A Dissertation on

A STUDY ON CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE IN YOUNG PATIENTS UNDERGOING CORONARY ANGIOGRAPHY

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032.

In partial fulfillment of the Regulations
For the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001.
APRIL 2015

CERTIFICATE BY INSTITUTION

This is to certify that **Dr. G.SURESH,** Post - Graduate Student (MAY 2012 TO APRIL 2015) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**A STUDY ON CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE IN YOUNG PATIENTS UNDERGOING CORONARY ANGIOGRAPHY"** under our guidance and supervision in partial fulfillment of the regulations laid down by the Tamil Nadu Dr.M.G.R. Medical University,Chennai for M.D (General Medicine), Degree Examination to be held in April 2015.

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DECLARATION

I **Dr. G.SURESH** declare that I carried out this work on "**A STUDY ON CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE IN YOUNG PATIENTS UNDERGOING CORONARY ANGIOGRAPHY**"at the CARDIOLOGY HUTTON WARD of Government Stanley Hospital during the period NOVEMBER 2013 to SEPTEMBER 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

Dr.G.SURESH

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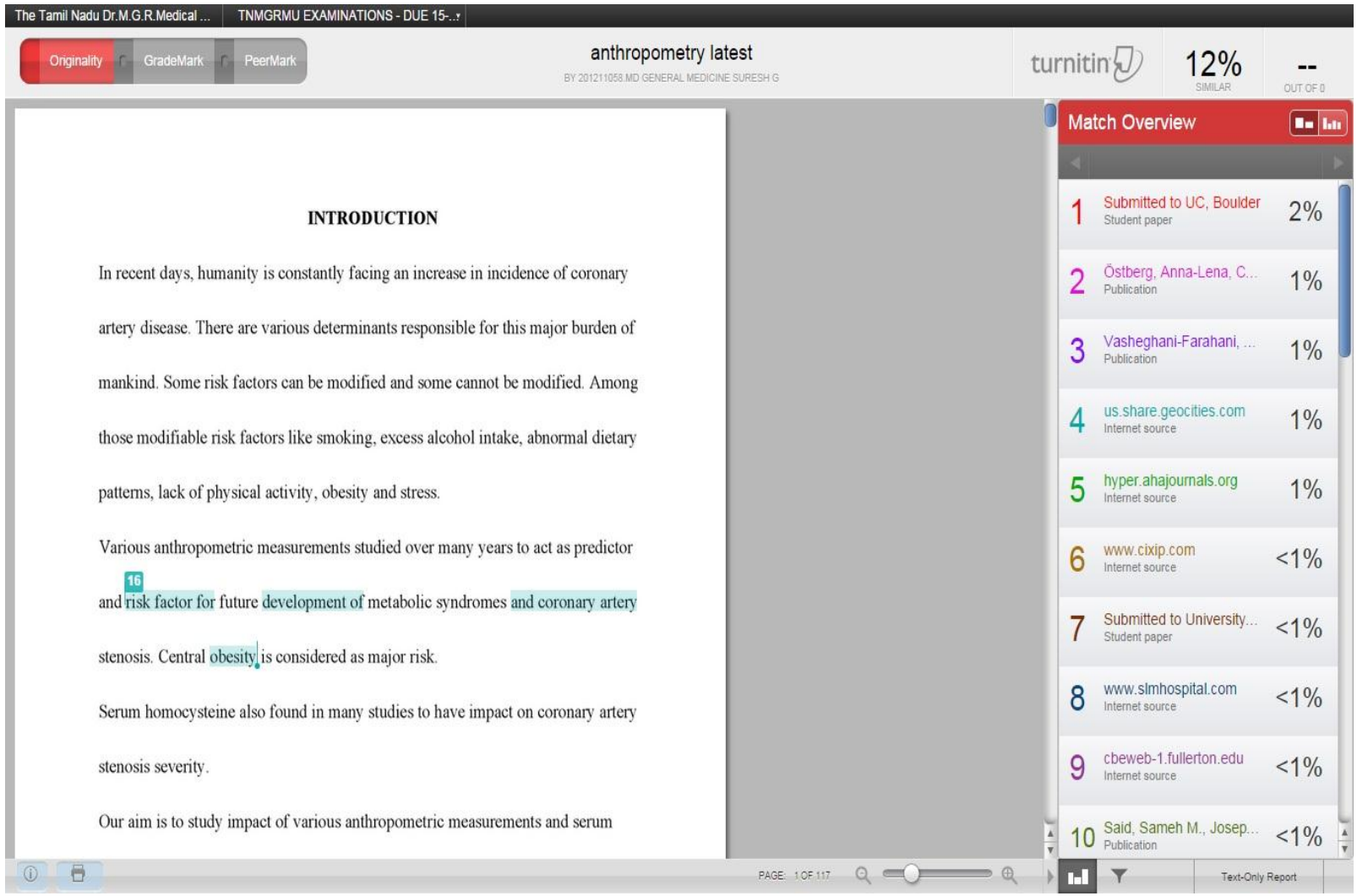
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INTRODUCTION
In recent days, humanity is constantly facing an increase in incidence of coronary artery disease. There are various determinants responsible for this major burden of mankind. Some risk factors can be modified and some cannot be modified. Among those modifiable risk factors like smoking, excess alcohol intake, abnormal dietary patterns, lack of physical activity, obesity and stress.
Various anthropometric measurements studied over many years to act as predictor and risk factor for future development of metabolic syndromes and coronary artery stenosis. Central obesity is considered as major risk.
Serum homocysteine also found in many studies to have impact on coronary artery stenosis severity.
Our aim is to study impact of various anthropometric measurements and serum
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ABBREVIATIONS

CAD – CORONARY ARTERY DISEASE
CAG – CORONARY ANGIOGRAM
LAD – LEFT ANTERIOR DESCENDING
LCX – LEFT CIRCUMFLEX
RCA – RIGHT CORONARY ARTERY
HT – HEIGHT
WT – WEIGHT
BMI – BODY MASS INDEX
SAD – SAGITTAL ABDOMINAL DIAMETER
TSFT – TRICEPS SKIN FOLD THICKNESS
WHR – WAIST HIP RATIO
LDL – LOW DENSITY LIPOPROTEIN
HDL – HIGH DENSITY LIPOPROTEIN
VLDL – VERY LOW DENSITY LIPOPROTEIN
TGL – TRIGLYCERIDES
HT – HYPERTENSION
DM – DIABETES
CKD – CHRONIC KIDNEY DISEASE
CVA – CEREBROVASCULAR ACCIDENT
ECG ELECTROCARDIOGRAPHY
ECHO – ECHOCARDIOGRAPHY
CT- COMPUTED TOMOGRAPHY
AGE - ADVANCED GLYCATION END PRODUCTS
BP – BLOOD PRESSURE
COA – COENZYME A
ACC – AMERICAN COLLEGE OF CARDIOLOGY
AHA – AMERICAN HEART ASSOCIATION

ABSTRACT :

AIMS AND OBJECTIVES

1. TO STUDY CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE.
2. TO IDENTIFY STRONG ANTHROPOMETRIC PARAMETER CORRELATING WITH THE SEVERITY OF CORONARY ARTERY DISEASE USING CAG.

KEY WORDS: Anthropometry, Sagittal abdominal diameter, Triceps skin fold thickness, Serum homocysteine, Modified gensini scoring, Coronary angiography

MATERIALS AND METHODS

PLACE OF STUDY:

HUTTON WARD, DEPARTMENT OFCARDIOLOGY, STANELY MEDICAL COLLEGE AND HOSPITAL, CHENNAI**.**

DURATION: NOV 2013 TO SEP 2014

STUDY DESIGN

PROSPECTIVE AND OBSERVATIONAL STUDY

SOURCE OF DATA:

PATIENTS WITH HISTORY OF CHEST PAIN WHO FULFILL THE INCLUSION AND EXCLUSION CRITERIA, GETTING ADMITTED AT CARDIOLOGY HUTTON WARD, STANLEY MEDICAL COLLEGE, CHENNAI DURING THE PERIOD OF NOV 2013 TO SEPTEMBER 2014 AND PLANNED FOR CORONARY ANGIOGRAM.

SAMPLE SIZE: 75

INCLUSION CRITERIA:

1. ALL PATIENTS UNDER 50YRS OF AGE , ADMITTED IN CARDIOLOGY DEPARTMENT HUTTON WARD, STANLEY MEDICAL COLLEGE FOR UNDERGOING CORONARY ANGIOGRAM

EXCLUSION CRITERIA:

1. PATIENTS KNOWN TO HAVE RHEUMATIC HEART DISEASE, ISCHEMIC DCMP.
2. PATIENTS WHO ARE ABOVE THE AGE OF 50 YRS.
3. PATIENTS WHO ARE CHRONIC ALCOHOLICS.
4. PATIENTS WITH CHRONIC KIDNEY DISEASE.
5. PATIENTS WITH ASCITES AND OTHER INTRABDOMINAL PATHOLOGY.
6. PATIENTS WITH HB < 11GM%

METHODOLOGY

Patients admitted for angiography between November 2013 to September 2014 will be included in the study.

DATA COLLECTED USING A PROFOMA MEETING THE OBJECTIVES OF THE STUDY.

DETAILED HISTORY, PHYSICAL EXAMINATION AND NECESSARY INVESTIGATIONS WILL BE UNDERTAKEN.

They are subjected for anthropometry measurements including height, weight, BMI, hip and waist circumference, WHR, sagittal abdominal diameter, triceps skin fold thickness and SAD/TSFT ratio measured. Also their serum homocysteine level, glycemic status, Presence or absence of hypertension and diabetes, smoking and alcohol intake with duration, life style & physical activity, frequency of chest pain, ECG findings and echo status will be noted prior to angiogram.

Post angiogram evaluation of severity of coronary artery stenosis made my Modified Gensini Score.

THE PURPOSE OF THE STUDY WILL BE EXPLAINED TO THE PATIENT AND INFORMED CONSENT OBTAINED

CONCLUSION

In my study using 75 patients under 50 years, I am able to come to a conclusion of some anthropometric variables better correlating with the severity of coronary stenosis and some not showing much correlation as listed below

1) Increased Waist-hip ratio, sagittal abdominal diameter, triceps skin fold thickness and SAD/TSFT ratio has got better correlation with the severity of coronary artery disease, with SAD/TSFT and WHR has got maximum significance of all anthropometric measurements.

2) Increased BMI poorly correlating with the severity of CAD as shown by p value more than 0.05.

3) Increased homocysteine level better correlates with the severity of coronary artery disease.

4) Increased LDL cholesterol value had good correlation with CAD severity butdecreased HDL cholesterol not correlating much with coronaryartery disease.

5) Duration of smoking but not mere smoking history better correlates with the severity of coronaryartery disease.Smoking is found to be an independent high risk factor even in patients with low BMI, low waist or hip circumference and low WHR or SAD/TSFT ratio.

6) Presence of hypertension and duration of diabetes got positive correlation on coronary artery disease severity.

7) Left ventricular ejection fraction (LVEF) also correlates with the severity of coronary artery disease with an inverse relation between them.

8) Modified gensini score – higher the modified gensini score, severe is the coronary artery disease.

9) It appears that the old concept of BMI being considered as major risk factor in the past will not apply nowadays as an important determinant for CAD risk assessment, as it is shown in our study and in many major studies to be statistically less significant and it's better to give importance to central Vs peripheral obesity ratio methods to assess future risk of CAD.

10) We feel great emphasis should be given on cessation of smoking compared to control of obesity as it's a major atherosclerotic risk factor even in thin individuals and those patients are prone for CAD at very young age.

INTRODUCTION

In recent days, humanity is constantly facing an increase in incidence of coronary artery disease. There are various determinants responsible for this major burden of mankind. Some risk factors can be modified and some cannot be modified. Among those modifiable risk factors like smoking, excess alcohol intake, abnormal dietary patterns, lack of physical activity, obesity and stress.

Various anthropometric measurements have been studied over many years to act as predictor and risk factor for future development of metabolic syndromes and coronary artery stenosis. Central obesity is considered as major risk.

Serum homocysteine has also been found in many studies to have impact on coronary artery disease severity.

Our aim is to study impact of various anthropometric measurements and serum homocysteine level and their correlation with coronary artery disease.

REVIEW OF LITERATURE

Coronary artery disease is basically a disease affecting those blood vessels that supply blood to heart. Atherosclerosis of coronary vessels is the forerunner for formation of coronary artery disease. It became significant when the narrowing of coronary vessels exceeds 50 % in any of coronary vessels. Basically there are 3 major coronary vessels and more than 8 minor branches of coronary arteries.

Myocardial ischemia or infarct patients presents with any of the following symptoms including typical retrosternal chest pain which is compressive or unexplainable vague pain, dyspnea and angina equivalents like sweating, back pain, palpitation and vomiting.

Hereby I am studying the correlation of various anthropometric measures and serum homocysteine level in patients under fifty years comparing with the coronary artery disease severity using modified gensini scoring method.

There are various risk factors attributed to the formation of plaques in the coronary vessels. Those include smoking, lack of exercise, obesity,diabetes,hypertension,hyperhomocysteinemia and so on.

Development of coronary artery disease can be diagnosed by invasive and noninvasive methods.

Invasive methods include those of CT angiogram using cardiac catheters and intravascular ultrasonography.

Noninvasive methods include simple ECG,echocardiography,treadmill test&cardiac MRI angiography.

ECG acts as the basic first line investigation for those presenting for the first time with cardiac chest pain. It will provide information on ischemia,injury or infarct of a particular cardiac wall involvement and also the concomitant presence of bundle branch blocks and nodal involvement. The problem with ECG is it will not detect all cases of ischemia as it has got low sensitivity and there are many bias possible by means of normal variants and by coincidental electrolyte disturbances in same patient.

Exercise testing

It is a cardiovascular stress test that uses treadmill bicycle exercise with ECG and blood pressure monitoring. Testing is a diagnostic procedure in which cardiovascular stress induced by pharmacologic agents is demonstrated in patients with decreased functional capacity or in patients who cannot exercise.

Both of which helps to identify those at risk of stress induced coronary vasospasm and helps to warn them to avoid strenuous exercise and activities and need for coronary angiography.

Echocardiography is a gold standard test to identify hypokinesia and akinesia of a particular portions of myocardium that could be basal, mid or apical portion of the left ventricular anterior wall, inferior wall, posterior wall or lateral wall. It also helps to identify the LV ejection fraction.

It can also help to exclude other causes of cardiac pain other than frank coronary vascular stenosis including septal defects, aortic and other valve involvement like stenosis or valve prolapse.

Obesity and type -2 diabetes are the major risk factors attributed in coronary stenosis. Population most affected by these are men in south East Asia particularly India and Middle East.

There are also genetic predilection to the development of atherosclerosis.

METABOLIC SYNDROME

The metabolic syndrome is closely related with development of diabetes, insulin resistance and high chances of development of CAD.

Its diagnosis comprises following components

1) Waist circumference >90 in Indian men and >80 in Indian women with
2) Two or more of the following
    a. Fasting triglycerides > 150 mg% , HDL<40mg% for men and <50mg% for women on diagnosis
    b. BP >130 mm of hg systolic , > 85mmm of hg diastolic on diagnosis
    c. Fasting plasma glucose >100mg% or known diabetic.

The primary pathogenesis for metabolic syndrome being increased insulin resistance. High free fatty acids in serum leads to substrate availability and thus produces insulin resistance by down streaming signals. Excess fatty acids impairs insulin mediated glucose uptake and accumulation of triglycerides in muscles and also increased glucose and TGL production in liver increased.

Various anthropometric measurements

Height – measured with standard ruler with patient in standing position with abdominal relaxation and with feet's together and whole body attached closely to the wall.

Weight -measured with weighing machine with patient looking forward, and in straight position without leaning forward.

BMI - body mass index is the ratio of weight in kilograms to the height in meter squares.

$$\mathrm{BMI} = \frac{weight(\mathrm{kg})}{height^2(\mathrm{m}^2)}$$

For Indian population, BMI more than 23 is considered overweight and more than 37.5 is considered morbid obesity.

	WHO CRITERIA	RECOMMENDATION FOR INDIANS
Normal	less than 25 kg per m^2	less than 23
Overweight	more than 25 kg per m^2	more than 23
Obese	more than 30 kg per m^2	more than 27.5
Severe obesity	more than 35 kg per m^2	more than 32.5
Morbid obesity	more than 40 kg per m^2	more than 37.5

Fat distribution in human body can in form of visceral or subcutaneous fat deposits.

Among those two , visceral fat deposits poses major health risk as those fatty acids derived from visceral fat will be delivered to liver compared to subcutaneous fat which will be delivered to peripheral circulation mostly. It is also established that increased visceral fat and waist circumference is directly related to the future development of CAD.

Even in the subcutaneous fat distribution, central abdominal fat is more dangerous than peripheral body subcutaneous fat. Even peripheral fat considered to have beneficial effect.

Visceral fat is more in cases of Asian / Indians. Waist circumference, waist-hip ratio, sagittal abdominal circumference (SAD), SAD/triceps skin fold thickness are among parameters which determines central obesity.

Waist circumference is the measure of circumference measured at the level of midway between iliac crest and lower rib margin. For Indian population, those with WC > 90 cm in men and > 80 cm in women is considered as abnormally high waist circumference and are at high risk for development of insulin resistance, diabetes and coronary artery stenosis.

Hip circumference is being measured at the level of greater trochanter of femur, this measurement is being used for comparison with waist circumference, to measure waist/hip ratio.

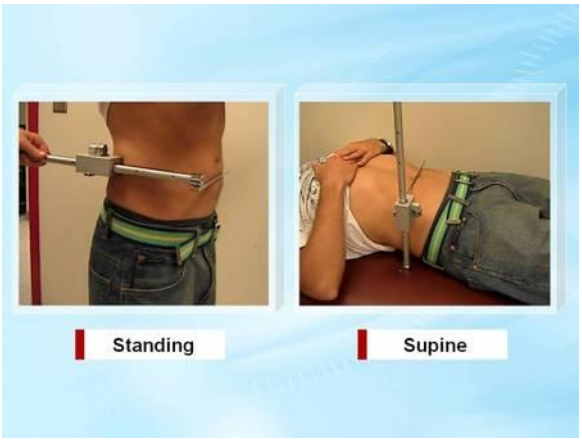
Waist-hip ratio (WHR) is the measure of waist circumference divided by hip circumference. This ratio projects the central obesity. The value must not exceed more than 0.9 in men and 0.8 in women.

Sagittal abdominal diameter (SAG) is an important reflection of visceral obesity.-

SAD was measured as the height of the abdomen in the supine position from the table measured at the waist after gentle expiration, by use of portable sliding-beam caliper.

The upper arm of the caliper was at the upper abdominal surface at iliac crest level, which approximates the level of the L4-L5 interspace.

For a person within normal limits of BMI, sagittal abdominal diameter should be within 25 cms. SAD above 30 correlated with high incidence of diabetes, metabolic syndrome, hypertension and coronary artery disease.



Triceps skin fold thickness is a measure of peripheral fat mass. It can be measured by pinching the vertical skin fold over mid-triceps region pinching away from muscle midway between acromial process and

olecranon process and measuring the thickness using vernier caliber or other instruments.

Normal adult triceps skin fold thickness

Male - 11-12.5 mm

Female – 15-16.5 mm

Boys at the age of 2 years have a median triceps skin fold of 10 mm after which it decreases gradually and by the age of eight years, reaches its lowest value of 8mm. After that it shows gradual increase, so that by age of twelve years, reaches a peak of 10 mm.

In case of females, stable value between 2 to 8 years, with average of sixteen millimeter by 15 years. By adulthood have normally 17 to 22 mm.

By comparing the central obesity factors with the peripheral fat distribution, we are able to bring a better predictor of future dyslipidemia, diabetes and CAD occurrence.

Often the peripheral fat distribution is safe than that of central fat distribution as shown in many studies like increased ‘waist-hip ratio’ and ‘waist-thigh ratio’ has got worse outcome as evident in coronary angiogram. Waist-hip ratio is one among such predictor. Many studies have shown a good correlation between ‘waist hip ratio’ with the incidence of coronary artery disease in the form of angina(either stable or unstable), myocardial ischemia and infarction.

Sagittal abdominal diameter / triceps skin fold thickness (SAD/TSFT)

Another such good variable to comparing central with peripheral adiposity is the sagittal abdominal diameter / triceps skin fold thickness. This has been recently concentrated and found as better anthropometric variable to predict CAD and correlates with the severity and extend of coronary stenosis as measured by using modified gensini scoring system.

Anatomy of coronary arteries

Coronary artery are those vessels that around the heart arising from the coronary sinus at aortic root level. There are right coronary artery and left main coronary arteries arising respectively from right and left aortic sinuses.

Left main coronary artery first gives of left circumflex coronary artery that runs in left anterior atrioventricular groove and then continues down anteriorly as left anterior descending artery in the anterior interventricular groove.

Right coronary artery has its own course starting from anterioratrioventricular groove and runs to posterior atrioventricular groove and descends usually in posterior interventriculargroove**.**

Dominant artery is the one coronary artery that give rise to posterior interventricular branch and supplies the posterior part of the ventricular septum and sometimes posterolateral wall of the left ventricle.

The dominant artery is usually the right coronary and less commonly from left circumflex artery, which after passing posterior atrioventricular groove runs down in posterior interventricular groove.

Anastomoses between right and left coronary arteries are more during fetal life and the same is much reduced by the end of the first year of life. Anastomoses provides collateral circulation when either of the artery is subjected to stenosis. They become prominent in scenarios of hypoxia and CAD. Collateral circulation also given by tiny branches from mediastinal and bronchial vessels.

The artery to the Sino atrial node is usually from the anterior circumflex segment and less common from the circummarginal branch.

Stenosis of this branch is crucial as it causes SA node to dysfunction and that results in an events like sick sinus syndrome with very low heart rate as other conduction system below the level of SA node will take over the intrinsic cardiac impulse generator and that have an intrinsic rate less than that of SA node.

Those subsidiary pacemakers includes conduction fibers namely Bachmann, wenkebach and thorel pathways that connects SA node with AV node, atrial musculature itself, AV node, bundle of his, right and left bundle branch and left anterior or posterior fascicular fibers and ventricular

myocytes themselves. Hence bradycardia sets in if nodal arterial branch got thrombosed.

There are several branches given off from these main coronary arteries. These include

1) **Form the left coronary artery–**

   Left main coronary artery arising from the left aortic sinus and that gives rise to

   a) **Left anterior descending branch** which runs in anterior interventricular groove and end up after winding in posterior interventricular groove.

   b) Left circumflex coronary artery – which is also a main branch from left main coronary artery and that runs in anterior atrio-ventricular region and winds to end in posterior atrio-ventricular groove.

   c) **Left obtuse marginal branch** is usually a sub branching from left circumflex artery.

   d) Left diagonal branch arises from the left anterior descending artery**.**

e) **Intermediate artery –** found in around one third of patients arising between LAD and LCX and one runs along anterolateral aspect of the left ventricle.

f) There can be more than one marginal branches in few people possible.

2) **Right coronary artery -** arises from the right aortic sinus of aorta and

   a) Runs along the right atrioventricular groove and winds up from inferior portion and runs along the posterior interventricular grove to supply the myocardium adjacent to it.

   b) Right acute marginal branch arise when the right coronary artery winds in the inferior surface of the heart.

   c) A **branch to the Sino atrial node.**

   d) Several other minor anatomic variant branches can occur.

Communication between the Left and Right Coronary Arteries

1. The artery of the conus with left anterior descending artery.
2. At the **interventricular septum,** with septal perforators of left anterior descending artery and posterior descending artery.
3. At the **apex** of the heart, between left anterior descending and posterior descending artery.

4. At the **crux**, between the left circumflex and posterior descending artery.

Coronary microvasculature – it includes the microcapillary circulation which runs in the form of coronary capillaries that are parallel to the myocytes.

Distribution of Blood supply of the heart

Heart has got three important layers including epicardium, myocardium and endocardium.

The epicardium can be supplied by micro branches from epicardial vessels.

The myocardial blood supplied is briefed here-

Most of the left ventricular surface except near posterior interventricular septum is supplied by left anterior descending artery.

Most of the right ventricle except near anterior interventricular septum is supplied by posterior descending artery.

Both atria are supplied by branches form circumflex and right coronary artery.

Coronary oxygen supply-

Myocardium unlike other tissues the percentage 02 demand per gram of myocardium is very high compared to any other cells.

