutamine myocardial scintigraphy in patients with LBBB may be misleading for the diagnosis of coronary artery disease, since up to 84.21% of patients may have false-positive septal perfusion defects[25]. A high heart rate associated with dobutamine myocardial scintigraphy increases the proportion of diastolic filling that is lost, leading to a further reduction in septal coronary flow and therefore more obvious perfusion defects.

When stress is performed with dipyridamole or adenosine alone it is less likely to observe such perfusion abnormalities probably because the effect of vasodilators on the heart rate and septal contraction is less than that of dynamic exercise. It has been shown that specificity with vasodilator stress improves by up to 75% and this is achieved without a reduction in sensitivity. Hence scintigraphy has a limited value in diagnosing CAD in the presence of LBBB[24-25].

False Positive Scintigraphy study in patient with LBBB and normal coronaries

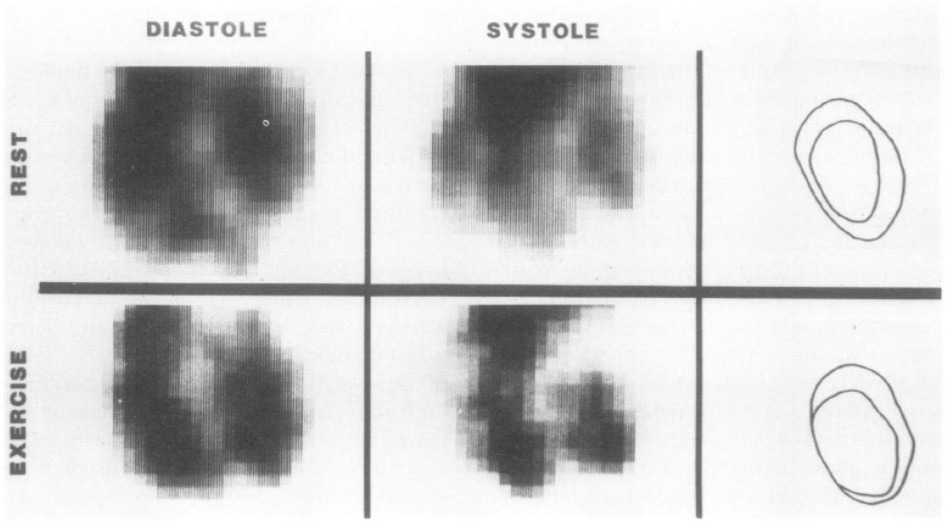


Fig. 2 False-positive RNV study in patient with LBBB and normal coronary arteries. Resting and exercise end-diastolic and end-systolic LAO images are shown. Superimposed systolic and diastolic silhouettes are shown in the third column, demonstrating mild septal hypokinesis at rest, progressing to septal akinesia and apical hypokinesia during exercise.
RNV = radionuclide ventriculography; LBBB = left bundle branch block
LAO = left anterior oblique

Echocardiography Versus Scintigraphy

Stress echocardiography and scintigraphy seek different end points—ventricular asynergy and perfusion abnormalities, respectively—but with a common purpose: the identification of coronary artery disease. Patients with perfusion abnormalities (positive scintigraphy) and no ventricular asynergy (negative echocardiography) have an imbalance strong enough to create perfusion abnormalities but not to precipitate

ventricular asynergy. Whether there is heterogeneity of perfusion with no ischaemia or with ischaemia that is too slight or too brief to be detected by current echocardiographic techniques is far from clear. It has recently been suggested that the presence or absence of ventricular asynergy during dobutamine infusion is not related to the extension of perfusion abnormalities.[24]

Several studies have compared stress echocardiography with nuclear perfusion imaging [28]. Overall, the sensitivity and specificity of the two techniques are comparable and range between 70% and 80%. Quinones and colleagues [24, 28] reported that the overall sensitivity and specificity of stress echocardiography were 85% and 88%, respectively, compared with 85% and 81% for exercise thallium testing. The sensitivity of exercise echocardiography and exercise thallium testing for CAD in patients with one-, two-, or three-vessel disease was also similar (58%, 86%, and 94% versus 61%, 86%, and 94%, respectively). It is a well-tolerated and valuable procedure for noninvasive evaluation of CAD.

However, the limitations of scintigraphic techniques in patients with hypertension and left ventricular hypertrophy[6,24,28] and with left bundle branch block are well known. Several studies have demonstrated that stress echocardiography is to be preferred in these subgroups of patients.[24] However, stress echocardiography is highly operator dependent, the technique chosen should depend on the expertise of each centre in stress echocardiography and scintigraphy.

Noninvasive assessment of CAD

1. Comparison Of The Different Tests For The Diagnosis Of Coronary Artery Disease

Performance	Exercise Stress	Echocardiography		Scintigraphy
		Dipyradamole	Dob	Thallium
Sensitivity	**66%**	**81%**	**78%**	**87%**
Specificity	**80%**	**90%**	**88%**	**70%**
Accuracy	**70%**	**86%**	**82%**	**81%**
Predictive Valve				
Positive	**91%**	**96%**	**92%**	**85%**
Negative	**49%**	**73%**	**69%**	**72%**

Patients with LBBB were excluded from Exercise Stress testing and scintigraphy analysis**.**

2.Sensitivity Obtained With The Different Diagnostic Tests According To Clinical Characteristics Of The Patients

CAD	EST	Dipyridamole	Dobutamine	Scintigraphy
LBBB	**-**	**50%**	**75%**	**-**

Patients with LBBB were excluded from Exercise Stress testing and scintigraphy analysis. EST – Exercise stress testing,

Hence, chest pain in patients with LBBB represents a significant challenge to the emergency practitioner. Currently, no single or combination diagnostic approach exists which will reliably reveal CAD in timely fashion.

Recent literatures show that Tissue Doppler Imaging (TDI) provides a quantitative analysis of regional myocardial function through the analysis of myocardial velocities [8-16 29-32.]

TDI - based modalities pulsed tissue Doppler imaging, strain and strain rate imaging have been introduced to measure regional shortening fraction and shortening rate and to diagnose ischemia [29-32]

Tissue Doppler Imaging For the Diagnosis of Coronary Artery Disease in Patients With Left Bundle Branch Block

Tissue Doppler imaging (TDI)

TDI is a new ultrasound technique that is based on color Doppler imaging principles and allows quantification of intramural myocardial velocities by detection of consecutive phase shifts of the ultrasound signal reflected from the contracting myocardium.[8,9]. Doppler tissue imaging uses the same principles as colour flow Doppler mapping, applying standard autocorrelation processing but reversing high velocity and low amplitude filters such that the high amplitude/low velocity motion of tissue is

displayed in preference to blood flow.

Principles of Tissue Doppler Imaging

Unlike conventional Doppler signals that are typified by high velocity and low amplitude, myocardial motion is characterized by relatively low velocity and high amplitude signals. To record low wall motion velocity, gain amplification is reduced and high pass filters are bypassed with the tissue signal directly entered into the autocorrelator. The thresholding and filtering algorithms are changed to reject the low-amplitude echoes from the blood pool. As cardiac structures move in a velocity range 0.06 to 0.24 m/s, some 10 times slower than myocardial blood flow, and have an amplitude approximately 40 decibels higher, it is possible to obtain images of tissue Doppler motion of high resolution without significant artifact originating from the blood pool. During image acquisition, it is important to optimise the frame rate using an image sector as narrow as possible and to select the appropriate velocity scale.

Modalities of Tissue Doppler Imaging

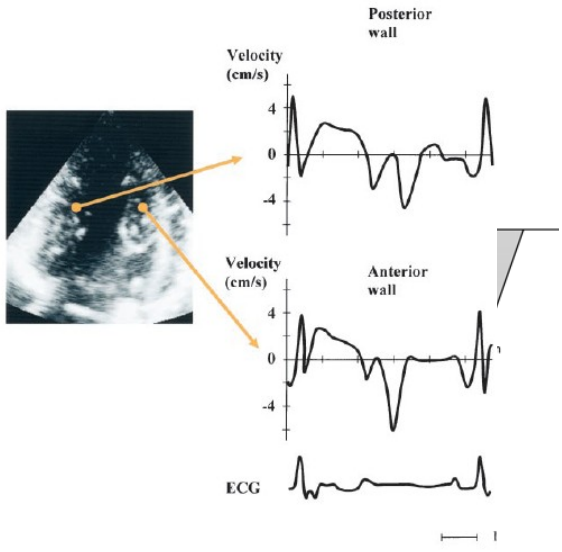
TDI has three modalities: spectral pulsed wave Doppler, two dimensional, and M mode colour Doppler.

