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PROJECT REPORT

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NAME : **DR. KRISHNAKUMAR NAIR**

PROGRAMME : **DM CARDIOLOGY**

MONTH & YEAR OF SUBMISSION : **NOVEMBER 2002**

CERTIFICATE

I, Dr. KRISHNAKUMAR NAIR hereby declare that I have actually carried out the two projects under report.

Signature.....

Place : Trivandrum

Name in capital letters

Date : 12-11-02

Dr. KRISHNAKUMAR NAIR

Forwarded. He has carried out the two projects under report.

Signature
Head of the Department

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	Date	

PROJECT WORKS DONE

1. CORONARY ARTERIAL AND VENOUS ANATOMY

2. EVALUATION OF VENTRICULAR FUNCTION USING TISSUE DOPPLER ECHOCARDIOGRAPHY

DR. KRISHNAKUMAR NAIR
DM Cardiology
Department of Cardiology

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY TRIVANDRUM 695 011	Name	
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PROJECT REPORT

(PROJECT NO. 1)

TITLE

CORONARY ARTERIAL AND VENOUS ANATOMY

NAME : DR. KRISHNAKUMAR NAIR

PROGRAMME : D M CARDIOLOGY

MONTH & YEAR
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SREE CHITRA TIRUNAL INSTITUTE FOR
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CORONARY ARTERIAL AND VENOUS ANATOMY

KRISHNAKUMAR NAIR

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CORONARY ARTERIAL AND VENOUS ANATOMY

INTRODUCTION

Left Ventricular(LV) pacing improves patients with severe CHF. For LV pacing, a permanent pacing electrode is placed in a coronary sinus(CS) tributary. This therapeutic method has caused renewed interest in the precise angiographic anatomy of the CS and more precisely of its tributaries. Also,coronary venous system has been used for radiofrequency catheter mapping and catheter ablation. Data on cardiac venous anatomy in the Indian population is lacking.

Angiographic data on normal coronary size and branching patterns in the Indian population is not available. There is no significant data on correlation between coronary arterial and venous diameters.

CORONARY VENOUS SYSTEM

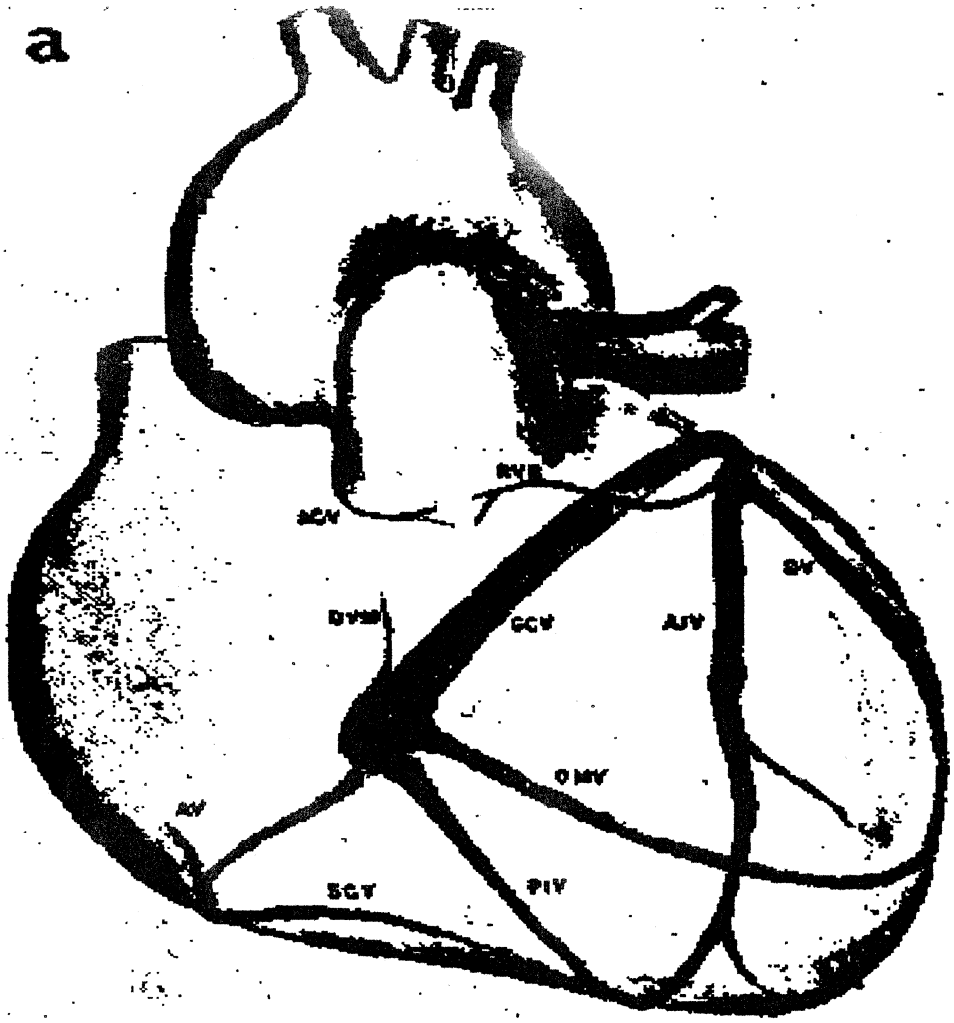
The venous system can be represented by two large triangles having their apexes on either side of the heart, and hinged at their bases on the great cardiac vein(GCV) and coronary sinus(CS). Small cardiac vein (SCV) is a tail like appendage that joins the CS at its ostium.

1st Triangle – Larger and more medially located

Sides – Anterior interventricular vein(AIV)and middle cardiac vein MCV(PIV-posterior interventricular vein))

2nd Triangle – Smaller and located on the free wall of the LV

Sides-(L) marginal vein(diagonal vein) , PLV(posterior left ventricular vein or obtuse marginal vein).The hinges run along the posterior IV groove are GCV + the coronary sinus1



3 coronary venous drainage systems can be considered.2

I. CORONARY SINUS (CS)

85% of the coronary venous blood, including the drainage from the interventricular septum(IVS),LV, atria, some of the right ventricle(RV), are carried by this elaborate system of veins.1

It is located near the crux of the heart. It extends 2 – 3 cm within the posterior AV grove between the left atrium(LA) and LV before it opens into the inferomedial aspect of the right atrium(RA).2 The CS orifice which is located between the orifices of the inferior

vena cava(IVC) and the septal tricuspid leaflet(STL) is guarded by a crescent shaped, rudimentary valve – the Thebesian valve. For anatomists , the CS ends at the Thebesian valve and the GCV ends in CS at Vieussens valve.3-5

CS receives venous blood from

- 1. Great cardiac vein
- 2. Middle cardiac vein
- 3. Small cardiac vein
- 4. Posterior and lateral veins of the LV
- 5. Left atrial oblique vein of Marshall-- all except the last have valved orifices.2
-

1. <u>Great Cardiac Vein (GCV)</u>

The *anterior inteventricular vein* lies in the anterior interventricular sulcus parallel to the LAD. It originates in the majority of the cases from the lower or middle third of the IV sulcus. It ascends to near the bifurcation of the LMCA, and then turns leftward to circle posteriorly under the LA in the left AV sulcus, where it is referred to as *the great cardiac vein.* Along its posterior course, the GCV receives venous blood from the large marginal and posterior LV branches and then becomes the CS near the posterior margin of the LA. It enters CS at an approximately 180 angle. It's ostium coincides, in the majority of cases, with the Valve of Vieussens. at approx the level of the Marhall vein (when the latter was visible)6.

2. <u>Middle cardiac vein (MCV)</u>

Synonym 'posterior interventricular vein'. Arises near the posterior aspect of the cardiac apex. Ascends in the posterior IV sulcus next to the PDA. Drains into (1) RA directly (2) or into CS just before it empties into the RA. Its mean diameter was 2.6 +/- 1.3 (0.7 to 6 mm)6

3. **<u>Oblique vein of Marshall</u>** It runs along the posterior LA and joins the GCV at the point where the latter becomes the CS. It is small in humans and is

the residua of the embryonic (L) superior cardinal vein .The oblique vein is continuous above with the Ligament of the left vena cava.2

4. **Small Cardiac Vein(SCV)**

The SCV winds around the right side of the heart in the AV sulcus between the RA and RV. It receives (1) some branches from the back of the RV and RA and(2)Right marginal vein. It terminates either in the Coronary sinus-near it's atrial end or in the RA.2

5. Right marginal vein

It passes right along the inferior cardiac margin (acute border) and drains adjacent parts of the RV. It is often grouped with the venae cordae minimae. It may

(1) join the small cardiac vein in the coronary sulcus

(2) but more often opens directly into RA.

(3) join the anterior cardiac veins (4) less often, joins the CS.2

6. Posterior and Lateral Veins of LV

LV venous branches arise from the lateral and posterior aspects of the LV

These drain into –(a)The great cardiac vein--- *lateral veins/lateral marginal veins*
(b)Middle of the CS – *posterior veins/left posterior veins* 6

II ANTERIOR RV VEINS

2-5 anterior cardiac veins7 originate in and drain (1) the anterior RV wall(2) and a region around the right cardiac border where the right marginal vein joins this group.They travel superiorly to cross the right atrioventricular(AV) sulcus, passing superficially or deep to the RCA.They enter (1) into RA anteriorly – separately/in various combinations
or (2) into a collecting vein at the base of the RA2

Venae cordis parva

These are small veins draining the right atrium and the right ventricle .They may be subdivided into(1)-those descending to the right atrio-ventricular sulcus and ending in the right atrium or in the small cardiac vein (2)and those opening by the Lannelongue foramina into the right atrium.8

III.THEBESIAN VEINS / VENAE CORDIS MINIMAE

These are tiny venous outlets draining the myocardium and are highly variable in number and size.They are derived from the intertrabecular spaces of the developing heart.They drain into primarily RA and RV, and to a lesser extent into the LA and LV .2

Venous bridges-

Nguyen et al reported that the coronary sinus may be covered by myocardial bundles. Myocardial bridges were detected above the anterior interventricular vein or its tributaries in 8% of specimens in Ortale's study.9

CORONARY ARTERIES

Diameters of the coronary arteries-

The diameters of the coronary arteries, both main stems and larger branches, have been reported. Such figures are of limited value since the technique is not always stated, physiological state is ignored and measurement of external and internal diameters is not clearly distinguished. Most measurements have been made on arterial casts or angiograms.10

Cherian B reported the luminal diameter of normal coronary arteries and their branches in normal young adult population from Trivandrum based on an autopsy study of 100 non coronary trauma victims(62male and 38 female). The total cross sectional area was calculated from the first segments by the formula 3.14X $D^2/4$.There was significant difference in total cross sectional area with regard to sex. The difference did not however persist after adjusting for body surface area. The coronary arteries in Caucasian males were significantly larger than those reported by Cherian et.al.. The difference persisted even after adjusting for body surface area. 11

DOMINANCE-

The term is misleading, since the left coronary artery almost always supplies a greater volume of tissue. There are multiple slightly differing definitions of dominance according to different studies. Lerer PK et al 12-16 ,Hutchins et al 17-18 and Chandrasekhar B19,20,21 have studied arterial dominance.

For identifying the dominant coronary artery, we adopted Hutchin's definition. In Hutchins' study17,18 left dominance was judged present when the posterior descending coronary artery and the posterior and lateral descending branches of the left ventricle arose from the left circumflex coronary artery. Right coronary arterial dominance was judged present when those vessels arose from the right coronary artery. Equal dominance was judged present when the posterior descending branch arose from the right coronary artery but did not extend beyond the posterior interventricular groove to give off descending branches to the left ventricle.

AIM AND OBJECTIVES OF THE STUDY-

1. To investigate the size and location of the coronary sinus and its tributaries in patients with normal coronaries, undergoing routine coronary angiography by screening for the venous phase .

2. To study the coronary artery pattern and distribution in patients with normal coronary arteries and to measure the sizes of major branches by quantitative coronary angiography(QCA).

3. To correlate between coronary arterial and venous diameters.

4. To compare coronary artery diameters in our population with published data of western population

MATERIALS AND METHODS-

The patients who were evaluated in SCTIMST with coronary angiogram to exclude/diagnose coronary artery disease formed the basis of our study.

EXCLUSION CRITERIA

Patients who were having CHD and VHD were excluded. Patients with significant stenosis of any coronary artery were also excluded.

Arterial study

Coronary angiograms were performed by standard Judkins technique in PHILIPS INTURIS 5500 Cath lab system. The projections used for the LCA were (1) anteroposterior caudal 35^0(2) 30° right anterior oblique (3) anteroposterior (4)60° left anterior oblique projection (5) left lateral projection and (6) LAO 40^0 Caudal 40^0 .

For RCA-60° left anterior oblique projection, anteroposterior cranial 30 ° and 30° right anterior oblique projections were used.Arteries smaller than 1.5mm in diameter were excluded from analysis .

Venous phase

8-10 ml of contrast was given into the left coronary artery and the venous phase which occurred 5-10 seconds after the injection was recorded and analysed. Venous phases were recorded in RAO 30, LAO 60, AP and lateral views.

For the right coronary artery,6-8 ml of contrast was given and the venous phase was recorded as above.

Measurements were made as follows

(1) CS – at its widest point

(2) Major tributaries-2cm from their draining point into the CS.

Veins smaller than 1.5mm in diameter were excluded from analysis .

Two observers made QCA measurements, on the digital acquisition system of the cardiac catheter suite (Philips, Netherlands). The (known) catheter diameter was used as the calibration object to assess the size of vessels,

with the settings of the image intensifier constant. Measurements were uniformly taken in diastole. Each artery was measured in defined segments (figure), and measurements were taken of the widest dimension in each segment. The left main (LM), left anterior descending (LAD) and left circumflex (LCX) coronary arteries were measured in the 30° right anterior oblique 20° caudal projection. The right coronary artery (RCA) was measured in the 60° left anterior oblique projection.

Segments of each epicardial coronary artery were measured as follows:

- LM (left main stem)-The length was measured from the ostium to the bifurcation. The diameter was measured in the mid portion.
- The LAD artery was divided into three segments, the proximal LAD (PLAD) segment (before the first septal), the mid-LAD (MLAD) segment (between the first septal and the second diagonal), and the distal LAD (DLAD) segment after the second diagonal branch of the LAD.
- The LCX was also divided into three segments, the proximal LCX (PCX) segment before the first obtuse marginal, the distal LCX (DCX) segment after the origin of the second obtuse marginal branch . The major obtuse marginal was also measured.
- RCA was divided into three segments, the proximal RCA (PRCA)before the major RV branch,the mid RCA upto the acute marginal and the distal RCA upto the PDA origin.The PDA was measured at its origin.
- We did not make any systematic attempt to canulate the conus artery seperately whenever it was not seen as a branch of the RCA. We have therefore not collected any data on separate origin of the conus artery.The total lumen diameter was calculated by adding the first segments of the three arteries (LAD, RCA, LCX)
- Body surface area was calculated using the formula of Dubois and Dubois from height and weight

STATISTICAL ANALYSIS

Data was analysed using the SPSS (versions 6 and 10) statistical software. Continuous variables were expressed as mean +/-standard deviation (SD).Comparisons of means with the mean coronary artery diameters of 2 different Caucasian populations was performed using the one-sample t-test. The coronary artery diameters indexed to body surface area were also compared with similar Caucasian indexed data using the same test.

Tests of correlation were performed between coronary artery and venous diameters, body surface area, myocardial wall thickness and left ventricular end diastolic volume. A probability of $p<0.05$ was considered statistically significant. The relation between arterial dominance and arterial supply to the sinus node and the AV node was found out using crosstabs. Gender differences between artery dimensions were looked for using the independent t-test.

RESULTS

We analysed the angiograms of 235 patients(68 females). The mean age was 50.55+/- 10.46 years.

CORONARY VENOUS ANATOMY-

The mean diameter of the coronary sinus(CS) was 7.37 +/- 1.92 mms. The mean diameters of the anterior inter-ventricular vein (AIV) and middle cardiac veins(MCV) were 2.78+/-0.74 and 3.29+/- 0.98 respectively. The mean distance from the middle cardiac vein (MCV) to the CS ostium was 11.21+/- 3.76 mms.

15 patients out of the 235, had a dimunitive anterior interventricular vein(AIV), the drainage area of which was by a larger MCV. 13.4%(6/235) of patients had only 1 vein between the great cardiac vein(GCV) and the middle cardiac vein .47.6%had 2 ,31.7% had 3,6.1% had 4 and only 1 patient had 5 veins arising between the GCV and the MCV.

The mean number of the posterior left ventricular vein(PLV) was 1.67+/- 0.59 and that of the lateral marginal vein(LMV) was 1.09+/-0.35. The mean diameter of the former was 2.85+/- 0.85 and that of the latter was 2.37+/- 0.50 mms, respectively. 96% of the patients had a PLV measuring at least 2mm. 64.5% of the patients had a LMV of at least 2 mm in size. Only 2 % of patients had no PLVs or LMVs more than 2 mm in diameter which could be used for LV pacing.

SMALL CARDIAC VEIN-

The small cardiac vein was clearly visualized only in 63 patients. The mean diameter of the small cardiac vein was 2.12+/-0.49 mm. The mean diameter of the right marginal vein was 2.46+/-1.03 mm. The small cardiac vein drainage was in 4 patterns-

1.To RA-26 patients

2.To CS-23 patients

3.Both to RA and CS-3

4.RMV and CS-11

The right marginal vein drainage was in 5- patterns-

1.To RA-23 patients via a common opening with the SCV

2.To CS-11

3.To RA and CS-3

4.MCV to CS-1

5.Separate opening to RA-1

In cases with a common opening of SCV and RMV to RA, another venous tributary could be identified in 18 patients.

VEIN OF MARSHALL

It was identified in 61 patients.

CORONARY ARTERY ANATOMY-

Out of the 235 patients studied, 67.5%(158/235) patients had a right dominant system, 18.4%(44/235)had left dominant and 14%(33/235) had co-dominant coronary arteries.

LMCA-The mean diameter of the LMCA was 3.998+/-0.843 mm and the mean length was 10.91+/-5.08 mms.

LAD -The diameter of the LAD in its proximal, mid and distal segments were 2.97+/-0.74 mm,2.61+/-0.68 mm and 2.27 +/-0.61 mm respectively. Regarding the LAD type, 9%(4/235) was Type I, 4.7%(11/235) was Type II and 93.3 % (219/235) was Type III .

The LAD gave off a mean of 2.28 +/-0.7 septals (greater than 1.5 mm in diameter)per patient.9 %(20/222) patients had 1 septal,57.7 %(128/222) had 2 septals,29.7 %(66/222) had 3 septals,2.7 %(6/222) had 4 septals and 0.9%(2/222) had 5 septals.

An average of 2.39 +/-0.78 diagonals (greater than 1.5 mm in diameter) were given off from the LAD. 10.09%(22/218) patients had 1 diagonal,49.5%(108/218) had 2 diagonals,35.78%(78/218) had 3 diagonals,3.7%(8/218) had 4 and 0.9%(2/218) had 5 diagonals. The lateral recurrent branch of LAD was prominent in 32 patients.

LCX - The diameter of the LCX in its proximal, mid and distal segments were 2.89+/-0.73 mm,2.53+/-0.78 mm and 2.257 +/-0.71 mm respectively. The major OM was 2.25 +/- 0.66 mm in diameter. The LCX gave off a mean of 2.32 +/-0.78 OM's(greater than 1.5 mm in diameter) per patient.13.9 %(30/216) patients had 1 OM, 45.4 %(98/216) had 2 OM's,35.2 %(76/108) had 3 OMs and 5.6 %(12/216) had 4 OM s.

RCA - The diameter of the RCA in its proximal,mid and distal segments were 2.88+/-0.83 mm,2.64+/-0.75 mm and 2.36 +/-0.68 mm respectively. The PDA was 2.18 +/- 0.524 mm in diameter.60.2%(130/216) patients had 1 RV branch ,31.5% (68/216) had 2 RV branches and 8.3%(18/216) had 3 RV branches. 93.2%(192/206) patients had 1 acute marginal, 5.8%(12/206) had 2 acute marginals and 0.97%(2/206) had 3 acute marginals.

SA-NODAL ARTERY-

The sinus nodal artery took origin from the RCA in 43.89%(97/221) cases ,from the LCX in 31.22 %(69/221) cases and from the LACX (from the LCX) as a branch in 9.5(21/221) % cases.13.6 %(30/221) patients had 2 SAN branches 1 from the RCA and 1 from the LCX.0.9%(2/221) had 1 SAN from the PLV branch of the RCA. 0.9%(2/221) had 2 SAN branches-1 from the distal RCA and 1 from the LCX respectively.

AVNODAL ARTERY-

The AV nodal artery arose from the RCA in 80%(177/221) cases and from the LCX in 18.2 % cases . 0.9 %(2/221) cases had 1 AVN branch from the PLVB from the LCX and 0.9 %(2/221) had 2 AVN arteries both from the RCA.

LEFT ATRIAL CIRCUMFLEX ARTERY-

The LACX artery took origin from the LCX in 97.2%(214/221) cases and from the RCA in 0.9 %(2/221) cases .1.8%(4/221) patients had 2 LACX both from the LCX.

POSTERIOR DESCENDING CORONARY ARTERY-

The PDA arose from the RCA in 67.5%(157/233) cases and from the LCX in 15.4%(36/233) cases.4 different combinations of double (2) PDA's were seen. Two PDA's arose both from the RCA in 4.3%(10/233) cases,one from the RCA and one from the RV branch in 3.4%(8/233) cases,one from the RCA and one from the acute marginal branch of the RCA in 1.7% (4/233)cases,and one from the RCA and one from the LCX in 6.9%(16/233) cases.

POSTERIOR LEFT VENTRICULAR BRANCHES(PLVB)

PLVB's arose from the RCA alone in 71.2%(160/225) cases, from the LCX alone in 22.5%(50/225)cases and from both the RCA and LCX in 4.5%(10/225)cases.

The total lumen diameter which is the sum of the diameters of the proximal segments of the LAD,LCX and RCA was 8.74mm

CORRELATIONS

Myocardial wall thickness was not correlated with any venous or arterial diameter tested. The CS diamater was positively correlated with the diameters of the LMCA(P=0.0001,correlation coefficient=0.261),proximal LCX (P=0.0001,correlation coefficient=0.5037) and the proximal LAD(P=0.0001,correlation coefficient=0.3119). The great cardiac vein had no correlation with the LAD diameter. There was no correlation between MCV and PDA diameter.

The diameters of GCV and MCV had no relation with the number of PLV's, the number of LMV's and the total number of PLV's + LMV's.

RELATION BETWEEN ARTERIAL DOMINANCE AND ARTERIAL SUPPLY TO THE SINUS NODE(SAN) AND THE AV NODE(AVN)-

For identifying the dominant coronary artery, we adopted Hutchin's definition. In right dominant coronary arterial system, the SAN artery arose from the RCA in 41.1%,from the LCX in 37.7% and from both the RCA and the LCX in 21.2% cases. In left dominant systems, the SAN artery arose from the RCA in 61%,from the LCX in 27.8% and from both the RCA and the LCX in 11.1% cases.

In codominant systems, the artery of origin for the SAN was the RCA in 34.5%,LCX in 44.8% and both RCA and LCX in 20.7% cases.

Regarding the AV nodal artery, in right dominant coronary arterial systems, it arose from the RCA in 97.3%,from the LCX in 2.7% cases. In left dominant systems, the AVN artery arose from the RCA in 15.8% and from the LCX in 84.2% cases. In co- dominant systems, the artery of origin for the AVN was the RCA in 81.5% and LCX in 18.5% cases.

MEAN ARTERIAL AND VENOUS DIAMETERS INDEXED TO BODY SURFACE AREA

Variable	Mean	Std Dev	Minimum	Maximum
LMCA-DIAM	2.37	.53	1.41	3.53
LMCA-LENGTH	6.64	3.07	2.08	13.45
LAD-PROX	1.79	.45	1.02	2.71
LAD-MID	1.55	.42	.80	2.47
LAD-DISTAL	1.35	.40	.49	2.29
LCX-PROX	1.69	.42	.88	2.72
LCX-MID	1.48	.42	.63	2.47
LCX-DISTAL	1.31	.40	.52	2.47
OM	1.31	.37	.61	2.32
RCA-PROX	1.74	.46	.76	2.95
RCA-MID	1.61	.47	.76	2.83
RCA-DISTAL	1.42	.42	.65	2.41
PDA	1.31	.34	.65	2.27
CS	4.39	1.03	2.69	7.41
GCV	1.66	.43	.87	2.97
MCV	2.04	.61	.99	3.68
PLV	1.76	.47	1.03	2.97
LMV	1.49	.40	.32	2.69

CORRELATION OF BODY SURFACE AREA AND MEAN ARTERIAL AND VENOUS DIAMETERS

-- CORRELATION COEFFICIENTS --

	CS	GCV	MCV	PLV	LMV	LMCA-DIAM
BSA	.3089	.2308	.1263	.1450	.2555	.1275
	(117)	(133)	(131)	(131)	(69)	(137)
	P= .001	P= .008	P= .151	P= .098	P= .034	P= .138
	LMCA-LENGTH	LAD-PROX	LAD-MID	LAD-DIST	LCX-PROX	LCX-MID
BSA	-.0153	.1979	.3191	.2203	.4204	.5024
	(137)	(137)	(137)	(137)	(137)	(137)
	P= .859	P= .020	P= .000	P= .010	P= .000	P= .000
	LCX-DIST	OM	RCA-PROX	RCA-MID	RCA-DIS	PDA
BSA	.4672	.4838	.2251	.1926	.1411	-.0656
	(133)	(133)	(135)	(133)	(133)	(77)
	P= .000	P= .000	P= .009	P= .026	P= .105	P= .571

Among the venous diameters, body surface area was positively but weakly correlated with the coronary sinus, great cardiac vein and lateral marginal vein diameters but not with the middle cardiac vein and posterior left ventricular veins.

Among, the arterial diameters, body surface area was positively but weakly correlated with the proximal, mid and distal LAD and LCX, major OM, proximal and mid RCA diameters but not with the LMCA diameter, RCA distal and PDA diameters.

CORONARY ARTERY DIAMETERS –COMPARISON TO SERIES FROM THE WEST

Compared to data from Baroldi's series of Caucasian population(1983),there was a statistically highly significant difference from LAD,LCX and RCA diameters of our population. However,there was no significant difference between LMCA length and diameter.

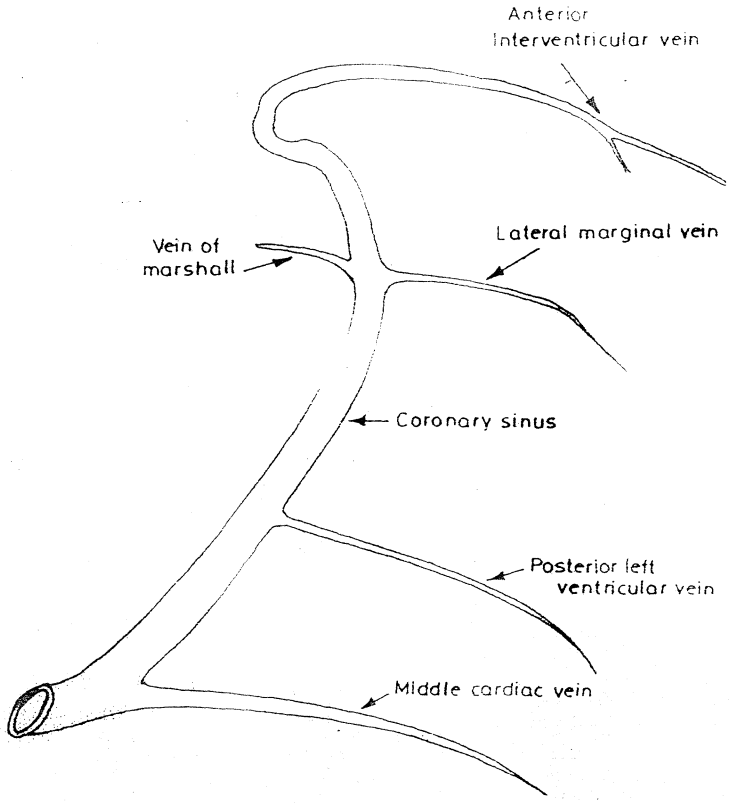
Compared to the recent Caucasian series by Lip (1999),the arterial diameters of the LMCA, LAD-proximal, mid and distal, LCX-proximal and distal and proximal RCA were significantly smaller.

After indexing to BSA, no significant difference was seen between diameters of the LMCA, LCX and proximal RCA. However LAD diameters remained smaller significantly, even after indexing to BSA.

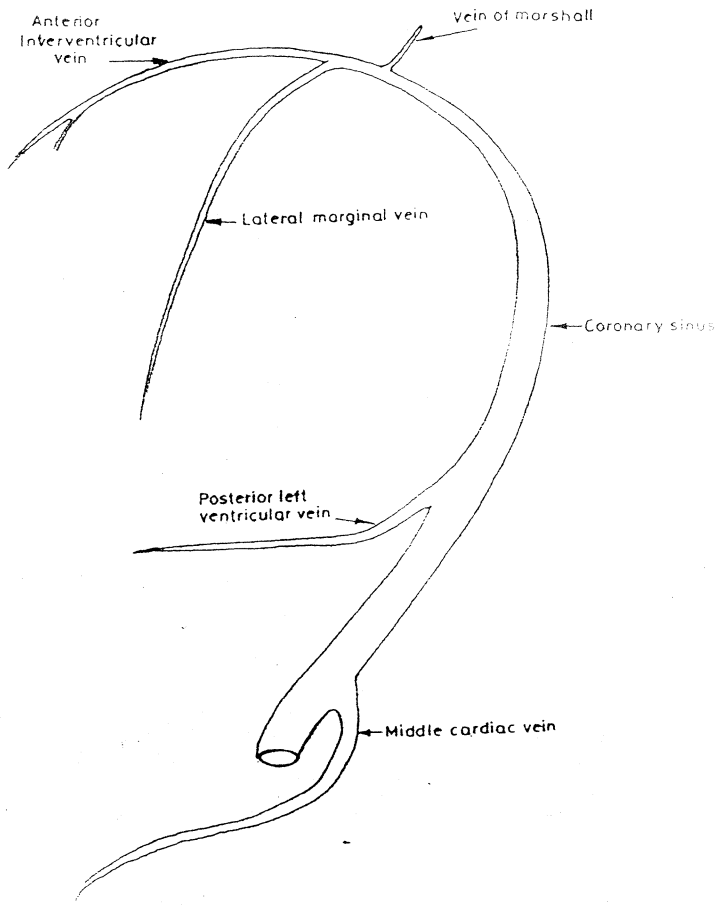
At the same time, the diameters of the major OM and the PDA were significantly larger in our population,with and without division by BSA.