It is dependent on pa02 content of blood & hemoglobin concentration.

The main factors responsible for increased oxygen demands includes heart rate, systolic blood pressure and left ventricular contractility factor.

The coronaries are being filled during diastolic phase of the cardiac cycle and so when the heart rate increases (which shortens diastolic phase) coronary arterial filling decreases and myocardium suffers from hypoperfusion and undergoes ischemia and if the same status continues, it will end up in infarct.

Anatomic Region of Heart	Coronary Artery (most likely associated)
Inferior	Right coronary
Anteroseptal	Left anterior descending
Anteroapical	Left anterior descending (distal)
Anterolateral	Circumflex
Posterior	Right coronary artery

Coronary Collateral Circulation

After total coronary occlusion, residual myocardium gets perfused through native coronary collateral channels that open when an intercoronary pressure gradient between origin and distal vessel drops. There is large individual variability in the function of coronary collaterals in patients with chronic stenosis. In those without coronary collaterals, coronary pressure distal to balloon angioplasty comes down to 10 mm Hg. In other patients, collaterals proliferate to the point where they are sufficient not only to

maintain normal resting perfusion but also to prevent stress-induced ischemia at submaximal cardiac workloads. Ischemia does not develop during PCI balloon occlusion when fractional flow reserve (based on coronary wedge pressure during occlusion minus venous pressure) is greater than 0.25. A large observational cross-sectional study has shown that patients with high distal coronary pressure attained through collaterals, fractional flow reserve > 0.25 have a lower CAD rate.

The volume of residual coronary flow through collaterals or by subtotal coronary occlusion has got most influence on the actual time of irreversible injury in patients with chronic CAD. The relationship between infarct size and the area at risk of ischemia during a total occlusion is inversely related to collateral flow.so this gives us the importance of collateral vessel function in determining prognosis. When subendocardial collateral flow is more than approximately 30% of resting flow values, more than 1 hour of protection to the myocardial injury is for sure. More moderate subendocardial ischemia from a subtotal occlusion with flow reduction of

less than 50% from baseline can prevent for about 5 hours the irreversible injury of the myocardium. This explains the reason behind less symptomatology and absence of ECG finding in early stages without producing much of myocardial necrosis. It also explains the clinical observation that late coronary reperfusion with ongoing ischemia can salvage myocardium beyond the 6-hour time limit predicted from experimental models of infarction.

Venous drainage of the heart:-

The heart is drained from all around and final drainage is via coronary sinus that open up in the right atrium between the opening of the inferior vena cava and the right atrioventricular orifice.

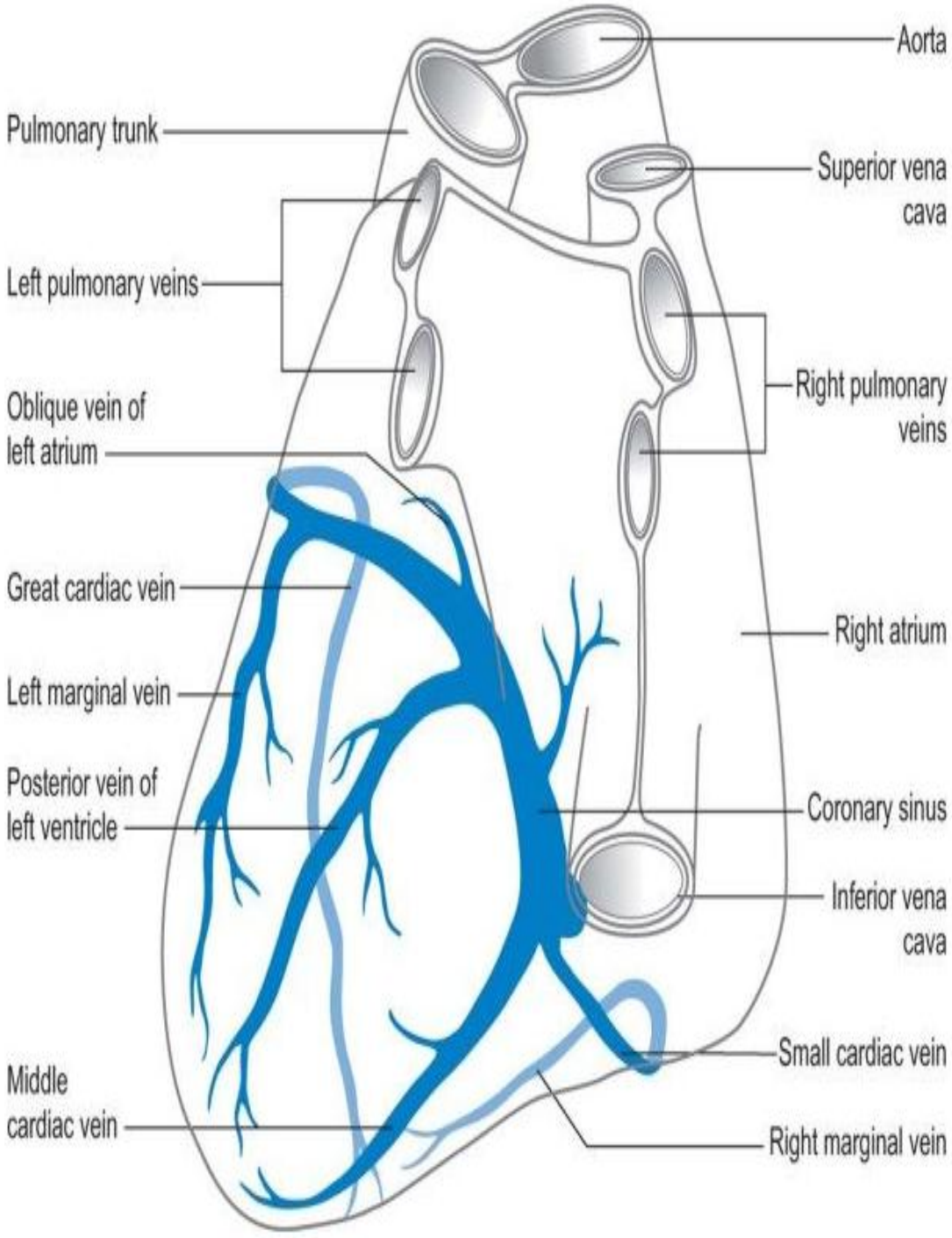
The tributaries of coronary sinus includes great, small and middle cardiac veins, the posterior vein of the left ventricle and the oblique vein of the left atrium

Great cardiac vein – drains left atrium and both ventricles.

Small cardiac vein – drains Posterior part of right atrium and right ventricle.

Middle cardiac vein – drains from cardiac apex.

Posterior vein of left ventricle and oblique vein of left atrium drains the corresponding chambers. Anterior cardiac vein in anterior part of right ventricle and joins coronary sinus. Multiple perforating veins draining in individual chambers drains corresponding myocardium.


Aorta
Pulmonary trunk
Superior vena cava
Left pulmonary veins
Right pulmonary veins
Oblique vein of left atrium
Great cardiac vein
Right atrium
Left marginal vein
Posterior vein of left ventricle
Coronary sinus
Inferior vena cava
Small cardiac vein
Middle cardiac vein
Right marginal vein

ATHEROSCLEROSIS

Progressive accumulation of smooth muscle cells and lipids within the intima.

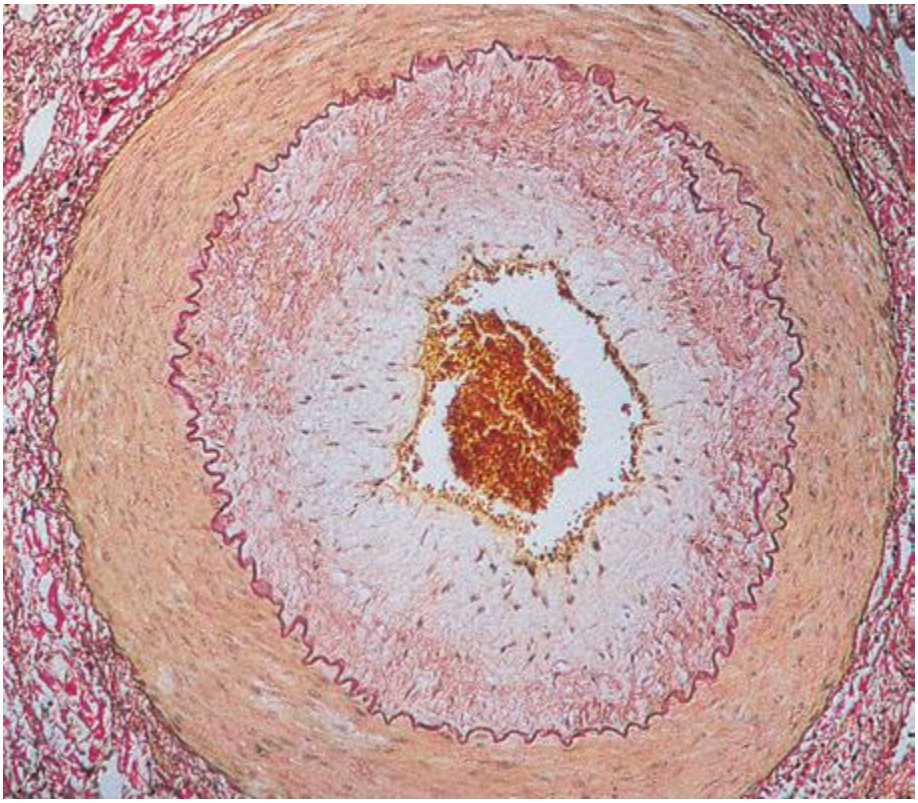
Also starts with stretching of vessels causing smooth muscle cells to arrange in parallel rows and reactive deposits of fibrin and over the course of time, this causes vessels to became fibrotic due to fibrin generation by epithelial cells and became stiff not yielding to recoil and finally the cardiac output with its full systolic pressure will be transmitted directly to the offending organs and capillary damage sets in.

Progression of atherosclerosis:

Early stages: Proliferation of smooth muscle cells and accumulation of lipid.

Later: macrophages, lymphocytes influx and connective tissue enlargement.

Very late stage: thrombus with canals within the thrombus.



MORPHOLOGY:

Initial lesion of atherosclerosis

Fat streaks: can be found in young children as well as adults at any point.

Intimal cell masses: found in branching places.

Characteristic lesion of atherosclerosis:

Fibrous fatty plaque and fibrous cap which constitutes layer of fibrous connective tissue overlying the atheroma which has foam cells (A modified macrophages) and smooth muscle cells, and also fibroblasts.

Atheroma: lipid rich necrotic center of the lesion.

Risk factors include

Hypertension

- Dyslipidemia
- Chronic smoking
- Diabetes
- Obesity with high BMI ,central obesity plays a major role
- increasing age
- male sex
- physical inactivity
- Stressful life patterns.

COMPLICATIONS OF ATHEROSCELOROSIS

Those atheroma more prone for complications include those with a) calcification b) large thrombus c) neovascularization d) ulceration

Most common complications depending on the organs involves

1) hypertension ,
2) Chronic kidney disease
3) Hypertensive retinopathy
4) Chronic headache ,
5) Hypertensive small vessel ischemic changes in brain
6) Stroke – either ischemic or hemorrhagic stroke
7) Neuropathy
8) Aneurysm formation
9) Myocardial ischemia or infarction

HYPERTENSION:

A person is labelled as hypertensive if systolic bp > 140 mmhg and diastolic > 90 mmhg.

Systolic cutoff is increased to 150 persons older than 50 years to start on antihypertensive medications.

Hypertension can be

a) Benign hypertension –with hyaline arteriosclerosis,without end organ damage and which remain asymptomatic or

b) Malignant hypertension – with end organ damage. Morphologically showing onion peel vessel damage and with fibrinoid necrosis.

Basically hypertension can be

1) Essential hypertension – without well known cause

2) Secondary hypertension – due to any of the below causes

Renovascular Ischemia: Renovascular stenosis or atherosclerosis of Renal Artery or arterioles which result in renin release resulting in hypertension.

Fibro muscular Dysplasia of Renal Artery is a congenital disorder, with

Progressive concentric thickening of the Renal Artery which occurs in young females.

Cushing's syndrome – primary hypersecretion of cortisol.

Conn's Syndrome - Primary hypersecretion of aldosterone.

Pheochromocytoma- Adrenal medullary tumor. Norepinephrine is secreted in spurts, so patient will have wild paroxysmal elevations in blood pressure.

DYSLIPIDEMIA

Lipoproteins are those substances which contains mixed protein content and lipid substances.

It can be

Chylomicrons

Low density lipoprotein

Intermediate density lipoprotein

Very Low density lipoprotein

High density lipoprotein

Apoproteins are the proteins associated with lipoproteins

There are numerous apoproteins. Apoproteins of importance are

B48,B100, A1, A2, A4, C1, C2, C3 and E

VARIOUS LIPOPROTEINS ARE

CHYLOMICRONS – source being intestinal wall. Carries exogenous triglycerides to the liver. It's around 90-1000 nm diameter. Density being <0.95 g/ml. main content being triglycerides. With Apo A1 A2 A4 B48 C1 C2 C3 E

CHYLOMICRON REMNANTS– formed after chylomicron acted upon by lipoprotein lipase with removal of most apoproteins with addesapoB48, E and cholesterol.

VLDL - smaller than chylomicron, again from intestinal origin with triglycerides being major component, with Apo B100 C1 C2 C3

IDL – derivative of VLDL after acted upon by lipase with removal of C1 C2 C3 and Apo E add up.

LDL – derived from VIDL and IDL after utilization in liver and removal of Apo E. contains loads of cholesterol.

HDL – derived from liver, intestine and from chylomicrons VLDL with Apo A1 A2 A4 C1 C2 C3 D and E. has more triglycerides and less cholesterol.

Free / albumin bound fatty acids

HDL cholesterol is named good cholesterol as it has got less cholesterol and has got reverse cholesterol transport bringing the cholesterol from periphery to liver for action by HMG CoA reductase and generation of free fatty acids for utilization and gluconeogenesis. Provides protection from coronary artery disease.

LDL transports and delivers free cholesterol to the periphery and thus acts as bad lipoprotein as this assists in the atherogenesis process. Causative for CAD.

VLDL – Provides endogenous triglycerides from liver to peripheral tissue.

Table 25–1. Composition of the Lipoproteins in Plasma of Humans.

				Composition			
Lipoprotein	**Source**	**Diameter (nm)**	**Density (g/mL)**	**Protein (%)**	**Lipid (%)**	**Main Lipid Components**	**Apolipoproteins**
Chylomicrons	Intestine	90–1000	< 0.95	1–2	98–99	Triacylglycerol	A-I, A-II, A-IV,[1] B-48, C-I, C-II, C-III, E
Chylomicron remnants	Chylomicrons	45–150	< 1.006	6–8	92–94	Triacylglycerol, phospholipids, cholesterol	B-48, E
VLDL	Liver (intestine)	30–90	0.95–1.006	7–10	90–93	Triacylglycerol	B-100, C-I, C-II, C-III
IDL	VLDL	25–35	1.006–1.019	11	89	Triacylglycerol, cholesterol	B-100, E
LDL	VLDL	20–25	1.019–1.063	21	79	Cholesterol	B-100
HDL	Liver, intestine, VLDL, chylomicrons					Phospholipids, cholesterol	A-I, A-II, A-IV, C-I, C-II, C-III, D,[2] E
HDL_1		20–25	1.019–1.063	32	68		
HDL_2		10–20	1.063–1.125	33	67		
HDL_3		5–10	1.125–1.210	57	43		
Preβ-HDL[3]		< 5	> 1.210				A-I
Albumin/free fatty acids	Adipose tissue		> 1.281	99	1	Free fatty acids	

TABLE 356-3 Fredrickson Classification of Hyperlipoproteinemias

Phenotype	I	IIa	IIb	III	IV	V
Lipoprotein, elevated	Chylomicrons	LDL	LDL and VLDL	Chylomicron and VLDL remnants	VLDL	Chylomicrons and VLDL
Triglycerides	↑↑↑	N	↑	↑↑	↑↑	↑↑↑
Cholesterol (total)	↑	↑↑↑	↑↑	↑↑	N/↑	↑↑
LDL-cholesterol	↓	↑↑↑	↑↑	↓	↓	↓
HDL-cholesterol	↓↓↓	N/↓	↓	N	↓↓	↓↓↓
Plasma appearance	Lactescent	Clear	Clear	Turbid	Turbid	Lactescent
Xanthomas	Eruptive	Tendon, tuberous	None	Palmar, tuberoeruptive	None	Eruptive
Pancreatitis	+++	0	0	0	0	+++
Coronary atherosclerosis	0	+++	+++	+++	+/–	+/–
Peripheral atherosclerosis	0	+	+	++	+/–	+/–
Molecular defects	LPL and ApoC-II	LDL receptor, ApoB-100, PCSK9, LDLRAP, ABCG5 and ABCG8		ApoE	ApoA-V	ApoA-V and GPIHBP1
Genetic nomenclature	FCS	FH, FDB, ADH, ARH, sitosterolemia	FCHL	FDBL	FHTG	FHTG

Abbreviations: ADH, autosomal dominant hypercholesterolemia; Apo, apolipoprotein; ARH, autosomal recessive hypercholesterolemia; FCHL, familial combined hyperlipidemia; FCS, familial chylomicronemia syndrome; FDB, familial defective ApoB; FDBL, familial dysbetalipoproteinemia; FH, familial hypercholesterolemia; FHTG, familial hypertriglyceridemia; LPL, lipoprotein lipase; LDLRAP, LDL receptor associated protein; GPIHBP1, glycosylphosphatidylinositol-anchored high density lipoprotein binding protein1; N, normal

Fatty acid metabolism

Fatty acid synthesis

This takes place in cytoplasm. Acetyl Co enzyme A is primer for synthesis of palmitic acid. Propionyl CoA is primer for synthesis of long chain fatty acids having odd number of carbon atoms.

First and rate limiting step is the formation of acetyl CoA by acetyl CoA carboxylase by fixing CO2.

Fatty acid elongation occurs both in mitochondria and microsomal system.

O2 required for microsomal pathway.

Formed fatty acids released free in circulation or in protein bound form.

Fatty acids after activated by coenzyme A is being utilized in following biosynthesis

a) Citric acid cycle via citrate synthase

b) Cholesterol synthesis via HMG CoA synthase.

c) Triglyceride biosynthesis.

Fatty acid oxidation occurs via Beta oxidation in either mitochondria or peroxisomes.

Mitochondrial oxidation is for long and short chain fatty acids and result in ATP production.

Peroxisomal oxidation is for very long chain fatty acids and is to shorten long FA's and remove side chain of cholesterol. No ATP is produced here.

OBESITY / DIABETES / HYPERTENSION INTERRELATION

Obesity mainly the central fat mass acts as main contributor for insulin resistance and future diabetes development.

Diabetes and hypertension are closely linked in such a way many factors acts as common for diabetes and hypertension.

a) **Platelet dysfunction**

Platelet dysfunction occurs due to end glycation product's impact on the platelet function and adherence properties which accounts for early atherogenesis and development of hypertension.

Abnormalities of Platelet Function in Diabetes Mellitus and Hypertension	
1.	Increased platelet adhesiveness
2.	Increased platelet aggregation
3.	Decreased platelet survival
4.	Increased platelet generation of vasoconstrictor prostanoids
5.	Reduced platelet generation of prostacyclin and other vasodilator prostanoids
6.	Altered platelet divalent cation homeostasis (ie, decreased $[Mg^{2+}]_i$ and increased $[Ca^{2+}]_i$)
7.	Increased nonenzymatic glycosylation of platelet proteins
8.	Decreased platelet polyphosphoinositide content

b) **Altered lipoprotein metabolism** in diabetes accounting for hypertension can occur due to following abnormalities in lipoprotein metabolism and include

Coagulation and Lipoprotein Abnormalities Seen in Patients With Hypertension and Diabetes Mellitus	
1.	Elevated plasma levels of VLDL, LDL, and lipoprotein(a)
2.	Decreased plasma HDL cholesterol
3.	Elevated plasma triglyceride levels
4.	Increased lipoprotein oxidation
5.	Increased lipoprotein glycation
6.	Increased small, dense LDL cholesterol products
7.	Decreased lipoprotein lipase activity
8.	Increased fibrinogen and PAI-1
9.	Decreased fibrinolytic activity
10.	Decreased angiotensin III, protein C and S levels

c) **Endothelial dysfunction** - various anatomic and functional abnormalities of the vascular endothelium are associated with both diabetes mellitus and hypertension. In patients with insulin resistance, endothelial lipoprotein lipase activity is decreased, so conversion of VLDL to LDL is reduced ant high chances of atherosclerosis is there as VLDL has got rich content of cholesterol which is injurious to endothelial cells. Hyperglycemia appears to contribute to endothelial dysfunction as well.Hyperglycemia promote protein kinase C in endothelial cells,which causes increased production of vasoconstrictor prostaglandins, endothelia and ACE. Increased PDGF and VGF appears, which enhances vasomotor constriction and vascular remodeling and growth. Also hyperglycemia alters endothelial cell matrix production, and basement membrane thickening. There is decreased endothelial nitric oxide production and effect. Increased expression of endothelial thrombomodulin.

Alterations in Vascular Endothelium Associated With Diabetes Mellitus and Hypertension	
1.	Elevated plasma levels of von Willebrand factor
2.	Elevated expression, synthesis, and plasma levels of endothelin-1
3.	Diminished prostacyclin release
4.	Decreased release of endothelium-derived relaxing factor (NO) and reduced responsiveness to NO
5.	Impaired fibrinolytic activity
6.	Increased endothelial cell procoagulant activity
7.	Increased endothelial cell surface thrombomodulin
8.	Impaired plasmin degradation of glycosylated fibrin
9.	Increased levels of advanced glycosylated end products

d) **Ill effects of advanced glycation end products** includes those directly and some indirectly related to AGE(advanced glycation end products)

Chronic hyperglycemia with advance glycation product at high concentrations has a direct toxic effect on vascular endothelial cells which may lead to decreased endothelium mediated vascular relaxation, increased vasoconstriction, promotion of vascular smooth muscle cell hyperplasia, vascular remodeling, and atherosclerotic events. High glucose proven to induce the overexpression of fibronectin and collagen IV in cultured human vascular endothelial cell leading to endothelial cell dysfunction

and it causes the glomerular basement membrane to thicken and meningeal hyperplasia.

Advanced glycation end products can do following detrimental effects as shown in below table

1.	Glycosylation of collagen occurs, increasing rigidity.
2.	Glycosylation increases DNA synthesis and proliferation.
3.	AGE protein receptor is identifiable on macrophages.
4.	AGE proteins enhance macrophage secretion of TNF-α and interleukin-1.
5.	Macrophage receptor for AGE proteins is upregulated by TNF-α in an autocrine manner.
6.	Enhanced free radicals are generated (early glycosylation products primarily responsible).
7.	AGE proteins are chemotactic for monocytes.
8.	AGE proteins enhance PDGF secretion.
9.	AGE proteins increase endothelial cell procoagulant activity and suppress endothelial cell surface thrombomodulin.
10.	AGE proteins increase endothelial cell transcytosis and monolayer permeability.
11.	Glycosylated collagen exhibits increased protein binding, including LDL, and increases platelet aggregation.
12.	Nonenzymatically glycosylated LDL enhances macrophage cholesterol ester synthesis and reduces degradation.

