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TITLE T.T.T ASSESMENT LMLASTENOSIS

DM CARDIOLOGY

MADURAI MEDICAL COLLEGE

AUGUST 2010

ASSESMENT OF LEFT MAIN CORONARY STENOSIS BY TRANSESOPHAGEAL ECHOCARDIOGRAPHY

INTRODUCTION

Quantitative evaluation of coronary stenosis is clinically important. Quantitative coronary angiography is usually performed for estimating the severity of coronary stenosis. Intracoronary blood flow velocity measurements using Doppler catheters or Doppler ultrasound guide systems have also been proposed as an alternative method for evaluating the functional severity of coronary stenosis at baseline as well as for assessing the results of interventional procedures . Johnson et al. demonstrated, in a canine model, that the cross-sectional area (CSA) of the coronary stenosis can be calculated with a Doppler catheter using the continuity equation, which was originally introduced for measuring stenotic valve area More recently, Nakatani et al. showed, in 13 patients with mild to moderate stenosis, that application of the continuity equation to Doppler catheter measurement of coronary flow velocity can be used to successfully compute the severity of coronary stenosis. These methods, however, remain invasive, requiring cardiac catheterization, and cannot be repeated without risk during serial follow-up studies. Furthermore, in a consecutive series of 52 patients undergoing percutaneous transluminal coronary angioplasty, Di Mario et al. found that, although the percent CSA stenosis derived from the intracoronary guide wire Doppler measurements based on the continuity equation were significantly correlated with the corresponding quantitative angiographic measurements, this determination could be achieved

in only 16% of cases. Recently, it has been demonstrated that coronary blood flow velocity can be recorded in the proximal part of the left coronary artery (LCA) with the use of transesophageal Doppler echocardiography (TEDE). In the present study, we tested whether the percent reduction of CSA of the stenosis can be quantitated by TEDE using the continuity equation.

REVIEW OF LITERATURE:

Discussion

The epicardial coronary artery system consists of the left and right coronary arteries, which normally arise from ostia located in the left and right sinuses of Valsalva, respectively In about 50% of humans a "third coronary artery" ("conus artery") arises from a separate ostium in the right sinus. The left main (LM) coronary artery ranges in length from 1 to 25 mm before bifurcating into the left anterior descending (LAD) and left circumflex (LC) branches. The LAD coronary artery measures from 10 to 13 cm in length, whereas the usual nondominant LC artery measures about 6 to 8 cm in length. The dominant right coronary artery (RCA) is about 12 to 14 cm in length, before giving rise to the posterior descending artery(PDA) The subepicardial coronary arteries run on the surface of the heart embedded in various amounts of subepicardial fat. Portions of the epicardial coronary arteries may dip into the myocardium ("mural artery " or "tunneled artery") and be covered for a variable length (1 to several mm) by ventricular muscle ("myocardial bridge"). However, the coronary artery size was greater in the male patients as compared to females in the left coronary system.

Interestingly, the diameter of the right coronary artery and its branches were comparable in both males and females.

Coronary artery size in Indians has been reported to be significantly smaller when compared to that of the western population8,15,16 . This has been attributed to body habitus, build & the body surface area. Lip8 reported that though the unadjusted angiographically estimated mean diameters of various coronary artery segments in the western population among Caucasians were higher than those of Indian Asians there was no statistically significant difference when these were indexed to the body surface area leading them to the conclude that the smaller size of the coronaries in Indian Asians is attributable to their relatively smaller body surface area. In terms of graftable arteries during CABG, Indian patients may have an LAD which is smaller, no significantly smaller obtuse marginal branches but indeed a larger RCA and branches. Similar findings have been reported by Dhawan4, 15.The smaller dimension of some coronary arterysegments has important diagnostic and therapeutic implications since for any interventional procedure the absolute size of the coronary arteries matters12. It has been reported that occlusion or thrombosis is more common in vessels less than 2.5 mm in diameter8. A moderate (60%) stenosis in a 2.5 mm vessel would have more effects on flow than the same degree of stenosis in a 3.5 mm vessel as the cross sectional area in the former would be reduced to 1.76 mm2 as compared to 3.46 mm2 in the larger vessel. Thus a moderate plaque would cause significant obstruction in

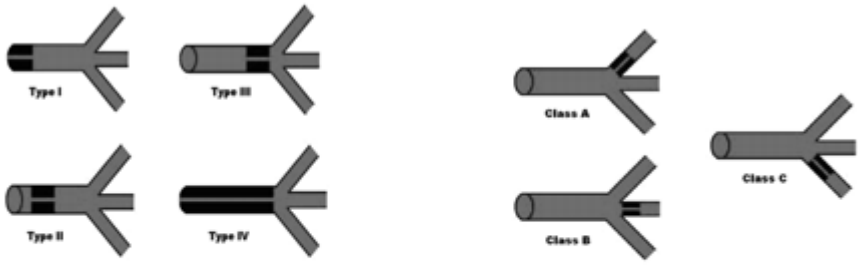
a small vessel with significant implications in coronary revascularization. Among patients with stable angina, certain findings on exercise testing, such as early or pronounced ischemic ECG changes in stage I or II of the Bruce protocol or at heart rates less than 120 beats/min or a high risk Duke treadmill score, can identify patients who are more likely to have left main coronary artery disease

Significant, defined as a greater than 50 percent narrowing, left main coronary artery disease, is found in 4 to 6 percent of all patients who undergo coronary arteriography [1]. When present, it is associated with multivessel coronary artery disease (MVCAD) over 70 percent of the time [2]. Most patients are symptomatic and at high risk of cardiovascular events, since occlusion of this vessel compromises flow to at least 75 percent of the left ventricle, unless they are protected by collateral flow or a patent bypass graft to either the left anterior descending or circumflex artery. Studies performed before revascularization with CABG became the standard of care revealed a poor prognosis for these patients, with three-year survival as low as 37 percent [3]. stenosis of the left main coronary artery (LMCA) is found in 3%–5% of patients undergoing coronary angiography.1 Total occlusion of the LMCA, defined as the complete absence of antegrade flow of contrast beyond the bifurcation of the LMCA, is rare. The rarity of this condition in an angiographic series may be due to the high mortality in this subgroup. Traditionally, coronary artery bypass graft surgery has been considered the treatment of choice for these patients. However, percutaneous revascularization is being increasingly performed in such patients.

Causes of left main coronary artery disease

a) Atherosclerosis
b) Non atherosclerotic causes:
c) Irradiation
d) Takayasus arteritis
e) Syphilitic aortitis
f) Rheumatoid arthritis
g) Aortic valve disease
h) Kawasaki disease
i) Injury after left main coronary intervention or cardiac surgery
j) Idiopathic causes

DeLago's LMCA stenosis Classification



Type I: Osteal Left Main
Type II: Mid Left Main
Type III: Distal Left Main
Type IV: Diffuse Left Main

Class A = Osteal LAD
Class B: Ramus intermedius
Class C: Osteal Lt. CX

2. Normal flow was laminar with a distinctly phasic character (diastolic predominance).

Mean values of peak coronary flow velocity (in cm/sec) for systole and diastole were:

Artery	Sys	Dia
LMCA	36	71
LAD	31	67
LCX	36	75
RCA	25	29

Coronary artery bypass graft surgery (CABG) has usually been recommended for left main disease in symptomatic patients. As will be described below, CABG is associated with a significant improvement in important cardiovascular outcomes compared to medical therapy, including mortality [4]. Percutaneous coronary intervention (PCI) has usually been restricted to patients considered inoperable, at high risk for CABG, or with prior CABG and at least one patent graft to the left anterior descending or circumflex artery (so-called "protected" left main disease). Graft patency is important in this setting in the event of acute or late closure

after PCI. The prevalence of significant (> 50%) isolated LMCA stenosis varies from 0.25 to 1.3 percent in patients undergoing diagnostic catheterization ([24]). In about 80 percent of patients with LMCA stenosis there is concomitant significant atherosclerosis in other major coronary vessels. The reported prevalence of LMCA stenosis varies from 2.5 to 10 percent depending upon the cohort of patients studied in different series ([25],[26]). The prevalence of significant LMCA stenosis has been reported to be approximately nine percent in patients undergoing bypass surgery, approximately five percent in patients with chronic angina and about seven percent in patients with AMI ([27],[28]).

Protected Versus Unprotected LMCA Stenosis

Left main trunk (LMT) or LMCA is defined as "Protected" when there is atleast one patent bypass graft to the left circumflex (LCX) or left anterior descending (LAD) artery. In the absence of such a patent graft, LMCA (LMT) is said to be unprotected. The intervention for protected or unprotected LMD may be elective or in emergency. Unfortunately, in the literature there is lot of mix-up about the results of these groups ([29]). The distinction becomes important when outcome from different studies are to be compared. The interventions performed in emergency usually are in critically ill patients presenting with haemodynamic collapse. In contrast, the elective interventions are performed in relatively stable patients. During the last few years, the number of elective interventions in unprotected LMD have increased. Initially, LMCA elective interventions were performed only in CABG ineligible

patients or in those with limited life expectancy. In the post stent era, the indications of elective LMCA interventions have widened and even includes CABG eligible patients with normal or reduced left ventricular (LV) function.

Percutaneous reperfusion of LMCA stenosis complicated by AMI

There is a paucity of data on outcomes of patients undergoing emergency percutaneous or surgical revascularisation of LMD complicated by AMI. Initial studies on percutaneous revascularisation in patients with AMI and LMD reported very poor in-hospital results. In a series of 6 patients reported by Chauhan et al ([31]), the in-hospital mortality was 83% (5/6 patients) and led the authors to conclude that there is a prohibitive risk for percutaneous revascularisation in LMD complicated by AMI. Quigley et al. ([32]) reported the in-hospital outcome of 34 patients with AMI and LMD. Out of these, 16 patients were in cardiogenic shock; 7 patients were treated medically, 4 were managed with PTCA, and 5 with CABGS. The overall mortality rate was 100% (4/4 patients) in the PTCA group and 100% (7/7 patients) in the medical treatment group; the surgical revascularisation group mortality rate was 89% (8/9 patients).

In contrast to poor results in earlier studies, the outcomes in ULTIMA registry ([33]) and in study by Neri et al ([34]) are more favourable. The better results in these series can be attributed to use of intracoronary stents, newer antiplatelet therapy and mechanical support. In

ULTIMA (Unprotected left main trunk intervention multicenter assessment) multicentric registry from 13 countries ([33]), 40 patients underwent percutaneous LMCA interventions for AMI with in hospital mortality of 55% and 18% requiring CABGS. Neri et al ([34]) reported results of PCI in 22 patients with most (82%) of the patients presenting in cardiogenic shock. The primary success of PCI was 91% and primary stenting was performed in 17 patient (77%). The overall in-hospital mortality was 50% (11/22 patients) and all deaths were due to refractory shock. The 6-month survival rate was 41% 1%, while the event-free survival was 27% 10%. At 6-month follow up, the mortality rate increased to 59%, the target vessel revascularisation rate was 14%. The results of ULTIMA registry and data of Neri et al ([33],[34]) suggest that emergency percutaneous revascularisation in patients with LMD and AMI is technically feasible. The benefit of percutaneous coronary interventions on mortality is likely. However, the small number of patients prevents any definitive conclusion.

With respect to the emergent CABGS for LMD complicated by AMI, the only reported data are those of Nakanishi et al. ([35]) and are based on a series of 13 patients. The mortality rate was 46% for the entire group and 53% for the patients presenting with cardiogenic shock. These results are comparable to those observed by Neri et al ([34]) and by ULTIMA registry experience ([33]). However, the paucity of data does not allow any conclusion on whether percutaneous or surgical reperfusion is preferred in patients with LMD complicated by AMI. Adjunctive intra-aortic balloon pump (IABP) is mandatory when PCI of LMD is performed during emergency, during AMI, in cardiogenic shock and in haemodynamically unstable

patients. Emergency LMCA intervention has also been performed using retrograde perfusion via the coronary sinus and retroinfusion of coronary veins by using extra corporal membrane oxygenation pump.

Long Term Clinical Outcomes

Park SJ et al ([39]) in a recent study reported long term outcome of 127 consecutive cases who underwent elective stenting of unprotected LMCA. At two years the cumulative survival rate was 97.0 1.7% and the cardiac event-free survival rate was 86.9 3.3%. Similar figures have been reported by Silvestri and colleagues ([17]) in a low risk population. One-year mortality reported by Park and colleagues was 5.7% and the same figures have been reported in low-risk group CABGS series ([40]). The mortality rate in this series over two-year followup was 3.1%, which is acceptable.

For patients with restenosis, CABG was recommended first. However, other modalities of treatment included repeat angioplasty using RA with or without radiation therapy. In this series of Park et al ([39]) after six months, there were no cardiac deaths or target lesion revascularizations, indicating that the long-term clinical course may be excellent after unprotected LMCA stenting in selected patients with normal left ventricular function. This result is consistent with previously published data showing that the restenotic process after stenting is time-limited and that little progression occurs beyond six months.

Tan WA et al. ([37]) on behalf of ULTIMA investigators reported long term clinical outcome after PCI of unprotected LMCA in 279 patients. Thirty (13.7%) patients died in hospital, and the rest were followed up for a period of 19 months. The 1-year incidence was 24.2% for all-cause mortality, 20.2% for cardiac mortality, 9.8% for myocardial infarction, and 9.4% for CABGS. Independent correlates of all-cause mortality were LVEF 30%, mitral regurgitation grade 3 or 4, presentation with myocardial infarction and shock, creatinine 2.0 mg/DL, and severe lesion calcification. For the 32% of patients (low risk group) < 65 years old with left ventricular ejection fraction > 30% and without shock, the prevalence of these adverse risk factors was low. No periprocedural deaths were observed in this low-risk subset, and the 1-year mortality was only 3.4%. On the basis of this data it becomes obvious that patients undergoing unprotected LMCA PCI are the ones with serious comorbidities and consequently have high event rates. PCI may be an alternative to CABG for a select proportion of elective patients (low risk group) and may also be appropriate for highly symptomatic inoperable patients.

Finally, on the basis of the 2% per month death rate among hospital survivors noted over the first 6 months after hospital discharge, probably partly a result of restenosis, it is strongly recommended to have surveillance coronary angiography at 2 and 4 months post PCI.

Role Of CABGS In LMCA Stenosis

CABGS has been standard of care for LMD ever since the veterans administrations cooperative study established its superiority over medical treatment with regards to survival ([41]). PCI was shown in randomized clinical trials in the 1990's to be equivalent to CABGS in terms of rates of survival and infarct free-survival in a growing number of patients with coronary artery disease. It is highly unlikely that there will be randomized clinical trials to compare results of PCI and CABGS in LMCA stenosis because of logistic considerations of prohibitive sample size and cost requirements.

From the available data, based on clinical studies and registries there is no doubt that PCI is an alternative to CABGS in selected cases. Judicious patient selection remains critical for both the interventionalist and cardiac surgeon, and further studies are needed to define which patients are truly inoperable, who among these patients still may benefit from PCI, and those in whom revascularisation attempts will be futile. Unfortunately, patients who are good candidates for surgery are typically the same ones who will do well with other invasive procedures, and poor surgical risks often mean poor global risks. It is fair to say that CABGS is still the first choice for the majority of patients with LMD, but PCI is a viable option in select circumstances **:** those presenting with AMI, the highly symptomatic but inoperable patient, and perhaps the low-risk patient group discussed above.

This concern about closure has been minimized by stent implantation, which has led to increasing evaluation of PCI for left main disease. Interest in elective left main stenting has further intensified with the availability of drug-eluting stents (DES), which dramatically reduce the incidence of in-stent restenosis The diagnosis of left main is usually made by angiography. However, certain findings on exercise testing or, in patients with acute coronary syndromes on the ECG, are suggestive of left main disease. Coronary stents are widely used to overcome the limitation of balloon angioplasty and may be useful for treating unprotected left main coronary artery (LMCA) stenosis.

Stenting in distal LMCA bifurcation disease is technically more complex than in ostial or shaft lesions.

With drug –eluting stents, simple (i.e., extending the stent across the circumflex artery) or complex stenting techniques (i.e., multiple stent placement, such as kissing stenting, T stenting, or crush technique) can be used for the treatment of bifurcation LMCA lesions according to vascular size and lesion morphology.

In bifurcation LMCA lesions with a normal circumflex Artery, simple stenting strategy using a crossover technical may be a more effective strategy for reducing the restenosis rate than complex stenting techniques.

Intravascular ultrasound is a useful adjunct in unprotected LMCA intervention to assess actual vessel size, disease extent of the main vessel and the side branch, and final stent optimization.

Routine use of debulking atherectomy is not recommended in drug- eluting stent implantation for unprotected LMCA stenosis. However, its selective role is being studied.

Although and intra – aortic balloon pump is not routinely recommended during the procedure, it should be considered for prevention of hemodynamic collapse in patients with severely depressed left ventricular function.