The dimensions of the coronary arteries in the different series ,compared to our own, are listed in the table below.

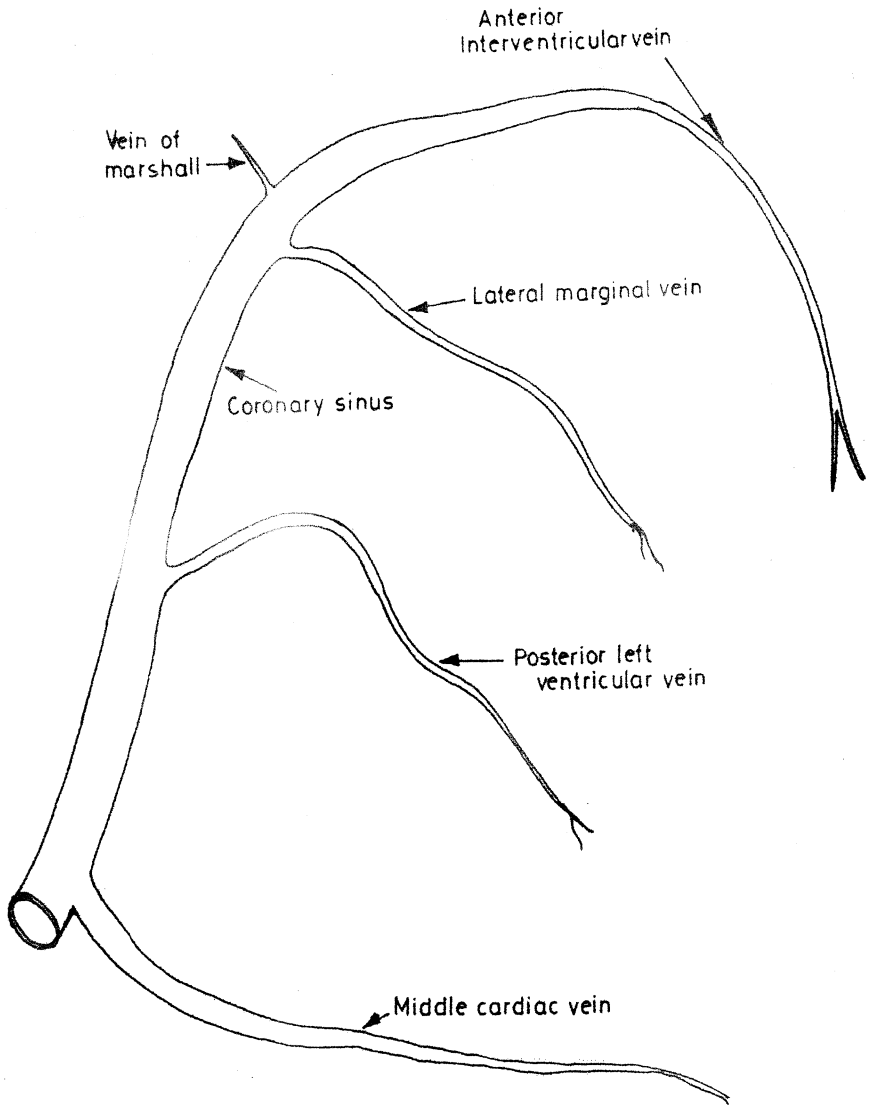
	CheriaN male	Cherian Female	Hurst-Caucasian	Lip-Caucasian	Lip-Indoasian	Our Series	Pvaluewith Hurst	Pvalue with Lip
LMCA DIAM			4(2-5.5)	4.44+/-*0.91*	3.98+/-.67	3.997+/- .84	.954	.000
LMCA LENGT	8.7+/-0.38	7.7+/-0.42	8.7-male 7.7-fem	----	------	10.91+/-5.08		
LAD-PROX	3.48+/-0.5	3.21+/-0.5	3.6(2-5)	5.53+/-0.69	3.22+/.56	2.98+/-.737	.000	.000
LAD-MID	2.54+/-0.61	2.33+/-0.34		3.13+/-0.68	2.77+/-.56	2.61+/-.68		.000
LAD-DISTAL	1.59+/-0.36	1.56+/-0.3		2.44+/-0.62	2.26+/-.6	2.27+/-.61		.000
LCX-PROX	2.78+/-0.54	2.71+/-0.52	3(1.5-5.5)	3.17+/-0.63	3.01+/-.66	2.8842+/-.732	.023	.000
LCX-MID	2.13+/-0.59	1.98+/-0.46		---	=			
LCXDISTAL				2.47+/-0.58	2.37+/-.67	2.25+/-.71		.000
OM				2.08+/-.62	1.96+/-.53	2.25+/-.65		.000
RCA-PROX	3.4+/-0.65	3.03+/-0.63	3.2(1.5-5.5)	3.35+/-.69	2.98+/-.63	2.8779+/-.823	.000	.000
RCA-MID	3.06+/-0.78	2.67+/-0.72		---	====			
RCA-DISTAL	2.31+/-0.84	2.06+/-0.58		--	---			
PDA				2.01+/-.5	1.69+/-.48	2.17+/-.52		.002

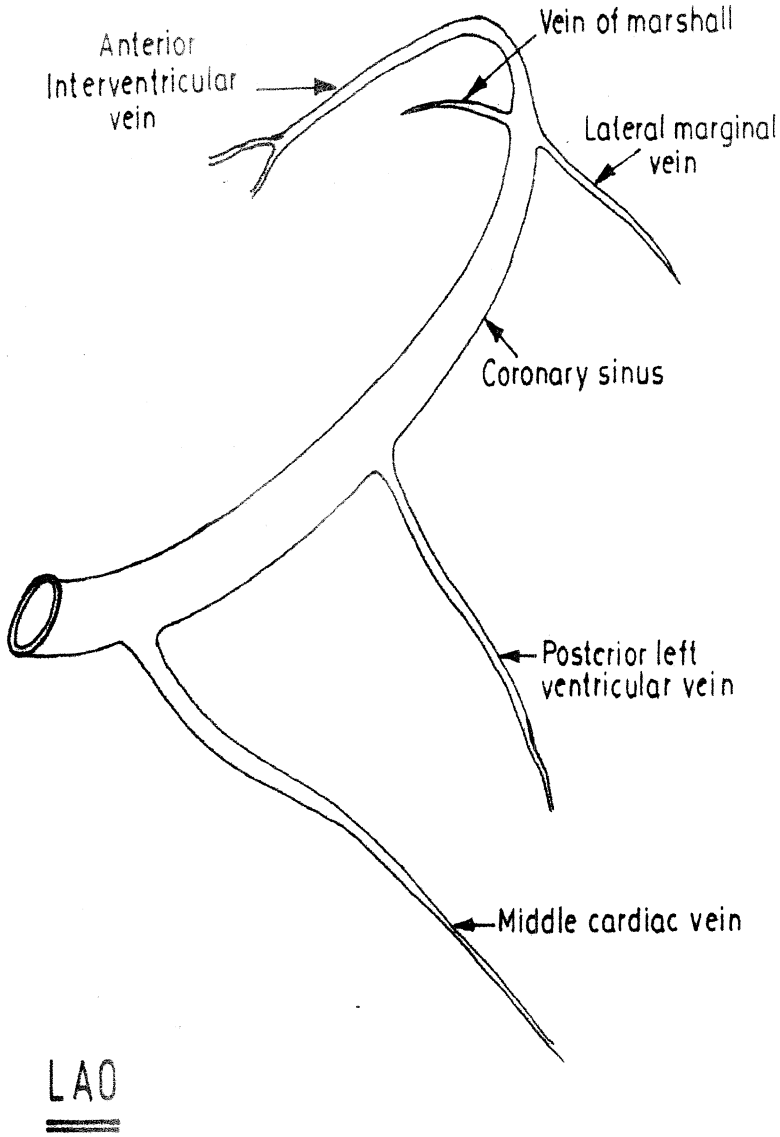


ANTEROPOSTERIOR VIEW



LATERAL VIEW


Anterior
Interventricular vein
Vein of
marshall
Lateral marginal vein
Coronary sinus
Posterior left
ventricular vein
Middle cardiac vein
RAO


Anterior
Interventricular
vein
Vein of marshall
Lateral marginal
vein
Coronary sinus
Posterior left
ventricular vein
Middle cardiac vein
LAO

DISCUSSION

LV pacing which is increasingly used now a days, has renewed interest in studying the cardiac venous anatomy. We used the levophase images to study the venous system. Out of the 2 methods of visualization of the coronary venous system, direct canulation and studying the levophase of the coronary arteriogram, each method has its own merits and demerits.

The advantages of studying the venous phase of CAG compared to retrograde venography are 1,6

(1) simple

(2) canulation of CS and a separate venous puncture is not necessary.

(3) most important – the diameters and position of the tributaries of the CS remain more natural since the study does not require acute occlusion of the CS – a procedure that clearly modifies the dimensions of the vessels and probably their angulation at points of anastomosis.

But the visualization of the veins on direct cannulation is superior to the levophase technique. Studying the venous phase of arterial injection may be a less accurate visualisation of the small coronary venous vessels as only 'major' veins will be visualized. 6

For pacing the left ventricle, the options available are MCV, LMV or PLV. But lead insertion into MCV may not be easy because

(1) Of its 90 degree angulation at its junction with the CS,

(2) It's proximity to the coronary ostium – causing the lead to pull back before entering the MCV

(3) Pacing through the MCV may not be optimal – pacing the LV from the MCV or the posterior veins close to it may pace the diaphragm

(4) The absence of demonstrated hemodynamic improvement at this very posterior site.6

The mean distance from the middle cardiac vein (MCV) to the CS ostium is important because if this distance is short, it will be technically difficult to canulate the MCV and pass the lead through the CS because of the angulation between the veins.6

In pacing therapy for CHF, the posterior or postero-inferior base is the area of choice. That site is the easiest to reach by transvenous route, by inserting the lead into a lateral or posterolateral vein over the LV free wall. 22

Regarding the veins useful for pacing (PLV's and LMV's), 96% of the patients in our study had a PLV measuring at least 2mm which is the minimum size which can accommodate a left ventricular (LV) pacing lead. 64.5% of the patients had a LMV of at least 2 mm in size. Only 2 % of patients had no PLVs or LMVs more than 2 mm in diameter which could be used for LV pacing.

In Gillard's study, the size of the left posterior veins were 2.25 +/- 1.2 mms and In <5%, absent LP or LM veins limited the ability to pace the LV endovenously in that study.6

Meisel et al did retrograde venography after ICD implant in 129 pts (86 pts, optimal visualization). A therapy that requires lead placement in a LMV or a PLV may be limited since LMV occurred in 71 (82%) and PLV occurred only in 47 (55%) cases. But, if either a LMV or a PLV was acceptable ,a high percentage of pts 85 (99%) would be eligible.23

The mean MCV diameter was significantly larger than the mean GCV diameter in our population. However,in Gillard's study,the mean GCV diameter(3.55 +/- 1.24 mm) was larger than the mean MCV diameter(2.62 +/- 1.26 mm).6

15 patients out of the 235, had a dimunitive anterior interventricular vein(AIV), the drainage area of which was by a larger MCV. From our search of literature, we could not find any similar report.

The dimensions of the GCV and MCV were negatively correlated with the number of posterior veins in 2 previous studies. 4,6This may have practical, though undesirable implications-- the more the tributaries, the less accessible they are with available pacing leads. However, in our study, the GCV diameter and the MCV diameter had no relation with the number of PLV's, the number of LMV's and the total number of PLV's + LMV's

2 tributaries of the CS were consistently present in all our patients- the AIV and the MCV-as was also reported in Gillard's study.6

AIV - AIV was always present in our study as reported in previous studies. In most previous studies, it had a diameter>2 mm allowing lead placement. However, in our study, 15 patients out of the 235, had a dimunitive anterior interventricular vein(AIV), the drainage area of which was by a larger MCV .

MCV- The other vein which was usually seen was the MCV.

SMALL CARDIAC VEIN-

We could identify the SCV only in 63 patients. This small number may be partly because of inadequate filling and partly because the small cardiac vein itself was small in some cases.It may be absent in some patients as already described.

In Ortale's study using anatomical dissection, the small cardiac vein was present in 54% of specimens9.In another autopsy study,the SCV could be identified only in 36% cases.24

The small cardiac vein drainage was noted to have 4 patterns and the right marginal vein drainage was noted to have 5 patterns. We are not aware of any other angiographic study which has commented upon the RCA venous drainage.

Because of the small numbers, we have not looked for any statistical correlation between the different vessel diameters(SCV,RMVand RCA).

SYSTOLIC NARROWING OF CORONARY SINUS AND TRIBUTARIES

Nguyen et al. and Ortale 9 has reported that the coronary sinus may be covered by myocardial bundles. A similar finding was noted in 37 of our patients - but we are not sure if these are true bridges analogous to the coronary arterial myocardial bridges. This finding could be

a) due to squeezing in the AV ring

b) due to emptying and filling of the CS due to ascent and descent of the AV ring.

CORONARY ARTERY

The mean length of the LMCA in our series was 10.91+/-5.08 mm .Green et al and Fox et al have showed that in normal hearts, the left main coronary artery is approximately 10mm in length.24,25 In Cherian's study,the average Left Main Stem artery length in the male was 0.87 cm and in the female 0.77 cm.11 Both were significantly lesser than the corresponding Western figures.

Most patients had Type III LAD arteries.

The sinus nodal artery took origin from the RCA in 40.9%(90/221) cases, from the LCX in 38.1 %(64/221) cases. 13.6 %(30/221) patients had 2 SAN branches 1 from the RCA and 1 from the LCX..Older studies note that this artery's origin is variable: from the left coronary in about 35 % (Hutchinson 1978), arising from its circumflex branch and from the RCA in the rest. 26

The AV nodal artery arose from the RCA in 80%(177/221) cases and from the LCX in 17.3 % cases. Autopsy studies have observed this artery to arise from the RCA in 80% of hearts, according to Hutchinson (1978).26

The total lumen diameter was 8.94mm in males and 8.4mm in females in our study. In Cherians study, it was found to be 9.65 mm in the male and 8.94 mm in the female. Cherian's total lumen diameters were established to be significantly lesser than those of Caucasians even when adjusted for body surface area. 11

Asians are said to have significantly smaller total vessel diameter compared to Caucasians. They have smaller body surface area too. This may have important implications in this group suffering excess mortality from coronary artery disease.27

The origin and size of the coronary arteries in the north-west Indians have been studied at autopsy in 500 adult hearts. The Left Coronary artery was found to be significantly smaller than the Western figures and the Right Coronary artery only marginally so.

Dominance

Ours was predominantly right dominant coronary system(67.5%).The prevalence of left dominant pattern of supply is reported as 8 % in patients undergoing

cardiac catheterization21.Hutchins et al have reported that in their series, 70% had a right dominant pattern,20% had a codominant system and 10 % had a left dominant system.14

We compared the diameter of coronary arteries in our study subjects to the data from Baroldi's series of Caucasian population(1983)28,29 and Lip's recent Caucasian series(1999)27. It was found that our major coronary arterial diameters were significantly smaller but after indexing to BSA, no significant difference was seen between diameters of the LMCA, LCX and RCA.

However LAD was found to be smaller even after indexing to BSA. At the same time, the diameters of the major OM and the PDA were significantly larger in our population, with and without division by BSA. We hypothesize that the LAD may be smaller because of the larger size of the OM and the PDA.

Lip G Y H et al27 compared the size of atheroma-free proximal and distal epicardial coronary arteries of Indo-Asians and Caucasians from normal coronary angiograms from 77 Caucasians and 39 Indo-Asians. The two groups were comparable for dominance of the coronary arteries. Indo-Asian patients had generally smaller coronary arteries, however, after correction for body surface area, none of these differences in size were statistically significant. Thus, the smaller coronary arteries in Indo-Asian patients were explained by body size alone and were not due to ethnic origin *per se*. Saldana *et al* found that the calibre of grafted vessels were smaller in Indo-Asians with a mean diameter of 1.5 mm in 55% of their cohort28. Dhawan and Bray also found Indo-asian coronaries to be smaller29.

Our present study is broadly consistent with previous reports of smaller coronary arteries in Indo-Asians28,29. In conclusion, Indo-Asians as a whole have generally smaller coronary arteries than Caucasians, although this may be a reflection of their smaller stature rather than a true size difference. This finding has important therapeutic implications since smaller coronary arteries may result in technical difficulties during bypass graft and intervention procedures such as PTCA, stents and atherectomy. In smaller arteries, atheroma may also result in more severe disease on haemodynamic grounds, compared with larger diameter arteries.

Table 2 Coronary artery segment sizes in Indo-Asians and Caucasians

	LMS	*PLAD*	*MLAD*	*DLAD*	*PCX*	*DCX*	*OM*	*PRC*	*DRC*
(a) Unadjusted mean diameter (mm) of coronary artery segments									
Indo-Asians	3.98	3.22	2.77	2.26	3.01	2.37	1.96	2.98	1.69
SD	0.67	0.56	0.56	0.60	0.66	0.67	0.53	0.63	0.48
Caucasians	4.44	5.53	3.13	2.44	3.17	2.47	2.08	3.35	2.01
SD	0.91	0.69	0.68	0.62	0.63	0.58	0.62	0.69	0.50
p	0.003	0.012	0.005	0.13	0.22	0.46	0.31	0.006	0.003
(b) Coronary artery segment size adjusted for individual body surface area (mean coronary artery diameter/body surface area)									
Indo-Asians	2.26	1.83	1.57	1.28	1.71	1.34	1.12	1.70	0.97
SD	0.41	0.34	0.29	0.31	0.39	0.37	0.29	0.39	0.27
Caucasians	2.38	1.89	1.68	1.31	1.71	1.32	1.10	1.79	1.06
SD	0.47	0.37	0.37	0.32	0.32	0.29	0.31	0.39	0.26
p	0.15	0.44	0.10	0.63	1.0	0.78	0.86	0.25	0.09

Abbreviations: LMS - left main stem; PLAD/MLAD/DLAD - proximal/mid/distal left anterior descending; PCX/DCX - proximal/distal circumflex; OM - obtuse marginal; PRC/DRC - proximal/distal right coronary artery.

LIMITATIONS OF THE STUDY-

1. This study is limited by its cross-sectional nature of patients attending a city centre teaching hospital and its results may not be generalisable to the population as a whole.

2. In addition, we have studied normal coronary angiograms in patients who had reasons to undergo the investigation, such as chest pain and such patients may not represent a 'normal' population. Nevertheless, undertaking coronary angiography on apparently healthy asymptomatic controls would be unethical.

3. The influence of age, sex, left ventricular mass and vasomotor tone on size of coronary arteries has been previously recognised. The poorer outcome of women undergoing PTCA or CABG may be related to their smaller coronary arteries. However, we did not quantify left ventricular mass or vasomotor tone as part of this study.

4. It is reported that diameters measured by angiography in vivo are smaller than the ones measured at autopsy.

5. Studying the venous phase of arterial injection may be a less accurate visualisation of the small coronary venous vessels. Only 'major' veins were visualized However, this is of little importance, since our study was designed with a view to insertion of a permanent electrode.

6. The study was on normal hearts. Whether these findings apply to markedly dilated hearts, in which LV pacing is often considered is not clear

CONCLUSIONS –

CORONARY VENOUS ANATOMY

1.The pattern of normal coronary venous drainage in our population is established.

2. A posterior vessel for lead introduction for LV pacing was available in 96% of patients, and a posterior or lateral vessel was available in 98% of the patient population.

CORONARY ARTERY ANATOMY-

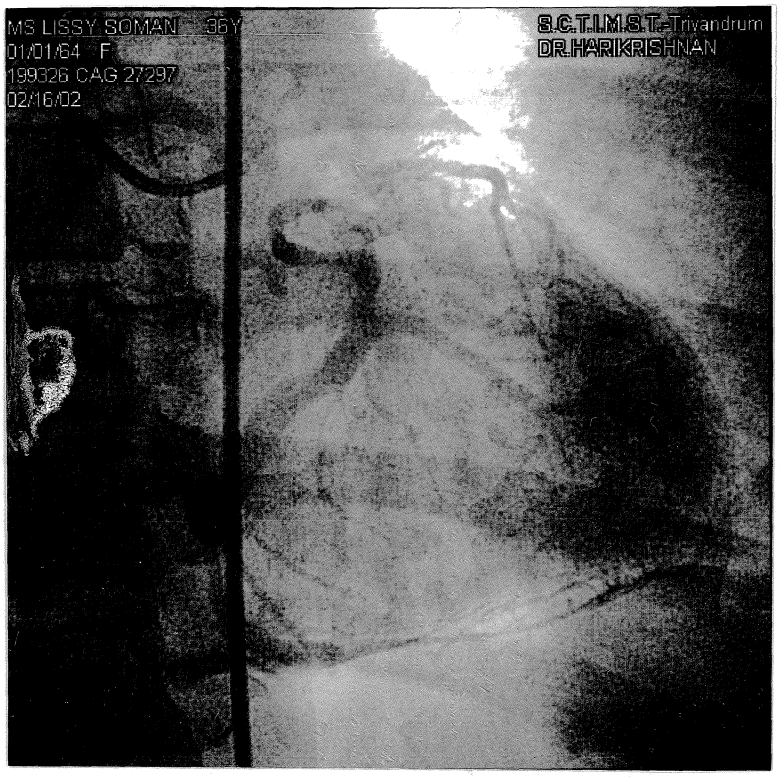
1. The normal coronary arterial and branch sizes were established for our study population which comprised of a normal adult South Indian population

2. Coronary arterial diameters in our population are significantly smaller than the Caucasian population.

3. But when indexed to BSA, the coronary artery sizes become comparable to the Caucasian population except the LAD.

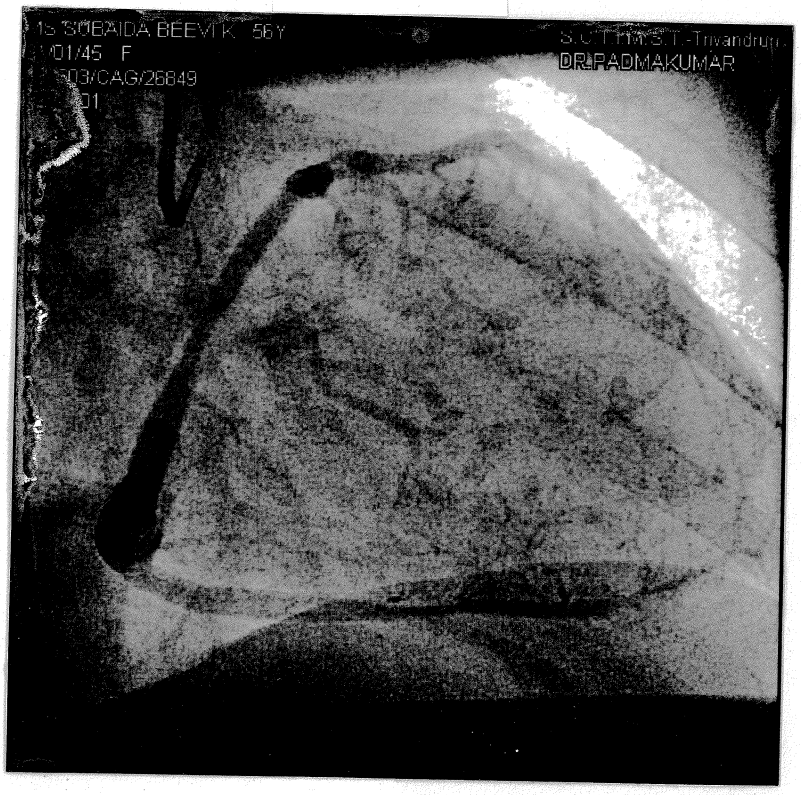
4. The LAD artery is small in South Indians, possibly because of the large sizes of the OMs and PDA which share the supply of adjacent areas.