Pulsed Spectral Doppler

Spectral pulsed TDI has the advantage of online measurements of velocities and time intervals and an excellent temporal resolution (8 ms). According to the Doppler principle[8], tissue velocities moving toward the transducer are positive, whereas velocities moving away from the transducer are negative. The spectral PW-TDI method provides higher temporal resolution and resolves all peak velocities. With this modality a sample volume is placed within the myocardium (either in the endocardium or the epicardium)

and the low Doppler shift of frequencies recorded from the heart wall moving through the sample volume during the cardiac cycle is recorded. The pattern (Fig. 1) can be divided into two parts systolic and diastolic, from which several measurements can be obtained: 1. The systolic phase is characterized by a positive wave (S) preceded by the time taken for regional isovolumic contraction (RIVCT); 2. The diastolic phase, which is complex, is composed of 4 periods: a) regional isovolumic relaxation (RIVRT); b) the rapid filling period characterized by a negative wave (E); c) diastasis, and d) filling due to atrial contraction, represented by a second negative wave

Figure 1: Schema of the tissue Doppler imaging pattern of the left ventricular mitral annulus. A_m, late diastolic wave; CT_m, myocardial contraction time; E_m, early diastolic wave; PCT_m, myocardial precontraction time; RT_m, myocardial relaxation time; S_m, myocardial systolic wave.



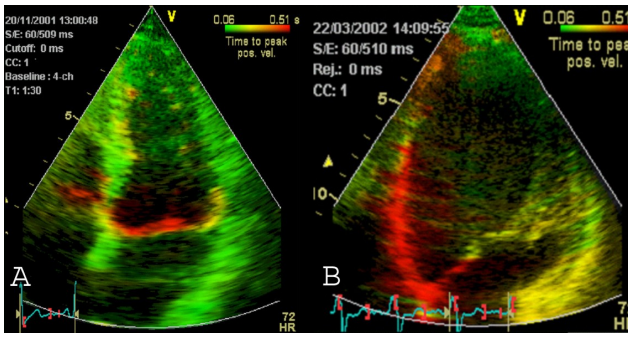
Colour Doppler and M Mode

In colour TDI, red encodes wall motion towards the transducer (positive velocities), whereas blue encodes wall motion away from the transducer (negative velocities). On each side of the scale, the brightest shades correspond to the highest velocities. Colour images require digital

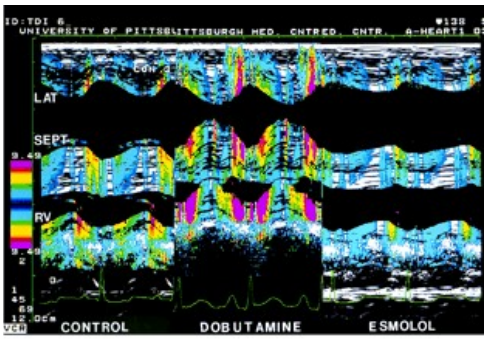
acquisition and storage for off-line post-processing analysis. In contrast to spectral Doppler, endocardial and epicardial layers can be separately analysed. Peak and mean velocities, time velocity integral, and regional time intervals can be measured in each myocardial segment, in each myocardial layer, and in each phase of the cardiac cycle.

M mode colour encoded TDI has a high temporal resolution (5–10 ms). Colour two dimensional imaging has been limited by a slow frame rate, but parallel processing and advances in beam formation technology have increased the frame rate to a level adequate for analysis of most cardiac events (temporal resolution 10–100 ms)

Color Doppler



M- Mode

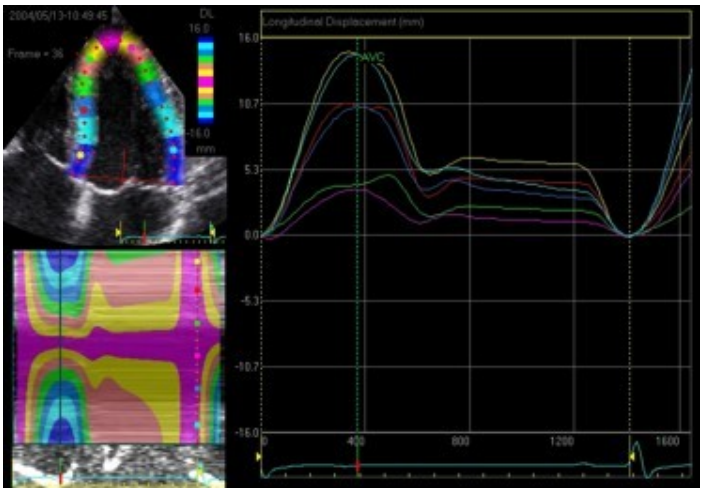


Strain and Strain Rate Echocardiography

Strain and strain rate are TDI derived modalities that are now available in real time. Strain rate measures the rate of deformation of a tissue segment. Peak systolic strain rate represents the maximal rate of deformation in systole. An algorithm calculates spatial differences in tissue velocities between neighbouring samples within the myocardium aligned along the Doppler beam. A sample distance of 5 to 11 mm has been previously used. Strain is obtained by integrating strain rate over time and represents deformation of a tissue segment over time. Strain is expressed as the per cent change from the original dimension. Systolic strain represents the magnitude of deformation between end diastole used as a reference point and end systole.

Systolic strain is positive and blue encoded when there is regional expansion. This is thickening in parasternal views and lengthening in apical views. Negative systolic strain is yellow to red encoded to denote regional compression, which is thinning in parasternal views and shortening in apical views. Infarcted myocardial tissue does not demonstrate shortening or lengthening activity and shows no or minimal systolic strain rate or strain, which is displayed as green. The technique of raw data storage and reconstruction permits the measurement of tissue velocity, peak systolic strain rate, peak early and late diastolic strain rate, and peak systolic strain from the same sample volume within the same cardiac cycle. Simultaneous interrogation of multiple myocardial segments and curved M mode colour display are also applicable to strain and strain rate.

Strain And Strain Rate Imaging



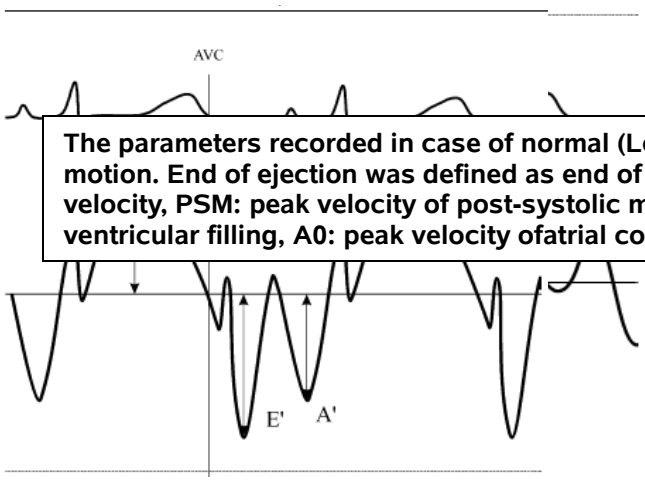
Detection of Ischemia Using TDI

Experimental and clinical studies have shown that during acute ischaemia, myocardial peak systolic velocity and strain rate were notably reduced or reversed within 5 seconds after coronary occlusion and were delayed. In addition, there was positive velocity after the end of ejection [Post systolic velocity].[8-9] . Post-systolic shortening or

thickening can be easily recognised by pulsed tissue doppler imaging and high velocity, strain rate or strain occurring during the isovolumic relaxation period, often extending into the early filling period.[29-32]

Post Systolic Motion

The first documentation of PSM was made in the 1970s in experimental studies after myocardial infarction[8]. The regional myocardial function was assessed in open chest animal models using sono-micrometry and a decrease in systolic shortening with an increase in PSM was described as an effect of progressive myocardial ischemia[29].



The parameters recorded in case of normal (Left) and abnormal (Right) myocardial motion. End of ejection was defined as end of T wave on ECG. S: peak systolic velocity, PSM: peak velocity of post-systolic motion, E0: peak velocity of early ventricular filling, A0: peak velocity ofatrial contraction.

More recently, other studies performed in animal models demonstrated that post-systolic contraction occurs both in case of moderate ischemia (when the myocardium is hypokinetic or akinetic and it should be due to active contraction) as well as in case of severe ischemia (when the myocardium is dyskinetic and it should be due to an entirely passive mechanism)[30].