When patients with LMCA stenosis had well – preserved left ventricular systolic function and were good candidates for coronary bypass graft surgery, the procedural success rates and in hospital outcomes after the use of bare metal stents were favorable.

Use of drug eluting stents significantly decreased in stent restenosis when stenting was done for unprotected LMCA stenosis compared with the use of bare metal stents.

Ongoing randomized studies comparing the safety and efficacy of drug eluting stents with bypass surgery will determine whether stenting can be an alternative to bypass surgery in patients with unprotected LMCA stenosis.

Left main coronary artery (LMCA) stenosis has several causes . LMCA stenosis is considered an attractive target for balloon angioplasty because of the vessels large caliber, the lack of tortuosity, and the short lesion length. Histologically, the LMCA ha the most elastic tissue of the coronary vessels, accounting for the poor response of the LMCA to simple balloon angioplasty. However, coronary stents have been shown to reduce the immediate need for coronary artery bypass surgery (CABG) for abrupt vessel closure and the likelihood of restenosis after balloon angioplasty. Newer devices are widely used to overcome the limitations of balloon angioplasty and may also be useful for treating unprotected LMCA stenosis in some patients. Stenting of unprotected LMCA stenosis if therefore considered a therapeutic option in selected patients.

TECHNICAL CONSIDERATIONS IN UNPROTECTED LEFT MAIN CORONARY ARTERY STENTING

Ostial lesions

The ostial LMCA lesion is dilated and stented with the tip positioned in the aortic sinus. The proximal end of the stent protrudes slightly to the left (1 to 2 mm) outside the ostium and is expanded against the aortic wall as in stenting of any aorto – ostial lesion. Predilatation before stenting is necessary and is usually performed with undersized, conventional

angioplasty balloons. The stent is then deployed by inflating the stent delivery balloon at a nominal or high pressure. After deployment of the stent, the stented segment is often dilated again using high – pressure for the balloon inflation to achieve angiographically confirmed optimal results. Stent size is selected based on the reference artery size and lesion length. Slotted – tube stents, rather than coil stents, are preferable for treatment of LMCA ostial disease because of their strong radial force. Balloon inflations should be brief (<30 seconds) and multiple (>3) to avoid prolonged global is ischemia and ischemia related complications.

Shaft lesions

The lesions in the mid shaft of the LMCA can be pre dilated and then stented as done for any discrete lesion in other branches. As for ostial LMCA lesions, debulking is commenced in suitable lesion.

Approximately two thirds of all significant lesions in the LMCA involve the distal bifurcation. Stenting of distal LMCA bifurcation disease is the most technically complex and potentially high- risk anatomic variant of LMCA intervention. Distal LMCA bifurcation stenting should therefore be performed only by highly skilled interventionists. Patients must also be informed and must fully comprehend the risks and benefits of the percutaneous approach compared with surgical alternatives.

Balloon angioplasty of the distal LMCA bifurcation has been associated with a high rate of complications and restenosis. In the contrast, numerous reports suggest that stenting in the bifurcation lesion may result in predictable short term outcomes with dreadful effects. Side branch occlusion due to plaque shifting during balloon angioplasty of a parent vessel is common, and in LMCA bifurcation stenosis, intervention may result in predictable short term outcomes with durable effects. Side branch occlusion due to plaque shifting during balloon angioplasty of a parents vessel is common, and in LMCA bifurcation stenosis, interventions may results in occlusion of the ostium of the left anterior descending artery (LAD) or left circumflex artery (LCX), with disastrous clinical consequences. Various interventional techniques have branch occlusion in bifurcating coronary lesions. Plaque debulking with directional and rotational atherctomy has been proposed for bifurcation lesions to reduce plaque shift and side branch compromise. Many bifurcation stent techniques have been explored to prevent side branch occlusion including T stenting, reverse – Y stenting, trouser leg stenting, V stenting , culotte stenting, and crush stenting. No single interventional technique has been found to guarantee preserved patency of the parent vessel and side branch. Bifurcation stenting (with or without debulking) is technically demanding, requiring considerable expertise. The complexity of these techniques depends on the specific anatomy of the bifurcation, the approach used, and the stents employed. There is no single optimal technical approach to LMCA bifurcation disease. Moreover because of the serious clinical consequences of major side branch occlusion during LMCA bifurcation stenting, not all

interventionists agree that percutaneous interventions for LMCA bifurcation stenosis is warranted.

At the Asan medical center, stenting for LMCA bifurcation stenoses was performed in selected patients between November 1995 and march 2002 80 consecutive patients with unprotected LMCA bifurcation lesions underwent stent placement. Stenting was larger performed with or without debulking atherectomy at the operators discretion. If the artery was larger than 3.0 mm in diameter and had no calcification, directional coronary atherectomy (DCA)was usefully performed using a 7 Fr catheter. Rotational atherectomy using a step burr approach was performed for calcified lesions. Four major stenting strategies were used, determined by the specific lesion characteristic and anatomy of the distal LMCA bifurcation, including crossover stenting of the LCX, T(Y) stenting, kissing stenting and bifurcation stenting.

Methods of stenting

Crossover technique

Tube stents may be deployed from the LMCA to the proximal portion of LAD if the LCX is diminutive (<2.5mm) or normal (diameter of stenosis <50%). After stent placement, the LCX is dilated through the first implanted stent strut, as necessary . in the study park and

colleagues, the LCX ostium was typically covered by a stent without risk of occlusion if it was diminutive or normal. Fifty – four percent of patients were successfully treated with stent placement across the LCX ostium, suggesting that this technique may be widely used for treatment of LMCA bifurcation lesions. However, progression of disease in the side branch lesion spanned by a stent may be difficult to treat, and a possible risk of side branch occlusion remains an important limitation of this strategy.

T or Y stenting with or without simultaneous kissing stenting

T or Y stenting with or without simultaneous kissing stenting is performed if the LCX is large and has significant ostial disease. In the bare metal stent (BMS) era, in the T technique (or Y technique, depending on the angle of the bifurcation), after coil stenting (or open – cell design) from the LMCA to the LCX, a slotted – tube stent was sequentially implanted into the LAD through the struts of the coil stent .

The efficacy of T stenting was challenged in the era of the drug – eluting stent (DES). The study of the sirolimus – Eluting stent in the Treatment of patients with long de novo lesions in small native coronary arteries (SIRIUS bifurcation study) was a multicenter, randomized trail to asses the feasibility and safety of sirolimus – eluting stent (SES) implantation for bifurcation lesions. In this study, 22 of 43 patient assigned to single SES implantation in the main vessel (group B) were crossed over to T stenting with two SESs,

implanted in the main vessel and the side branch (group A) because of flow impairment or residual stenosis of more than 50% of the diameter of the side branch after stent implantation in the main vessel. Sixty – three patients were treated with single stent implantation. Although the high number of crossover patients has made direct comparison of the two groups difficult, the restenosis rates for the two treatment groups were comparably very low (2.3% in group A versus 5.0% in group B). the use of a second stent did not improve the restenosis rate of the side branch (19.2% in group A versus 21.1% in group B, p= NS). Overall, the restenosis rate for group A was higher than for group B (25.0% versus 10.0%, p=.20). but it did not achieve statistical significance. The investigators of this study postulated that the relatively high restenosis rate at the side branch in the two – stents implantation group was caused by incomplete coverage of the bifurcation using the T stenting technique. They suggested that T stenting might leave a gap between the two stents at the bifurcation. Other techniques, such as the crush technique of kissing stenting, have been introduced to overcome the problem of T stenting.

Kissing stenting

Kissing stenting is also a two stent implantation technique similar to kissing balloon inflation. A minimum 8 Fr guiding catheter is needed for this technique. The two stents are deployed simultaneously, which creates a double lumen in the main vessel proximal to the bifurcation site. Adjunctive sequential alternative balloon dilatation with final kissing balloon

dilatation may be required for optimal stent expansion, especially in the distal part of the bifurcation. This technique is useful for bifurcation lesions with two large side branches with a large diameter of the LMCA trunk. Park suggested that this technique would be appropriate in treating large LMCA lesions (>4.5mm). a practical concern of this technique is the difficulty of re-accessing both branches in case of rest enosis. However, in the era of DES implantation as one of the appropriate stenting techniques for LMCA bifurcation lesions with a very proximal vessel. This technique is very safe because access to both branches is always maintained, and it allows complete lesion coverage quick performance and easy execution are the major advantages for this technique.

Bifurcation stenting with side branch access stenting

The AST SLK – View bifurcation stent (advanced stent technologies, san Francisco, CA) was designed to preserve side branch access and complete the bifurcation stent procedure. the AST SLK – View stent consists of a stainless steel stent with a widened section in the struts located between the proximal and distal ends. A side branch wire exit port passes through this hole, allowing access to the side branch after stenting the main branch. The AST SLK – view stent represents an main branch an attractive new approach for treatment of bifurcation lesions. However, further studies are needed to compare this stent with conventional stents for treatment of LMCA bifurcation lesions. Preliminary efficacy of this stent system, including outcomes for 11 cases of LMCA bifurcation stenosis, has been presented. In this

study, the binary restenosis rates at the 6 months follow up evaluation were 28.3% and 37.7% for the main vessel and the side branch, respectively.

Crush technique

The crush technique was introduced by Colombo and colleagues. With the use of DESs, this techniques has several advantages. The crush techniques ensures the complete circumferential coverage of the side branch ostium. A practically advantages is that the techniques is quite safe and relatively simple to execute. This approach gives an immediately successfully results with patency of both branches without special technical maneuvers. An important concern about this technique is whether the traditional final kissing balloon dilatation is required. Colombo and colleagues suggested that final kissing balloon inflation is very important to achieve long – term patency. Ormiston and coworkers supported the idea with the use of a phantom model. This study demonstrated that is important to postdilate both stents with appropriately sized balloons. One study achieved a 49% relative reduction in the restenosis rate in the side branch by routine use of final kissing balloon dilation compared with the results of the SIRIUS bifurcation study. the significant reduction of late lumen loss in the side branch after kissing balloon dilation can be explained by better strut contact with the vessel wall and better drug strut contact with the vessel wall and better drug delivery. Kissing ballon dilation may also correct stent deformation and ensure optimal stent scaffolding.

Adjunctive Devices in Unprotected Left Main Coronary Artery Stenting

Intravascular Ultrasound

Although intravascular ultrasound (IVUS) provides unique quantitative and qualitative information on coronary artery lesions, the impact of IVUS on long term clinical outcomes after stent implantation has been controversial. The can routine ultrasound influence stent expansion (CRUISE) study demonstrated that IVUS –guided stent implantation in non-LMCA lesions ensures more effective stent expansion and a larger minimal stent area, resulting in less frequent target vessel revascularization. These results may apply to LMCA intervention as well for several reasons. It is often difficult to evaluate the actual size of the LMCA by angiography. The left main trunk often is short and lacks a normal segment for comparison. Contrast blowback in the aortic cusp may obscure the ostium, and streaming of contrast may result in a false impression of luminal narrowing. Angiography may underestimate stenosis severity, because diffuse disease in the proximal and distal reference segments adjacent to a focal stenosis may be interpreted as normal dimensions, leading to stent undersizing. Preinterventional IVUS examination also provides important information about the underlying lesion morphology and may guide treatment strategy, especially in helping to decide when debulking is necessary or is complete.

Angiographically unapparent severe calcification may be seen by IVUS, leading to the decision to performed high-speed rotational atherectomy before stenting to maximize stent expansion. Moreover, negative remodeling (defined as an external elastic membrane cross-sectional area at the lesion site that is smaller than that of the distal reference segment) may be documented in 91% of patients undergoing IVUS-guided stenting of ostial LMCA lesions. in such cases; in which plaque volume is actually reduced compared with non-remodeled or positively remodeled vessels, debulking is unnecessary, and stenting along should be performed.

Table: Advantages to Intravascular Ultrasound before and during Left Main Coronary Artery Intervention

Provides precise quantitative measurement

- Reference vessel diameter
- Minimal luminal diameter (before and after)
- Lesion cross-sectional area (before and after)
- Lesion length (automatic pullback)

Characterization of plaque

- Arterial remodeling (positive, negative)

Plaque stability vs. rupture

Plaque distribution (eccentric, concentric)

Plaque composition (soft, fibrous, calcified, mixed; depth of calcium)

Dissection after predilatation and stenting (length, severity of lumen compromise)

Accurate guidance of procedure

Decision about additional ballooning

Decision about treatment strategy in intermediate lesion by quantitative coronary angiography

Decision about debulking procedure

Evaluation of stent expansion

Evaluation of apposition

Performance of IVUS before and during stenting in LMCA stenoses may provide useful information for the selection of the appropriate diameter of balloons and stents, as well as the accurate amount and extent of calcification and need to debulk. Such information has resulted in changes in the planned procedure and treatment modalities for approximately 40% of non-LMCA lesions. IVUS may also help differentiate which borderline lesions require

intervention (with stenting or surgery). However, there are no absolute ultrasound criteria for intervening in a "critical" LMCA stenosis. Important considerations include the patient's symptom status, the presence of other lesions, and the amount of myocardium in jeopardy. Nevertheless, suggested IVUS criteria for significant LMCA disease are stenosis of more than 50% of the vessel diameter, stenosis of more than 60% of the area, and an absolute cross-sectional area less than $7mm^2$ in symptomatic patients or less than $6mm^2$ in asymptomatic patients.

One study evaluated 122 patients with intermediate LMCA disease (=42% diameter stenosis assessed by quantitative coronary angiography). The 1 year event rate was 14% when LMCA revascularization was deferred based on IVUS findings. When patients with an event were compared with patients without an event, there were no significant differences in left ventricular function or the angiographic diameter stenosis, but the group with events had greater cross-sectional narrowing (70% $\pm$ 14% versus 53% $\pm$ 18%, P=.04), smaller minimum LMCA lumen area (6.8 $\pm$ 4.4 versus 10.0 $\pm$ $5.3mm^2$, P=.01), and smaller minimal lumen diameter (MLD) (2.30 $\pm$ 0.69 versus 2.94 $\pm$ 0.81 mm, P = .001). Predictors of cardiac events at 1 year were diabetes mellitus (OR = 6.32; 95% CI: 1.82 to 22.04; P=.004), any epicardial vessel with an angiographic stenosis of 50% or more as assessed by quantitative analysis (3.80 [1.08 to 13.39]; P = .037), and the LMCA MLD as assessed by IVUS (or = 0.17[CI: 0.05 to 0.59]; p= .005). when IVUS is used to assess the severity of intermediate LMCA stenoses, decisions to defer to revascularization must consider absolute IVUS dimensions,

the presence of diabetes, and the presence of significant lesions in other major epicardial vessels.

The Asan medical center reported their experience in stenting 127 unprotected LMCA lesions with (n=77) or without (n=50) IVUS guidance. Debulking procedures before stenting were more frequently performed in the IVUS guided group (39% versus 20%, p=.02), primarily because of identification of severe calcification with a circumferential arc of more than 90 degree after IVUS evaluation. According to the IVUS criteria of stent optimization, additional high pressure balloon angioplasty was performed in 15 (19.5%) of the 77 lesions. As a consequence, the postintervention minimal stent cross- sectional area increased from 10.7+- 2.8mm to 13.0+-4.0 mm after additional balloon angioplasty. The final lumen diameter after stenting was significantly larger in the IVUS guided group as assessed by quantitative coronary analysis (4.2+-0.6 versus 4.0+-0.6 mm, p=.003) . However, the angiographic restenosis and target lesion revascularization rates were not different between the IVUS guided and angiography guided procedures in this study. This finding may be explained partly by the fact that the reference vessel size in the current series was large (>4.0 mm) and that the post stent MLD was large (>4.0 mm), even in the angiography guided group. A post stent MLD of more than 4.0 mm should be large enough to prevent binary restenosis at follow up. However, we continue to believe that IVUS guidance of unprotected LMCA lesion stenting should be considered, because optimal stent expansion and apposition (which can be verified only by IVUS) may prevent stent thrombosis in the LMCA, a

complication with a potentially fatal outcome. In a single center, a small study evaluating the impact of IVUS on unprotected LMCA intervention showed that the incidence of major adverse events was similar : 2(8%) of 24 in the IVUS group and 7 (20%) of 34 in the non IVUS group (p=.18). however, IVUS evaluation may be crucial in unprotected LMCA intervention for lesion assessment, which facilities selecting performing optimal stenting strategy.