ANTEROPOSTERIOR VIEW



LATERAL VIEW

RAO VIEW



LAO VIEW

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V1	V3	V5	V8	V9	V10	V14	V15	V16	V17	V18	V19	V20	V21	V22	V23	V24	V25	V26	V29	V30	V31	V32	V33	V34	V35	V36	V37	V38	V39	V40	V41	V42	V43	V44	V45	V46	V47	V48	V49	V50	V51	V52
Kanaksugathan	M		27108	12.9														L	3.7	5.1	1.8	1.6	1.6	3	1	2	2.3	2.1	2	1.5	3	1.2	1	0.8		2	1				X	
Kannan T K	M		27108	12.9														L	3.7	5.1	1.8	1.6	1.6	3	1	2	2.3	2.1	2	1.5	3	1.2	1	0.8		2	1				X	
Indira	F	1.76	27396	14.8		5	3.3	2.8	2	2.1	0	N		3	N	N	N	R	3.2	12	3.2	3.2	2	3	3	2	2.6	2.8	2.6	1.7	2	3.3	2.6	1.3	N	2	1	LCX	CX	RCA	R	R
Suseela	F	1.72	27397	12.5	90	7.2	2.9	5.3	2	2.5	1	2.2	13.2	5.2	Y	N	N	R	4.1	16.9	3.1	2.6	1.3	3	2	2	2.8	2.3	1.2	1.3	3	3.6	2.8	2.1	2	2	1	2PLV,RC	X	RCA	R	R
Nuasiba	F	1.54	27398		134	6.4	2.1	2.9	1	4.2	N	N	13.8	2.5	N	N	N	R	4.2	11	3.1	2.7	1.6	3	2	2	2.4	2.3	1.9	2.3	2	2.9	1.6	1.6	N	N	N	RCA,LC	X	RCA	R	R
Prem Kumari	F	1.62	27402	14.7	108	6.8	2.3	5.5	2	4.4	N	N	14.5	Y	N	N	N	R	3.9	24	2.4	2	2.1	3	2	3	2.4	2.1	1.7	2	2	3.4	3.4	2.6	N	2	2	R,LACX	X	RCA	R	R
Sarasamma	F		27403	11.5	125	4.7	3.3	3.5	2	2.8			17.4	2	Y		Y	R	3.3	16	1.8	1.8	1.3	3	5	3	1.9	1.5	1.4	1.4	2	1.6	2	2.2	N	1	1	X	X	R	2RCA,RVBRAN	R
Krishnamoney B	F	1.65	27527	15.3		5.7	2.4	3.1	2	2.6	1	N	11.2	Y	Y		Y	R	4.9	6.1	3.4	1.8	1.4	3	1	2	3.3	2.4	1.9	2.6	2	3.4	4.3	3.1	N	0	1	R,LACX	LCX	R	2R,R	N
Mary Mathew	F	1.52				4.5	2	1.8	2	2	N	N	18	2.2	Y			L	2.2	8.6	1.6	1.4	1.3	3	4	3	2.1	1.6	1.5	1.2	2	1.6	1.4	1.3	N	2	1	R	X	R	X	R
Leela Bhai	F	1.62	27382	18.1	110	8.2	5.4	5.3	2	2.1	N	N	15	Y	Y	N	N	R	4	5	3.3	3.8	1.8	3	1	2	3	2.6	2.8	1.7	2	3.3	2.7	2	N	1	1	R	X	R	R	R
Savithri	F		27387	7.2		6.7	4.3	4.4	1	2.3	1			Y			?	C	3.3	6.8	3.1	2.7	2.2	3	2	2	3.6	3.9	3.4	2.9	3	2.8	2.8	2	N	2	1	LCX	X	N	R,X	R
Rasheeda	F	1.46	27353	8	61	6.1	3.1	3.3	2	2.5			7.5		N	N	N	R	4.4	16.6	3.8	2	1.8	3	4	4	2.2	1.5	1	1.5	2	1.6	1.4	1.4		1	1	R	X	R	R,RVB	R
Sarada	F		27303				2.5	2.4	1	2.4	1	2.5		Y				L	5.4	21	2.8	2.4	2	3	2	3	2.8	2.8	2.8	2.5	3	3.2	1.7	1.7		1	1	R	X	X	X	X
Sathy amma	F		27310						1		2			Y	1									3	2	3					2					2	2	R,LACX	X	R	R	R
Saramma Daniel	F		27436	11.7		12	2.3	4.6	2	2.5	1	2.5	5					R	4.3	6.9	5.3	3.3	2.5	3	3	3	3.8	3.5	3.5	2.3	3	2.5	2.5	2.3		1	1	X	X	R	R	R
Sudha Raghavan	F		27461		89	8.9	2.4	3	2	2.2	1	2.3	11.3	Y	Y			L		9.7	3.3	2.5	2	3	2	1	2.7	2.2	2.2	2	3	2	2	2		1	1	X	X	X	X	X
Mary aloysius	F		27019			8.5	3.1	2.7	2	2.4	0		12.8					R	3.5	11.5	2.6	2.6	2.4	3	2	3	2.6	2.6	2.6	2.2	2	3.2	3	3	3	2	1	R	X	R	2R	R
Renani	F		27010	8.1	79	6.7	2.6	2.4	1	2.6	1	2.6	7	Y				R	2.9	4.9	2.2	2.3	2	3	2	3	2.9	2.9	2	2.4	3	3.2	3	3		2	1	X	X	R	R	R
Radha	F		27007	7.2		8.3	2.6	2.6		4.9	1	2.4	4.5	Y				L	5.4	12.1	4.4	4	2.5	3	3	3	4	2.9	2.6	2.3	3					2	1	R,X	X	X	R,X	X
Vijayamma	F		26993						2		1				Y	Y	Y	L						3	2	3										1	1	X	X	X	X	X
Knaka rathinam	F		27004							1		1						CD						3	2	2					3					1	BIF	R,X	X,X	R	R	R,X
Remadei	F		26975						1		1													3	2	3					2					1		X	X	R	R	R
Valsamma balan	F	1.75	27346	7.4		4.9	3.5	2.1	2	1.9								R	3.3	17	3.4	1.5	1.5	3	1	2	1.7	1.4		1.9	1	2.8	2.6	2.4		1	1	R	X	R	2R,R	R
Sosamma	F		27345			11.3	3.5	2.9	2	3.3	1							R	5.7	15.1	2.9	2.9	2.7	1	2	2	2.7	2.4	2.2	2	2	2.7	2.3	1.7		1	1	X	X	R	R	R
Devi	F	1.37	27344						2									L						3	2	2					2					1	1	R	X	X	X	X
Dr Cynthia	F	1.45	27342			6.3	2.2	2.8	2	2.7								R	2.8	19.5	2.2	2.2	1.8	3	2	3	1.6	1.4	1.4	1.4		2.6	2.5	2.5		2	2	R	X	R	R	R
Retnamma	F	1.58	27321	16		8.9	1.8	3.2	3	3.9						Large	CS Br	R	3.8	8.7	3.7	1.7	1.2				2.7	1.4	1.4	2.2	2	3.2	2.8	2.8		1	1	R	X	R	R	R
Lissy soman	F	1.55	27297	15		8.4	2	2.3	2	2.7	1	2					Uturn	L	3.1	7.4	2.6	3.1	2.8		2	2	2.8	2	1.8	2	1	1.8	1.8	1.6	1.6	1	1	X	X	X	X	X
Santha	F	1.60	27229	15.1		6	2.8	2.6	1	3.1					Y			R	2.5	6.8	3.2	3.2	3	3	2	2	3.5	3	2.7	3.2	2	2.9	2.8	2	2	1	1	X	X	R	R,ACM	R
Isha Beevi	F	1.76	27276	15.2	84	10.8	2.9	2.8	2	4	1		15.3		Y	CS B		R	4.4	16	3.5	3.2	3	3	2	1	3.1	3	2.4	2.4	1	2.8	2.8	2.6	2.6	1	1	X	X	R	R	R
Lekshmi	F	1.45	27196	10.7	129	6.6	2.3		2	2.4			11.3					R	4.2	9.4	3	2.8	2.8	3	2	2	1.7	1.7	1.5	1.5	3	2.8	2.8	2.6		1	1	X	X	R	R	R
Sarojini amma	F	1.50	27208	14	96	6.4	2.3	3.9	2	2	1	2	8	toCS	Y	OMV	Csbrdi	R	4.1	11.8	1.9	1.8	1.8	3			2.1	2	2	1.8	4	2.7	2.4	2.4	2	1	1	R		R	R	R
Padmavathy K	F		27192			4.5	1.7	3.7	2	5.1	1	1.4		SCVT			Y	R	4.4	12.7	2.3	2	2	3	1	1	2.3	2.3	2.3	2	3	2.3	2.2	2.2	1.3	1	1	R,X	X	R	R	R
Sobha	F	1.27	27154	11.5	93	7.5	2.4	2.6	1	2.2	1		6.5	Y				R	3.2	6.3	1.5	1.4	1.3	3	2	3	1.7	1.4	1.4	1.2	2	2.6	2.6	2.5	2.2	1	1	X	X	R	R	R
Visalakshi	F		27164			6	3.2	2.1	2	3	2		16	DILAT				L	5.7	21.6	3	3	2.8	3	1	1	3.6	4	3.4	2.3	3	1.5	1	1		1	1	R,LACX	X	X	X	X
Manamma	F	1.66	27172	7		6.8	2.3	4.4	1	3.5	1	2.4		Y				CD	5.5	17.1	4.3	4.1	3.8	3	3	3	3.3	2.8	2.6	2.8	2	2.3	2.1	2	2	3	1				R,X	X
Devi	F	1.60	17145	11.5	151	6	3.2	4.4	2	2.3	1					Y	Y	R	4.5	6.3	3.7	3.5	3.5	3	2	2	2.8	2.5	2	2.3	2	3.3	3	2.6	2	1	2	R	X	R	R	R
Rema S Nair	F	1.72	27129	10		6.5	1.7	1.7	3	2.3	1	2.1	10	Y	Y			R	3.4	9.2	2.3	2.3	2	2	1	1	2.8	2	2	2.8	1	3	2.8	2.8	2	1	1	X	X	R	RR	R
Stella	F	1.66	27127	14.7		6.1	2.4	2.6	1	2.8	1	3	8.9	2 TO I				R	4.4	10.4	3.6	3.4	3	3	3	2	2	1.8	1.8	1.6	2	3.6	3.6	3.2	3.2	2	1	LACX	X	R	R	R
Saraswathy	F	1.62	27130	13.2	87	8.6	1.9	3.6	2	1.8			13	Y	CS B			R	4.8	7.5	3.8	3.2	3		3	2	2.3	2	2	1.6	2	3	2.6	2.6	2	1	1	R	X	R	R	R
Subhadra	F		27050	13.1		10.9/2	2.8	5.2	2	3.1	2	2.6	10					CD	2.8	8	2.7	2.6	2.2	3	2	2	3.5	3.2	3	3.5	2					1	1	R	X	X	X	?R
Radhamony	F	1.70	27051	9		6/1.8	2.3	3.5		2.4		2.4	5.6					R	2.9	10.4	1.8	1.7	1.7	3	3	2	2.2	2.2	2	2.2	3	3.4	3.2	2.3	2.3	3	1	R	X	R	R	R
Ammini	F		27445			6.7/2.5	2.3	2.3	1	3.8	1	2.8	14					L	4.8	13.8	1.8	1.7	1.6		2	1	2.5	2.5	2.3	2.5	3							X	X	X	X	X
Valsmma	F	1.62	27516	13.5		6.3/2	2.6	2.5	2	2.4	1	2.2	9.8				Y	R	5.3	5.6	3.6	3.6	2	3	2	3	3	2.5	2.5	1.8	1	2.6	2.6	2.2	2	1	1	LACX	X	R	R	R
Manamma	F	1.52	27558	11		9/2.3	2.3	2.5	1	2.6	1	2.5			Y			R	3	5.2	2	2	1.9	3	2	2	2.1	2.1	2	2	2	2	2	2	1.8	3	1	R,X	X	R	R	R
Sasi kumar	F	1.85	27475	15.5	99	13.7	3.2	3.5	1	3.5	1	3.2			Y			R	4	9.5	3.9	3.9	2		3	3	4.5	4.5	2.2	4.3	2	2.3	2.2	2.2	2.2	3	1	LACX	X	R	R	R
Francis P	F	1.78	27396	14.8		5	3.3	2.8	2	2.1	0	N		3	N	N	N	R	3.2	12	3.2	3.2	2	3	3	2	2.6	2.8	2.6	1.7	2	3.3	2.6	1.3	N	2	1	LCX	CX	RCA	R	R
Vasumathy	F	1.72	27397	12.5	90	7.2	2.9	5.3	2	2.5	1	2.2	13.2	5.2	Y	N	N	R	4.1	16.9	3.1	2.6	1.3	3	2	2	2.8	2.3	1.2	1.3	3	3.6	2.8	2.1	2	2	1	2PLV,RC	X	RCA	R	R
Sathi	F	1.54	27398		134	6.4	2.1	2.9	1	4.2	N	N	13.8	2.5	N	N	N	R	4.2	11	3.1	2.7	1.6	3	2	2	2.4	2.3	1.9	2.3	2	2.9	1.6	1.6	N	N	N	RCA,LC	X	RCA	R	R
Leela L	F	1.82	27402	14.7	108	6.8	2.3	5.5	2	4.4	N	N	14.5	Y	N	N	N	R	3.9	24	2.4	2	2.1	3	2	3	2.4	2.1	1.7	2	2	3.4	3.4	2.6	N	2	2	R,LACX	X	RCA	R	R
Ambika	F		27403	11.5	125	4.7	3.3	3.5	2	2.8			17.4	2	Y		Y	R	3.3	16	1.8	1.8	1.3	3	5	3	1.9	1.5	1.4	1.4	2	1.6	2	2.2	N	1	1	X	X	R	2RCA,RVBRAN	R
Mohammed M	F	1.65	27527	15.3		5.7	2.4	3.1	2	2.6	1	N	11.2	Y	Y		Y	R	4.9	6.1	3.4	1.8	1.4	3	1	2	3.3	2.4	1.9	2.6	2	3.4	4.3	3.1	N	0	1	R,LACX	LCX	R	2R,R	N
Narayanan V	F	1.27	27356			4.5	2	1.8	2	2	N	N	18	2.2	Y			L	2.2	8.6	1.6	1.4	1.3	3	4	3	2.1	1.6	1.5	1.2	2	1.6	1.4	1.3	N	2	1	R	X	R	X	R
Harikumar	F		27050	13.1		10.9/2	2.8	5.2	2	3.1	2	2.6	10					CD	2.8	8	2.7	2.6	2.2	3	2	2	3.5	3.2	3	3.5	2					1	1	R	X	X	X	?R
Thankappan S	F	1.70	27051	9		6/1.8	2.3	3.5		2.4		2.4	5.6					R	2.9	10.4	1.8	1.7	1.7	3	3	2	2.2	2.2	2	2.2	3	3.4	3.2	2.3	2.3	3	1	R	X	R	R	R
Mohan Kumar	F	1.82	27362	18.1	110	8.2	5.4	5.3	2	2.1	N	N	15	Y	Y	N	N	R	4	5	3.3	3.8	1.8	3	1	2	3	2.6	2.8	1.7	2	3.3	2.7	2	N	1	1	R	X	R	R	R
Arifa Beevi	F		27387	7.2		6.7	4.3	4.4	1	2.3	1			Y			?	C	3.3	6.8	3.1	2.7	2.2	3	2	2	3.6	3.9	3.4	2.9	3	2.8	2.8	2	N	2	1	LCX	X	N	R,X	R
Vijayan	F	1.46	27353	8	61	6.1	3.1	3.3	2	2.5			7.5		N	N	N	R	4.4	16.6	3.8	2	1.8	3	4	4	2.2	1.5	1	1.5	2	1.6	1.4	1.4		1	1	R	X	R	R,RVB	R
Girija	F		27303				2.5	2.4	1	2.4	1	2.5		Y				L	5.4	21	2.8	2.4	2	3	2	3	2.8	2.8	2.8	2.5	3	3.2	1.7	1.7		1	1	R	X	X	X	X
Sheels	F		27310						1		2			Y	1									3	2	3					2					2	2	R,LACX	X	R	R	R
Kochumma Nair	F		27436	11.7		12	2.3	4.6	2	2.5	1	2.5	5					R	4.3	6.9	5.3	3.3	2.5	3	3	3	3.8	3.5	3.5	2.3	3	2.5	2.5	2.3		1	1	X	X	R	R	R
Appukuttan	F		27461		89	8.9	2.4	3	2	2.2	1	2.3	11.3	Y	Y			L		9.7	3.3	2.5	2	3	2	1	2.7	2.2	2.2	2	3	2	2	2		1	1	X	X	X	X	X
Ranganatha Pillai	F		27019			8.5	3.1	2.7	2	2.4	0		12.8					R	3.5	11.5	2.6	2.6	2.4	3	2	3	2.6	2.6	2.6	2.2	2	3.2	3	3	3	2	1	R	X	R	2R	R
BalakrishnanT	F	1.60	17145	11.5	151	6	3.2	4.4	2	2.3	1					Y	Y	R	4.5	6.3	3.7	3.5	3.5	3	2	2	2.8	2.5	2	2.3	2	3.3	3	2.6	2	1	2	R	X	R	R	R
Omana K	F	1.72	27129	10		6.5	1.7	1.7	3	2.3	1	2.1	10	Y	Y			R	3.4	9.2	2.3	2.3	2	2	1	1	2.8	2	2	2.8	1	3	2.8	2.8	2	1	1	X	X	R	RR	R
Rosamma B	F	1.66	27127	14.7		6.1	2.4	2.6	1	2.8	1	3	8.9	2 TO I				R	4.4	10.4	3.6	3.4	3	3	3	2	2	1.8	1.8	1.6	2	3.6	3.6	3.2	3.2	2	1	LACX	X	R	R	R
Vallikutty	F	1.62	27130	13.2	87	8.6	1.9	3.6	2	1.8			13	Y	CS B			R	4.8	7.5	3.8	3.2	3		3	2	2.3	2	2	1.6	2	3	2.6	2.6	2	1	1	R	X	R	R	R
Mohanan S	F		27010	8.1	79	6.7	2.6	2.4	1	2.6	1	2.6	7	Y				R	2.9	4.9	2.2	2.3	2	3	2	3	2.9	2.9	2	2.4	3	3.2	3	3		2	1	X	X	R	R	R

Shahul Hameed	F		27007	7.2		8.3	2.6	2.6		4.9	1	2.4	4.5	Y				L	5.4	12.1	4.4	4	2.5	3	3	3	4	2.9	2.6	2.3	3					2	1	R,X	X	X	R,X	X
Meenakshi Mani	F		26993						2		1				Y	Y	Y	L						3	2	3					2					1	1	X	X	X	X	X
Babu Rajan	F		27004							1		1						CD						3	2	2					3					1	BIF	R,X	X,X	R	R	R,X
Rajamma joseph	F		27192			4.5	1.7	3.7	2	5.1	1	1.4		SCV T			Y	R	4.4	12.7	2.3	2	2	3	1	1	2.3	2.3	2.3	2	3	2.3	2.2	2.2	1.3	1	1	R,X	X	R	R	R
Ismailkunju	F	1.27	27154	11.5	93	7.5	2.4	2.6	1	2.2	1		6.5	Y				R	3.2	6.3	1.5	1.4	1.3	3	2	3	1.7	1.4	1.4	1.2	2	2.6	2.6	2.5	2.2	1	1	X	X	R	R	R
Prathapachandran	F		27164			6	3.2	2.1	2	3	2		16	DILAT				L	5.7	21.6	3	3	2.8	3	1	1	3.6	4	3.4	2.3	3	1.5	1	1		1	1	R,LACX	X	X	X	X
Bala Krishnan	F	1.66	27172	7		6.8	2.3	4.4	1	3.5	1	2.4		Y				CD	5.5	17.1	4.3	4.1	3.8	3	3	3	3.3	2.8	2.6	2.8	2	2.3	2.1	2	2	3	1				R,X	X
Remadei	F		26975						1		1													3	2	3					2					1		X	X	R	R	R
ISMAIL	F	1.75	28347	7.4		4.9	3.5	2.1	2	1.9								R	3.3	17	3.4	1.5	1.5	3	1	2	1.7	1.4	SAMLL	1.9	1	2.8	2.6	2.4		1	1	R	X	R	2R,R	R
KANAKAMMA	F		28408			11.3	3.5	2.9	2	3.3	1							R	5.7	15.1	2.9	2.9	2.7	1	2	2	2.7	2.4	2.2	2	2	2.7	2.3	1.7		1	1	X	X	R	R	R
JEYABALAN	F	1.37	28407						2									L						3	2	2					2					1	1	R	X	X	X	X
MAHARAJAN	F	1.45	28350			6.3	2.2	2.8	2	2.7								R	2.8	19.5	2.2	2.2	1.8	3	2	3	1.6	1.4	1.4	1.4		2.6	2.5	2.5		2	2	R	X	R	R	R
VAVACHAN	F	1.58	28676	16		8.9	1.8	3.2	3	3.9						Large	CS Br	R	3.8	8.7	3.7	1.7	1.2				2.7	1.4	1.4	2.2	2	3.2	2.8	2.8		1	1	R	X	R	R	R
ANANDAVALLY	F	1.76	28529	15.2	84	10.8	2.9	2.8	2	4	1		15.3		Y	CS B		R	4.4	16	3.5	3.2	3	3	2	1	3.1	3	2.4	2.4	1	2.8	2.8	2.6	2.6	1	1	X	X	R	R	R
SRIKUMAR	F	1.45	28565	10.7	129	6.6	2.3		2	2.4			11.3					R	4.2	9.4	3	2.8	2.8	3	2	2	1.7	1.7	1.5	1.5	3	2.8	2.8	2.6		1	1	X	X	R	R	R
BABY	F	1.50	28675	14	96	6.4	2.3	3.9	2	2	1	2	8	IoCS	Y	OMV	Csbrdi	R	4.1	11.8	1.9	1.8	1.8	3			2.1	2	2	1.8	4	2.7	2.4	2.4	2	1	1	R		R	R	R
ARUNAGIRI	F	1.55	28616	15		8.4	2	2.3	2	2.7	1	2					Utum	L	3.1	7.4	2.6	3.1	2.8		2	2	2.8	2	1.8	2	1	1.8	1.8	1.6	1.6	1	1	X	X	X	X	X
NABEESA BEEVI	F	1.60	28585	15.1		6	2.8	2.6	1	3.1					Y			R	2.5	6.8	3.2	3.2	3	3	2	2	3.5	3	2.7	3.2	2	2.9	2.8	2	2	1	1	X	X	R	R,ACM	R
SAROJINI L	F		28704			6.7/2.5	2.3	2.3	1	3.8	1	2.8	14					L	4.8	13.8	1.8	1.7	1.6		2	1	2.5	2.5	2.3	2.5	3							X	X	X	X	X
RAMESH g	F	1.62	28706	13.5		63/2	2.6	2.5	2	2.4	1	2.2	9.8				Y	R	5.3	5.6	3.6	3.6	2	3	2	3	3	2.5	2.5	1.8	1	2.6	2.6	2.2	2	1	1	LACX	X	R	R	R
RAVINKUTTY	F	1.52	28477	11		9/2.3	2.3	2.5	1	2.6	1	2.5			Y			R	3	5.2	2	2	1.9	3	2	2	2.1	2.1	2	2	2	2	2	2	1.8	3	1	R,X	X	R	R	R
SAIDU MOHAMM	F	1.85	28392	15.5	99	13.7	3.2	3.5	1	3.5	1	3.2			Y			R	4	9.5	3.9	3.9	2		3	3	4.5	4.5	2.2	4.3	2	2.3	2.2	2.2	2.2	3	1	LACX	X	R	R	R
J. Menon	m		27008		125	10.1	3.7	2.6	1	2.7	1	1.9	12.9					R	4.9	11.5	2.3	2.5	2.3	3	3	2	3	2	2.6	2.4	4	3	2.6	2.6		2	1	R	X	R	R	R
Babu	m		26990		126				1		1							R						3	3	4					3					1	1	R	X	R	R	R
Komal Kumar	m		27053			6	2.6	2.5	2	2.7	2	2.7	7		y			R	3.7	11.3	3.1	3	2.5	3	2	3	2.4	2.3	2.2	2.2	3	3	2.8	2.8		1	1	R	X	R	R	R
Thankappan	m		27008		125	10.1	3.7	2.6	1	2.7	1	1.9	12.9					R	4.9	11.5	2.3	2.5	2.3	3	3	2	3	2	2.6	2.4	4	3	2.6	2.6		2	1	R	X	R	R	R
Aboobacker	M	1.82	27400	15.5	148	6	3.9	3.6	2	2.4	0	N	22.7	3.1	N	N	N	R	3.6	8.1	3.4	2.7	1.8	3	3	2	2.6	2.8	2.4	3	2	4.9	4.4	3.9	3	2	1	RCA	X	RCA	2R	R
PANKAJAKSHAN	M	1.30	27357	16.2	163	3.5	2.3	2.9	2	2.4	N	N	11.9	Y				R	4.3	16.9	3.3	2.8	2.3		2	4	1.8	1.6	1.3	1.9	1	1.5	1.5	1.4	N	2	N	LACX	2XX	L	R	R
Raju	M	1.48	27361			5.7	3.6	3.3	2	3.1	1	N	13.3	Y	Y	N	N	R	2.6	6.8	2.2	2	1.7	3	3	3	1.3	1.4	1	0.9	2	1.9	2.7	1.5	1.4	1	1	R	X	R	2R	2R
Muraleedharan	M	1.62	27365	20	178	5.1	1.5	2.1	2	1.7	N	N	6	Y	N	N	N	R	3.4	5.9	2.2	1.3	0.8	3	2	3	2.1	1.4	1.4	1	4	3	1.5	1.4	1.7	1	1	R,LACX	X	R	R	R
Gopalan	M	1.55	27376						2									R	3.4	9.2	2.2	1.7	1.8	3	3	3	2.2	1.6	0.8	2.1	1	2.4	2.1	2.2	N	1	1	LCX	X	R	R	R
Jacob	M	1.96	27382	13.6		11.1	3.7	4.3	1	3.2	1	N	6.5	6.5	N	N	N	R	3.5	4.7	3.8	3.8	2.1	3	2	2	4.6	4.8	2.2	3.5	1	3.7	4	4	N	2	1	LACX	X	R	R	R
Vasudea	M		27395			6.6	2.4	2.4	1	2.3	1	2.2						L	4.2	6.7	3	2.6	2.2	3	3	3	2.8	2.8	2.6	2.4	3	2.3	2.3	2		1	1	R	X	X	X	X
Jugal Singh	M		27460			6	3.2	2.9	2	2.8	1		7.1	Y				R	3.5	2.6	2.8	2.6	2	3	3	4	2.8	2.8	2.6	2	4	3.8	3.6	2.5	2.5	2	1	R	X	R	R	R
Balachandran	M		27470	14	159	7	2.3	2.2	2	2	1	2	7.8		Y			CD	4.5	9.5	3.8	2.4	2.5	3	4	3	4.1	4	2	3.2	3	2.8	2.6	2		1	1	R	X	R	R	X
Shamsudden	M		27535	10.9	98	8.2	2	3.6	1	2.2	1	2.2	6.9	Y				R	5	15.1	3.9	3.2	3.2	3	2	2	2.9	2.9	2.9	2.5	2	3.2	3.2.	3	2.4	1	1	R,X	X	R	R	R
Purushothman	M		27016		133	9	3	3.2	1	2.9	1	2.1	13					R	3.5	3.5	3.5	3.4	3.3	3	2	4	3.2	3	2.6	2.4	3	4.3	4	3.6		2	1	R	X	R	R	R
Mohd Shafi	M		27017		89	8	2.4	2.4	1	2.6	1	2.5	14					R	4.3	5.8	3.5	3.5	2.5	3	2	3	2.4	2.4	2.4	2	3	4	3.6	3.6	3.2	2	1	LACX	X	R	R	R
Rajan	M		27038	8	72	4.6	2	3.4	1	2	1	2						R	3	7	1.7	1.7	1.5	3	3	2	1.7	1.7	1.3		3	2.3	2.3	2				R	X	RR	R	R
Sahadevan	M		27026	17	96				1		1							R						3	2	2					3					2	1	X	X	R	R	R
Damodaran	M		26991						1		1							CD						3	3	3					2					1	1	R	X	R	R	R,X
Abdul LATHEEF	M		26979						1		1							R						3	3	2					2					1	1	R,X	X	R	R	R
Ramachandran	M		26986						1		1							R						2	1	2					1					2	1	LACX	X	R	R	R
Ouseph	M		26808															R						3	2	2					1							Distal R,L	X	R	R	R
Thomas	M		26977							1	1							R						3	3	2					3							R,X		R	R	R
Abdul Hakem	M	1.73	27326			7	2.6	2.3	3	3								R	4.2	13.6	2.7	2.5	2.5	3	3	2	2.6	2.6	Small			3.3	3.9	3.3		3	1	X	X	R	R	R
Dr Chellappan	M	1.58	27319	7		8.4	2.9	4	1	3.3								R	4.2	16.4	3.2	3	3	3	3	5	2.5	1	1	1.9	3	3.3	3.3	2.6		1	1	LACX	X	R	R	R
Vallikkannu	M	1.50	27323	15.2	108	7.8	2.2	3.8	3	2.5								L	3.6	6.5	3.1	2	2	2	2	1	3.5	3.2	2.9	2.4	2	2	1.8	1.6		1	1	R	X	X	X	X
Ramachandran pill	M	1.58	27317	25.7					2									R	4.9	11.5	4.2	3	2		2	2	3	2.8	2	1.3	2	3.1	2.1	2.5		1	1	R	X	R	R	R
Mathai	M		27312			9.2	3.5	3.6		3.6			13					R	5.5	14.1	4	2.2	2.1	3	2	1	2.3	0.9	0.9	1.6	1	3	2.6	2.6	2.6	1			X	R	R	R
Jose	M		27314						1									R	4.4	7.7	2.7	2.8	2	3	2	2	3.9	2.4	1.8	1.8	3	5.7	2.9	3.1	2	1	1BIF	X	X	R	R	R
Sushena Kumar	M	1.70	27308	11.8	94	8.6	3	4	1	3			12	3.7		Bifurc	I	R	5.7	17.1	4.6	2.2	1.7	3	2	1	4.1	3.2	2.5	2.3	2	4.1	1.8	1.6		1	1	R,X	X	R	R	R
Gopala	M	1.69	27291	14	127	6.5	2.9	4.5	2	3	1		11.6	3.1	OMV			L	3.8	6.4	2.5	1.7	1.7	3			2.3	1.9	1.4	1.4	3	2.1	2.3	2	2	3	1				R,X	X
Sundaresan	M	1.65	27289	19.3		7.4	3.3	2.8	2	3.6			12.7	Y		CS B		R	3	6.7	2.4	2	2	3	2	2	2.5	2.4	2	2.2	2	3.3	3.2	3.3	2.6	2	1	X	X	R	R	R
Sundarwesn	M		27267						1									L	2.7	11.1	2.6	2.4	2	2	2	2	2.3	2.,3	2.2	1.9	3	2.3	2.3	2	2	1	1	R	X	X	X	X
Janardhana	M		27214						1									R	4.2	8.1	3.2	3.2	3		2	2	3.4	3.2	3.2	3.2	2	2	2	1.6	1.4	1	1	R			R	R
Prabhakaran	M		27217						2					Y	Y	CSBr																										
Babu	M	1.92	27233			10.2	4.6	3.8	2									R	3.8	11.4	3.4	3.4	3.2	3	2	2	2.1	2	2	2	1	3.4	3.4	3.2		1	1	X	X	R	R	R
Mohd Hussain	M	1.83	27205	14		6.7	1.6	3.4	2	3.5			11		Small			CD	4.7	17.8	2.2	2	2	3	2	2	3.5	3	2.8	2.5	3	2.8	2.6	2.6		1	1	X	X	R	RX	X
kunjumohd	M	1.44	27183			8	2.7	5.3	1	3.4	1	1.8	11.7		Y		Y	L	2.9	3	1.9	1.8	1.5	3	2	2	1.9	1.8	1.1	1.1	1								X	X	X	X
Vikraman T	M	1.80	27189		121	7	2.5	5.1	2	5	1						Uluret	R	[illegible]	22.1	3.2	3	2.7		2	2	2.3	2	2	2	3	3.9	3.9	3.7		1	1	LACX	X	R	R	R
Mohanaa	M		27191	11	103	12	3.8	3	1	3.2			20				CS Br	R	[illegible]	[illegible]	3.4	3	2.6	1	2	2	3.9	3.4	3.2	2.5	3	3.2	3	3	2.5	2	1	R	R	R	R	
Madahavan	M	1.70	27194	10.2	104	8	3	4.7	2	3.6			10.7				CS bri	R	[illegible]	[illegible]	2.9	2.5	2.3	3	1	3	2.8	2.4	2.4	2.4	2	1.9	1.8	1.7	1.8	1	1	R	X	PLB	R,ACM FROM I	X
Vigneswara pillai	M	1.52	27152			7	2.5	2.3	1	2.6	1	2	7	OMV				R	5.7	9.7	2.9	2.7	2.7		2	2	3.1	2.7	2.7	2	3	2.5	2.5	2.4	2.4	1	1	R	X	R	R	R
Sundaram	M	1.65	27163	15		8.1	3.1	3.5	2	4.9	1	2.3	7	Y	Y	UTUF		R	4.2	13.3	2.7	2.6	2.6	3	3	2	2.6	2.5	2.2	2.6	2	3	2.8	2.6	2.6	1	2	R			R	
Salim Ibrahim	M		27140			12.5	2	2.1	2	3.3			9.5					R	4.8	25	3	2.5	2.2	3	3	2	3.6	3.5	3.3	2.2	4	3.2	3.5	3.2	1.3	1	1	R	X	R	R	R
Perumal	M	1.62	27141			8.1	3.9	4.5	3	3.1	1	2.8	12				y	R	3.6	11.4	3.7	3.4	3.2	3	2	2	4.4	4	4	2	3	2.2	2.2	2	2	1	1	R	X	R	R	R
Mohanan	M	1.85	27150	12.8	116	6	2.8	3.1	2	2.4	1	3.3	8.6	TO RA	Y	CSBF		L	2.6	5.2	2.4	2.3	2.2	3	2	2	2.1	2	2	2		1.4	1.4	1.2	1.2	2	1	R	X	X	X	X
Neelakatan pillai	M	1.50	27151			10.1	4	5.5	1	4.1			11.2	Y				CD	5.3	14.9	2.4	1.5	1.5	3	2	3	3.3	2	2	2.7	2	3.8	3.8	3.6	3.4	1	1	R,X	X	R	R,X	R