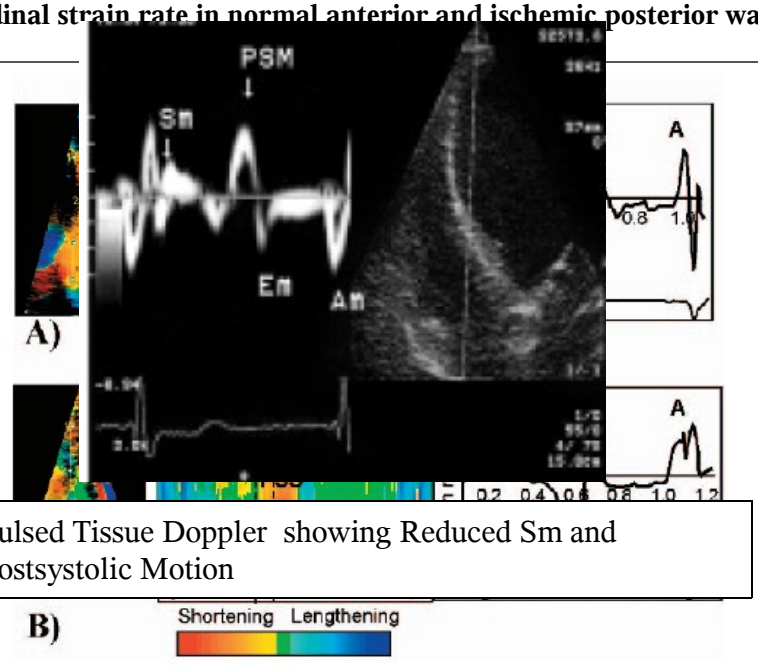
This asynchronous contraction occurring after normal left ventricular (LV) systolic ejection, during a prolonged relaxation period, has been observed in humans at ventriculography[8]. This phenomenon, defined as postsystolic shortening or thickening or

postsystolic motion (PSM), has been recently also described at Tissue Doppler[9], an ultrasound modality which allows the quantification of both the systolic and diastolic segmental myocardial wall velocities[8,9].

Postsystolic shortening or thickening can be easily recognized by pulsed tissue doppler and high, abnormal strain rate during the isovolumic relaxation period, often extending into the early filling period[10-12, 15,16]. Accurate timing of the aortic valve closure is critically important for correct recognition of postsystolic shortening. Earlier studies have shown that the extent of myocardium that exhibits postsystolic shortening approximates the extent of myocardium at ischemic risk [17].

Longitudinal strain rate in normal anterior and ischemic posterior wall

Figure 3.

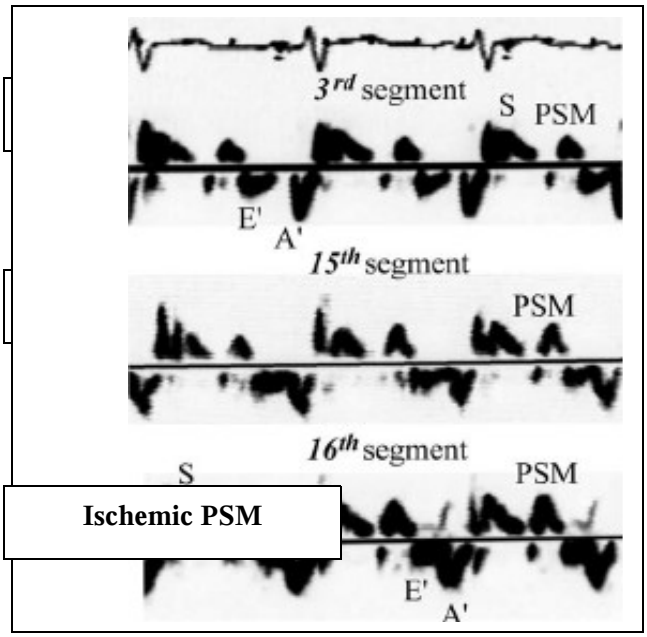


Pulsed Tissue Doppler showing Reduced Sm and Postsystolic Motion

Longitudinal strain rate (SR) maps in normal anterior (A) and ischemic posterior (B) left ventricular (LV) wall in one animal during left circumflex coronary artery occlusion. SR maps were reconstructed as detailed in Figure 2. Right panels show SR profiles from the corresponding mid anteroseptal and posterior LV segments. Inverted systolic strain rates (dyskinesia) and postsystolic shortening (PSS) are readily visible in the acutely ischemic myocardium. In normal anteroseptal wall, SRs are homogeneously distributed from apex to base. The vertical lines indicate time of the aortic valve closure and mitral valve opening. IVC, isovolumic contraction; S, ejection; E, early diastole; A, late diastole.

However, small amplitudes of postsystolic motion or shortening have been also found in normal myocardium, LVH and in patients with LBBB without CAD. [15-17]

Recent studies have shown that post systolic motion occurring due to CAD is of higher amplitude, delayed [>100msec form aortic valve closure] and associated with decrease in systolic amplitude[29-32].

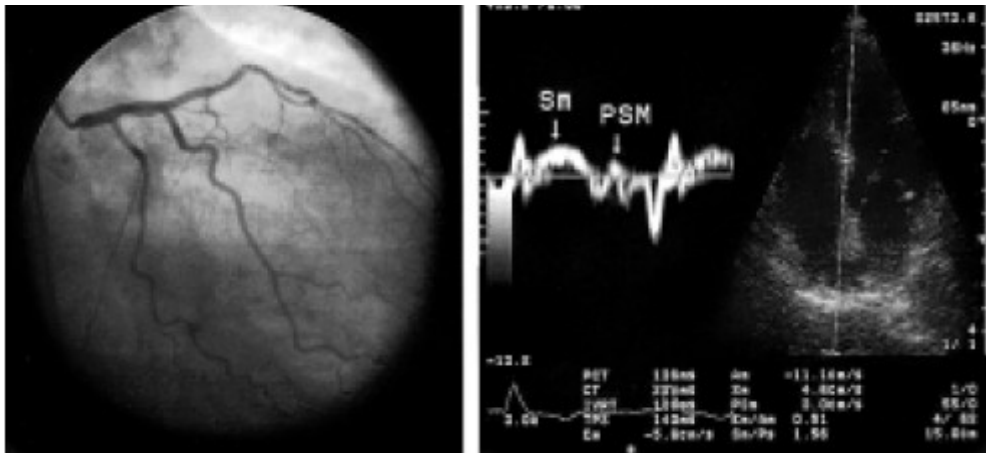


Differentiation of ischemic and non-ischemic Post-systolic motion

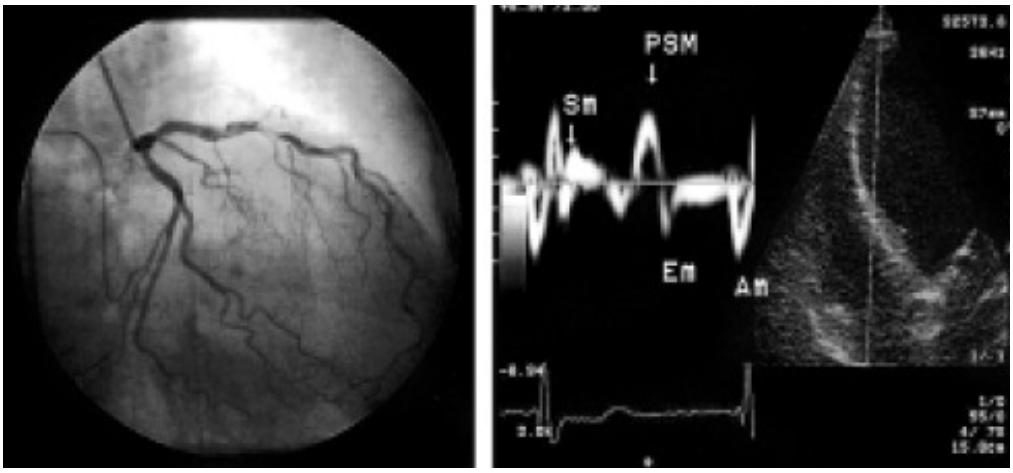
TDI:	PSM non ischemic	PSM ischemic
Systolic Velocity[Sm]	Normal	Reduced
PSM	Small Amplitude	Large ampitude
Diastolic velocities [Em]	Normal	Normal or Em reduced

Figure Showing Coronary angio and TDI : LBBB with out LAD Disease

Doppler pattern of the mid septal wall in a patient with left bundle branch block but without left anterior descending coronary artery stenosis.
The post-systolic motion (PSM) is lower than the myocardial systolic peak velocity (Sm) (Sm/PSM > 1).



Doppler pattern of the posterior septal wall in a patient with left bundle branch block and significant left anterior descending coronary artery stenosis. Note the higher amplitude of the post-systolic motion (PSM) and the myocardial systolic peak velocity (Sm)/PSM ratio < 1.) Am = myocardial atrial peak velocity; Em = myocardial early peak velocity.



Review of Literatures

PSM in Experimental Studies

Since in 1958 a delayed relaxation of fibers after release of a coronary ligature in canine ischemic myocardium was reported, an abnormal forward movement occurring during IVRT and following a reduced systolic excursion of the posterior wall was observed in dogs in both ischemic and marginal segments after circumflex coronary artery occlusion3 and after posterior MI by M-mode echocardiography. By analyzing regional wall motion by sonomicrometry in the intact dog heart, two different contractility patterns were observed by **Doyle et al,**[33] the first after gradual or abrupt coronary occlusion, characterized by a bulge during IVRT; and the second one during gradual occlusion, characterized by an early systolic lengthening followed by a diastolic PSM. By the same method used in open chest anaesthetized dogs, PSM paralleled a systolic shortening impairment, either immediately after coronary occlusion or during progressive stenosis of left anterior descending coronary artery, and persisted with the development of dyskinesis and during reperfusion.[5] This finding proposed PSM as possible marker of early recovery in myocardial function after acute ischemia.