Debulking atherectomy

Debulking with directional or rotational atherectomy does not completely eliminate acute recoil and active vessel remodeling after directional. The angio graphic restenosis rate after directional atherectomy was found to be similar to that of balloon angioplasty alone despite a smaller post procedural, however optimally deployed coronary stents the rate of restenosis compared with balloon angioplasty (or directional atherectomy) alone by preventing acute elastic recoil and negative chronic remodeling (although neointimal hyperplasty increased). Debulking combined with stenting compared with stenting alone may result in a postprocedural lumen gain and subsequently angiographic restenosis. Studies have shown that residual plaque burden is an important predictive intimal hyperplasia in stented lesions and aggressive debulking with directional atherectomy before stenting might reduce the residual plaque burden and subsequently reduce the extent of restenosis. This combined approach therefore may be optimal approach for the management of unprotected LMCA

stenosis with a large plaque burden the asan medical center, debulking especially is performed before stenting if the lesion is suited rotational atherectomy before stenting is also performed if the plaque has diffuse superficial calcification. At the Asan Medical Center, the degree debulking using directional atherectomy was compatible with that of other reports (e.g stenosis after Optimal Lesion Debulking [SOLD] Registry compared with stent implantation without atherectomy, debulking before stenting in unprotected LMCA lesions was associated with a significant restenosis of angiographically confirmed restenosis target lesion revascularization as assessed by analysis the reduction in the rate of restenosis was most striking in LMCA ostial stenosis. However debulking atherectomy was not an independent predictor of freedom from restenosis as assessed by multivariate analysis. The most likely explanation is that because of the limited atherectomy device size, the degree of debulking achieved may be insufficient in large LMCA vessels. (i.e., mean reference vessel diameter of 4.0 mm). in the non debulking group it was possible to achieve just as large and MLD by high pressure balloon inflations.

DCA may facilitate successful stent placement by removing the plaque and may reduce restenosis rate by improving acute results. A nonrandomized study using BMSs showed that debulking before stenting resulted in significant reduction of angiographic restenosis. ($p= .049$) as assessed by univariate analysis. Hu and associaties found similar results for 67 low to high risk patients with unprotected LMCA stenosis with distal bifurcation involvement treated with IVUS guided directional atherectomy. The all cause

mortality, angiographic restenosis, and target lesion revascularization rates at 6 months were 7%, 24% and 20%, respectively. However it is unknown whether debulking atherectomy would be advantageous in the era of DES implantation. In practice the use of debulking has been decreased by the remarkable reduction of restenosis by DESs. Additional research on the effects of DCA and DES implantation is warranted.

Intra aortic Balloon Pump

Patients with normal left ventricular function are tolerant of global ischemia during balloon occlusion although the intra aortic balloon pump is not routinely recommended during the procedure, it should be considered for prevention of hemodynamic collapse in patients with severely depressed left ventricular function.

BARE METAL STENT IMPLANTETION FOR UNPROTECTED LEFT MAIN CORONARY ARTERY STENOSIS.

With the explosive growth of coronary stenting in the 1990sintervention in the diseased LMCA was again attempted The results of these series demonstrated that when patients with LMCA stenosis had well – preserved left ventricular systolic function and were good candidates for bypass graft surgery, the procedural success rates and in hospital outcomes after stenting were favorable.

Unprotected left main trunk intervention

A multicenter registry of 107 patients from 25 centers was used to examine the procedural safety and the midterm outcomes of patients who may be considered for percutaneous intervention of unprotected LMCA stenosis. Stents were used in 50% directional atheractomy in 24%, balloon angioplasty in 20% and rotational atherectomy in 6% of patients. Technical success was achieved in 96.4% of cases, but 20.6% of a patients died while in the hospital, and 10% had nonfatal Q- wave myocardial infarctions (MIs). After post – hospital discharge, outcomes were also unfavorable. Left ventricular function was the most important determinant of survival after LMCA intervention. The unprotected left main trunk intervention multicenter Assessment (ULTIMA). Registry data are difficult to interpret because of inclusion of a very heterogeneous group of patients, including those with poor of good left ventricular function, various degrees of severity of coronary artery disease, and different types of intervention used.

The ULTIMA registry was extended to 279 patients undergoing percutaneous intervention of unprotected LMCA stenosis between july 1993 and july 1998 to examine which patients might have favorable outcomes. Forty six percent of these patients were deemed inoperable of high risk surgical candidates. Thirty eight patients (13.7%) died in the hospital, and the remaining patients were followed for a mean of 19 months. The 1 year incidence of all cause mortality was 24.2% (with 20.2% cardiac mortality), with a 9.8% rate

of bypass surgery. By multivariate analysis, the independent correlates of death during and after hospitalization were left ventricular ejection fraction of 30% or less grade 3 to 4 mitral regurgitation, clinical presentation of MI with cardiogenic shock, serum creatinine level of 2.0mg/dl or higher, and severe lesion calcification . decreasing left ventricular ejection fraction was inversely related to events in a nonlinear fashion, with an apparent inflection point at 30%. Except for lesion calcification the predictors of cardiac death were similar although different in magnitude: mitral regurgitation grade 3 or 4 (hazard ratio [HR] = 5.0); left ventricular ejection fraction of 30% or lower (HR = 4.9); MI with cardiogenic shock (HR = 4.8; and serum creatinine level of 2.0 mg/ dL or higher (HR=3.2). on the basis of this analysis, 32% of the patients could be identified with three clinical features: age younger than 65 years left ventricular ejection fraction of 30% or more and absence of cardiogenic shock from acute MI; they comprised a low risk group with a 3.4% 1 year mortality rate after LMCA intervention and a 2.3% risk of MI . there were no periprocedural deaths in this subgroup, and there were no additional deaths or MIs beyond 4 months after discharge (up to 35 months). During the 1 year follow up of this low risk group, 24.5% of patients required additional revascularization procedures including repeat percutanenous intervention in 20.4% and bypass surgery in 11.4% Similar data were reported by silvertri and colleagues, who defined a low risk group as younger than 75 years with no prior bypass great surgery with a left ventricular ejection fraction of 35% or higher and with the absence of renal failure for these patients the 1 Year mortality rate after unprotected LMCA stenting was 7% and the

need for revascularization was 28%. To put these data in perspective, the in hospital mortality rate of patients with LMCA disease undergoing bypass graft surgery was 3.9% as reported by the Cleveland clinical Foundation, with a 1 year mortality rate of 11.3%. In the latter report, the 1 year morality rate after bypass surgery. For a low risk group similar to that defined in ULTIMA (age < 65 years with New York Heart Association congestive heart failure class < 2) was 5.7%. patients with well preserved systolic ventricular performance may have acceptable outcomes after LMCA stenting.

Experience of the Asan Medical Center

The initial report from the ULTIMA Registry demonstrated a relatively high short term cardiac mortality rate of a heterogeneous group of patients. Many of these patients were high risk or ineligible for bypass surgery, and left ventricular ejection fraction was directly related to early and late survival. In the Asan Medical Center experience as reported by park and associates, only patients with a left ventricular function of 40% or higher were considered for LMCA intervention.

Until January 2001, unprotected LMCA stenting was performed in 156 consecutive patients with normal left ventricular function. The procedural success rate was 99.1% and 13% underwent multivessel angioplasty during the intervention. There were no procedure related deaths. However, one patients developed a coronary perforation after DCA, which

was successfully treated with a stent graft. During the hospital stay, angiographically documented stent thrombosis occurred in one patient (0.6%) at 3 days after intervention and was complicated by a Q –wave acute MI and the need for elective bypass graft surgery 30 days later. For the remaining patients, the in hospital clinical outcome was uneventful. Angiographic follow up data were obtained for 100 of 104 eligible patients (96%) Restenosis was angiographically documented in 19 patients (19%). In the Asan Medical Center study, when restenosis developed after stenting, it required repeat revascularization, typically within 6 months, and thereafter, most patients were free of symptoms without major adverse cardiac events.

As expected, a smaller reference vessel size was related to a greater likelihood of restenosis, because late lumen loss may be greater in stents implanted in large vessels. As in previous studies of non LMCA lesions and protected LMCA stenting, the post stent MLD and minimal lumen cross sectional area as assessed by IVUS were the most powerful predictors of angiographic restenosis. Target lesion revascularization at the 2 year follow up was independent of lesion location within the LMCA as assessed by univariate analysis, there were trends for lower restenosis rates in patients undergoing debulking and in those achieving was the only independent predictor of angiographically observed restenosis in the study. The angiographically confirmed restenosis rate was statistically higher in vessels with a reference diameter of less than 3.6 mm. this cutoff level of 3.6 mm is an arbitrary lower

threshold. Although the 31% restenosis rate for these vessels may be slightly higher than that expected in non LMCA stenting for similar sized arteries, it still is acceptable.

The results to LMCA bifurcation stenting from the Asan Medical Center were published in 2002. Sixty three consecutive patients were included.DCA was performed in 32 patients (51%). The procedure was successful in all patients, and a prophylactic intraaortic balloon pump was used in only two patients. In hospital events did not occur for any patient. Angiographic follow-up was performed for 43 (86%) of the 50 eligible patients reaching the 6 month follow up point. The angiographic restenosis rate was 28% (including the parent vessel only [LMCA to LAD], 14%; LCX only, 9%; and both, 5%). Restenosis in the parent vessel occurred less frequently in the debulking group than in the non-debulking group (5% versus 33%, P=.02). Smaller reference vessel diameter and not having performed debulking were significant univariate predictors of restenosis in the parent vessel, but debulking was the only independent predictive factor for freedom from restenosis (OR=0.10; 95%, CI: 0.01 to 0.90; P=.04). No factors were predictive of restenosis at the side branch. Mean follow-up duration was 19.9 $\pm$ 13.7 months (range, 0.23 to 64.6 months). There were low noncardiac deaths but no instances of MI during follow-up. Target lesion revascularization was performed in 6 patients (10%), including repeated percutaneous intervention (n=5) and bypass surgery (n=1). The event free survival rate was 86% $\pm$ 6%. In this study, DCA might have been useful for treatment of LMCA bifurcation lesions because a large plaque burden was usually present, and debulking might have prevented plaque shift.

Experience of the ULTRA Registry

The unprotected left main trunk, angioplasty (ULTRA) study was a multicenter, prospective registry of patients undergoing emergent or elective percutaneous coronary intervention (PCI) for unprotected LMCA stenoses (n=284), which was performed in Japan, and the results were presented at the complex Catheter Technique meeting in Japan in 2002. This study included very-high-risk patients, including those with acute MI (17%) and patients undergoing emergent intervention for acute coronary syndromes (35%). Coronary stenting, DCA, and other techniques (including conventional balloon angioplasty, cutting balloon angioplasty in acute MI were very poor, as expected, with a clinical success rate of 64% and an in hospital mortality rate of 34%. Patients without acute MI, however, had excellent initial and long term outcomes. In these patients, procedural success was achieved in 99.6% of patients, and major in hospital complications (i.e., death, Q-wave MI, and emergent bypass surgery) occurred in only 6% of patients. In patients undergoing elective intervention for LMCA disease (n=183), the major in hospital complication rate was only 1%. During 30$\pm$11 months of follow up, the restenosis rate was 22% and the 1 year event free survival rate was 89%. These very good initial and long term outcomes are similar to those of other studies of stenting for low risk patients with unprotected LMCA stenosis.

Experience of the Multicenter European Study

Black and associates reported the results of stenting unprotected LMCA stenoses in 92 patients from Australia and France. Angiographic success was achieved in 100% of patients, and the mean final stent diameter was 3.9±0.51 mm. Four (4.3%) procedure-related deaths occurred. Neither MI nor emergency bypass surgery occurred during the index hospitalization. During follow up (7.3±5.8 months; median, 239 days; range, 49 to 1477 days), there were six additional deaths (6.5%): one sudden death was presumed to have a cardiac cause, one was caused by ventricular arrhythmia, three patients died of congestive heart failure, and there was one noncardiac death from lung cancer. The Kaplan-Meier survival estimates found 500- and 1000-day survival estimates of 89% and 85% respectively. Of the 82 (89%) patients surviving at 6 months, 4 had symptomatic LMCA restenosis and were treated by repeat balloon angioplasty within the stent, Nine other patients had repeat percutaneous intervention in other vessels, and two patients had bypass surgery for restenosis (one for LMCA disease, one for LAD disease). The results differed dramatically, depending on whether LMCA stenting was performed in patients in whom bypass surgery was or was not contraindicated (i.e., high and low risk groups, respectively). The total mortality rate at 6 months was significantly higher for patients who were not candidates for bypass graft surgery (20.5% versus 3.8%, P<.02). The final stent MLDs and diameters of stenoses were

predicative of mortality by univariate analysis. Lower left ventricular ejection fraction and the presence of three-vessel coronary artery disease also tended to be more common in patients who died of cardiac causes.

Silvestri and coworkers also examined the outcomes of low risk patient (n=93) and high risk patients (n=47) after LMCA stenting. The high risk group was composed of patients who were older than 75 years, had history of heart surgery, had a left ventricular ejection fraction less than 35% had renal failure, had inadequate distal coronary runoff, or had severe respiratory failure. The mortality rate at 1 month was 7% for the high risk group of patients and 0% for the low risk group. However, the rate of freedom from major adverse cardiac events at I year was similar for the two groups (66% in high risk group versus 72% in low risk group, p=NS).

Experience of the French multicenter registry

Lefevere presented the French data comparing coronary intervention (n=193), bypass surgery (n=233), and medical treatment (n=57) for LMCA stenoses at the complex catheter Technique meeting in Japan in 2002. Eleven centers in france enrolled 483 patients older than 75 years, with pulmonary failure, renal failure, severe peripheral disease, previous bypass graft surgery, previous stroke, and left ventricular ejection fraction less than 30%.

Coronary intervention, bypass surgery, or the patients discretion. High risk patients were more common in the coronary intervention group than in the bypass surgery group (45% versus 14%), although triple vessel disease was more common in the surgery groups (53% versus 68%). The in hospital mortality rate was higher after surgery than after coronary intervention (3.8% versus 0%, p<.001). at the 6 month follow up evaluation, the rates of mortality (6.4% with coronary intervention versus 8.1% after surgery) and MI (1.6% versus 1.6%) were similar in the two groups. However, the rate of the repeat revascularization of the LMCA was higher in the angioplasty group than the surgery group (15.2% versus 2.7%, p=.04). although these data were not from a randomized , controlled study, the result suggest that LMCA stenting may have comparable long term results in terms of freedom from death or MI compared with bypass surgery. These favorable initial and intermediate term outcomes of LMCA stenting for low risk patients (who would otherwise be candidates for bypass graft surgery) suggested the feasibility of unprotected LMCA stenting.

APPROACH IN STENT RESTENOSIS OF THE UNPROTECTED LEFT MAIN CORONARY ARTERY

In stent restenosis of the LMCA remains a challenging problem. Because unrecognized LMCA restenosis can manifest as cardiac death, most groups perform unprotected LMCA stenting recommends elective angiographic restudy at 4 to 6 months after stenting. Elective bypass graft surgery is usually recommended for the treatment of in stent restenosis of the

LMCA. Prior stenting in the LMCA does nor interfere with subsequent CABG, and it is the gold standard for the treatment of LMCA stenosis. Alternatively, repeated percutaneous interventions using rotational atherectomy or radiation therapy, or both in selected patients who refuse surgery have been performed successfully. Further studies and follow up are needed. The role of drug- eluting stents for treating LMCA in stent restenosis after bare metal stenting has not been reported.

ELECTIVE INTERVENTION FOR PROTECTED LEFT MAIN CORONARY ARTERY STENOSIS

Over the past 25 years, bypass surgery has provided excellent short term and long term clinical results for patients with LMCA disease, and the treatment of this lesion has therefore largely remained in the province of the cardiovascular surgeon. However LMCA stenosis often require treatment after bypass surgery disease of bypass graft failure. In most cases, a bypass graft is patent to the LAD or to the LCX (or one of their respective branches), resulting in intervention being required for a protected LMCA lesion; the implication is that such a procedure is lower risk than in an unprotected LMCA lesion because ischemia due to LMCA occlusion does not compromise a large portion of the left ventricular myocardium, conventional balloon angioplasty is often ineffective for a heavy plaque burden and calcification in the unprotected (or protected) LMCA lesion. However, several studies have shown that stenting a protected LMCA stenosis can be safely performed with a high success

rate and favorable clinical outcomes Kornowski and coworker reported that stents reduce major in hospital complication but might not significantly reduce repeat revascularisation of major cardiac events at 1 year compared with nonstent LMCA procedures. In their study, diabetes mellitus (OR=3.2, P=.04) independently predicated target lesion revascularization, and the final lumen diameter (OR=0.3, P=.017) was negatively associated with target lesion revascularization. Nevertheless, the use of stents, alone or after initial rotational atherectomy, produces the best immediate angiographic results. Technical considerations are similar to those for unprotected LMCA intervention, and precise positioning of the stent is critical, as described previously. although not proved.

AIM AND OBJECTIVES OF THE STUDY

The purpose of the study was the utility of transesophageal echocardiography in assessing left main coronary artery stenosis

Materials and methods:

The study was conducted in the department of cardiology Government Rajaji Hospital Madurai between Jan 2009 to April 2010.All patients underwent Coronary angiogram &Transesophageal echocardiography was done and left main coronary artery visualized at midesophageal transverse view at base of the heart and at the level of left sinus of valsalva and flow was recorded with pulsed wave Doppler.