Bobby Mlhew	M	1.76	27137	14.5	101	7.9	3.3	5.5	2	5			11.3	Y	Y	CS B		CD	4.2	5.3	3.3	3.2	3	3	2	3	2.6	2.6	2.4	2	3	2.8	2.2	2	2	1	2	X	X	R	R	X
Sulaiman	M	1.76	27107	13		5.2	2.2	2.3	1	2.8	1	2.2	12.5		Y			CD	3.4	21.2	1.8	1.8	1.6	3	3	3	2.7	2.1	2	2.7	3	3.1	3	2.6		2	1	R	HIL	R	R	R,X
Reghu TK	M		27109	13.7		5.6	3.2	1.9	1		1							CD	3.3	17.5	2.6	2.4	2.4	3	3	2	2.4	2.2	1.5	2	3	2.5	2.8	2.5	2	1	1	X	X	R	R,X	R,X
Muraleedharakuru	M	1.80	27118	15.6	86	9	3.8	3.3	2	2.4	1	2.3	7.9	Y	Y	CSBR		CD	3.7	10	3	2.6	2.6	3	2	2	2.8	2.6	2.4	2.4		2.1	2	1.8	1.5			X	X	X	R	X
Sankaran NAIR	M	1.65	27112	13.6	143	5.1	2.2	2.3	2	2.7	1	2.5	9.5					CD	2.8	8.8	2	2	1.8	3	3	2	2.6	2.6	2.6	2.,5		2.7	2.5	2	1.6	3	1	R	X	R	R	X
MlahewPJ	M		27121						1									CD	4.1	13.7	3.1	3	2.8	3	3	3	3.3	2.8	2.6	2.8	2	2.3	2.3	2.5		1	1	R,X	X	R	R	X
Jnardhanan	M	1.46	27690	10.8		6.7	2.4	2.5	1	3.3	1	3.2	4.8	Y	Y			R	4.4	18.9	3.5	3.2	3	3			2.1	1.8	1.6	1.8		3.8	3.8	3.2		2	1	R	X	R	R	R
Mohanan	M	1.85	27097	13	157	8.5	2.6	3.8	1	1.9	2	2.3	13.4	Y				R	4.8	10.6	2.5	2	2	3	2	3	4.3	3.5	3.5	3.8	2	4.9	3.8	1.9	1.9	2	1	R	X	R	R	R
Remanan	M		27103	14.3		9.1	3	4	3	2.2	2	2.6	16				Y	R	4	10.4	4.1	3.8	3.6	3	3	4	3.2	2	2	3		4	3.6	2.5	2.4	2	1	R	X	R	R	R
Shanmugn	M		27106						1						1			R	3	1.3	3.2	3	3	3	2	3	3.2	1.6	1.6	1.6	2					1	1	R X	X	R	R .	R
Sulaiman	M	1.76	27107	13		5	3.9	2.5	2	2.7	2	3.1	13.3	Y	Y			R	3.3	20.5	2	2	1.8	3	3	2	2.5	2	2	2.8	2	2.8	2.8	2.5		3	1	R	X	R	R	R
Ajithmuar	M	1.66	27087	12		6.8	2.7	4.1	2	2.2	1	2.2	13					R	4.4	13.5	3	2.8	2.6	3			3.6	3.5	3.2	3	2	4.8	4.7	4	3.5	3	1	R X	X	R	R	R
Rajan	M		27053			6	2.6	2.5	2	2.7	2	2.7	7		y			R	3.7	11.3	3.1	3	2.5	3	2	3	2.4	2.3	2.2	2.2	3	3	2.8	2.8		1	1	R	X	R	R	R
LUKAS	M	1.77	27061	16		10.9	2.5	2.7	2	2.8	1	3.6		Y				L	2.7	5.2	2.3	2.3	2	3	3	3	4.2	3.5	3.2	2.5	2	1.7	1.5	1.5	2.5	1	1	R	X	X	X	X
AbdulRasheed	M	1.84	27084	17		6.5	1.8	2.7	2	3	1	2.9	10.1					R	3.6	4	4.2	3.8	3.6	3	2	1	4.2	4.1	4	4	2	3.5	2.7	1.4	1.9	2	1	R X	X	R	R	R
Balasubramainapill	M	2.15	27065			7.1	2	3.4	2	2.8	1	2.4	11.2	Y				R	5.8	10.7	3.4	3.3	3.3	3	3	3	3.3	3.2	3	2.5	3	2.8	2.6	2.6	2.3	1	1	X	X	R	R R	R
Murukan	M	1.92	27089			7.5/2.8	3.3	2.9	3	2.7	1	2.5	8.5		Y			L	3	8.3	2.6	2.6	2.5	3	2	1	2.7	2.5	2.5	2.5	2	1.8	1.6	1.6		1	1	X	X	X	X	
Ashraf	M	1.45	21789	10.9	98	8.2/3	2	3.6	1	2.2	1	2.2	6.9					R	5.1	15.1	3.9	3.2	3.2	3	2	2	2.9	2.9	2.9	2.5	2	3.2	3.2	3	2.4	1	1	R,X	X	R	R	R
Radhakrishnan	M		27041	14.5	125	8.2/3.5	3.5	4.2	2	2.9	1	1.6	12.5		Y		Y	R	4.6	9	3.4	3	2.5	3	2	3	3.9	1.5	1.5	3.5	3	2.2	2	2		1	1	R,LACX	X	R	R	R
Anirudhan	M	1.68	27044	15.6	64	6.3	3	2.3	2	2.5	1	2.8	13					CD	4.8	10.6	4	4	3	3	2	3	4	4	4	2.4	4	2.8	2.3	2		2			X		R,X	X
Appukuutan nair	M	1.92	27049		106	10.2/3	2.2	2.2	2	2.2	1	1.6	11					CD	3.8	9.8	2.1	2	1.6	3	2	3	2.6	2.6	2.2	1.5	1	2.8	2.5	2.5		2	1	X	X	R	R	X,R
Rajamohanna	M		27529	10.7	133	11.1/3	2L7	6.1	1	3.3	1	2.8	10		Y			R	5	16.5	3	2.5	2.3	3	2	3	3.3	3	2.5	2.5	2	3.5	3	3	2	2	1	X	X	R	R	R
Padmakumar	M	1.90	27526	14.2	170	9//3	2.5	3.4	1	4.4	1	3.9	14					L	4.2	4.5	3.4	3.2	3	3	3	3	4.1	4	4	3.2	3	2.5				2	0	R	X	X	X	X
Premkumar	M		27552			8	2.5	2.5	1	2.5	1	2.3						R	4.2	9.8	2.9	2.5	2.4	2	1	2	3.2	3.1	2	3.1	1	2.4	2.4	2		1	1	LACX	X	R	R	R
Aliyar	M	1.56	27477			9	4.2	4	1	4	1	4.2	18.2		Y			L	4.8	13.5	2.4	2	2	3	2	3	4.1	3	2	1.6		4.6	3.6	3	2.5	2	1	R	X	R	R	R
Govinda	M	1.60	27483	14.4	150	7.5	1.8	4.1	1	2.1	2	2.5	14		Y			R	3	6.3	3.2	2	2	3	3	2	3.1	2	2	1.7	2	2.3	2.5	2.3	2.3	1	1	X	X	R	R	R
Abdul	M	1.65	27405	12.6	133	9.9/5.8	3	2.4	2	3.1	1	2.5	17.9	DOUB				L	4.6	12	3.1	2.6	2.2	3	2	2	3.3	3.3	2	3.2	2	2.8	2.6	2.5	2.2	1	1	R	X	R	R	R
Peter Koshy	M	1.82	27400	15.5	148	6	3.9	3.6	2	2.4	0	N	22.7	3.1	N	N	N	R	3.6	8.1	3.4	2.7	1.8	3	3	2	2.6	2.8	2.4	3	2	4.9	4.4	3.9	3	2	1	RCA	X	RCA	2R	R
Bhaskaran N	M	1.30	27357	16.2	163	3.5	2.3	2.9	2	2.4	N	N	11.9	Y				R	4.3	16.9	3.3	2.8	2.3		2	4	1.8	1.6	1.3	1.9	1	1.5	1.5	1.4	N	2	N	LACX	2XX	L	R	R
Lekshmikutty K	M	1.48	27361			5.7	3.6	3.3	2	3.1	1	N	13.3	Y	Y	N	N	R	2.6	6.8	2.2	2	1.7	3	3	3	1.3	1.4	1	0.9	2	1.9	2.7	1.5	1.4	1	1	R	X	R	2R	2R
Vijayan Varghese	M	1.77	27061	16		10.9	2.5	2.7	2	2.8	1	3.6		Y				L	2.7	5.2	2.3	2.3	2	3	3	3	4.2	3.5	3.2	2.5	2	1.7	1.5	1.5	2.5	1	1	R	X	X	X	X
Ravindran K	M	1.84	27084	17		6.5	1.8	2.7	2	3	1	2.9	10.1					R	3.6	4	4.2	3.8	3.6	3	2	1	4.2	4.1	4	4	2	3.5	2.7	1.4	1.9	2	1	R X	X	R	R	R
Raghabhan M	M	2.15	27065			7.1	2	3.4	2	2.8	1	2.4	11.2	Y				R	5.8	10.7	3.4	3.3	3.3	3	3	3	3.3	3.2	3	2.5	3	2.8	2.6	2.6	2.3	1	1	X	X	R	R R	R
Unnamalai Amma	M	1.92	27089			7.5/2.8	3.3	2.9	3	2.7	1	2.5	8.5		Y			L	3	8.3	2.6	2.6	2.5	3	2	1	2.7	2.5	2.5	2.5	2	1.8	1.6	1.6		1	1	X	X	X	X	
Kochumani R	M	1.45	27535	10.9	98	8.2/3	2	3.6	1	2.2	1	2.2	6.9					R	5.1	15.1	3.9	3.2	3.2	3	2	2	2.9	2.9	2.9	2.5	2	3.2	3.2	3	2.4	1	1	R,X	X	R	R	R
Sasindran K	M		27041	14.5	125	8.2/3.5	3.5	4.2	2	2.9	1	1.6	12.5		Y		Y	R	4.6	9	3.4	3	2.5	3	2	3	3.9	1.5	1.5	3.5	3	2.2	2	2		1	1	R,LACX	X	R	R	R
Pareedumma	M	1.68	27044	15.6	64	6.3	3	2.3	2	2.5	1	2.8	13					CD	4.8	10.6	4	4	3	3	2	3	4	4	4	2.4	4	2.8	2.3	2		2			X		R,X	X
Somasekharan Ma	M	1.92	27049		106	10.2/3	2.2	2.2	2	2.2	1	1.6	11					CD	3.8	9.8	2.1	2	1.6	3	2	3	2.6	2.6	2.2	1.5	1	2.8	2.5	2.5		2	1	X	X	R	R	X,R
Sainaba S	M	1.62	27365	20	178	5.1	1.5	2.1	2	1.7	N	N	6	Y	N	N	N	R	3.4	5.9	2.2	1.3	0.8	3	2	3	2.1	1.4	1.4	1	4	3	1.5	1.4	1.7	1	1	R,LACX	X	R	R	R
Paramanan	M	1.55	27376						2									R	3.4	9.2	2.2	1.7	1.8	3	3	3	2.2	1.6	0.8	2.1	1	2.4	2.1	2.2	N	1	1	LCX	X	R	R	R
Joseph K U	M	1.96	27382	13.6		11.1	3.7	4.3	1	3.2	1	N	6.5	6.5	N	N	N	R	3.5	4.7	3.8	3.8	2.1	3	2	2	4.6	4.8	2.2	3.5	1	3.7	4	4	N	2	1	LACX	X	R	R	R
Mariam Beevi	M		27395			6.6	2.4	2.4	1	2.3	1	2.2						L	4.2	6.7	3	2.6	2.2	3	3	3	2.8	2.8	2.6	2.4	3	2.3	2.3	2		1	1	R	X	X	X	X
Sivakami	M	1.80	27118	15.6	86	9	3.8	3.3	2	2.4	1	2.3	7.9	Y	Y	CSBR		CD	3.7	10	3	2.6	2.6	3	2	2	2.8	2.6	2.4	2.4		2.1	2	1.8	1.5			X	X	X	R	X
Sukumaran Nair	M	1.65	27112	13.6	143	5.1	2.2	2.3	2	2.7	1	2.5	9.5					CD	2.8	8.8	2	2	1.8	3	3	2	2.6	2.6	2.6	2.,5		2.7	2.5	2	1.6	3	1	R	X	R	R	X
Suhara	M		27121						1									CD	4.1	13.7	3.1	3	2.8	3	3	3	3.3	2.8	2.6	2.8	2	2.3	2.3	2.5		1	1	R,X	X	R	R	X
Sivadesan	M	1.46	27690	10.8		6.7	2.4	2.5	1	3.3	1	3.2	4.8	Y	Y			R	4.4	18.9	3.5	3.2	3	3			2.1	1.8	1.6	1.8		3.8	3.8	3.2		2	1	R	X	R	R	R
Yesudasan	M	1.85	27097	13	157	8.5	2.6	3.8	1	1.9	2	2.3	13.4	Y				R	4.8	10.6	2.5	2	2	3	2	3	4.3	3.5	3.5	3.8	2	4.9	3.8	1.9	1.9	2	1	R	X	R	R	R
Devaki Amma	M		27103	14.3		9.1	3	4	3	2.2	2	2.6	16				Y	R	4	10.4	4.1	3.8	3.6	3	3	4	3.2	2	2	3		4	3.6	2.5	2.4	2	1	R	X	R	R	R
Mary thomas	M		27106						1						1			R	3	1.3	3.2	3	3	3	2	3	3.2	1.6	1.6	1.6	2					1	1	R X	X	R	R	R
Leela A	M	1.76	27107	13		5	3.9	2.5	2	2.7	2	3.1	13.3	Y	Y			R	3.3	20.5	2	2	1.8	3	3	2	2.5	2	2	2.8	2	2.8	2.8	2.5		3	1	R	X	R	R	R
Subaida Beevi	M	1.66	27087	12		6.8	2.7	4.1	2	2.2	1	2.2	13					R	4.4	13.5	3	2.8	2.6	3			3.6	3.5	3.2	3	2	4.8	4.7	4	3.5	3	1	R X	X	R	R	R
Ravindran nair	M		27460			6	3.2	2.9	2	2.8	1		7.1	Y				R	3.5	2.6	2.8	2.6	2	3	3	4	2.8	2.8	2.6	2	4	3.8	3.6	2.5	2.5	2	1	R	X	R	R	R
Fr John Peeliyani	M		27470	14	159	7	2.3	2.2	2	2	1	2	7.8		Y			CD	4.5	9.5	3.8	2.4	2.5	3	4	3	4.1	4	2	3.2	3	2.8	2.6	2		1	1	R	X	R	R	X
Thankappan M A	M		27535	10.9	98	8.2	2	3.6	1	2.2	1	2.2	6.9	Y				R	5	15.1	3.9	3.2	3.2	3	2	2	2.9	2.9	2.9	2.5	2	3.2	3.2.	3	2.4	1	1	R,X	X	R	R	R
Bhadran K	M		27016		133	9	3	3.2	1	2.9	1	2.1	13					R	3.5	3.5	3.5	3.4	3.3	3	2	4	3.2	3	2.6	2.4	3	4.3	4	3.6		2	1	R	X	R	R	R
Pavithran	M		27017		89	8	2.4	2.4	1	2.6	1	2.5	14					R	4.3	5.8	3.5	3.5	2.5	3	2	3	2.4	2.4	2.4	2	3	4	3.6	3.6	3.2	2	1	LACX	X	R	R	R
Velayudhan S	M		27038	8	72	4.6	2	3.4	1	2	1	2						R	3	7	1.7	1.7	1.5	3	3	2	1.7	1.7	1.3		3	2.3	2.3	2				R	X	RR	R	R
Kunjukunju K M	M		27026	17	96				1		1							R						3	2	2					3					2	1	X	X	R	R	R
Estha Perera	M	1.85	27150	12.8	116	6	2.8	3.1	2	2.4	1	3.3	8.6	TO RA	Y	CSBR		L	2.6	6.2	2.4	2.3	2.2	3	2	2	2.1	2	2	2		1.4	1.4	1.2	1.2	2	1	R	X	X	X	X
Varghese K V	M	1.50	27151			10.1	4	5.5	1	4.1			11.2	Y				CD	5.3	14.9	2.4	1.5	1.5	3	2	3	3.3	2	2	2.7	2	3.8	3.8	3.6	3.4	1	1	R,X	X	R	R,X	R
Binish kumar	M	1.76	27137	14.5	101	7.9	3.3	5.5	2	5			11.3	Y	Y	CS B		CD	4.2	5.3	3.3	3.2	3	3	2	3	2.6	2.6	2.4	2	3	2.8	2.2	2	2	1	2	X	X	R	R	X
Madhusoodhanan	M	1.76	27107	13		5.2	2.2	2.3	1	2.8	1	2.2	12.5		Y			CD	3.4	21.2	1.8	1.8	1.6	3	3	3	2.7	2.1	2	2.7	3	3.1	3	2.6		2	1	R	HIL	R	R	R,X
Sainaba Beevi	M		27109	13.7		5.6	3.2	1.9	1		1							CD	3.3	17.5	2.6	2.4	2.4	3	3	2	2.4	2.2	1.5	2	3	2.5	2.8	2.5	2	1	1	X	X	R	[illegible]	R,X
Marykutty Abraha	M	1.80	27118	15.6	86	9	3.8	3.3	2	2.4	1	2.3	7.9	Y	Y	CSBR		CD	3.7	10	3	2.6	2.6	3	2	2	2.8	2.6	2.4	2.4		2.1	2	1.8	1.5			X	X	X	R	X
Babu P V	M		26990		126				1		1							R						3	3	4					3					1	1	R	X	R	R	R
Sumedha P	M		26991						1		1							CD						3	3	3					2					1	1	R	X	R	R	R,X
Kunjumani	M		26979						1		1							R						3	3	2					2					1	1	R,X	X	R	R	R
Ganesh Kumar	M		26986						1		1							R						2	1	2					1					2	1	LACX	X	R	R	R
Rajamma	M		26808															R						3	2	2 .					1							Distal R,U	X	R	R	R

Bhoo	M		26977							1	1							R						3	3	2					3							R,X		R	R	R
Komalavally	M		27191	11	103	12	3.8	3	1	3.2			20				CS Br	R	4.1	9	3.4	3	2.6	1	2	2	3.9	3.4	3.2	2.5	3	3.2	3	3	2.5	2	1	R	R	R	R	
Rajendra kumar	M	1.70	27194	10.2	104	8	3	4.7	2	3.6			10.7				CS bn	R	4.9	10.8	2.9	2.5	2.3	3	1	3	2.8	2.4	2.4	2.4	2	1.9	1.8	1.7	1.8	1	1	R	X	PLB	R,ACM FROM I	X
Sadandan	M	1.52	27152			7	2.5	2.3	1	2.6	1	2	7	OMV				R	3.7	9.7	2.9	2.7	2.7		2	2	3.1	2.7	2.7	2	3	2.5	2.5	2.4	2.4	1	1	R	X	R	R	R
Sudarsanan	M	1.65	27163	15		8.1	3.1	3.5	2	4.9	1	2.3	7	Y	Y	UTUR		R	4.2	13.3	2.7	2.6	2.6	3	3	2	2.6	2.5	2.2	2.6	2	3	2.8	2.6	2.6	1	2	R			R	
Ponnayan C	M		27140			12.5	2	2.1	2	3.3			9.5					R	4.8	25	3	2.5	2.2	3	3	2	3.6	3.5	3.3	2.2	4	3.2	3.5	3.2	1.3	1	1	R	X	R	R	R
Perumal	M	1.62	27141			8.1	3.9	4.5	3	3.1	1	2.8	12				y	R	3.6	11.4	3.7	3.4	3.2	3	2	2	4.4	4	4	2	3	2.2	2.2	2	2	1	1	R	X	R	R	R
VASUMATHY AM	M	1.73	28409			7	2.6	2.3	3	3								R	4.2	13.6	2.7	2.5	2.5	3	3	2	2.6	2.6	Small			3.3	3.9	3.3		3	1	X	X	R	R	R
KOCHAPPAN	M	1.58	28678	7		8.4	2.9	4	1	3.3								R	4.2	16.4	3.2	3	3	3	3	5	2.5	1	1	1.9	3	3.3	3.3	2.6		1	1	LACX	X	R	R	R
SUBAIDA	M	1.50	28508	15.2	108	7.8	2.2	3.8	3	2.5								L	3.6	6.5	3.1	2	2	2	2	1	3.5	3.2	2.9	2.4	2	2	1.8	1.6		1	1	R	X	X	X	X
KRISHANPILLAI	M	1.58	28511	25.7					2									R	4.9	11.5	4.2	3	2		2	2	3	2.8	2	1.3	2	3.1	2.1	2.5		1	1	R	X	R	R	R
RAMALABEEVI	M		28531						1									L	2.7	11.1	2.6	2.4	2	2	2	2	2.3	2.,3	2.2	1.9	3	2.3	2.3	2	2	1	1	R	X	X	X	X
SARASWATHY	M		28564						1									R	4.2	8.1	3.2	3.2	3		2	2	3.4	3.2	3.2	3.2	2	2	2	1.6	1.4	1	1	R			R	R
VELU	M		28555						2					Y	Y	CSBr																										
PREMACHANDR/	M	1.92	28505			10.2	4.6	3.8	2									R	3.8	11.4	3.4	3.4	3.2	3	2	2	2.1	2	2	2	1	3.4	3.4	3.2		1	1	X	X	R	R	R
SASIDHARAN	M	1.83	28673	14		6.7	1.6	3.4	2	3.5			11		Small			CD	4.7	17.8	2.2	2	2	3	2	2	3.5	3	2.8	2.5	3	2.8	2.6	2.6		1	1	X	X	R	RX	X
KAMALAKSHI	M	1.44	28679			8	2.7	5.3	1	3.4	1	1.8	11.7		Y		Y	L	2.9	3	1.9	1.8	1.5	3	2	2	1.9	1.8	1.1	1.1	1								X	X	X	X
PUROSHOTHAM/	M	1.80	28615		121	7	2.5	5.1	2	5	1						Uluren	R	3.7	22.1	3.2	3	2.7		2	2	2.3	2	2	2	3	3.9	3.9	3.7		1	1	LACX	X	R	R	R
SADASIVAN k	M		28621			9.2	3.5	3.6		3.6			13					R	5.5	14.1	4	2.2	2.1	3	2	1	2.3	0.9	0.9	1.6	1	3	2.6	2.6	2.6	1			X	R	R	R
SIVADASAN CHA	M		28617						1									R	4.4	7.7	2.7	2.8	2	3	2	2	3.9	2.4	1.8	1.8	3	5.7	2.9	3.1	2	1	1BIF	X	X	R	R	R
MARIAMMA	M	1.70	28620	11.8	94	8.6	3	4	1	3			12	3.7		Bifurc	I	R	5.7	17.1	4.6	2.2	1.7	3	2	1	4.1	3.2	2.5	2.3	2	4.1	1.8	1.6		1	1	R,X	X	R	R	R
SIVASANKAN g	M	1.69	28614	14	127	6.5	2.9	4.5	2	3	1		11.6	3.1	OMV			L	3.8	6.4	2.5	1.7	1.7	3			2.3	1.9	1.4	1.4	3	2.1	2.3	2	2	3	1				R,X	X
SASIDHARAN NA	M	1.65	28709	19.3		7.4	3.3	2.8	2	3.6			12.7	Y		CS B		R	3	6.7	2.4	2	2	3	2	2	2.5	2.4	2	2.2	2	3.3	3.2	3.3	2.6	2	1	X	X	R	R	R
JOHN KC	M		28509	10.7	133	11.1/3	2L7	6.1	1	3.3	1	2.8	10		Y			R	5	16.5	3	2.5	2.3	3	2	3	3.3	3	2.5	2.5	2	3.5	3	3	2	2	1	X	X	R	R	R
NOOR JAHAN	M	1.90	28409	14.2	170	9//3	2.5	3.4	1	4.4	1	3.9	14					L	4.2	4.5	3.4	3.2	3	3	3	3	4.1	4	4	3.2	3	2.5				2	0	R	X	X	X	X
GEORGE S	M		28349			8	2.5	2.5	1	2.5	1	2.3						R	4.2	9.8	2.9	2.5	2.4	2	1	2	3.2	3.1	2	3.1	1	2.4	2.4	2		1	1	LACX	X	R	R	R
KAMALAKSHI	M	1.56	28679			9	4.2	4	1	4	1	4.2	18.2		Y			L	4.8	13.5	2.4	2	2	3	2	3	4.1	3	2	1.6		4.6	3.6	3	2.5	2	1	R	X	R	R	R
SIVASANKARAN	M	1.60	28503	14.4	150	7.5	1.8	4.1	1	2.1	2	2.5	14		Y			R	3	6.3	3.2	2	2	3	3	2	3.1	2	2	1.7	2	2.3	2.5	2.3	2.3	1	1	X	X	R	R	R
RAMAKRISHNAP	M	1.65	28504	12.6	133	9.9/5.8	3	2.4	2	3.1	1	2.5	17.9	DOUE				L	4.6	12	3.1	2.6	2.2	3	2	2	3.3	3.3	2	3.2	2	2.8	2.6	2.5	2.2	1	1	R	X	R	R	R

PROJECT REPORT
(PROJECT NO. 2)

TITLE

EVALUATION OF VENTRICULAR FUNCTION USING TISSUE DOPPLER ECHOCARDIOGRAPHY

NAME : DR. KRISHNAKUMAR NAIR

PROGRAMME : D M CARDIOLOGY

MONTH & YEAR OF SUBMISSION : NOVEMBER 2002

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY TRIVANDRUM 695 011

Name	
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SREE CHITRA TIRUNAL INSTITUTE FOR
MEDICAL SCIENCES & TECHNOLOGY

EVALUATION OF VENTRICULAR FUNCTION USING TISSUE DOPPLER ECHOCARDIOGRAPHY

KRISHNAKUMAR NAIR

TABLE OF CONTENTS-

EVALUATION OF VENTRICULAR FUNCTION USING TISSUE DOPPLER ECHOCARDIOGRAPHY

INTRODUCTION-

Doppler ultrasound has been traditionally applied for the measurement of blood flow velocities across intravascular structures. Moving red blood cells reflect low amplitude, high velocity doppler signal. In contrast, moving tissue such as the myocardium typically reflects low velocity, but a high amplitude doppler signal. In a conventional Doppler system a high pass filter is incorporated to eliminate these low velocity signals and the gain settings are increased to amplify the signals reflected by moving blood.

Mitral pulsed Doppler echocardiography provides information of blood velocities in a specific location, reflecting both LV relaxation and LV compliance. On the other hand, the velocities obtained in tissue doppler represent the myocardial motion parallel with the Doppler beam, which reflect the rate of LV volume change in the long axis when measured at the mitral annulus.

Tissue Doppler-imaging methods require modifications in signal processing of the returned Doppler signals. To display tissue velocities, two relatively simple alterations in Doppler signal processing are required: 1) the high pass filter is bypassed and 2) a lower gain amplification is used to eliminate the weaker intensity blood flow signals.

The available literature on tissue Doppler imaging lists several small studies ,each studying a few variables. This study included almost all the variables studied so far (and some new ones) ,and applied it to the same population. Of the available tissue Doppler modalities, this study used the pulse doppler technique in our study. This is the easiest way to measure myocardial velocities. It has been used for interrogation of myocardial or mitral annular velocities.

AIM AND OBJECTIVES

The aim of this study was to study the utility of tissue Doppler in the assessment of systolic and diastolic function.The specific objectives were as follows-

I.The first objective of this study was to determine the utility of Doppler tissue echocardiography in the evaluation of diastolic filling and in discriminating between normal subjects and those with various stages of diastolic dysfunction.

II.To assess the suitability of tissue Doppler in the evaluation of systolic function and in discriminating between normal subjects and those with various stages of systolic dysfunction.

III.To assess the correlation, if any, between tissue Doppler signals directly from the lateral segment in comparison to the lateral mitral annulus in patients with and without wall motion abnormalities.

IV.To examine the relationship between velocities at various myocardial segments recorded by pulsed tissue Doppler imaging and LV regional wall motion in patients with previous myocardial infarction (MI).

V.To assess the relevance of the modified Tei 's index

REVIEW OF LITERATURE

Spectral pulse wave Doppler method provides the highest 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.

A typical waveform of PW-TD is shown in Figure 1.

The pattern can be divided into 2 parts, systolic and diastolic, from which several measurements can be obtained [1,2]

The systolic phase-is characterized by

1.a positive wave "Sm" or Sw2

2. preceded by the isovolumic contraction "IVC" or Sw1 velocity

The diastolic phase comprises of 4 periods:

1. Isovolumic relaxation (IVR) velocity

2. Rapid-filling period, characterized by a negative wave ("Em")

3. Diastasis

4. Filling caused by atrial contraction, represented by a second negative wave ("Am").

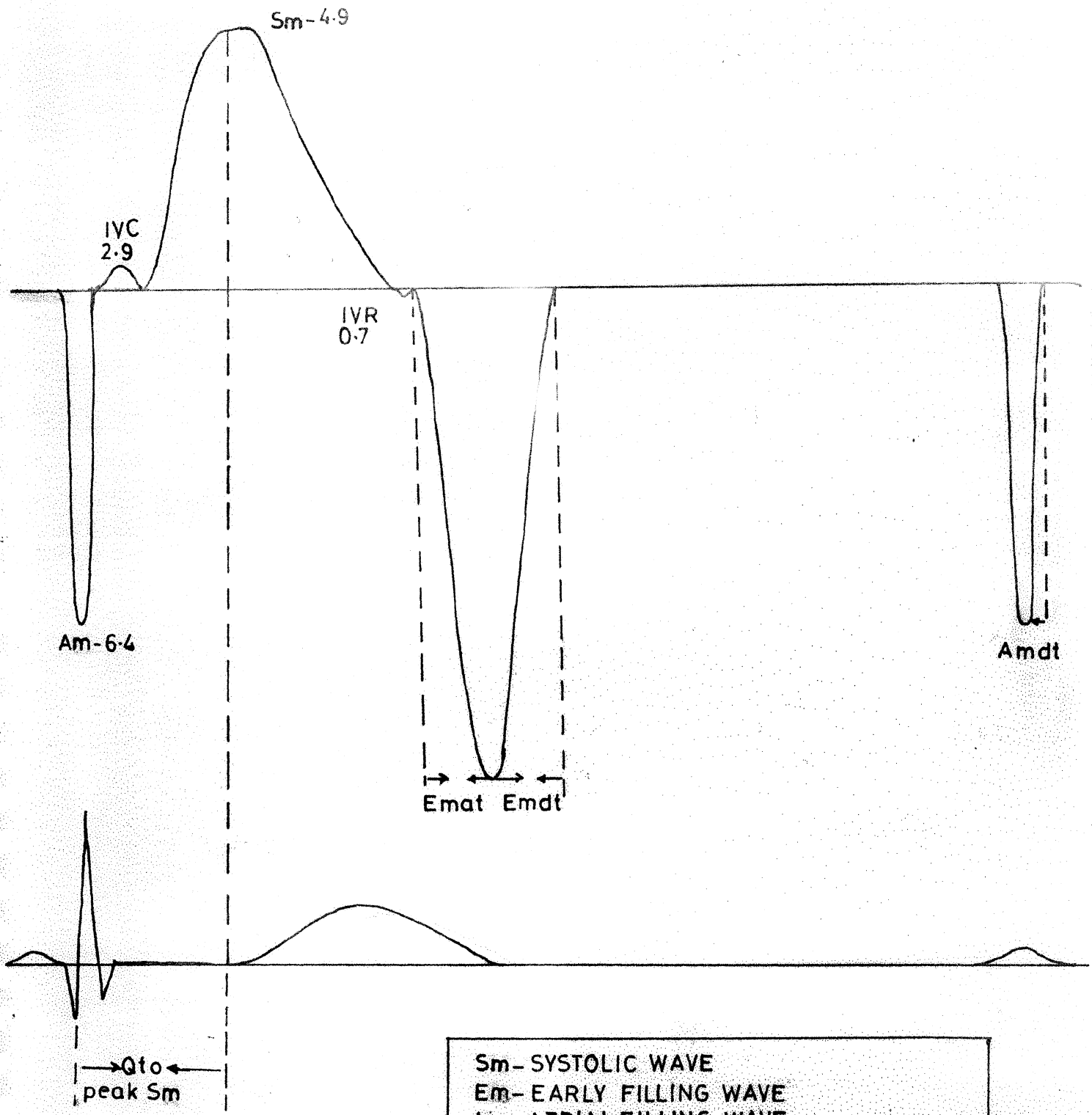
Garcia and colleagues[3] reported that biphasic velocity components can be observed during both isovolumic contraction and relaxation in parasternal long-axis views, and they are the result of the asynchronous myocardial activation between the base and the apical regions of the heart. The multiphasic signals frequently detected during isovolumic contraction and relaxation, which are normally not apparent from two-dimensional echocardiography are thought to be caused by rapid small geometrical changes that occur in the LV and by right ventricular interdependence .