Consistent with this experience, **Takajama et al**,[34] found in a canine model that the amplitude of PSM detected immediately before reperfusion is associated with the magnitude of preocclusion systolic shortening and to the magnitude of systolic shortening measured both 30—60 minutes and 2—3 weeks after reperfusion. The authors concluded that PSM is a sensitive index of myocardial ischemia in both dyskinetic and hypokinetic segments but represents also a predictor of early and late functional recovery of ischemic myocardium. A delayed myocardial relaxation was hypothesized as the mechanism

underlying PSM during ischemia. These results were confirmed by **Leone et al**[35]. who reported a gradual increase of PSM amplitude from moderate to severe ischemia, parallel to the decrease of systolic shortening and to the influence exerted by changes in afterload.

PSM in the Clinical Setting

In the clinical setting, the first evidence of PSM was provided by **Gibson et al**.[36] who described prolonged inward movement of the ischemic segment during IVRT by cineventriculograms, 4 hours after MI, and attributed this phenomenon to residual contractility of ischemic segment. More recently by a computerized method analyzing wall motion through cardiac cycle during ventriculography, the magnitude of PSM before reperfusion was associated with the late recovery of regional wall motion and with the improvement of ejection fraction in 35 patients with anterior MI undergoing primary coronary angioplasty." This study proposes PSM as an indicator of myocardial viability. PSM has been visualized also by M-mode echocardiography in 23 patients with critical coronary stenosis. In this study the evidence of PSM at rest was related to the evidence of contractile recovery during low dose dobutamine stress and to a higher dipyridamole SPECT early-redistribution thallium activity.

Is post-systolic motion the optimal ultrasound parameter to detect induced ischemia during dobutamine stress echocardiography?

Jelena Celutkiene et al. European Heart Journal (2004) 25, 932–942

In this study the authors used dobutamine stress echocardiography using pulsed-wave TDI to investigate both standard systolic and diastolic parameters, but more specifically to address the phenomenon of post-systolic motion (PSM) as a marker of

acquired ischaemia. The study investigators examined 60 patients without previous myocardial infarction who underwent DSE. Peak systolic, post-systolic, early and late diastolic velocities were measured at rest and during stress. Myocardial segments were divided into ischemic and non-ischemic groups according to the presence of significant angiographic coronary stenosis.

Ischemic segments compared with non-ischemic segments demonstrated a reduced increase of systolic velocity, prominent PSM and reduced early diastolic velocity during stress.

They study revealed that the peak velocity of PSM was the most accurate index of induced ischemia (sensitivity 73–100%, specificity 82–97%) compared to systolic and early diastolic velocities (sensitivity 52–77% and 63–68%, specificity 63–77% and 59–81%, respectively).

Left bundle branch block with and without coronary artery disease: which value for a tissue Doppler-derived post-systolic motion?

Rodolfo Citro et al. Ital Heart J 2003; 4 (10): 706-712)

This study used pulsed tissue doppler to differentiate PSM of the interventricular septum in LBBB with or without stenosis of the left anterior descending coronary artery (LAD). Forty-two patients with chronic, complete LBBB and tissue Doppler-derived septal PSM were studied.

Septal tissue Doppler showed lower myocardial systolic (Sm) and atrial peak velocities, a higher PSM, and a longer relaxation time and pre-contraction time in patients with LAD stenosis.

A Sm / PSM ratio < 1 was detected in 86% of patients with LAD stenosis and in 22% without LAD stenosis (sensitivity 73%, specificity 77%, positive predictive value 64%, and negative predictive value 84%). In addition, by a stepwise forward multiple linear regression analysis, the evidence of left anterior descending artery stenosis was an independent predictor of the PSM amplitude.This study revealed that Tissue Doppler may be useful to distinguish septal myocardial asynchrony in LBBB with and without LAD stenosis.

Differential diagnosis of left bundle-branch block using tissue Doppler echocardiography: importance of both right and left ventricular systolic velocities and isovolumic relaxation times.

Saha S k et al ; Euro Echo 2004

The authors investigated the role of color tissue Doppler echocardiography (TDE) in the differentiation these conditions. The study population included 86 subjects using standard and tissue Doppler. The subjects were 20 controls (G1), 21 with idiopathic LBBB (G2), 19 with LBBB in presence of ICM (G3), and 26 LBBB with DCM (G4).

TDI profiles were obtained in 4 left ventricular (LV) basal segments (septal, lateral, inferior and anterior) & in the right ventricular (RV) base by digital post-processing. Peak systolic velocity (PSV, cm/s) and the isovolumic relaxation time (IVRT, ms) were computed. The duration of IVRT was universally prolonged in all groups compared with controls .G1, longest duration being observed in those with LBBB and dilated cardiomyopathy (G4.). The right ventricular peak systolic velocity (RV PSV) was similar in G1 and G2 but significantly lower in both G3 and G4. This study concluded

that QRS duration, LV dimension, LVEF do not differentiate LBBB. PSV and IVRT not only differentiate between isolated and pathologic LBBB, they also help to distinguish DCM from ICM. This study suggests that both LV and RV studies using TDI are helpful to differentiate LBBB of diverse etiology.

Myocardial Postsystolic Motion in Ischemic and Not Ischemic Myocardium: The Clinical Value of Tissue Doppler

Rodolfo Citro, M.D.* et al. Echocardiography Volume 22 Issue 6 Page 525 - July 2005

In this study the authors analyzed the pathophysiologic mechanisms underlying PSM and the contribution of tissue Doppler for its understanding. The authors used strain and strain rate (SR), obtainable by off-line color tissue Doppler to identify the mechanisms underlying PSM since these measurements reflect, respectively, the intrinsic rate and the percentage of deformation of a given myocardial segment, and are relatively independent of both overall cardiac movement and tethering of the neighboring LV segments. This study revealed that the ratio of PSM to regional systolic longitudinal strain can be used to separate ischemic from non ischemic PSM and appears the best quantitative parameter to identify ischemia during dobutamine stress.

To summarize, current literatures clearly indicates that Tissue Doppler derived post systolic motion and myocardial velocities appears to be a sensitive, reproducible, accurate, noninvasive echocardiographic technique for diagnosing myocardial ischemia.

The clinical implementation of TDI, however, has been relatively slow, and most echocardiographic laboratories do not apply TDI as a routine diagnostic method. This may in part be attributed to a lack of established criteria for how to analyze and interpret the TDI velocity trace, which reflects the relatively limited insight into the etiology of the different velocity components

In view of the above, the present study was designed

- to assess the usefulness of TDI
- to characterize the Post systolic motion among LBBB patients with or without angiographic evidence of stenosis of the left anterior descending coronary artery (LAD).

Among the available ultrasound modalities, Strain/strain rate imaging better reflects true myocardial thickening/thinning[15-17, 29-32], we used of **Pulsed Tissue Doppler imaging** for myocardial velocities because **it is far more practical, as the analysis can be performed on-line and does not require sophisticated time-consuming post-processing** and which makes the method useful for daily clinical practice.

Usefulness Of Tissue Doppler Imaging For the Diagnosis of Coronary Artery Disease in Patients With Left Bundle Branch Block

AIM

The aims of this study were;

- To assess the usefulness of Pulsed tissue Doppler imaging for the diagnosis of CAD in the presence of LBBB
- To characterize the Post systolic motion among LBBB patients with or without CAD.

Methods

Selection of the study group.

This was a prospective study done between June 2004-September 2005 at the Department of Cardiology, Government General hospital, Chennai. We enrolled 30 consecutive patients who were referred to our department for the evaluation of CAD.

Patients with chest pain and permanent, complete left bundle branch block were eligible for the study if they did not meet any of the following excluding criteria: hemodynamically significant valvular heart disease, congenital heart disease, previous myocardial infarction, unstable angina, pressure or volume right ventricular overload, permanent pacemaker and abnormal atrioventricular pathways. (Figure 1)

The original cohort comprised 30 patients with chronic (> 6 months), complete LBBB and recurrent angina pectoris or atypical chest pain, but without any exclusion criteria. From this cohort, 28 patients (16 males, 12 females, mean age 62 years) were selected, after their informed consent. All patients underwent Tissue Doppler echocardiography within 1 month of coronary angiography, without any interceding clinical event.

Mean age of the patients was 62 $\pm$ 4 years (range 58 to66), 16 were men (57.14%) and 12 were females. Thirteen patients (46.4%) had typical angina, 15 patients (53.5%) had atypical angina. Mean pre-test probability of coronary artery disease, calculated from age, gender and chest pain characteristics, was 74%$\pm$21%. Relevant demographic data of all patients are displayed in table 1

Definition of LBBB

Complete left bundle branch block was defined as a notched or slurred QRS with duration more than 120 ms, with an initial R wave, absent septal Q wave in left sided leads and displacement of the ST segment, the T wave, in a direction opposite to that of the principal QRS deflection. Electrocardiographic measurements from a standard 12-lead electrocardiogram included QRS duration, measured to the nearest 10 ms, and frontal QRS axis, measured to the nearest 15^0. A frontal QRS axis between $-30^0$and $+90^0$ was considered normal.