Homocysteine metabolism and its role in coronary artery disease:

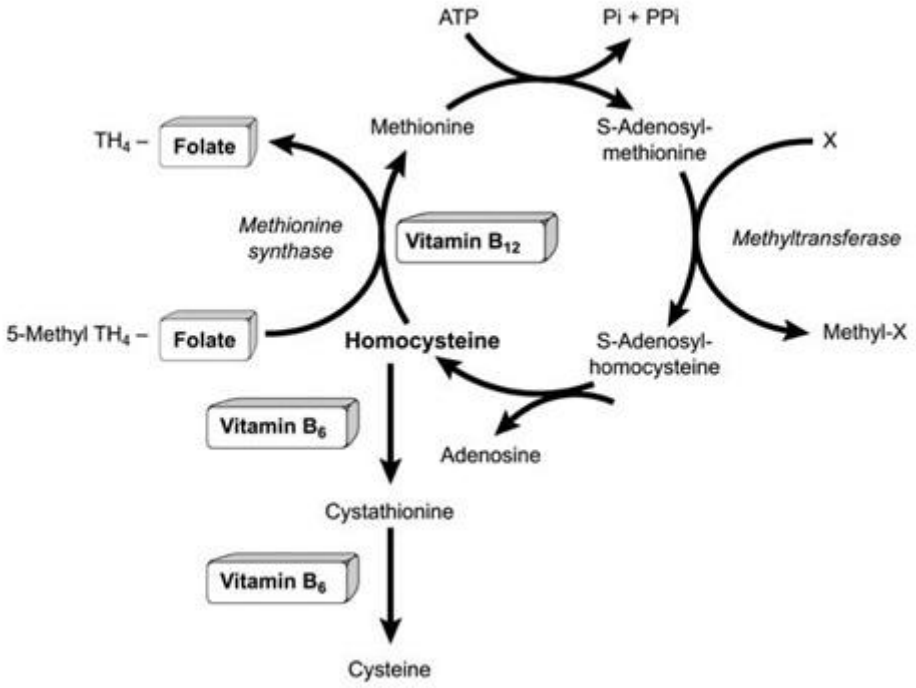
Homocysteine is a non-essential sulfur containing amminoacid, which has got its role in intermediate of two main pathways:

1) Remethylation to methionine, which requires folate and vitamin B12
2) Transsulfuration to cystathionine, which requires pyridoxal-5′-phosphate.

Both pathways are coordinated by *S*-adenosylmethionine, which is as an allosteric inhibitor of the methylenetetrahydrofolate reductase reaction and as an activator of cystathionine β-synthase.

Hyperhomocysteinemia is recently been studied extensively associated with increased risk of cardio-vascular disease. Severe hyperhomocysteinemia is due to rare genetic disorders

with defectsin enzymes namely cystathionine beta synthase, methylenetetrahydrofolate reductase, or in enzymes involved in methyl-B_{12} synthesis and homocysteine methylation



Conversion of homocysteine to methionine requires methionine synthase enzyme with vitamin b12 as cofactor, which parallels the conversion of methylene tetrahydrofolate to tetrahydrofolate.

Normal values

MALE = 5.46-16.2 umol/lit

FEMALE = 4.44-13.56 umol/lit

Any conditions which results in B12 deficiency like those of atrophic gastritis ,post gastrectomy, intrinsic factor deficiency , chronic alcoholics lead on to hyperhomocysteinemia and thus accelerated atherosclerosis including coronary arteries and early onset myocardial ischemia and infarct sets in even without other risk factors.

Hyperhomocysteinemia leading to CVD risk is explained in many hypothetical methods but none proved. The proposed mechanisms by which hyperhomocysteinemia leads to cvd and overall atherogenesis includes direct endothelial injury , endothelial dysfunction , increase in intima medial thickness, increased activity of procoagulants and immune dysfunction been proposed.

The addition of methyl group to cytosine in genome is essential to decide gene product to be active or not. Defect in this process leads to inactive gene products and thus functional capacity of endothelium is lost along with its protective mechanisms. This is accounted for increase CVD risks and also stroke events in high incidence.

Vitamin B12 deficiency

It can result in hyperhomocysteinemia. The reasons behind b12 deficiency could be due to

A) Pernicious anemia – an autoimmune disease where autoantibodies are directed against one's own bowel cells and there is destruction of those gastric mucosal cells that produces intrinsic factor.

B) Short bowel syndrome – any operational or disease process which leads to small bowel loops especially the distal segments to shorten will leads to low b12 absorption from GIT.

C) Chron's disease – which makes small bowel mucosal dysfunction.

D) Vegetarian diet – which is defective in b12 levels.

E) Chronic alcoholics

Endothelial dysfunction as a marker of future coronary artery disease

Endothelium acts in many ways to influence the effect of the

Endothelium function in a paracrine manner to adjust in case of shear stress situations like increase systolic pressure causing stretch of vascularity and

endothelium tend to get damaged and this being prevented by intrinsic capacity of vascular endothelial cells to secrete vasodilators like NO (nitric oxide) and prostanoids.

NO is produced by potassium activated calcium channels which acts during periods of stress force, so that when potassium channel opens up calcium entry occurs and that causes activating of endothelial nitric oxide synthase – eNO's.

TheseeNO's causes vasodilatation and thus accommodates the stress force and prevents the endothelial damage and thrombus formation.

Nitric oxide is the vasodilator substance being internally synthesized in endothelium as a paracrine hormonal response in case of localized vasoconstriction events. It will be generated in response to hypoxia and produced by endothelial nitricoxide synthase which can be inducible or

Myocardial ischemia / infarct evaluation and treatment

Any patient presenting with retrosternal chest pain is considered as acute coronary syndrome until proved otherwise. Initially patients are subjected to electrocardiogram and if no finding consistent with acute coronary event in ECG, we should not presume the patient is having non-cardiac chest pain. If patient has typical constant compressive or unexplainable chest pain, he should to subjected to echocardiography after initial hemodynamic evaluation and ECG. Patient should be started on antiplatelet therapy unless it is contraindicated. Hemodynamic stabilization should be the prime goal of treatment of acute chest pain patient. Serum marker of myocardial injury should be sent. Additional diuretic and antihypertensive treatment depends on the presentation. Statin therapy is nowadays is must that too in very high doses of 80 mg atorvastatin in initial therapy to reduce acute inflammation and should continued in low dose to maintain in isolipidemic state.

Here is a simple algorithm to explain the initial evaluation of any patient with cardiac pain

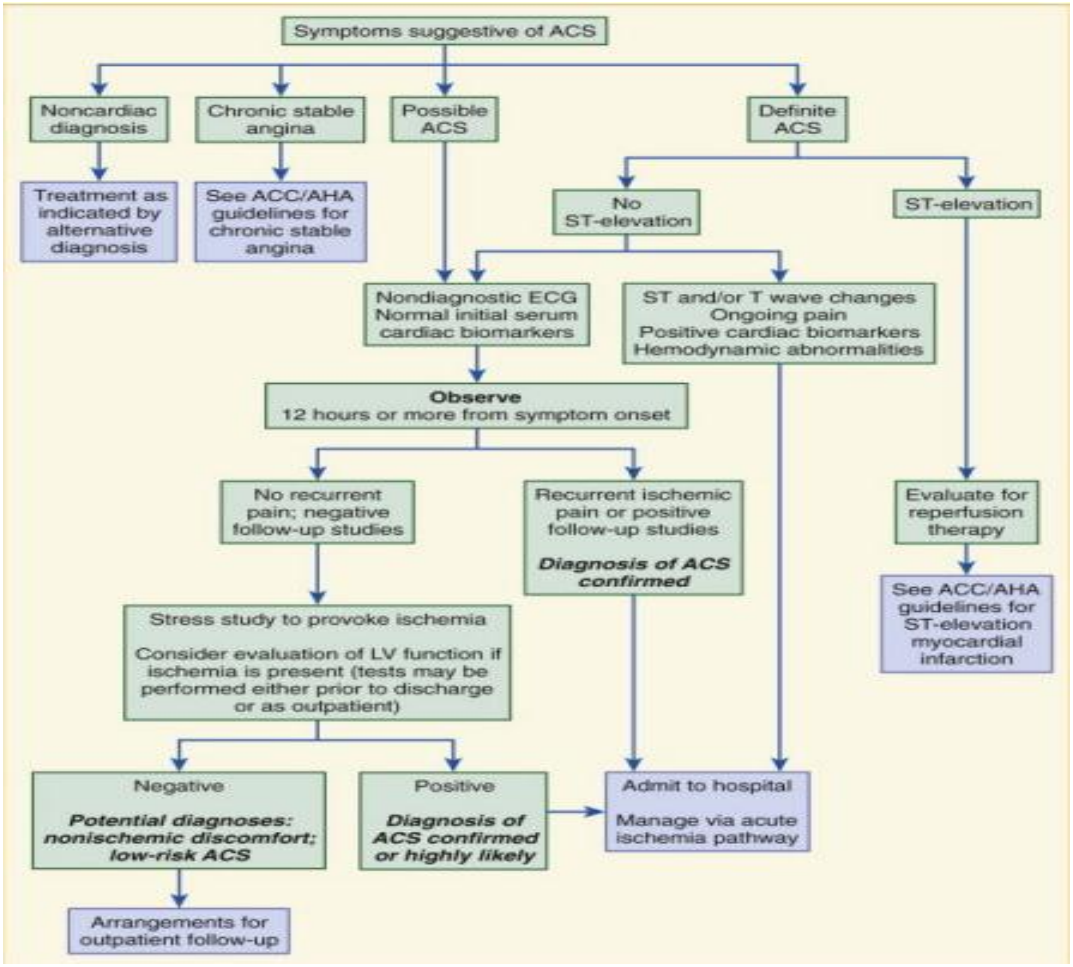
It mainly focuses on to first exclude myocardial infarction (MI)

It is not common to encounter with normal ECG, echo but those with inducible ischemia can be picked up with treadmill test.

Even with pharmacological test, if a person comes with normal result, we should subject the patient to angiography if the symptoms recurs or persists and rule out early significant coronary stenosis and continue the patient on antiplatelet therapy if needed.

ACC/AHA guideline algorithm for acute coronary syndrome

This guide line highlights the importance of stress test to provoke ischemia changes in a patient with normal resting ECG with no significant findings like ST, T changes or new bundle branch block.Assess LV function along with the stress test or after the stress study to assess future risks.



Initial evaluation in the emergency department focuses on identification of STEMI, early therapy, and reperfusion strategy. Selection of reperfusion strategy depends on hospital and patient characteristics, time to reperfusion therapy strongly determines outcomes in STEMI. Patients presenting within 90 minutes to a hospital with percutaneous coronary intervention (PCI) capability should undergo PCI. Those with STEMI presenting to a hospital

not capable of doing PCI should be transferred to a PCI capable hospital based on patient characteristics, time from onset of symptoms, and time to reachhospital with PCI therapy.

Those STEMI patients presenting to a hospital without PCI capability, who cannot be transferred to a center capable of doing PCI and where PCI can't be done within 90 minutes of first medical contact should be offered fibrinolysis therapy in 30 minutes of hospitalization provided no contraindications exists for thrombolysis for that particular patient.

The management of patients with hypotension, pulmonary edema, arrhythmias, or shock depends on the most likely underlying disorder. LV function and the presence of any aneurysm, septal rupture should be assessed by echocardiograph.

For those with low blood pressure treatment should include inotropic support if it's due to right ventricular MI. suppose right ventricular MI is present, intravenous fluid rushing should be the first line of treatment and

later to decide on thrombolytic therapy. Other treatment options to save coronary perfusion includes intraaorticbaloon counter pulsation, mechanical reperfusion with percutaneous coronary intervention and once stabilized to undergo coronary artery bypass graft (CABG).

Any aneurysm or septal infarct with communication should subjected to surgery immediately.

Beta blockers or calcium channel blockers should not be administered in low cardiac output states.

Contraindications for thrombolysis includesthose

With intracranial hemorrhage

AV malformations

Ischemic stroke within 3 months

Any Active bleeding diathesis

Some are considered relative contraindications as listed below

Absolute Contraindications

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 mo *except* acute ischemic stroke within 3 hr
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 mo

Relative Contraindications

- History of chronic severe poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP >180 mm Hg or DBP >110 mm Hg)[†]
- History of prior ischemic stroke > 3 mo, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Recent (within 2-4 wk) internal bleeding
- Noncompressible vascular punctures
- For streptokinase, anistreplase: Prior exposure (>5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

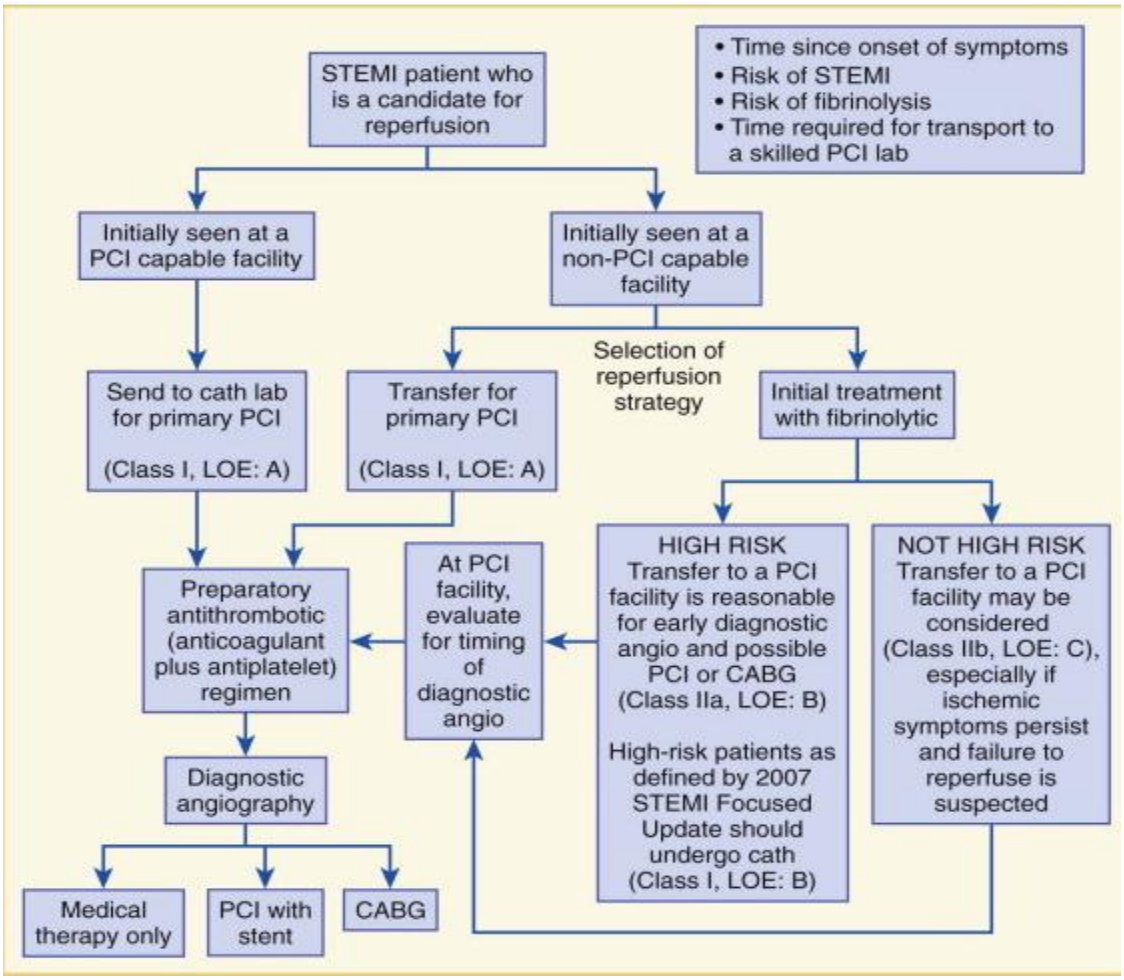
Decision to select patient fit for reperfusion therapy should be made in quick succession so that any patient who is candidate to undergo percutaneous intervention should not fail to get treatment.

Keep ready with thrombolytic agent and proceed with angiogram and depending on the circumstances do medical intervention , PCI with stent insertion or if situations persists do primary CABG (coronary artery bypass graft) using the suitable vessel. Most commonly used one is left internal thoracic artery that is anastomosed to the left anterior descending artery.

Other vessels used includes right internal thoracic artery, great saphenous vein and radial artery if necessary.

The reason left internal thoracic artery preferred is due to its long patency and easy end to end anastomosis so that long duration of surgery also be minimized and ability to withstand shear force. Great saphenous vein has got poor patency but got an advantage multiple level harvesting possibility in that vein.

Algorithym for treatmet of a patient diagnosed with STEMI.



Methods to evaluate coronary stenosis

Coronary stenosis can be measured by means of both invasive and non-

Invasive Methods.

Invasive methods include

Invasive angiogram.

Intravascular ultra sonogram.

Non-invasive methods include

MRI angiogram - which also gives myocardial contractive characteristics and ejection fraction.

Single photon computed tomography

128 slice MDCT -Multislice computed tomography – allows calcium scoring also.

PET scan - which is expensive.

Thallium201 myocardial imaging with rest exercise assessment of left ventricular function using radionuclide methods.

CT ANGIOGRAM

Apart from providing information regarding calcification, this also gives angiogram of coronary arterial tree.

This gives the total calcium score. Provides information only for established or chronic stable angina and not in cases low risk cardiac pain patients. The benefit should weighted against risk of radiation exposure. When combined with PET scan termed PET-CT, myocardial perfusion and viability.

Cardiac MRI (CMR)

This helps to additional imaging of aorta and major vessels status. When this combined with the pharmacological stress test, appears best comparable to SPECT. Provides information on anatomy of coronaries, LV function, blood flow, viability of myocardium.

Stress Nuclear Myocardial Perfusion Imaging–

Exercise perfusion imaging with concomitant ECG analysis is better than exercise ECG alonefor identifying CAD, vessel localization, and assessing extend of myocardial ischemic and infarcted regions. Exercise single-photon emission computed tomography (SPECT) yields an average sensitivity and specificity of 89% and 71%, compared with 67% sensitivity and 76% specificity for exercise ECG alone.Higher specificity of around 90% obtained after avoiding referring bias. Also helps in identifying viable tissue and assessing prognosis.

Pharmacologic Nuclear Stress Testing

For those unable to exercise with effort, in older patients and those with PVD, COPD, arthritis, obesity individual, pharmacologic vasodilator stress with dipyridamole or adenosine derivatives is useful. This form of imaging is very much comparable to exercise perfusion imaging. But still treadmill testing is preferred for patients who can able to do exercising as exercise provides additional diagnostic and prognostic information, including ST-segment changes, effort tolerance and symptomatic response, and HR and blood pressure monitoring.

Vasodilator stress agents are also used with positron emission tomography to diagnose CAD and assess its severity.

Stress Echocardiography

2D echocardiography is useful to assess the chronic CAD patients as it can assess focal and regional LV function during normal and in ischemic conditions and also to assess the left ventricular hypertrophy and also cardiac valvular disease. Stress echocardiography may be done by usingboth exercise andpharmacologic agent's helps us to identify the areas with regional wall motion abnormalities. Best images and diagnosis can be made in around 84% of patients. The test is a reproducible oneseveral studies have proved that stress echocardiography is superior to exercise ECG and stress cardiac imaging alone. This is also best in picking up areas of ischemic myocardium and localizing it. This has got good prognostic assessment capability. Pharmacologic stress ECHO using dobutamine should reservedfor patients unable to exercise, those unable to get required heart rate with exercise, and those who got poor images after exercise.

Stress echocardiography is best avoided in cases of patient having features of COPD.

Patient should not smoke, eat or drink water four hours prior to the procedure. The procedure will take around half hour to one hour depending on patient's cooperation and need to repeat in cases of errors.

Drug induced stress echocardiography also takes around one hour for the procedure to complete.

Nuclear Stress Test

A Nuclear Stress Test is similar to a standard exercise treadmill stress test, using an isotope Cardiolite or Thallium to evaluate coronary blood flow. Also provides information on recoverable status of heart myocytes recovers after exercise, and arrhythmia.

High Risk (>3% Annual Mortality Rate)

1. Severe resting left ventricular dysfunction (LVEF < 0.35)
2. High-risk treadmill score (score ≤ −11)
3. Severe exercise left ventricular dysfunction (exercise LVEF < 0.35)
4. Stress-induced large perfusion defect (particularly if anterior)
5. Stress-induced multiple perfusion defects of moderate size
6. Large, fixed perfusion defect with LV dilation or increased lung uptake (thallium-201)
7. Stress-induced moderate perfusion defect with LV dilation or increased lung uptake (thallium-201)
8. Echocardiographic wall motion abnormality (involving more than two segments) developing at low dose of dobutamine (≤10 ?g/kg/min) or at low heart rate (<120 beats/min)
9. Stress echocardiographic evidence of extensive ischemia

Intermediate Risk (1%-3% Annual Mortality Rate)

1. Mild or moderate resting LV dysfunction (LVEF = 0.35-0.49)
2. Intermediate-risk treadmill score (−11 < score < 5)
3. Stress-induced moderate perfusion defect without LV dilation or increased lung intake (thallium-201)
4. Limited stress echocardiographic ischemia with a wall motion abnormality only at higher doses of dobutamine involving two segments or less

Low Risk (<1% Annual Mortality Rate)

1. Low-risk treadmill score (score ≥ 5)
2. Normal or small myocardial perfusion defect at rest or with stress*
3. Normal stress echocardiographic wall motion or no change of limited resting wall motion abnormalities during stress*

Cardiac catheterization and angiography:

Definitive diagnosis of CAD and precise assessment of its anatomic severity can be made by using catheterization and angiography only. The catheter can be accessed via radial or femoral artery and reached till the ascending aorta until cusps under the guidance of CT angiogram, where radioactive

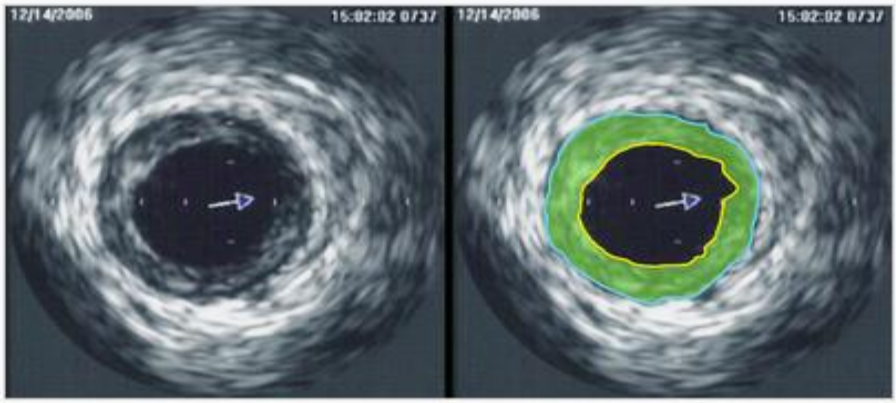
dye is being injection after the catheter tip is inserted into origin of left coronary sinus and left coronary anatomy is first visualized guiding with dye injection into the left main coronary artery and after visualizing the left coronary anatomy with clear delineation in multiple views so as to visualize till the distal branches, the catheter is turned around and further inserted into the right coronary cusp and entered the right coronary sinus and dye is being injected into the right coronary artery with parallel view of the anatomy of right coronary system with the guidance of computed tomography.

In patients with chronic stable angina, around 25% each have single, to triple vessel disease in coronary angiogram.

INTRAVASCULAR ULTRSONOGRAPHY

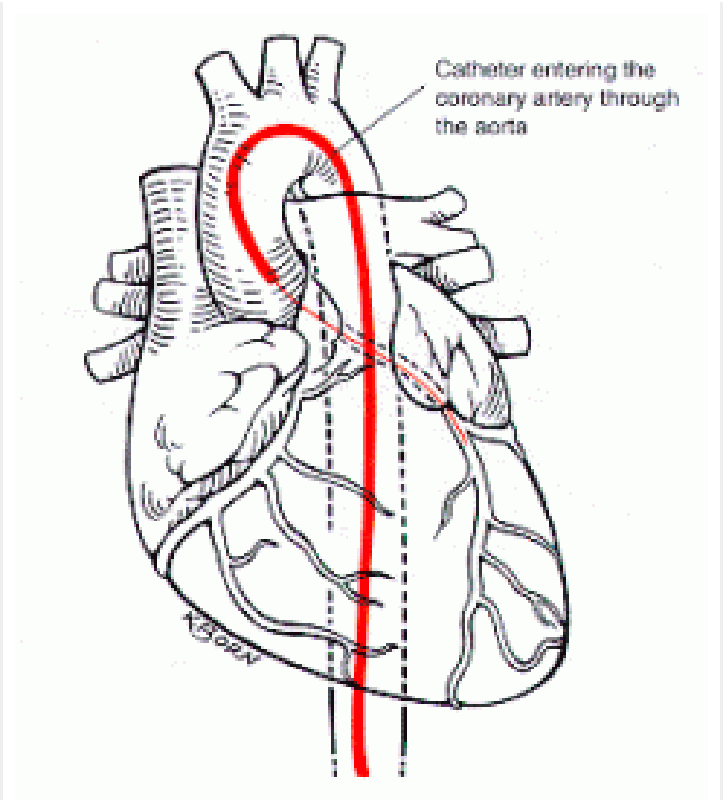
This one done with a mini ultrasound probe attached at the tip of the catheter and images is viewed in a monitor connected outside.it helps to visualize not only the plaque inside the lumen but also the hidden plaques within the vascular wall.