Directionality of the TDI velocity display is dependent on location of the pulsed Doppler sample volume relative to the myocardial site.

When using pulsed TDI,the Nyquist limit should range from –20 to 30 cm/s, with minimum gain and low wall filter settings.The monitor sweep speed should be set at 50 to 100 mm/s to optimize the spectral display of myocardial velocities.

ASSESSMENT OF DIASTOLIC DYSFUNCTION

Diastolic dysfunction can be defined as the inability of the heart to maintain normal diastolic pressures during left ventricular filling.Conventional clinical evaluation of left ventricular relaxation involves determining the time constant of pressure decay during isovolumic diastole, as calculated from the left ventricular pressure curve.



Sm - SYSTOLIC WAVE
Em - EARLY FILLING WAVE
Am - ATRIAL FILLING WAVE
IVC - ISOVOLUMIC RELAXATION VELOCITY
Emat - Em ACCELERATION TIME
Emdt - Em DECELERATION TIME
Amdt - Am DECELERATION TIME

FIGURE - 1

The gold standard- for evaluation of diastolic dysfunction has been the direct measurement of left ventricular pressure with simultaneous left ventricular volume estimation. This allows us to construct pressure-volume curves and determine the exact relationship between these parameters. But, this requires invasive placement of intraventricular catheters with sophisticated micromanometers, which make this method unsuitable for routine use or for serial studies.

Doppler echocardiography has become the non-invasive technique of choice for evaluating diastolic function. However, as several physiological variables—including volume status, left atrial pressure, and the rate of myocardial relaxation—affect Doppler flow velocities simultaneously, it is often difficult to determine which individual variables are responsible when a specific Doppler pattern is observed, unless other relevant clinical information is available.

UTILITY AND LIMITATIONS OF STANDARD DOPPLER FILLING INDICES

Transmitral Doppler velocities are related to transmitral pressure gradients as determined by Bernoulli's equation of flow [4,5]. Standard pulsed-wave Doppler velocities may be applied only to determine the convective component of the pressure gradient by using the simplified form of this equation. Doppler-derived pressure gradients are clinically accurate in diseased valves with restrictive orifices. However, in conditions of pulsatile flow across a nonrestrictive orifice such as a normal mitral valve, the relative contribution of the inertial component to the pressure gradient has been shown to be significant [6,7] and therefore true pressure gradients are underestimated.

PULMONARY VEIN DOPPLER-

In an attempt to overcome the limitations of transmitral flow Doppler indices of LV filling, several investigators have incorporated routinely the assessment of PV flow [8,9,10]. Doppler indices of PV flow have been used in the assessment of LA pressure, differentiation of constrictive pericarditis from restrictive cardiomyopathy and assessment of the severity of mitral regurgitation 11-14 and to evaluate different indices of diastolic function, including left ventricular filling, pressure, relaxation, and stiffness.

Most normal adult patients exhibit a prominent systolic (S) flow and a systolic-to-diastolic (S/D) ratio >1. In patients with elevated LV filling pressure, reduced LA and LV compliance or with severe mitral regurgitation there is blunting of the pulmonary

venous S wave and increased D flow. This pattern, in addition to prominent atrial reversal (AR) flow velocity, has been used to distinguish normal from pseudonormal transmitral Doppler filling [15].

Normal filling pattern of LV-

The normal filling pattern is seen in patients with normal LV relaxation rate, compliance and filling pressures. Atrial contribution to LV filling is minimal. Thus, standard Doppler indices of LV filling and PV flow are characterized by high E, E/A ratio <1, IVRT <100 ms and early filling deceleration time (DT) <220 ms. In young adults, athletes and pregnancy the very rapid relaxation rate results in rapid and near-complete LV filling during early diastole, causing very short IVRT, prominent E and short DT. The LA behaves primarily as a reservoir and conduit. Since LA volume before atrial contraction is minimal, LA contractility is reduced, resulting in low ejection volume, A and AR velocities, reduced LA relaxation force and consequently low S. As atrial contribution to LV filling increases with age, A and S become more prominent and S/D ratio becomes >1. Color M-mode Vp is fast, in our experience >55 cm/s in younger and >45 cm/s in older adults. Tissue Doppler echocardiography Em velocity measured in the LV long axis plane is >10 and >8 cm/s, respectively.

STAGES OF DIASTOLIC DYSFUNCTION [16]

Stage I (delayed relaxation)-

Patients with coronary artery disease, advanced age, hypertension and early restrictive cardiomyopathy typically exhibit a Doppler pattern of "delayed relaxation." This pattern is characterized by decreased early filling (E) and increased atrial contraction (A) mitral flow velocities. This pattern is seen in patients with reduced LV relaxation rate but relatively normal compliance and filling pressures. Atrial contribution to LV filling is increased, frequently >30% of the stroke volume. The delayed relaxation pattern is characterized by an E/A ratio <1, prolonged DT (>220 ms) and IVRT (>100 ms). Pulmonary venous flows show S > D with usually prominent AR. Color M-mode Vp is reduced (<45 cm/s), as well as TDE Em (<8 cm/s).). Patients typically have only mild symptoms or are asymptomatic and may have mild LA enlargement

Stage II (pseudonormal)

This pattern is often the most difficult to recognize since, as its name implies, Doppler filling indices resemble those found in normal subjects. Left ventricular relaxation rate and compliance are reduced, but filling pressure is now increased as a

compensatory or overcompensatory mechanism to maintain cardiac output.. The elevated LA pressure results in earlier opening of the mitral valve and, thus, shorter IVRT. Increasing filling pressure increases early transmitral gradient and transmitral flow velocity and reduces early flow deceleration time and atrial flow velocity. The reduced LV compliance causes rapid increase in LV pressure with cessation of LV filling and reduced DT. Atrial contribution to LV filling is reduced due to the increased end-diastolic LV stiffness resulting in reduced A and pulmonary venous S/D <1. Pulmonary venous AR is >35 cm/s unless atrial mechanical failure is present. Since LV relaxation is impaired, color M-mode Vp remains reduced, <45 cm/s, as well as TDE Em (<8 cm/s). Patients have mild-to-moderate symptoms of pulmonary vascular congestion and various degrees of LA enlargement depending upon the chronicity of disease. Other evidence of structural heart disease, such as increased LV volumes and mass and reduced ejection fraction, is also commonly present

Stage III (restrictive filling)-

The last clinical filling pattern is seen in the presence of profound abnormalities of LV compliance and markedly increased filling pressure. Left ventricular relaxation is reduced, perhaps with the only exception being patients with isolated constrictive pericarditis. Patients have overt heart failure and moderate-to-severe LA enlargement depending upon the chronicity of disease. Echocardiographic features of advanced structural heart disease are evident by now. Standard Doppler filling indices are characterized by an increased E/A ratio (>2), short DT (<150 ms) and IVRT (<60 ms). Pulmonary venous flow usually shows markedly blunted S. Prominent AR is frequently not present, probably because of atrial mechanical failure, and usually carries a poor prognosis Color M-mode Vp and TDE Em are the lowest, except in patients with constrictive pericarditis in whom LV relaxation is normal.

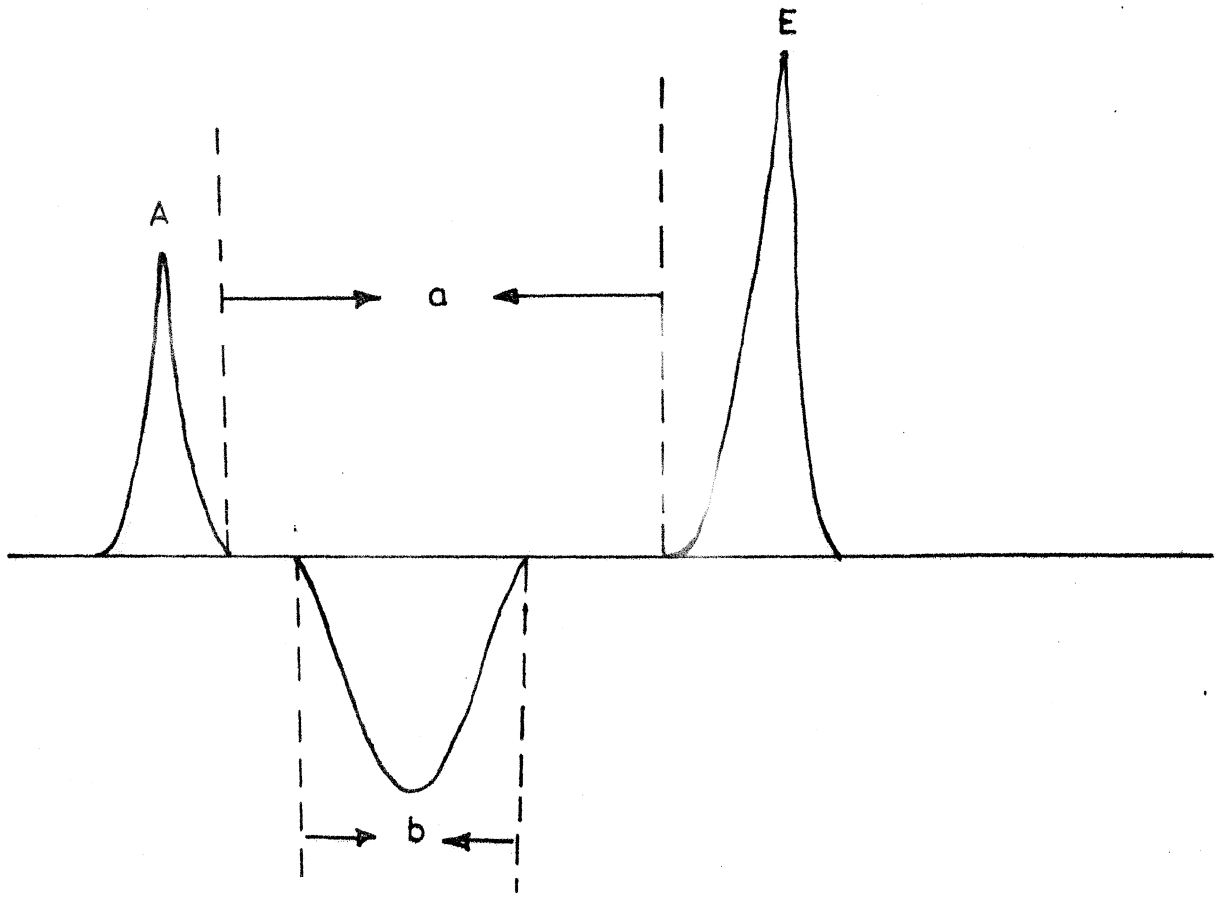
However, in normal young adults and athletes in whom atrial contribution to LV filling is minimal and the LA behaves more as a "passive" conduit, blunting of the S wave is also common in pulmonary vein Doppler. Although normal patients may be recognized by their AR velocities of shorter amplitude and duration [17]. AR velocities are also frequently diminished in patients with restrictive filling, possibly because of atrial mechanical failure [18,19]. In addition, AR amplitude and duration are often difficult to measure in many patients from transthoracic echocardiography.

To date, only a few publications have studied the utility of Doppler tissue velocities in the assessment of diastolic function. In healthy normal subjects, the motion of the

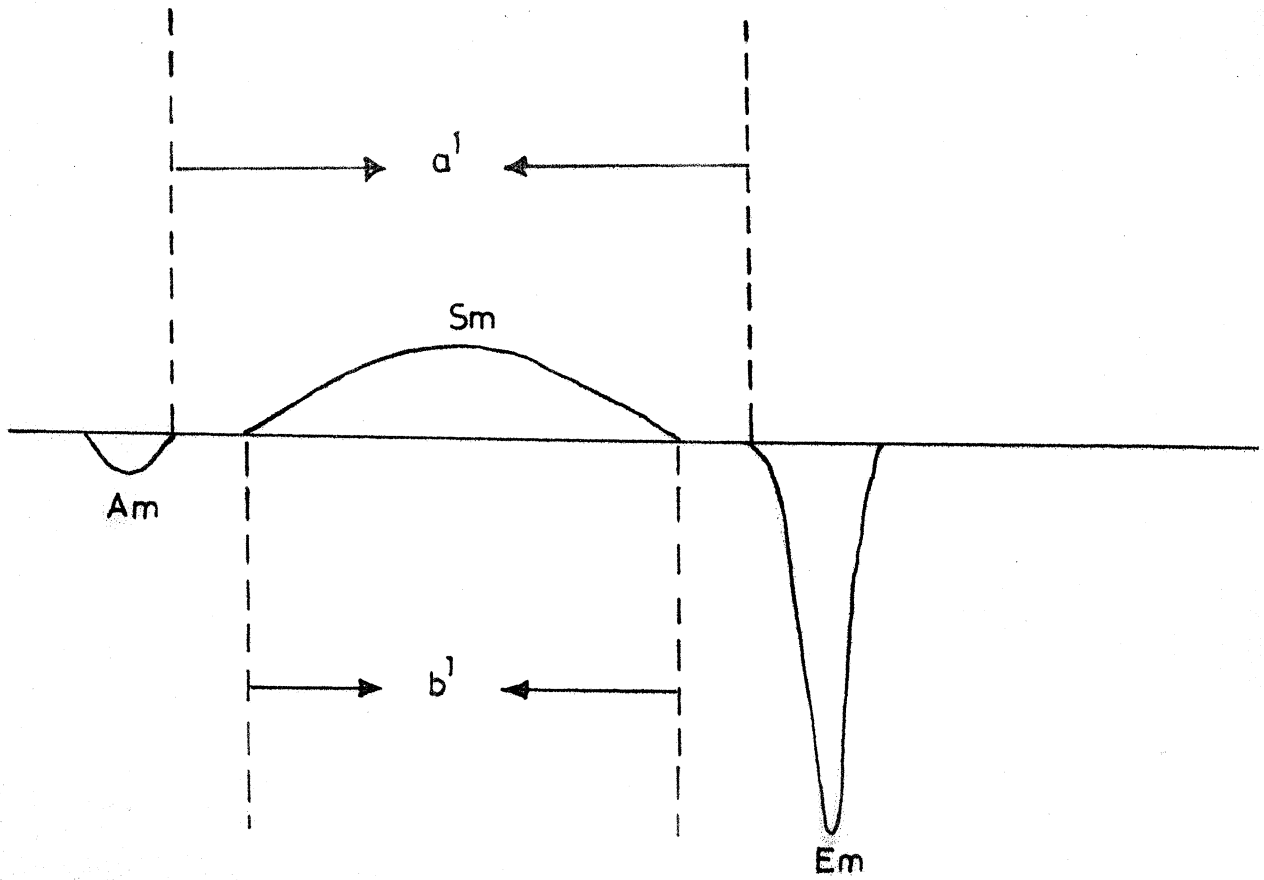
myocardium during diastole appears as a mirror image of the transmitral Doppler flow Tissue Doppler echocardiography (TDE) can provide accurate quantitative information about myocardial motion during the cardiac cycle.

THE MODIFIED TEI INDEX [20]

The Tei Index is an easily measured Doppler index of combined systolic and diastolic ventricular myocardial performance .It was proposed as a potentially useful predictor of global cardiac function. 21-30. This index is defined as (a _ b)/b, where a is the interval between end and onset of the mitral in- flow, and b is the ejection time of left ventricular (LV) outflow (Figure 1)There is an important limitation in that the interval between the end and the onset of mitral inflow and ejection time is measured sequentially and not on the same cardiac cycle. Because of this, results are probably less reliable in the presence of heart rate fluctuation.Tissue Doppler imaging (TDI) enables us to simultaneously measure contraction and relaxation velocities from myocardium.[31-34] Using TDI, the time interval between the end and the onset of mitral annular velocities during diastole (a' minus the duration of the S wave (b') divided by b (i.e., (a'- b')/b') (Figure 2) may approximate the Tei index obtained by the conventional Doppler method



Tei index = $\frac{a-b}{b}$



Modified Tei index = $\frac{a^1-b^1}{b^1}$

FIGURE - 2

METHODS

STUDY POPULATION

I.Diastolic dysfunction-

The study group consisted of 52 patients with abnormal diastolic function (38- impaired relaxation and pseudonormal groups combined,and 14 - the restrictive pattern group) referred to our laboratory for echocardiographic evaluation. 25 age and sex matched normals served as a control group (group 1).

The consensus opinion of 2 experts blinded to DTE results was used to classify patients into each of the different stages of diastolic dysfunction by using published criteria accepted by the Canadian Consensus on Diastolic dysfunction [35] (Table 1). All patients met at least 3 of the 6 criteria listed in this table.

STAGE	E/A	EDTms	IVRTms	S/D	ARcm/s	Other
DELAYED RELAXATION	<1	>220	>100	>/=1	<35	-
PSEUDONORMAL	1-2	150-200	60-100	<1	>/=35	Structural heart disease
RESTRICTIVE	>2.	<150	<60	<1	>/=35	Structural heart disease

TABLE I

II.Systolic dysfunction-

The study group consisted of 16 patients with ejection fraction 35% to 50% (group 2) and 13 patients with ejection fraction less than 35% (group1). 25 age and sex matched normals served as a control group (group 3).

III.Tissue Doppler signals directly from the lateral segment in comparison to the lateral mitral annulus in patients with and without wall motion abnormalities

There were 2 groups –Group 1-17 patients with lateral wall WMA (28males) and Group 2-83 patients with no lateral wall WMA (males)

IV.Wall motion abnormalities (WMA) -

There were 6 groups –Group 1-34 patients with no WMA (28males), Group 2-38 patients with anterior wall WMA (36males) ,Group 3-38 patients with interventricular septal WMA (37males),Group 4-17 patients with lateral wall WMA

(15males), Group 5-29 patients with inferior wall WMA (25males) and Group 6-15 patients with posterior WMA (14males).

V. Modified Tei 's index-

The modified tei index was computed in patients with different degrees of diastolic function , and in patients with different degrees of systolic function.

This index was computed in 100 patients-correlation was tested with the Tei's index

Echocardiographic Study

All patients were examined at rest in the left lateral decubitus position. Studies were performed with a commercially available ultrasound system equipped with DTE capabilities (System five, GEVingmed). A variable frequency transducer (1.5 to 4.0 MHz) was used in all 2-dimensional and Doppler examinations. Twodimensional studies were recorded from the parasternal long and short axes and from the apical 4- and 2-chamber views. LV end-diastolic and end-systolic volumes were traced from the apical 4-chamber view. Ejection fraction was calculated with M-Mode or off-line with the use of the area-length method.

Doppler Tissue Echocardiography

The spectral pulsed Doppler signal was adjusted to obtain a Nyquist limit of 15 or 20 cm/s, with the lowest wall filter settings and the minimum optimal gain to eliminate the signals produced by the transmitral flow. To record the velocities of LV motion in its longitudinal axis, 2-dimensionally guided, pulsed DTE of the lateral mitral annulus and mid interventricular septum in apical 4chamber view was done.To record the tissue velocities of individual myocardial segments, 2-dimensionally guided, pulsed DTE of the anterior,lateral, septal, inferior, and posterior myocardial segments were performed from the short axis views and their values were averaged. Ten consecutive beats were recorded at a sweep rate of 100 mm/s during apnea and stored on half-inch videotape for off-line analysis.

From the videotaped recordings,we measured peak velocities during systole (Sm), early diastole (Em),and late diastole (Am).Measurements were obtained from 10 consecutive beats by 2 experienced echocardiographers and averaged.

Standard Doppler Flows

1.Standard pulsed wave Doppler transvalvular flow measurements were obtained immediately before the DTE recordings. Normal gain and low-filter settings were

optimized to record the onset and cessation of flow.Mitral flows were recorded from the apical 4-chamber echocardiographic window, placing the sample volume at the level of the mitral valve leaflet tips. Peak early (E) and atrial (A) contraction velocities, early filling acceleration time and deceleration time,A deceleration time and isovolumic relaxation time (IVRT) were measured and averaged from 10 consecutive beats.

2.PV flow was recorded from the same acoustic window, placing a sampling volume 1 cm into the right upper pulmonary vein. Peak systolic (S), diastolic (D), and atrial reversal (AR) velocities were measured and averaged from 10 consecutive beats.

3.LV end-diastolic and end-systolic volumes were traced from the apical 4-chamber view.Ejection fraction was calculated with M-Mode or off-line with the use of the area-length method.

4.Wall motion abnormalities were recorded in the parasternal long and short axes and from the apical 4- and 2-chamber views.

PARAMETERS STUDIED-

Velocities going above the baseline have been marked as negative,and those going below the baseline marked as positive.

I.For assessment of diastolic dysfunction-

Standard Doppler Flow Indexes

1.Peak E velocity ,Peak A velocities and the E/A ratio

2. IVRT ,E-deceleration time and the A- deceleration time

3. Tei 's index

Doppler Myocardial Velocities

1. Sm velocity or Sw2.

2.Isovolumic contraction phase velocity or IVC or Sw1

3.Em velocities

4.Peak myocardial velocities during atrial contraction (Am)

5.The ratio Em/Am

6 Em/E :the ratio of early diastolic velocity at the lateral mitral annulus by tissue Doppler to the pulse Doppler mitral filling velocity.

7. (Sm + Em +Am)

8.The isovolumic phase relaxation velocity(IVR velocity)

9.The Em –acceleration time

10. The Em –decceleration time

11. The mean total Am –decceleration time

12.The mean total Em/acceleration time(Em/ACT)

13. The mean total Em/decceleration time(Em/DCT) velocities

14. The modified Tei index

II.For assessment of systolic dysfunction

1.Sm: The mean systolic velocity by tissue Doppler

2.IVC-Vel :The isovolumic contraction phase velocity by tissue Doppler

3.IVC-Vel / Sm: The ratio of the IVC-velocity and Sm

4.Q to peak of Sm time

5.Sm + Em + Am:

6.Em / E:

7.Tei and Modified Tei Index

III.Tissue Doppler signals directly from the lateral segment in comparison to the lateral mitral annulus in patients with and without wall motion abnormalities

1.Doppler Sm velocities

2.Em velocities

3.Peak myocardial velocities during atrial contraction (Am)

4.The ratio Em/Am

IV.Tissue Doppler and wall motion abnormalities

1.Doppler Sm velocities

2.Em velocities

3.Peak myocardial velocities during atrial contraction (Am)

4.The ratio Em/Am

V.The modified Tei 's index-

STATISTICAL ANALYSIS

Values are reported as mean ± SD. Myocardial velocities,Doppler indexes of transmitral filling and PV flows, LV ejection fraction, and LA areas were compared between

1)3 different groups by use of the *F* test (analysis of variance,ANOVA).

2)different variables in the same population using the paired t-test.

3)same variables in 2 different populations using the independent t-test.

Correlation was done between the modified Tei index and the Tei index.

A value was considered statistically significant at $P < .05$.Tests of correlation were done wherever indicated.Statistical analysis was performed with the use of commercially available software (SSPS version 10.0).

RESULTS

DIASTOLIC DYSFUNCTION-

STANDARD DOPPLER FLOW INDEXES

The mean and standard deviation of all the tested variables along with their P value are mentioned in Table II.

TABLE II.One wayANOVA-DIASTOLIC FUNCTION

Variable	Groups	N	Mean	Std.Devia	FVALUE	PVALUE
MV-E	normal	25	.8596	.1285	15.6087	0000
	impairedrel	39	.7124	.1772		
	restrictive	14	.9750	.1638		
	Total	78	.8079	.1889		
MV-A	normal	25	.6904	.1359	23.0628	.0000
	impairedrel	39	.8703	.2482		
	restrictive	14	.4536	.1412		
	Total	78	.7378	.2526		
E/A	normal	25	9.8420	42.9503	1.0744	.3467
	impairedrel	39	.8676	.2266		
	restrictive	14	2.4614	1.0241		
	Total	78	4.0301	24.3241		
IVRT	normal	25	95.3200	30.6278	.7627	.4700
	impairedrel	39	101.3846	28.0438		
	restrictive	14	91.1429	27.9501		
	Total	78	97.6026	28.7909		
EDT	normal	25	166.1520	64.1564	6.1683	.0033
	impairedrel	39	191.5128	67.2743		
	restrictive	14	124.8571	32.4721		
	Total	78	171.4205	65.5909		
ADT	normal	25	108.6957	23.9895	.1796	.8360
	impairedrel	39	114.2000	29.6646		
	restrictive	14	110.0000	62.8129		
	Total	78	111.6714	35.3946		

1.Peak E velocity decreased from .8596 =/-0.1285 m/s in normal subjects to.7124 +/- .1772 m/s in patients with delayed relaxation and increased to 0.975 ± 0.16 m/s in patients with restrictive filling, respectively .(F =15.6087 P< .00001)

2.Peak A velocities were highest in patients with delayed relaxation (.8703+/- .2482m/s) F = 23.0628 P< .0001

3.Both E/A and IVRT and were not significantly different between the three groups. F = 1.0744,P= .3467 and F=.7627 ,P= .4700 respectively±

4.The A-deceleration time was also not significantly different between the three groups.**F=.1796,P= .8360**

5.The E- deceleration time was significantly different between the three groups and highest in the group with impaired relaxation.(F =6.1683 ,P= .0033)

DOPPLER MYOCARDIAL VELOCITIES

The mean and standard deviation of all the tested variables along with their P value are mentioned in Table III.

TABLE III.ANOVA-DIASTOLIC FUNCTION

Variable	Groups	N	Mean	Std.Devia	FVALUE	PVALUE
Em	normal	25	9.3200	3.3257	10.6227	.0001
	impairedrel	39	5.6667	3.6153		
	restrictive	14	5.7143	1.5898		
	Total	78	6.8462	3.6399		
Am	normal	25	6.4000	1.9579	10.6689	.0001
	impairedrel	39	6.8462	2.9606		
	restrictive	14	3.3571	1.4991		
	Total	78				
Sm	normal	25	-4.9200	6.3961	.3831	.6830
	impairedrel	39	-5.3590	4.0878		
	restrictive	14	-4.0857	1.6487		
	Total	78	-4.9897	4.6557		
Em/Am	normal	25	2.0496	1.9123	5.6091	.0054
	impairedrel	39	.9595	.8723		
	restrictive	14	1.7362	.8659		
	Total	78	1.4509	1.3862		