Diagnostic Criteria used for LBBB [Braunwald]

Complete left bundle branch block
QRS duration ≥120 msec
Broad, notched R waves in lateral precordial leads (V_5 and V_6) and usually leads I and aV_l
Small or absent initial r waves in right precordial leads (V_1 and V_2) followed by deep S waves
Absent septal q waves in left-sided leads
Prolonged intrinsicoid deflection (>60 msec) in V_5 and V_6*

Figure 2 Left Bundle Branch Block

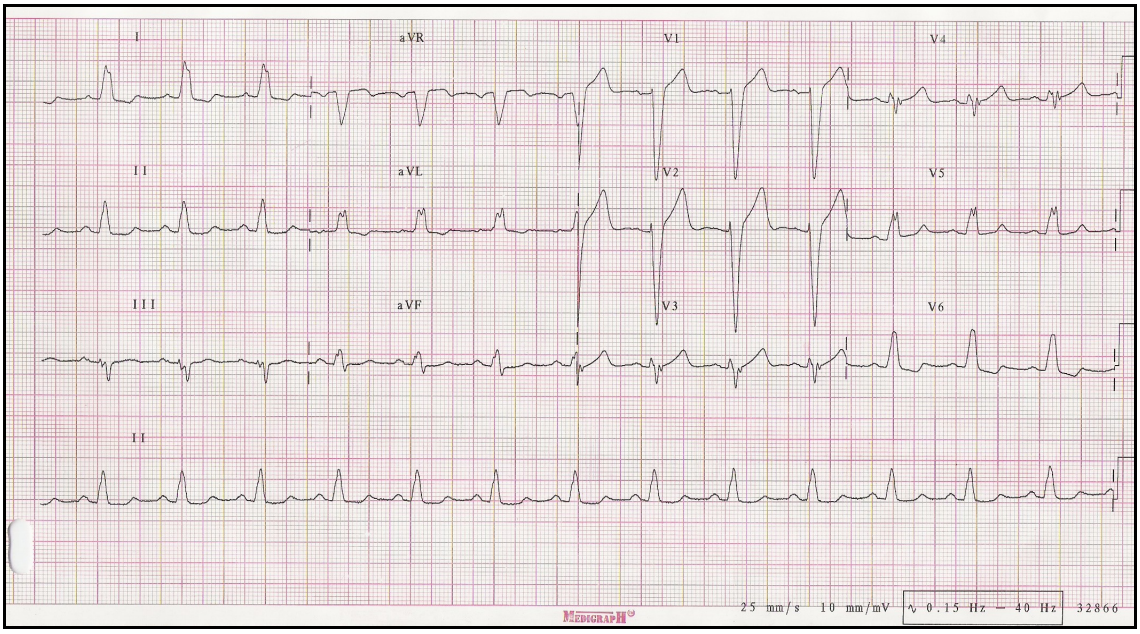


Figure 1: Inclusion and Exclusion Criteria

Inclusion Criteria

- *Patients with Chronic LBBB and Chest pain*

Exclusion Criteria

- *Hemodynamically significant Valvular Heart Disease,*
- *Congenital Heart Disease,*
- *Previous Myocardial Infarction,*
- *Unstable angina,*
- *Pressure Or Volume Right Ventricular Overload,*
- *Permanent Pacemaker and*
- *Abnormal Atrioventricular Pathways.*

Table 1. Base line Characteristics of the study population

Variable	LBBB with LAD	LBBB without LAD	p value
Gender (M/F)	/6	8/5	NS
Age (years)	62.4 ± 4.2	60.6 ± 4.5	NS
Body mass index (kg/m2)	27.1 ± 3.7	26.8 ± 2.5	NS
Systolic BP (mmHg)	138.0 ± 16.0	142.0 ± 20.0	NS
Diastolic BP (mmHg)	79.9 ± 11.0	83.3 ± 13.1	NS
Heart rate (b/min)	78.5 ± 14.5	71.7 ± 11.2	NS
QRS duration (ms)	142 ± 9.1	141 ± 8.6	NS
Risk factors			
Smoking	6	3	NS
Diabetes	4	2	<0·02
Hypertension	10	7	NS
Family history of CAD	4	5	NS
Dyslipidemia	3	5	NS

BP = blood pressure; LAD = left anterior descending coronary artery; LBBB = left bundle branch block. LAD- = patients without significant LAD stenosis; LAD+ = patients with significant LAD stenosis.

All the selected patients underwent standard Doppler and TD echocardiography and coronary angiography. Patients were divided into two groups on the basis of their

coronary angiography: 13 had no LAD stenosis whereas 15 had significant LAD stenosis (> 50%). Of note, among the patients with LAD stenosis, none presented significant stenosis of any coronary branches other than the LAD.

Standard Doppler echocardiography and Tissue Doppler Imaging.

Standard Doppler echocardiograms and pulsed TD were performed with the subjects in partial left decubitus, using the Philips IEE 32 and Aloka Trivitron both equipped with a variable-frequency phased-array transducer and TD capabilities. Two-guided, M-mode LV analysis and Doppler recording of the LV transmitral diastolic inflow was performed as previously described. The LV mass was calculated using the criteria of the American Society of Echocardiograph and normalized for body height. The two-dimensional LV end-diastolic and end-systolic volumes were calculated using the Simpson method and the LV ejection fraction was calculated using the following formula: end-diastolic volume end-systolic volume/end-diastolic volume x 100.

Pulsed TD was performed at transducer frequencies of 3.5-4.0 MHz, adjusting the spectral pulsed Doppler signal filters to obtain the Nyquist limits of 15 and 20 cm/s, and using the minimal optimal gain. In the apical 4-chamber view, the pulsed Doppler sample volume was subsequently placed in two different regions: middle interventricular septum (the perfusion of which is provided by the LAD) and LV lateral mitral annulus. (Figure 3 & 4)

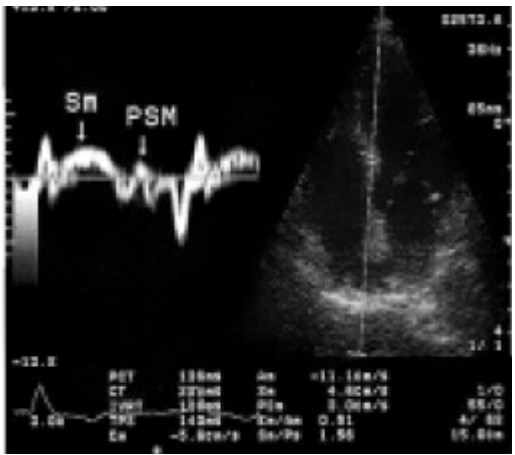
The apical 4-chamber view was chosen to obtain the quantitative assessment of the regional myocardial wall motion almost simultaneously to the Doppler LV inflow and to minimize the incidence angle between the Doppler beam and the LV longitudinal motion. The following TD measurements were determined as indexes of regional

myocardial function: myocardial systolic peak velocity (Sm, m/s), myocardial pre-contraction time (from the onset of the ECG QRS to the beginning of sm) and contraction time (from the beginning to the end of Sm) (all in ms) as systolic indexes and myocardial early (Em) and atrial (Am) peak velocities (m/s) and their ratios, and relaxation time (RTm) (ms) – corresponding to the time interval elapsing between the end of Sm and the onset of Em – as diastolic measurements. PSM was identified during RTm, using the previously mentioned criteria. The presence of TD-derived PSM of the interventricular septum was defined as any clear (>2 cm/s) myocardial velocity occurring upon the baseline between the end of the myocardial systolic velocity and the onset of the myocardial early diastolic velocity. Our TDI methods and reproducibility have been previously described [29-32] (Figure 3-5)

Coronary angiography.

Coronary angiograms using the Judkins technique were performed with a standard cineangiographic system in multiple views. Lumen diameter narrowing was graded as 0, < 25, 25, 50, 75, 90, and 100% in 15 arterial segments. In the present study, the definition of a significant anatomical stenosis implied a >50% luminal narrowing localized in the first or middle segments of the coronary arterial tree.

Figure 3 & 4: Apical 4-chamber view, the pulsed Doppler sample volume mid interventricular septum (the perfusion of which is provided by the LAD).