Methods

Study group

Fifty patients with a left main coronary artery (LMCA) stenosis were prospectively studied from January2009 to April2010. We chose patients with LMCA because, TEDE recordings are easier to obtain in these portions of the LCA. A high quality TEDE signal was obtained in 50 patients (45 men and 5 women, mean age 53 years [range 36 to 70]). Written informed consent for TEDE examination was obtained in all patients.

Coronary angiography and quantitative coronary angiography

Coronary angiography was performed using the standard Judkins method with the femoral artery approach. Coronary injections were performed using multiple views, and images were recorded on TOSHIBA flat panel direct digital acquisition system . This quantitative

coronary angiographic system has been validated previously (16). Quantitative analysis of stenosis was performed using the average of results obtained from two orthogonal projections, when available, or the most severe narrowing of several nonorthogonal angiographic projections. Three recognized quantitative variables of stenosis severity 17., 18. were automatically computed by the software: percent diameter stenosis (DS), minimal lumen diameter (MLD) and percent CSA stenosis.

Transesophageal Doppler echocardiographic measurements

Transesophageal echocardiography was performed with a 7-MHz probe connected to a Philips IE33 echocardiographic system within 24 h of the angiographic study. A multiplane probe was used in all patients. Transesophageal examination was performed in each patient after oropharyngeal anesthesia by lidocaïne. The LMCA was visualized by placing the transducer just above the aortic leaflets. Small adjustments in transducer orientation were necessary to visualize the bifurcation of the vessel into the LAD and circumflex artery.The length ,diameter of LMCA (at the level of ostium,shaft and distal LMCA)were measured. Prestenotic and transstenotic coronary flow velocities were then measured as follows: coronary blood flow was first visualized by color flow imaging and a localized color aliasing phenomenon corresponding to a local flow acceleration was searched; pulsed wave Doppler echocardiography was then sampled in the site immediately upstream from the area of color aliasing; second, the sample volume was moved slightly downward in the area of color

aliasing. High pulse repetition frequency or continuous wave Doppler echocardiography was used to quantitate the magnitude of transstenotic velocities if these velocities were too high to be measured by pulsed Doppler echocardiography without aliasing,. Small adjustments in the transducer orientation allowed alignment of the ultrasound beam with the long axis of the interrogated proximal portion of the LCA. The peak flow–velocity curve was traced from the outer border of the Doppler spectral signal, and the time–velocity integral (TVI) was obtained by planimetry as the area under this peak flow– velocity curve during diastole.other investigators have previously reported good interobserver and intraobserver reproducibility of coronary flow transesophageal Doppler velocity recording in the proximal part of the LCA 9., 10., 11., 12., 13., 14., 15..

The parameters assessed in T.E.E are

a) Diameter of LMCA at ostial level, shaft level, at the level of bifurcation

b)Length of left main coronary artery

c)Presence of atheroma.

d)Presence of turbulence

e) Diastolic flow velocity before level of stenosis,at the level of stenosis,and after the level of stenosis.

f)TVI before the level of stenosis and at the trans stenotic level were measured.

Statistical analysis

All data are expressed as the mean value ± SD The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2008)** developed by Center for Disease Control, Atlanta.

Using this software, range, frequencies, percentages, means, standard deviations and 'p' values were calculated. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATIONS

Table 1.: Age

Age group (in years)	Cases	
	(no)	%
Up to 40 years	4	8
41-50 yrs	16	32
51-60 yrs	21	42
> 60 yrs	9	18
Total	50	100
Range	36 – 70 years	
Mean	53.3 years	
SD	8.1 years	

AGE DISTRIBUTION

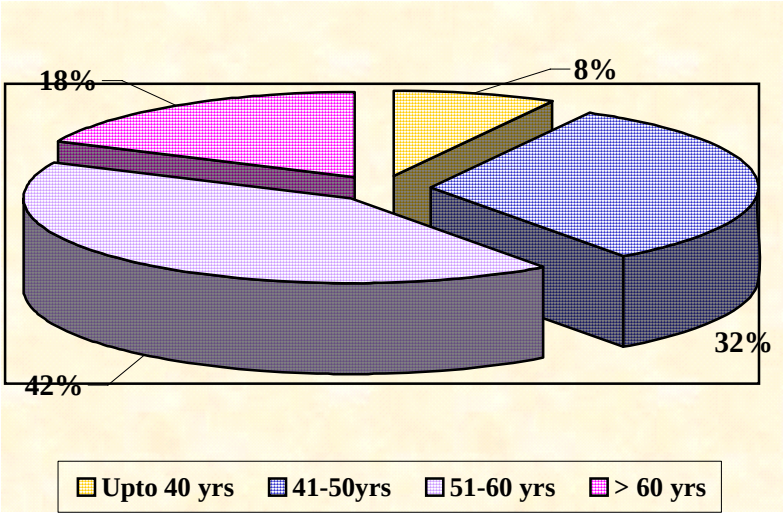


Table 2 : Sex distribution

Sex	Cases	
	No	%
Male	45	90
Female	5	10
Total	50	100

SEX DISTRIBUTION

Table 3.: Diagnosis

Diagnosis	Cases	
	No	**%**
Stable Angina	11	22
Unstable Angina	6	12
AWMI	17	34
IWMI	14	28
PWMI	1	2
Old IWMI	1	2
Total	50	100

DIAGNOSIS

Table 4 : D M

D M	Cases	
	No	%
Yes	15	30
No	35	70
Total	50	100

D M

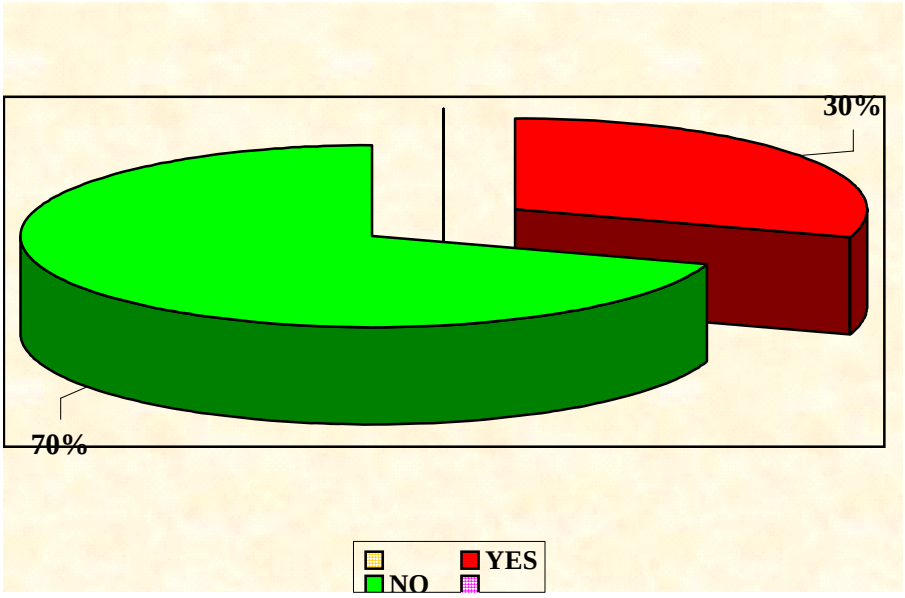


Table 5: LVID & LVEF

PARAMETER	Mean ± S.D	Range
LVID(D)	5.1±0.67	3.5 – 7.1
LVID(S)	3.8±0.86	1.9 – 6
LVEF(T)	46.5±13.9	17 – 73
LVEF(QLAB)	44.7±11.3	25 - 73

LVID & LVEF

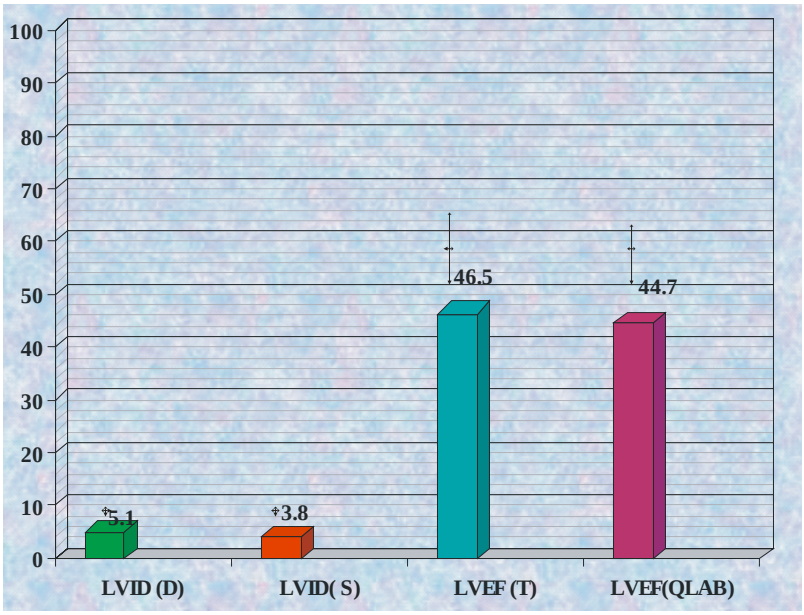


Table 6 : E / A / DT

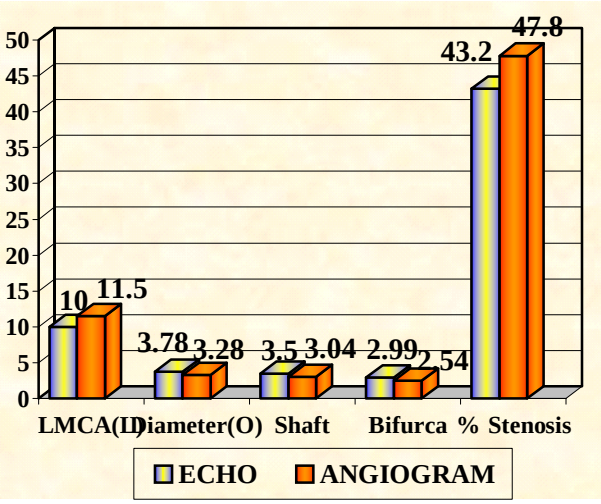
Parameter	Mean ±S.D	Range
E	64.2±15.4	36 – 110
A	61±10.9	27 – 79
DT	139±33.5	56 - 238

E / A / DT

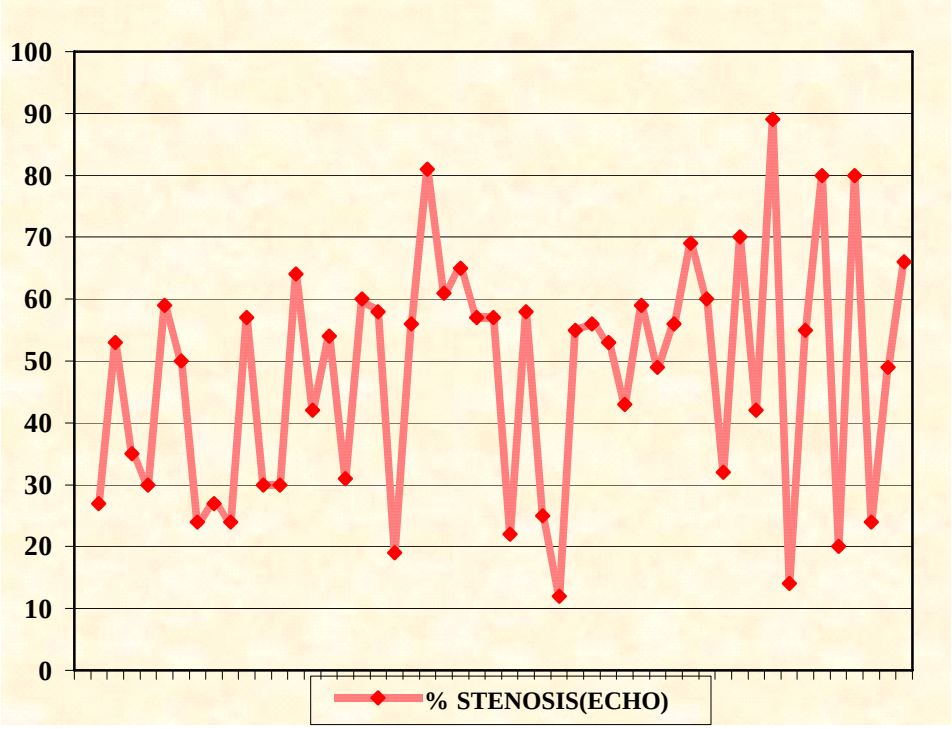
Table 7 : Correlation between Echo findings and Angiogram findings

Parametetr	Values as per						Correlation Coefficient Between Echo and Angiogram values
	Echo			Angiogram			
	Range	Mean	S.D.	Range	Mean	S.D.	
LMCA(L)	3.9-18.8	10	3.2	4.5-22	11.5	4.2	0.7137
Diameter(O)	2-6.9	3.78	1.12	0.96-5.31	3.28	0.84	0.3562
Shaft	1.7-5.2	3.5	0.97	1.4-5.03	3.04	0.89	0.0267
Bifurca	1.3-6.3	2.99	0.87	1.11-4.37	2.54	0.84	0.3817
% Stenosis	12-89	47.8	19.1	20-90	43.2	15.7	0.6007

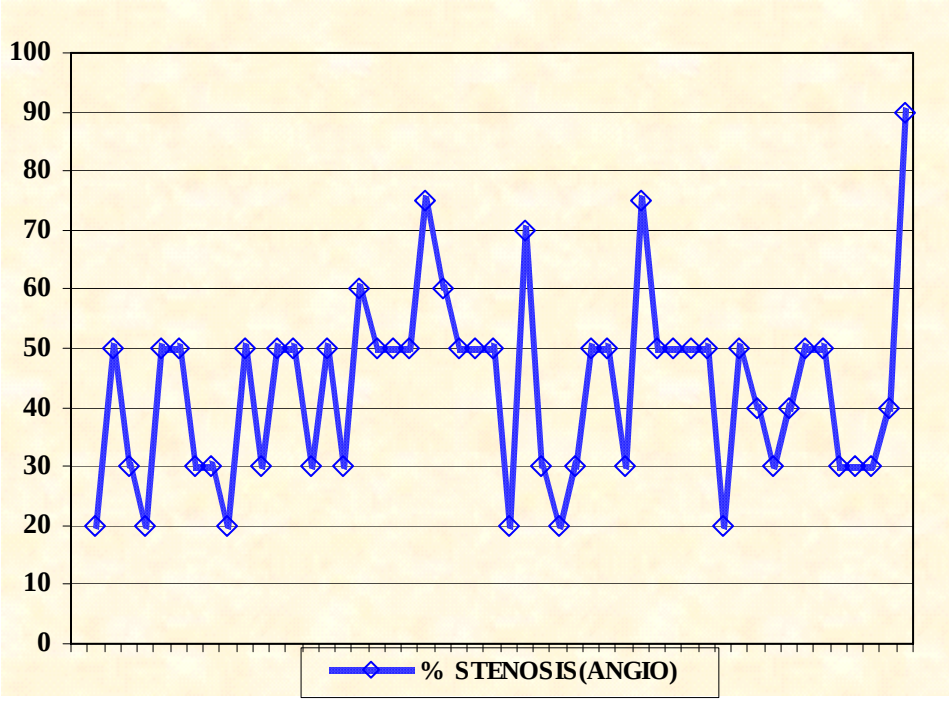
ECHO & ANGIOGRAM VALUES



% STENOSIS(ECHO)



% STENOSIS(ANGIO)



% STENOSIS(ECHO)
& % STENOSIS(ANGIO)

Table 8 : TVI (BEFORE) & TVI (AFTER) STENOSIS

PARAMETER	**TVI(BEFORE)**	**TVI(AFTER)**
Range	5-53	12-103
Mean	25.2	51.8
S.D.	11.4	21.4

TVI(BEFORE) & TVI (AFTER)STENOSIS

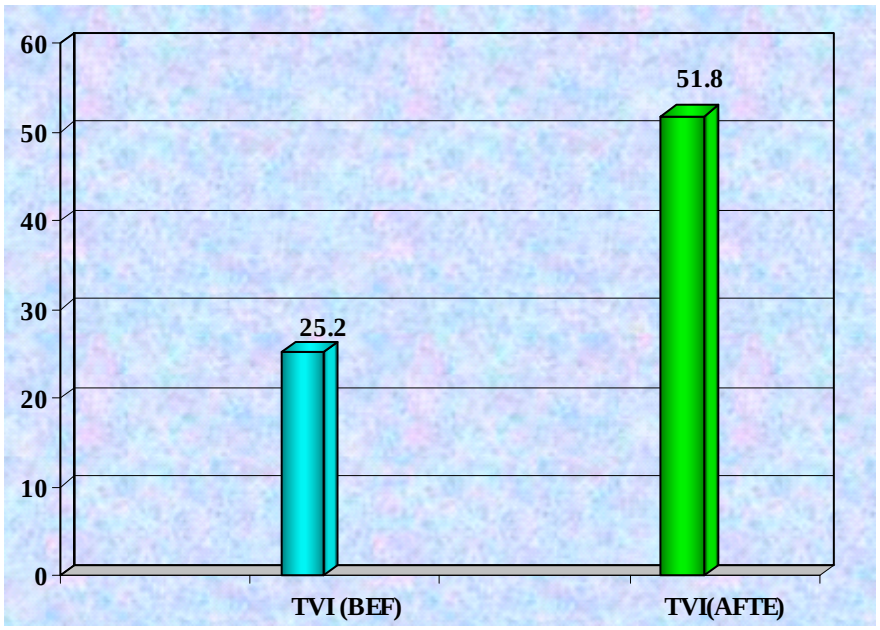


Table 9 : Relationship between pre/post TVI and % Stenosis as per Angiogram findings