TISSUE DOPPLER

NAME	HNO	AGE	SEX	LVI DD	LVI DS	E F	EFA RE AL	V 19	A W	L W	P W	IW	V2 4	V25	V26	EDT	IVR T	ADT	V3 0	V31	B	V33	V34	AM	SM	V37	IVR _VE L	IVS _A4 CE	V40	V41	V42	V43	V4 4	I WE M	V4 6	V4 7	V4 8	V4 9	V5 0	LW EM	V5 2	V5 3	AWE M	V5 5	V 66	V67	V68	V69	V70	EE M	IVCS M
SURENDRAN	199453	49	m	47	26	81		n	n	n	h	h	.6	.9	.67	206	116	190	I	370	310	.19	3	8	-6	2	.8	2	7	5	2	2	2	7	4	-3	3	4	-4	3	5	-3	1	5	3	50	100	50	110	.38	.05	17	.6	.3		550	180		2.1	.20	-.33
omanaamma	199041	63	f	34	23	67		n	n	n	n	n	1.0	1.1	.91	234	64	81	I	460	300	.53	4	8	-6	-3	-.1	5	8	-8	4	3	5	3	4	-3	3	2	-3	1	2	-6	2	2	5	60	130	90		.63	.04	18	.1	.1		470	170		2.8	.26	.50
appukuttan nair	199697	55	m	44	26	76		n	n	n	n	n	.6	.6	1.00	160	90		I				12	8	-11	-1	1.0	7	9	-6	4	3	-2	5	4	-4	6	3	-5	6	1	-6	-3	1	3	60	110	110	150	1.50	.20	31	1.5	1.1		500	150		2.3	.05	.09
radhalekshmy	201348	60	f	52	42	47		n	n	n	a	a	.9	1.0	.90	193	76	100	I	440	220	1.00	4	6	-5	-1	-2.0	3	7	-6	-2	-4	2	-2	4	-3	1	5	-4	5	2	-4	-3	-7	5	50	70	40	190	.67	.04	15	.8	.6		550	320		.7	.23	.20
sreedharan		68	m	44	26	80		n	n	n	n	n	.6	.6	.93	222	70	130	I	460	270	.70	8	7	-6	-2	-.6	6	4	-4	2	1	-2	3	3	-3	3	1	-2	3	3	-4	-4	-3	4	130	150	70	150	1.14	.14	21	.6	.5		400	140		1.9	.07	.33
sainudeen	201492	44	m	48	31	74	50	h	a	n	n	n	.7	.8	.90	195	76		I	390	240	.63	3	5	-4	-1	.9	3	4	-4	6	4	-5	5	4	-5	7	4	-6	4	6	-4	9	2	-6	20	60	50	100	.60	.04	12	1.5	.5		490	200		1.4	.24	.25
gopinathan	9010250	60	m	46	27	86		n	n	n	n	n	.7	.9	.78	280	66	130	I	370	290	.70	8	9	-5	-3	1.0	5	6	-3	2	6	-1	2	5	-3	5	6	-6	3	3	-2	-1	-3	2	50	130	60	170	.89	.11	22	1.6	.6		630	340		.9	.09	.60
sasikumar	201806	36	m	46	29	74		h	h	n	n	n	.6	.7	.86	95	81	70	I	370	240	.54	10	3	-11	-5	2.0	8	5	-4	5	7	-7	3	5	-4	4	2	-4	3	5	-4	3	-2	-4	60	120	30	110	3.00	.17	24	1.3	.8		430	270		.5	.06	.45
basheerkoya	201805	54	m	40	22	82		n	n	n	n	n	.6	.7	.86	128	76	120	I	390	270	.44	5	1	-5	-2	2.0	4	6	-4	3	2	-2	3	4	-3	3	3	-3	3	4	-1	-2	-2	2	50	150	60	160	5.00	1.00	11	1.0	.3		510	250		1.0	.12	.40
youniskutty	197555	57	m	41	24	79		n	n	n	n	h	.7	.9	.78	240	93		I	350	210	.67	8	11	-10	-5	1.0	4	9	-3	-4	-2	2	4	5	-4	2	4	-3	3	6	-3	-3	4	3	90	80	50	80	.73	.11	29	.9	1.3		460	300		.6	.09	.50
kuttan	201944	67	m	55	41	60	43	h	a	n	n	n	1.0	1.1	.91	170	93	140	I	410	240	.71	4	9	-6	-1	-2.0	3	5	-3	-5	-3	4	3	4	-4	5	3	-5	4	7	-2	3	-2	2	40	180	90	110	.44	.04	19	1.0	.2		530	260		1.0	.25	.15
chacko	9901052	71	m	48	31	73		n	n	h	h	h	.6	.8	.75	240	100	110	I	460	250	.92	3	6	-5	14	3.0	2	4	-2	-2	-1	3	3	6	-4	3	5	-4	2	5	-3	-3	-2	3	90	140	50	110	.50	.03	14	.3	.2		580	270		1.2	.20	-2.80
abdulrazak	202160	58	m	53	35	60		h	h	n	n	n	.4	.5	.80	228	115		I	510	250	1.04	6	8	-8	-2	1.0	4	6	-4	3	2	3	3	6	-4	7	2	-3	4	6	-6	2	1	1B	130	180	70	90	.75	.15	22	.5	.3		560	300		.8	.07	.25
kunjiraman	198946	57	m	60	49	45		a	d	a	n	n	.9	.8	1.13	110	105	150	I	470	300	.57	6	6	-5	-3	-2.0	5	5	-6	-4	-1	5	5	4	-4	7	1	-3	-4	6	-2	-3	3	2	60	90	40		1.00	.07	17	.8	.7		440	270		.6	.15	.60
narayanannair	192627	55	m	37	20	86	64	h	h	n	n	n	.7	.8	.88	210	110	90	I	410	300	.37	5	8	-5	-2	-.6	6	8	-3	-1	2	2	3	5	-4	6	6	-5	3	5	-5	-1	2	2	40	140	60	170	.63	.07	18	1.3	.4		540	280		.9	.14	.40
vijayarajan	194319	45	m	40	34	64	43	n	n	h	h	h	.6	.8	.75	175	105	80	I	480	320	.33	5	6	-2	-2	.8	2	5	-3	-3	1	2	2	2	-2	4	6	-5	3	2	-3	-3	-2	3	50	150	60	110	.83	.08	13	1.0	.3		590	270		1.2	.12	1.00
kochuraman			m	53	44	42	40	h	a	n	n	n	.5	.6	.83	180	82	130	I	350	220	.59	6	10	-6	-3	1.0	6	4	-2	-4	3	2	2	3	-2	5	3	-4	3	3	-2	-2	1	1B	40	130	40	70	.60	.09	22	1.0	.8		480	260		.8	.08	.50
sreedharan KP			m	56	41	60	25	a	d	n	n	h	.6	.7	.85	207	93	110	I	370	250	.46	6	8	-6	-4	-3.0	7	4	-2	3	3	-2	7	5	-4	6	3	-4	7	7	-4	2	2	-3	60	80	60	100	.75		20				480	230		1.1	.10	.67
georgejacob	202830	66	m	42	25	71	55	h	h	n	n	n	1.1	1.2	.90	205	93	70	I	420	220	.91	7	6	-5	-3	1.0	7	5	-3	3	7	-3	4	8	-5	4	7	-4	3	6	-3	-1	-1	7	60	110	70	120	.55	.06	18	1.2	.6		490	270		.8	.16	.60
jalaludden	9508256	45	m	58	49	38	39	h	a	n	n	n	.8	.6	1.27	111	87	80	I	390	300	.30	4	6	-8	-1	-2.0	14	1	-12	8	7	-7	9	3	-5	15	2	-6	6	2	-5	-4	-1	1B	50	160	80	120	.67	.05	16	.8			500	230		1.2	.19	.10
vincentnetto	9605675	57	m	56	47	40	35	h	h	n	n	n	.5	.7	.71	270	110	100	I	430	270	.59	6	6	-2	-3	-4.0	1	1	-1	9	3	-2	3	3	-3	6	2	-5	3	4	-7	1	3	-2	40	80	50		1.00	.12	14	1.5	.8		560	210		1.2	.08	1.50
cruz	203387	70	m	46	28	78		n	n	n	n	n	.8	1.0	.80	208	70	160	I	530	270	.96	5	9	-5	-5	-1.0	4	8	-6	3	6	-6	5	9	-7	5	6	-6	4	5	-5	3	6	-3	50	130	40	130	.56	.06	19	1.0	.4		490	260		.9	.16	1.00
sreedharan c r	203377	50	m	59	44	60		h	n	n	n	n	.9	1.1	.82	200	80	160	I	560	280	1.00	4	8	-5	-2	-.5	3	6	-4	-3	-1	3	3	2	-5	2	5	-2	-3	-1	2	20	40	50	190	.50	.04	17	2.0	1.0		520	270		.9	.23	.40
sadanandan tha	9809861		m	60	43	48	48	a	a	n	n	n	.8	.9	.89	220	100		I	540	260	1.08	8	10	-7	-3	1.0	8	3	-5	3	4	1	3	6	3	8	7	-8	4	3	-3	1	-1	-2	60	60	50	130	.80	.10	25	1.3	1.3	540	540	260		1.1	.10	.43
r.mohankumar	203792		m	49	39	51		h	n	n	n	n	.8	.9	.80	175	173	90	I	450	310	.45	4	7	-7	1	1.0	17	13	-12	-2	3	10	5	3	-3	10	4	-5	6	6	-6	-1	-1	1	130	170	40	110	.57	.05	18	.3	.2	550	550	280		1.0	.19	-.14
janardhanan ac	198649	60	m	46	32	67		a	a	n	n	n	.7	1.1	.64	240	95	150	I	520	240	1.17	4	12	-10	-3	3.0	-6	5	1	-11	-4	10	0	0	0	0	0	#	9	-4	-11	-2	-1	6	60	70	60	120	.33	.06	26	.7	.6	500	500	220		1.3	.18	.30
sivadas k v	204144	55	m	48	27	83		n	n	h	h	n	.7	1.0	.70	204	93	120	I	550	260	1.12	6	7	-6	-1	1.0	3	-4	-4	-3	-2	4	0	0	0	0	0	-4	4	-2	3	-2	-4	3	60	150	60	80	.86	.09	19	1.0	.4	510	510	250		1.0	.12	.17
shereef	203813	46	m	40	30	56		n	n	n	n	n	1.0	.8	1.31	122	87	80	I	340	230	.46	15	9	-11	-4	2.0																			60	60	20	110	1.67	.15	35	2.5	2.5	480	480	290		.7	.07	.36
kunnalan	202368	50	m	44	23	80		n	n	n	n	n	.4	.8	.50	167	210	130	I	520	240	1.17	3	5	6	2	1.0																			160	120	50	90	.60	.08	14	.2	.3	570	570	340		.7	.13	.33
preman	204260	55	m	44	23	80		n	n	n	n	n	.7	.8	.88	250	70	110	I	570	290	.97	9	8	-8	-6	2.0	2	5	-3	4	-5	5	0	0	0	0	0	3	2	-5	-2	-3	3	3	50	110	40	110	1.13	.13	25	1.8	.8	500	500	290		.7	.08	.75
ahamed k m	204520	50	m	55	48	38		a	a	n	n	n	.5	.7	.71	140	100	120	I	590	260	1.26	3	-5	-3	2	3.0	3	-2	2	4	-2	4	0	0	0	0	0	2	3	-6	2	3	3	-3	70	50	50	90	.43	.06	15	.4	.6		580	260		1.3	.17	-.67
padmanabhan s	204568	68	m	51	30	60		n	n	n	n	n	.7	1.0	.70	133	93	150	I	360	240	.50	7	11	-7	-2	2.0	3	8	-4	3	3	-1	0	0	0	0	0	-4	5	9	-5	-2	-3	4	90	90	110	130	.04	.10	25	.8	.8		490	270		.8	.10	.29
krishnan nair	203225	52	m	61	48	52,48		h	a	a	h	h	.6	.8	.77	138	122	90	I	420	270	.56	6	4	-5	-1	-2.0	14	3	-5	5	1	-2	0	0	0	0	0	-2	-2	1	1	-2	-1	1	60	70	40	170	1.50	.10	15	1.0	.9		530	310		.7	.10	.20
rosy mathew	9500512	77	f	44	26	78		n	n	n	n	n		.7	.90	460	130	130	I	660	410	.66	2	5	-4	-3	1.0	2	4	-2	-5	-3	-3	0	0	0	0	0	1	3	1	-4	-2	-2	3	50	160	80	180	.40	.03	11	.4	.1		610	310		1.0		.75
shibili m	6807455	53	m	52	43	26		n	n	n	n	n	.5	1.0	.55	94	116	110	I	500	200	1.30	3	8	-8	-3	1.0	3	8	-4	5	6	-5	0	0	0	0	0	-4	3	5	-5	-5	-6	5	40	60	20	120	.38	.06	17	.8	.5		530	200		1.7	.17	.50
younus kunju	9704410	58	m	61	39	74		n	n	h	h	h	.9	1.9	1.00	170	134	90	I	460	320		5	5	-4	-4	-2.0	4	7	-5	2	3	-3	0	0	0	0	0	-2	3	2	1	-3	-3	5	50	110	40	210	1.00	.06	14	1.0	.5		580	290		.5	.18	1.00
hamsa	201390	65	m	50	35	55		n	n	n	n	n	.8	1.1	1.00	155	111	80	I	540	270		8	7	-8	-3	1.0	5	7	-6	-1	-2	1	0	0	0	0	0	-6	3	1	-4	-2	-2	2	110	120	40	120							510	300		.7	.10	.38
mariamma thon	204799	69	f	44	26	67		n	n	n	n	n	.6	1.0	.60	200	93	110	I	650	300	1.17	-9	6	12	-3	3.0	4	9	-6	4	7	-4	2	4	-3	3	4	-3	5	4	-3	-2	-3	3	50	80	80	130	50	.10	27	.1	.1		580	320		.8	-.07	-.25
pushpangathan	205587	60	m	48	28	79		n	n	n	n	n	.8	.8	1.00	175	140	90	I	500	320	.56	9	7	-5	-2	-2.0	6	8	-5	-2	-2	3	7	4	-5	9	5	-5	3	5	-3	3	3	-2	90	120	40	180	1.29	.11	21	1.0	.8		600	360		.7	.09	.40
mohdsharif	205582	26	m	56	37	70	43						.9	.5	1.80	134	90	146	I	430	240	.79	9	4	-5	-2	-3.0	7	3	-2	2	5	-3	5	3	-6	3	2	-7	3	5	-5	-5	-9	4	50	80	60	80	2.25	.10	18	1.8	1.1		540	270		1.0	.10	.40
sasi			m	54	39	63		n	n	n	h	h	1.1	.8	1.36	137			N	460	300	.53	13	8	-7	-1	2.0	2	4	-2	4	1	-1	6	-1	-3	1	3	-5	6	3	-4	4	-1	-1	90	60	80	120	1.62	.12	28	1.4	2.2		470	150		2.1	.08	.14
prabhakaran	201343	63	m	57	44	52		n	n	n	h	a	.8	.5	1.60	146	64	170	N	460	300	.53	3	4	-4	-3	.7	7	4	-4	3	2	-2	8	5	-1	2	2	-3	2	2	-3	-4	-2	3	40	40	110	90	.75	.04	11	.8	.8		420	160		1.3	.27	.75
uthamannair	201808	41	m	52	38	59		n	n	n	a	h	.8	.6	1.33	196	86	290	N	440	260	.69	6	8	-7	-5	.8	3	6	-3	2	7	-4	5	9	-7	9	6	-6	6	2	-3	2	1	0.6	60	120	60	120	.75	.08	21	1.0	.2		540	280		.9	.13	.71
narayanakurup	201635	60	m	45	28	76		n	n	n	n	h	1.1	.6	1.83	175	70		N	370	270	.37	9	7	-9	-1	-1.0	7	4	-4	5	3	-2	5	6	-5	9	4	-5	7	3	6	-4	-2	4	60	90	50	70	1.29	.08	25	1.1	1.0		500	300		.7	.12	.11
sukumaran	9406625	61	m	46	26	82		n	n	n	n	n	.8	.6	1.43	170	90	90	N	500	320	.56	8	7	-6	-3	1.0	3	6	-5	9	6	-3	4	3	-4	7	2	-4	5	4	-7	3	3	-2	60	90	60	130	1.14	.10	21	1.3	.9		600	330		.8	.10	.50
krishnan	202183	50	m	43	24	76		a	h	n	n	n	1.0	.7	1.46	160	100	120	N	530	300	.77	7	8	-8	4	3.0	6	5	-5	6	5	-5	2	2	2	3	4	-5	3	4	-5	2	-1	2	60	90	80	80	.88	.07	21	1.2	.8		560	270		1.1	.14	-.67
joseph	9707844	49	m	50	27	76		n	n	n	n	h	.9	.5	1.80	113		160	N	510	270	.89	10	5	-6	-2	4.0	9	4	-4	2	2	-2	4	4	-4	7	5	-6	2	3	-2	1	-2	2	60	70	30	130	2.00	.11	21	1.3	1.4		630	310		1.0	.09	.33
sankaranmk	202366		m	48	34	64		n	h	h	n	n	.9	.6	1.50	190	70	150	N	350	210	.67	9	4	-4	-3	1.0	4	3	-3	6	3	-2	-4	2	3	3	3	-3	-3	2	4	1	3	-4	30	140	60	80	.70	1.00	25	1.4	.5		600	310		.4	.10	.75
ishak k	202467		m	54	36	71	52	n	a	n	n	n	.8	.8	1.00	140	93	100	N	540	230	1.34	7	8	-5	-2	2.0	4	5	-3	3	4	-2	4	6	-5	7	3	-5	5	5	-4	2	3	-1	50	120	50	70	.88		20				480	250		.9	.11	.40
sreedharapanick	202748		m	41	27	72		n	n	n	n	h	.7	.7	1.00	125	110	140	N	480	320	.50	6	6	-5	-2	-.8	5	2	-2	5	3	-1	6	2	-2	9	4	-3	6	7	-4	4	2	1	60	70	40	90	1.00	.09	17	.8	.9		620	330		.9	.12	.40
murugan	202749	55	m	54	37	24	24	d	d	d	n	h	1.0	.8	1.16	165	76	100	N	400	270	.48	6	4	-6	-1	3.0	6	3	-4	3	3	-4	11	2	-4	3	2	-9	4	1	-13	-2	-2	2	110	70	40	410	1.50	.06	16	.6	1.5		670	320		1.1	.17	.17
thomas v t	203333	33	m	55	43	54		n	n	n	h	h	1.1	.7	1.60	160	88	120	N	480	240	1.00	7	6	-5	-2	-1.0	6	5	4	-4	1	5	6	3	-5	5	4	-4	3	-1	-4	-6	-4	5	50	80	40	170	.06	.05	18	.1	1.4		550	290		.9	.16	.40
lekshmikutty	186290	69	f	41	30	63		n	n	h	n	h	1.3	1.2	1.08	159	93	80	N	490	300	.63	3	6	-5	-1	-1.0	4	5	-3	-4	-2	4	3	4	-2	2	1	-1	4	-2	-3	-6	-2	6	40	140	80	80	.08	.08	14	.8	.2		530	300		.8	.43	.16
omanakuttan	203545	50	m	54	33	77		n	n	n	n	n	.9	.8	1.17	180	110	110	N	400	280	.43	6	7	-7	-2	-3.0	7	5	-4	5	0	4	2	2	-3	3	3	-3	5	4	-3	-6	-4	2	60	80	50	180	.86	.07	19	1.0	.2		550	300		.8	.15	.29
babyissac	203130	38	m	58	48	36	35	h	h	n	n	n	.8	.6	1.33	100	105	90	N	360	230	.57	6	6	-3	-1	-1.0	12	12	-7	13	13	-8	5	2	-4	9	3	-5	11	11	-13	-4	-1	3	60	40	40	180	1.00	.08	15	1.0	1.5		520	310		.7	.13	.33
vilasini	203554	58	f	40	32	35	29	a	h	a	n	n	.9	.7	1.30	70	160	50	N	310	220	.41	8	9	-8	-2	3.0	6	2	-2	2	2	-4	2	3	-3	3	3	-5	2	3	-3	3	1	-1	40	20	40		1.60	.09	16	2.0	4.0		460	260		.8	.11	.67
sadasivan	203608	79	m	61	49	49	58	n	n	n	a	a	1.3	1.0	1.25	170	110	60	N	420	290	.45	4	6	-5	-4	4.0	4	3	-3	1	2	-4	7	4	-3	3	2	-2	4	3	-3	-2	4	-5	40	70	60		.67	.03	15	1.0	.6	540	540	410		.3	.31	-1.33
robin thomas	201579	17	m	45	15	75		n	n	n	n	n	.9	.7	1.34	190	93	100	N	470	270	.74	12	5	-5	-4	1.0	8	3	-5	-6	-2	5	5	3		5	2	-5	10	3	-8	-4	-2	4	60	120	50	80	.75	.13	26	2.0	1.0	480	480	270		.8	.08	.56
rasalamma	201070	50	f	49	28	81		n	h	a	n	n	.8	.8	1.00	186	116	120	N	430	280	.54	8	6	-6	-3	1.0	14	11	-6	3	3	-4	4	5	-4	3	3	-2	10	10	-9	1	2	-2	60	110	20	180	1.33	.10	19	1.3	.7	550	550	320		.7	.10	.20
vijayan	9109191	36	m	52	32	65		n	n	n	n	n	1.0	.6	1.67	160	134	140	N	450	300	.50	14	7	-13	-2	1.0	11	12	-13	1	3	-4	15	4	-9	15	5	-8	16	16	25	-2	-2	2	140	60	70	100	2.00	.14	34	1.0	2.3	540	540	270		1.0	.07	.15
elizebeth thoma	204096	63	f	53	34	72		n	n	n	n	n	1.1	1.1	1.00	130	76	120	N	580	260	1.23	10	6	-6	1	2.0																			50	150	150	190	1.67	.09	22	2.0	.7	600	600	310		.9	.11	-.17
mohammed isn	9901233	67	m	49	38	52		h	h	a	n	n	.8	.7	1.20	220	80	120	N	670	340	.97	7	5	-7	-1	3.0	6	8	-6	-3	-2	2	3	5	-3	7	4	-5	3	3	-4	-4	5	-4	70	120	60	220	1.40	.09	19	1.0	.6	670	670	350		.9	.11	.14
kunhikannan	9902617	53	m	40	35	57		n	n	n	n	h	.4	.5	.80	170	52	140	N	510	290	1.22	7	5	-6	-3	1.0	2	3	-2	3	3	-3	0	0	0	0	0	-6	4	3	-3	-2	-1	2	100	60	60	110	1.40	.18	20	.7	.9	510	510	260		1.0	.06	.38
vikraman	203019	50	m	54	30	74		n	n	n	n	n	.9	.7	1.29	200	160	90	N	480	300	.60	8	5	-4	-5	1.0																			130	70	50	110	1.60	.08	17	.6	1.1	610	610	330		.9	.11	1.25
divakaran pillai	203999	50	m	51	33	72		n	n	n	n	h	.8	.6	1.33	170	180	100	N	420	300	.47	9	9	-10	3	3.0	7	6	-5	3	4	-2	0	0	0	0	0	-3	7	6	-7	2	3	-4	50	110	90	120	1.13	.11	27	1.8	.8	590	590	290		1.0	.09	-.30

d pillai	203999	50	m	51	33	72		n	n	n	n	h	.9	.7	1.29	180	80	180	N	560	290	.93	11	10	-13	-1	2.0	5	8	-4	5	5	-5	0	0	0	0	0	-7	5	6	-8	3	4	-3	110	90	50	70	1.10	.12	34	1.0	1.2	540	540	270		1.0	.08	.08
devakiamma	203415	60	f	32	18	75		n	n	n	n	n	.9	.9	1.00	160	110	140	N	550	370	.49	7	6	-7	-1	-1.0																			120	90	60	130	1.17	.08	20	.6	.8	640	640	320		1.0	.13	.14
velappan nair	9600363	66	m	61	44	40	40	n	n	n	h	a	1.1	.7	1.70	167	116	120	N																																										
sukumaran nair	192798	50	m	62	49	50		a	a	n	n	n	.8	.5	1.65	160	95	120	N	570	290	.97	4	6	-5	-2	1.0	4	2	-4	-3	1	3	0	0	0	0	0	-4	4	1	-4	-3	-1	2	50	60	40	110	.67	.05	15	.8	.7	550	550	270		1.0	.20	.40
remasan p	204517	64	m	56	46	38		a	a	n	n	n	.8	.8	1.07	106	93	80	N	360	220	.64	3	9	-4	-2	-1.0	3	4	-2	-2	-1	2	0	0	0	0	0	-4	5	-5	2	-1	-1	1	50	60	30	120	.33	.04	16	.6	.5		430	190		1.3	.27	.50
varkey varghes	203061	70	m										.9	.8	1.09	153	70	90	N	370	270	.37	10	10	-9	-6	3.0																			90	110	50	100	.97	1.08	12	1.1	8.8		470	260		.8	.09	.67
ramesh	204517	64	m	56	46	38		a	a	n	n	n	.9	.7	1.40	111	93	110	N	340	200	.70	5	5	-5	-1	-1.0	3	5	-1	1	5	-0.0	0	0	0	0	0	-5	5	2	-3	1	1	-1	80	50	80	80	1.00	.06	15	.6	1.0		480	210		1.3	.18	.20
mohanan pillai	204482	40	m	43	31	61		a	n	n	n	n	.9	.8	1.13	200	81	90	N	480	270	.78	10	5	-7	1	1.0																			110	90	60	100	2.00	.11	22	.9	1.1		580	300		.9	.09	-.14
yesudasan	204201	47	m	40	29	56		n	n	n	n	n	.8	.6	1.33	216	60	120	N	550	320	.72	9	5	-6	1	1.0	6	5	-3	-4	-2	3	0	0	0	0	0	-4	3	-1	-3	-4	-2	3	90	110	60	80	1.80	.11	20	1.0	.8		550	300		.8	.09	-.17
vijayan n	205394	51	m	49	28	72		n	n	n	n	n	1.0	.9	1.20	160	76	150	N	570	330	.73	8	7	-7	-1	1.0	8	4	-4	4	3	-2	0	0	0	0	0	-5	5	5	-5	-4	-3	4	60	90	30	130	1.14	.08	22	1.3	.9		600	300		1.0	.13	.14
karunakaran un	205432	70	m	53	40	55	34	h	h	n	n	n	.9	.6	1.50	130	220	90	N	500	330	.52	10	4	-6	-3	-1.0	6	3	-5	-4	-3	3	0	0	0	0	0	-5	3	3	-9	-1	-1	1	80	80	90	90	2.50	.11	20	1.3	1.3		620	330		.9	.09	.50
vijayalekshmi	204522	48	f	45	33	62		n	n	n	n	h	.9	.9	1.10	170	128	90	N	500	300	.67	5	9	-5	-1	-2.0	4	7	-4	-8	-7	4	0	0	0	0	0	-2	3	2	-3	-4	-2	2	50	90	70	150	.56	.06	19	1.0	.6		570	270		1.1	.18	.20
sasidhara kurup	205551	46	m	46	28	68		n	n	n	n	n	1.1	.9	1.20	172	111	100	N	420	270		8	9	-9	-2	-1.0	7	7	-5	3	3	-3	0	0	0	0	0	-6	7	3	-5	-3	-2	2	70	80	20	110							590	330		.8	.14	.22
jayan b	205584	41	m	58	44	56	33	a	a	n	n	n	.5	.6	.90	86	110	60	N	380	200	.90	4	7	-6	-5	-1.0	2	3	-3	3	2	-3	5	6	-6	1	5	-7	5	4	-4	-2	2	-9	80	50	40		.57	.08	17	.5	.8		470	190		1.5	.13	.83
ratheesan J	203467	40	m	44	28	73		n	n	n	n	h	.7	.9	.84	130	169	100	N				9	1	-7	-2	1.0	6	6	-5	4	3	-4	2	3	-3	5	2	-3	5	2	-4	3	3	-5	120	60	50	120	9.00	.12	17	.8	1.5		540	320		.7	.08	.29
edwin	192189	49	m	45	26	74		n	n	n	n	n	1.0	.9	1.08	168	70	130	N	370	250	.54	10	8	-7	-1	-1.0	7	5	-6	4	4	-3	8	7	-8	8	7	-7	5	5	-4	-3	-2	1	110	100	40	100	1.25		25	.9	1.0		460	260		.8	.10	.14
kochuhasankun	205564	70	m	42	29	67		n	n	n	n	n	.8	.8	1.00	145	111	110	N	510	320	.59	5	7	-7	-1	-1.0	4	-5	-4	5	6	-7	5	6	-7	6	4	-5	3	3	-3	4	4	-8	100	100	50	100	.71	.06	19	.5	.5		580	330		.8	.16	.14
ahmedrawther	9808173	62	m	52	34	72		n	n	n	n	n	.9	.9	1.00	129	128	80	N	470	210	1.24	8	7	-6	-1	-.7	2	4	-2	-1	-2	1	2	3	-3	3	2	-3	2	2	-1	-1	-1	1	80	130	80	250	1.14	.09	21	1.0	.6		600	360		.7	.11	.17
ravi	205583	49	m	47	31	72		n	n	n	n	n	1.0	.6	1.70	103	122	50	N	480	260	.85	6	8	-5	-1	-1.0	4	5	-3	-2	2	2	3	4	-4	6	6	-5	3	7	-3	-3	-3	3	90	110	70	130	.75	.06	19	.7	.6		580	310		.9	.17	.20
susheela	9809856	66	f	52	38	63		n	n	n	n	h	1.1	.8	1.30	140	93	120	N	430	270	.59	8	7	-7	-2	-1.0	6	4	-5	-3	-6	4	3	2	-3	3	2	-3	6	5	-5	-2	-5	2	60	80	60	190	1.14	.08	22	1.3	.9		570	300		.9	.14	.29
Unni E P	206228	62	m	51	35	60		n	n	n	n	n	1.1	.7	1.60	146	82	80	N	540	270	1.00	11	5	-6	-2	2.0	5	4	-5	3	-3	2	3	4	-2	2	3	-3	3	-2	-3	-4	-3	3	60	60	40	200	2.20	.10	22	1.4	1.8		550	280		1.0	.10	.33
hameed kunju	201268	60	m	53	47	31		n	n	n	a	h	.9	.3	2.50	90	70		R	400	310	.29	5	2	-4	-2	1.0	7	5	-6	-7	-2	2	3	2	-3	2	-1	2	3	2	-3	-8	-2	3	50	80	40	110	2.50	.05	11	1.0	.6		500	190		1.6	.17	.50
jisha jose	199183	23	f	53	47	32	28	n	n	n	n	n	.9	.4	2.14	90	70	270	R	500	200	1.50	6	1	0	-1	.6	4	2	-4	-3	-1	2	2	1	-2	3	1	-3	-2	-1	3	-6	-1	5	70	40	60	120	.85	1.50	7	.9	1.5		590	150		2.9	.15	3.50
prakasam	201901	49	m	50	40	43		a	d	a	n	n	.8	.5	1.60	128	47	70	R	510	300	.70	6	4	-5	-7	7.0	4	5	-2	-2	-2	3	8	2	-3	1	2	-5	3	4	-4	2	3	-1	50	90	60	110	1.50	.08	15	1.2	.7		510	280		.8	.13	1.40
xavierraj	202066	43	m	62	50	36		h	a	h	n	h	1.1	.3	3.75	128	116	80	R	310	160	.48	7	2	-4	-1	-2.0	3	1	-2	-7	-2	2	6	2	-4	15	8	-5	5	-2	3	2	1	-4	80	80	60	130	3.50	.07	13	.9	.9		470	260		.8	.16	.25
chellappan pillai	27649	75	m	64	45	66		a	a	n	n	n	1.3	.4	3.25	120	105	60	R	400	250	.60	5	3	-4	-1	-.9	2	5	-2	2	2	-3	3	5	-3	5	2	-4	5	2	-4	2	3	-2	70	40	80	90	1.67	.04	12	.7	1.3		540	250		1.2	.26	.25
mohd.moideen	190923	55	m	56	48	34	32	a	a	h	h	h	.9	.5	2.00	80	130	100	R	420	220	.91	3	4	-3	-2	-1.0	2	1	-1	-2	-2	1	6	-1	-3	9	5	-3	2	-2	2	1	1	1	70	40	40	160	.75	.03	10	.4	.8		550	300		.8	.30	.67
sadasivan	9102477	62	m	56	47	40	.35	a	a	h	h	h	.9	.8	1.12	165	111	90	R	460	250	.84	5	3	-5	-2	3.0	5	5	-5	-4	1	-7	2	1	-2	4	2	2	4	4	2	-2	3	-2	80	60	50	390	1.67	.06	13	.6	.6		670	130		4.2	.17	.40
antony philip	202275		m	58	46	47	31	a	a	n	n	n	1.3	.4	4.85	129	122	40	R	470	260	.81	6	2	-5	-1	-1.0	12	6	-7	6	4	-7	6	3	-5	9	1	-5	5	4	-9	-3	-1	-1	80	60	70	130	3.00	.05	13	.8	1.0		530	250		1.1	.21	.20
sivankuuly	203105	35	m	51	37	60	42	a	a	h	n	n	1.0	.4	2.90	130	110	140	R	400	270	.48	6	3	-4	-2	-1.0	9	5	-8	6	6	-6	3	2	-4	11	2	-7	8	6	-6	1	2	1B	70	30	50	90	2.00	.07	13	.9	2.0		510	230		1.2	.17	.50
nesayyan	203291	65	m	42	27	63		n	n	m	n	n	1.1	.7	1.60	210	93		R	390	220	.77	10	6	-7	-7	1.0																			80	120	60			.09	23	1.3	.8		570	290		.8	.11	1.00
antony philip	202275			58	46	47		h	a	h	n	n	1.0	.4	2.70	110	50	150	R	410	230	.78	4	3	-4	-1	.7	5	2	-4	-4	3	4	3	5	-3	11	2	-5	2	2	-9	1	1	2	30	30	60	110	1.33	.04	11	1.3	1.3		480	240		1.0	.25	.25
subbaih	203543		m	51	42	47	30	a	a	h	n	n	.8	.6	1.25	120	100	130	R	350	180	.94	5	6	-6	-1	1.0	2	3	-1	-3	-3	3	7	4	-5	11	4	-7	1	1	-7	-1	-1	1	50	80	70	160	.60	.04	11	.6	.4		570	180		2.2	.16	.17
abiraman s	204917	47	m	51	42	44		n	n	n	n	n	.8	.4	2.00	130	53	140	R	340	220	.55	6	3	-4	-3	1.0	-6	4	3	-5	3	1	0	0	0	0	0	-3	3	-2	-3	-5	1	1	50	40	60	130	2.00	.08	14	1.2	1.5		390	250		.6	.13	.75
ramaswamy pil	205471	59	m	50	41	44	32	h	h	n	n	n	1.0	.4	2.80	118	99	50	R	340	200	.41	6	5	-2	-1	-3.0	2	4	-1	-2	-2	2	0	0	0	0	0	-3	-2	-1	3	-2	-1	3	50	40	30	190	1.20	.06	13	1.2	1.5		480	200		1.4	.17	.50
sasikumar	1	45	m	44	32	61		n	n	n	n	n	1.0	.8	1.10	196	93	60	N	430	270	.59	12	7	-8	-3	1.0	10	7	5	4	3	4	15	4	4	13	5	5	5	3	-4	-3	-3	2	100	80	40	80	1.70	.13	27	1.2	1.5		520	300		.7	.08	.38
Jacob Thomas	2	56	m	43	28	72		n	n	n	n	n	.8	.7	1.14	260	76	110		530	270	.46	9	4	-6	-5	3.0																			110	100	50	90	2.25	8.90	19	.8	.9		470	280		.7	.09	.83
Thankachan	3	55	m	47	32	69		n	n	n	n	n	.7	.8	.88	290	60	160		500	290	.72	7	11	-5																					50	120	50	170	.64	.10	23	1.4	.6		530	250		1.1	.10	
Anil	4	34	m	43	30	66		n	n	n	n	n	1.0	.6	1.67	115	70	120		480	260	.85	12	7	-11																			-6	2	110	80	60	90	7.71	.12	30	1.1	1.5		460	310		.5	.08	
Dinesh	5	27	m	54	38	65		n	n	n	n	n	.6	.4	1.50	250	90	140		500	250	1.00	13	5	-12																			-4	4	80	90	30	110	2.60	.22	30	1.6	1.4		540	270		1.0	.05	
Krishnakumar	6	30	m	50	34	70		n	n	n	n	n	.6	.5	1.10	110	93						13	5	9																			-7	2	50	90	40	120	2.60	.22	27	2.6	1.4		540	270		1.0	.05	
Harish	7	18	m	43	26	77		n	n	n	n	n	1.0	.5	1.89	118	134	80		443	198	1.24	12	3	-9																			-3	2	110	80	70	80	4.00	.12	24	1.1	1.5		570	290		1.0	.08	
Ganeshan	8	33	m	50	32	73		n	n	n	n	n	1.0	.6	216.00	2	105	120		-380	-260	.46	9	7	5																			-3	-1	80	70	30	100	1.29	.09	21	1.1	1.3		520	290		.8	.11	
Vijayamma	9	57	f	49	27	85		n	n	n	n	n	.9	.9	.95	230	93	130		440	280	.57	6	8	-5																			-3	9	110	90	50	90	.75	.07	19	.6	.7		620	310		1.0	.15	
Sonny	10	37	m	49	31	75		n	n	n	n	n	.7	.5	1.37	156	105	130		380	230	.65	9	8	10																			-4	3	110	90	50	40	1.13	.12	27	.8	1.0		510	270		.9	.08	
Usha	11	38	f	40	25	77		n	n	n	n	n	1.0	.8	1.25	135	87	110		370	250	.48	14	6	-9																			-2	1	110	80	40	80	2.33	.14	29	1.3	1.8		480	290		.7	.07	
Mathachan	12	47	m	44	32	61		n	n	n	n	n	1.0	.8	1.10	196	93	60	N	430	270	.59	12	7	-8	-3	1.0	10	7	5	4	3	4	15	4	4	13	5	5	5	3	-4	-3	-3	2	100	80	40	80	1.70	.13	27	1.2	1.5		520	300		.7	.08	.38
Sivan	13	59	m	43	28	72		n	n	n	n	n	.8	.7	1.14	260	76	110		530	270	.46	9	4	-6	-5	3.0																			110	100	50	90	2.25	8.90	19	.8	.9		470	280		.7	.09	.83
Abhiraman	14	38	m	43	30	66		n	n	n	n	n	1.0	.6	1.67	115	70	120		480	260	.85	12	7	-11																			-6	2	110	80	60	90	7.71	.12	30	1.1	1.5		460	310		.5	.08	
Aliyar	15	38	m	49	31	75		n	n	n	n	n	.7	.5	1.37	156	105	130		380	230	.65	9	8	10																			-4	3	110	90	50	40	1.13	.12	27	.8	1.0		510	270		.9	.08	
Selvi	16	42	f	40	25	77		n	n	n	n	n	1.0	.8	1.25	135	87	110		370	250	.48	14	6	-9																			-2	1	110	80	40	80	2.33	.14	29	1.3	1.8		480	290		.7	.07	