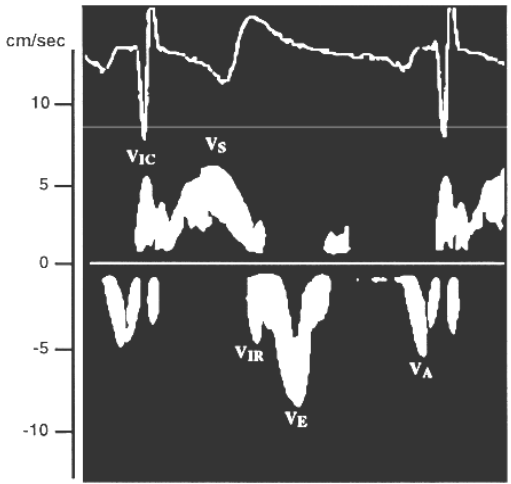
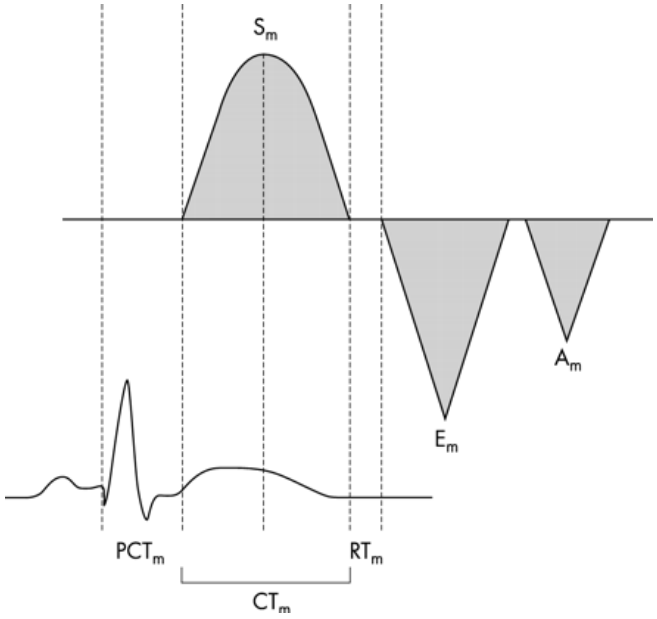


Figure: 5 TDI pattern including the normal myocardial velocities, one positive myocardial systolic velocity and two negative diastolic velocities. Post systolic motion occurs during a prolonged myocardial relaxation time.

Sm = myocardial systolic velocity, Em = early diastolic velocity, Am = myocardial atrial velocity, RTm = myocardial relaxation time, PSM = Post systolic motion



Statistical analysis.

Variables are presented as mean ± 1 SD. Analysis of variance was performed to estimate intergroup differences. Linear regression analyses and partial correlation testing using Pearson's method were used to assess univariate relations. The prediction of PSM was made using stepwise, forward, multiple regression analyses that included potential confounding variables not obviously related to each other. The null hypothesis was rejected for $p < 0.05$.

Results

The study population included patients with arterial systemic hypertension (17 / 28; 60%), mild calcific aortic valve disease (5/28; 17.8%) and isolated LBBB (6/28; 21.4%). With regard to the 15 patients with LAD stenosis, coronary angiography showed a narrowing of 84.5 ± 6.0%. Among these patients, 10 were hypertensive, 5 had mild aortic regurgitation, and 1 mild aortic valve stenosis.

The demographic characteristics of the groups are listed in table I. No differences in gender, age, body mass index, heart rate, blood pressure and ECG-derived QRS duration were found between the two groups. The results of Doppler echocardiographic analysis are reported in table II. Patients with LAD stenosis had a significantly lower septal wall thickness. ($p < 0.01$). No differences of the two-dimensional-derived LV end-diastolic diameter, LV ejection fraction and Doppler indexes of LV diastolic function were found between the two groups. Table III shows the results of pulsed TD analysis of both the middle posterior septum and LV lateral mitral annulus. At the level of the mid inventricular septum, patients with LAD stenosis had higher amplitude PSM ($p < 0.005$), a longer RTm ($p < 0.02$) and PCTm ($p < 0.05$) and a lower Am and Sm peak velocity (both $p < 0.05$). Of note, we did not find any difference of the PSM amplitude in relation to the different degrees of LAD narrowing in this subgroup.

An Sm / PSM ratio < 1 was found in 93.3 % (14 / 15)of patients with LAD stenosis. None of the patients without LAD stenosis had Sm / PSM < 1 (sensitivity 93.3%, specificity 100%, positive predictive value 64%, negative predictive value 84%). 30%of LBBB patients without CAD also had post systolic motion, but of small amplitude (PSM < 4 cm).

Figures 6 and 7 show two examples of LBBB, without and with LAD stenosis respectively: the Sm /PSM ratio is > 1 in patients without coronary artery disease and < 1 in the patient with LAD stenosis. At the level of the LV mitral annulus, patients with LAD stenosis had a longer RTm ($p < 0.01$) and PCTm ($p < 0.05$) and a lower Am peak velocity ($p < 0.05$), without any difference in the Em/Am ratio and Sm peak velocity. The PSM peak velocity was positively related to LV ejection fraction. No significant relation of PSM was found with the body mass index, heart rate, systolic and diastolic blood pressure, and with the septal Sm and RTm. Using a multiple linear regression analysis LAD stenosis and ejection fraction were found to be independent predictors of the PSM amplitude. (cumulative $r2 = 0.27$, standard error 2.11 cm/s, $p < 0.002$).

Discussion

LBBB is characterized by a delay in the onset and completion of the systolic ejection phase and lengthening of the LV diastolic period. This abnormally prolonged activation is often associated with late asynchronous contraction of the interventricular septum that often alters the assessment of the LV global and segmental functions by usually employed noninvasive imaging methods. Limitations of currently available non invasive diagnostic tests are well known. All the recently reported studies visualized PSM as a marker of ischemia. On these grounds, this study investigated whether pulsed tissue Doppler derived myocardial velocities and PSM could be useful to distinguish isolated and ischemic left bundle branch block.

Tissue Doppler Imaging in patients with coronary artery disease.

TDI permits the measurement of the systolic and diastolic velocities of the regional myocardial walls [8,9] and there by helps in the evaluation of ischemia. Recent studies done recently has clearly established the role of TDI in the diagnosis of CAD in patients with LBBB. Saha et al and Rodolfo Citro et al have clearly showed that high amplitude PSM and reduced myocardial systolic velocities are hallmark of ischemia due to LAD disease

The findings of the our study clearly indicate that septal PSM and reduced Sm can be documented by means of this technique[TDI] in LBBB and utilized to distinguish LBBB patients with or without coronary artery disease.

However, PSM of small amplitude also occurs in subsets of patients free of coronary artery stenosis, including those with LV hypertrophy and LBBB. All the recently reported studies visualized PSM as a marker of ischemia. Hence, it has to be

taken into account that the mere presence of PSM is not a specific sign of ischemia.

Differences of post-systolic motion in left bundle branch block with or without left anterior descending coronary artery stenosis.

In our study population, 93.3% with LAD disease had PSM, whereas 30% of patients without LAD disease exhibited PSM. The group with LAD stenosis was characterized by a high amplitude PSM peak velocity and a lower Sm peak velocity. PSM occurs during the myocardial relaxation period which, according to previous observations[8,9,10-12], is lengthened in the presence of coronary stenosis. The following features are used to differentiate ischemic PSM from non ischemic PSM.

1) overall systolic deformation,
2) the coexisting reduction in systolic velocity;
3) the exceeding of post systolic motion with respect to total systolic velocity >20% and
4) the timing of occurrence of PSM (>90 ms after aortic valve closure).

In LBBB associated with LAD stenosis, a lower Sm is an obvious consequence of a reduced myocardial contraction while the higher PSM amplitude could be explained by the additive effect of the delayed myocardial tension which is more prolonged during the relaxation phase.

Of interest, a Sm/PSM ratio < 1 was evident in 93.3% of patients with LAD stenosis but none in those without coronary artery disease. This ratio might be useful to exclude significant LAD stenosis in LBBB patients since it showed good sensitivity and specificity and a negative predictive value of 92.9%.

With regard to temporal analysis, a prolonged precontraction time and RTm were

found at the level of both the septal wall and LV mitral annulus in patients with LAD stenosis. Asynchronous contraction associated with LBBB and the presence of CAD could be the explanation for delayed myocardial relaxation. Except for minor changes of the Am, the systolic and diastolic peak velocities of the LV mitral annulus were not significantly modified in patients with LAD stenosis.

Associations of post-systolic motion in the overall left bundle branch block population.

PSM was seen in 67% of our study population. Our results are similar to earlier studies in that, only 30% of LBBB patients without LAD exhibited PSM. In non ischemic conditions, PSM could be simply secondary to a wall motion heterogeneity due to prolonged intraventricular conduction. Potential hypothesis and rationale of PSM in healthy subjects find support in experimental observation showing that few LV myocardial segments are truly isometric during relaxation period, some of these developing a post ejection shortening and others and early re-extension.[29-32]

In addition, the PSM amplitude was positively associated with LV ejection fraction, i.e. to LV systolic function. This finding suggests a possible effect of a good LV global systolic function in sustaining a prolonged delayed motion of the asynchronous myocardium by the neighboring normally contracting LV walls.