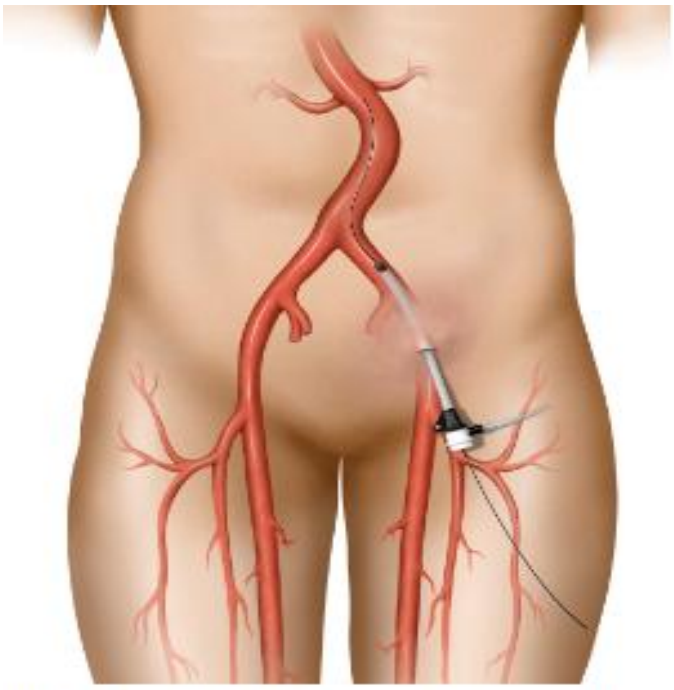
The Advanced invasive imaging like as intravascular ultrasonography provide a cross-sectional view of the coronary artery. This provides enhanced view and helps quantify coronary atherosclerosis, as identify those vessels got chance for future coronary atheroma. Studies incorporating both coronary angiography and IVUS have proved that the severity of coronary artery disease may be underestimated by angiography alone.



Intravascular ultrasound image of a coronary artery (left), with color-coding on the right, delineating the lumen (yellow), external elastic membrane (blue) and the atherosclerotic plaque burden (green). The percentage stenosis is defined as the area of the lumen (yellow) divided by the area of the external elastic membrane (blue) times 100. As the plaque burden increases, the lumen size will decrease and the degree of stenosis will increase.

Intravascular optical coherence tomography, angioscopy, and thermography are newer techniques and will be made routine in future for complete characterization of coronary atherosclerosis.



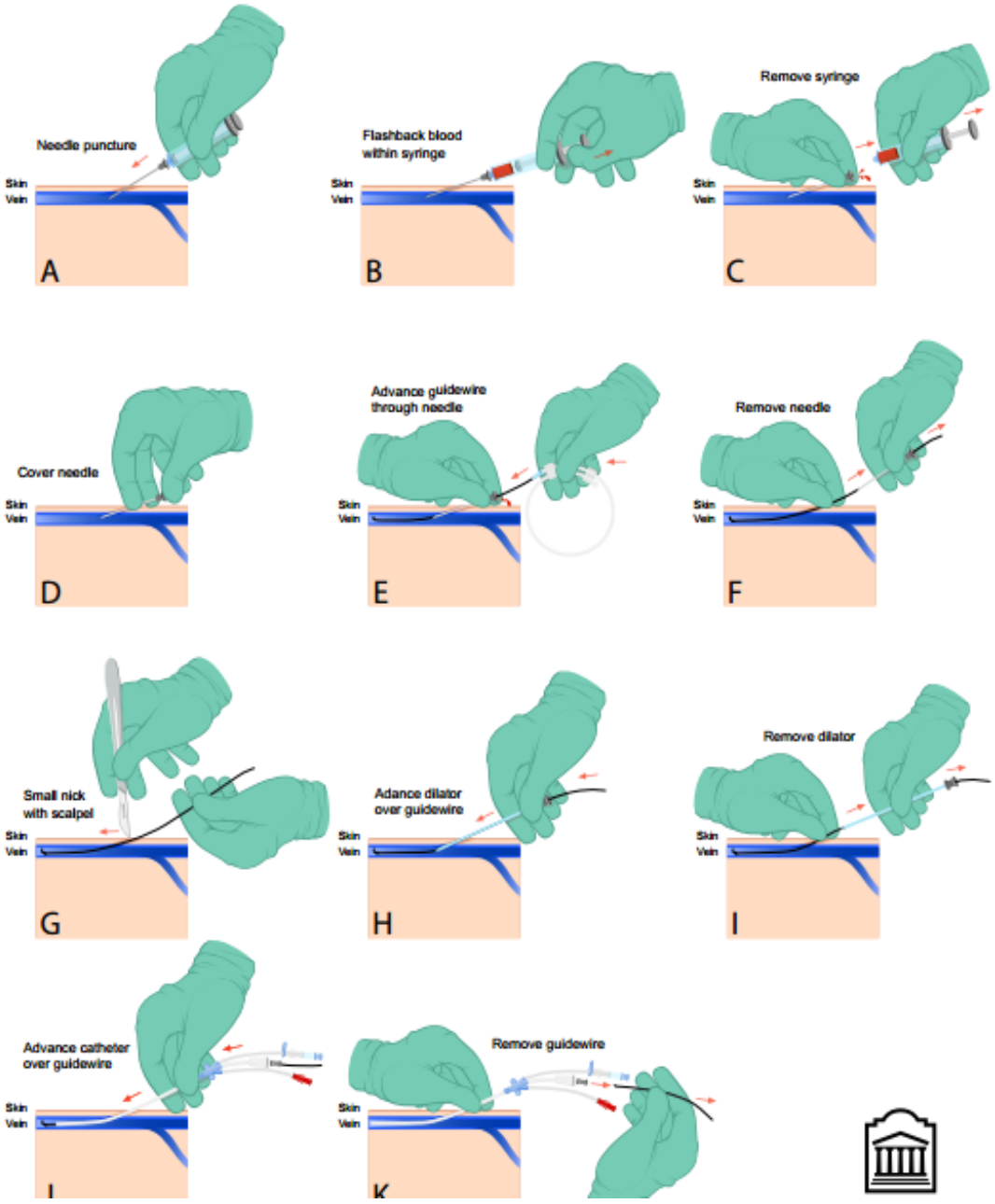


Introducer Sheath during Catheterization

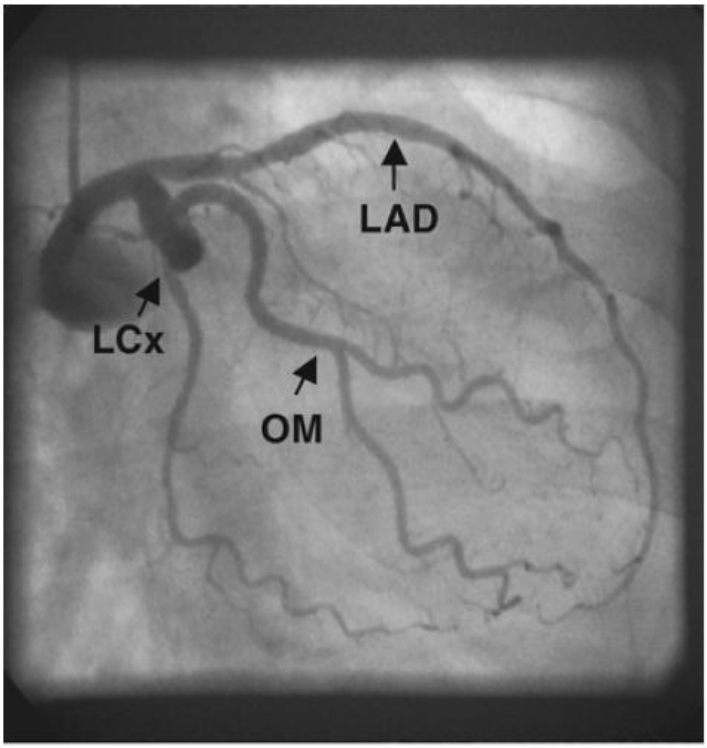
Cardiac catheter insertion

Cardiac catheterization using seldinger technique

MODIFIED SELDINGER TECHNIQUE



Left coronary artery in coronary angiogram



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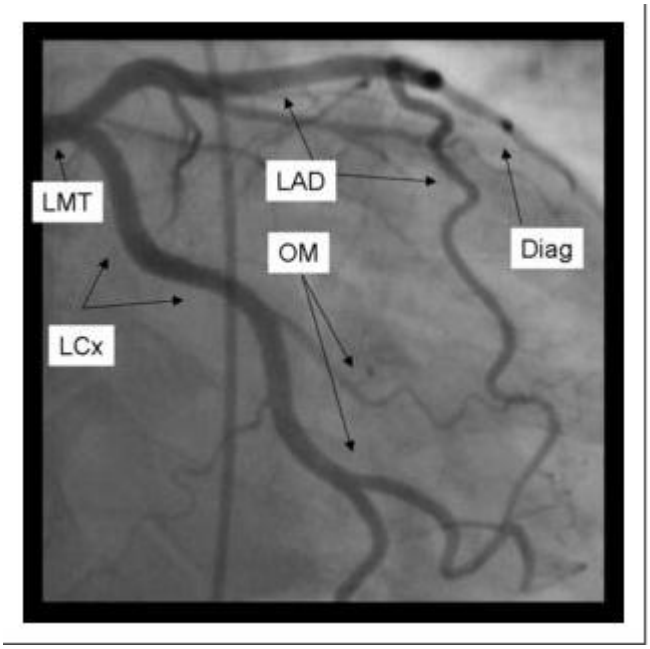
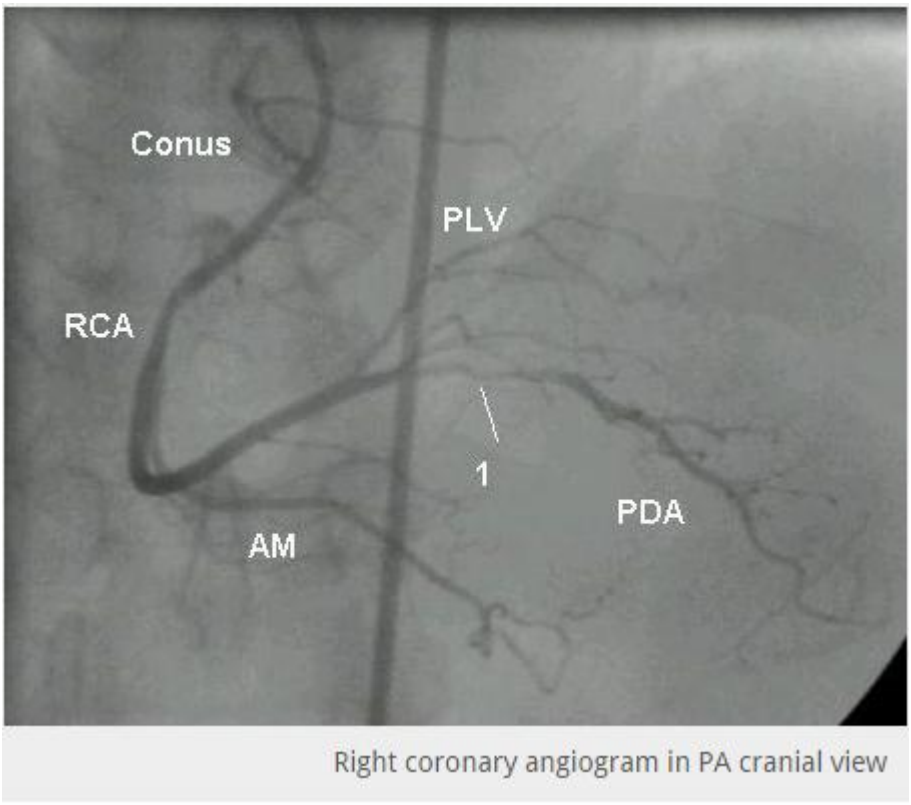


Right coronary artery in coronary angiogram



Right coronary angiogram in PA cranial view

Right coronary angiogram in PA cranial view showing lesion in proximal posterior descending artery.

Modified gensini scoring

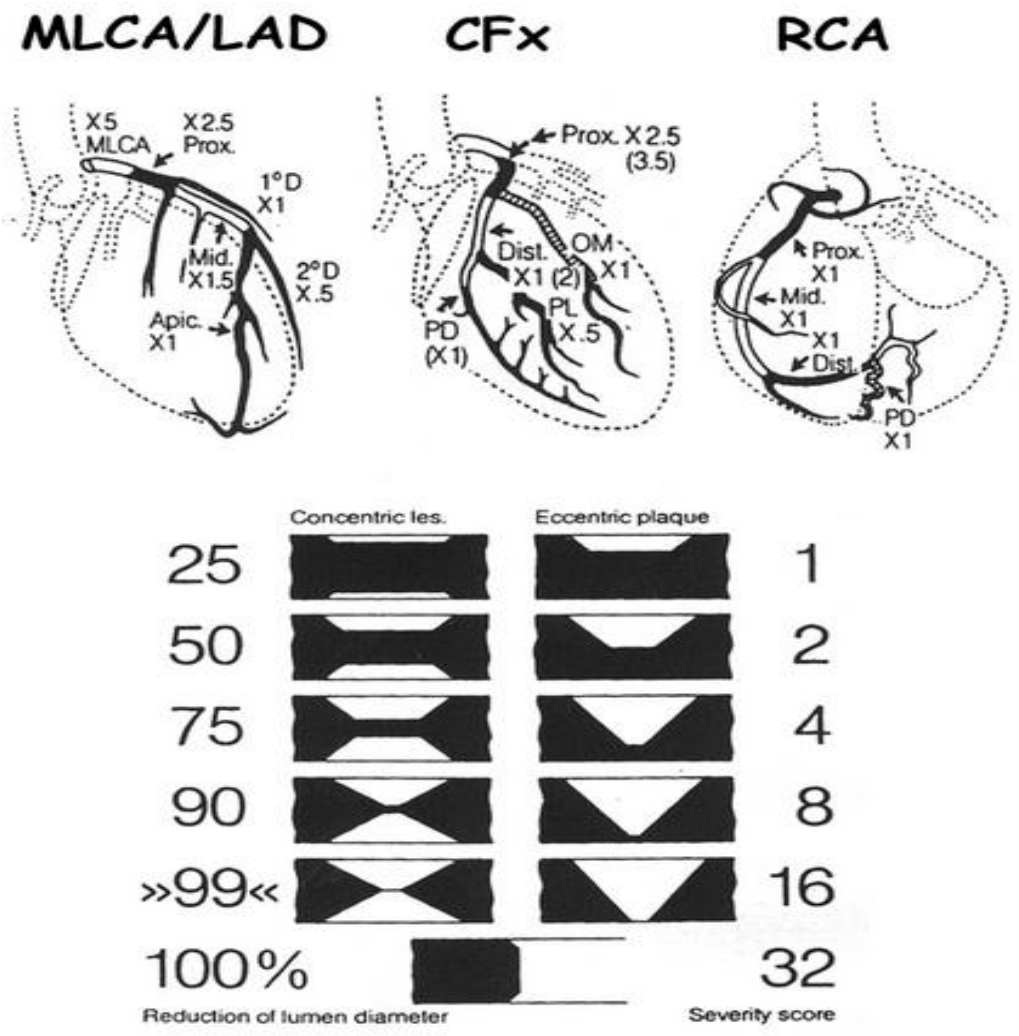
The severity of coronary stenosis is assessed by using modified gensini scoring.

This involves assigning fixed scoring based on percentage of compression of the lumen in coronaries and then multiplying it with the fixed factor

depending on the location of stenosis. This also describes the extent of lesion.

The proximal portions of the coronaries caries more multiplying factor compared to the distal portions of coronaries.20% lumen narrowing carries a score of 1 and it gradually increased in terms of 50,75,90,99 and 100 percent stenosis with the score of 2, 4,8,16and 32 respectively.

This score concentrates not on the number of branches involved but the region of artery involved and the percentage of luminal occlusion which is more important than the number of vessels involved. This gives more importance to those vessel which supplies majority of myocardium and resultant percentage of myocardium being infarcted.



The multiplying factors is given as follows

Left main stem – 2.5 x

LAD proximal – 2.5 x

LAD mid – 1.5 x

LAD distal – 1 x

First degree diagonal – 1x & Second degree diagonal -0.5

Left circumflex proximal – 2.5x, distal – 1x

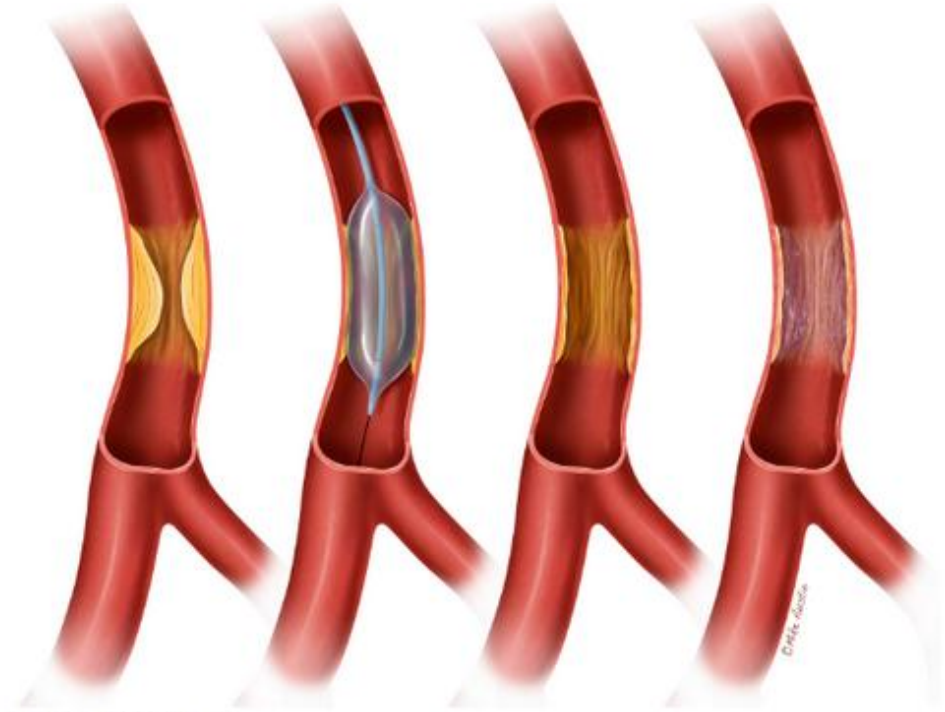
Left diagonal and marginal branch – 1x

Left poster lateral branch – 0.5x

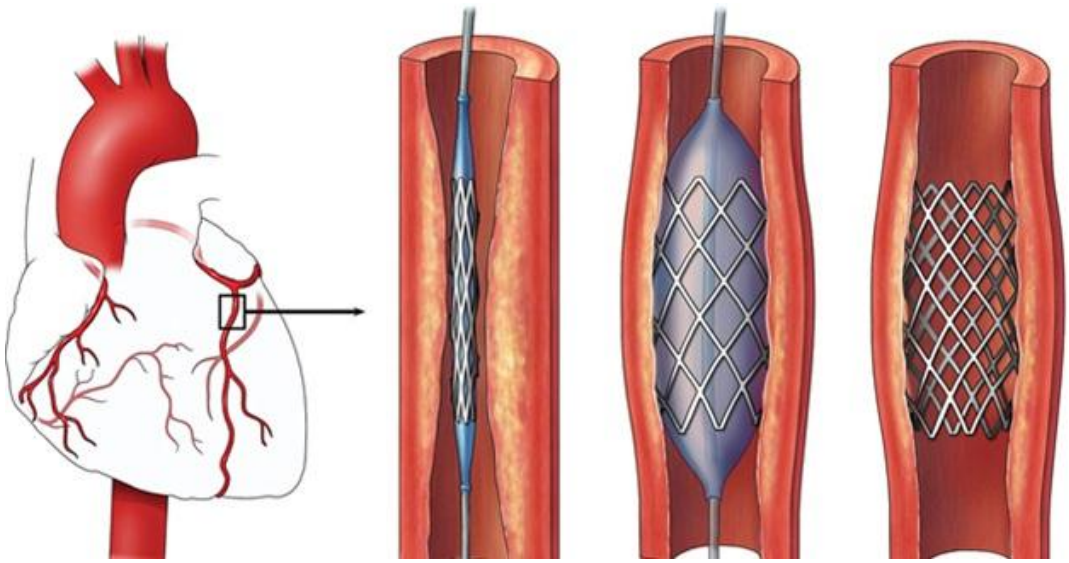
Right coronary artery proximal, mid and distal – 1x

Balloon angioplasty

This procedure involves cardiac catheterization as usual and balloon which is inflated after assessing the plaque size and the appropriate air inflation is done so as to compress the plaque and attain the desired lumen size post ballooning. The balloon in inflated for sixty to ninety seconds and repeated if required.



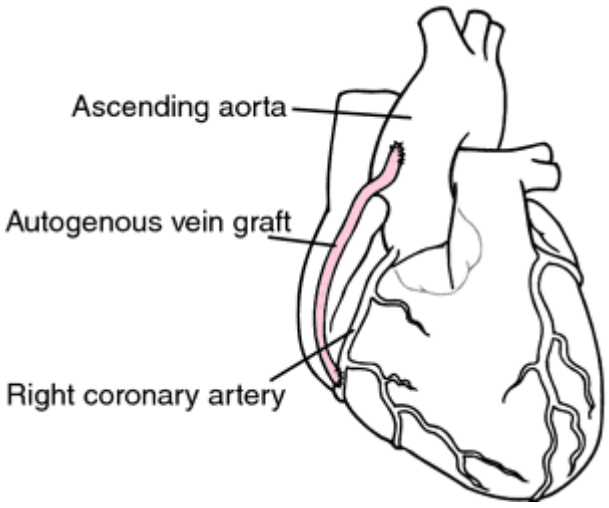
Balloon Angioplasty

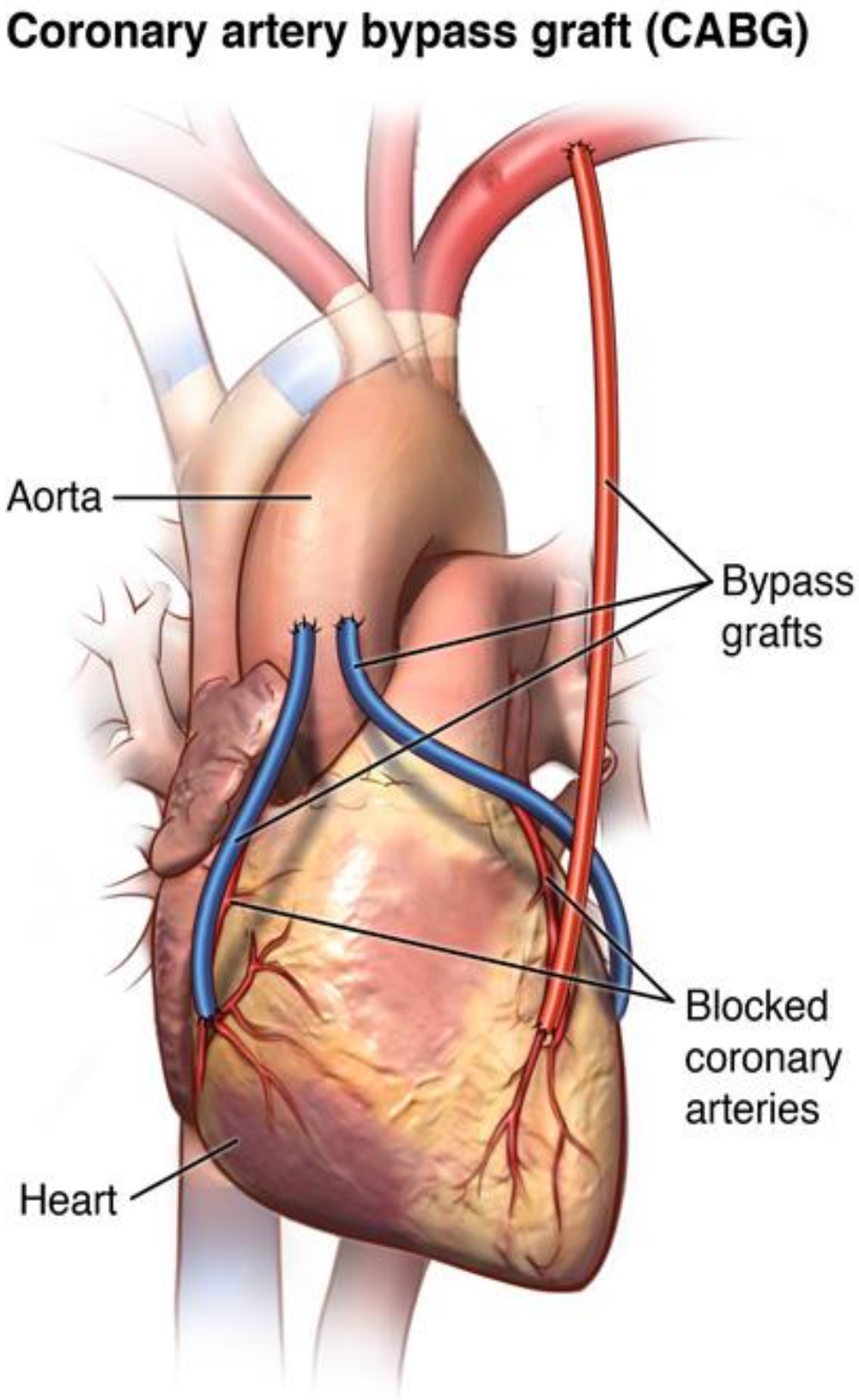


Coronary stent placement using balloon guidance

Coronary artery bypass graft

This is the last sort of treatment when either medication and balloon angioplasty fails to relieve disease or can be a part of primary treatment depending on the severity of lesion and persistence of symptoms.