Sm+Em+Am	normal	25	22.6400	5.6633	15.7958	.0000
	impairedrel	39	19.5263	5.4858		
	restrictive	14	12.8000	3.5091		
	Total	78	19.3143	6.1938		
IVR_VEL	normal	25	.6846	1.9731	.2389	.7882
	impairedrel	39	.2513	1.8624		
	restrictive	14	.4571	2.4295		
	Total	78	.3803	1.9891		
EM-ACT	normal	25	86.8000	26.0960	4.9545	.0095
	impairedrel	39	67.9487	30.2776		
	restrictive	14	62.8571	15.8980		
	Total	78	73.0769	28.2984		
EM-DCT	normal	25	87.2000	18.1475	14.4520	.0000
	impairedrel	39	111.0256	37.9608		
	restrictive	14	60.7143	27.5860		
	Total	78	94.3590	36.1307		
AM-DCT	normal	25	50.4000	14.2829	.9638	.3861
	impairedrel	39	56.4103	20.9620		
	restrictive	14	56.4286	13.3631		
	Total	78	54.4872	17.8484		
EM/ACT	normal	25	1.1362	.4624	1.2875	.2821
	impairedrel	39	.9643	.5391		
	restrictive	14	.9207	.2812		
	Total	78	1.0128	.4790		
EM/DCT	normal	25	1.3808	1.6066	4.2244	.0184
	impairedrel	39	.6314	.4587		
	restrictive	14	1.0604	.4595		
	Total	78	.9613	1.0431		
E/Em	normal	25	10.89	5.49	6.6493	.0022
	Impairedred	39	12.85	6.46		
	restrictive	14	18.11	5.42		
	Total	78	13.17	6.41		

mTeia'	normal	25	518.4000	58.2151	.1166	.8901
	impairedrel	39	524.6154	50.6196		
	restrictive	14	525.7143	65.9504		
	Total	78	522.8205	55.3594		
b'	normal	25	280.8000	38.3970	5.6354	.0052
	impairedrel	39	265.1282	49.7823		
	restrictive	14	228.5714	51.5688		
	Total	78	263.5897	49.5165		
mTEIa'-b'/b'	normal	25	.8672	.2301	5.3266	.0069
	impairedrel	39	1.0511	.4923		
	restrictive	14	1.4771	1.0074		
	Total	78	1.0686	.5926		

1-Doppler tissue myocardial velocities were obtained in 100% of cases.

2-The amplitude of Doppler Sm velocities decreased progressively from normal control subjects to patients with restrictive filling with P value not significant. F=.3831,P=.6830

3.Em velocities were significantly different between the normal group and the diastolic dysfunction groups. Standard Doppler E velocity has a bimodal distribution, while Em is reported to have a unimodal distribution, unaffected by preload, decreasing significantly and steadily from normal to impaired relaxation to restrictive. This was not seen in our patients- the Em velocities were changed from 3.3257 +/- .6651 in the normal group to 3.6153 +/- .5789 in the impaired relaxation group to 1.5898 +/-.4249 cm/s in the restrictive group significantly (F=10.6227,P= .0001)

4.Peak myocardial velocities during atrial contraction (Am) were significantly different between the normal group and the diastolic dysfunction groups .Am increased from 6.4+/- 1.9579 in normal subjects to 6.8462 +/- 2.9606 cm/s in patients with delayed relaxation but were reduced in patients with restrictive filling (3.357+/- 1.4991 cm/s,F=10.6689 P=.0001).

5.The ratio Em/Am was maximum in the normal group, and minimum in the delayed relaxation group with P significant. F=5.6091P=.0054.

This was because the Em velocity was also maximum in the normal group, and minimum in the delayed relaxation group .

6.The E/Em was significantly different between the 3 groups,progressively increasing from the normal group to increasing grades of diastolic dysfunction. .F= 6.6493 P= .0022

7.The value of(Sm + Em +Am) was maximum in the normal group and decreased progressively as the gradation of diastolic function worsened. F=15.7958 P<.0001

8.The isovolumic phase relaxation velocity(IVR velocity) was not significantly different between the 3 groups. F=.2389,P=.7882 .

9.The Em –acceleration time decreased progressively from the normal group to the restrictive filling group. F=4.9545 P= .0095 Change in this variable was therefore associated with gradation of severity of diastolic dysfunction.

10. The Em –deceleration time increased from 87.2000 +/-8.1475 cm/s in normal subjects to 111.0256 +/-37.9608 cm/s in patients with delayed relaxation and then was reduced in patients with restrictive filling 60.7143 +/-27.5860 cm/s (F=14.4520, $P < .0001$).

11. The Am –deceleration time was 57.56+/-23.76 ms and was not significantly different in the 3 groups.F=.9638 P= .3861

12.The Em/acceleration time (Em/ACT) decreased progressively from normal to the restrictive group,but was not significantly different in the 3 groups.F=1.2875 P=.2821.

13. The Em/ deceleration time (Em/DCT) velocities decreased from 1.3808 +/- 1.6066 in the normal group to .6314 +/- .4587 in the impaired relaxation group and increased to 1.0604 +/- .4595 cm/s in the restrictive group significantly (F=4.2244, P= .0184)

14. The modified Tei index increased progressively from .8672 +/-.2301 in the normal group to 1.0511 +/- .4923 in the impaired relaxation group to 1.4771 +/-1.0074 in the restrictive group .F=5.3266 P= .00695 Change in this variable was therefore associated with gradation of severity of diastolic dysfunction.

II.RESULTS-SYSTOLIC FUNCTION-

The mean and standard deviation of all the tested variables along with their P value are mentioned in Table IV. **TABLE IV-ANOVA-SYSYTOLIC FUNCTION**

VARIABLE	GROUPS	N	Mean	Std.Devn	F	P
Sm	EF<35%	13	-4.1692	1.9593	.1861	.8308
	EF35-50%	16	-4.1250	2.3058		
	EF>50%	25	-4.9200	6.3961		
	Total	54	-4.5037	4.5882		
IVC-VEL	EF<35%	13	-2.1308	1.3060	1.0993	.3432
	EF35-50%	16	-2.0313	1.9348		
	EF>50%	25	-2.9231	1.8010		
	Total	54	-2.3381	1.7284		
IVC/SM	EF<35%	13	.7897	.8863	2.1601	.1289
	EF35-50%	16	.3039	.6207		
	EF>50%	25	.4354	.2387		
	Total	54	.4950	.6561		
TimeQRSToSm	EF<35%	13	189.0000	114.5474	7.7175	.0013
Peak	EF35-50%	16	117.8571	34.0087		
	EF>50%	25	100.0000	39.3700		
	Total	54	123.2653	68.8412		
Sm+Em+Am	EF<35%	13	13.7077	3.4657	19.0698	.0000
	EF35-50%	16	16.0000	3.5777		
	EF>50%	25	22.6400	5.6633		
	Total	54	18.5222	6.0390		
E/Em	EF<35%	13	15.9900	5.4300	6.1936	.0039
	EF35-50%	16	16.7000	6.3700		
	EF>50%	25	10.8900	5.4900		
	Total	54	13.8400	6.2800		
mTEI_-a'	EF<35%	13	543.0769	69.4484	1.1696	.3187
	EF35-50%	16	510.6250	50.9207		
	EF>50%	25	518.4000	58.2151		
	Total	54	522.0370	59.2844		

mTEIb'	EF<35%	13	216.1538	54.8541	9.8894	.0002
	EF35-50%	16	275.0000	43.5125		
	EF>50%	25	280.8000	38.3970		
	Total	54	263.5185	51.1438		
mTEI a-b/b	EF<35%	13	1.6569	.9640	11.2826	.0001
	EF35-50%	16	.8975	.2705		
	EF>50%	25	.8672	.2301		
	Total	54	1.0663	.6066		

1.**Sm:** The mean systolic velocity by tissue Doppler was not statistically different between the three groups. (F=.1861 P= .8308).

2.**IVC-Vel** :The isovolumic contraction phase velocity by tissue Doppler was also not statistically different between the three groups (F=1.0993 P= .3432).

3.**IVC-Vel / Sm**: There was no statistically significant difference in the ratio of the IVC-velocity and Sm (F=2.1601 P= .1289)

4.**Q to peak of Sm** :This variable increased progressively from the normal group to the EF 35 to 50group to the EF < 35 group significantly (F=7.7175 ,P= .0013). Change in this variable was therefore associated with gradation of severity of systolic dysfunction.

5.**Sm + Em + Am:** This derived variable decreased from its maximum value in the normal EF group to its minimum value in the EF < 35 group highly significantly (F= 19.0698 P<.0001).Change in this variable was therefore associated with gradation of severity of systolic dysfunction.

6.**E / Em**:The value varied significantly among the groups,with no clear relationship with increasing severity of systolic dysfunction. .F= 6.1936,P= .0039

7. **Modified Tei Index-**

The modified Tei index by tissue Doppler (F=11.2826 ,P=.0001) and its component b'(F=9.8894 ,P=.0002) were significantly different between the three groups.The modified Tei index increased and the component b' decreased progressively with increasing grades of systolic dysfunction.The component variable a' was not statistically significantly different between the three groups.

III.TISSUE DOPPLER AT THE LATERAL MITRAL ANNULUS AND DIRECTLY OVER THE LATERAL MYOCARDIAL SEGMENT IN SHORT AXIS VIEW-

TABLE IV

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	M_ANN-Em	6.32	91	2.97	.31
	LWEM	3.95	91	2.77	.29
Pair 2	AM	6.14	91	2.62	.27
	Am	3.07	91	3.40	.36
Pair 3	SM	-5.56	91	3.03	.32
	SM	-3.26	91	4.50	.47

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	M_ANN-Em & LWEM	91	.209	.047
Pair 2	AM & Am	91	.283	.007
Pair 3	SM & SM	91	-.014	.898

Em at lateral mitral annulus correlated weakly and positively with lateral segment tissue Doppler. Em corelation is 0.209 and P= 0.047.

Am at lateral mitral annulus correlated weakly and positively with lateral segment tissue Doppler. Am corelation is 0.283 and P= 0.007.

Sm at the two sites did not correlate.

IV.WALL MOTION ABNORMALITIES-

Independent sample t-test was done for each myocardial segment comparing the variables –Sm,Em and Am-in patients with WMA in that particular segment versus patients with no WMA in that segment(or any segment).As already shown in this study,there was correlation between tissue Doppler at the lateral mitral annulus and that directly over the lateral segment(in Em and Am).Em ,Am and Sm in each segment was taken with the cursor directly over the said segment.

The mean and standard deviation of all the tested variables along with their P value are mentioned in Table V.

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Am at lateral mitral annulus correlated weakly and positively with lateral segment tissue Doppler. Am corelation is 0.283 and P= 0.007.

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Independent sample t-test was done for each myocardial segment comparing the variables –Sm,Em and Am-in patients with WMA in that particular segment versus patients with no WMA in that segment(or any segment).As already shown in this study,there was correlation between tissue Doppler at the lateral mitral annulus and that directly over the lateral segment(in Em and Am).Em ,Am and Sm in each segment was taken with the cursor directly over the said segment.

The mean and standard deviation of all the tested variables along with their P value are mentioned in Table V.

TABLE V-t-tests for Independent Samples

SEGMENT	NOWMA	NOWMA	WMA	WMA		
	MEAN	SD	MEAN	SD	STD.ERROR	P Value
IVS NO:	29		33			
Em	5.1724	3.714	4.8485	3.701	0.943	0.733
Am	5.7586	3.313	4.2121	2.484	0.738	0.040
Sm	-4.6552	2.919	-3.2727	2.254	0.658	0.040
AW NO:	29		38			
Em	-2.1379	2.761	0.0789	2.655	0.666	0.001
Am	-1.2345	3.114	0.7605	1.88	0.613	0.002
Sm	1.9655	2.771	-0.4118	3.276	0.772	0.003
LW NO:	29		17			
Em	4.1724	3.095	2.2353	2.969	0.932	0.043
Am	3.2793	3.85	1.8235	2.698	1.062	.177
Sm	-2.7931	5.747	-2.1765	4.707	1.647	0.71
IW NO:	29		28			
Em	3.0866	3.234	3.7218	3.092	0.839	0.452
Am	2.7683	2.359	2.615	2.446	0.636	0.811
Sm	-2.9382	2.655	-2.6511	1.797	0.606	0.637
PW NO:	29		15			
Am	2.4534	2.224	3.0047	2.585	0.748	0.465
Sm	-4.1034	2.2257	-2.9333	2.314	0.734	0.114
Em	3.7424	3.644	3.0740	2.857	1.082	0.540

I. OF THE (IVS) INTERVENTRICULAR SEPTUM-

1.Em:mean em at the ivs in apical 4 chamber view in patients with and without ivswma was not significantly different.

2.Am:mean am at the ivs in apical 4 chamber view in patients with ivswma was significantly different from that in patients with no wma.4.21+/- 2.48 and 5.76+/- 3.31 p=0.040

3.Sm: sm at the ivs in apical 4 chamber view in patients with ivswma was significantly different from that in patients with no wma.-3.27+/-2.25 and -4.66+/-2.91 p=0.040

2. AWWMA AND NO WMA-INDEPENDENT SAMPLE t-test

1.Em:mean em at the aw in short axis view in patients with awwma was significantly different from that in patients with no wma. 0 .0789 +/- 2.655 and -2.1379 +/- 2.761 p=0.001

2Am:mean am at the the aw in short axis view in patients with awwma was significantly different from that in patients with no wma. 0.7605 +/- 1.880 and -1.2345 +/- 3.114p=0 .002

3.Sm: sm at the the aw in short axis view in patients with awwma was significantly different from that in patients with no wma. -0.4118 +/-3.276 and 1.9655 +/- 2.771 P=0.003

3. LWWMA AND NO WMA-INDEPENDENT SAMPLE t-test

1.Em:mean em at the lw in short axis view in patients with lwwma was significantly different from that in patients with no wma 2.2353 +/- 2.969 and 4.1724 +/- 3.095 p=0.043

2.Am:mean am in patients with and without lwwma was not significantly different.

1.8235 +/- 2.698 and 3.2793 +/- 3.850 p=0.177

3.Sm: sm in patients with and without lwwma was not significantly different.

- 2.1765 +/- 4.707 and -2.7931+/- 5.747 p=0.710

4.IWWMA AND NO WMA-INDEPENDENT SAMPLE t-test

Mean Em, Am and Sm in patients with and without iwwma was not significantly different.

p= 0.45, 0.81 and 0.637 respectively

5.PWWMA AND NO WMA-INDEPENDENT SAMPLE t-test

Mean Em, Am and Sm in patients with and without pwwma was not significantly different.(p= 0.54, 0.465 and 0.114 respectively.)

V.MODIFIED TEI INDEX-

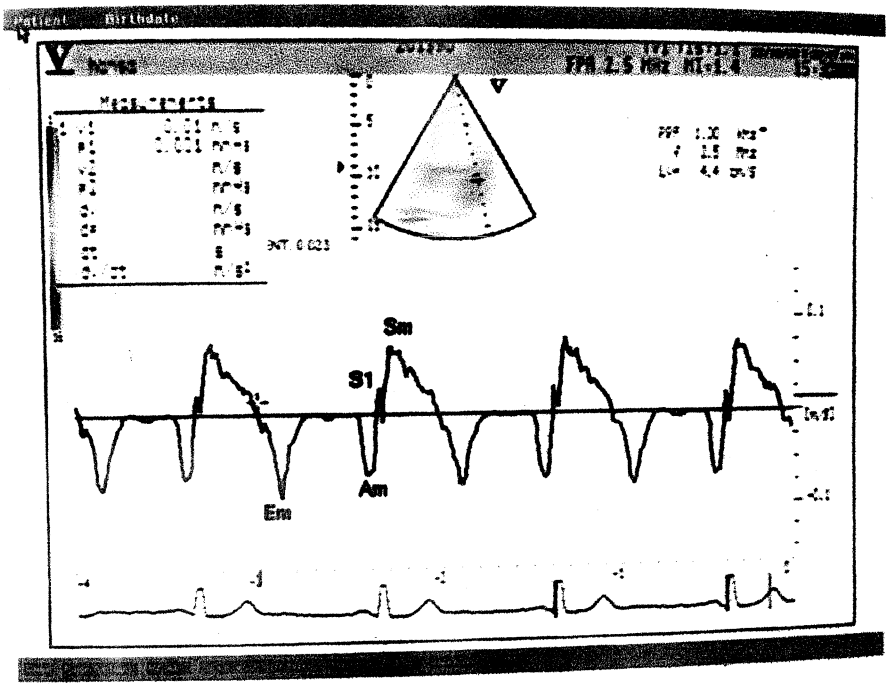
Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	TEI_-a & mTeia'	97	.432	.000
Pair 2	B & b'	97	.305	.002
Pair 3	a-b/b & a'-b'/b'	94	.106	.309

Paired Samples Test

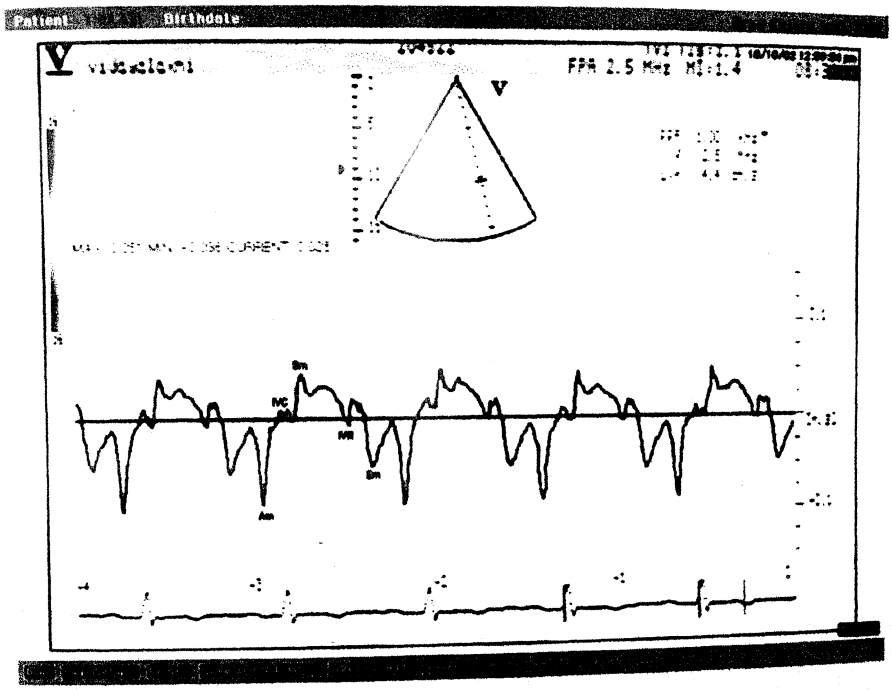
		Paired Differences					t	df	Sig. (2-tai
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	TEI_-a - mTeia'	-80.72	76.00	7.72	-96.04	-65.40	-10.461	96	.
Pair 2	B - b'	-4.02	56.34	5.72	-15.38	7.33	-.703	96	.
Pair 3	a-b/b - a'-b'/b'	-.3410	.5771	5.952E-02	-.4592	-.2227	-5.728	93	.

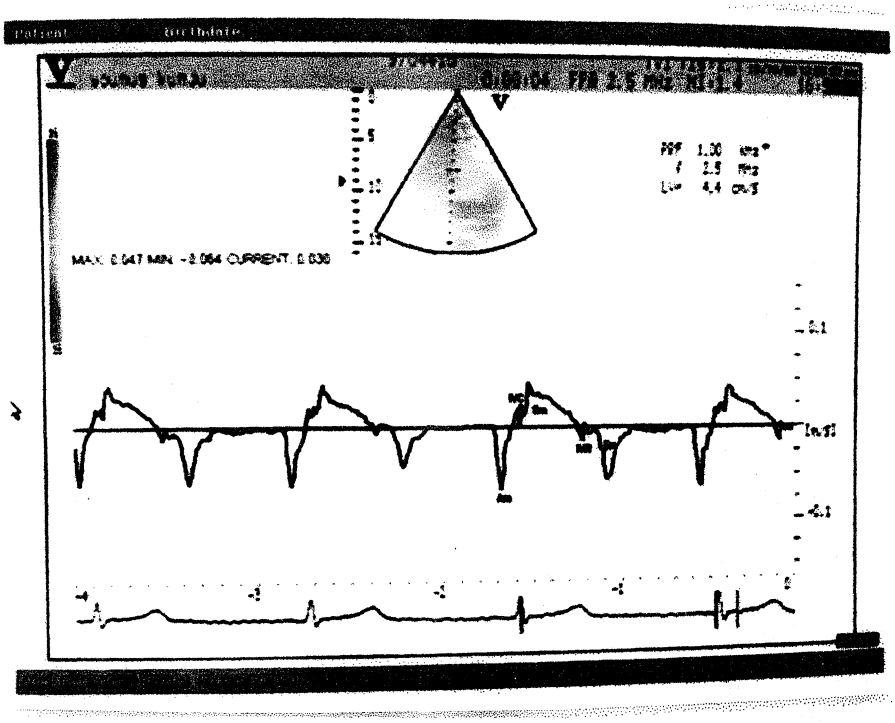
The mean values of the tei and the modified tei indices were significantly different .There was no statistically significant relation between these 2 indices.



NORMAL TISSUE DOPPLER WAVE PATTERN IN A PATIENT

TISSUE DOPPLER –DIASTOLIC DYSFUNCTION :Em < Am





DISCUSSION-

In this prospective study, we have tested the utility of tissue Doppler in the assessment of diastolic function,systolic function and wall motion abnormalities. We also examined the utility of a new index of global left ventricular function-the modified Tei index. We also looked for correlation between standard tissue Doppler parameters measured at the lateral mitral annulus, and the same parameters measured with the cursor directly over the lateral myocardial segment.

DIASTOLIC FUNCTION BY TDI-

Standard Doppler Flow Indexes

Carlos et al [36] have shown that all Doppler indices of transmitral and PV flow present a parabolic distribution during progression from normal to advanced diastolic dysfunction. Although careful analysis of several combined standard Doppler flow indexes in addition to 2-dimensional echocardiographic and clinical data permits the correct assessment of diastolic function in many patients, it is frequently inconclusive in others. In our study, similar to Farias's study, the peak E velocity had a bimodal distribution. Similarly, the peak A was highest in patients with diastolic dysfunction. Also, the E- deceleration time was significantly different between the three groups and highest in the group with impaired relaxation . However, in difference from Farias's study, the E/A ratio and the IVRT was not significantly different between the three groups. We also studied a little studied variable "the A-deceleration time " which was not significantly different between the three groups.Tenenbaum [37] has shown that that the Doppler derived a deceleration time is am important predictor of the left ventricular end diastolic pressure .

There was no significant difference in the Tei 's index (F=.8224,P= .44350) between the three groups.

Doppler Myocardial Velocities

1.Em and Am-

The results of our study indicate that early diastolic myocardial velocities and peak myocardial velocities during atrial contraction measured by DTE are significantly changed in patients with abnormal diastolic function.

It has been reported earlier that in contrast to standard Doppler indexes of

LV filling, Doppler myocardial velocities show a steady decline from normal to advanced diastolic dysfunction and do not revert during stages characterized by

increased preload compensation. However, in our series, this was true for most indices except Em. Em was maximum in the impaired relaxation group and minimum in the restrictive group. Em therefore was not unimodal as reported earlier .

Several studies have correlated Em with indices of diastolic function. Oki and coworkers[42] noted that Em correlated inversely with the fall in LV pressure during early diastole (peak negative dP/dt) or the time constant of LV relaxation (tau). Ohte et al [40] evaluated 50 patients with CAD and 8 patients without coronary disease with M-mode color TDI of the inferoposterior LV wall at the mitral annulus.Tissue Doppler imaging measurements were compared with LV volumes (angiography) and pressures obtained by catheter-tip micromanometry. The Em correlated inversely with peak negative dP/dt and LV end systolic volumes. These studies confirm that Em is related both to LV relaxation and elastic recoil.

Additional work by Sohn et al [41] also demonstrated a correlation between Em and tau, a load-independent measure of relaxation. In a later publication, Em was an accurate measure of abnormal LV diastolic function, despite the presence of atrial fibrillation.[43]

However, the concordance observed between diastolic myocardial velocities in the axial plane and ventricular inflow patterns is disrupted in various disease states. In patients with restrictive cardiomyopathy, low Em values have been found in the presence of high pulsed Doppler E velocities and frequently an Em/Am ratio less than 1 in patients with pseudonormalized or restrictive filling (E/A ratio >1). Similar discordance between LV filling ratios and myocardial velocities may be found in patients with hypetrophic, dilated, and hypertensive cardiomyopathies. [38]

More recently, Oki et al [39] recorded transmitral flow and myocardial Doppler velocities and measured tau in patients undergoing cardiac catheterization.

The time constant (tau) correlated well with IVRT and other various parameters calculated from transmitral flow Doppler except in those patients with elevated LV end-diastolic pressure who had a pseudonormal filling pattern. In contrast, all patients with heart disease who had abnormal LV relaxation (prolonged tau) had a low peak early myocardial (Em) velocity. Moreover, tau correlated well with Em in all subjects regardless of their LV filling pressure.These findings have suggested that Em velocity provides a relatively preload-independent assessment of LV relaxation.

In a recent study, Sohn et al[55] measured myocardial velocities in normal volunteers and in patients with delayed relaxation and pseudonormal LV filling as assessed by

Doppler mitral inflow variables and invasive hemodynamic measurements. In patients with a mitral Doppler inflow pattern of delayed relaxation, the infusion of volume resulted in a change toward pseudonormalization, with shortening of the E-wave deceleration time and increased E/A ratio. However, in the same patients, Em velocity did not change significantly with this maneuver. On the other hand, patients with normal or pseudonormal filling at baseline demonstrated a significant reduction in mitral inflow E velocity and E/A ratio after nitroglycerin infusion but no significant change in Em velocity, confirming that this parameter is less sensitive to preload changes.

