A STUDY ON CAROTID ARTERIAL REMODELING IN PATIENTS WITH OBSTRUCTIVE CORONARY ARTERY DISEASE AND DISEASE-FREE CONTROL SUBJECTS

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CERTIFICATE

This is to certify that the Dissertation entitled "**A STUDY ON CAROTID ARTERY REMODELING IN PATIENTS WITH OBSTRUCTIVE CORONARY ARTERY DISEASE AND DISEASE FREE SUBJECTS"** is the bonafide original work of **Dr. M.DINAKARAN** in partial fulfillment of the requirements for D.M. Branch – II (CARDIOLOGY) Examinations of the Tamil Nadu Dr. M.G.R. Medical University to be held in August 2009.

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CONTENTS

DECLARATION

I Dr. M. DINAKARAN solemnly declare that the dissertation titled "A STUDY ON CAROTID ARTERIAL REMODELING IN PATIENTS WITH OBSTRUCTIVE CORONARY ARTERY DISEASE AND DISEASE – FREE CONTROL SUBJECTS"is a bonafide work done by me at Govt. Stanley Medical College & Hospital during January 2007 to Dec.2008 under the guidance & suggestion of my Prof. Dr. R. Subramanian, M.D., D.M., Prof. & Head of the Dept. of Cardiology.

The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of D.M. Degree Branch-II Cardiology.

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DR. M. DINAKARAN

INTRODUCTION

Decades of silent arterial wall alterations precede vascular clinical events, which then reflect advanced atherosclerotic disease. The first morphological abnormalities of arterial walls can be visualized by B-mode ultrasonography. This high-resolution, noninvasive technique is one of the best methods for the detection of early stages of atherosclerotic disease, because it is rapidly applicable, readily available and demonstrates the wall structure with better resolution than any other similar technique .Accordingly, ultrasound has been used in a number of studies to monitor the intima-media thickness (IMT) of the carotid arteries, a measurement which has consequently been shown to be associated with cardiovascular risk factors and the incidence of cardiovascular disease. Furthermore, there are diverse approaches for measuring IMT, and some of these may lead to divergent results. Moreover, there are no unified criteria for distinguishing atherosclerosis as seen in early plaque formation from thickening of the intimal-medial complex. This is because IMT reflects not only early atherosclerosis, but also non atherosclerotic compensatory enlargement with largely medial hypertrophy as a result of smooth muscle cell hyperplasia and fibro cellular hypertrophy. This differentiation is important because epidemiological studies have shown that wall thickening as depicted by

ultrasonographic measurements of IMT is different from atherosclerotic plaque regarding localization, natural history, risk factors and predictive value for vascular events. As IMT is being increasingly used in clinical trials to serve as a surrogate end point for determining the success of interventions that lower risk factors for atherosclerosis, it is imperative that standardized methods be used to allow homogenous data collection and analysis. This would help to improve the power of such studies and to facilitate the merging of large databases for meta-analyses. In our study correlation of intima medial thickness with carotid artery remodeling and coronary artery disease status is assessed

AIM OF THE STUDY

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To study the carotid arterial remodeling in patients with coronary artery disease

To study the relationship between carotid arterial inter adventitial and lumen diameters to the intima medial thickness

To evaluate carotid arterial diameters of individuals with coronary artery disease and how it differed from those of CAD-free controls and

To study the relationship between carotid artery remodeling and severity of cornary artery disease

REVIEW OF LITERATURE

Many clinical studies support the opinion that carotid intima-media thickness (IMT) assessed by B-mode ultrasound in carotid arteries, either in a single segment (common carotid) or in multiple segments (aggregate of measures in common carotids, bifurcations, and internal carotids), is a valuable mirror of coronary risk status and a worthwhile predictor of subsequent coronary heart disease (CHD), the leading source of cardiovascular death. Indeed, traditional and emerging risk factors for CHD have been found mostly to be closely correlated with increased carotid IMT. Also, increased carotid IMT is associated with substantially increased risk of a future CHD event in primary and secondary prevention populations. Thus, by analyzing the proportion of subjects developing a CHD event according to the carotid IMT level at the onset of the follow-up in two pioneering prospective population-based studies, it has been shown that markedly increased IMT above 1 mm was associated with a 12–16% 10-year CHD incidence depending on age, sex, and carotid segment(s) measured, whereas the lowest percentiles of IMT distribution were associated with a CHD incidence of consistently <0.5%.

Carotid intima-media thickness (IMT) is a marker for early atherosclerosis. Because it can be measured relatively simply and non invasively, it is well suited for use in large-scale population studies. Ultrasonic measurements correlate well with histology, and increased IMT is associated with vascular risk factors and the presence of more

advanced atherosclerosis, which includes coronary artery disease. IMT is being increasingly used for risk stratification in individuals and as an end point in intervention studies. An important precondition for this application of IMT is that it can predict future risk of clinical vascular events. A number of longitudinal studies have examined the relationship between IMT and future events, most frequently the incidence of cardiac events (myocardial infarction [MI], angina pectoris, and coronary intervention) and cerebrovascular events (stroke or transient ischemic attack [TIA]).

Carotid intima-media thickness (IMT) measurements are being applied widely as a measure of atherosclerosis in studies on determinants of presence and progression of atherosclerosis and in studies on atherosclerosis as determinant of cardiovascular disease. Carotid IMT has been shown to be related to cardiovascular risk factors, prevalent cardiovascular disease, and atherosclerosis in the peripheral, coronary, and femoral arteries. Recently, evidence became available indicating that an increased carotid IMT is a strong predictor of coronary heart disease and stroke

Carotid intima-media thickness (CIMT) is a non-invasive alternative marker of atherosclerotic disease that has been used extensively since 1987. CIMT is defined as the distance between the lumen-intima interface, which corresponds to the inner and outer echogenic lines seen on the B-mode ultrasound image. Increased CIMT

has consistently been shown to predict future vascular events. In addition, change in CIMT over time is currently used in randomized controlled trials (RCTs) as an alternative (surrogate) end point for cardiovascular events to evaluate the efficacy of interventions because of its advantage of considerable reductions in sample size and duration of follow-up in comparison to traditional morbidity-mortality event trials.

ARTERIAL REMODELING

The process of arterial remodeling, i.e., the change in structural arterial properties through time in response to atherogenic and/or hemodynamic alterations within the arterial environment, is thought to be an adaptive phenomenon aimed at maintaining circumferential wall and shear stresses within certain limits of operation and possibly at preserving compliance. Arterial remodeling is characterized by wall thickening (indicated by an increase in intima-media thickness (IMT)] and diameter widening [indicated by an increase in interadventitial diameter [IAD]).At the biochemical level, arterial remodeling is characterized by a complex set of interactions between vasoactive molecules (eg, nitric oxide), enzymes (eg, matrix metalloproteinase), and inflammatory cells (monocytes/macrophages).

Three different patterns of arterial remodeling have been identified: inward (characterized by a decrease in lumen diameter [LD] resulting from a greater change in IMT than in IAD), outward (characterized by an increase in LD caused by a greater change in IAD than in IMT), and

compensatory (characterized by LD preservation despite changes in IMT and IAD) remodeling. Recent studies suggested that inward remodeling and outward remodeling are associated with distinct forms of plaque formation, ie, inward with stable plaque formation and outward with unstable plaque formation. As such, inward remodeling and outward remodeling have been recognized as markers of CVD risk. Yet, it is unclear to what extent the process of remodeling is driven by atherogenic factors (i.e., the presence of atherosclerosis) compared with hemodynamic factors (i.e., increased blood pressure). This is of particular importance because increased IMT could be the consequence of an unfavourable atherogenic milieu, subsequently leading to increases in IAD (i.e., the increase in IMT drives the increase in IAD), whereas alternatively IAD could be the consequence of an unfavorable hemodynamic milieu, which in turn could lead to increases in IMT (i.e., the increase in IAD drives the increase in IMT). Alternatively, changes in IMT and IAD may be driven by different processes and thus be unrelated

Mannheim Carotid Intima-Media Thickness Consensus [32]

IMT and Early Plaque

In the absence of atherosclerotic plaque, B-mode ultrasound displays the vascular wall as a regular pattern that correlates with anatomical layers. The intima-media portion of this pattern is represented by the area of tissue starting at the luminal edge of the artery and ending at the

boundary between the media and the adventitia. This interface is well depicted by ultrasound. With increasing age, this pattern has been shown to thicken in a uniform way in straight arterial segments. Thickening of the intima-media is accelerated and enhanced in the presence of risk factors of atherosclerosis, particularly high blood pressure, and by inherited genetic factors. As a mirror of these processes, IMT was identified as a tool to investigate normal aging and preclinical atherosclerosis. Later stages of atherosclerosis (plaque, stenosis, occlusion) can also be identified by ultrasound imaging either in the absence of or coincident with increasing IMT. However, there are intermediate stages between increased IMT and atherosclerotic plaque formation that cannot clearly be differentiated either by ultrasound or by histological examination. Such conditions are common at the bifurcation and the origin of the internal carotid artery, but occur only occasionally in the common carotid artery (CCA). Epidemiological and intervention studies have shown that although both share some common atherosclerosis risk factors, the natural history, patterns of risk factors and the prediction of cardiac and cerebral events are different for carotid IMT and carotid plaque.

The consensus recommends the following definitions for ultrasound characterization of IMT and atherosclerotic plaque:

(1) IMT is a double-line pattern visualized by echo tomography on both walls of the CCA s in a longitudinal image. It is formed by two parallel lines, which consist of the leading edges of two anatomical boundaries: the lumen-intima and media-adventitia interfaces (2) Plaque is a focal structure encroaching into the arterial lumen of at least 0.5 mm or 50% of the surrounding IMT value, or demonstrates a thickness >1.5 mm as measured from the media-adventitia interface to the intima-lumen interface.

These definitions will allow classification of the vast majority of carotid lesions observed with ultrasound.

Carotid examination includes visualization of common, internal and external carotid arteries. The different ultrasound devices are continuously evolving, providing higher spatial and density resolutions, and improving information on all the structures visualized is waiting for a scientific validation of the superiority of 3-dimensional over 2-dimensional imaging. We propose some rules to improve 2-dimensional image acquisition based on ultrasound physics and basic anatomy.

Ultrasound physics principles:

- Ultrasound beam is a virtual biconcave lens which is thicker on the near and distal part of the field represented on the screen. For that reason, the

best resolution is commonly obtained in the mid part of this field. Perpendicularity between the ultrasound beam and the structures to be visualized provides the best information as the reflection of the incident beam is optimal. The energy of the ultrasound beam decreases with the distance and is usually reduced in the far part of the field. Frequency of emission determines the resolution. The higher the frequency, the higher the resolution, however, frequency is inversely proportional to the depth to explore. Linear transducers activate piezo -electric crystals simultaneously resulting in a synchronized propagation of the ultrasound wave. Sector scanners are typically activating crystals sequentially in pre-defined time intervals, causing asymmetric shifts and reception of skewed wave fronts. Linear ultrasound transducers therefore provide best image quality for superficial arteries, while electronic sector scanners are better used for deep structures with less accessibility.

Physiological and patho physiological principles:

Common and internal carotid arteries are quite homogeneous in structure and hemodynamics. Delimited at the origin by the brachio-arterial cephalic trunk on the right side and the aortic arch on the left side, the CCA ends at the bifurcation which is represented anatomically by the point of divergence of the walls of the CCA .The bifurcation is rather heterogeneous between individuals. Since atherosclerosis usually starts in

the carotid bulb, delineation of the different segments is required. The continuous related progression of vascular wall changes best monitored in CCA IMT studies is different from discontinuous focal lesions (plaque) which are characteristic of atherosclerotic disease. Therefore, distinction between IMT and plaque must be clearly specified in the scanning protocols.

Methods of measuring the intima medial thickness

The IMT can be assessed in nearly every patient. Successful examination of the internal carotid artery and of the carotid bulb depends both upon the anatomical topography of the patient and on sono grapher expertise.

(1) Measurement of IMT is most simply performed in a region free of plaque where the double-line pattern is observed - this is advantageous as measurements are easier, more accurate, and reproducible and can be standardized by computerized analyses.

(2) IMT can be measured in the CCA, at the bulb and the origin of the internal carotid artery.

(3) In study designs that include wall thickness, values obtained from different sites of the carotid arteries should be documented separately.

Methods of analyzing the data

CCA acquisition with ultrasound should be conducted as follows.

Standard equipment includes a high-resolution B-mode system operating with preferentially linear ultrasound transducers at frequencies >7 MHz. Appropriate depth of focus (e.g. 30-40 mm), frame rate optimally 25 Hz (>15 Hz), and gain settings (minimal intra luminal artifacts) are recommended to obtain optimal image quality. Log gain compensation should be around 60 dB. They must be adjusted to obtain a symmetrical brightness on near and far wall, decreasing if necessary the gain in the mid part of the field to avoid intraluminal artifacts.

Carotid IMT scanning and reading protocol recommendations:

(1) The arterial wall segments should be assessed in a longitudinal view, strictly perpendicular to the ultrasound beam, with both walls clearly visualized in order to achieve diameter measurements. Lateral probe incidence is recommended as it offers the best view in the middle field, where the resolution is known to be greater than in the near or far field. A horizontal display of the arterial image is also recommended to obtain the optimal interfaces between blood and vascular structures, on the longest possible segment. The localization of the end of the CA is necessary to help in repositioning during the follow-up.

(2) IMT should be measured preferably on the far wall. This is because IMT values from the near wall depend in part on gain settings and are less reliable. If taken on the near wall, IMT should be measured separately from IMT of the far wall.

(3) Along a minimum of 10 mm length of an arterial segment, a high-quality image acquisition is required for serial reproducible measurements. Due to vessel tortuosity, IMT measurement could only be possible at a shorter vessel segment, especially in the carotid bifurcation or the ICA bulb.