Coronary artery bypass graft (CABG)
Aorta
Bypass grafts
Blocked coronary arteries
Heart

AIMS AND OBJECTIVES

1. TO STUDY CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE.

2. TO IDENTIFY STRONG ANTHROPOMETRIC PARAMETER CORRELATING WITH THE SEVERITY OF CORONARY ARTERY DISEASE USING CAG.

MATERIALS AND METHODS

PLACE OF STUDY:

HUTTON WARD, DEPARTMENT OFCARDIOLOGY, STANELY MEDICAL COLLEGE AND HOSPITAL, CHENNAI**.**

DURATION:

NOV 2013 TO SEP 2014

STUDY DESIGN

PROSPECTIVE AND OBSERVATIONAL STUDY

SOURCE OF DATA:

PATIENTS WITH HISTORY OF CHEST PAIN WHO FULFILL THE INCLUSION AND EXCLUSION CRITERIA, GETTING ADMITTED AT CARDIOLOGY HUTTON WARD, STANLEY MEDICAL COLLEGE, CHENNAI DURING THE PERIOD OF NOV 2013 TO SEPTEMBER 2014 AND PLANNED FOR CORONARY ANGIOGRAM.

SAMPLE SIZE:

75

INCLUSION CRITERIA:

1. ALL PATIENTS UNDER 50YRS OF AGE , ADMITTED IN CARDIOLOGY DEPARTMENT HUTTON WARD, STANLEY MEDICAL COLLEGE FOR UNDERGOING CORONARY ANGIOGRAM

EXCLUSION CRITERIA:

1. PATIENTS KNOWN TO HAVE RHEUMATIC HEART DISEASE, ISCHEMIC DCMP.
2. PATIENTS WHO ARE ABOVE THE AGE OF 50 YRS.
3. PATIENTS WHO ARE CHRONIC ALCOHOLICS.
4. PATIENTS WITH CHRONIC KIDNEY DISEASE.

5. PATIENTS WITH ASCITES AND OTHER INTRABDOMINAL PATHOLOGY.
6. PATIENTS WITH HB < 11GM%

METHODOLOGY

Patients admitted for angiography between November 2013 to September 2014 will be included in the study.

DATA COLLECTED USING A PROFOMA MEETING THE OBJECTIVES OF THE STUDY.

DETAILED HISTORY, PHYSICAL EXAMINATION AND NECESSARY INVESTIGATIONS WILL BE UNDERTAKEN.

They are subjected for anthropometry measurements including height, weight, BMI, hip and waist circumference, WHR, sagittal abdominal diameter, triceps skin fold thickness and SAD/TSFT ratio measured. Also their serum homocysteine level, glycemic status, Presence or absence of hypertension and diabetes, smoking and alcohol intake with duration, life style & physical activity, frequency of chest pain, ECG findings and echo status will be noted prior to angiogram.

Post angiogram evaluation of severity of coronary artery stenosis made my Modified Gensini Score.

THE PURPOSE OF THE STUDY WILL BE EXPLAINED TO THE PATIENT AND INFORMED CONSENT OBTAINED

THE ANALYSIS OF THE DATA WILL BE DONE USING APPROPRIATE STATISTICAL METHODS.

Results and discussion

The study includes total number of 75 patients. Data were collected and final analysis were made.

Study Groups

Study Groups	Name of Group	CAG Findings	Number of Subjects
Group A	Non critical CAD	Normal and Non obstructive (without a noncritical coronary Lesion)	21
Group B	Critical CAD	SVD, DVD, TVD (having at least one lesion more than 50% within the main branches of the coronary arteries)	54

Statistics

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with Unpaired t-test and categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010.

Sample Size Calculation

Sample size was determined on the basis of a pilot study in which the presence increased percentage of homocysteine levels was measured at 5%. We calculated a minimum sample size of 73 patients was required, assuming a type 1 error (two-tailed) of 0.05 and a margin of error of 10%. Therefore, the final sample selected was n=75.

$$\mathbf{n} = \frac{\mathbf{t^2} \times \mathbf{p(1-p)}}{\mathbf{m^2}}$$

Description:

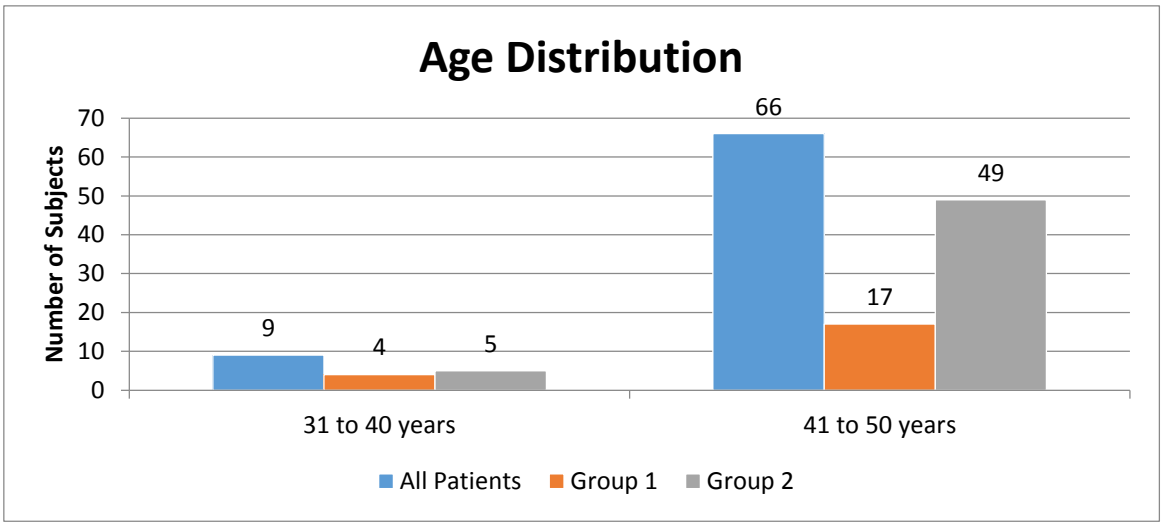
n = required sample size
t = confidence level at 95% (standard value of 1.96)
p = estimated prevalence of malnutrition in the project area
m = margin of error at 10% (standard value of 0.05)

$$\mathbf{n} = \frac{\mathbf{(1.96)^2} \times \mathbf{0.05(1-0.95)}}{\mathbf{(0.05)^2}}$$

$$\mathbf{n} = \frac{\mathbf{3.8146} \times \mathbf{0.0474}}{\mathbf{0.0025}}$$

$$\mathbf{= 73}$$

Age

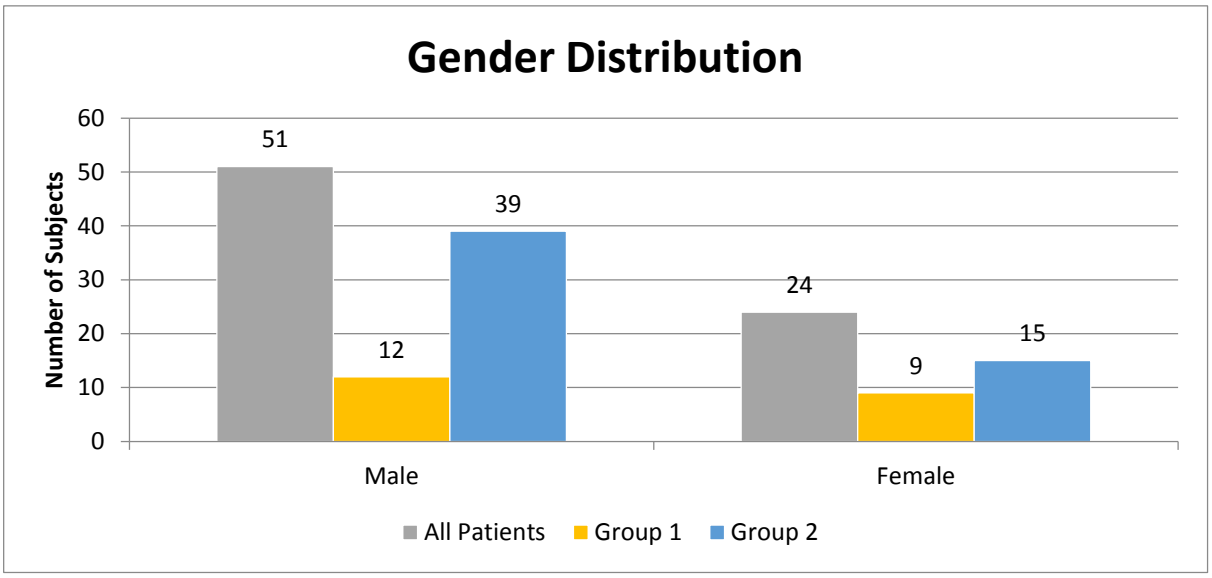


Age Distribution	All Patients	%	Group 1	%	Group 2	%
31 to 40 years	9	12.00	4	19.05	5	9.26
41 to 50 years	66	88.00	17	80.95	49	90.74
Total	75	100	21	100	54	100

Age Distribution	Group 1	Group 2
N	21	54
MEAN	44.62	46.07
SD	3.81	3.32
t test	0.134229994	

By conventional criteria the association between the study groups and age is considered to be not statistically significant since $p > 0.05$

Gender

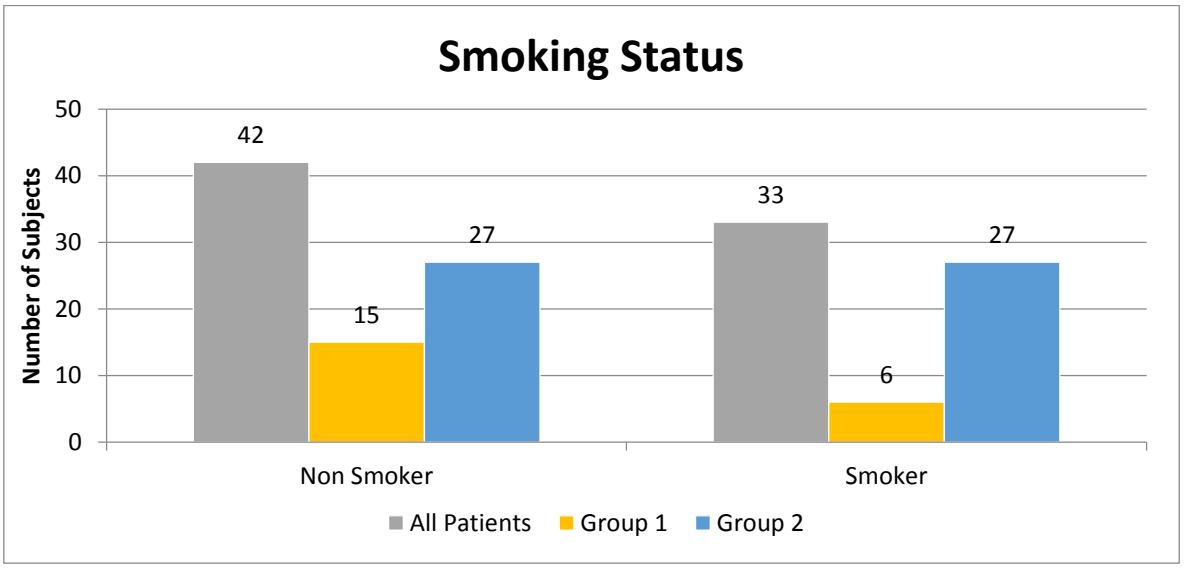


Gender Distribution	All Patients	%	Group 1	%	Group 2	%
Male	51	68.00	12	57.14	39	72.22
Female	24	32.00	9	42.86	15	27.78
Total	75	100	21	100	54	100
Chi-square value				1.58		
Degrees of freedom				1		
P value Chi Squared Test				0.209		

By conventional criteria the association between the study groups and gender is considered to be not statistically significant since $p > 0.05$.

Since age and gender is not statistically significant, it means that there is no difference between the groups. In other words the groups contain subjects with the same basic demographic characteristics.

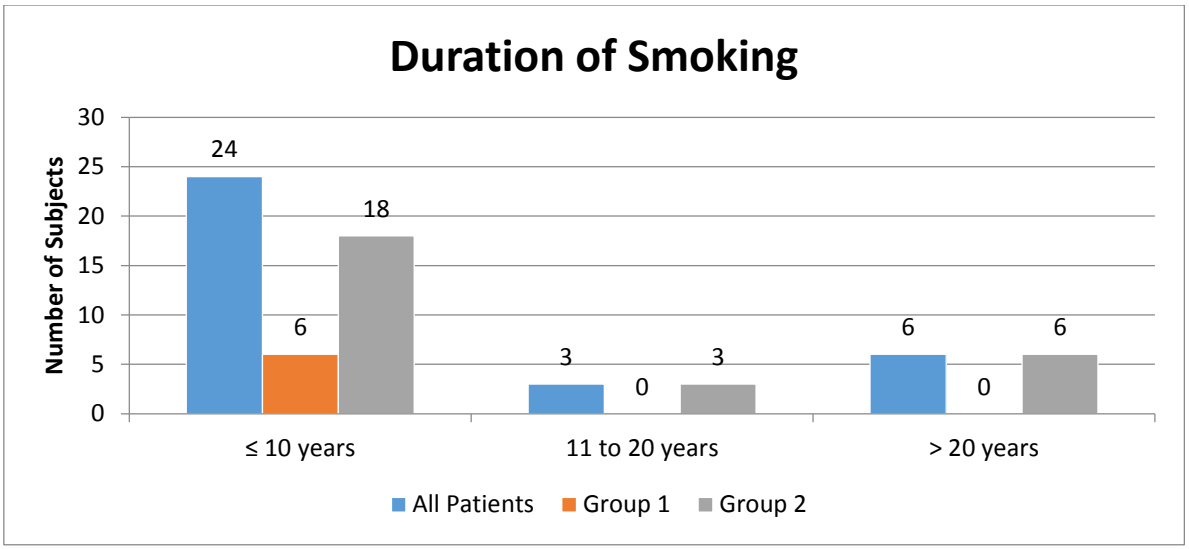
Smoking



Smoking Status	All Patients	%	Group 1	%	Group 2	%
Non Smoker	42	56.00	15	71.43	27	50.00
Smoker	33	44.00	6	28.57	27	50.00
Total	75	100	21	100	54	100
Chi-square value				2.82		
Degrees of freedom				1		
P value Chi Squared Test				0.093		

By conventional criteria the association between the study groups and smoking among study subjects is considered to be statistically not significant since $p > 0.05$.

Duration of Smoking



Duration of Smoking	All Patients	%	Group 1	%	Group 2	%
≤ 10 years	24	72.73	6	100.00	18	66.67
11 to 20 years	3	9.09	0	0.00	3	11.11
> 20 years	6	18.18	0	0.00	6	22.22
Total	33	100	6	100	27	100

Duration of Smoking	Group 1	Group 2
N	6	27
MEAN	2.00	9.93
SD	0.89	11.02
t test	0.0009	

By conventional criteria the association between the study groups and duration of smoking among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the duration of smoking in

Group 1 (2 years) is predominantly less when compared to Group 2 (9.93 years). It is statistically significant with a p-value of 0.0009 according to unpaired t test.

Clinical Significance

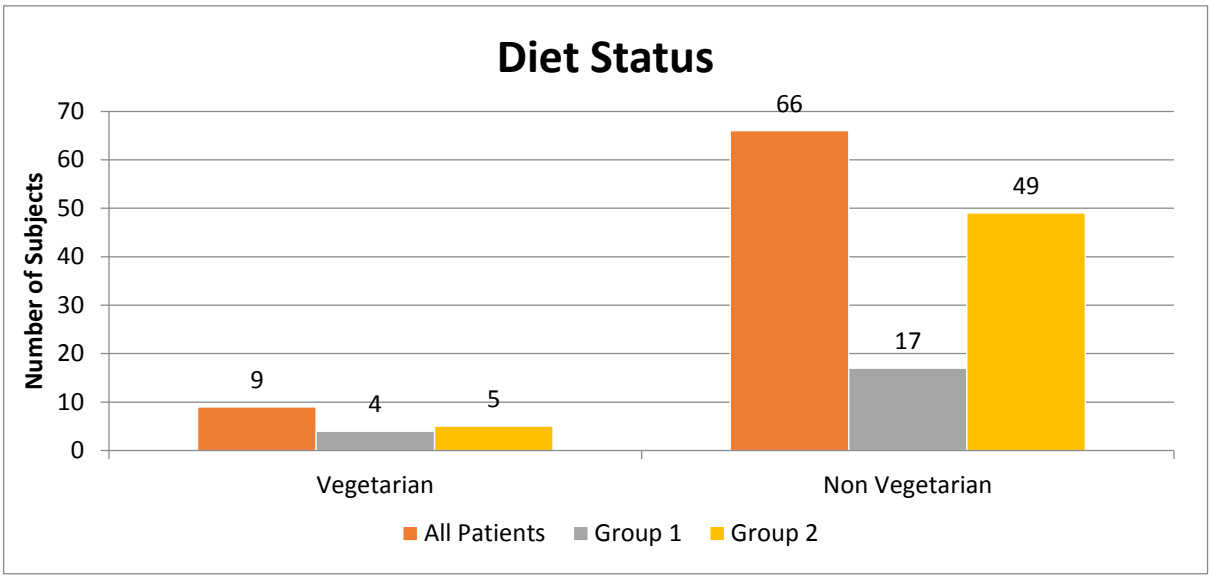
The duration of smoking in Group 1 is meaningfully less than Group 2 by 4.96 times with a mean difference of 7.93 years.

This difference is true and significant and has not occurred by chance.

Conclusion

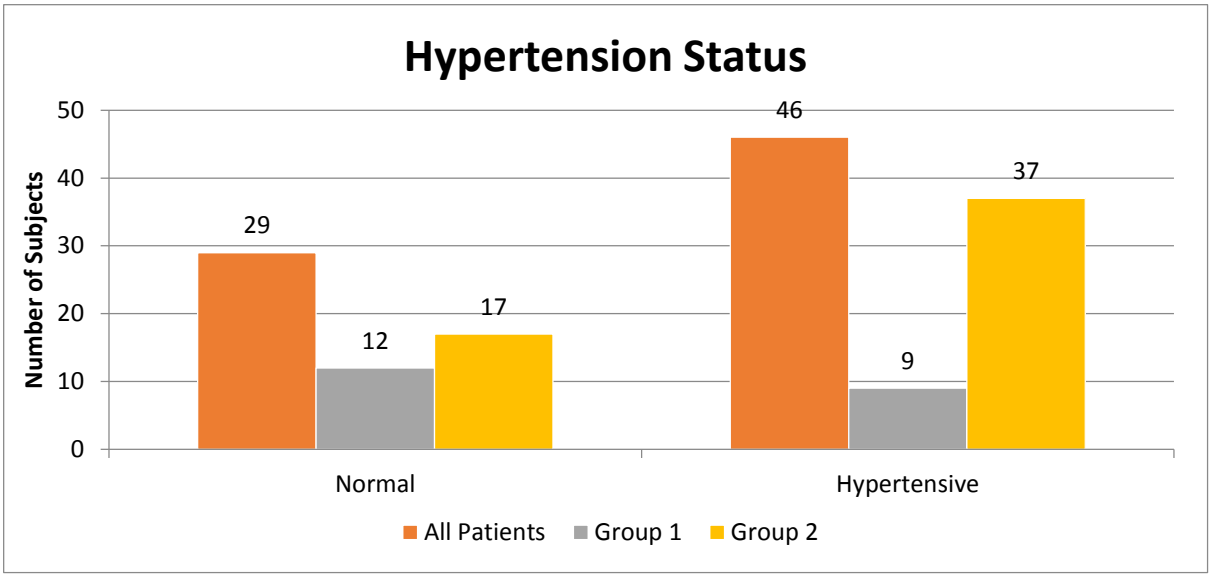
We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in smokers with increased duration of smoking among our study subjects

Diet



Diet Status	All Patients	%	Group 1	%	Group 2	%
Vegetarian	9	12.00	4	19.05	5	9.26
Non Vegetarian	66	88.00	17	80.95	49	90.74
Total	75	100	21	100	54	100
Chi-square value				1.03		
Degrees of freedom				1		
P value Chi Squared Test				0.309		

By conventional criteria the association between the study groups and diet status among study subjects is considered to be statistically not significant since $p > 0.05$.



Hypertension Status	All Patients	%	Group 1	%	Group 2	%
Normal	29	61.33	12	57.14	17	31.48
Hypertensive	46	38.67	9	42.86	37	68.52
Total	75	100	21	100	54	100
Chi-square value				4.20		
Degrees of freedom				1		
P value Chi Squared Test				0.040		

By conventional criteria the association between the study groups and hypertension distribution status among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the hypertension distribution

status in Group 1 (42.86% hypertensives) is predominantly less when compared to Group 2 (68.52% hypertensives). It is statistically significant with a p-value of 0.040 according to chi squared test.

Clinical Significance

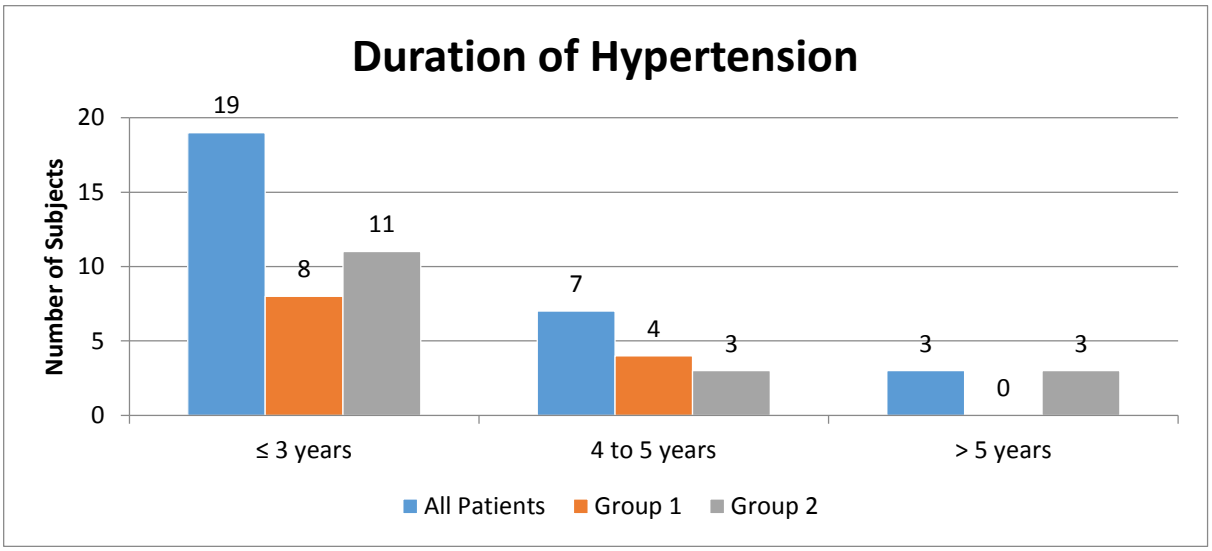
The hypertension distribution status in Group 1 is meaningfully less than Group 2 by 1.60 times with a mean difference of 25.66 percentage points.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in hypertensive among our study subjects.