Pre / Post TVI	No. of cases	% Stenosis as per Angiogram			
		< 50%		≥ 50%	
		No.	%	No.	%
< 0.5	27	3	11.1	24	88.9
≥ 0.5	23	19	82.6	4	17.4
'p'		0.0001			
		Significant			

RELATIONSHIP BETWEEN

PRE/POST TVI &

% STENOSIS

(Angiogram values)

% STENOSIS(ECHO

Table 10: Difference in the values of % Stenosis between Echo findings and Angiogram findings

% Stenosis difference between Echo findings and Angiogram values	Cases	
	No.	%
< 10 %	30	60
> 10%	20	40
Total	50	100
Range	-31% - (+ 59%)	
Mean	4.6%	
S.D.	15.8%	

Transesophageal Doppler echocardiographic determination of percent area stenosis

According to the continuity equation, coronary flow volume at the prestenotic segment is equal to that at the stenotic segment in the absence of branches between the two segments. As flow volume is derived from the product of CSA with the TVI, thus,

$$\text{Prestenotic CSA} \times \text{Prestenotic TVI} = \text{Stenotic CSA} \times \text{Stenotic TVI} \quad (1)$$

The percent area stenosis (%CSA) is written as

$$\%\text{CSA} = 100 \times (1 - \text{Stenotic CSA} / \text{Prestenotic CSA}) \quad (2)$$

Rearranging (1), (2) leads to

$$\%\text{CSA} = 100 \times (1 - \text{Prestenotic TVI} / \text{Stenotic TVI}) \quad (3)$$

Coronary angiographic data

The length of Left main coronary artery in angiogram varies between 4.5 to 22 mm with a mean of 11.5 and a standard deviation of 4.2.The diameter of left main coronary artery at ostial level ranges from 0.96 to 5.31 mm with a mean of 3.28 mm and standard deviation of 0.84. The diameter of left main coronary artery at shaft level ranges from 1.4 to 5.03 mm with a mean of 3.04 mm and standard deviation of 0.89. The diameter of left main coronary artery at distal level ranges from 1.11 to 4.37 mm with a mean of 2.54 mm and standard

deviation of 0.84. The calculated percent DS ranged from 20% to 90% (mean 43.2 and standard deviation of 15.7);

Transesophageal Doppler echocardiographic data

A localized increase in velocity appeared on Doppler color flow mapping as a localized area of aliased and disturbed signal in all 50 patients studied. In all patients, peak diastolic velocity and diastolic TVI at the prestenotic site were obtained by pulsed Doppler echocardiography; transstenotic diastolic peak velocity and TVI were obtained in all patients with the use of either pulsed Doppler echocardiography or high pulse repetition frequency Doppler or continuous wave Doppler echocardiography. The peak diastolic velocity at the stenotic region was 12 to 103 cm/ with a mean of 51.8 and standard deviation of 21.4 and was significantly higher than that measured at the prestenotic segment 5 to 53 cm/ with a mean of 25.2 and standard deviation of 11.4.. A good linear correlation was found between the catheterization-derived and TEDE-derived percent CSA stenosis (correlation coefficient of 0.6007)(significant>0.5)and length of left main coronary artery(correlation coefficient of 0.7137(significant>0.5).A good linear relation was also found between the catheterization-derived percent DS and the simple prestenotic to stenotic TVI ratio, which was a good discriminator for distinguishing patients with ≥50% diameter reduction from those with <50% diameter reduction. All patients with ≥50% diameter reduction stenosis at catheterization had a TVI ratio ≤0.5 and only four of the 50 patients with <50% diameter

reduction had a TVI ratio ≤0.5. Thus, a TVI ratio ≤0.5 predicted ≥50% diameter reduction with 90% sensitivity and 85% specificity. The diameter of the coronary vessels did not correlate because the lateral resolution of the two-dimensional sector scan is too low to allow reliable measurements of dimensions of coronary arteries. The present study demonstrates that velocity measurements derived from TEDE can be used for quantitating stenosis of the LMCA . .

Use of the continuity equation

Three previously published reports 1., 2., 8. based on invasive Doppler measurements have proposed the application of the continuity equation to estimate the severity of coronary stenosis. However, the methods used in these previously published studies 1., 2., 8. remain invasive, requiring cardiac catheterization, and cannot be repeated without risk during serial follow-up studies. Furthermore, in their consecutive series of 52 patients undergoing percutaneous transluminal coronary angioplasty, Di Mario et al. (8) found that, although accurate for quantitation of lesion significance, use of the continuity equation employing intracoronary guide wire Doppler measurements is difficult and impractical for clinical application because high quality intrastenotic Doppler signals are obtained in only a minority of cases.

In our study, we used a noninvasive approach—TEDE—which can be used more easily in a clinical setting. We found a good linear relation between catheterization-derived and TEDE-

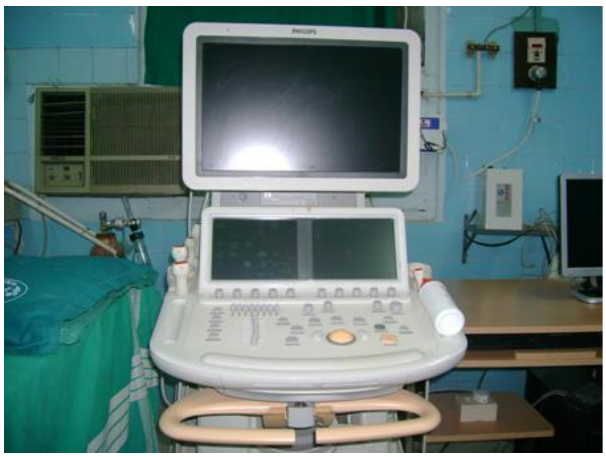
derived percent CSA stenosis using the continuity equation. Despite this good linear relation, TEDE measurements significantly underestimated the actual percent CSA stenosis. This discrepancy between transesophageal Doppler measurements and the actual percent CSA may be explained by differences in the cross-sectional velocity profile that may occur between the prestenotic and stenotic segment sites. Fluid mechanics theory and previous published experimental studies suggest that cross-sectional velocity profile in a small conduit, like coronary arteries, is parabolic at a low Reynolds number, but flattens when velocities increase, like in a stenosis where flow becomes turbulent 20., 21., 22., 23., 24.. We have recently confirmed in a clinical study, based on computer analysis of digitally transferred transesophageal color coronary flow maps, that the cross-sectional velocity profile is parabolic in the normal proximal LAD, whereas it becomes flatter when velocities increase, like at the site of stenosis or after intravenous injection of dipyridamole (25). For clinical purposes, however, the simple TVI ratio may be used for predicting with good accuracy the percent DS, which is also a well recognized variable of stenosis severity.

Clinical implications

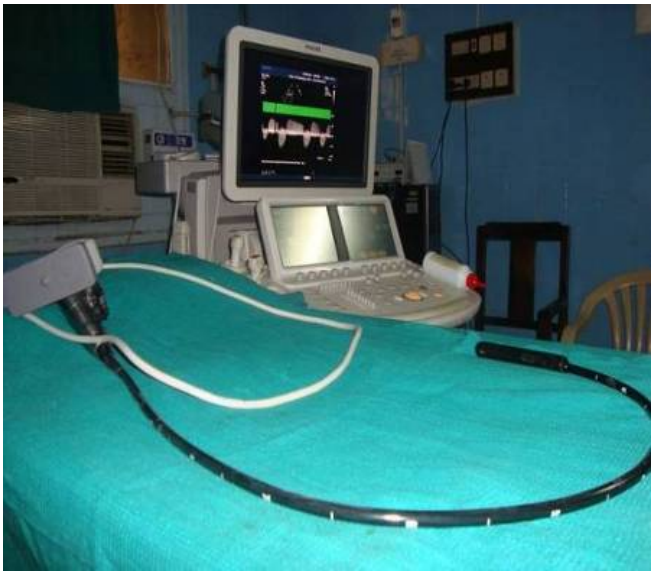
Our data suggest that TEDE allows quantitation of stenosis of the LMCA .This method offers the advantage of a noninvasive technique, which can be applied in many echocardiographic laboratories. Our TEDE method might also represent an adjunct to coronary angiography to evaluate mild to moderate stenosis. Conventional angiography with visual interpretation, as currently used in many catheterization laboratories, has significant limitations in the

assessment of coronary stenosis (26). In patients with severe coronary diameter reduction on the angiogram, there is usually no difficulty in ascertaining the functional severity of the lesion and in making clinical decisions. In contrast, in some patients with angiographically documented mild to moderate stenosis, it is sometimes difficult to evaluate the actual physiologic consequences of the obstruction. Also, contrast angiography, even when using quantitative angiography, is not necessarily suitable for evaluating the results of catheter-based interventions owing to the eccentricity of the vascular lumen after angioplasty (27). As TEDE measurements using the continuity equation do not rely on any geometric assumption, it might help to confirm the functional severity of stenosis visualized by angiography, especially in cases of mild to moderate lesions and after catheter-based interventions. TEDE also provides a method for quantitating the severity of the stenosis without inserting any catheter or guide wire into the stenotic segment. In contrast, Doppler catheters or guide wires reduce the actual CSA of the stenosis and may disturb flow field, thus leading to some errors in measurements.

PHILIPS iE 33 – ECHOCARDIOGRAPHY SYSTEM



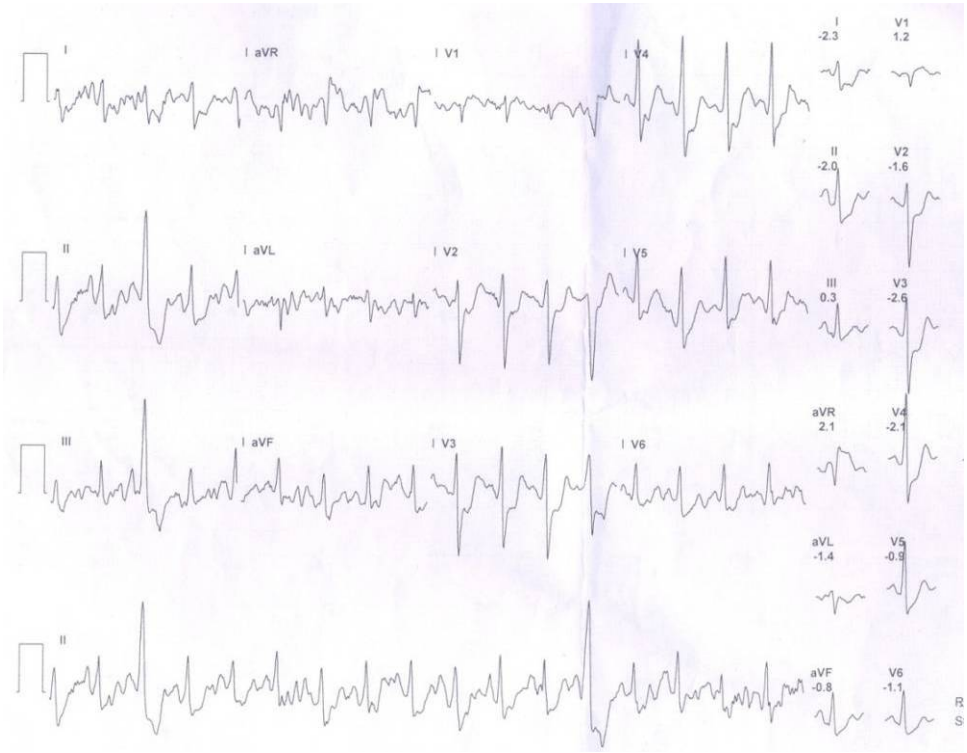
TRANSESOPHAGEAL ECHOCARDIOGRAPHY



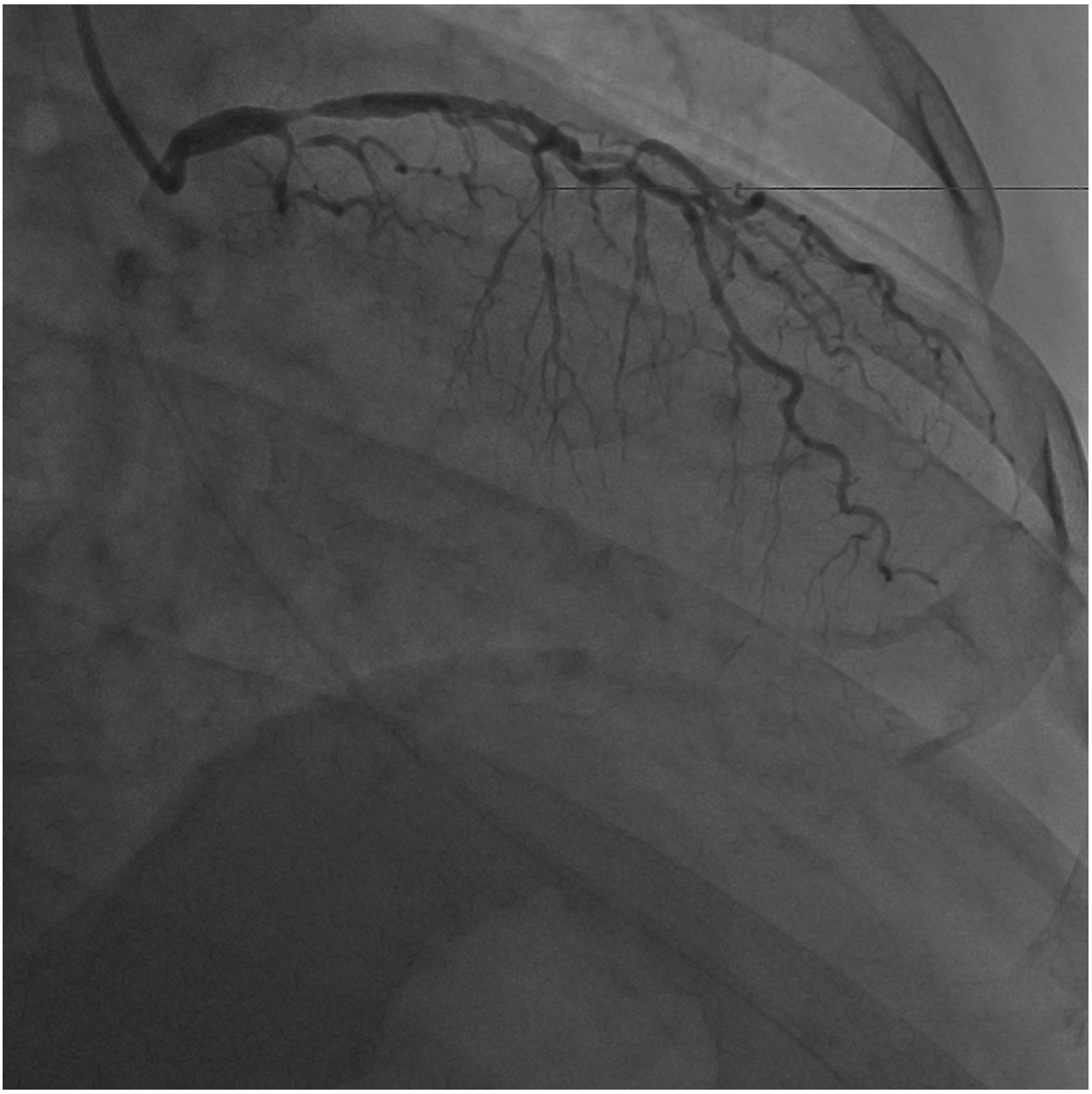
TOSHIBA DIGITAL FLAT PANEL RECORDER

CATH LAB CONSOLE

Two examples of LMCA stenosis patients with ECG,CAG,typical phasic coronary flow–velocity signals recorded by TEDE in the prestenotic and transstenotic regions are illustrated below

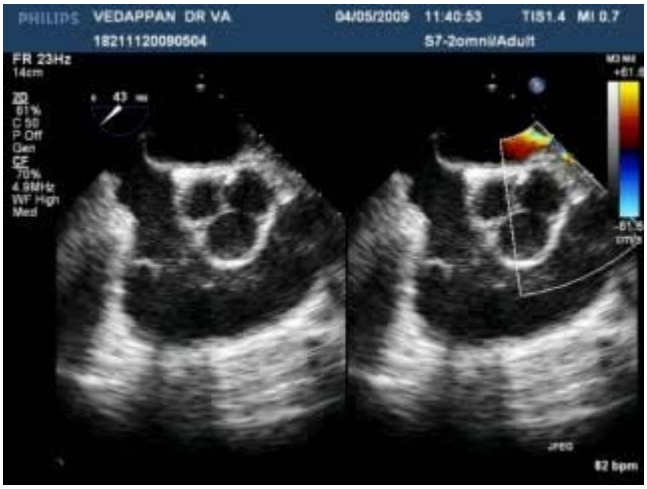


Patient underwent TMT with BRUCE PROTOCOL achieved 95% of target heart rate and10.7 METS achieved. He had hypertensive response during exercise and developed 2mm ST depression in anterior chest leads V2-V6& ST elevation in aVR that persisted 2 minutes into recovery.