(4) Edge detection systems that are properly calibrated provide accurate measurements of IMT. Observations made by readers may be equally valid, but they require rigorous quality control and quality assurance. In addition, manual and semi-manual reading methods are extremely time consuming in comparison to automated systems, which can provide the mean maximal value of 150 measurements performed on a 10-mm segment of CA in a very short time (<0.1 s).

(5) Adventitia-to-adventitia diameter and intraluminal diameter of CA must also be measured as IMT is significantly correlated with the arterial diameter.

(6) Another important issue is the question which IMT measure (e.g. mean IMT value, maximal IMT value, and composite measures from the left and the right side or from different arterial sites) should be used. There is no definite answer to this question. It seems to be plausible that mean IMT values as averaged across the entire distance are less susceptible to outliers, whereas the maximal IMT may reflect more advanced stages with focal thickening towards plaque formation. It is accepted that IMT values from the left and right side could be averaged although there is a significant difference between the left and right CA IMT, with higher values on the left side .Composite scores including both plaque and IMT measure should be avoided. In conclusion, data acquisition should be as detailed as possible, and data analysis can be restricted to single measures to reduce multiple testing problems.

(7) Each vascular laboratory must perform periodical quality control of their equipment (phantom scans) and reliability studies of scans and measurements for ultra sonographers and readers. The intra class correlation coefficient should be evaluated for intra- and inter observer variability, both for IMT and plaque measurements.

Population to be investigated

Standard and regular use of IMT measurements are recommended in all epidemiological and interventional trials dealing with vascular diseases to better characterize the population investigated (similarly to the documentation of other risk factors). Determination of 'normal values' should help to better characterize 'intermediate' risk populations in the future. The moderate but significant correlation between IMT and Framingham score observed in a large population (PARC Study) raises the question of the best indicator for vascular event in individuals. IMT measurement is the first candidate to evaluate against conventional evaluation of cardiovascular risk. In some countries recommendations already suggest that carefully performed IMT measurement can add incremental information to traditional risk factor assessment (European Society of Cardiology Guidelines).

Ancillary studies in clinical trials should be sufficiently powered to contribute to evaluating the predictive value of IMT on reduction of clinical events.

There is no recommendation up to date neither to ‘treat IMT values’ nor to monitor IMT values in individual patients.

Recent studies have shown that reduction of IMT values is significantly correlated with risk reduction and improvement of risk factor profiles in

large populations. However, neither positive nor negative predictive values on ischemic risk reduction are known in individual subjects treated successfully for specific risk factors. Thus, although IMT has been suggested to represent an important risk marker, it does not fulfill the characteristics of an accepted risk factor. At present, carotid IMT is not an FDA approved surrogate end point of vascular events. The moderate but significant correlation between IMT and Framingham score observed in a large population (PARC Study) raises the question of the best indicator for vascular event in individuals. IMT measurement is the first candidate to evaluate against conventional evaluation of cardiovascular risk in prospective studies. Clinical trials aimed to show that the efficacy of various classes of drugs on the reduction of IMT translates into the reduction of vascular events are needed and are currently underway

CAROTID INTIMA-MEDIA THICKNESS MEASUREMENTS

Manual B-mode versus automated Radio-Frequency measurements.

The approaches to measure CIMT can be classified generally in two major Categories: manual versus automated measurements. Manual measurement can be subdivided in offline and online measurements. In off-line measurements B-mode ultrasound images are stored on videotape or a digital medium. The CIMT measurements are performed later, with either manual tracing or an automated edge detection program. In routine clinical practice this approach may seem elaborate and time-consuming. Alternatively, in clinical practice CIMT can be measured manually and online with calipers of the ultrasound machine which provides a direct estimate of CIMT. Disadvantages of manual measurements are that it can be relatively time-consuming. Furthermore reader subjectivity due to personal interpretation of the lumen-intima and media-adventitia borders on the B-mode image, reflectivity of the structures, gain setting and compression characteristics of the ultrasound system are other disadvantages. A technically different approach is the M-mode ultrasonography, an on-line automated measurement approach which uses radiofrequency (RF) signals. In this approach, the diameter and relative increase in cross-sectional area are obtained with a wall track system which processes the raw RF signals that are received along a single line of observation (M-line processing).The RF measurements are performed in the distal common carotid artery 2 cm proximal to the origin of the

carotid bulb . Automated RF is more time efficient and involves a minimum of user interaction .RF calculates mean IMT locally over a cardiac cycle instead of mean IMT over a 10-mm segment at end-diastole. With RF simultaneous assessment of arterial wall properties such as distensibility can be obtained at the same location, facilitating detailed study of the intrinsic wall characteristics Variability in CIMT due to sonographers in the RF approach remains but the reproducibility of the automated RF approach has been shown to be good, as reported in previous studies).

MEAN OR MEAN MAXIMUM CAROTID INTIMA-MEDIA THICKNESS AS PRIMARY OUTCOME IN LIPID LOWERING INTERVENTION STUDIES

The mean maximum CIMT is a summary measure that is computed as the mean of the single maximum CIMT measurements measured in 6 to 12 standard carotid artery walls (far or near wall of the3 distinct carotid segments: the common carotid segment (CCA), the carotid bifurcation (BIF) and the internal carotid artery (ICA) segment) at both the left or right side. A mean maximum CIMT ultrasound assessment also enables the reader to acquire the mean common CIMT measure. The choice for one or the other CIMT measure as primary outcome is generally based on personal preference and/or expert opinion. An evaluation of the published data to support the use of either measure is lacking, but its availability may facilitate an evidence based decision. Arguments in favor of a common CIMT measurement over a mean maximum CIMT measurement

generally include higher reproducibility, more complete measurement assessment, an equally strong relation with future events, a stronger relation between progression rates and lipid levels, a higher susceptibility for lipid lowering treatment, and a more rapid ultrasound protocol. Support for mean maximum CIMT measurement above common CIMT measurement includes the view that reproducibility, completeness, risk prediction, and lipid level relations are similar to that of the common CIMT, but that a mean maximum CIMT provides a more complete coverage of the extent of carotid atherosclerosis. Furthermore, CIMT progresses differently over the carotid segments and it appears unpredictable at which segment lipid modifying treatment might have its effect. In addition, when mean maximum CIMT measurement is chosen, also information on the mean common CIMT is collected so one can use both the mean maximum CIMT and the mean common CIMT as outcome measurement. Based on the literature, reproducibility and lipid relations appear similar for both the mean maximum and mean common CIMT measurement, but, the mean maximum CIMT results most often are congruent with event findings. Therefore, the mean maximum CIMT as primary outcome is preferred. An additional advantage is that information on mean common CIMT can also be obtained in protocols assessing the mean maximum CIMT, but not the other way around.

ULTRASOUND PROTOCOLS TO MEASURE CAROTID INTIMA-MEDIA THICKNESS

A comparison of reproducibility, rate of progression and treatment. Effect in asymptomatic subjects with mild to moderate sub clinical atherosclerosis the number and specific combination of segments, angles and walls interrogated are associated with differences in reproducibility; magnitude and accuracy of progression of CIMT over time and treatment effect. The best balance between these parameters was found in mean maximum CIMT protocols followed by the mean common CIMT protocols, that included measurements of both walls at multiple angles. Protocols in which only the far wall was measured performed worse than protocols in which both the near and far wall were measured.

COMMON CAROTID INTIMA-MEDIA THICKNESS AND ARTERIAL STIFFNESS SMART STUDY

In a study by Petra C. G. Simons et.al. , , Common carotid intima-media thickness (IMT) and distensibilityrelationship was studied, as markers of structural and functional vessel wall properties Common carotid IMT and distensibility are clear markers of cardiovascular risk in patients who already have vascular disease or atherosclerotic risk factors. IMT appears to discriminate between low- and high-risk patients better than distensibility. Arterial stiffness has been described by use of several parameters: distensibility coefficient, compliance coefficient,stiffness (ß), pressure-strain elastic modulus, Young's modulus, and pulse-wave

velocity. In most of these parameters, the relationships between distensibility (change in lumen diameter), pulse pressure, and **carotid** lumen diameter are included. **Riley et al** used component mathematical models in which diameter change was the dependent variable and pulse pressure and lumen diameter were covariates of the predictor variable rather than ratios to describe arterial stiffness. Simons have shown that there is no statistically significant associations observed between pulse pressure, lumen diameter, and distensibility; therefore, adjustments for lumen diameter and pulse pressure were redundant. For IMT, usually no adjustments are made for pulse pressure and lumen diameter despite their relationships to IMT. Adjustments for pulse pressure and lumen diameter may be justified if one wants to study associations with distensibility or IMT independent of pulse pressure and lumen diameter.

There is increasing evidence that increased IMT and decreased distensibility are associated with the presence of cardiovascular risk factors and cardiovascular disease and that increased IMT is associated with higher risk of future myocardial infarction and stroke. Furthermore, a positive relationship between IMT and risk scores was also reported for the British Heart Study, Framingham, and EPOZ risk scores.. Measures of IMT and distensibility may also prove to be useful as markers of increased cardiovascular risk in a population with extensive

cardiovascular disease. Common **carotid** IMT and distensibility are clear markers of cardiovascular risk in patients who already have cardiovascular disease or risk factors. Simons study indicates that Increased IMT discriminates between low and high-risk patients better than reduced distensibility.

CAROTID Artery Intima -Media Thickness, Plaques, and C - reactive protein With Future Cardiovascular Disease and All-Cause Mortality.[51]

Jie J. Cao et al ., in The Cardiovascular Health Study have shown that Elevated C-reactive protein (CRP) and increased carotid artery intima -media thickness (IMT) are both associated with the occurrence of stroke. Higher CRP is also associated with higher IMT,. It is not clear whether CRP and carotid IMT each play an independent role in the pathogenesis of stroke.

Inflammation mediates a key role in the pathogenesis of atherosclerosis. Various cytokines, growth factors, and inflammatory cells are abundant in atheromatous plaques. The atherosclerotic vessel wall is the source of soluble adhesion molecules that mark inflammation. Endarterectomy specimens from symptomatic patients compared with asymptomatic patients and those in areas with more extensive plaques

have higher expression of adhesion molecules. Thus, the extent of inflammation may reflect in part the propensity of atherosclerotic lesions to lead to clinical disease.

It appears that among inflammation-sensitive proteins studied to date in relation to cardiovascular disease risk, higher CRP concentration is most consistently and strongly related to risk. Higher CRP might be a marker of destabilizing plaques, and CRP itself might also participate in plaque destabilization. It has been proposed that CRP reflects the overall inflammatory burden of atherosclerotic disease However, there is also evidence for possible direct roles of CRP in the pathogenesis of atherosclerosis, such as induction of endothelial cell adhesion molecules, opsonization of native LDL to form foam cells, induction of monocyte tissue factor production, and recruitment of monocytes by receptor-mediated chemotaxis.

And from this study it is clear that CRP is a risk factor for stroke independent of **carotid** IMT and, because it might be a marker of plaque severity and instability, that CRP would be more strongly related to stroke among those with higher **carotid** IMT. in the Cardiovascular Health Study (CHS). Elevated CRP is related to the future occurrence of ischemic stroke in this elderly cohort, an association that is largely independent of atherosclerosis severity as measured by **carotid** IMT. The

association of CRP with stroke was less apparent among those with less advanced compared with more advanced **carotid** atherosclerosis. Although CRP and **carotid** IMT are closely correlated, each factor may be an independent integral in the risk of ischemic stroke. There is a trend to suggest that the association of CRP and stroke might be stronger in men than in women. These findings should be further explored both in the laboratory and in ongoing observational and interventional studies carotid IMT, plaque, and elevated CRP each independently contributed to the risk of CVD and all-cause mortality in models that included all 3 measures. However, elevated CRP was associated with CVD events and all-cause mortality only in those with detectable atherosclerosis. Addition of CRP or carotid atherosclerosis to conventional risk factors resulted in a modest increase in the ability to predict CVD on the basis of ROC analysis.

CAROTID INTIMAL-MEDIAL THICKNESS AND CORONARY CALCIFICATION [33]

Patricia H. Davis et al studied the noninvasive methods used to evaluate early carotid and coronary atherosclerosis that included measurement of carotid intimal-medial thickness (IMT) by B-mode ultrasound and detection of coronary artery calcification (CAC) by electron beam computed tomography (EBCT). Presence of calcification in the coronary arteries has been correlated with atherosclerotic changes in

the coronary arteries found on postmortem examination or coronary angiography. CAC has also been shown to be associated with known risk factors for CAD in adults. Their study provides new data concerning the distribution of carotid IMT in a cohort aged 33 to 42 years and the association between increased carotid IMT and CAC. Most epidemiological studies of risk factors for atherosclerosis and clinical trials for the prevention of disease have used the morbidity and mortality that results from coronary artery disease (CAD), stroke, and peripheral vascular disease as measures of the disease process in older adults. There is a need to identify young subjects at risk for premature cardiovascular disease so that preventive measures may be instituted before occlusive vascular disease occurs. In this study, the investigators have used noninvasive measures of **carotid** and coronary artery atherosclerosis to evaluate the early process in young asymptomatic adults and to examine the relationship to established cardiovascular risk factors. Presence of calcification in the coronary arteries has been correlated with atherosclerotic changes in the coronary arteries found on postmortem examination or coronary angiography. CAC has also been shown to be associated with known risk factors for CAD in adults. Increased carotid IMT is also associated with known cardiovascular risk factors in older adults, as well as with prevalent and incident CAD. In addition, small case-control studies have shown increased carotid IMT in children with

familial hypercholesterolemia. Since 1971, a cohort of children in Muscatine, Iowa, has been followed up, with risk factors measured in childhood (ages 8 to 18 years) and young adulthood (ages 20 to 33 years). In a prior study of this cohort, **Mahoney et al** demonstrated that in 197 men and 187 women aged 29 to 37 years, CAC was associated with concurrently measured body mass index (BMI), blood pressure, and low HDL cholesterol (HDL-C), as well as with BMI measured in childhood Cardiovascular risk factors are associated with increased carotid IMT in adults aged 33 to 42 years. An association between carotid IMT and CAC that supports the use of carotid ultrasound to identify young adults at risk for premature atherosclerosis not only in the carotid arteries but also in the coronary arteries. Identification of atherosclerosis at an early age could lead to the development of interventions to ameliorate the process before the development of symptomatic disease

Carotid intima-media thickness by B-mode ultrasound as surrogate of coronary atherosclerosis: correlation with quantitative coronary angiography and coronary intravascular ultrasound findings

High resolution B-mode ultrasound is a non-invasive technique widely used to assess atherosclerosis in superficial arteries. It allows the

accurate measurement of the distance between blood–intima and media–adventitia interfaces of the carotid wall, which is defined as carotid intima-media thickness (IMT). Several authors have suggested that carotid IMT is a marker of atherosclerosis in other vascular beds. Indeed, an increased carotid IMT has been associated with a number of atherosclerosis risk factors, with the prevalence and extent of coronary artery disease (CAD) and with the incidence of new coronary and cerebral events. In view of these relationships, carotid IMT has been proposed as a surrogate endpoint to be used in clinical trials as an alternative to coronary atherosclerosis.