Duration of HT

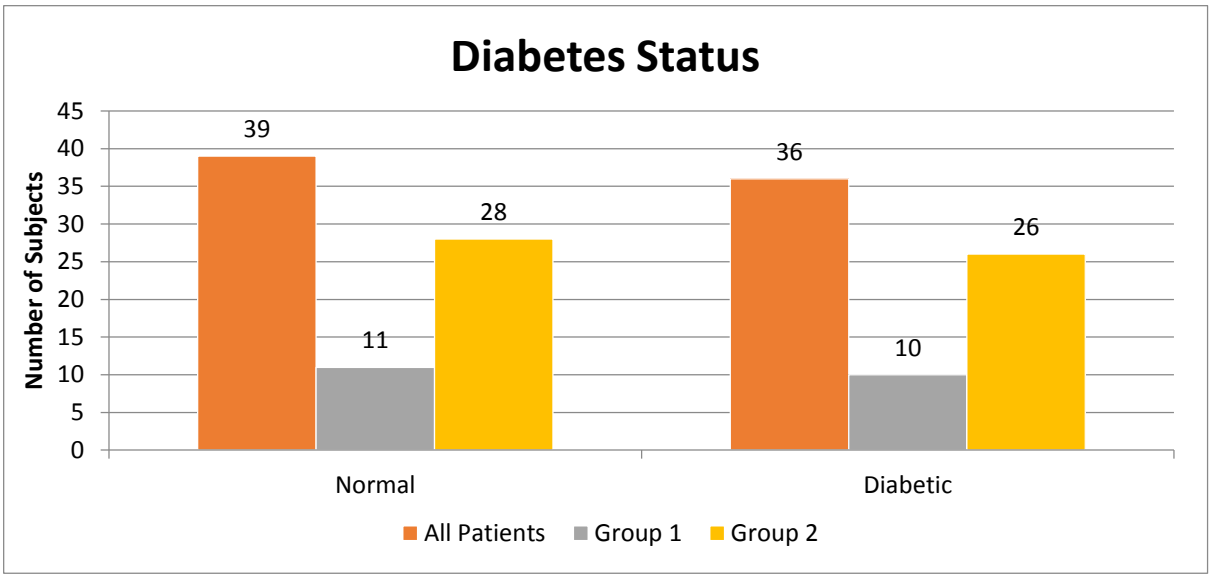


Duration of Hypertension	All Patients	%	Group 1	%	Group 2	%
≤ 3 years	19	65.52	8	66.67	11	64.71
4 to 5 years	7	24.14	4	33.33	3	17.65
> 5 years	3	10.34	0	0.00	3	17.65
Total	29	100	12	100	17	100

Duration of Hypertension	Group 1	Group 2
N	12	17
MEAN	3.00	3.06
SD	1.71	2.07
t test	0.9339	

By conventional criteria the association between the study groups and duration ofhypertension is considered to be not statistically significant since $p > 0.05$

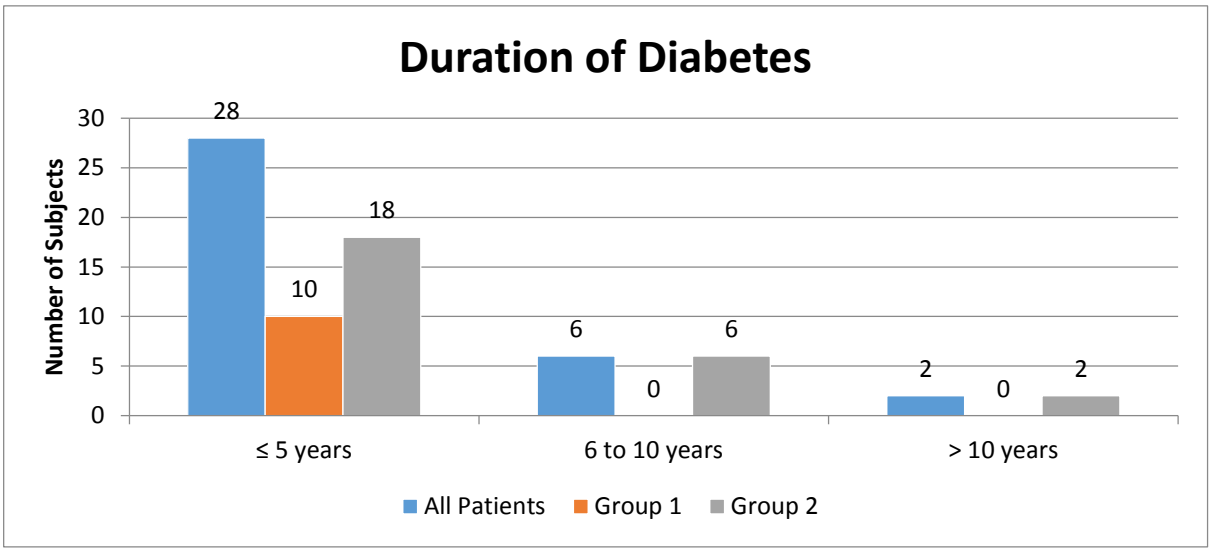
DM



Diabetes Status	All Patients	%	Group 1	%	Group 2	%
Normal	39	52.00	11	52.38	28	51.85
Diabetic	36	48.00	10	47.62	26	48.15
Total	75	100	21	100	54	100
Chi-square value				0.170		
Degrees of freedom				1		
P value Chi Squared Test				0.967		

By conventional criteria the association between the study groups and diabetes mellitus distribution status is considered to be not statistically significant since $p > 0.05$

DM Duration



Duration of Diabetes	All Patients	%	Group 1	%	Group 2	%
≤ 5 years	28	89.33	10	100.00	18	69.23
6 to 10 years	6	8.00	0	0.00	6	23.08
> 10 years	2	2.67	0	0.00	2	7.69
Total	36	100	10	100	26	100

By conventional criteria the association between the study groups and duration of diabetes mellitus among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the duration of diabetes mellitus in Group 1 (2.90 years) is predominantly less when compared to Group 2 (5.65 years). It is statistically significant with a p-value of 0.0162 according to unpaired t test.

Clinical Significance

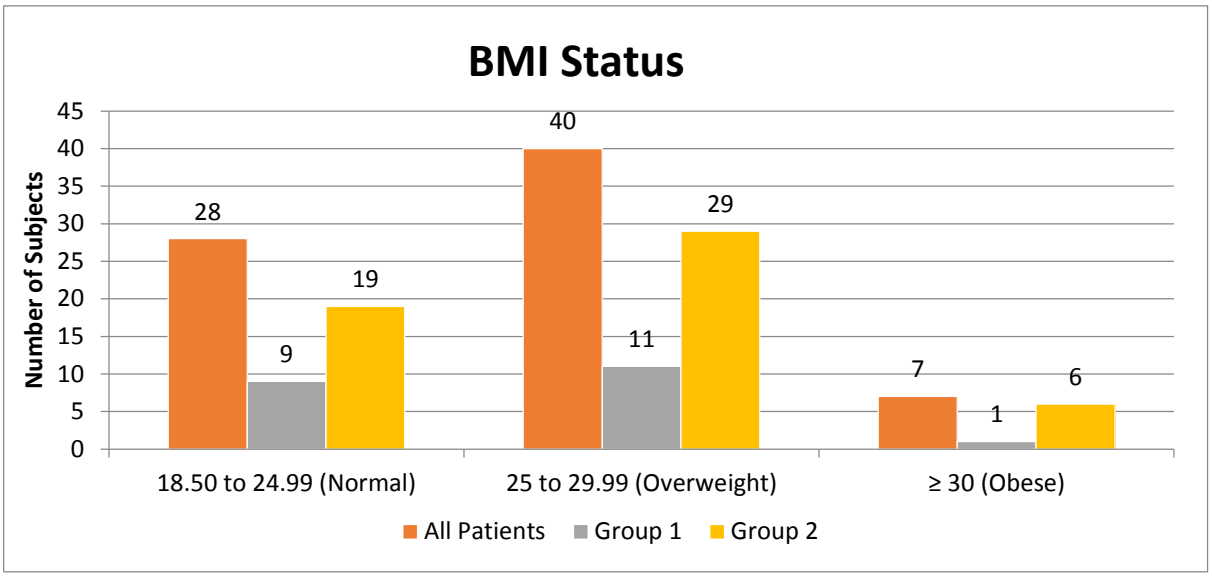
The duration of diabetes mellitus in Group 1 is meaningfully less than Group 2 by 1.95 times with a mean difference of 2.75 years.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased duration of diabetes mellitus among our study subjects

BMI

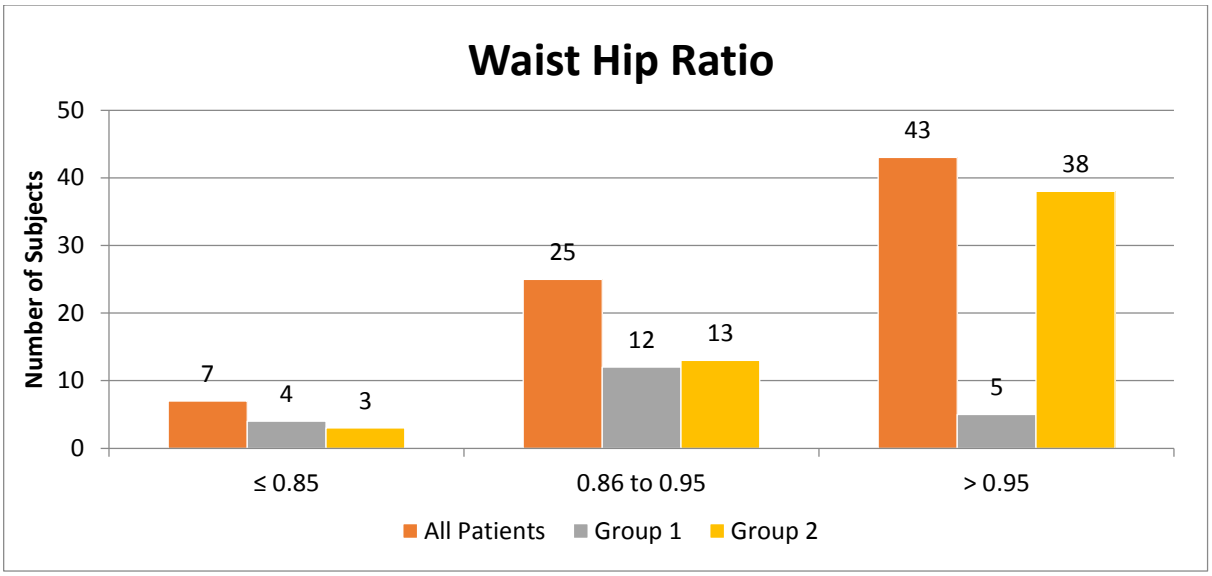


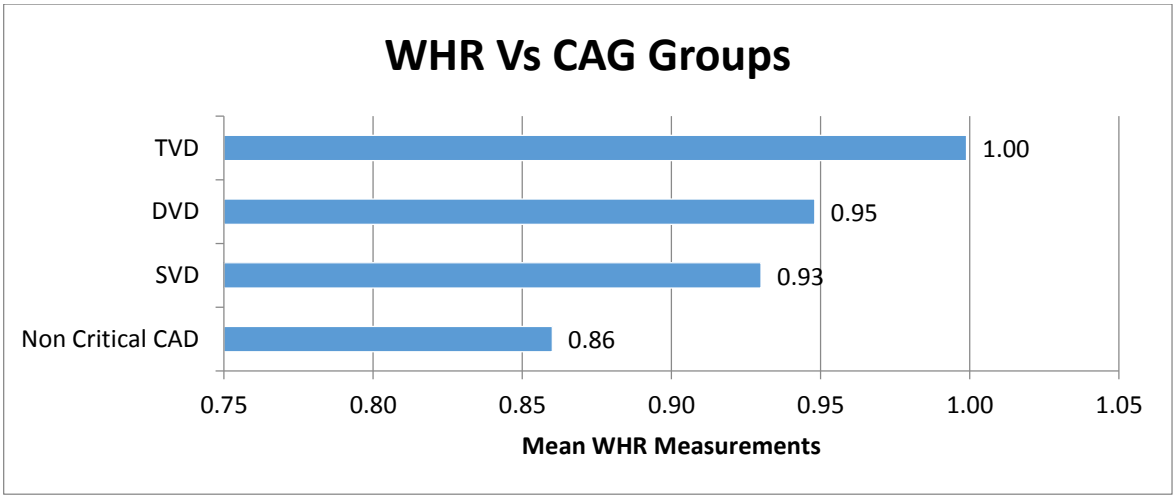
BMI Status	All Patients	%	Group 1	%	Group 2	%
18.50 to 24.99 (Normal)	28	37.33	9	42.86	19	35.19
25 to 29.99 (Overweight)	40	53.33	11	52.38	29	53.70
≥ 30 (Obese)	7	9.33	1	4.76	6	11.11
Total	75	100	21	100	54	100

BMI Status	Group 1	Group 2
N	21	54
MEAN	25.23	26.11
SD	2.85	3.54
t test	0.2688	

By conventional criteria the association between the study groups and BMI distribution status is considered to be not statistically significant since $p > 0.05$

WHR





Waist Hip Ratio	All Patients	%	Group 1	%	Group 2	%
≤ 0.85	7	9.33	4	19.05	3	5.56
0.86 to 0.95	25	33.33	12	57.14	13	24.07
> 0.95	43	57.33	5	23.81	38	70.37
Total	75	100	21	100	54	100

Groups	*Count*	*Mean*	*%*
Non Critical CAD	21	0.86	28
SVD	33	0.93	44
DVD	9	0.95	12

TVD	12	1.00	16
Total	75		100

Waist Hip Ratio	Group 1		Group 2
N	21		54
MEAN	0.91		1.04
SD	0.04		0.06
t test		0.0001	

By conventional criteria the association between the study groups and waist hip ratio among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the waist hip ratio in Group 1 (0.91) is predominantly less when compared to Group 2 (1.04). The waist hip ratio in non-critical CAD group (0.86) is predominantly less when compared to SVD(0.93), DVD(0.95) and TVD(1.00) groups It is statistically significant with a p-value of 0.0001 according to unpaired t test.

Clinical Significance

The waist hip ratio inGroup 1 is meaningfully less than Group 2 by 1.12 times with a mean difference of 0.13 measurement points.

The waist hip ratio in non-critical CAD group is meaningfully less than SVD group by 1.08 times with a mean difference of 0.07 measurement points.

The waist hip ratio in non-critical CAD group is meaningfully less than DVD group by 1.10 times with a mean difference of 0.09 measurement points.

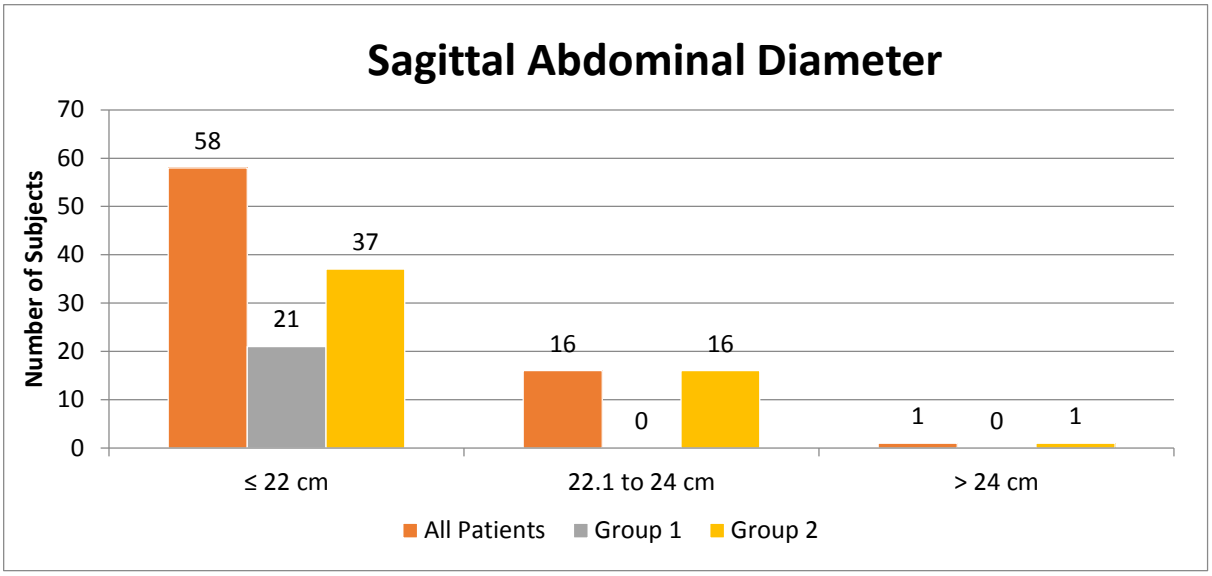
The waist hip ratio in non-critical CAD group is meaningfully less than TVD group by 1.16 times with a mean difference of 0.14 measurement points.

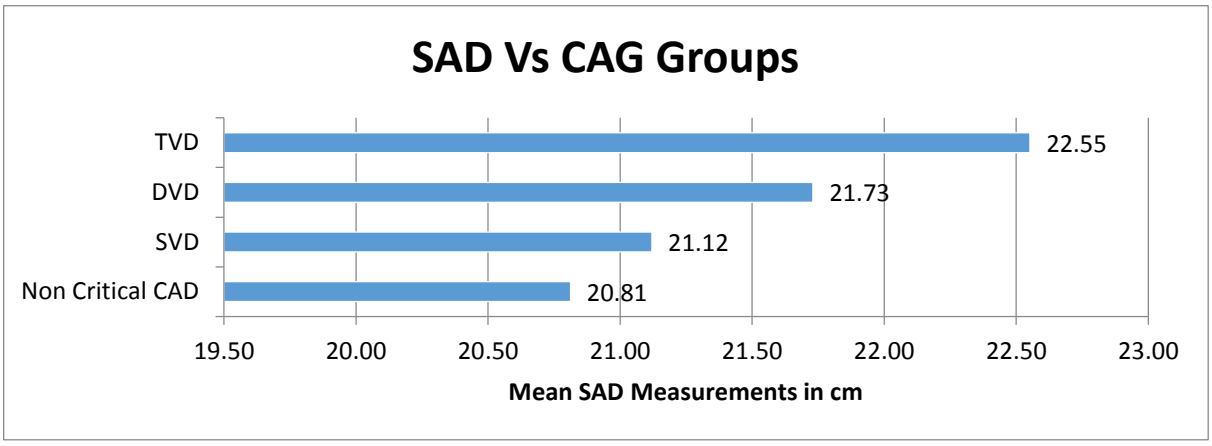
This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased waist hip ratio among our study subjects.

SAD





Sagittal Abdominal Diameter	All Patients	%	Group 1	%	Group 2	%
≤ 22 cm	58	77.33	21	100.00	37	68.52
22.1 to 24 cm	16	21.33	0	0.00	16	29.63
> 24 cm	1	1.33	0	0.00	1	1.85
Total	75	100	21	100	54	100

Groups	*Count*	*Mean*	*%*
Non Critical CAD	21	20.81	28
SVD	33	21.12	44
DVD	9	21.73	12
TVD	12	22.55	16
Total	75		100

Sagittal Abdominal Diameter	Group 1	Group 2
N	21	54
MEAN	20.81	21.80
SD	0.87	1.33
t test	0.0004	

By conventional criteria the association between the study groups and sagittal abdominal diameter among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the sagittal abdominal diameter in Group 1 (20.81 cm) is predominantly less when compared to Group 2 (21.80 cm). The sagittal abdominal diameter in noncritical CAD group(20.81 cm) is predominantly less when compared to SVD(21.12 cm), DVD(21.73 cm) and TVD(1.00) groups It is statistically significant with a p-value of 0.0001 according to unpaired t test.
It is statistically significant with a p-value of 0.0004 according to unpaired t test.

Clinical Significance

The sagittal abdominal diameter in Group 1 is meaningfully less than Group 2 by 1.05 times with a mean difference of 0.98 cm.

The sagittal abdominal diameter in non-critical CAD group is meaningfully less than SVD group by 1.01 times with a mean difference of 0.31 cm.

The sagittal abdominal diameter in non-critical CAD group is meaningfully less than DVD group by 1.04 times with a mean difference of 0.92 cm.

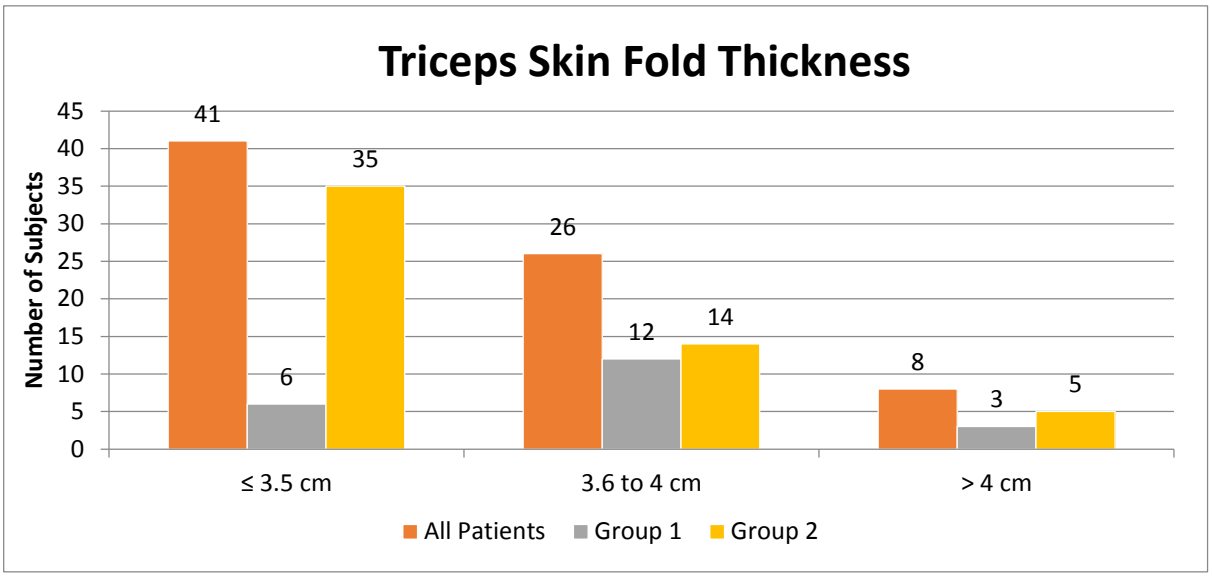
The sagittal abdominal diameter in non-critical CAD group is meaningfully less than TVD group by 1.08 times with a mean difference of 1.74 cm.

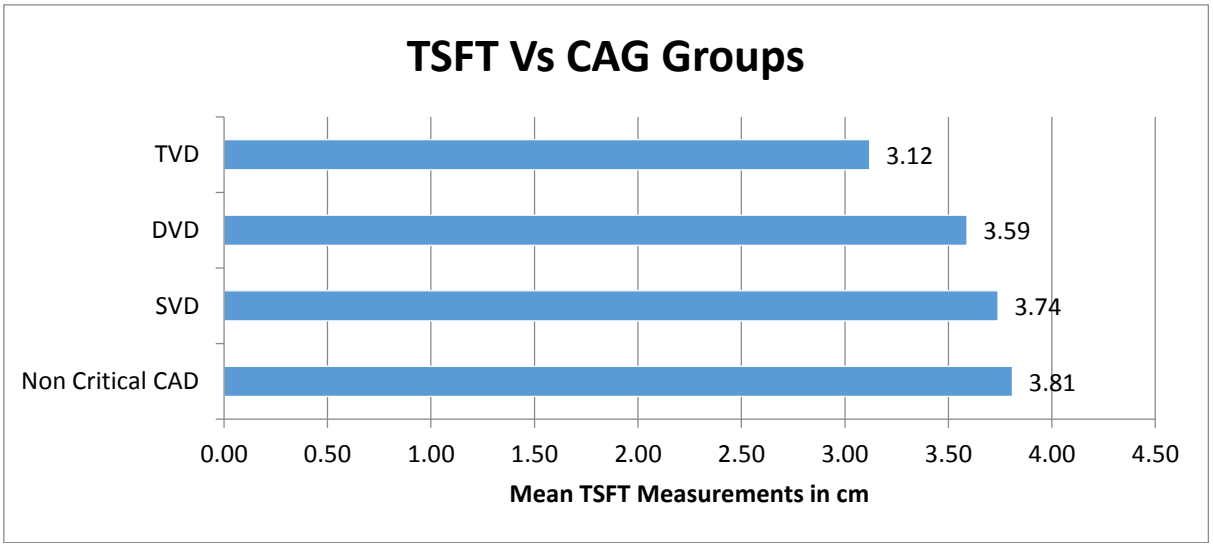
This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased sagittal abdominal diameter among our study subjects.