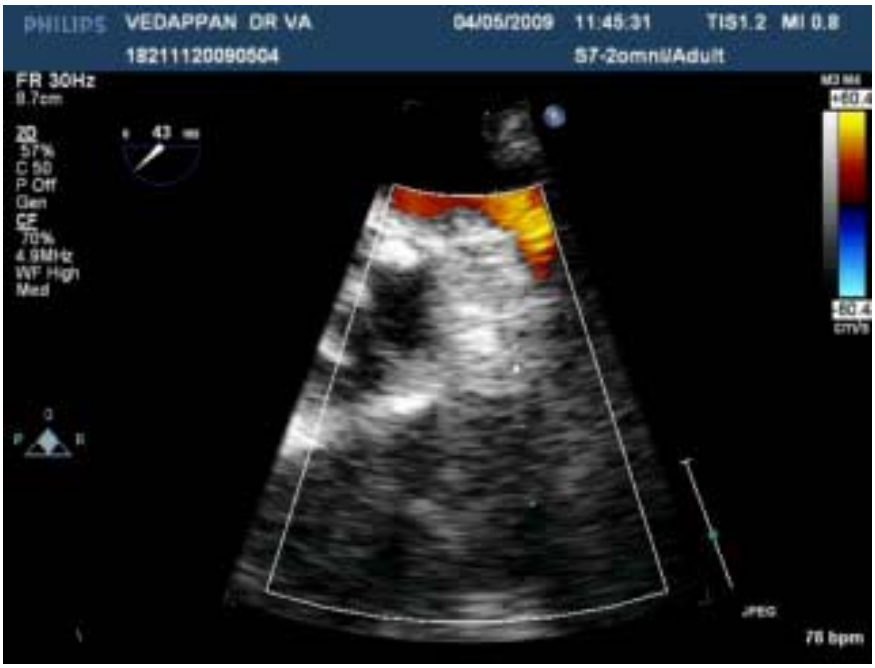


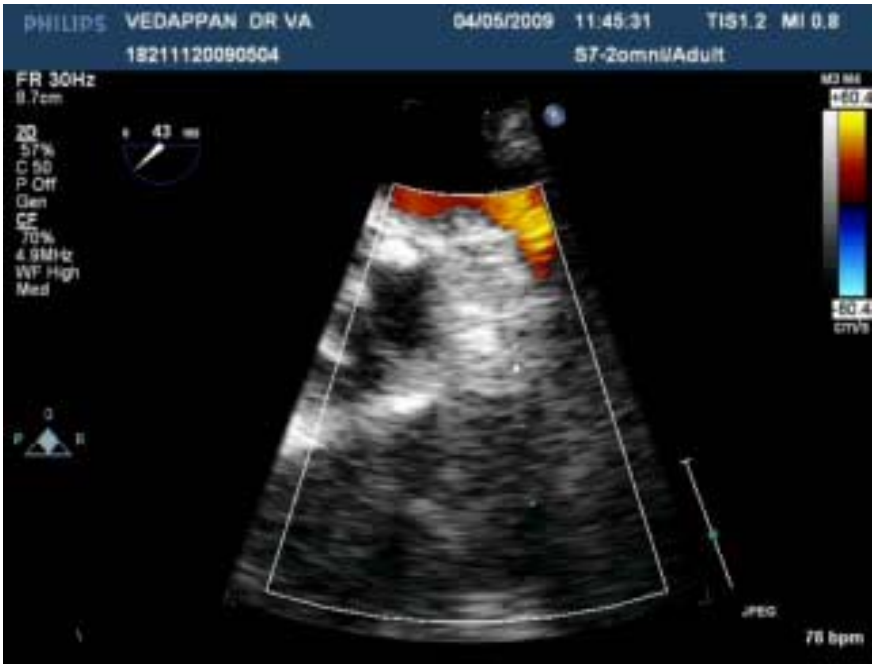
VEDAPPAN CAG

Trans Esophageal Echocardiogram



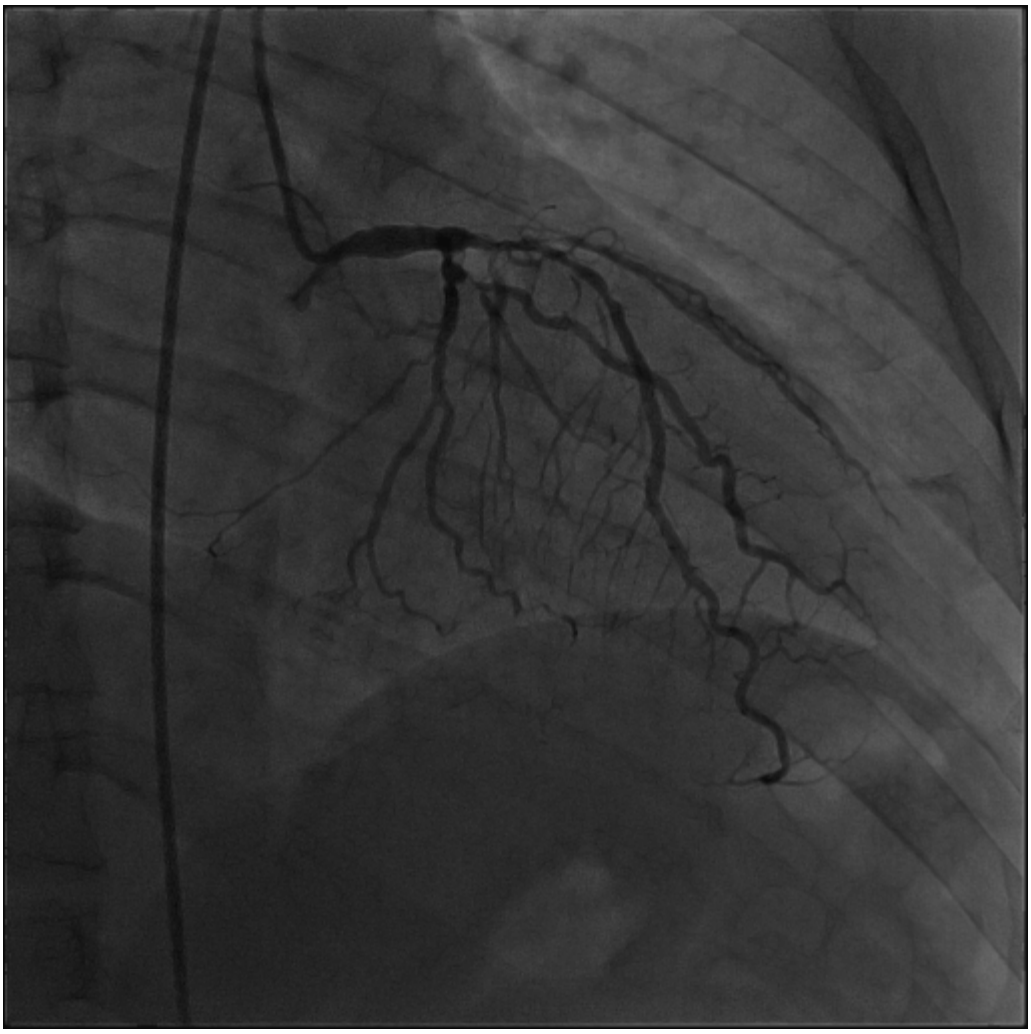
Turbulence in LMCA at branching point



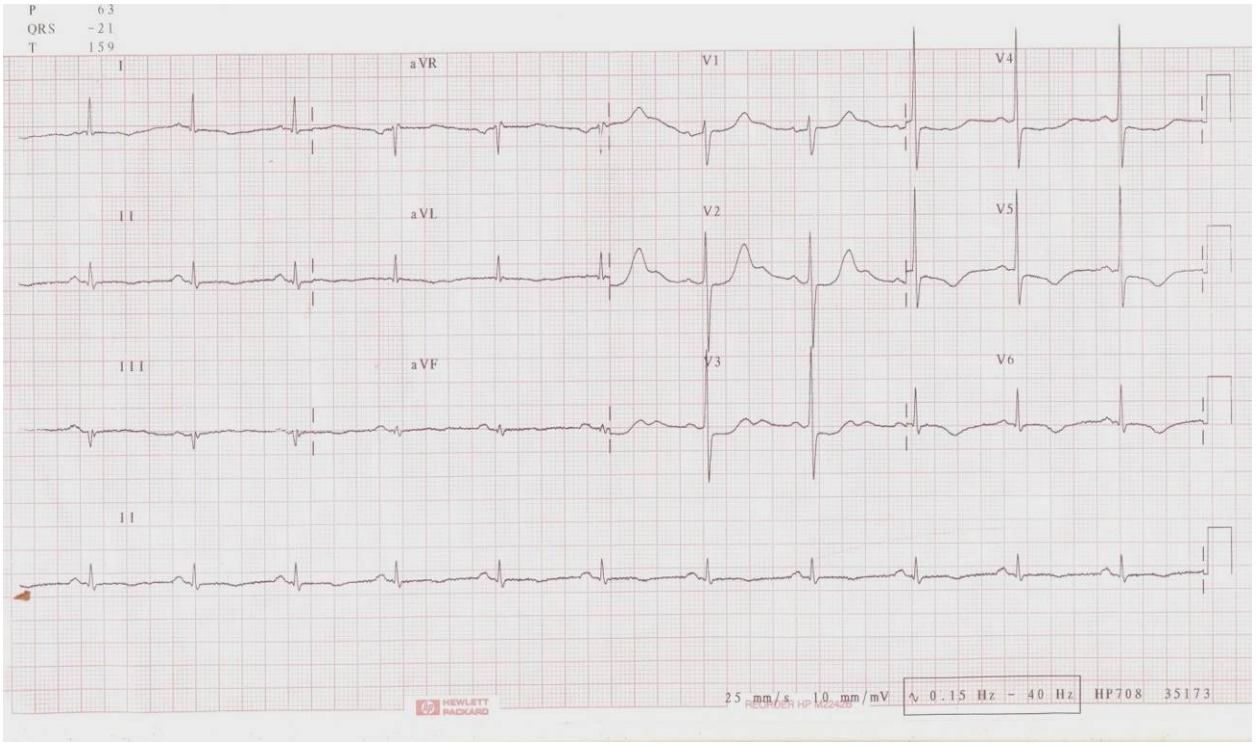


High velocity Jet in LMCA

CASE2

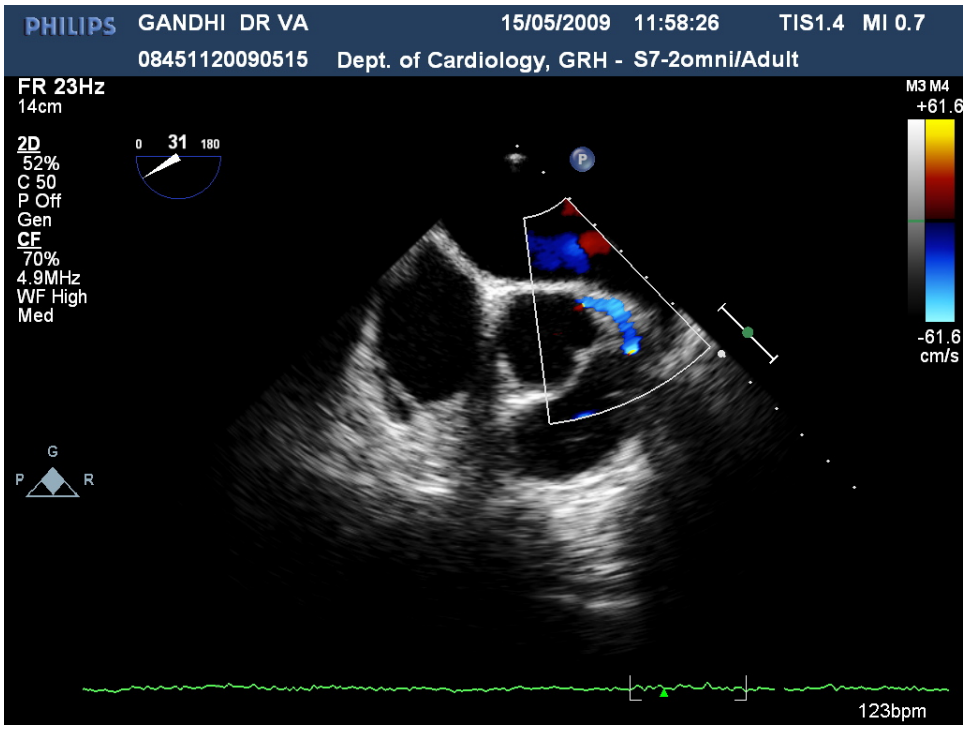


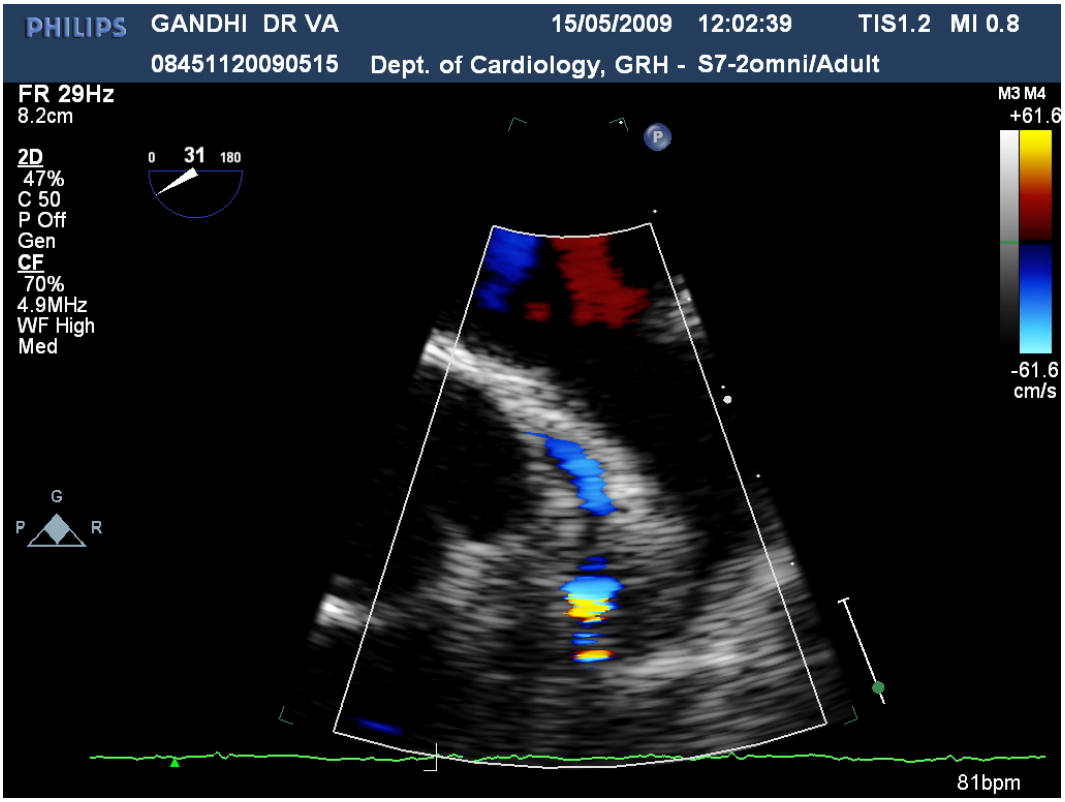
GANDHI CAG



ECG OF GANDHI

TEE

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C 50
P Off
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-61.6
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G
P R
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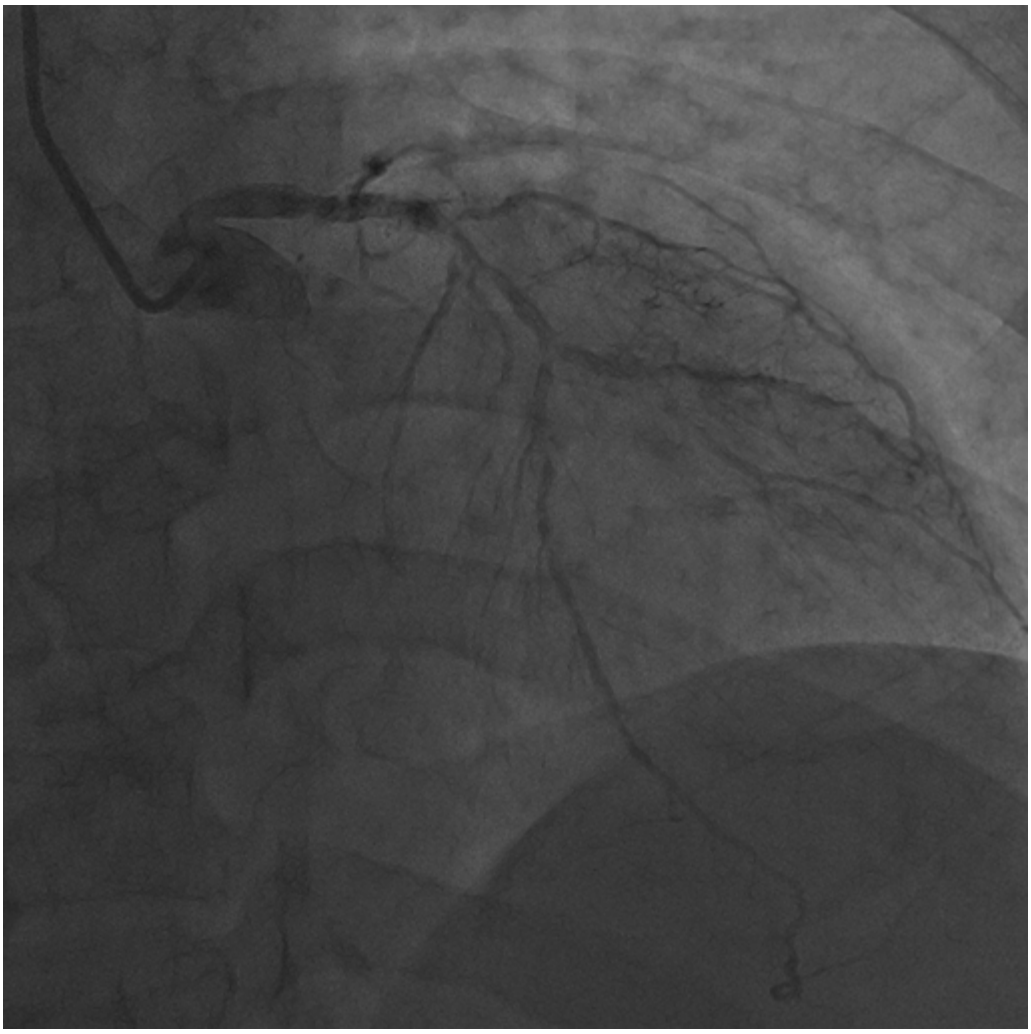
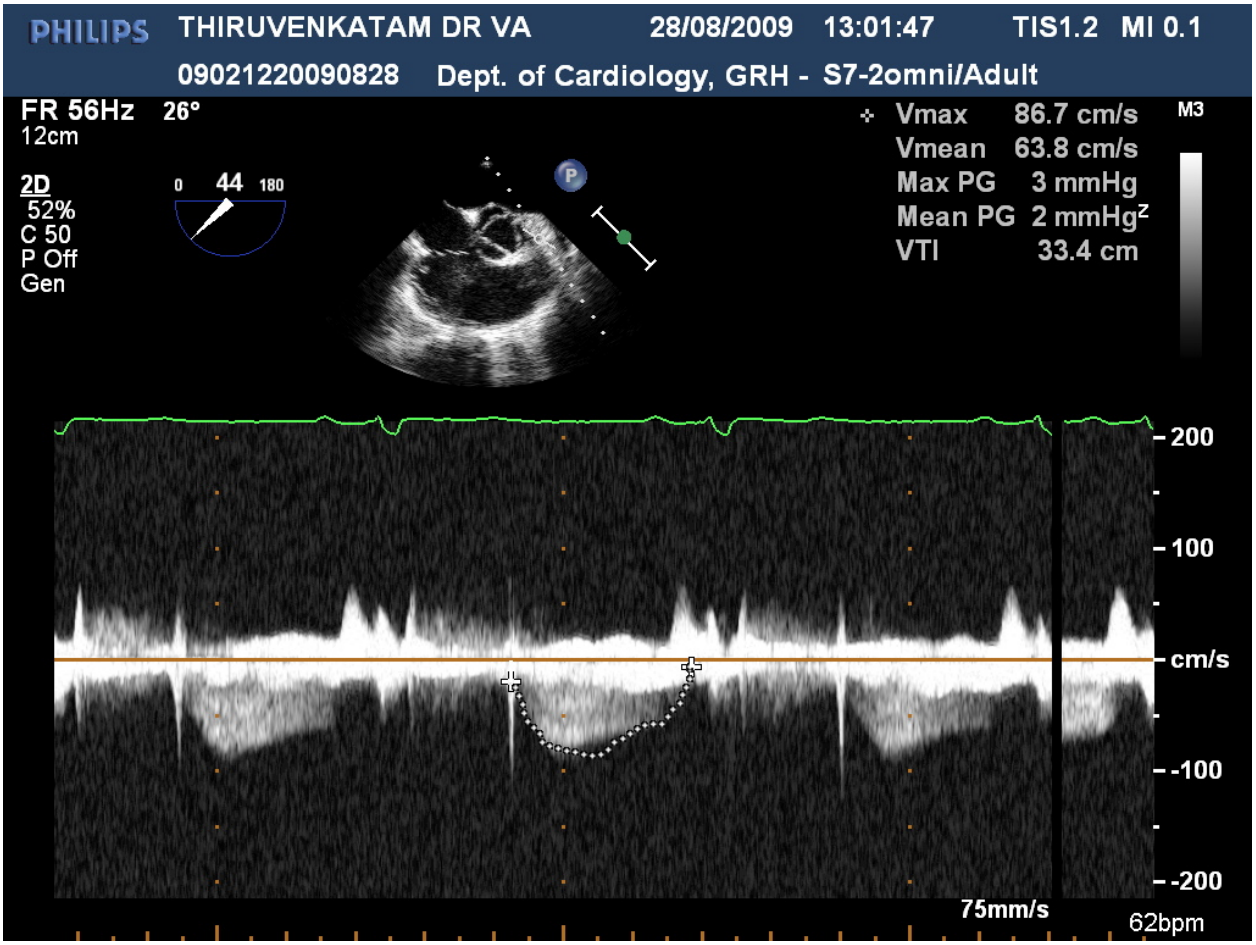


THIRUVENKATAM CAG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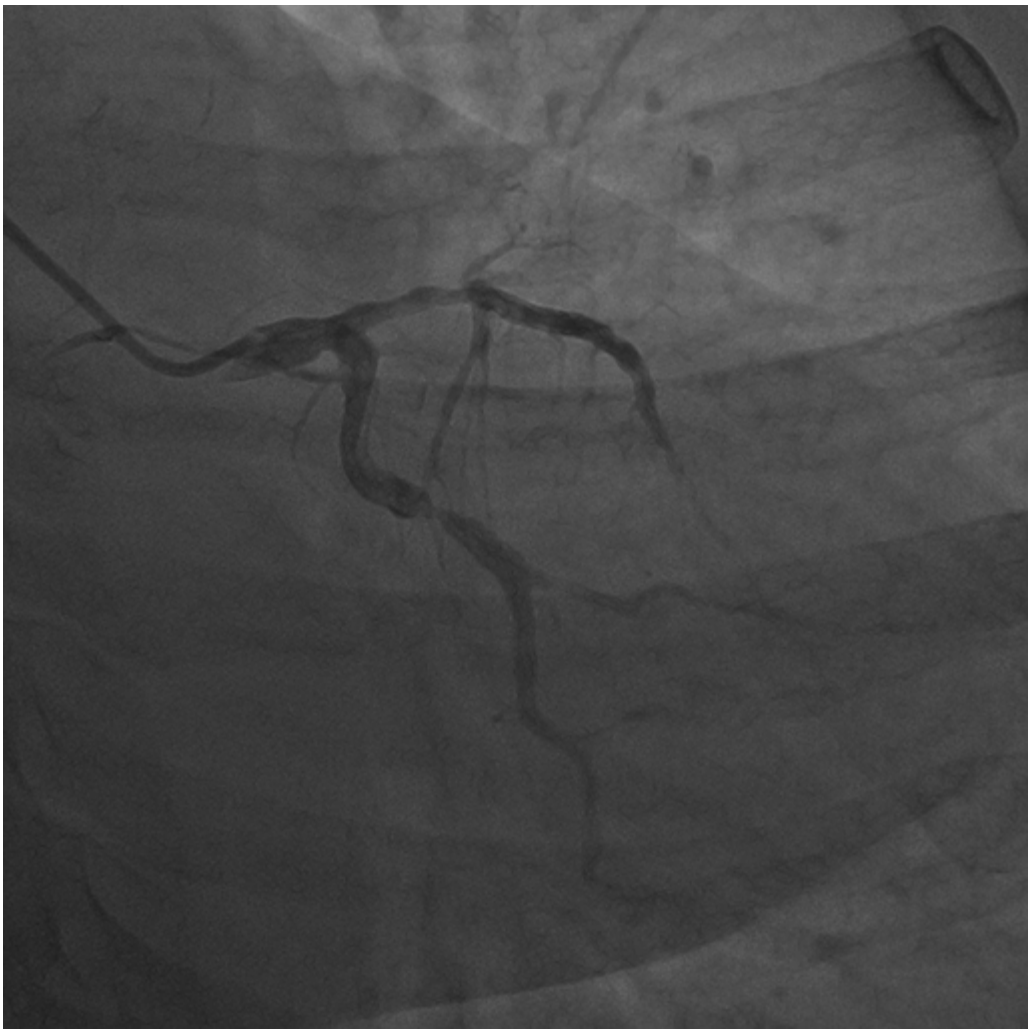
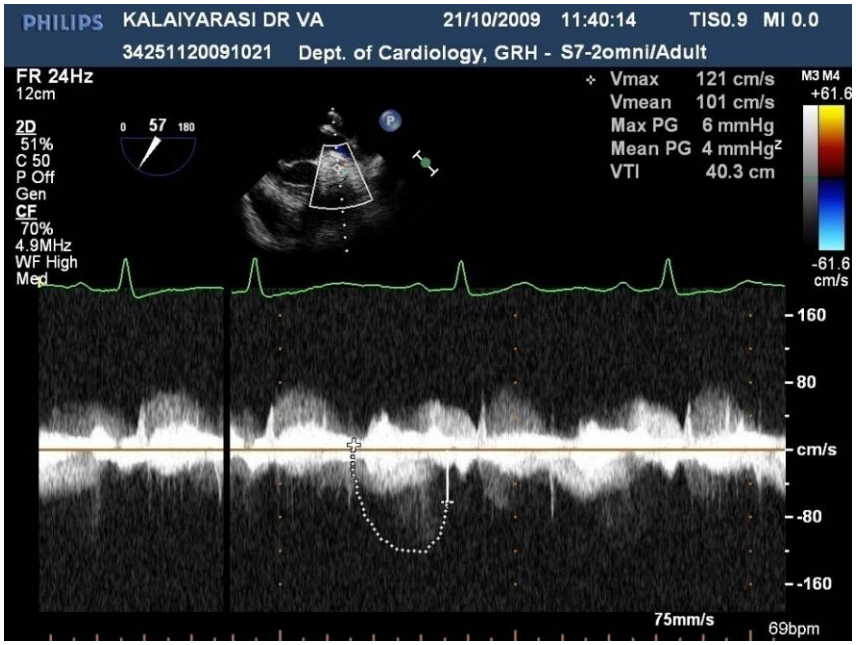
26°
12cm
2D
52%
C 50
P Off
Gen
0
44
180
Vmax 86.7 cm/s
Vmean 63.8 cm/s
Max PG 3 mmHg
Mean PG 2 mmHg
VTI 33.4 cm
M3
200
100
cm/s
-100
-200
75mm/s
62bpm



CAG KALAIARASI



Quantification of LMCA Stenosis

- 100(I–pre-stenotic TVI/stenotic TVI)
  = 100 (1 – 30/40)
  = 100 (1 – 0.75)
  = 25%

LIMITATIONS OF THE STUDY

Some limitations of this new technique must be addressed. In our study, we were able to obtain interpretable TEDE flow recordings at the site of both prestenotic and stenotic regions in only 50 of60 patients.

The present study was designed to test the ability of TEDE, in comparison with digital quantitative angiographic data, for quantitating proximal LCA stenosis based on the continuity equation. However, the accuracy of this method in detecting the absence or presence of a significant stenosis in the proximal LCA in patients with various heart diseases on a large screening basis remains to be determined.

Only patients with stenosis of the LMCA were studied, and no attempt was made to explore circumflex and right coronary arteries. Owing to more severe angulation and tortuosity of these vessels, adequate Doppler signals, as well as a good alignment between the ultrasound beam and the axial flow direction, appear to be more difficult to obtain in the right coronary artery and the circumflex artery. However, computation of severity of stenosis of the LMCA provides clinically important information, because a major amount of myocardium is perfused by these vessels.The lateral resolution of the two-dimensional sector scan is too low to allow reliable measurements of dimensions of coronary arteries.

Continuity equation in coronary artery stenosis

At any instant and at any site, the coronary flow rate (CFR) is equal to the product of spatial average velocity with the CSA:

$$\text{CFR} = \text{Spatial average velocity} \times \text{CSA}. \quad (4)$$

In the prestenotic segment, assuming a parabolic velocity profile—that is, spatial average velocity is equal to half the peak axial velocity—CFR is written as

$$\text{CFR} = \text{Spatial average prestenotic velocity} \times \text{Prestenotic CSA} \quad (5)$$

$$\approx \frac{1}{2}\text{Peak axial prestenotic velocity} \times \text{Prestenotic CSA}. \quad (6)$$

In the stenotic segment, assuming a flat velocity profile—that is, spatial average velocity is equal to peak axial velocity—CFR is written as

$$\text{CFR} = \text{Spatial average stenotic velocity} \times \text{Stenotic CSA} \quad (7)$$

$$\approx \text{Peak axial stenotic velocity} \times \text{Stenotic CSA}. \quad (8)$$

Rearranging (6), (8) demonstrates that the percent CSA reduction may be written as

$$\%\text{CSA} = 100(1 - \text{Stenotic CSA} / \text{Prestenotic CSA})$$

$$= 100\left(1 - \left[\frac{1}{2}\text{Peak axial prestenotic velocity}\right] / \text{Stenotic peak axial velocity}\right). \quad (9)$$

Owing to the width of the pulsed Doppler sample volume and of the continuous wave Doppler beam compared with the small diameter of the coronary vessel, one can assume that peak velocity measurements derived from TEDE correspond to the actual peak axial velocity

within the vessel at any instant. In our study, TVIs were measured by planimetry of the peak velocity curve obtained by tracing the outer border of the Doppler spectral display on the recording, so that Equation 9 can be rewritten by substituting peak velocity by TVI:

$$\%CSA = 100\left(1 - \left[\frac{1}{2}\text{Prestenotic TVI}\right] \;/\; \text{Stenotic TVI}\right) \quad (10)$$

Conclusion

Transoesophageal Doppler assessment of coronary blood flow is a highly sensitive and specific non invasive method in the diagnostics of stenotic and occlusive atherosclerosis of the main coronary arteries.

A modified continuity equation is haemodynamically correct and allows with application of Transoesophageal Doppler allows the accurate calculation of the coronary artery stenosis percentage.

The peak diastolic velocity of coronary blood flow (equal to 1.4 $m.s^{-1}$ in the LMCA,0.9 $m.s^{-1}$ in the LAD ,and 1.1 $m.s^{-1}$ in the LCX) alongside the aliasing phenomenon is a Doppler criterion of haemodynamically significant stenosis.

Break of colour mapping,absence of Doppler spectrum and registration of retrograde blood flow during late diastole are Doppler echocardiographic criteria for coronary coronary artery occlusion.