In spite of the widespread use of carotid IMT as a surrogate for CAD, validation studies evaluating the correlation between carotid IMT measured by external carotid ultrasound (ECU) and CAD measured by quantitative coronary angiography (QCA) showed a relatively poor correlation ($r < 0.36$ on average). This finding has cast doubt on the reliability of carotid atherosclerosis as a surrogate marker of coronary atherosclerosis. Postmortem studies, however, have shown a far greater degree of correlation between the two arterial districts, which suggests that the poor correlation observed in ECU vs. QCA studies, may be due more to technical issues than to differential effects of the traditional vascular risk factors on the carotid and coronary tissues.

Intravascular ultrasound (IVUS) is a unique imaging modality for the direct examination of vessel dimensions and arterial wall characteristics in live subjects. Like ECU, IVUS measures, in addition to lumen diameter, plaque area and any thickening of arterial walls. Carotid and coronary arterial districts can then be compared using the same arterial wall parameter, namely the IMT. Studies so far published addressing the relationship between IVUS-detected coronary atherosclerosis and carotid ultrasonic measurements, carotid IMT was correlated with coronary percent plaque area, and the correlation between IMT in the two vascular districts was not investigated. Researchers found correlations were stronger than those observed in QCA studies and closer to those observed in postmortem studies could be obtained by using the homogeneous variable IMT. And also found that carotid IMT would identify subjects with IVUS-detected evidence of coronary atherosclerosis among patients with a QCA diagnosis of no or intermediate coronary lesions.

Ankle-Brachial Index and Sub clinical Cardiac and Carotid Disease [37]

Mary McGrae Mc Dermott et. al. in The Multi-Ethnic Study of Atherosclerosis have shown that Lower-extremity peripheral arterial disease (PAD) can be detected non invasively with the ankle-brachial index (ABI), a ratio of Doppler-recorded systolic blood pressures in the

lower and upper extremities. In persons without PAD, arterial pressures increase with greater distance from the heart, because of increasing impedance with increasing arterial taper, resulting in higher systolic blood pressures at the ankle as compared with the brachial arteries. Thus, persons without atherosclerosis typically have an ABI greater than 1.00. An ABI less than 0.90 is highly sensitive and specific for angiographic ally diagnosed PAD

In general medical practice, the prevalence of ABI <0.90 is 25–30 percent among patients selected for older age or a history of diabetes or smoking. ABI <0.90 is associated with a two- to threefold increased risk of cardiovascular morbidity and mortality. The prevalence and significance of borderline ABI (i.e., ABI 0.90–0.99), low-normal ABI (i.e., ABI 1.00–1.09), and elevated ABI (i.e., ABI 1.30) are less well studied. In their study the prevalence of a priori defined categories of ABI: PAD (ABI <0.90), borderline ABI (ABI 0.90–0.99), low-normal ABI (ABI 1.00–1.09), and normal ABI (ABI 1.10–1.29), and high ABI (ABI 1.30) in an ethnically diverse group of men and women participating in the Multi-Ethnic Study of Atherosclerosis (MESA). Associations between these ABI categories and severity of sub clinical atherosclerosis in the carotid and coronary arterial beds. That borderline ABI, low-normal ABI, definite PAD, and possibly high ABI would be

associated with more severe sub clinical atherosclerosis in other vascular beds in comparison with normal ABI values. Whether there were ethnic differences in associations between ABI and sub clinical atherosclerosis in the carotid and coronary arterial beds. Borderline and low-normal ABI values were common in the MESA cohort. In both men and women, borderline ABI values were associated with a significantly higher prevalence of sub clinical atherosclerosis in comparison with normal ABI values (ABI 1.10–1.29). Low-normal ABI values were associated with a significantly higher prevalence of sub clinical atherosclerosis in men. Based on the findings reported here, further study is needed to determine whether persons with borderline ABI and men with low-normal ABI have a higher incidence of cardiovascular events than persons with normal ABI, and whether intensive atherosclerotic risk factor intervention comparable to that currently recommended for patients with clinically evident PAD is important for persons with borderline and low-normal ABI in order to prevent the progression of subclinical atherosclerosis

Glycated Hemoglobin Level is strongly related to the Prevalence of Carotid Artery Plaques with High Echogenicity in Non diabetic Individuals [36]

In The Tromso Study HbA_{1c} , an indicator of average glycemia over the previous 6 to 8 weeks was correlated with CMIT. THE level of

HbA_{1c} has been suggested as a diagnostic or screening tool for diabetes. Previous studies have shown that HbA_{1c} concentration is related to mortality and cardiovascular disease in non diabetic persons. In this population-based study examined the prevalence of carotid artery plaques, with emphasis on plaque morphology, in subjects at different HbA_{1c} levels. Increasing HbA_{1c} levels are related to an increased risk of carotid artery plaques and that the relationship depends on plaque echogenicity. And the results suggest that the degree of glycemia contributes to the development of hard echogenic plaques and that the process takes place already at modestly elevated levels of HbA_{1c}.

Association of increased carotid intima-media thickness with the extent of coronary artery disease

A Kablak-Ziembicka et al[32]., in their study have shown A significant correlation between mean IMT and advancing CAD ($p < 0.0001$). Four independent predictors of CAD were found in the discriminant analysis: age ($p = 0.0193$), hyperlipidaemia ($p < 0.0001$), smoking ($p = 0.0032$), and IMT ($p < 0.0001$). A significant increase in IMT was observed among patients with one, two, and three vessel CAD. A log normal distribution of IMT values showed that if mean IMT was over 1.15 mm, patients had a 94% probability of having CAD, with sensitivity of 65% and specificity of 80% in the patients with a high risk of CAD. The number of critically stenosed extra cranial arteries increased with advancing CAD. None of the patients with normal coronary arteries had severe stenosis of the extra cranial arteries. Severe carotid, vertebral, or subclavian stenosis was found in 16.6% of patients with three vessels CAD

Extra cranial Carotid Atherosclerosis Progression With Coronary Status and Risk Factors in Patients With and Without Coronary Artery Disease [53]

In the study by **Crouse, III, MD et al..** EXTRA CRANIAL **CAROTID** ATHEROSCLEROSIS PROGRESSION WAS STUDIED **.**Age, sex, hypertension,

smoking, diabetes, LDL and HDL cholesterol, as well as coronary status have been associated with extra cranial carotid disease, and investigators have suggested that they impact disease progression. Although direct quantification of the influence of risk factors on carotid intimal medial thickness (IMT) progression is more accurate than inference from cross-sectional studies, it also represents a greater technological challenge, because IMT changes slowly. Several epidemiological investigations have reported on IMT progression. This study compared progression in patients with and without coronary artery disease (CAD). It is clear from this study that patients with angiographic evidence for CAD would experience more rapid progression of carotid IMT than CAD-free individuals.

COMMON CAROTID INTIMA-MEDIA THICKNESS AND RISK OF STROKE AND MYOCARDIAL INFARCTION [45]

In The Rotterdam Study **Michiel L. Bots, MDet.al.,** have demonstrated increased common carotid intima-media thickness relates to future cardiovascular and cerebrovascular events. This study provides supportive evidence for the use of intima-media thickness measurements as an intermediate or proxy end point in observational studies and trials

Common Carotid Artery Intima-Media Thickness and Brain Infarction [45]

Carotid ultrasonographic methods capable of visualizing the arterial wall have been used to obtain measures of intima-media thickness (IMT). Increased IMT is generally considered an early marker of atherosclerosis. Cross-sectional associations have been reported between IMT and cardiovascular risk factors, prevalent cardiovascular disease, and peripheral atherosclerosis. Many prospective studies showed that an increased carotid IMT was associated with an increased risk of incident myocardial infarction. Two studies also showed that an increased carotid IMT was associated with an increased risk of stroke.. the authors in this study distinguished brain infarction (BI) from hemorrhages It has been suggested that IMT may, in part, reflect an adaptive response to changes in tensile and shear stress, we also studied IMT in association with CCA lumen diameter.; this study clearly demonstrate that the relation between cerebrovascular disease and IMT was not linear, with a significant association only present for the highest IMT values .In the Cardiovascular Health Study, 4476 participants (65 years) were followed for a median duration of 6 years; 284 strokes were ascertained. An association between increased IMT and the risk of incident stroke was observed. The relative risk increased in a linear fashion with increasing IMT, and it was of the same magnitude as the relative risk for myocardial

infarction this study shows a highly significant association between BI and CCA-IMT in the atherothrombotic group. The relation between CCA-IMT and BI remained after adjustment for main cardiovascular risk factors. is likely that an adjustment for lumen diameter is insufficient to adjust for wall stress; other measures (i.e., tensile stress and CCA end-diastolic lumen diameter) may be more meaningful. And it has been clearly shown that an increased CCA-IMT was associated with BI, both overall and in its main subtypes. Use of IMT as an outcome measure in observational studies and intervention trials relies on the view that IMT is a marker of atherosclerosis and reflects cardiovascular risk.

CAROTID INTIMA-MEDIA THICKNESS IS ASSOCIATED WITH PREMATURE PARENTAL CORONARY HEART DISEASE [43]

A family history of premature coronary heart disease (CHD) is an independent risk factor for cardiovascular events. The mechanisms underlying this familial clustering have not been established, but may include an increased susceptibility to atherosclerosis, an increased tendency for thrombosis, or other factors. Important insight may be gained by understanding how offspring of parents with premature CHD differ from offspring of parents without premature CHD. Evidence supporting a higher burden of sub clinical atherosclerosis in individuals with a parental history of premature CHD would implicate pathways

involved in atherogenesis and point to the potential utility of sub clinical disease measures as "intermediate phenotypes" for mapping CHD susceptibility genes. Elucidating this association may also have clinical implications, because it has been suggested that asymptomatic adults with a positive family history may benefit from sub clinical disease screening to determine need for primary preventive therapies.

High-resolution carotid ultrasound provides a noninvasive assessment of atherosclerotic burden. Increased intima-media thickness (IMT) is associated with prevalent coronary heart disease, peripheral vascular disease, and incident cardiovascular events. There is also evidence that carotid IMT may be a heritable trait. An association between carotid IMT or plaque and a parental history of CHD has been studied in one hospital-based study and several community-based studies. **The Framingham Heart Study** provides an opportunity to study the association between parental history and offspring sub clinical atherosclerosis, using a population-based cohort of families in which CHD events have been validated prospectively in both parents and offspring. It is also possible to examine whether this association is modified by parental sex or age of disease onset, offspring sex, or offspring cardiovascular disease risk factors

Thomas J. Wang, MD and colleagues performed ultrasound of the common carotid and brachial arteries in 40 offspring (mean age 19 years) recruited from patients hospitalized with premature acute myocardial infarction. Compared with age- and sex-matched controls, the offspring had increased common carotid IMT and more abnormalities in brachial artery reactivity. **Whtey et.al.,** found an association between parental premature CHD and offspring carotid IMT in this large, prospective family-based cohort. Their results and those of prior studies strongly support the existence of anatomic and functional vascular abnormalities in offspring of parents with premature CHD, independent of known vascular risk factors. Further investigation may help to clarify the relative contribution of shared genetic background versus shared environmental influences to this familial predisposition. In this regard, carotid IMT may be a useful phenotype for the study of the genetic transmission of CHD

CAROTID INTIMA-MEDIA THICKNESS AND HYDROXYMETHYLGLUTARYL COENZYME A REDUCTASE INHIBITORS.[40]

Allen J. Taylor, MD et.al. ,, compared the Effects of Atorvastatin and Pravastin on IMT . Marked LDL reduction to values substantially below 100 mg/dL treatment with atorvastatin provides superior efficacy for atherosclerosis regression in the distal common carotid artery at 1 year compared with an LDL of 110 mg/dL achieved with pravastatin. This early effect on CIMT, a surrogate for clinical benefit of cholesterol

lowering therapies, supports the hypothesis currently being tested in ongoing randomized clinical trials that marked LDL reduction with synthetic statins may provide enhanced reduction in clinical coronary event rates. statin on Carotid Intima Medial Thickness.