TSFT





Triceps Skin Fold Thickness	All Patients	%	Group 1	%	Group 2	%
≤ 3.5 cm	41	54.67	6	28.57	35	64.81
3.6 to 4 cm	26	34.67	12	57.14	14	25.93
> 4 cm	8	10.67	3	14.29	5	9.26
Total	75	100	21	100	54	100

Groups	*Count*	*Mean*	*%*
Non Critical CAD	21	3.81	28
SVD	33	3.74	44

DVD	9	3.59	12
TVD	12	3.12	16
Total	75		100

Triceps Skin Fold Thickness	Group 1	Group 2
N	21	54
MEAN	3.74	3.52
SD	0.33	0.58
t test	0.0457	

By conventional criteria the association between the study groups and Triceps Skin Fold Thickness among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the Triceps Skin Fold Thickness in Group 1 (3.74 cm) is predominantly more when compared to Group 2 (3.52cm). The Triceps Skin Fold Thicknessin non-critical CAD group (3.81cm) is predominantly more when compared to SVD (3.74 cm), DVD (3.59 cm) and TVD (3.21 cm) groups. It is statistically significant with a p-value of 0.0457 according to unpaired t test.

Clinical Significance

The Triceps Skin Fold Thickness in Group 1 is meaningfully more than Group 2 by 1.06 times with a mean difference of 0.22 cm.

The Triceps Skin Fold Thickness in non-critical CAD group is meaningfully more than SVD group by 1.02 times with a mean difference of 0.07 cm.

The Triceps Skin Fold Thickness in non-critical CAD group is meaningfully more than DVD group by 1.06 times with a mean difference of 0.22 cm.

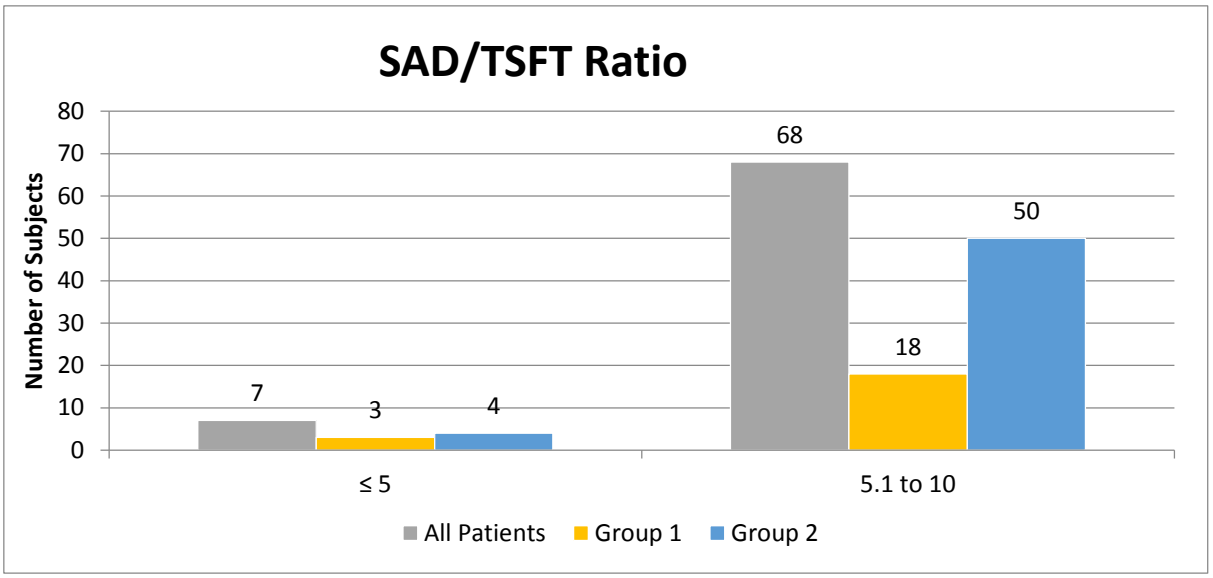
The Triceps Skin Fold Thickness in non-critical CAD group is meaningfully more than TVD group by 1.22 times with a mean difference of 0.69 cm.

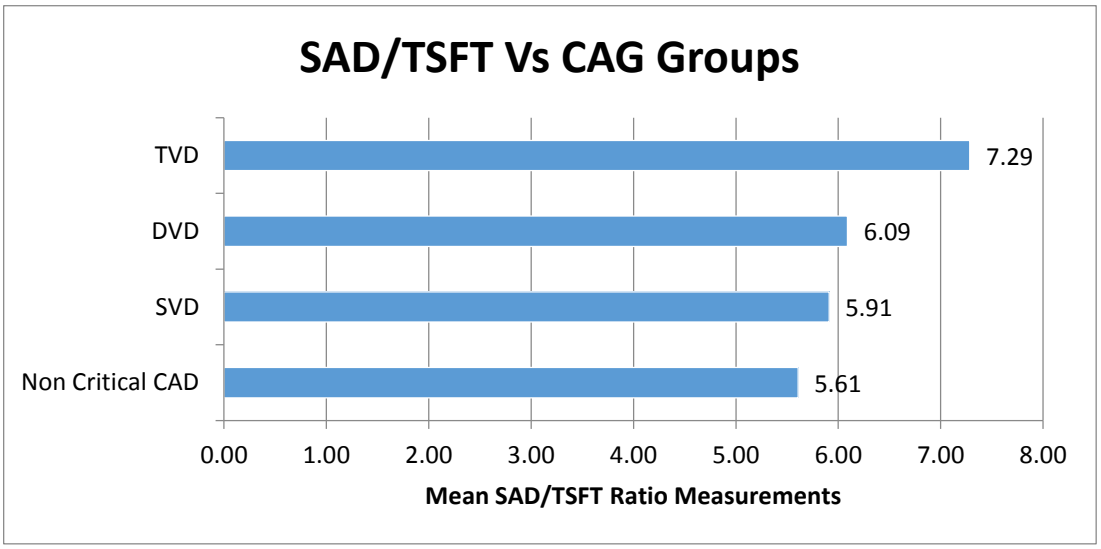
This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with decreased Triceps Skin Fold Thickness among our study subjects.

SAD/TSFT





SAD/TSFT	All Patients	%	Group 1	%	Group 2	%
≤ 5	7	9.33	3	14.29	4	7.41
5.1 to 10	68	90.67	18	85.71	50	92.59
Total	75	100	21	100	54	100

Groups	*Count*	*Mean*	*%*
Non Critical CAD	21	5.61	28

SVD	33	5.91	44
DVD	9	6.09	12
TVD	12	7.29	16
Total	75		100

SAD/TSFT Ratio	Group 1	Group 2
N	21	54
MEAN	5.61	6.32
SD	0.55	0.96
t test	0.0001	

By conventional criteria the association between the study groups and SAD/TSFT ratio among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the SAD/TSFT ratio in Group 1 (5.61) is predominantly less when compared to Group 2 (6,32). The SAD/TSFT ratio in noncritical CAD group(5.61) is predominantly less when compared to SVD(5.91), DVD(6.09) and TVD(7.29) groups .It is statistically significant with a p-value of 0.0001 according to unpaired t test.

Clinical Significance

The SAD/TSFT ratio in Group 1 is meaningfully less than Group 2 by 1.13 times with a mean difference of 0.72 measurement points.

The SAD/TSFT ratio in non-critical CAD group is meaningfully less than SVD group by 1.05 times with a mean difference of 0.30 measurement points.

The SAD/TSFT ratio in non-critical CAD group is meaningfully less than DVD group by 1.09 times with a mean difference of 0.48 measurement points.

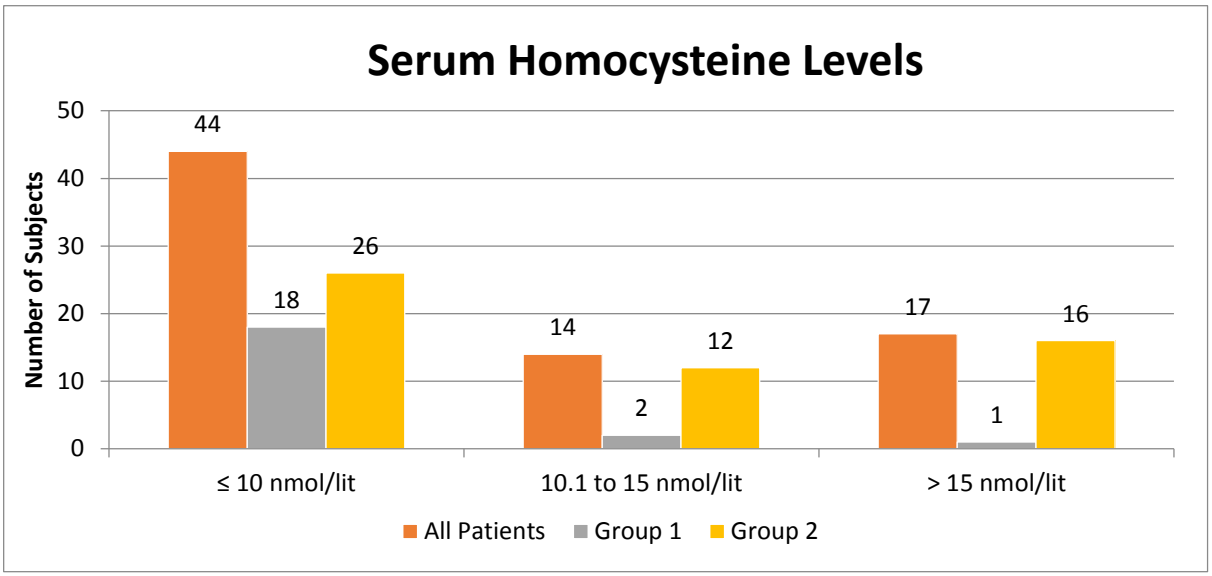
The SAD/TSFT ratio in non-critical CAD group is meaningfully less than TVD group by 1.30 times with a mean difference of 1.68 measurement points.

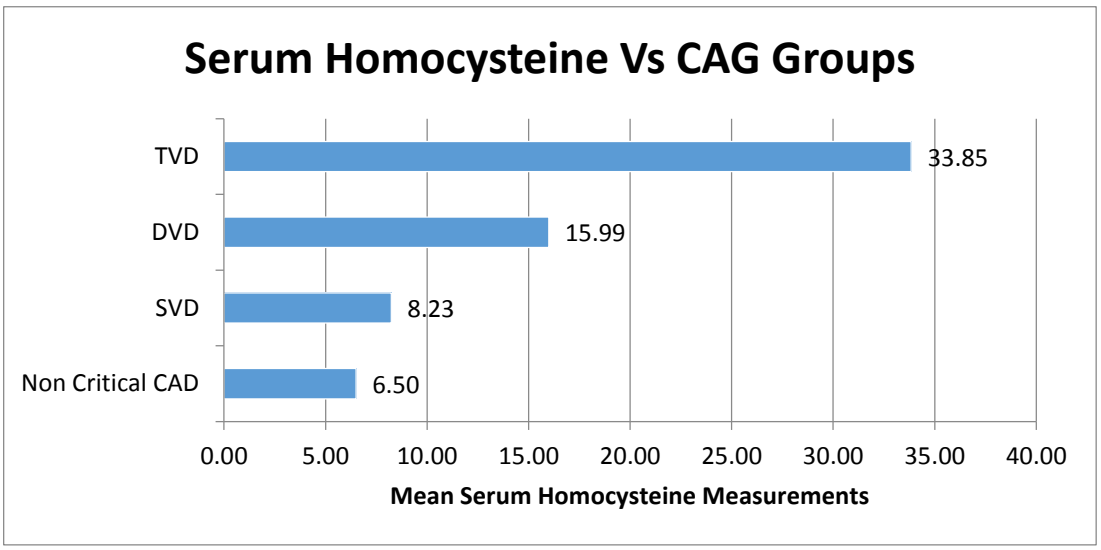
This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased SAD/TSFT ratio among our study subjects.

HOMOCYSTEINE





Homocysteine Levels	All Patients	%	Group 1	%	Group 2	%
≤ 10 nmol/lit	44	58.67	18	85.71	26	48.15
10.1 to 15 nmol/lit	14	18.67	2	9.52	12	22.22
> 15 nmol/lit	17	22.67	1	4.76	16	29.63
Total	75	100	21	100	54	100

Groups	*Count*	*Mean*	*%*
Non Critical CAD	21	6.50	28
SVD	33	8.23	44
DVD	9	15.99	12
TVD	12	33.85	16

Total	75	100

Homocysteine Levels	Group 1	Group 2
N	21	54
MEAN	6.50	15.22
SD	3.59	13.63
t test	0.0000	

By conventional criteria the association between the study groups and serum homocysteine levels among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum homocysteine level along with the severity of coronary stenosis, the serum homocysteine levels in Group 1 (6.50 nmol/lit) is predominantly less when compared to Group 2 (15.22 nmol/lit). The serum homocysteine levels in noncritical CAD group(6.50 nmol/lit) is predominantly less when compared to SVD(8.23 nmol/lit), DVD(15.99 nmol/lit) and TVD(33.85 nmol/lit) groups .It is statistically significant with a p-value of 0.0000 according to unpaired t test.

Clinical Significance

The serum homocysteine levels in Group 1 is meaningfully less than Group 2 by 2.34 times with a mean difference of 8.72 nmol/lit.

The serum homocysteine levels in non-critical CAD group is meaningfully less than SVD group by 1.27 times with a mean difference of 1.73 nmol/lit.

The serum homocysteine levels in non-critical CAD group is meaningfully less than DVD group by 2.46 times with a mean difference of 9.49 nmol/lit.

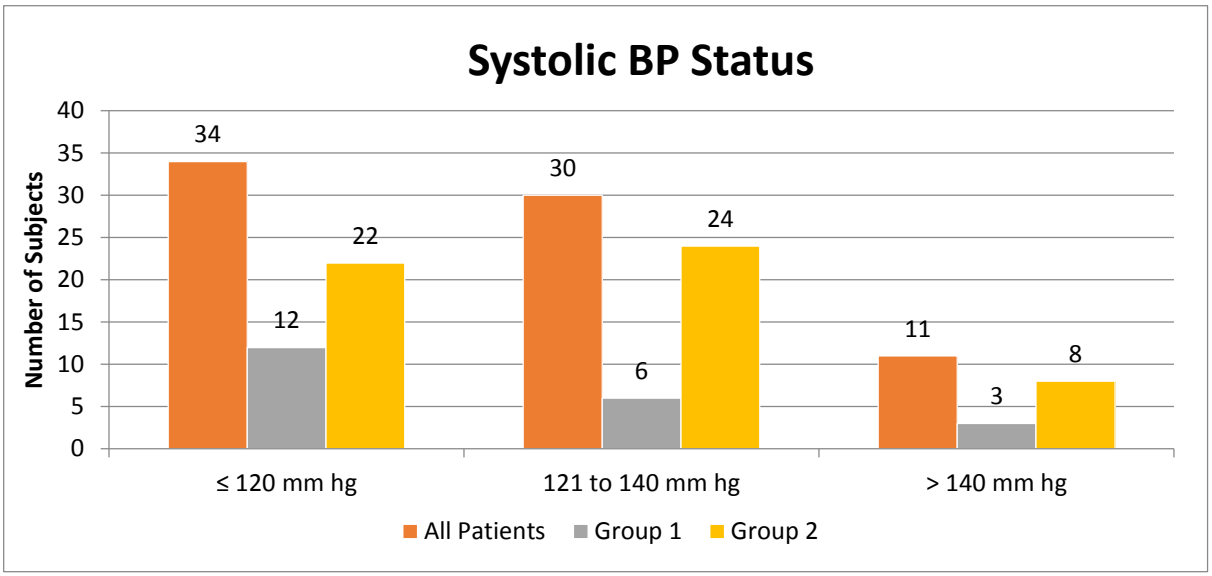
The serum homocysteine levels in non-critical CAD group is meaningfully less than TVD group by 5.20 times with a mean difference of 27.35 nmol/lit.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased serum homocysteine levels among our study subjects.

SBP

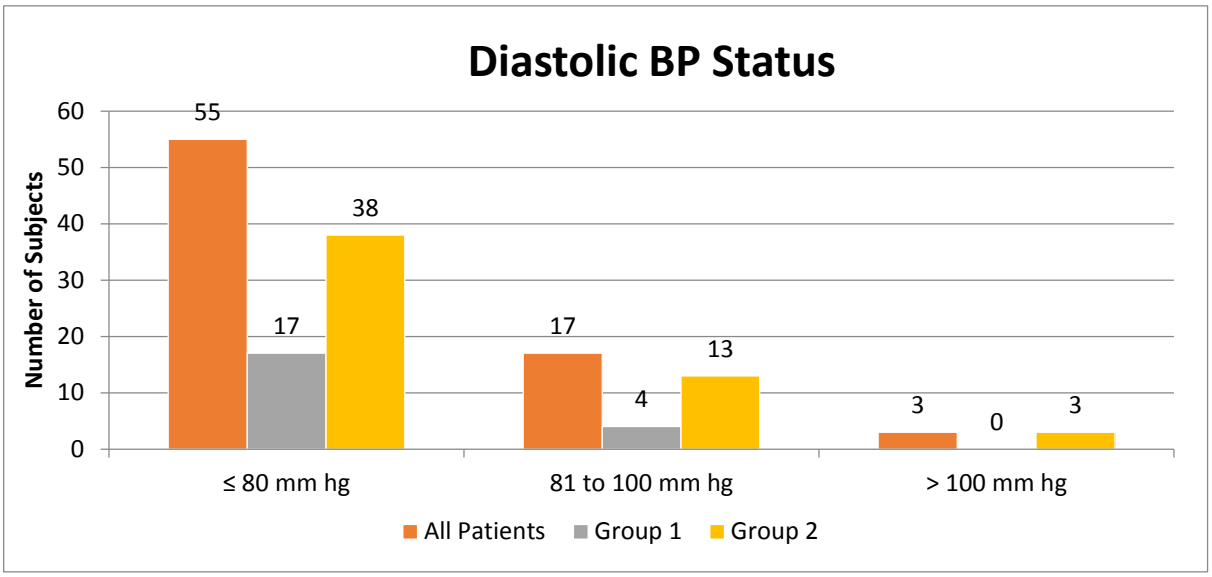


Systolic BP Status	All Patients	%	Group 1	%	Group 2	%
≤ 120 mm hg	34	46.67	12	57.14	22	40.74
121 to 140 mm hg	30	38.67	6	28.57	24	44.44
> 140 mm hg	11	14.67	3	14.29	8	14.81
Total	75	100	21	100	54	100

Systolic BP Status	Group 1	Group 2
N	21	54
MEAN	126.48	131.22
SD	14.24	18.58
t test	0.2420	

By conventional criteria the association between the study groups and systolic BP distribution status is considered to be not statistically significant since $p > 0.05$

DBP

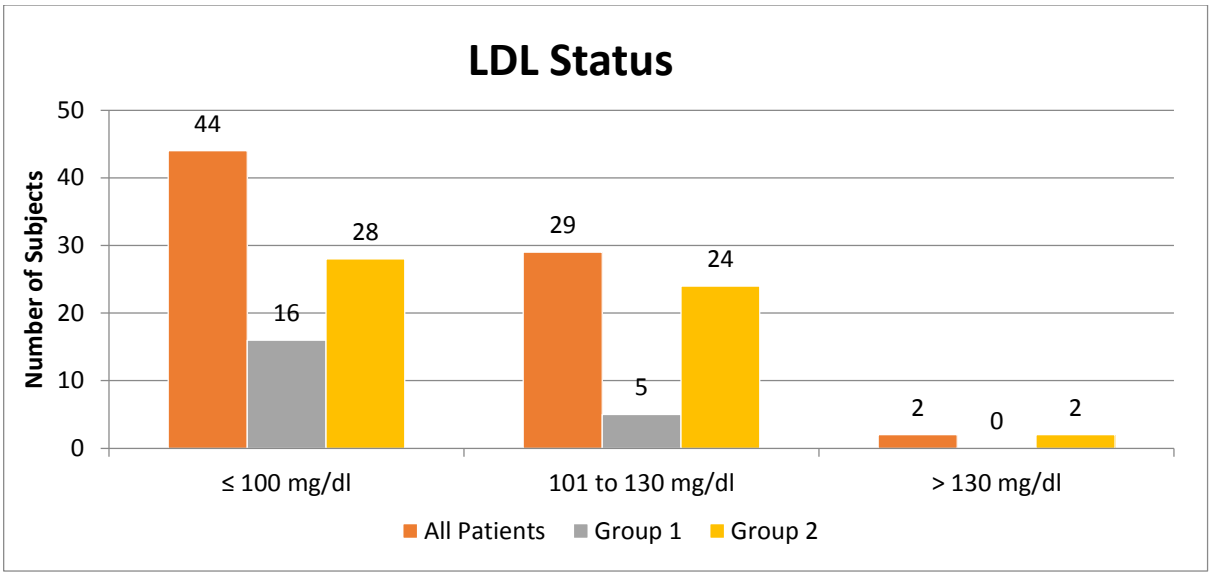


Diastolic BP Status	All Patients	%	Group 1	%	Group 2	%
≤ 80 mm hg	55	73.33	17	80.95	38	70.37
81 to 100 mm hg	17	21.33	4	19.05	13	24.07
> 100 mm hg	3	5.33	0	0.00	3	5.56
Total	75	100	21	100	54	100

Diastolic BP Status	Group 1	Group 2
N	21	54
MEAN	77.81	81.15
SD	10.99	10.07
t test	0.2351	

By conventional criteria the association between the study groups and diastolic BP distribution status is considered to be not statistically significant since $p > 0.05$

LDL



LDL Status	All Patients	%	Group 1	%	Group 2	%
≤ 100 mg/dl	44	64.00	16	76.19	28	51.85
101 to 130 mg/dl	29	36.00	5	23.81	24	44.44
> 130 mg/dl	2	0.00	0	0.00	2	3.70
Total	75	100	21	100	54	100

LDL Status	Group 1	Group 2
N	21	54
MEAN	94.24	100.52
SD	9.18	12.76
t test	0.0217	

By conventional criteria the association between the study groups and serum LDL levels among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum LDL level along with the severity of coronary stenosis, the serum homocysteine levels in Group 1 (9.24 mg/dl) is predominantly less when compared to Group 2 (100.52 mg/dl). It is statistically significant with a p-value of 0.0217 according to unpaired t test.

Clinical Significance

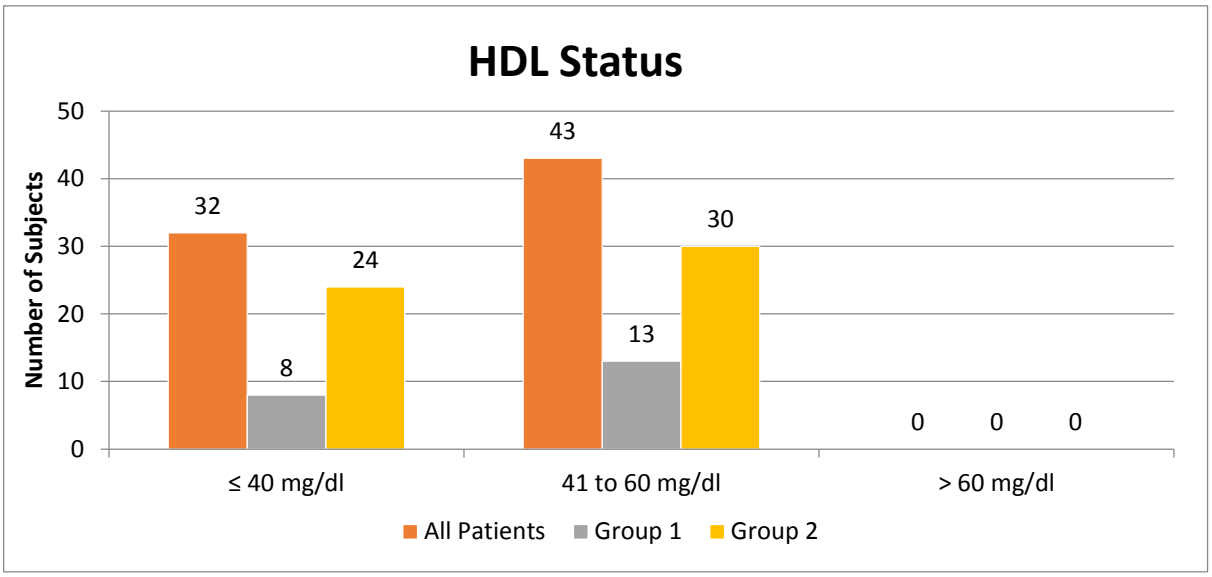
The serum LDL levels in Group 1 is meaningfully less than Group 2 by 1.07 times with a mean difference of 6.28 mg/dl.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased serum LDL levels among our study subjects.