Our study results are similar to recent assessment by means of strain Doppler echocardiography which supports the hypothesis that the abnormal contraction developing during isovolumic relaxation may reflect the work performed on that area by other myocardial segments. By this analysis, the presence of LAD stenosis and, to a lesser extent, LV ejection fraction were the only independent predictors of the PSM

amplitude. The independent association between evidence of significant coronary stenosis and a higher PSM peak velocity is consistent with previous studies [29-32] and suggests a potential clinical role of TD in the identification of coronary artery disease in patients with LBBB.

Study limitations.

In the present study, among the LV myocardial walls pulsed TD sampling was limited to the interventricular septum and to the LV lateral mitral annulus, which corresponds to the overall LV longitudinal motion. Although it is well known that the contraction asynchrony of LBBB is particularly overt at the septal level, it could have been more comprehensive to evaluate even other LV myocardial segments, possibly influenced by the abnormal electrical conduction present in LBBB. Another limitation is due to the assessment exclusively of the LV longitudinal motion (since our TD recording was performed only in the apical 4- chamber view), without considering the circumferential shortening (in the parasternal short-axis view). Our choice, however, was mainly due to the assumption that, because of a reduced translation movement, the pulsed TD septal pattern is more easily obtainable in the apical views. Further studies including larger cohorts of patients with LBBB are needed to confirm our data.

Advantages:

1. The findings of the present study indicate that septal PSM can be documented by means of this technique[TDI] in LBBB and utilized to distinguish LBBB patients with or without coronary artery disease.
2. **Pulsed Tissue Doppler imaging** for myocardial velocities and Post systolic motion **is far more practical, as the analysis can be performed on-line and does not require sophisticated time-consuming post-processing** and which makes the method useful for daily clinical practice.
3. **The proposed method is technically simple and can be easily performed using any echo machine equipped with conventional pulsed Doppler myocardial imaging**.

Conclusions

- The present study demonstrates the usefulness of Pulsed Tissue Doppler for diagnosing CAD in the presence of LBBB.
- Pulsed TDI could be a relevant, sensitive, specific and non invasive tool for the detection of the coexistence of coronary artery disease in patients with left bundle branch block.
- Post-systolic motion presents different characteristics in patients with and without significant LAD stenosis. [High amplitude PSM = LAD disease]
- Sm / PSM < 1 differentiate ischemic from nonischemic LBBB with greater sensitivity and specificity, might be a useful and simple adjunct to standard dobutamine stress testing for detecting CAD in patients when LBBB is present.
- Pulsed TD derived echocardiographic methods are quantifiable, reproducible, and noninvasive techniques for assessing the presence of CAD in the patients with LBBB.

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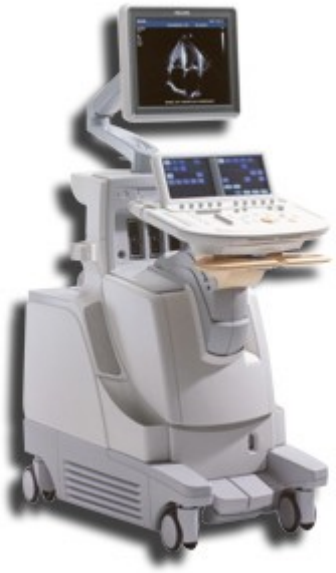
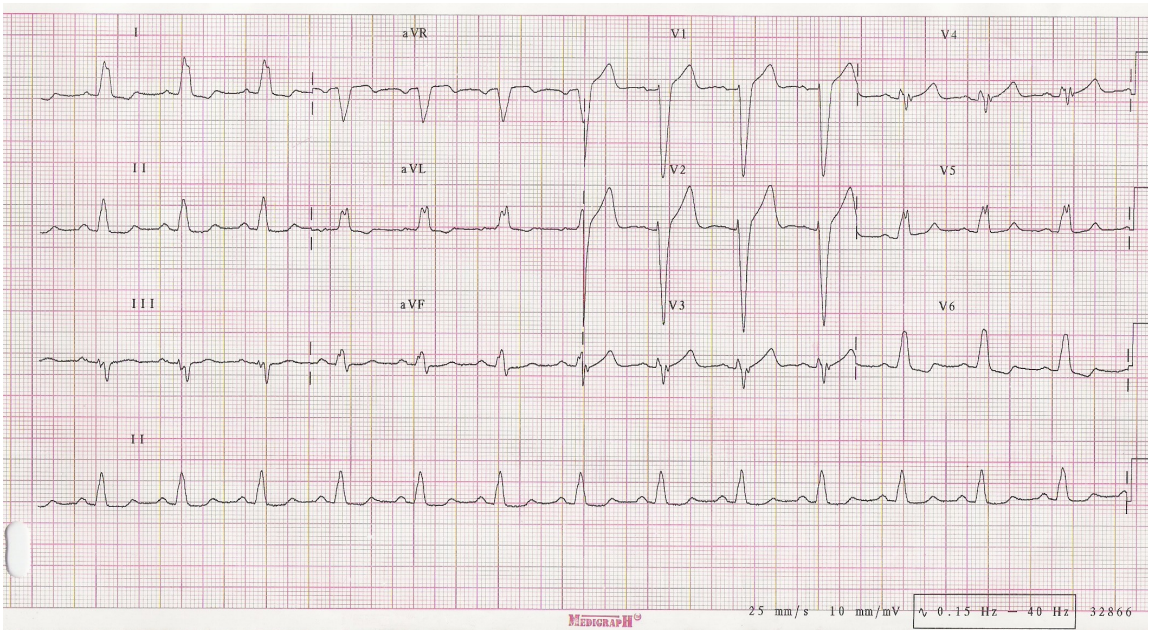
Appendix

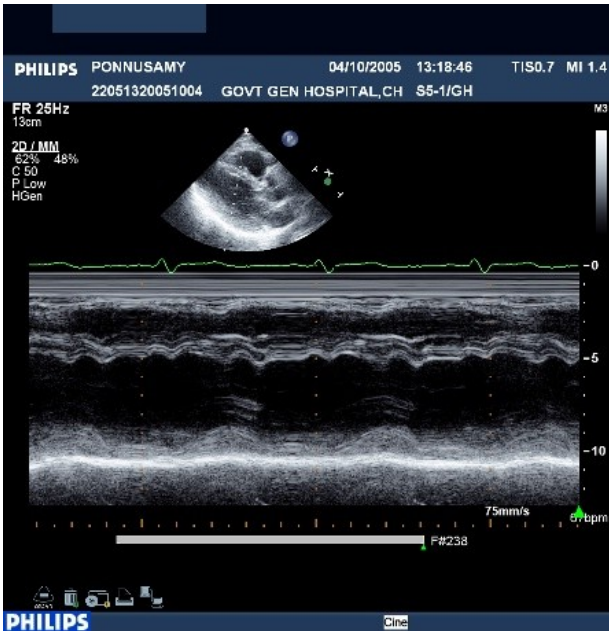


Philips IEE 32

ECG showing LBBB



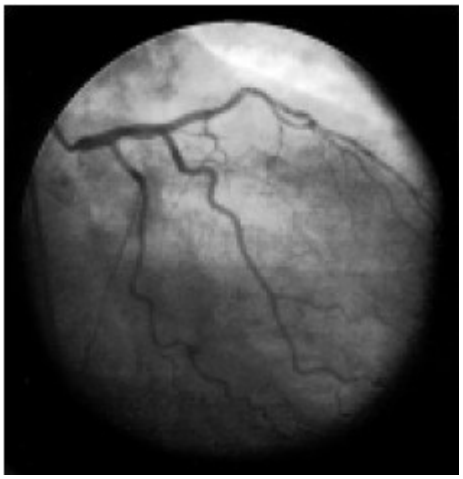
M-Mode showing Parodoxical septal motion



Pulsed Tissue Doppler Pattern showing reduced Sm

Figure.6: LBBB with out LAD Disease

Coronary angiography – Normal LAD



Pulsed Tissue Doppler

Doppler pattern of the mid septal wall in a patient with left bundle branch block but without left anterior descending coronary artery stenosis.
The post-systolic motion (PSM) is lower than the myocardial systolic peak velocity (Sm) (Sm/PSM = 1.56).