Effect of Supplementary Antioxidant Vitamin Intake on Carotid Arterial Wall Intima-Media Thickness in a Controlled Clinical Trial of Cholesterol Lowering.[44]

There is accumulating experimental, epidemiological, and clinical evidence of an association between antioxidant vitamin intake and reduced risk of coronary heart disease. Using data from the Cholesterol Lowering Atherosclerosis Study (CLAS), the association of self-selected supplementary antioxidant vitamin intake on the rate of progression of early preintrusive atherosclerosis Supplementary vitamin E intake appears to be effective in reducing the progression of atherosclerosis in subjects not treated with lipid-lowering drugs while the process is still confined to the arterial wall (early preintrusive atherosclerosis).

Elevated Carotid Artery Intima-Media Thickness Levels in Individuals Who Subsequently Develop Type 2 Diabetes

Although previous studies have established that coronary heart disease (CHD) risk factors are elevated before the clinical onset of diabetes and a recent study has reported increased cardiovascular disease

risk before the clinical onset of diabetes, documented. Documenting not only increased cardiovascular risk factors but increased atherosclerosis.

Results from the current study suggest again that increased atherosclerosis is responsible for elevated cardiovascular disease before the onset of clinical diabetes. If, THIS STUDY suggest, the association between diabetes and CHD stems mainly from common antecedents, one way to prevent the development of atherosclerosis would be to identify and intervene in those at high risk of developing diabetes.

EVIDENCE OF CAROTID ARTERY WALL HYPERTROPHY IN HOMOZYGOUS HOMOCYSTINURIA

Jean- Louis Megnien, MDet.al.,., have shown that Homozygous homocystinuria was associated with common carotid wall hypertrophy, whereas heterozygous disease was not. Such hypertrophy may reflect a smooth muscle proliferation induced by hyperhomocysteinemia and represent a promising target for testing vascular effects of therapeutic measures to lower homocysteine

.DIETARY ANTIOXIDANTS AND CAROTID ARTERY WALL THICKNESS

Stephen B. Kritchevsky,[41] PhD in the The ARIC Study have shown that dietary antioxidants may prevent atherosclerotic disease. The relationship between the intake of dietary and supplemental vitamin C, tocopherol, and provitamin A carotenoids and average **carotid** artery wall

thickness was studied in 6318 female and 4989 male participants 45 to 64 years old in the Atherosclerosis Risk in Communities Study. These data provide limited support for the hypothesis that dietary vitamin C and tocopherol may protect against atherosclerotic disease, especially in individuals >55 years old.

Intermediate-Density Lipoproteins and Progression of Carotid **Arterial Wall Intima-Media Thickness**[53]

Howard N. Hodiset.al .., in the Monitored Atherosclerosis Regression Study (MARS). Provide data that further give evidence for the role of triglyceride-rich lipoproteins in the progression of atherosclerosis and support the evidence that indicates that the risk of atherosclerosis attributable to LDL-C may in part be the result of lipoproteins in the IDL fraction (S_f 12 to 20) that is included within the traditional measurements of LDL-C.

ASSOCIATION OF MENOPAUSE AND HORMONE REPLACEMENT THERAPY WITH CAROTID ARTERY INTIMA-**MEDIA** [44]

Atherosclerotic CVD is the major cause of morbidity and mortality in older women. Although the incidence of CVD increases with age, evidence that natural menopause is an independent risk factor for CHD or strokes not convincing. In contrast, several studies have suggested that surgical menopause (bilateral oophorectomy) increases CHD risk. HRT

appears to afford protection from CHD, especially in women with bilateral oophorectomy, but there was little evidence that HRT prevents stroke until several recent studies were reported. This study evaluated the effect of stroke risk, cmit in postmenopausal women and found a significant reduction in CMITand stroke risk reduction on HRT therapy

LOW-DOSE METOPROLOL CR/XL AND FLUVASTATIN SLOW PROGRESSION OF CAROTID INTIMA-MEDIA THICKNESS [39]

Incidence of cardiovascular events in both secondary and primary prevention trials has been reduced by statins. The objective of the placebo-controlled ß-Blocker Cholesterol-Lowering Asymptomatic Plaque Study (BCAPS) by **B. Hedblad, MD, PhD et .al** ..,.was to evaluate the effect of bet blocker on CMIT. Main Results From the ß-Blocker Cholesterol-Lowering Asymptomatic Plaque Statins reduce cardiovascular events and progression of carotid intima-media thickness (IMT). ß-Blockers are also known to reduce cardiovascular events, but less is known about their effects on carotid IMT.study (BCAPS) this is the first randomized trial to show that a ß-blocker can reduce the rate of progression of carotid IMT in clinically healthy, symptom-free subjects with carotid plaque. This suggests that ß-blockers may have a favorable effect on atherosclerosis development.

INCREASED CAROTID ARTERY INTIMAL-MEDIAL THICKNESS IN ASYMPTOMATIC OLDER SUBJECTS WITH EXERCISE-INDUCED MYOCARDIAL ISCHEMIA [50]

Asymptomatic coronary artery disease (CAD) is prevalent in the general population, and individuals with CAD are at greater risk than those who are disease free to progress to symptomatic CAD and cardiac death. Yoji Nagai, MD et. al., in their study have shown CCA IMT is increased in older subjects with asymptomatic myocardial ischemia as evidenced by exercise ECG alone or in combination with thallium scan. Carotid ultrasound may help to identify asymptomatic individuals with CAD

MATERIALS AND METHODS

CAROTID artery lumen diameter, IA diameter, and intima-media thickness (IMT) were measured using B-mode ultrasound ALOKA prosound SSD 4000 machine with a 7.5 MHz probe in the common and internal carotid arteries of 123 CAD case patients and 125 disease-free control subjects. All patients have undergone CAG for evaluation CAD study was done strictly adhering to study protocol at the department of cardiology government Stanley medical college. Study was done after getting approval from local ethics committee

EXCLUSION CRITERIA

1. Patients with coronary stenoses of <50% were excluded
2. Patients were excluded who were alcohol, drug abusers
3. Clinically unstable Patients
4. Previous coronary bypass surgery, angioplasty, or carotid endarterectomy.
5. Controls who were on medications lipid-lowering drugs, thyroid medication, or cortisone
6. Patients who have hepatic disease, cancer, or renal failure.

CLINICAL EVALUATION

1. Medical history
2. Vascular disease risk factors
3. Menopausal status
4. Medication
5. Height, weight, and blood pressure

STUDY PROTOCOL

1. Informed consent
2. ECHO cardiographic evaluation
3. Selection of cases with optimal images
4. Lipid profile
5. Symptom limited exercise Stress test
6. Patients underwent Coronary angiogram in our cath lab

Risk Factors in Patients With CAD and in CAD-Free Control Subjects

Risk Factors in Patients With CAD and in CAD-Free Control Subjects									
	All Patients			Male Patients (n=124)			Female Patients (n=124)		
	CAD Cases (n=123)	Disease-Free Control Subjects (n=125)	*P*	CAD Cases (n=61)	Disease-Free Control Subjects (n=63)	*P*	CAD Cases (n=61)	Disease-Free Control Subjects (n=63)	*P*
TC, mg/dL	217±39	207 ±39	0.037	207±37	196 ±37	0.084	227±39	217±38	0.17
TG, mg/dL	185±109	177±149	0.618	181±108	179 ±145	0.922	189±111	175±155	0.541
HDL, mg/dL	43 ±12	47±16	0.016	38±11	41±16	0.23	47±12	52 ±14	0.017
LDL, mg/dL	141±32	130±34	0.006	135 ±31	123±33	0.02	146±33	136±33	0.1
Smoking, pk-y	25±31	19±25	0.045	36±33	28±29	0.154	15 ±25	9±17	0.112
DM, %	22	9	0.002	14	6	0.102	30	11	0.007
HTN, %	53	41	0.041	42	32	0.001	64	50	0.088
Age, y	62±8	56±8	0.001	61±7	56 ±9	0.002	63±9	56±7	0.001

The data shows the risk factor profile of cases and controls

METHODOLOGY

Arterial IMT and dimensions were measured with **ALOKA** prosound SSD4000, 7.5 MHZ probe. For the present analysis, 2 carotid segments were identified on both the left and right sides: the distal 1 cm of the common carotid proximal to the bifurcation and the proximal 1 cm of the internal carotid. 2 interfaces were identified on each wall: on the near wall, the first interface (interface 2) is the adventitial-medial boundary, and the second (interface 3) is the intima-lumen boundary; on the far wall, the first interface (interface 4) is the lumen-intima boundary, and the second (interface 5) is the media-adventitia boundary. Thus, 2 to 3 and 4 to 5 define IMT on the near and far walls, respectively. Examiner optimized the near and far walls separately (multiple focus zones) to define the maximum IMT for each of the 8 sites.

Arterial dimensions were measured at the maximum 2 to 3 or 4 to 5 IMT within each segment. Reference points were placed at interfaces 2, 3, 4, and 5 when available, and the distance was computed between interfaces 2 and 3 (near-wall IMT), 4 and 5 (far-wall IMT), 3 and 4 (lumen), and 2 and 5 (interadventitial [IA] diameter). In some cases, because of the tortuosity of the internal segment, the intima/lumen interface (interface 3 or 4) opposing the site of maximum IMT could not be visualized and direct lumen measurement was not possible. If interfaces adjacent to this site (with missing **intima**/lumen interface) were defined, we measured the IMT there after recording the maximum IMT

and IA diameter measurements. In such circumstances, an indirect measurement of lumen diameter was computed by subtracting the adjacent IMT plus the maximum IMT from the IA diameter measured at the maximum IMT site

STATISTICAL METHODS

Preliminary analyses suggested no differences in the distributions of IMT, lumen diameter, and IA diameter for right versus left carotid artery; therefore, the mean of right- and left-side measures was analyzed for each variable. For data presentation and analysis, age and IMT were categorized separately into quartiles by ranking these data without regard to CAD status. General linear regression models (SAS GLM procedure) were used to test associations between arterial dimensions and case-control status, age quartile, and IMT quartile. For arterial measures, means and SEMs (SAS least-square means procedure) are reported after adjustment for age, sex, and, where appropriate, IMT. Because physical stature is known to contribute to arterial diameter, height was included in all multivariable models for arterial dimensions.

RESULTS AND DATA ANALYSIS

The age was associated with structural measures (IMT, IA diameter, and lumen diameter) similarly in CAD cases and controls with the exception of the association between age and internal carotid lumen diameter.

Logistic Models to Predict CAD Status as a Function of Age and Each of the 11 Segment-Specific and Aggregate IMT Measures

Model	ß	SEM (ß)	x^2	*P*	Probability of Correct Class	
					Median	Inner-Quartile Range
ALL PATIENTS						
Mean common	2.411	0.545	23.37	<.001	0.607	0.29
Mean bifurcation	1.595	0.305	34.06	<.001	0.648	0.31
Mean com/bif	2.679	0.514	34.4	<.001	0.639	0.31
Mean bif/int	1.946	0.384	33.05	<.001	0.64	0.31
Mean internal	1.327	0.318	21.55	<.001	0.602	0.26
Mean agg	2.631	0.499	35.77	<.001	0.649	0.31
Mean agg far wall	1.854	0.38	30.78	<.001	0.638	0.3
Max common	0.767	0.194	18.49	<.001	0.608	0.28
Max bifurcation	0.751	0.162	24.82	<.001	0.613	0.29
Max internal	0.699	0.176	18.46	<.001	0.597	0.26
Max agg	0.738	0.156	25.84	<.001	0.625	0.29

MALE PATIENTS

Model	ß	SEM (ß)	x^2	*P*	Probability of Correct Class	
					Median	Inner-Quartile Range
Mean common	2.308	0.735	11.5	0.001	0.596	0.3
Mean bifurcation	1.301	0.382	13.35	<.001	0.621	0.3
Mean com/bif	2.325	0.66	14.88	<.001	0.62	0.29
Mean bif/int	1.828	0.506	16.09	<.001	0.645	0.29
Mean internal	1.573	0.442	16.14	<.001	0.643	0.27
Mean agg	2.413	0.637	17.77	<.001	0.646	0.33
Mean agg far wall	1.557	0.492	12.16	<.001	0.608	0.26
Max common	0.618	0.249	6.93	0.009	0.588	0.24
Max bifurcation	0.5	0.2	6.73	0.009	0.592	0.26
Max internal	0.696	0.233	10.4	0.001	0.567	0.42
Max agg	0.461	0.193	6.1	0.013	0.595	0.25
FEMALE PATIENTS						
Mean common	2.507	0.826	11.28	0.001	0.613	0.3
Mean bifurcation	2.054	0.521	21.42	<.001	0.656	0.31
Mean com/bif	3.209	0.844	19.52	<.001	0.655	0.31
Mean bif/int	2.098	0.601	16.59	<.001	0.636	0.32
Mean internal	1.025	0.467	5.71	0.017	0.574	0.25
Mean agg	3.004	0.823	17.72	<.001	0.65	0.33
Mean agg far wall	2.228	0.602	18.91	<.001	0.658	0.33
Max common	0.974	0.316	12.04	<.001	0.622	0.3
Max bifurcation	1.152	0.288	21.19	<.001	0.665	0.31
Max internal	0.704	0.278	7.61	0.006	0.585	0.26
Max agg	1.171	0.275	23.84	<.011	0.687	0.3

Among all participants, both common and internal carotid IMTs were greater in each quartile of increasing age ($P<0.01$ for each artery segment, adjusted for CAD status). In the common carotid artery, both lumen and IA diameter were greater in association with age ($P=0.003$ and $P<0.001$, respectively, adjusted for CAD status and height). Associations of age with common carotid lumen or IA diameter did not differ by case status (tests of CAD status–by-age interactions were not significant). In contrast, internal carotid IA diameter was not associated with age ($P=0.17$ adjusted for CAD status and height), whereas lumen diameter was smaller in older individuals ($P=0.002$), and the association varied by CAD case status ($I_{\text{nteraction}}=0.005$).

ASSOCIATIONS OF ARTERIAL DIMENSIONS WITH AGE AND CAD.