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PROFORMA

ASSESMENT OF LEFT MAIN CORONARY STENOSIS BY TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Name of the patient : Age : Sex:

CD no : Address :

Clinical diagnosis :

Brief history :

General examination :

Build and nourishment

Height

Weight

Waist circumference

Hip measurement

Waist hip ratio

Pulse :

Blood pressure :

Cardiovascular system examination :

Respiratory system examination :

Abdomen :

CNS examination:

Investigations :

Urine - Albumin

- Sugar

- Deposits

Blood - Hb

- ESR

Blood urea

Blood sugar - F

- PP

Serum creatinine

Fasting lipid profile

Total – C LDL – C HDL – C

TGL VLDL

ECG

Echocardiogram :

2 D and M mode echo :

LVID (d) LVID (s)

EF

Doppler echo :

Mitral inflow : E

A

DT

E/A

TDI - S' E'

A' E/E^1

3 D echo:

TRANSESOPHAGEAL ECHOCARDIOGRAPHY:

LENGTH OF LEFT MAIN ARTERY:

DIAMETER OF LMCA:

OSTIUM LEVEL:

SHAFT LEVEL:

DISTAL LEVEL:

TVI BEFORE STENOSIS:

TVI AFTER STENOSIS:

PRESTENOTIC/POST STENOTIC RATIO:

% OF STENOSIS OF LMCA:

CORONARY ANGIOGRAPHIC DATA:

LENGTH OF LEFT MAIN ARTERY:

DIAMETER OF LMCA:

OSTIUM LEVEL:

SHAFT LEVEL:

DISTAL LEVEL:

% OF STENOSIS OF LMCA:

S.NO	NAME	AGE	SEX	DIAGNOSIS	DM	ECG	ST↑	ST↓	T↓	LVID(D)	LVID(S)	LVEF(T)	LVEF(QLAB)	MR	RWMA	E	A	DT	S'	E'	A'	E/E'
										(CM)	(CM)	(%)	(%)			cm/s	cm/s	msec	cm/s	cm/s	cm/s	(ratio)
1	Vedappan	53	M	Stable angina	Nil	ST↑avR	avr	v2- v6	v2	4.6	2.68	72	51	nil	nil	63	61	126	7	8	6	8
2	Gandhi	50	M	PWMI	nil	QV8V9	V8V9	_	_	4.5	3.3	51	65	yes	IWPW	62	47	134	7	6	6	10
3	Singadurai	50	M	IWMI	nil	Qii,iii,avf	ii,iii,avf	_	L1avl	3.7	2.6	55	49	Gri	IW,IS	91	69	115	6	4	7	22
4	Leelavathy	42	F	AWMI	Nil	QV1-V6	V1-V6	ii,iii,avf	iii,avf	5.6	5.2	26	25	Gri	AW,AS	79	51	154	4	4	6	20
5	Thangamuthu	50	M	IWMI	Nil	ST↑iiiiiavf	ii,iii,avf	_	_	5.2	4.4	30	26	nil	IW.IS	71	73	137	6	7	8	10
6	Thaniperavi	40	M	IWMI	Nil	QsLiii,avf	_	V3V4	iii,avf	4.7	2.8	60	56	Gri	Iw	65	72	124	7	6	6	11
7	Shikh hussain	52	M	AWMI	nil	ST↑V2-V4	V2-V4	Li,avl	V1-V2	5.2	4.2	44	37	nil	AW,AS	67	54	113	6	5	5	13
8	Marimuthu	48	M	AWMI	yes	ST↑V1-V6	V1-V6	Li,avl	L1avl	5.6	5.1	27	30	nil	AW,AS	67	69	129	7	9	8	7
9	Ramakrishnan	55	M	IWMI	Nil	Qii,iii,avf	ii,iii,avf	_	L1avl	4.7	2.8	69	60	nil	IW.IS	42	76	145	6	6	9	7
10	Annadurai	43	M	AWMI	yes	QSV1-V3	no	V1-V3	V1-V2	4.1	3.3	40	45	nil	AW,AS	49	58	174	5	5	9	10
11	Mohammedali	46	M	AWMI	Nil	QSV1-V3	no	V1-V3	V1-V3	5.2	4.4	42	45	nil	AW,AS	65	54	120	7	4	6	16
12	Ayyavu	58	M	AWMI	Nil	QSV1-V3	no	V1-V3	V1-V3	4.5	3.3	50	46	nil	AW,AS	50	58	174	5	5	9	10
13	Ramalingam	50	M	Stable angina	Nil	Normal	no	nil	nil	6	3.8	65	69	nil	IW,IS	76	71	140	5	5	7	15
14	Thangaraj	70	M	IWMI	Nil	QLiii,avf	ii,iii,avf	nil	nil	4.8	3.4	41	45	nil	IW,IS	75	73	117	6	8	7	9
15	Krishnamoorthy	65	M	Stable angina	Nil	ST↓V4V6	no	V4-V6	V4-V6	3.8	1.9	68	73	nil	nil	62	53	130	7	4	6	15
16	Thiruvengadam	68	M	AWMI	Nil	QV1-V4	V1-V4	nil	nil	5.8	4.5	45	49	nil	AW,AS	42	68	132	6	4	7	10
17	Balaguru	56	M	AWMI	Nil	QV1-V4	no	V2-V6	V2-V6	7.1	6	32	38	nil	AW,AS	65	45	133	4	5	5	13
18	Krishnamoorthy	58	M	Stable angina	Nil	Normal	no	nil	nil	4.9	3.5	54	48	nil	nil	62	53	127	7	4	6	15
19	Elango	45	M	IWMI	yes	QLiii,avf	Lii,iii	nil	nil	4.3	3.6	37	35	Gri	IW,IS	72	52	113	5	7	7	10
20	Padmavathy	66	F	IWMI	Nil	QLiii,avf	no	Liii.avf	V5,V6	4.5	2.5	73	56	Gri	IW,IS	72	64	136	5	6	8	12
21	Sakthivel	50	M	Stable angina	Nil	Normal	no	nil	nil	5	3.9	60	48	Gri	nil	75	73	133	6	8	7	9
22	Babu	48	M	AWMI	Nil	ST↑V1-V6	V1-V4	nil	nil	5.2	4.2	27	32	nil	AW,AS	110	38	120	6	7	6	15
23	Shanmugam	61	M	IWMI	yes	ST↑Liiiavf	Lii,iii	nil	nil	5.1	4	38	43	Gri	IW,IS	46	60	145	6	5	9	9
24	Murugesan	58	M	IWMI	Nil	QLiii,avf	_	Liii.avf	Liii.avf	5.4	4	42	36	nil	IW,IS	43	76	143	6	6	9	7
25	Ramiah	60	M	IWMI	Nil	QLiii,avf	no	Liii.avf	Liii.avf	4.4	3.6	26	34	nil	IW,IS	78	79	187	5	4	7	19
26	Ramakrishnan	55	M	IWMI	Nil	QLiii,avf	_	Liii.avf	Liii.avf	4.8	4.2	28	34	Gri	IW,IS	36	46	56	5	5	5	7
27	Sethuraman	52	M	IWMI	Nil	QLiii,avf	Lii,iii	nil	nil	5.3	3.8	54	51	Gri	IW,IS	64	67	132	5	6	8	10
28	Kalaiarasi	50	F	Stable angina	yes	T↓V5V6	_	V5,V6	V5,V6	3.5	2	69	64	Gri	nil	65	62	156	4	4	7	16
29	Radhakrishnan	53	M	AWMI	Nil	QV3-V6	no	nil	nil	5.4	3.6	61	48.8	Gri	AW,AS	66	61	145	5	4	8	16
30	Jeyaraj	43	M	IWMI	yes	QLiii,avf	_	Liii.avf	Liii.avf	5.6	3.9	58	52	Gri	IW,IS	82	70	122	6	5	7	16
31	Kathiresan	36	M	AWMI	yes	QV1-V4	no	nil	V1-V3	4.9	3.3	50	45	Gri	AW,AS	58	59	132	6	10	6	5
32	Mookiah	51	M	Unstable ang	Nil	ST↓V4V6	no	V4-V6	V5,V6	4.6	2.9	54	50	nil	nil	52	71	220	6	4	8	13
33	Govindaraj	48	M	AWMI	Nil	QV1,V2	no	V1-V3	V1-V3	4.9	3.3	42	38	Gri	AW,AS	50	66	132	5	9	8	5
34	Abdulaziz	59	M	Stable angina	yes	Normal	no	nil	nil	5	3.4	59	60	nil	nil	64	67	122	5	6	8	10
35	Jeyakodi	38	M	Stable angina	yes	Normal	no	nil	nil	5.7	4.1	50	54	nil	Iw	68	69	132	5	5	11	13
36	Vembu	52	M	AWMI	yes	ST↑V1-V6	V1-V4	Liii.avf	Liii.avf	5.9	5	31	36	Gri	AWAS	52	62	132	9	5	6	10
37	Natarajan	62	M	IWMI	Nil	QLiii,avf	no	Liii.avf	Liii.avf	5.4	4.9	17	25	Gri	AWAS	79	27	96	4	2	5	40
38	Sunderrajan	54	M	Stable angina	Nil	ST↓V4V6	no	V4-V6	V4-V6	4.8	3.3	48	39	nil	nil	51	70	238	5	3	8	17
39	Ravi	40	M	Unstable ang	Nil	ST↓V4V6	no	V4-V6	V4-V6	5.2	3.2	52	40	nil	HYAW	50	60	198	7	8	8	6
40	Sivasubramanian	58	M	Stable angina	Nil	ST↓Liavl	no	Liavl	Liavl	5.4	3.2	60	62	nil	nil	36	62	146	6	5	9	7

41	Navaneethammal	66	F	Unstable ang	yes	ST↓V4V6	no	V4-V6	V4-V6	4.3	3.7	27	30	nil	IW,IS	92	61	126	7	6	5	16
42	Mookan	60	M	Unstable ang	Nil	ST↓V4V6	no	V4-V6	V4-V6	5.2	3.8	44	39	Gri	AWAS	66	61	162	5	4	8	16
43	Indhurani	56	F	Unstable ang	Nil	ST↓V4V6	no	V4-V6	V4-V6	5	3.9	39	46	Gri	AWAS	38	47	65	5	5	5	7
44	Muthiah	65	M	Unstable ang	yes	ST↓V4V6	no	V4-V6	V4-V6	6	5.1	33	46	Gri	AWAS	58	59	137	6	10	6	5
45	Mohanganapathy	56	M	AWMI	yes	ST↑V1-V6	V1-V4	nil	nil	5	3.6	53	43	nil	AW,AS	87	42	148	6	7	8	12
46	Jebamalai	59	M	AWMI	Nil	ST↑V1-V6	V1-V4	nil	V1-V3	6	5	35	43	Griii	AWAS	68	73	129	7	8	8	8
47	Krishnasamy	48	M	AWMI	yes	ST↑V1-V6	V1-V4	nil	nil	6	5	35	39	nil	AWAS	78	71	146	6	8	8	9
48	Ramamoorthy	59	M	AWMI	yes	QV1-V6	no	V1-V3	V1-V3	5.3	4.3	48	26	Gri	AWAS	60	50	227	5	5	5.1	12
49	Marri	52	M	Stable angina	Nil	Normal	no	nil	avl	4.3	2.98	60	45	nil	nil	82	66	221`	7	8	7	8
50	Syed	62	M	OldIWMI	Nil	QLiii,avf	_	Liii.avf	Liii.avf	5.4	4.6	42	47	nil	IW,IS	58	56	111	7	6	6	9

S.NO	NAME	TRANSESOPHAGEAL ECHOCARDIOGRAPH								CORONARY ANGIOGRAM				
		LMCA(L)cm	DIAMETER(O)cm	SHAFT cm	BIFURcm	TVI(BEF)	TVI(AFTE)	%STENOS)	pre/potvi	LMCA(L)cm	DIA(O)cm	DIA(S)cm	DIA(B)cm	%STENOS
1	Vedappan	7.46	3.2	3.8	3.1	17	23	27	0.73	8.39	3.67	4.22	3.96	20
2	Gandhi	8.12	2.6	2.2	2	25	53	53	0.47	9.4	4.07	4.53	3.44	50
3	Singadurai	18.8	4.2	3.9	1.7	28	43	35	0.65	21.85	4.4	4.17	3.05	30
4	Leelavathy	9	2.6	1.9	4.2	22	31	30	0.7	7.64	2.15	1.87	2.04	20
5	Thangamuthu	14	3	2.1	3	38	91	59	0.41	18.48	3.73	2.91	4.25	50
6	Thaniperavi	6.8	4.3	4.3	3.9	42	83	50	0.5	7.96	4.05	4.21	3.29	50
7	Shikh hussain	6.2	5.2	5	4.7	43	56	24	0.76	16	5.31	5.03	3.59	30
8	Marimuthu	8.1	4.2	5.2	4.1	31	42	27	0.73	7.37	2.78	3.96	4.24	30
9	Ramakrishnan	7.5	2.9	3	2.6	29	38	24	0.76	7.35	3	2.99	2.27	20
10	Annadurai	10	5	4.2	2.5	28	64	57	0.43	11	3.5	3.14	2.27	50
11	Mohammedali	10.1	5	5	3.4	42	54	30	0.71	10.5	2.93	3.29	2.19	30
12	Ayyavu	5.5	2	3.7	2.9	47	67	30	0.73	17	4.5	2.9	3.7	50
13	Ramalingam	14	4.4	4.1	3	23	63	64	0.36	15.7	3.88	3.88	2.3	50
14	Thangaraj	7	2	2.2	2.5	17	29	42	0.58	8.48	3.28	2.61	3.04	30
15	Krishnamoorthy	9	4	2.8	3	23	49	54	0.46	12	4.07	2.32	3.77	50
16	Thiruvengadam	15	2.5	3.9	3	27	39	31	0.69	16	2.82	3.32	1.97	30
17	Balaguru	3.9	4.1	2.5	2.6	29	72	60	0.4	6.97	3.96	3.68	2.23	60
18	Krishnamoorthy	11	4.4	2.7	2.3	30	70	58	0.42	12.62	4.33	2.44	3.55	50
19	Elango	7.4	2.7	3.7	3	49	60	19	0.81	4.55	2.48	2.04	1.29	50
20	Padmavathy	11.2	3.9	3.2	2.8	30	66	56	0.44	11.18	4.07	4.07	2.94	50
21	Sakthivel	12.1	5.8	4.9	2.2	16	83	81	0.19	9.37	2.9	3.16	1.9	75
22	Babu	10.4	3	4	2.2	27	68	61	0.39	11.1	2.84	2.87	1.45	60
23	Shanmugam	13.2	5	4.2	3.9	18	51	65	0.35	14	4.65	4.98	3.12	50
24	Murugesan	6.2	3.2	2.1	3.4	29	66	57	0.43	4.82	2.76	1.53	2.47	50
25	Ramiah	8.4	5.5	4.1	3	24	55	57	0.43	7.99	4.3	2.13	2.31	50
26	Ramakrishnan	10.1	6.9	5.2	6.3	15	19	22	0.78	9.48	3.9	3.41	3.95	20
27	Sethuraman	14.1	5.7	2.9	2.4	32	76	58	0.42	16.47	3.78	3.82	2.4	70
28	Kalaiarasi	8.4	4.6	4.3	3.2	30.8	40.2	25	0.75	7.7	4.01	4.02	2.29	30
29	Radhakrishnan	10	2.5	2.1	2.2	16	18	12	0.88	11.13	3.13	2.75	2.91	20
30	Jeyaraj	7	2.5	3.6	2.3	21.6	47	55	0.45	5.73	2.4	2.4	1.85	30
31	Kathiresan	12	3	4	3.6	25	56	56	0.44	14.77	2.51	2.3	3	50
32	Mookiah	13	3	2.9	2.2	32	68	53	0.47	14.24	3	2.12	1.52	50
33	Govindaraj	14	5.7	5	3.3	11	19	43	0.57	13.63	3.09	3.38	2.48	30
34	Abdulaziz	13	2.8	4.3	3.8	29.5	70.5	59	0.41	14.35	3.92	4.14	2.2	75
35	Jeyakodi	18.7	4	3.8	3	53	103	49	0.51	21	3.08	2.8	1.54	50
36	Vembu	10	4.1	3.2	3.4	20	45	56	0.44	9.59	3.03	1.77	1.77	50
37	Natarajan	7	3	2.8	2.1	10	31.7	69	0.31	6.67	3.6	3.16	2.25	50
38	Sunderrajan	9	4.7	4.9	4	17.5	50.5	60	0.34	13.51	3.59	3.81	1.96	50
39	Ravi	8	3.4	2.9	2.6	14.3	21	32	0.68	8.87	2.73	2.77	1.83	20
40	Sivasubramanian	11	4	3.9	3	11	36	70	0.3	7.9	3.39	2.57	1.68	50

41	Navaneethammal	10	2.4	3.2	3	7	12	42	0.58	8.46	1.92	2.32	2.56	40
42	Mookan	7.8	3	2.1	2	5	46	89	0.11	11.38	2.47	2.21	1.86	30
43	Indhurani	14	3	1.7	1.9	26	30	14	0.86	22	2.77	1.48	2.46	40
44	Muthiah	11.6	2.8	3.6	3	23	51	55	0.45	11.75	0.96	2.45	2.3	50
45	Mohanganapathy	8.6	4	3.7	2.3	15.6	95.4	80	0.16	12.84	2.31	3.09	1.89	50
46	Jebamalai	7.5	3	2.3	3.7	12.3	15.2	20	0.8	14.98	4.06	3.27	4.39	30
47	Krishnasamy	8	5	4.5	4	7	61	80	0.11	9.21	2.99	3.32	2.31	30
48	Ramamoorthy	9	3.8	2.9	3.8	46.2	60.6	24	0.7	9.87	2.06	1.4	2.38	30
49	Marri	6	3.8	3.8	1.3	28.7	57.3	49	0.5	9.6	2.1	2.37	1.49	40
50	Syed	9.1	3.8	2.9	2.2	15	43.2	66	0.34	12.39	2.88	2.81	1.11	90