In a preliminary study ,a cutoff pulsed Doppler Em velocity of 8 cm/s allowed complete separation between patients with constriction and restriction. These results have been recently confirmed in a larger, prospective cohort .

Ohte et al[40] noted that in older patients (mean age 60 years), all of whom had coronary arteriography to detect CAD and a mitral Doppler E/A ratio <1, Em was lower in those with significant coronary obstruction than in those patients with chest pain but no CAD. The Em provided clear separation of patients with CAD and prolonged tau, compared with patients without CAD. End-systolic volume index and ejection fraction was lower in CAD patients versus those without obstructive lesions. Thus a relation likely exists between impaired LV systolic function and diastolic relaxation and has also been described in patients with dilated cardiomyopathy evaluated by TDI[4]

Am --

Carlos et al [36] have reported that peak myocardial velocities during atrial contraction (Am) were similar in normal subjects and in patients with delayed relaxation (11.0 ± 2.1 vs 11.7 ± 2.4 cm/s) but were reduced in patients with pseudonormal filling (8.5 ± 2.7 cm/s) and those with restrictive filling (3.9 ± 1.3 cm/s, $P < .001$).We found that Am increased mildly from the normal to delayed relaxation group,but was reduced in the restrictive group(P-significant).

2.Em/Am

Since the Em was bimodal in distribution, this ratio was maximum in the normal group, and minimum in the delayed relaxation group with P significant .

In patients with restrictive cardiomyopathy low Em is seen in the presence of either low or high pulsed Doppler E velocities and frequently a Em/Am ratio <1 in patients with pseudonormalized or restrictive filling (E/A ratio >1)

A fall in the ratio of the myocardial Em velocity to Am velocity with age has also been described.[44] Garcia-Fernandez et al [45] observed a reduced Em and Em/Am ratio in a subset of patients with significant CAD but normal segmental LV function.

3.Sm-

is not truly an index of diastolic function .A progressive decrease was noted without statistical significance, which needs further study as to whether it reflects the link between systolic and diastolic function in myocardium.

4.E/Em-

The E/Em was significantly different between the 3 groups,progressively increasing from the normal group to increasing grades of diastolic dysfunction.We are not aware of any other study that compared this variable in different degrees of diastolic function.

The mitral E wave velocity is directly influenced by left atrial pressure and inversely altered by changes in the time constant of relaxation [46,47]. So,by itself, the E wave velocity relates poorly with left atrial pressure [48-52], given that abnormal relaxation and high filling pressures commonly coexist in the cardiac patient. Logically,correcting E wave velocity for the influence of relaxation will improve its relation with left atrial pressure. Studies using the early propagation velocity of LV inflow by color M-mode echocardiography support this hypothesis. The propagation velocity behaves as an index of LV relaxation [53-55], and the ratio of E wave velocity to propagation velocity (or its inverse) relates well with mean PCWP [56]

Similarly,by dividing the E wave velocity by Em provides an alternative method to correct the transmitral velocity for the influence of relaxation. In comparison to propagation velocity, Ea is easily recorded and measured with DTI and is independent of systolic LV function, whereas propagation velocity is currently measured with different methods [53-55] and appears to have some relation to systolic performance [57].

The use of a combination of Doppler mitral inflow and DTE parameters (E/Em ratio) has been shown to improve the estimation of capillary wedge pressure.[61] Yong-Jin Kim et al showed in 200 patients that the E/E′ ratio correlated well with pre–Awave pressure (r = 0.74, P < .001), and the correlation was not dependent on the left ventricular systolic function (ejection fraction [EF] ≥50%: r = 0.74, P < .001 versus

EF <50%: $r = 0.70$, $P < .001$). The E/E′ ratio of ≥9 best discriminated elevated (>12 mm Hg) from normal left ventricular pre-A pressure with a sensitivity of 81% and a specificity of 80%.This study showed that quantitative estimation of left ventricular pre-A pressure could be suggested by the simplified equation of *pre-A pressure = E/E′ + 4* with reasonable accuracy. The ratio of early mitral inflow to early mitral annular velocity (E/E′) was therefore suggested as a useful index in the estimation of left ventricular filling pressure.[60]

Nagueh et al demonstrated good correlation between the E/E′ ratio and pulmonary capillary wedge pressure (PCWP) in 60 patients with a mitral inflow pattern of relaxation abnormality or pseudonormalization ($r = 0.87$, $P < .001$)12 and also in 120 patients with sinus tachycardia in whom mitral E and A waves were fused ($r = 0.86$, $P < .001$). In both studies, the value of >10 best predicted the PCWP of >12 mm Hg.[58,59] The ratio of mitral E-wave velocity to Em (E/Em) >/10 has been shown to be associated with elevated filling pressures.[60]

LAP = (E/Ea x 1.25) + 1.9

Similarly, several regression equations have been proposed by previous investigators to estimate left-sided filling pressures using one or more variables derived from the transmitral or pulmonary vein velocity, or both [60]. To date, there are no studies comparing the accuracy of one method with another. The 95% confidence limit (2 SD) of E/Em was 7.6 mm Hg, and thus, this method provides only an estimate of filling pressures close to that provided by several of the published equations. However, the E/Em is relatively simple to obtain and conceptually has the potential for providing a reasonable estimate of filling pressures throughout a wide range of relaxation abnormalities. Furthermore, an E/Em ratio >10 may be used to detect patients with high filling pressures.

5.Sm +Em + Am- The (Sm + Em +Am-the isovolumic contraction and relaxation velocities excluded) was maximum in the normal group and decreased progressively in group 2 togroup 3 with significant P value.

6. Change in the **Em –acceleration time** was associated with gradation of severity of diastolic dysfunction.

Oki et al [42] demonstrated a significant increase in mitral early diastolic filling acceleration time in patients with mitral E/A < 1. They measured mitral early diastolic filling acceleration time as 89 ± 11 ms in patients with mitral E/A > 1.This time interval in the cardiac cycle is the same as the interval used to determine EmDT

7.The **isovolumic phase relaxation velocity(IVR velocity) and the Am – decceleration time** were not significantly different in the 3 groups. Bolognesi et al 84 showed that Am deceleration was not significantly different between patients with coronary artery disease and diastolic dysfunction than in the control patients.

Am dec (cm/s2) 2.1 ± 0.4 and 1.8 ± 0.5 P=NS

8. As has been shown earlier for E decceleration time by standard pulse Doppler, the **Em –deceleration time** increased from normal subjects to patients with delayed relaxation and then was reduced in patients with restrictive filling significantly in our patients.

Sinan Dagdelen et al [62] studied 3 groups of patients who were proven by angiography to be free of significant coronary artery lesions (<40% stenosis) with an LVEDP < 10 mm Hg (group A: n = 24; 16 men, 18 women; mean age ± SD = 55 ± 13 years), an LVEDP of 10 to 15 mm Hg (group B: n = 21; 17 men, 4 women; mean age 56 ± 11 years), or an LVEDP > 15 mm Hg (group C: n = 35; 20 men, 15 women; mean age 58 ± 9 years). The EmDT obtained non invasively by left ventricular tissue Doppler imaging was found to be useful in the estimation of LVEDP.

Naqvi et al [63] noted in that in LVH, atrial contraction velocity(Am), Em/Am ratio, and DT of early filling (EDt) wave were the only variables that correlated with measures of LVH.

9.The **Em acceleration= Em/acceleration time (Em/ACT)** decreased progressively in the 3 groups but there was no significant difference between them.

Bolognesi et al [64] showed that Em acceleration was significantly lower and peak Em acceleration time significantly longer in patients with coronary artery disease than in control patients, whereas E acceleration and peak E acceleration times were similar between the two groups .These findings suggest that tissue Doppler has a somewhat higher sensitivity (when compared with the conventional transmitral Doppler flow) in detecting minor impairments of left ventricular filling in patients with coronary artery disease.

10. The **Em deceleration= Em/deceleration time(Em/DCT)** decreased from the normal group to the impaired relaxation group and increased in the restrictive group significantly .

Bolognesi et al [64] showed that E deceleration was significantly lower and E deceleration time significantly longer in patients with coronary artery disease than in

the control patients; the profiles of these indexes are related to left ventricular altered relaxation observed in ischemic patients.

11. The modified Tei index increased progressively from .8672 +/-.2301 in the normal group to 1.0511 +/- .4923 in the impaired relaxation group to 1.4771 +/-1.0074 in the restrictive group . The mean of this index was significantly different between the 3 groups-so, we conclude that this index is useful to distinguish between different grades of diastolic function.

One study [20] has demonstrated that the modified Tei index correlates well with the Tei index by pulsed Doppler and can be used as a simple method of assessing LV myocardial performance.The modified Tei index has the advantage of simultaneous recording of systolic and diastolic velocity patterns. Thus, the modified Tei index provides an alternative to the Tei index by the pulsed Doppler for assessing LV myocardial performance.

DISCUSSION-SYSTOLIC FUNCTION:

I.Sm: The mean systolic velocity by tissue Doppler was not statistically different between the three groups .Older studies have shown a correlation with ejection fraction .The small sample size could be the reason for this difference in our study.

Ejection fraction and Sm-

Nagueh et al [58] also reported that the lateral base Sm had a good correlation with LV ejection fraction, as determined by 2-dimensional echocardiography.

Gulati and coworkers [65] reported that pulsed TDI Sm, measured as an average from 6 sites in the apical 2- and 4-chamber views and the longaxis view,correlated well (r = 0.85) with the LV global ejection fraction.

A second study(Fukuda et al) has shown that the peak Sm along the long and short axes were all significantly lower in DCM than in the control . [66]

Alam et al [67,68] showed that the peak amplitude of mitral annular motion was significantly lower in subjects with ischemic heart disease than in healthy subjects. Furthermore, they emphasized that LVEF could be predicted to be higher than 55% (92% sensitivity, 87% specificity) in patients in whom the mean amplitude of mitral annular motion at the 4 sites in the 2D 4-chamber and 2-chamber echocardiograms is greater than 10 mm.

In addition, LVEF could be predicted to be less than 30% (92% sensitivity, 67% specificity) in patients with chronic heart failure in whom the mean amplitude of mitral annular motion was lower than 7 mm.[69]

Sm and Stress echocardiography-

Several experimental studies have been performed to define the pattern of myocardial velocities during regional ischemia and reperfusion.[70-78] All these studies have demonstrated that measures of myocardial velocity and velocity gradient with the use of TD can be applied to assess noninvasively the alterations in left ventricular function induced by either physical or pharmacologic inotropic stimulation.

DI has been used to assess myocardial velocities during pharmacologic or postexercise stress echocardiography.Gorscan et al [79] observed that systolic velocities increased with only a minimal dosage (3 microg/kg per minute) during dobutamine infusions, but without significant changes in myocardial wall thickening or LV ejection fraction by conventional 2-dimensional echocardiography.[80]

Yamada et al 82 reported in dobutamine stress echocardiography that myocardial segments with ischemia had a <90% increase in Sm from baseline to peak dobutamine dose, with 83% sensitivity and 87% specificity. A lack of increase in Sm after exercise was reported to be a sensitive indicator of ischemia in studies by Pasquet and colleagues[82,83]

This approach could hold promise for improved sensitivity in the detection of significant coronary stenosis. In addition,TDI may be helpful in studies of myocardial viability to distinguish stunned or hibernating myocardium during dobutamine infusions. Further investigations are needed to address this potentially useful application of TDI for regional and global LV systolic function.

Normal values-

Normal values of pulsed TDI-derived Sm have been reported [84,85,86]. Each of these studies has reported regional differences in the various myocardial segments analyzed. Sm recorded at the base is greater than that in the mid left ventricle or the apex. Lateral and inferior segments have higher velocities than the anterior wall and septum when obtained from apical windows. In general, values greater than 9 cm/s are considered normal.

dp/dt and Sm- Other investigators have noted that Sm, measured by pulsed TDI from the parasternal or apical approach, correlated with peak positive dP/dt and LV

ejection fraction in patients with dilated cardiomyopathy [87] and hypertensive heart disease.[88]

II.Q to peak of Sm :

In our study, this variable increased progressively from the normal group to the EF 35 to 50group to the EF < 35 group significantly.This variable could therefore be used to differentiate between gradations of systolic dysfunction.

A study on ischemic and dilated cardiomyopathy(Fukuda et al) [66] has previously shown that the mean Q-Sw was significantly longer in the MI groups than in the control group. The mean Q-Sw at all 6 sites in the ischemic and dilated cardiomyopathy groups were significantly lower and longer, respectively, than those of the control group. There were significant correlations between the EF and the means of the Q-Sw values at the sites corresponding to the infarct regions in the MI groups. In the ischemic and dilated cardiomyopathy groups,significant correlations existed between the EF and the means of the Q-Sw values at all 6 sites.

Hirotsugu Yamada et al [89] showed in 65 patients that the peak systolic velocity was lower and the time from the electrocardiographic Q wave to the peak of the systolic wave for the posterior wall was longer in the hypertensive heart disease (5.9 6 0.5 cm/sec and 215 6 21 msec, respectively), hypertrophic cardiomyopathy cardiomyopathy (6.2 6 0.9 cm/sec and 217 6 17 msec, respectively), and dilated cardiomyopathy (5.2 6 0.8 cm/sec and 235 6 26 msec, respectively) groups than in the noncardiac chest pain (7.7 6 0.9 cm/sec and 187 6 24 msec, respectively) and the ischemic heart disease (7.6 6 0.8 cm/sec and 184 6 22 msec, respectively) groups. In all groups, the peak systolic velocity and the time from the electrocardiographic Q wave to the peak of the systolic wave for the posterior wall correlated directly and inversely, respectively, with the %EF (r50.59, p<0.0001;r 5 20.59, p < 0.0001) and the peak dP/dt (r 5 0.75, p<0.0001; r520.68, p<0.0001). Both tissue Doppler variables for the ventricular septum did not correlate with the %EF but roughly correlated with peak dP/dt.

III.IVC-Vel : In a previous study, the peak Swl (isovolumetric contraction velocity) was significantly lower in the DCM group than in the control group. Also, the peak isovolumetric contraction velocity along the long axis, correlated well with the peak dP/dt in all patients.

However, the isovolumic contraction phase velocity by tissue Doppler was not found to be a useful variable in systolic function assessment in our study .

IVC velocity along the long axis reflects LV myocardial contractility during the isovolumic contraction. The increase in the peak IVC-velocity along the long axis in normal subjects reflects more profound shortening of longitudinal fibers than circumferential fibers during early systole, resulting in a more spherical change in the LV cavity. As indicated by previous reports, [7,20] circumferential fibers play a more important role in LV systolic function than longitudinal fibers during the ejection phase. Indeed, Sm along the short axis and IVC velocity along the long axis correlated well with peak dP/dt and LV ejection fraction in a study. However, because the endocardium is impaired in the early stages of disease conditions such as ischemia or hypertension, contractile abnormalities in longitudinal fibers of the endocardial layer at early systole precede those in circumferential fibers of the mid-wall [4,61]. So, recording of the IVC velocity along the long axis measured by pulsed TDI may be a useful parameter for understanding the LV contractility in patients with myocardial diseases at early stages.

IV.IVC-Vel / Sm:

There was no statistically significant difference in the ratio of the IVC-velocity and Sm

V.Sm + Em + Am:

Change in this variable was therefore associated with gradation of severity of systolic dysfunction.

VI.E / Em:

The value varied significantly among the groups,with no clear relationship with increasing severity of systolic dysfunction.

In a study, echocardiography was performed on days 1 and 5, and 1 and 3 months after a first myocardial infarction in 67 consecutive patients. Flow propagation velocity correlated well with Em ($r = 0.72$, $P < .0001$). The ratio of peak E-wave velocity (E) to flow propagation velocity also correlated well with E/Em ($r = 0.87$, $P < .0001$). Thus E/Em was closely related after myocardial infarction and appear to have similar prognostic information. [90]

VII. Modified Tei Index-

The modified Tei index increased and the component b' decreased progressively with increasing grades of systolic dysfunction(P-significant).

Harada et al [20] showed in 46 children (37 healthy children and 9 patients with heart disease) that

1. the time interval between the end and onset of mitral annular velocities (a) correlated highly with the mitral closing-to-opening time (a) (r = 0.98, p =0.0001). The a was significantly longer than the a (390 +/- 69 vs 368+/- 65 ms, p _0.001). The mean

difference between a and a was 22 +/- 14 ms (Figure 3).

2.There was an excellent correlation between the duration of the S wave (b) and LV ejection time (b) r= 0.96, p=0.0001). The b obtained from TDI was significantly longer than the b (263 +/- 40 vs 250+/- 37 ms, p =0.001). The mean difference between b and b was 13 +/- 12 ms (Figure 4).

3. The modified Tei index obtained from TDI correlated well with the Tei index by the pulsed Doppler method (r = 0.96, p=0.0001). The mean difference between the modified Tei index and the Tei index by pulsed Doppler was 0.011 +/- 0.052 . In 9 patients with heart failure, the Tei index was significantly increased compared with that in normal subjects (0.77+/- 0.19 vs 0.40 +/- 0.07, p =0.0001). Similarly, the modified Tei index in the 9 patients was significantly higher than in normal subjects (0.79+/- 0.18 vs 0.41 +/- 0.06, p =0.0001) (Figure 6). The LV ejection fraction was significantly lower in these 9 patients than in normal children .

The Tei index has earlier shown to be a promising parameter for predicting global myocardial performance, which also can be used for quantitative assessment of LV function.21-30 It has been correlated with severity and clinical outcome in patients with dilated cardiomyopathy, amyloidosis, or anthracycline-induced cardiomyopathy and for serial evaluation of patients with congenital heart disease[24,26,27]

A major limitation of assessing the Tei index is that the interval between end and onset of mitral inflow and ejection time cannot be measured simultaneously. According to a previous report[91] changes in heart rate of _10% during transmitral inflow recordings were observed in 24 of 41 infants (59%). Because of this limitation, results are probably less reliable in the presence of physiologic heart rate changes during examination. TDI as used in the present study can simultaneously

record systolic and diastolic mitral annular velocities. In contrast to the pulsed Doppler method, the time interval between the end and onset of diastolic annular velocities and the duration of the S wave can be measured on the same cardiac cycle. However, the timing and length of mitral annular velocities during diastole and systole may differ from those of mitral inflow and LV outflow.

In Harada's study, the time interval from the end to the onset of mitral annular diastolic velocities was 6.0% longer than the mitral closing-to-opening time. The mechanism of this prolongation was that the late diastolic wave of TDI usually ended before the end of mitral late diastolic flow, despite the fact that early diastolic waves seen in TDI and mitral inflow begin simultaneously, as reported previously[92] Similarly, the duration of the S wave was 5.3% longer than the LV ejection time, because the S wave ends later than the LV outflow wave, despite the fact that the S and LV outflow waves begin at the same time .

In conclusion, this study demonstrated that the modified Tei index is an useful and simple method of assessing LV myocardial performance. The modified Tei index has the advantage of simultaneous recording of systolic and diastolic velocity patterns.

Thus, the modified Tei index provides an alternative to the Tei index by the pulsed Doppler for assessing LV myocardial performance.It increases progressively with increasing severities of both systolic and diastolic dysfunction.

IV.TISSUE DOPPLER AT THE LATERAL MITRAL ANNULUS AND DIRECTLY OVER THE LATERAL MYOCARDIAL SEGMENT

Em and Am at the lateral mitral annulus correlated weakly and positively with the lateral segment tissue Doppler Em and Am respectively.However,Sm at the 2 sites did not correlate.

Most of the previous studies on WMA have utilized TDI at the corresponding site of the mitral annulus.Our study shows possibly for the first time a weak but positive correlation between 2 Doppler parameters at these 2 sites—lateral mitral annulus and directly over the lateral myocardial segment.This could mean that there is a correlation between the long axis function of the heart and localized segmental contraction.

V.WALL MOTION ABNORMALITIES-

Tissue Doppler analysis has been mainly used in the assessment of regional systolic ventricular wall motion in patients with coronary artery disease. In fact, coronary artery disease is a process typically characterized by regional rather than global left ventricular dysfunction. Most of the previous studies on WMA have utilized TDI at the corresponding site of the mitral annulus.Ours is possibly the first study that has studied wall motion with the cursor directly over myocardial segments.

In our study,the myocardial tissue velocities at the inferior and posterior segments had no relation with corresponding wall motion abnormalities.However,all the myocardial tissue velocities-Em,Am and Sm –at the anterior wall were different in patients with AW-WMA compared to those with no wall motion.Em alone at the lateral wall ,and Am and Sm at the interventricular septum were also significantly different in patients with the corresponding wall motion abnormalities.We feel that tissue Doppler was less sensitive at the inferoposterior segments because most patients had only hypokinesia at these sites,while in other segments more severe WMA like akinesia and dyskinesia were present.Further studies are recquired to clarify the role of this modality in the objective assessment of diastolic function.

None of the other studies have taken tissue Doppler velocities directly over the myocardial segments.

1.Palmes P P [90] compared myocardial velocity profiles were taken at the annulus, basal, mid, and apical segments of the septal and lateral walls in the apical view between normal segments in healthy subjects and abnormal segments in patients with MI. The Sm and Em velocities and the Em/Am ratio were significantly reduced in the abnormal segments in patients with MI.He concluded that TVI objectively quantifies directional and incremental changes in myocardial movement that are useful in evaluating global and regional myocardial function, and it may play a role in the detection of early myocardial ischemia.

2. Fukuda et al [66] studied 45 patients with wall asynergies, 3 with ischemic cardiomyopathy, 8 with dilated cardiomyopathy, and 15 healthy control subjects.He showed that the mean Sw at the sites corresponding to the infarct regions was significantly lower and the mean Q-Sw was significantly longer in the MI groups than in the control group. The mean Sw and Q-Sw at all 6 sites in the ischemic and dilated cardiomyopathy groups were significantly lower and longer, respectively,

than those of the control group. There were significant correlations between the EF and the means of the Sw and Q-Sw values at the sites corresponding to the infarct regions in the MI groups. In the ischemic and dilated cardiomyopathy groups, significant correlations existed between the EF and the means of the Sw and Q-Sw values at all 6 sites.

These findings were subsequently confirmed in other studies.[90]

3. In an early study by Bach et al[91] TDI was found to be a sensitive indicator of myocardial function These investigators reported that Sm obtained from short-axis views in patients who had evidence of hypokinesis by conventional 2-dimensional imaging before percutaneous transluminal angioplasty in a stenotic vessel promptly increased after reperfusion.These increases in Sm after angioplasty were thought to be the result of hyperemia and increased catecholamine response.

4.Recent work by Derumeaux and colleagues [92,93] with pulsed and color TDI in 2 separate animal models showed that blunting of the Sm occurred during acute ischemia.After reperfusion, recovery of Sm was observed in the infarct zones, but velocities did not return to baseline values.

5.No WMA on 2-D Echo with coronary stenosis-Palmes et al [90] demonstrated that decreases in Sm can be recorded in the lateral segments of patients with evidence of significant left circumflex coronary artery disease, even though they have normal wall motion by 2-dimensional echocardiography.

Regional diastolic function-

Studies have shown that a regional variation in LV diastolic myocardial velocities exists; the variation is similar to that seen in systolic velocities and is dependent on the segment evaluated.[94,95]The Em is greater in lateral, inferior, and posterior segments than in the anterior and septal segments, and normal values are considered to be greater than 9 cm/s (Table 2).The Em is greater at the base than at the mid left ventricle, whereas the Am is more uniform throughout the left ventricle. However, the Am is slightly lower in the mid left ventricle compared with the basal LV segments.

LIMITATIONS-

Diastolic dysfunction-

1.In the current study, patients were classified on the basis of established clinical and echocardiographic grounds alone,[96] without invasive confirmatory data. Previous studies have shown the validity of these combined criteria in selected groups of patients. The important finding in this study is that despite the fact that we used standard Doppler indexes to classify these patients, indices obtained by DTE emerged as strong discriminating factors. In clinical practice, this would aid in establishing the diagnosis in those patients in whom the standard Doppler data alone may be inconclusive. One example would be patients after heart transplantation in whom a high E/A ratio, short

DT, and blunted S could be either normal because of the young age of the donor heart or could represent diastolic dysfunction in a pseudonormal pattern. In this particular example, LA dimension would also be increased in both cases. It has been reported that

Doppler-derived estimation of capillary wedge pressure with mitral and PV inflow patterns cannot be used to reliably predict capillary wedge pressure in heart transplantation recipients.[97] In another study, the use of a combination of Doppler mitral inflow and DTE parameters (E/Em ratio) improved the estimation of capillary wedge pressure.[98]

OtherLimitations—

1.The effect of varying preload conditions, heart rate and atrioventricular conduction on these variables remains to be rigorously tested.

2.The impact of variable transducer and pulse repetition frequencies needs to be studied to determine whether the suggested range of normal values is comparable among different ultrasound systems. Automated algorithms are being developed to facilitate these measurements.

3.The major limitation of TDE measurements is their angle dependency. Although several algorithms, such as the measurement of myocardial velocity gradients, have been suggested to correct for the Doppler beam angle of incidence, the application of this technique is still limited to interrogating a limited number of myocardial segments from the different acoustic windows. It has been particularly problematic to study the apical myocardial segments with this technique.

CONCLUSIONS-

I.DIASTOLIC DYSFUNCTION-

.Myocardial velocities emerge as new indexes of diastolic function, providing additional value over mitral or PV flow indexes. The results of our study indicate that early diastolic myocardial velocities and peak myocardial velocities during atrial contraction measured by DTE are significantly changed in patients with abnormal diastolic function.

Alone or in combination with standard Doppler and 2-D echocardiographic indexes, Doppler myocardial velocities permit the differentiation between normal subjects and patients with different degrees of diastolic dysfunction regardless of the preload compensatory stage.

Therefore pulsed TDI provides a superior method for the identification of patients with impaired LV relaxation, regardless of the mitral inflow velocity flow pattern measured by conventional pulsed Doppler

Parameters found to be useful in diastolic function in our study were-

.1.Em velocities were significantly different between the normal group and the diastolic dysfunction groups but did not have a unimodal distribution as previously reported.

2.Peak myocardial velocities during atrial contraction (Am)

3.The ratio Em/Am.

4. Sm + Em +Am

5. Em –acceleration time

6.The Em –decceleration time

7.The E/Em

8.The Em/decceleration time(Em/DCT)

9. The modified Tei index.

Parameters not found useful in diastolic function-

1.The Doppler Sm velocities

2.The isovolumic phase relaxation velocity(IVR velocity)

3.The Am –decceleration time

4.The Em/acceleration time(Em/ACT)

II.This method can be applicable for measurements of **global LV systolic function**, and it provides additional information on regional abnormalities

Parameters found useful in systolic function assessment-

1.Q to peak of Sm :

2.Sm + Em + Am:Change in this variable was therefore associated with gradation of severity of systolic dysfunction.

3.The modified Tei index and the left ventricular ejection time(b')

4.E / Em:The total mean value varied significantly among the groups

Parameters not found useful in systolic function assessment-

1.Sm: The systolic velocity by tissue Doppler

2.IVC-Vel

3.IVC-Vel / Sm: The ratio of the IVC-velocity and Sm

III.The mean values of the Tei and the modified Tei indices were significantly different .There was no statistically significant relation between these 2 indices.

IV. In our study,the myocardial tissue velocities at the inferior and posterior segments had no relation with corresponding wall motion abnormalities.However,all the myocardial tissue velocities-Em,Am and Sm –at the anterior wall were different in patients with AW-WMA compared to those with no wall motion.Em alone at the lateral wall ,and Am and Sm at the interventricular septum were also significantly different in patients with the corresponding wall motion abnormalities.We feel that tissue Doppler was less sensitive at the inferoposterior segments because most patients had only hypokinesia at these sites, while in other segments more severe WMA like akinesia and dyskinesia were present.

We conclude that Doppler tissue imaging is an objective method of assessing wall motion abnormalities especially in the septal.anterior wall and lateral wall segments.

V. Most of the previous studies on WMA have utilized TDI at the corresponding site of the mitral annulus. Our study shows possibly for the first time a weak but positive correlation between 2 Doppler parameter at these 2 sites—lateral mitral annulus and directly over the lateral myocardial segment.

FUTURE DIRECTIONS

Areas of future research might include the establishment of normal values in children and elderly patients, and the effects of conduction disturbances (bundle branch block) on global and regional TDI myocardial velocities.

We conclude that myocardial velocities can be obtained easily by pulsed Doppler techniques in most patients and should be incorporated into the noninvasive echocardiographic assessment of diastolic function.

GLOSSARY (Figures 1,2 and 3)

Standard Doppler Flow Indexes

1.Peak E velocity

2.Peak A velocity

3. E/A ratio

4. IVRT –isovolumic relaxation time-from the end of the aortic velocity signal to the onset of the mitral early filling velocity signal.

5.E-deceleration time

6.A- deceleration time

7. Tei 's index -This index is defined as (a - b)/b, where

- a is the interval between end and onset of the mitral inflow, that is ,it is the closing to opening time in the left ventricular inflow.
- b is the ejection time of left ventricular (LV) outflow (Figure 2)

Doppler Myocardial Velocities

Measured values-

1. Sm velocity or Sw2-a positive wave

2. The isovolumic contraction "IVC" or Sw1 velocity-preceedes Sw2 or Sm

3. Isovolumic relaxation (IVR) velocity

4. Rapid-filling period characterized by a negative wave ("Em")

5. Filling caused by atrial contraction, represented by a second negative wave ("Am").

6. Em –acceleration time –time from the baseline at the beginning of the Em wave to its peak .

7. Em –deceleration time-time from the peak of the Em wave to the baseline .

8. Am –deceleration time time from the peak of the Am wave to the baseline

9.Q to peak of Sm time –measured from the onset of the q wave in the electrocardiogram to the peak of the Sm(or Sw2) wave.

Derived values-

1. The ratio Em/Am

2. The E/Em :the ratio of early diastolic pulse Doppler mitral filling velocity to the early diastolic tissue velocity at the lateral mitral annulus by tissue Doppler .

3. Em/acceleration time(Em/ACT)
4. Em/decceleration time(Em/DCT)
5. Sm + Em +Am
6. IVC-Vel / Sm: The ratio of the IVC-velocity and Sm
7. The modified Tei index-

The time interval between the end and the onset of mitral annular velocities during diastole (a') minus the duration of the S wave (b') divided by b' (i.e., (a'-b')/b') (Figure 3) may approximate the Tei index obtained by the conventional Doppler method

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