The following Data shows that, among all participants, height-adjusted common carotid IA diameter was greater in CAD cases than controls (P=0.004), whereas lumen diameters did not differ between cases and controls Overall, CAD cases had reduced internal carotid lumen and IA diameters compared with controls (P<0.001 and P=0.01, respectively) despite markedly greater IMT in the former.

Arterial Measures in mean Mean±SEM for CAD Cases and Controls by Quartiles of Age

Internal	45–51 (55 Controls; 15 Cases	52–59 (35 Controls; 35 Cases)	60–65 (32 Controls; 38 Cases)	66–79 (17 Controls; 53 Cases)	*P P* for age trend	*P P* for age-by–case status interaction	All Participants (123 Controls; 125 Cases)
Lumen							
All	6.53±0.17	6.70±0.15	6.00±0.15	5.94±0.16	0.002	0.005	
Controls	6.87±0.16	6.74±0.21	6.32±0.23	7.04±0.33	0.41		6.75±0.12
Cases	6.22±0.29	6.72±0.20	5.49±0.20	5.33±0.19	<0.001		5.93±0.11
P	0.06	0.95	0.01	<0.001			<0.001
IA							
All	8.15±0.16	8.43±0.13	7.93±0.13	8.04±0.15	0.17	0.44	
Controls	8.28±0.14	8.49±0.19	8.15±0.19	8.30±0.30	0.89		8.30±0.10
Cases	8.10±0.26	8.40±0.18	7.54±0.17	7.67±0.17	0.05		7.93±0.10
P	0.55	0.74	0.02	0.08			0.01
IMT							
All	0.99±0.07	1.13±0.06	1.22±0.06	1.36±0.07	0.01	0.4	
Controls	0.91±0.04	0.93±0.09	1.12±0.10	1.12±0.15	0.04		0.99±0.05
Cases	1.07±0.08	1.34±0.09	1.31±0.09	1.53±0.08	0.03		1.37±0.04
P	0.1	0.003	0.17	0.02			<0.001

The relationships between arterial diameters and case status were essentially unaffected by adjustment for age and sex (data not shown). In the internal carotid, associations of prevalent CAD with smaller IA and lumen diameters persisted ($P<0.01$ and $P<0.001$, respectively) in multivariable models that included age, sex, and IMT.

Arterial Measures in mean Mean±SEM for CAD Cases and Controls by Quartiles of Age

Common	45-51 (55 Controls; 15 Cases)	52-59 (35 Controls; 35 Cases)	60-65 (32 Controls; 38 Cases)	66-79 (17 Controls; 53 Cases)	PP for age trend	PP for age-by-case status interaction	All participants (123 controls; 125 Cases)
Lumen							
All	6.00±0.11	5.89±0.09	6.18±0.09	6.32±0.10	0.003	0.88	
Controls	5.98±0.09	5.85±0.11	6.05±0.13	6.30±021	0.006		6.05±0.07
Cases	6.08±0.18	6.06±0.11	6.20±0.12	6.32±0.12	0.12		6.16±0.07
P	0.64	0.17	0.38	0.92			0.23
IA							
All	7.67±0.11	7.67±0.10	8.11±0.10	8.38±0.12	<0.001	0.8	
Controls	7.53±0.11	7.53±0.13	7.86±0.14	8.25±0.24	<0.001		7.80±0.08
Cases	7.87±0.21	7.97±0.13	8.24±0.13	8.41±0.13	0.03		8.12±0.13
P	0.17	0.02	0.06	0.56			0.004
IMT							
All	0.86±0.03	0.94±0.03	0.99±0.03	1.07±0.03	<0.001	0.52	
Controls	0.84±0.02	0.86±0.04	0.96±0.04	1.04±0.07	<0.001		0.90±0.02
Cases	0.88±0.04	1.02±0.04	1.03±0.04	1.11±0.04	0.03		1.04±0.02
P	0.26	0.01	0.17	0.36			<0.001

CASE-CONTROL DIFFERENCES IN ARTERIAL DIMENSIONS

Height-adjusted common **carotid** IA diameter was greater in CAD cases than controls (P=0.004), whereas lumen diameters did not differ between cases and controls. Overall, CAD cases had reduced internal **carotid** lumen and IA diameters compared with controls (P<0.001 and P=0.01, respectively) despite markedly greater IMT in the former The internal **carotid**, associations of prevalent CAD with smaller IA and lumen diameters persisted (P<0.01 and P<0.001, respectively) in multivariable models that included age, sex, and IMT.

Arterial dimensions CAD Cases and Controls by severity of coronary artery disease

Height-adjusted common **carotid** IA diameter was greater in triple vessel disease than in patients with single vessel disease (P=0.01), whereas lumen diameters did not differ between 3vessel Disease and 1vessel Disease. Overall, 3 VD cases had reduced internal carotid lumen and IA diameters compared with controls (P<0.01 and P=0.02, respectively) despite markedly greater IMT in the former the internal carotid, associations of prevalent CAD with smaller IA and lumen diameters persisted (P<0.01 and P<0.001, respectively) in multivariable models that included age, sex, and IMT.

The following data shows the relation ship between carotid artery dimensions and severity of coronary artery disease

Arterial Measures (mm)in Mean±SEM for CAD Cases and Controls by severity of coronary artery disease

CAG	single vessel disease= 38	double vessel disease=43	Triple vessel T disease N=42
Common Lumen			
Controls	5.98±0.09	5.85±0.11	6.05±0.13
Cases	6.08±0.18	6.06±0.11	6.20±0.12
P	0.64	0.17	0.38
IA			
Controls	7.53±0.11	7.53±0.13	7.86±0.14
Cases	7.87±0.21	7.97±0.13	8.94±0.13
P	0.17	0.02	0.01
IMT			
Controls	0.84±0.02	0.86±0.04	0.96±0.04
Cases	0.88±0.04	1.02±0.04	1.03±0.04
P	0.26	0.01	0.17

Arterial Measures (mm)in Mean±SEM for CAD Cases and Controls by severity of coronary artery disease

CAG	single vessel disease	Double vessel disease	Triple vessel disease
Internal Lumen			
Controls	6.87±0.16	6.74±0.21	6.32±0.23
Cases	6.22±0.29	6.72±0.20	5.49±0.20
P	0.06	0.95	0.01
IA			
Controls	8.28±0.14	8.49±0.19	8.15±0.19
Cases	8.10±0.26	8.40±0.18	7.54±0.17
P	0.55	0.74	0.02
IMT			
Controls	0.91±0.04	0.93±0.09	1.12±0.10
Cases	1.07±0.08	1.34±0.09	1.31±0.09
P	0.1	0.003	0.17

ASSOCIATION OF LUMEN AND IA DIAMETER WITH IMT

The relationships between IMT and lumen and IA diameters in the common and internal carotid arteries, respectively. Demonstrates that age- and height-adjusted common carotid IA diameters were larger with each quartile of increasing IMT in both cases and controls. Common carotid IA diameter was strongly associated with IMT (P<0.001 for age, height, and CAD status–adjusted model), and the association was consistent in cases and controls (P=0.79 for case status by IMT interaction). The marginally significant association of common carotid lumen diameter with increased IMT (P=0.06) likewise did not vary by CAD status ($I_{nteraction}$=0.98).

Mean Segment-Specific IMT in Patients With CAD and in CAD-Free Control Subjects

	All Patients		***T***	***P***	**Male Patients**		***T***	***P***	**Female Patients**		***T***	***P***
IMT measure												
Mean common	1.221± 0.025	1.049 ± 0.025	4.82	<.001	1.238 ± 0.034	1.068 ± 0.035	3.37	<.001	1.199 ± 0.035	1.032± 0.034	3.3	0.001
Mean bifurcation	1.742± 0.045	1.357 ± 0.045	5.84	<.001	1.761± 0.064	1.416 ± 0.065	3.7	<.001	1.717± 0.064	1.302 ± 0.063	4.5	<.001
Mean bifurcation + common	1.368 ± 0.027	1.135± 0.027	5.85	<.001	1.394± 0.039	1.175 ± 0.040	3.85	<.001	1.336± 0.038	1.099 ± 0.038	4.3	0.001
Mean bifurcation + internal	1.465 ± 0.037	1.154± 0.038	5.72	<.001	1.491± 0.052	1.184 ± 0.053	3.99	0	1.434± 0.053	1.126 ± 0.053	4	<.001
Mean internal	1.320± 0.044	1.028 ± 0.044	4.57	<.001	1.410± 0.062	1.050 ± 0.063	3.97	<.001	1.214± 0.061	1.011 ± 0.059	2.3	0.021

As suggested by the above data, internal carotid IA diameter was not associated with IMT (P=0.27 for age, height, and CAD status–adjusted model). However, internal carotid lumen diameter was smaller in individuals with greater IMT (P<0.01 for age, height, and CAD status–adjusted model). The trend was present in CAD cases and controls alike, with lumen diameter smaller by almost 1 mm in patients in the lowest compared with those in the highest quartile of IMT (6.53±0.16 versus 5.59±0.16 mm, P<0.001, in the lowest and highest quartiles, respectively, adjusted for age, height, and CAD status). Mean internal carotid lumen diameters for CAD cases tended to be smaller than those of controls at each IMT cut-point; however, within quartiles, case-control comparisons reached significance only in the upper quartile of IMT. Within the internal carotid, CAD cases and disease-free controls showed similar associations of lumen and IA diameter with IMT (P=0.32 and 0.28 for tests of CAD

DISCUSSION

Segment-specific associations of carotid artery dimensions with IMT were reported previously in a cross-sectional analysis of data from the population-based ARIC study.[3] Common carotid wall thickening in the ARIC study was associated with larger IA diameters in both men and women; however, there was no significant association between IMT and IA diameter in the internal segment.[3] The ARIC analysis suggested that common carotid lumina were smaller only when mean far wall IMT exceeded 1.2 mm (observed in 1.5% of women and 3.2% of men), but internal carotid lumina were smaller over a broad range of increasing IMT.[3]

Our study describes segment-specific differences in carotid artery lumen and IA dimensions in individuals with obstructive CAD and CAD-free controls. Although common carotid IMT was greater in CAD cases than controls (as previously reported[11–13]), IA diameters were increased proportionately; thus, lumen diameters were similar in CAD cases and controls. In the internal carotid**,** CAD cases also had greater IMT than controls, but both lumen and IA diameters of the internal segment were significantly smaller in cases than controls.

Of interest, Pasterkamp et al[15] reported what the authors called "paradoxical constriction" of femoral arteries associated with atherosclerosis. Their cross-sectional study suggested that stenoses of

<25% were most often associated with local arterial enlargement in the femoral arteries, whereas reduced arterial diameter accompanied more significant stenoses.[15] A previous longitudinal study using B-mode ultrasound also suggested that the absence of compensatory enlargement contributed to lumen compromise during rapid focal progression of carotid plaques in areas of high hemodynamic forces such as the internal carotid.[7] The cross-sectional design of the present study does not permit us to determine whether case-control differences in internal carotid IA diameters accompanied atherosclerosis or whether individuals with CAD had smaller internal carotid arteries ab initio.

Because previous studies have described associations of age,[7,16,17] sex,[3,5,16–18] and physical stature[2,7,16–18] with arterial diameters, we adjusted for these factors in multivariable models. These covariates explained neither the case-control differences in arterial dimensions nor the segment-specific associations of IMT with arterial dimensions. Moreover, inclusion of IMT in multivariable models failed to explain reduced internal carotid lumen and IA diameters associated with prevalent CAD. Pasterkamp et al[15] also reported that reductions in femoral artery lumen and IA diameters were not explained by increased IMT.

It has been postulated that variation in the compensatory remodeling mechanism could influence individual susceptibility to developing symptomatic, obstructive cardiovascular disease.[2] Indeed, we found that participants with prevalent CAD had smaller mean internal

carotid lumen and IA diameters than disease-free controls; however, we could detect no statistically significant effect of case status on the association of IMT with either lumen or IA diameter (P>0.2 for tests of interactive effects of case status on association of IMT with lumen and IA diameters). It is possible that our sample size limits the statistical power to detect interactive effects with case status, although suggest that large differences in the association of carotid artery atherosclerosis with arterial dimensions are unlikely between CAD cases and controls.

Anatomic and physiological differences in the carotid segments may partially explain segment-specific differences in associations of arterial dimensions with IMT. The internal carotid is a muscular artery, whereas the common segment is an elastic conducting artery.[19] Elastic arteries, such as the common carotid, are exposed to relatively high blood pressure and laminar blood flow, ostensibly curtailing intima exposure to circulating risk factors. Within the internal carotid, turbulent blood flow and locally reduced shear stress prevail[20] and advanced plaques are more frequent. Longitudinal follow-up suggests that procoagulant risk factors such as fibrinogen and lipoprotein (a) contribute to rapid focal progression of atherosclerosis in the internal carotid, where blood flow is turbulent.[7] Moreover, these data showed that rapid focal atherosclerosis progression within the internal carotid favored lumen narrowing over arterial enlargement and lumen preservation.[7,8] In both the coronary and carotid artery systems, associations between atherosclerosis and lumen and IA dimensions are probably modulated by myriad growth factors,

cytokines, and enzymes secreted from both resident cells and cells recruited from the circulation.[9,10,21] the present data suggest that differences in arterial enlargement may contribute to carotid segment–specific variation in the occurrence of occlusive lumen stenosis. Common carotid occlusion is rare, accounting for <1% of carotid disease cases.[22] The present data, as well as cross-sectional ARIC data,[3] suggest that increased common carotid IMT is strongly associated with enlarged IA diameter and little or no decrease in lumen diameter. However, at the other extreme, our data suggest that increased internal carotid IMT is associated with decreased lumen diameter in the absence of IA diameter enlargement.