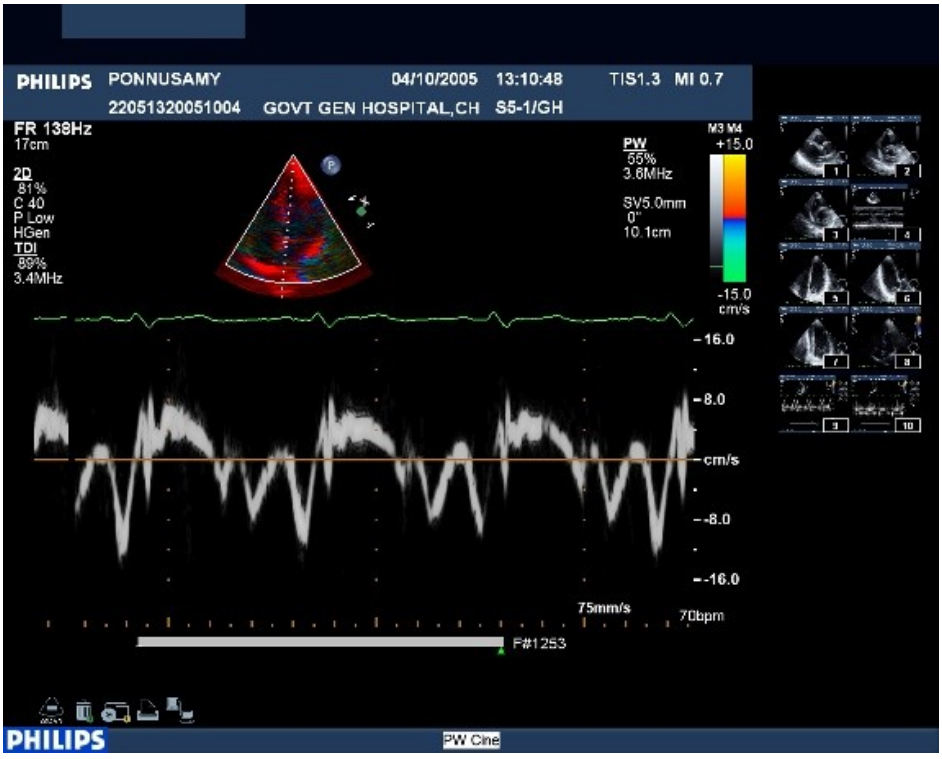
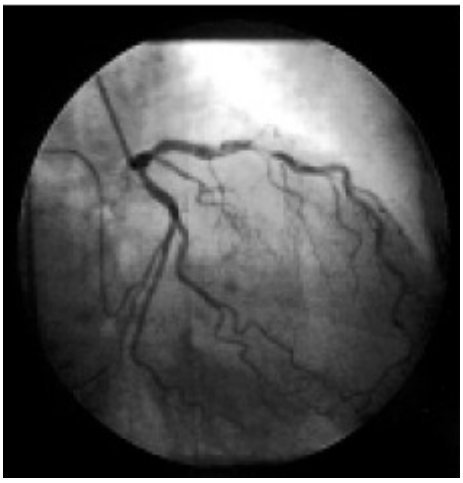


Figure 7a: LBBB with LAD Disease

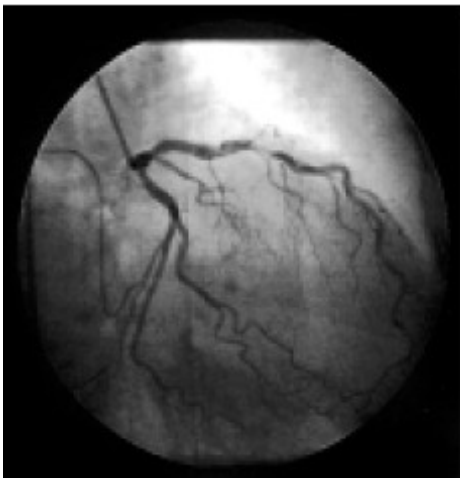
Coronary angiography – showing LAD Disease



Doppler pattern of the posterior septal wall in a patient with left bundle branch block and significant left anterior descending coronary artery stenosis. Note the equal amplitude of the post-systolic motion (PSM) and the myocardial systolic peak velocity (Sm)=PSM ratio). Am = myocardial atrial peak velocity; Em = myocardial early peak velocity.

Figure.7 b LBBB with LAD Disease

Coronary angiography – showing LAD Disease



Doppler pattern of the posterior septal wall in a patient with left bundle branch block and significant left anterior descending coronary artery stenosis. Note the higher amplitude of the post-systolic motion (PSM) and the myocardial systolic peak velocity (Sm)/PSM ratio < 1.) Am = myocardial atrial peak velocity; Em = myocardial early peak velocity.

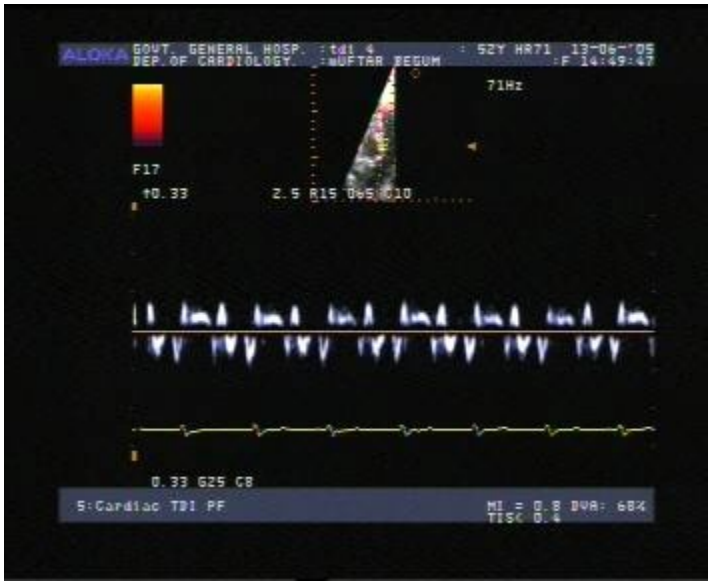
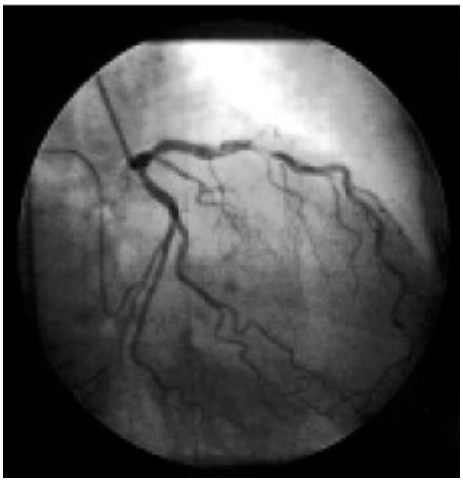


Figure 7c . LBBB with LAD Disease

Coronary angiography – showing LAD Disease



Doppler pattern of the posterior septal wall in a patient with left bundle branch block and significant left anterior descending coronary artery stenosis. Note the higher amplitude of the post-systolic motion (PSM) and the myocardial systolic peak velocity (Sm)/PSM ratio < 1. Am = myocardial atrial peak velocity; Em = myocardial early peak velocity.

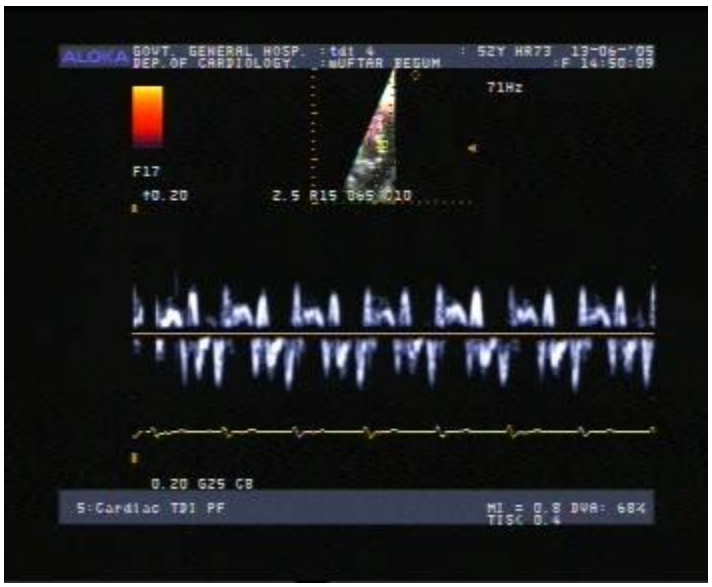
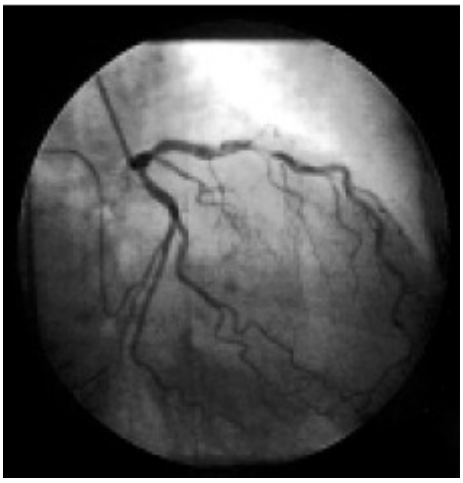


Figure 7c . LBBB with LAD Disease

Coronary angiography – showing LAD Disease



Doppler pattern of the posterior septal wall in a patient with left bundle branch block and significant left anterior descending coronary artery stenosis. Note the higher amplitude of the post-systolic motion (PSM) and the myocardial systolic peak velocity (Sm)/PSM ratio < 1. Am = myocardial atrial peak velocity; Em = myocardial early peak velocity.