Fisher[23,24] noted in the early 1950s that internal carotid stenosis and occlusion were more frequent than previously believed. Fisher et al[25] later reported that among 178 autopsied cases, they found all cases of occlusive carotid disease (indeed, all significant stenoses) to occur distal to the common segment. In both the Cardiovascular Health Study (CHS) and the Framingham Study, researchers found that among asymptomatic participants >65 years old, 8% had significant carotid stenosis >50%.[26,27] Although the carotid arteries are the source of perhaps only 20% of all ischemic strokes,[28] overall stroke incidence in asymptomatic patients with significant carotid lumen stenosis ranges from 2% to 8% per year, depending on the degree of lumen obstruction.[22] Moreover, longitudinal follow-up within the ARIC and Cardiovascular Health Study populations suggests a graded increase in risk of incident ischemic stroke with

increasing carotid IMT measured by B-mode ultrasound.[29,30] The present data are consistent with the hypothesis that part of the risk of occlusive disease within the internal carotid is related to a relative lack of compensatory enlargement in that segment.and Remodeling accompanies significant progression of atherosclerosis in both the carotid[4,5] and coronary[6] arteries.

CONCLUSIONS

1. Common carotid atherosclerosis is associated with larger IA diameter and no reduction in lumen diameter POSITIVE REMODELING**.** Conversely, in the internal carotid**,** greater IMT is associated with smaller lumina in the absence of IA diameter enlargement. NEGATIVE REMODELING

2. Case-control comparisons suggest differences in arterial dimensions: common carotid IA diameter was greater in cases than controls, whereas internal carotid lumen and IA diameter were both reduced in CAD cases compared with controls. However, interactive effects of case status on the associations between IMT and arterial dimensions did not reach significance. Patients with triple vessel disease had significant remodeling of carotids when comparing patients with single vessel disease

3. The data is consistent with the concept that lack of arterial enlargement of the internal carotid during atherosclerosis progression partly explains the well-documented association of this carotid segment with symptomatic cardiovascular disease and the difference in the arterial biologic, physiologic and atherogenic prones properties of the different segments of carotid arteries

BIBILOGRAPHY

1. Glagov S, Weisenberg E, Zarins CK, et al. Compensatory enlargement of human atherosclerotic coronary arteries**. N Engl J Med. 1987; 316: 1371–1375.**

2. Clarkson TB, Prichard RW, Morgan TM, et al. remodeling of coronary arteries in human and nonhuman primates**. JAMA. 1994; 271: 289–294.**

3. Crouse JR, Goldbourt U, Evans G, et al. Arterial enlargement in the Atherosclerosis Risk in Communities (ARIC) cohort: in vivo quantification of **carotid** arterial enlargement**. Stroke. 1994; 25: 1354–1359.**

4. Steinke W, Els T, Hennerici M. Compensatory **carotid** artery dilatation in early atherosclerosis. **Circulation. 1994; 89: 2578–2581.**

5. Kiechl S, Willeit J, for the Bruneck Study Group. The natural course of atherosclerosis, II: vascular remodeling. **Arterioscler Thromb Vasc Biol. 1999; 19: 1491–1498.**

6. Shircore AM, Mack WJ, Selzer RH, et al. Compensatory vascular changes of remote coronary segments in response to lesion progression as observed by sequential angiography from a controlled clinical trial. **Circulation. 1995; 92: 2411–2418**.

7. Kiechl S, Willeit J, for the Bruneck Study Group. The natural course of atherosclerosis, I: incidence and progression. **Arterioscler Thromb Vasc Biol. 1999; 19: 1484–1490.**

8. Pasterkamp G, Schoneveld AH, van Wolferen W, et al. The impact of atherosclerotic arterial remodeling on percentage of luminal stenosis varies widely within the arterial system: a postmortem study. **Arterioscler Thromb Vasc Biol. 1997; 17: 3057–3063.**

9. Falk F, Shah PK, Fuster V. Coronary plaque disruption. **Circulation. 1995; 92: 657–671.**

10. Ward MR, Pasterkamp G, Yeung AC, et al. Arterial remodeling: mechanisms and clinical implications. **Circulation. 2000; 102: 1186–1191.**

11. Crouse JR III, Craven TE, Hagaman AP, et al. Association of coronary disease with segment-specific intima**l-**medial thickening

of the extracranial carotid artery. **Circulation. 1995; 92: 1141–1147.**

12. Terry JG, Howard G, Mercuri M, et al. Apolipoprotein E polymorphism is associated with segment-specific extracranial carotid artery intima-media thickening. **Stroke. 1996; 27: 1755–1759.**

13. Espeland MA, Tang R, Terry JG, et al. Associations of risk factors with segment-specific intima-media thickness of the extracranial carotid artery. **Stroke. 1999; 30: 1047–1055.**

14. SAS Institute Inc. SAS/STAT® software: changes and enhancements through release 6.12. Cary, NC: SAS Institute Inc; 1997:571–702.

15. Pasterkamp G, Wensing PJW, Post MJ, et al. Paradoxical arterial wall shrinkage may contribute to luminal narrowing of human atherosclerotic femoral arteries. **Circulation. 1995; 91: 1444–1449**.

16. Crouse JR, Goldbourt U, Evans G, et al. Risk factors and segment-specific carotid arterial enlargement in the Atherosclerosis Risk in Communities (ARIC) cohort. **Stroke. 1996; 27: 69–75.**

17. Bonithon-Kopp C, Touboul PJ, Berr C, et al. Factors of carotid arterial enlargement in a population aged 59 to 71 years: the EVA Study**. Stroke. 1996; 27: 654–660.**

18. Jensen-Urstad K, Jensen-Urstad M, Johansson J. Carotid artery diameter correlates with risk factors for cardiovascular disease in a population of 55-year-old subjects. **Stroke. 1999; 30: 1572–1576**.

19. Heath D, Smith P, Harris P, et al. **The atherosclerotic human** carotid **sinus. J Pathol. 1973; 110: 49–58.**

20. Carallo C, Irace C, Pujia A, et al. Evaluation of common carotid hemodynamic forces: relations with wall thickening. **Hypertension. 1999; 34: 217–221.**

21. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990's. **Nature. 1993; 362: 801–809.**

22. Toole JF. Cerebrovascular Disorders, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1999.

23. Fisher M. Occlusion of the internal carotid artery**. AMA Arch Neurol Psychiatry. 1951; 65: 346–377.**

24. Fisher M. Occlusion of the carotid arteries. **AMA Arch Neurol Psychiatry. 1954; 72: 187–204.**

25. Fisher CM, Gore I, Okabe N, et al. Atherosclerosis of the carotid and vertebral arteries: extracranial and intracranial. **J Neuropathol Exp Neurol. 1965; 24: 455–476.**

26. O'Leary DH, Polak JF, Kronmal RA, et al. Distribution and correlates of sonographically detected carotid artery disease in the Cardiovascular Health Study. **Stroke. 1992; 23: 1752–1760.**

27. O'Leary DH, Anderson KM, Wolf PA, et al. Cholesterol and carotid atherosclerosis in older persons: the Framingham Study. **Ann Epidemiol. 1992; 2: 147–153.**

28. Sacco RL, Ellenberg JH, Mohr JP, et al. Infarcts of undetermined cause: the NINCDS stroke data bank**. Ann Neurol. 1989; 25: 382–390.**

29. Chambless LE, Folsom AR, Clegg LX, et al. Carotid artery wall thickness is predictive of incident clinical stroke: The Atherosclerosis Risk in Communities (ARIC) Study. **Am J Epidemiol. 2000; 151: 478–487.**

30. O'Leary DH, Polak JF, Kronmal RA, et al. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. **N Engl J Med. 1999; 340: 14–22.**

31. Carotid Intima-Media Thickness Is Associated With Premature Parental Coronary Heart Disease The Framingham Heart Study Thomas J. Wang, MD; Byung-Ho Nam

32. Mannheim Carotid Intima-Media Thickness Consensus (2004-2006) An Update on Behalf of the Advisory Board of the 3rd and 4th Watching the Risk Symposium 13th and 15th European Stroke Conferences, Mannheim, Germany, 2004, and Brussels, Belgium, 2006

33. Carotid Intima-Media Thickness and Coronary Artery Calcium Score as Indications of Subclinical AtherosclerosisSteven J. Lester, MD, FRCPC,

34. Surrogate Markers for Cardiovascular Disease G. B. John Mancini, MD; Björn Dahlöf, MD; Javier Díez, MD, PhDCirculation. 2004;109:IV-22 – IV-30

35. Ankle-Brachial Index and Subclinical Cardiac and Carotid Disease The Multi-Ethnic Study of Atherosclerosis Mary McGrae McDermott[1], Kiang Liu[1], Michael H. Criqui[2], Karen Ruth[1], David Goff[3], Mohammed F. Saad[4], Colin Wu[5], Shunichi Homma[6] and A. Richey Sharrett[7]. American Journal of Epidemiology 2005 162(1):33-41

36. Glycated Hemoglobin Level Is Strongly Related to the Prevalence of Carotid Artery Plaques With High Echogenicity in Nondiabetic Individuals The Tromsø Study Lone Jørgensen, PhD; Trond Jenssen, MD, PhD; Oddmund Joakimsen, MD, PhDCirculation. 2004;110:466-470.)

37. Association of Carotid Artery Intima-Media Thickness, Plaques, and C-Reactive Protein With Future Cardiovascular Disease and All-Cause Mortality. The Cardiovascular Health Study Jie J. Cao, MD, MPH; Alice M. Arnold, PhD; Teri A. Manolio, MD, PhD

38. Carotid Arterial Remodeling A Maladaptive Phenomenon in Type 2 Diabetes but Not in Impaired Glucose Metabolism: The Hoorn Study

39. Low-Dose Metoprolol CR/XL and Fluvastatin Slow Progression of Carotid Intima-Media Thickness Main Results From the ß-Blocker Cholesterol-Lowering Asymptomatic Plaque Study (BCAPS) B. Hedblad, MD, PhD; J. Wikstrand, MD, PhD; L. Janzon, MD, PhD; H. Wedel, PhD; G. Berglund, MD, PhD. Circulation. 2001;103:1721

40. Carotid Atherosclerosis Is a Stronger Predictor of Myocardial Infarction in Women Than in Men A 6-Year Follow-Up Study of 6226 Persons: The Tromsø Study Stein Harald Johnsen, MD, PhD; Ellisiv B. Mathiesen, MD, PhD

41. Effect of Supplementary Antioxidant Vitamin Intake on Carotid Arterial Wall Intima-Media Thickness in a Controlled Clinical Trial of Cholesterol Lowering Stanley P. Azen, PhD; Dajun Qian, MS; Wendy J. Mack, PhD; Alex Sevanian, PhD Circulation. 1996;94:2369-2372

42. Carotid Intima-Media Thickness Is Associated With Premature Parental Coronary Heart Disease The Framingham Heart Study Thomas J. Wang, MD; Byung-Ho Nam, PhD; Ralph B. D'Agostino, PhD; Circulation. 2003;108:572-576

43. Carotid Intima–Media Thickness as an Independent Predictor of Coronary Artery Disease UdayM Jadhav, NN Kadam Department of Non-Invasive Cardiology, MGM New Bombay Hospital, New Mumbai. Indian Heart J 2001; 53: 458–462

44. Association of Menopause and Hormone Replacement Therapy With Carotid Artery Intima-Media Thickness Circulation. 1996;94:1857-1863

45. Common Carotid Intima-Media Thickness and Risk of Stroke and Myocardial Infarction. The Rotterdam Study MichielL.Bots,MD,PhD;ArnoW.Hoes,MD,PhD2007;116:32-38.)

46. Ankle-Brachial Index and Subclinical Cardiac and Carotid Disease The Multi-Ethnic Study of Atherosclerosis Mary McGrae McDermott[1], Kiang Liu[1], Michael**American Journal of Epidemiology 2005 162(1):33-41**

47. The relationship between carotid intima-media thickness and coronary atherosclerosis revisited Alain Simon[*] and Gilles Chironi European Heart Journal 2007 28(17):2049-2050

48. Carotid Intima-Media Thickness in Japanese Type 2 Diabetic Subjects ,MD,PHDDiabetesCare23:1310–1315,2000

49. Associations of Extracranial **Carotid** Atherosclerosis Progression With Coronary Status and Risk Factors in Patients With and Without Coronary Artery Disease John R. Crouse, III, MD*Circulation.* 2002;106:2061.)

50. Predictors of progression and relationship with incident corIncreased Carotid Artery Intimal-Medial Thickness in Asymptomatic Older Subjects With Exercise-Induced Myocardial Ischemia Yoji Nagai, MD; E. Jeffrey Metter, MD; Christopher J. Earley, MD, PhDonary YOSHIMITSUYAMASAKI Circulation. 1998;98:1504-1509

51. C-Reactive Protein, Carotid Intima-Media Thickness, and Incidence of Ischemic Stroke in the Elderly The Cardiovascular Health Study Jie J. Cao, MD, MPH; Chau Thach, PhD; TeriCirculation. 2003;108:166-170

52. Intermediate-Density Lipoproteins and Progression of **Carotid** Arterial Wall **Intima**-Media **Thickness** Howard N. Hodis, MD; Wendy J. Mack, PhD; Meleana Dunn, MS; Chao-ran Liu, MDCirculation.1997;95:2022-2026

53. Associations of Extra cranial Carotid Atherosclerosis Progression With Coronary Status and Risk Factors in Patients With and Without Coronary Artery Disease JohnR.Crouse,III,MD Circulation. 1995; 92:1141-1147

PROFOMA

A STUDY ON CAROTID ARTERIAL REMODELING IN PATIENTS WITH OBSTRUCTIVE CORONARY ARTERY DISEASE AND DISEASE-FREE CONTROL SUBJECTS

NAME

AGE

SEX

OCCUPATION

CD NUMBER

SOCIOECONOMIC STATUS

RISK FACTOR PROFILE

HYPERTENSION

DIABETES

HYPERLIPEDIMIA

SMOKING

OBESITY

PREVIOUS CAD

FAMILY HISTORY OF CAD

CLINICAL PROFILE

VITAL PARAMETERS PR BP

TREADMILL TEST

CORONARY ANGIOGRAM REPORT

MEASUREMENTS

COMMON CAROTID

1. LUMINAL DIAMETER
2. IMT
3. INTRADVENTITIAL DIAMETER

INTERNAL CAROTID

1 .LUMINAL DIAMETER

2. IMT

3. INTRADVENTITIAL

SERIAL NO.	COMMON CAROTID	1.LUMINAL DIAMETER	2.IMT	3.INTRADVENTITIAL DIAMETER
	INTERNAL CAROTID	1.LUMINAL DIAMETER	2. IMT	3.INTRADVENTITIAL DIAMETER

CD num	AGE	SEX	HEIGHT	WEIGHT	HTN	DM	SMOKING	ECG	ECHO	TC	TG	HDL	IC LD	IC IMT	IC IA	CC LD	CC IMT	CC IAD	TMT	CAG
134536	43	M	162	54	N	Y	Y	SR WNL	NORMAL LV FUNCTION	234	156	48	5.94±0.04	0.85±0.02	7.53±0.11	6.77±0.16	0.93±0.04	8.28±0.14	NEGATIVE	normal
178755	54	M	150	63	Y	N	Y	SR WNL	NORMAL LV FUNCTION	256	140	32	5.92±0.03	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
124545	34	F	148	46	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	276	148	34	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
152111	54	M	155	64	N	Y	Y	SR,BAE,LVH,OLDawmi	RWMA IN LV ANT WAL ,EF 53%	256	145	33	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
123474	54	F	164	75	N	N	N	SR ,prwp,T inv V1-v3	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	246	134	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
123434	66	M	156	43	N	N	Y	SR WNL	NORMAL LV FUNCTION	264	121	42	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
134545	54	M	176	43	Y	Y	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	274	111	55	6.06±0.11	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
134546	44	F	134	65	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	356	110	45	6.20±0.12	0.88±0.04	7.87±0.21	6.22±0.29	1.27±0.08	8.12±0.26	NEGATIVE	DVD
165625	76	F	147	53	N	N	N	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	352	151	34	6.32±0.12	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.24±0.13	POSITIVE	TVD
135534	54	M	163	54	Y	N	Y	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	342	151	32	6.05±0.13	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143415	44	F	145	44	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	234	145	33	6.30±021	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.33±0.14	POSITIVE	SVD
143457	34	M	162	43	N	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	321	134	35	6.05±0.07	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.33±0.26	POSITIVE	DVD
176678	56	M	145	55	N	N	Y	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	233	127	42	6.16±0.07	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.965±0.13	POSITIVE	DVD
143143	67	M	164	66	Y	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	232	118	32	6.09±0.18	0.89±0.04	7.86±0.21	6.06±0.18	0.93±0.04	8.37±0.14	POSITIVE	SVD
134347	65	F	155	66	Y	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	244	151	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.20±0.26	POSITIVE	DVD
175453	66	F	148	54	N	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	231	157	33	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.84±0.13	POSITIVE	TVD
165676	55	M	145	76	N	Y	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	149	35	6.06±0.11	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
176767	45	F	176	86	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	245	136	42	6.20±0.12	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.38±0.14	POSITIVE	SVD
176456	67	F	133	65	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	266	121	55	6.32±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
145634	54	F	136	56	N	Y	N	SR WNL	NORMAL LV FUNCTION	233	111	45	6.05±0.13	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.84±0.13	POSITIVE	TVD
134545	54	F	137	54	Y	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	356	110	34	6.30±021	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.15±0.26	POSITIVE	DVD
174563	54	M	156	54	Y	N	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	352	134	32	6.05±0.07	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
124543	66	F	176	64	N	Y	N	SR WNL	NORMAL LV FUNCTION	342	121	34	6.16±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.26±0.14	POSITIVE	SVD
167543	54	M	134	64	N	Y	Y	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	234	111	33	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.12±0.26	POSITIVE	DVD
145678	44	M	147	45	N	N	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	321	117	35	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.96±0.13	POSITIVE	TVD
124565	76	M	163	65	Y	N	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	171	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134535	54	F	145	67	Y	Y	N	SR WNL	NORMAL LV FUNCTION	232	151	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156743	65	M	162	65	N	Y	Y	SR WNL	NORMAL LV FUNCTION	244	145	45	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.27±0.14	POSITIVE	SVD
123432	66	M	145	43	Y	N	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	231	134	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.92±0.04	NEGATIVE	normal
175433	55	F	164	43	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	276	171	55	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.27±0.14	POSITIVE	SVD
187654	45	M	155	65	N	Y	Y	SR WNL	RWMA IN INFERO POST SEGMENT.EF 48%	256	127	45	5.88±0.09	0.85±0.02	7.56±0.11	6.86±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176543	67	F	148	53	N	N	N	SR ,LVH	NORMAL LV FUNCTION	246	118	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143546	54	M	145	54	Y	Y	Y	SR WNL	NORMAL LV FUNCTION	246	151	32	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
176542	54	M	176	44	Y	N	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	264	157	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
143567	54	F	133	43	N	Y	N	SR WNL	NORMAL LV FUNCTION	274	149	35	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
156563	54	F	136	55	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	356	136	42	5.88±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156743	66	M	137	66	Y	N	Y	SR ,LVH	NORMAL LV FUNCTION	352	121	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
134532	54	M	150	66	Y	N	N	SR WNL	NORMAL LV FUNCTION	342	111	55	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
156754	44	F	148	54	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	234	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176543	76	F	155	76	Y	Y	N	SR WNL	NORMAL LV FUNCTION	321	110	34	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
134344	54	F	164	86	Y	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	233	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
134543	44	M	156	65	N	N	Y	SR ,LVH	NORMAL LV FUNCTION	232	151	33	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
134353	34	F	176	54	N	Y	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 50%	244	121	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134354	56	M	134	54	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	231	111	42	6.06±0.11	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
154453	67	M	147	64	Y	N	N	SR ,OLD ASMI	NORMAL LV FUNCTION	233	110	32	6.20±0.12	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
143444	65	M	163	64	Y	Y	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	245	134	55	6.32±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143455	66	F	145	45	N	Y	N	SR WNL	NORMAL LV FUNCTION	266	121	45	6.05±0.13	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143413	55	F	147	65	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	233	111	34	6.30±021	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
134576	45	M	163	67	Y	Y	N	SR Tinv in 2,3,avf	RWMA IN INFERO POST SEGMENT.EF 50%	356	110	32	6.05±0.07	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
165366	66	M	145	65	N	N	Y	SR WNL	NORMAL LV FUNCTION	352	151	33	6.16±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD

165675	54	F	162	43	N	Y	N	SR Tinv in 2,3,avf	NORMAL LV FUNCTION	233	151	35	6.32±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
135546	44	M	145	43	Y	Y	Y	SR WNL	NORMAL LV FUNCTION	232	145	42	6.05±0.13	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
155355	76	F	164	65	Y	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	244	134	32	6.30±021	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
154554	54	M	155	53	N	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	231	121	55	6.05±0.07	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
135554	65	F	148	54	N	Y	Y	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	276	110	45	6.16±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
156656	66	F	145	44	Y	Y	N	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	256	151	34	6.06±0.11	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
135544	55	F	176	54	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	246	151	32	6.20±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
146443	45	M	133	54	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	246	145	33	6.32±0.12	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
145673	67	M	136	64	Y	Y	Y	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	264	110	32	6.05±0.13	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
187797	54	F	137	64	Y	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	274	134	55	6.30±021	1.31±0.09	7.54±0.17	6.21±0.11	1.01±0.14	8.84±0.12	NEGATIVE	TVD
186585	54	M	176	45	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	276	121	45	6.05±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
145788	54	M	134	65	N	Y	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	256	111	34	6.16±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
198675	55	F	147	67	N	Y	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	246	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156756	45	M	163	65	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	264	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134536	67	F	145	43	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	274	151	35	6.06±0.11	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
178755	54	M	162	43	N	N	N	SR WNL	NORMAL LV FUNCTION	356	145	42	6.20±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
124545	54	M	145	65	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	352	134	32	6.32±0.12	0.88±0.04	7.87±0.21	6.21±0.11	1.01±0.14	8.84±0.12	POSITIVE	SVD
152111	54	F	164	53	Y	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	342	121	55	6.05±0.13	0.88±0.04	7.87±0.21	6.20±0.12	1.03±0.04	8.94±0.15	POSITIVE	SVD
152111	54	F	155	54	N	N	N	SR WNL	NORMAL LV FUNCTION	234	110	45	6.30±021	0.84±0.02	7.53±0.11	6.25±0.12	1.05±0.04	8.94±0.13	NEGATIVE	normal
123434	66	M	148	44	N	N	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	321	151	34	6.05±0.07	0.84±0.02	7.53±0.11	6.26±0.12	1.03±0.04	8.96±0.13	NEGATIVE	normal
134545	54	F	145	43	Y	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	233	110	32	6.16±0.07	0.84±0.02	7.53±0.11	6.28±0.12	1.05±0.04	8.94±0.13	NEGATIVE	normal
134546	44	M	176	65	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	232	151	33	6.08±0.18	0.88±0.04	7.87±0.21	6.26±0.12	1.09±0.04	8.95±0.13	POSITIVE	SVD
165625	76	M	133	53	N	N	N	SR WNL	NORMAL LV FUNCTION	233	151	35	6.08±0.18	0.88±0.04	7.87±0.21	6.25±0.12	1.05±0.04	8.96±0.13	NEGATIVE	SVD
135534	54	M	136	54	N	N	N	SR WNL	NORMAL LV FUNCTION	232	145	42	5.98±0.09	0.84±0.02	7.53±0.11	6.25±0.12	1.06±0.04	8.94±0.13	NEGATIVE	normal
143415	44	F	137	44	Y	Y	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	244	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.24±0.12	1.05±0.04	8.96±0.13	NEGATIVE	normal
143457	34	F	150	54	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	231	121	55	6.06±0.11	0.84±0.02	7.53±0.11	6.23±0.12	1.03±0.04	8.94±0.13	NEGATIVE	normal
176678	55	M	148	54	N	N	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 48%	233	110	45	6.20±0.12	0.88±0.04	7.87±0.21	6.22±0.12	1.05±0.04	8.97±0.13	POSITIVE	SVD
143143	45	F	155	64	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	245	151	34	6.32±0.12	0.84±0.02	7.53±0.11	6.25±0.12	1.03±0.04	8.95±0.13	NEGATIVE	normal
134347	66	F	164	64	Y	Y	N	SR WNL	NORMAL LV FUNCTION	266	111	45	6.05±0.13	0.88±0.04	7.87±0.21	6.29±0.12	1.05±0.04	8.99±0.13	NEGATIVE	SVD
175453	54	F	163	45	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	233	110	34	6.30±021	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165676	44	F	145	65	N	Y	N	SR WNL	NORMAL LV FUNCTION	356	134	32	6.05±0.07	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176767	76	M	162	67	N	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	352	121	34	6.16±0.07	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	SVD
176456	54	F	145	65	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	342	111	33	6.08±0.18	0.88±0.04	7.87±0.21	6.25±0.12	1.03±0.04	8.95±0.13	POSITIVE	TVD
145634	65	M	164	43	Y	Y	N	SR WNL	NORMAL LV FUNCTION	234	110	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134545	66	M	155	65	N	N	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	321	151	42	6.06±0.11	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
174563	55	M	148	53	Y	N	Y	SR WNL	NORMAL LV FUNCTION	233	151	55	6.20±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
124543	45	F	145	54	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	232	145	45	6.32±0.12	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
167543	54	M	176	44	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	244	134	34	6.05±0.13	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
145678	54	M	133	54	N	N	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 50%	231	121	55	6.30±021	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
124565	54	F	136	54	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	276	110	45	6.05±0.07	0.84±0.02	7.53±0.11	6.26±0.12	1.09±0.04	8.95±0.13	POSITIVE	normal
134535	55	M	137	64	Y	Y	N	SR ,OLD ASMI	NORMAL LV FUNCTION	256	151	34	6.16±0.07	0.84±0.02	7.53±0.11	6.25±0.12	1.05±0.04	8.96±0.13	NEGATIVE	normal
156743	45	F	176	64	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	246	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.25±0.12	1.06±0.04	8.94±0.13	NEGATIVE	normal
123432	67	M	134	45	N	N	Y	SR WNL	NORMAL LV FUNCTION	246	145	33	5.98±0.09	0.84±0.02	7.53±0.11	6.24±0.12	1.05±0.04	8.96±0.13	NEGATIVE	normal
175433	54	M	147	65	Y	Y	Y	SR ,LVH	NORMAL LV FUNCTION	264	134	35	6.08±0.18	0.88±0.04	7.87±0.21	6.23±0.12	1.03±0.04	8.94±0.13	NEGATIVE	SVD
187654	54	F	163	67	Y	Y	N	SR Tinv in 2,3,avf	RWMA IN INFERO POST SEGMENT.EF 50%	274	121	42	5.49±0.20	1.31±0.09	7.54±0.17	6.22±0.12	1.05±0.04	8.97±0.13	POSITIVE	TVD
176543	54	F	145	65	N	N	N	SR WNL	NORMAL LV FUNCTION	356	134	32	6.06±0.11	0.88±0.04	7.87±0.21	6.25±0.12	1.03±0.04	8.95±0.13	NEGATIVE	SVD
143546	54	M	162	43	Y	Y	N	SR Tinv in 2,3,avf	NORMAL LV FUNCTION	352	121	55	6.20±0.12	0.88±0.04	7.87±0.21	6.29±0.12	1.05±0.04	8.99±0.13	NEGATIVE	SVD
176542	45	M	145	65	Y	N	Y	SR WNL	NORMAL LV FUNCTION	342	111	45	6.32±0.12	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143567	66	F	164	53	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	234	110	34	6.05±0.13	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156563	54	F	164	54	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	321	151	32	6.30±021	0.84±0.02	7.53±0.11	6.08±0.18	0.91±0.04	8.28±0.14	POSITIVE	normal
156743	44	F	155	44	N	N	N	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	233	151	33	6.05±0.07	0.84±0.02	7.53±0.11	6.25±0.12	1.03±0.04	8.95±0.13	NEGATIVE	normal
134532	76	M	148	43	Y	N	Y	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	232	121	35	6.16±0.07	0.88±0.04	7.87±0.21	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	SVD
156754	54	F	145	65	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	244	111	42	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD

176543	65	M	176	53	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	231	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134344	66	M	133	54	Y	N	Y	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	233	134	55	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
134543	55	M	136	44	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	245	121	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
134353	45	F	137	44	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	266	111	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134354	44	F	150	54	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	110	32	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
154453	76	M	148	54	Y	N	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	356	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143444	54	M	155	64	Y	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	352	151	35	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
143455	44	F	164	64	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	145	42	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
143413	34	M	156	45	N	N	Y	SR WNL	NORMAL LV FUNCTION	232	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	8.28±0.14	0.91±0.04	NEGATIVE	normal
134576	56	F	176	65	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	244	121	55	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
165366	67	M	134	67	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	231	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165675	65	F	147	65	N	Y	N	SR WNL	NORMAL LV FUNCTION	276	151	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
135546	66	F	163	43	Y	Y	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	256	121	45	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
155355	55	F	145	43	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	246	111	34	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
154554	45	M	147	65	N	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	246	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
135554	67	M	163	53	N	Y	Y	SR WNL	NORMAL LV FUNCTION	264	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156656	54	F	145	54	N	Y	N	SR WNL	NORMAL LV FUNCTION	274	151	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
135544	54	F	162	64	Y	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	276	145	42	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
146443	54	F	145	64	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	256	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
145673	66	M	164	45	N	Y	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 48%	246	121	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
187797	54	F	155	65	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	233	110	45	6.08±0.18	0.88±0.04	7.87±0.21	6.08±0.18	0.91±0.04	8.28±0.14	NEGATIVE	SVD
186585	44	M	148	67	Y	N	Y	SR WNL	NORMAL LV FUNCTION	232	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
145788	76	M	145	65	N	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	244	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
198675	54	M	176	43	N	Y	N	SR WNL	NORMAL LV FUNCTION	231	145	33	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
156756	65	F	133	65	Y	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	233	110	32	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
134536	66	F	136	53	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	245	134	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
178755	55	M	137	54	N	N	N	SR WNL	NORMAL LV FUNCTION	266	121	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
124545	45	M	176	44	N	Y	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	233	111	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
152111	67	F	134	43	Y	Y	N	SR WNL	NORMAL LV FUNCTION	356	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143143	54	M	147	65	Y	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	352	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
123434	54	F	163	45	N	N	N	SR ,LVH	NORMAL LV FUNCTION	342	151	35	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
134545	54	M	145	65	Y	Y	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 50%	234	145	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134546	54	F	162	67	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	321	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
165625	66	F	145	65	N	N	N	SR ,OLD ASMI	NORMAL LV FUNCTION	233	121	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
135534	54	F	164	43	N	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	232	110	45	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
143415	44	M	145	43	N	Y	Y	SR WNL	NORMAL LV FUNCTION	244	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
143457	76	M	164	65	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	231	121	45	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
176678	54	F	155	53	Y	N	N	SR Tinv in 2,3,avf	RWMA IN INFERO POST SEGMENT.EF 50%	276	111	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143143	44	M	148	54	N	Y	N	SR WNL	NORMAL LV FUNCTION	256	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134347	34	M	145	44	Y	Y	N	SR Tinv in 2,3,avf	NORMAL LV FUNCTION	246	151	33	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
175453	56	F	176	54	Y	N	N	SR WNL	NORMAL LV FUNCTION	246	151	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165676	67	M	133	54	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	264	145	42	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
176767	65	F	136	64	N	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	274	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176456	66	M	137	64	Y	Y	N	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	356	121	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
145634	55	M	150	45	Y	Y	Y	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	352	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134545	45	F	148	65	N	N	N	SR ,LVH	NORMAL LV FUNCTION	342	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
174563	66	F	155	67	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	234	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
124543	54	M	164	65	Y	Y	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	321	151	33	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
167543	44	F	163	43	Y	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	233	151	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
145678	76	M	145	65	N	N	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	232	145	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
124565	54	M	162	53	Y	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	244	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
134535	65	M	145	54	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	231	121	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
156743	66	F	164	44	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	233	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
123432	55	F	155	43	N	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	245	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
175433	45	M	148	65	N	Y	N	SR WNL	NORMAL LV FUNCTION	266	111	45	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD

187654	67	F	145	45	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	110	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
176543	54	F	176	65	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	356	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143546	54	F	133	67	N	Y	N	SR WNL	NORMAL LV FUNCTION	352	121	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176542	54	F	136	65	Y	N	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	233	111	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143567	55	M	137	43	Y	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	232	110	35	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
156563	45	F	176	67	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	244	151	42	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
156743	67	M	134	65	N	N	N	SR WNL	NORMAL LV FUNCTION	231	151	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134532	54	M	147	43	Y	N	N	SR WNL	NORMAL LV FUNCTION	276	145	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
156754	54	M	163	65	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	256	134	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
176543	54	F	145	53	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	246	121	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
134344	54	M	162	54	N	N	Y	SR WNL	RWMA IN INFERO POST SEGMENT.EF 48%	246	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134543	66	M	145	44	Y	N	Y	SR ,LVH	NORMAL LV FUNCTION	264	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134353	54	F	164	43	Y	Y	N	SR WNL	NORMAL LV FUNCTION	274	151	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
134354	44	M	164	65	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	276	145	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
154453	76	F	155	45	Y	N	N	SR WNL	NORMAL LV FUNCTION	256	134	35	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143444	54	M	148	65	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	246	121	42	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
143455	44	M	145	67	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	232	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
143413	34	F	176	65	N	N	N	SR WNL	NORMAL LV FUNCTION	244	121	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134576	67	F	133	43	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	231	111	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165366	54	M	162	45	Y	N	N	SR WNL	NORMAL LV FUNCTION	276	110	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
165675	54	M	145	65	Y	Y	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	256	151	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
135546	54	F	164	67	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	246	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
155355	55	F	155	65	Y	N	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 50%	246	121	35	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
154554	45	F	148	43	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	264	111	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
135554	67	M	145	65	N	Y	N	SR ,OLD ASMI	NORMAL LV FUNCTION	274	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156656	54	F	176	53	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	276	134	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
135544	54	M	133	54	Y	N	N	SR WNL	NORMAL LV FUNCTION	256	121	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
146443	54	M	136	44	Y	N	N	SR ,LVH	NORMAL LV FUNCTION	246	111	34	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
145673	54	M	137	43	N	Y	N	SR Tinv in 2,3,avf	RWMA IN INFERO POST SEGMENT.EF 50%	233	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
187797	66	F	176	65	N	Y	N	SR WNL	NORMAL LV FUNCTION	232	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	DVD
186585	54	F	134	45	Y	N	Y	SR Tinv in 2,3,avf	NORMAL LV FUNCTION	244	151	35	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	normal
145788	44	M	147	65	Y	Y	N	SR WNL	NORMAL LV FUNCTION	231	145	42	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
198675	76	M	163	67	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	233	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156756	54	F	145	43	Y	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	245	121	55	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134536	44	M	162	65	Y	Y	N	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	266	110	45	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
178755	34	F	145	53	N	N	N	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	233	151	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
124545	44	M	164	54	N	Y	Y	SR ,LVH	NORMAL LV FUNCTION	232	121	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
152111	34	F	164	44	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	244	111	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
134546	67	F	155	44	Y	N	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	231	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
123434	54	F	148	54	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	276	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134545	54	M	145	54	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	256	151	35	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
134546	54	M	176	64	Y	Y	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	246	145	42	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
165625	55	F	133	64	Y	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	246	134	32	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	POSITIVE	TVD
135534	45	M	136	45	N	N	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	264	121	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
143415	67	M	137	65	N	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	274	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
143457	54	F	150	67	Y	Y	N	SR WNL	NORMAL LV FUNCTION	276	151	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
176678	54	M	148	65	Y	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	256	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143143	54	F	155	44	N	Y	Y	SR WNL	RWMA IN LV ANT WALL,EF 45%	246	145	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134347	54	M	164	54	N	Y	N	SR WNL	NORMAL LV FUNCTION	233	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
175453	66	M	156	54	Y	N	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	232	134	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
165676	44	F	176	64	Y	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	244	121	45	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
176767	34	F	134	64	N	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	231	111	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176456	67	M	147	45	Y	Y	Y	SR WNL	NORMAL LV FUNCTION	233	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
145634	54	F	163	65	Y	Y	N	SR WNL	NORMAL LV FUNCTION	245	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
134545	54	M	145	67	N	N	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	266	151	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal

174563	54	M	147	65	N	N	N	SR ,LVH	NORMAL LV FUNCTION	233	145	42	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
124543	55	M	163	43	N	Y	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 48%	232	134	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
167543	45	F	145	65	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	244	121	55	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
145678	67	F	162	53	Y	N	N	SR WNL	NORMAL LV FUNCTION	231	110	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
124565	54	M	145	54	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	276	151	34	5.49±0.20	1.31±0.09	7.54±0.17	6.20±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
134535	54	F	164	44	Y	Y	N	SR WNL	NORMAL LV FUNCTION	256	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
156743	54	F	155	54	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	246	145	33	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
123432	54	F	148	54	N	N	N	SR ,LVH	NORMAL LV FUNCTION	246	134	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
175433	66	F	137	53	N	Y	N	SR WNL	NORMAL LV FUNCTION	264	121	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
187654	44	M	176	54	Y	Y	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	274	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
176543	34	F	134	44	Y	N	N	SR WNL	NORMAL LV FUNCTION	356	121	55	5.49±0.20	1.31±0.09	7.54±0.17	6.26±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
143546	67	M	147	44	N	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	352	111	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176542	54	M	163	54	N	N	N	SR ,LVH	NORMAL LV FUNCTION	342	110	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143567	54	M	145	54	Y	Y	N	SR WNL	RWMA IN INFERO POST SEGMENT.EF 50%	234	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
156563	54	F	162	64	Y	Y	N	SR ,LVH	NORMAL LV FUNCTION	321	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	POSITIVE	normal
156743	55	M	145	64	N	N	Y	SR ,OLD ASMI	NORMAL LV FUNCTION	233	121	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134532	45	M	164	45	Y	N	Y	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	232	111	42	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
156754	67	F	164	65	Y	Y	N	SR WNL	NORMAL LV FUNCTION	244	110	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
176543	54	M	155	67	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	231	134	55	5.49±0.20	1.31±0.09	7.54±0.17	6.26±0.12	1.09±0.04	8.95±0.13	POSITIVE	TVD
134344	54	F	148	65	N	N	N	SR Tinv in 2,3,avf	RWMA IN INFERO POST SEGMENT.EF 50%	233	121	45	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134543	54	M	145	53	N	N	N	SR WNL	NORMAL LV FUNCTION	245	111	34	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
134353	54	M	176	54	Y	Y	N	SR Tinv in 2,3,avf	NORMAL LV FUNCTION	266	110	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	POSITIVE	DVD
134354	66	F	133	44	Y	Y	N	SR WNL	NORMAL LV FUNCTION	232	151	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
154453	44	F	162	44	N	N	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	244	151	35	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143444	76	M	136	54	Y	Y	N	SR OLIWMI	RWMA IN INFERO POST SEGMENT.EF 48%	231	145	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
143455	54	M	137	54	Y	Y	N	SR,OLD HLMI	RWMA inANT,LAT WALL,EF42%	276	134	32	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
143413	44	F	150	64	N	N	N	SR,OLD ASMI	RWMA IN LV ANT WAL ,EF 53%	256	121	55	5.49±0.20	1.31±0.09	7.54±0.17	6.24±0.12	1.05±0.04	8.96±0.13	POSITIVE	TVD
134576	34	F	148	64	N	Y	N	SR ,LVH	NORMAL LV FUNCTION	246	145	33	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165366	44	F	155	45	Y	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	246	134	35	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
165675	34	M	164	65	Y	Y	N	SR ,OLD ASMI	RWMA IN LV ANT WALL,EF 45%	264	121	42	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal
135546	67	F	163	67	N	Y	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	274	111	55	5.49±0.20	1.31±0.09	7.54±0.17	6.25±0.12	1.03±0.04	8.94±0.13	NEGATIVE	TVD
155355	54	M	145	65	N	N	N	SR WNL	RWMA IN LV ANT WALL,EF 45%	356	110	45	6.08±0.18	0.88±0.04	7.87±0.21	6.29±0.12	1.05±0.04	8.99±0.13	POSITIVE	DVD
154554	54	M	162	65	Y	N	Y	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	352	151	34	6.08±0.18	0.88±0.04	7.87±0.21	6.22±0.29	1.07±0.08	8.10±0.26	NEGATIVE	DVD
135554	54	M	145	43	Y	Y	N	SR ,OLD ASMI	RWMA IN DISTAL IVS ,LV APEX,ANTLAT WALL EF 38%	342	151	32	5.98±0.09	0.84±0.02	7.53±0.11	6.87±0.16	0.91±0.04	8.28±0.14	NEGATIVE	normal