HDL

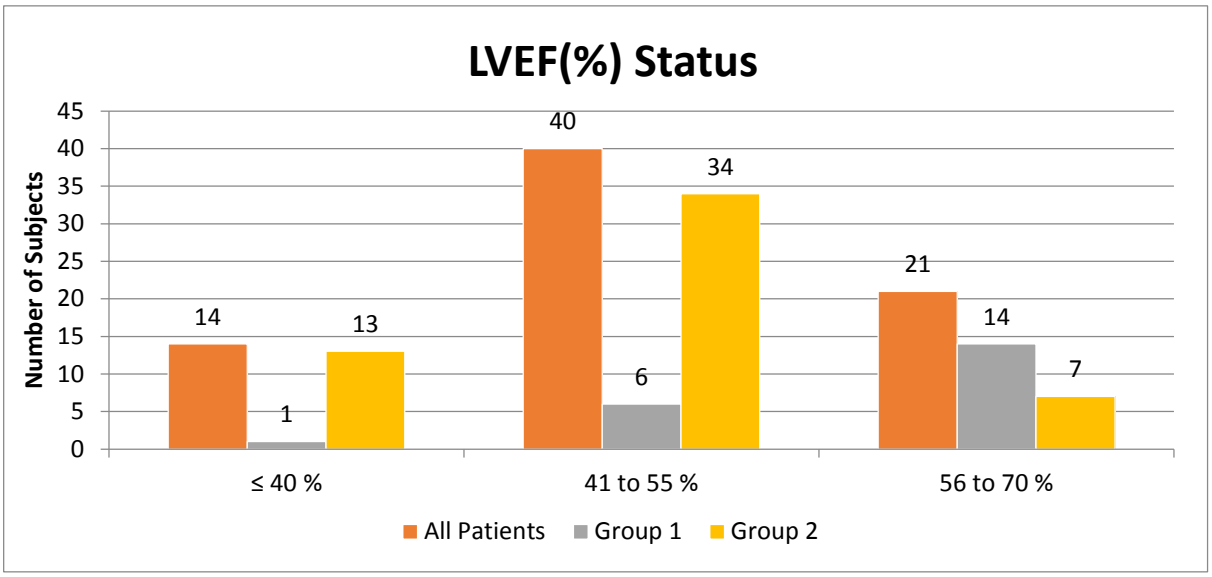


HDL Status	All Patients	%	Group 1	%	Group 2	%
≤ 40 mg/dl	32	42.67	8	38.10	24	44.44
41 to 60 mg/dl	43	57.33	13	61.90	30	55.56
> 60 mg/dl	0	0.00	0	0.00	0	0.00
Total	75	100	21	100	54	100

HDL Status	Group 1	Group 2
N	21	54
MEAN	41.71	41.56
SD	5.00	5.94
t test	0.9074	

By conventional criteria the association between the study groups and serum HDL levels is considered to be not statistically significant since $p > 0.05$

LVEF



LVEF (%) Status	All Patients	%	Group 1	%	Group 2	%
≤ 40 %	14	17.33	1	4.76	13	24.07
41 to 55 %	40	53.33	6	28.57	34	62.96
56 to 70 %	21	29.33	14	66.67	7	12.96
Total	75	100	21	100	54	100

LVEF (%) Status	Group 1	Group 2
N	21	54
MEAN	57.19	45.56
SD	6.92	6.56
t test	0.0000	

By conventional criteria the association between the study groups and LVEF% status among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum LDL level along with the severity of coronary stenosis, the LVEF% status in Group 1 (57.19%) is predominantly more when compared to Group 2 (45.56%). It is statistically significant with a p-value of 0.0217 according to unpaired t test.

Clinical Significance

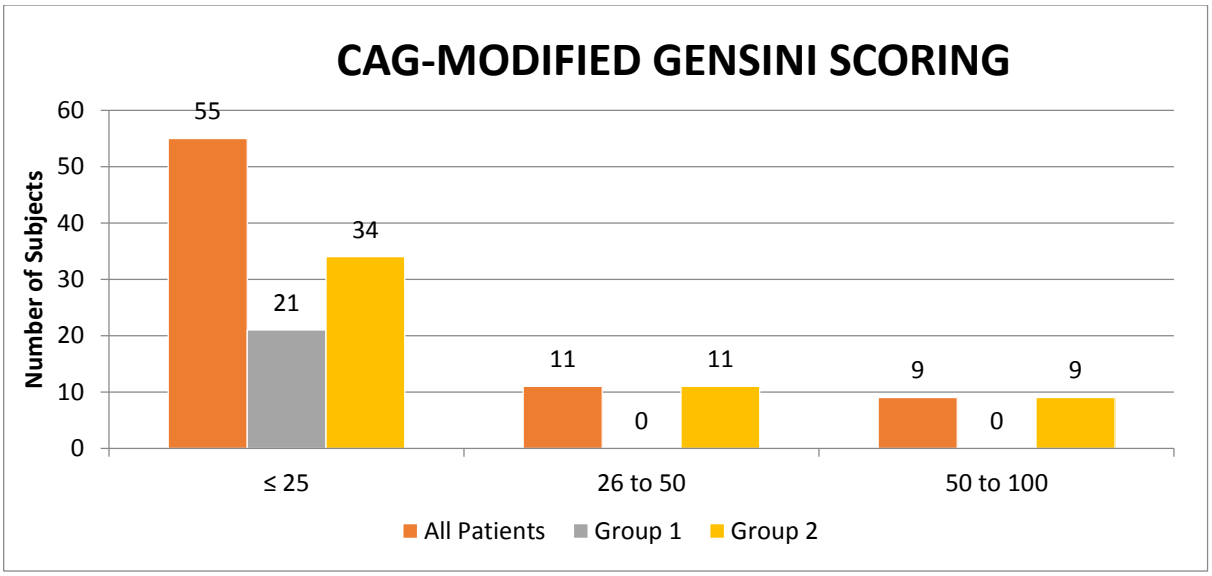
The LVEF% statusin Group 1 is meaningfully more than Group 2 by 1.25 times with a mean difference of 11.63 percentage measurement points.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with decreased LVEF% status among our study subjects.

GENSINI



CAG-MODIFIED GENSINI SCORING	All Patients	%	Group 1	%	Group 2	%
≤ 25	55	73.33	21	100.00	34	62.96
26 to 50	11	14.67	0	0.00	11	20.37
50 to 100	9	12.00	0	0.00	9	16.67
Total	75	100	21	100	54	100

CAG-MODIFIED GENSINI SCORING	Group 1	Group 2
N	21	54
MEAN	1.10	30.19
SD	1.37	23.32
t test	0.0000	

By conventional criteria the association between the study groups and Gensini scores among study subjects is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, when studying the association between various anthropometric measures and serum LDL level along with the severity of coronary stenosis, the Gensini scores in Group 1 (1.10 points) is predominantly less when compared to Group 2 (30.19 points). It is statistically significant with a p-value of 0.0000 according to unpaired t test.

Clinical Significance

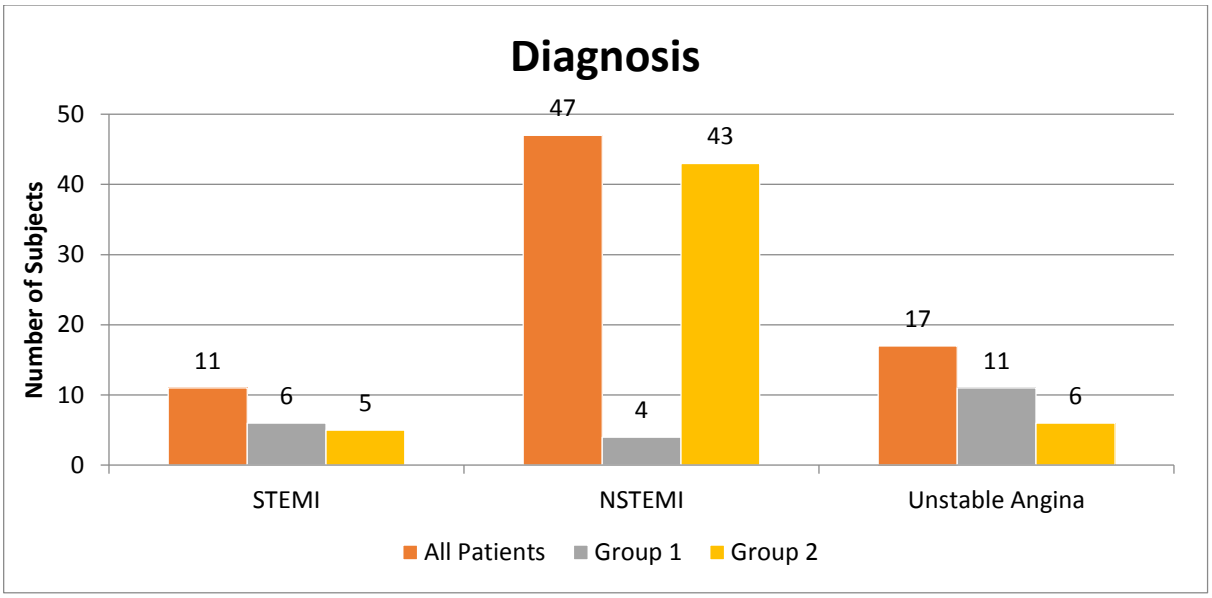
The Gensini scoresin Group 1 is meaningfully less than Group 2 by 27.56 times with a mean difference of 29.09 scoring points.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is meaningfully real increase in risk of developing increased coronary stenosis in patients with increased Gensini scores among our study subjects.

DIAGNOSIS



Diagnosis	All Patients	%	Group 1	%	Group 2	%
STEMI	11	61.33	6	28.57	5	9.26
NSTEMI	47	16.00	4	19.05	43	79.63
Unstable Angina	17	22.67	11	52.38	6	11.11
Total	75	100	21	100	54	100

Diagnosis	Group 1	Group 2
N	21	54
MEAN	2.24	2.02
SD	0.89	0.46
t test	0.2916	

By conventional criteria the association between the study groups and diagnosis at the time of admission is considered to be not statistically significant since $p > 0.05$

CITATIONS

A. Vasheghani-Farahani et al.showed SAD-to-TSF ratio, has a substantially better accuracythan do the known indices of obesity like body mass index, waist circumference, and waist-to-hip ratiofor the prediction of premature CAD. He found that sagittal abdominal diameter to triceps skinfold thickness ratio as a novel anthropometric index to predict premature coronaryAtherosclerosis.

AC Carlsson et alin his study tried to establish Novel and established anthropometric measures and the predictionof incident cardiovascular disease: a cohort study and found that WHHR, a measure reflecting body fat distribution, showed the highest risk estimates after adjustments forestablished CVD risk factors. These findings were verified in men but not women in an independent cohort.

R Abraham et al studied on homocysteine level and foundraised serum homocysteine levels in patients of coronary Artery disease and the effect of vitamin b12 and folate on its Concentration.

Kazemimb et al in one study found hyperhomocysteinemia is related to

CAD as an independent risk factor. In individuals without any risk factors a

linear correlation between homocysteine level and numbers of coronary

artery involvement was present

Kahn et al proposed that ADI might be more effective atestimating visceral adipose tissue and, hence, a better predictor ofCVD than WHR

Wilken et al published the first reportthat patients with coronary artery disease haveabnormal homocysteine metabolism.

CONCLUSION

In my study using 75 patients under 50 years, I am able to come to a conclusion of some anthropometric variables better correlating with the severity of coronary stenosis and some not showing much correlation as listed below

1) Increased Waist-hip ratio, sagittal abdominal diameter, triceps skin fold thickness and SAD/TSFT ratio has got better correlation with the severity of coronary artery disease, with SAD/TSFT and WHR has got maximum significance of all anthropometric measurements.

2) Increased BMI poorly correlating with the severity of CAD as shown by p value more than 0.05.

3) Increased homocysteine level better correlates with the severity of coronary artery disease.

4) Increased LDL cholesterol value had good correlation with CAD severity butdecreased HDL cholesterol not correlating much with coronaryartery disease.

5) Duration of smoking but not mere smoking history better correlates with the severity of coronaryartery disease.Smoking is found to be

an independent high risk factor even in patients with low BMI, low waist or hip circumference and low WHR or SAD/TSFT ratio.

6) Presence of hypertension and duration of diabetes got positive correlation on coronary artery disease severity.

7) Left ventricular ejection fraction (LVEF) also correlates with the severity of coronary artery disease with an inverse relation between them.

8) Modified gensini score – higher the modified gensini score, severe is the coronary artery disease.

9) It appears that the old concept of BMI being considered as major risk factor in the past will not apply nowadays as an important determinant for CAD risk assessment, as it is shown in our study and in many major studies to be statistically less significant and it's better to give importance to central Vs peripheral obesity ratio methods to assess future risk of CAD.

10) We feel great emphasis should be given on cessation of smoking compared to control of obesity as it's a major atherosclerotic risk factor even in thin individuals and those patients are prone for CAD at very young age.

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PROFORMA

NAME : SL. NO:
AGE /SEX:
OCCUPATION:
ADDRESS WITH CONTACT NUMBER:

IP NO:
DATE OF ADMISSION:
DATE OF DISCHARGE:

HISTORY:

CHEST PAIN

- **Duration**
- **Severity**
- **Pain radiation**

BREATHLESSNESS

ORTHOPNEA

PND

LEG SWELLING

FACIAL PUFFINESS

PAST HISTORY

- **HYPERTENSION**
- **DIABETES**
- **CAD**
- **CVD**
- **COPD**
- **CKD**

PERSONAL HISTORY

- **SMOKER**
- **ALCOHOLIC**

PHYSICAL EXAMINATION

PALLOR

ICTERUS

CYANOSIS

CLUBBING

PEDAL EDEMA

JVP

BP:

PR:

HEIGHT

WEIGHTS

BMI

WAIST CIRCUMFERENCE

HIP CIRCUMFERENCE

WAIST HIP RATIO

SAGITTAL ABDOMINAL CIRCUMFERENCE

TRICEPS SKIN FOLD THICKNESS

SAD/TSFT

CVS -

RS -

P/A -

CNS –

INVESTIGATIONS

CBC – TC

DC

ESR

HB

PLATELETS

BLOOD SUGAR

UREA

SERUM CREATININE

SODIUM

POTASSIUM

CHLORIDE

BICARBONATE

BLOOD GROUP

VIRAL MARKERS

SERUM HOMOCYSTEINE LEVEL

URINE EXAMINATION

TOTAL CPK

CPK-MB LEVEL

ECG

ECHOCARDIOGRAPHY

SEVERITY OF CORONARY STENOSIS USING MODIFIED GENSINI SCORING

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC: "A STUDY ON CORRELATION OF VARIOUS ANTHROPOMETRIC MEASURES AND SERUM HOMOCYSTEINE LEVEL WITH THE SEVERITY OF CORONARY ARTERY DISEASE IN YOUNG PATIENTS UNDERGOING CORONARY ANGIOGRAPHY"

PLACE OF STUDY: HUTTON WARD, DEPARTMENT OF CARDIOLOGY, STANELY MEDICAL COLLEGE AND HOSPITAL, CHENNAI**.**

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.

I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:

Signature/Thumb impression of the Volunteer

Date:

Witnesses:
(Signature, Name & Address)

Date:
Name and signature of investigator:

Date:

மனிதரளவையியல் அளவுருக்கள் மற்றும் இரத்த ஹோமோசிஷ்டின் அளவையும் கரோனரி தமனியின் குறுக்கத்தின் தீவிரத்தோடு ஒப்பிடும் ஒரு ஆய்வு

ஆய்வாளர்: **மரு.கோ.சுரேஷ்** (முதுநிலை பட்டமேற்படிப்பு மாணவர், பொது மருத்துவ பட்டபடிப்பு)

வழிகாட்டி: **மரு.க.தமிழ்செல்வன்** (இருதய மருத்துவ துணை பேராசிரியர்)
அரசு ஸ்டான்லி மருத்துவமனை.

<u>பங்கேற்பாளரின் தகவல் படிவம்</u>

நீங்கள் இந்த ஆய்வில் பங்கேற்க அழைக்கப்படுகிறீர்கள். இந்த ஆய்வில் பங்கேற்கும் முன், இதன் நோக்கத்தையும், முறைகளையும், இதனால் ஏற்படும் பின்விளைவுகளையும் நீங்கள் அறிந்து கொள்ள ஆய்வாளர் அளிக்கும் தகவல்:
உங்கள் நோயின் வரலாறும், உங்களின் முழு உடல் பரிசோதனையும் தெளிவாகவும் விரிவாகவும் பதிவு செய்யப்படும்.
இந்த ஆய்வின் முடிவுகள் மருத்துவ காரணங்களுக்காகவும், மருத்துவ கல்விக்காகவும் பயன்படுத்தப்படும். இந்த ஆய்வு பற்றிய சந்தேகங்களுக்கு உரிய முறையில் விளக்கமளிக்கப்படும்.
தங்களைப்பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும்.
இந்த ஆய்வில் இருந்து எப்போது வேண்டுமானாலும் தாங்கள் எவ்வித முன்னறிவிப்பின்றியும், எவ்வித சட்டசிக்கலும் இன்றி விலகிக்கொள்ளலாம்.
இந்த ஆய்வில் பங்கேற்குமாறு கேட்டுக்கொள்கிறேன்.
நன்றி,

ஆய்வாளர் கையொப்பம் நோயாளியின் கையொப்பம்
(மரு.**கோ.சுரேஷ்**) (பெயர்:)

மனிதரளவையியல் அளவுருக்கள் மற்றும் இரத்த ஹோமோசிஷ்டின் அளவையும் கரோனரி தமனியின் குறுக்கத்தின் தீவிரத்தோடு ஒப்பிடும் ஒரு ஆய்வு

ஆய்வாளர்: **மரு.கோ.சுரேஷ்** (முதுநிலை பட்டமேற்படிப்பு மாணவர், பொது மருத்துவ பட்டபடிப்பு)

வழிகாட்டி: **மரு.க.தமிழ்செல்வன்** (இருதய மருத்துவ துணை பேராசிரியர்)
அரசு ஸ்டான்லி மருத்துவமனை.

<u>சுயஒப்புதல்படிவம்</u>

பெயர்: வயது:

உள்ளிருப்பு எண்:

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களைக்கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும், எந்த கட்டத்திலும், எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்து கொண்டேன். என்னைப்பற்றிய தகவல்கள் இரகசியமாகப் பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப எவ்விதமாகப் பயன்படுத்திக்கொள்ளவும், அதனை பிரசுரிக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன், ஆய்வாளருக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

என் உடல்நலம் பாதிக்கப்பட்டாலோ அல்லது வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை தெரிவிப்பேன் என உறுதிகூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும், சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

நோயாளியின் கையொப்பம் ஆய்வாளர் கையொப்பம்/பெயர்

(பெயர்:)

(மரு**கோ.சுரேஷ்**.)

MASTER CHART

AGE	S	SMOKING	NV	HT	DM	BMI	WHR	SAD(cm)	SAD/TSFT	HOMO	SBP	DBP	LDL	HDL	EF	CAG	GEN
50	M	YES,10 YRS	YES	NO	NO	25	0.989	19.5	6.5	13.50	140	110	95	36	40	SVD	24
49	M	NO	YES	NO	NO	24	0.953	21.3	6.65625	1208.00	130	80	90	35	53	DVD	46
48	M	YES,20 YRS	YES	NO	NO	26.2	1.0117	22	9.565217	10.56	100	60	120	30	42	TVD	31
43	M	YES,40 YRS	YES	NO	NO	24.4418	0.9525	21	5.121951	6.46	130	80	89	39	45	SVD	4
49	M	YES,15 YRS	YES	NO	NO	29.01745	0.9533	19.8	5.657143	13.58	120	80	120	32	45	SVD	48
46	M	YES,10YRS	YES	NO	NO	23.56	0.957	22	6.470588	7.65	140	90	105	34	42	DVD	92
39	M	YES,20YRS	YES	NO	NO	24.53896	0.89	20.2	6.3125	7.52	140	80	110	32	55	NORMAL	
38	F	NO	YES	YES,3YRS	YES,6 MO	28.658	0.95	21	4.468085	4.70	110	70	98	42	60	NON OBS	2
48	F	NO	YES	NO	NO	26.535	0.91	19.6	5.090909	5.80	120	80	85	45	60	NORMAL	0
49	F	NO	YES	YES,6YRS	YES,6YRS	32.89474	1.008	21.2	5.520833	8.40	110	80	105	33	60	SVD	24
47	M	NO	YES	NO	YES,3YRS	27.43484	1.21	23	8.214286	29.58	120	90	142	41	33	TVD	68
42	F	NO	NO	NO	YES,3YRS	27.89	0.923	21.5	5.972222	9.50	120	80	86	40	62	NORMAL	0
48	F	NO	YES	YES,10YRS	NO	27.11111	0.9663	21	6.501548	12.40	150	70	89	50	60	DVD	21
47	F	NO	YES	YES,10YRS	YES,5YRS	26.557	0.914	20	4.878049	2.50	150	100	90	40	60	NON OBS	4
42	F	NO	YES	YES,3YRS	YES,5YRS	31.62628	0.875	21	5.185185	6.00	120	80	92	37	64	NON OBS	2
39	M	yes,3YRS	YES	YES,1YR	YES,1YR	24.856	0.895	19.4	4.974359	5.20	130	80	89	52	60	NON OBS	2.5
48	M	NO	NO	NO	NO	22	0.844	21.3	6.016949	5.10	130	80	87	43	50	NON OBS	1
49	M	YES,50YRS	YES	NO	NO	26.02617	0.9	22	6.470588	12.50	120	70	99	38	54	DVD	84
46	M	YES,20YRS	YES	NO	NO	27.6398	0.985	22.3	6.757576	11.00	120	70	99	45	45	SVD	12
47	M	YES,10YRS	YES	NO	NO	20.95717	0.85	19.5	4.875	10.10	110	70	87	42	50	NORMAL	0
47	M	NO	NO	NO	NO	23.11111	0.954	22	5.684755	5.32	120	80	95	39	45	SVD	6
45	M	YES,30YRS	YES	NO	NO	24.97704	0.923	21	5	4.90	120	80	110	46	45	SVD	24
50	F	NO	YES	NO	YES,3YRS	37.195	0.9846	21.1	5.861111	3.58	190	110	104	40	41	SVD	40
38	M	NO	YES	YES,1YR	NO	28.13366	0.9762	21.2	6.347305	3.87	100	70	83	46	45	SVD	8
50	M	NO	YES	YES,6MON	YES,18YRS	27.73438	0.9584	21	6.501548	6.50	140	90	88	48	40	SVD	12
48	M	YES,10YRS	YES	NO	YES,2YRS	30.40529	0.9623	20.5	5	6.48	140	90	108	50	48	SVD	6
46	M	YES,25YRS	YES	NO	YES,10YRS	25.6	1.042	21.2	7.066667	54.20	150	80	91	46	42	TVD	48
45	M	NO	YES	YES,5YRS	NO	27.4	1.08	22	6.567164	23.98	120	80	132	43	40	TVD	17
43	M	YES,5YRS	YES	NO	NO	19.60716	0.85	20.8	5.621622	6.12	140	80	105	48	40	SVD	10
47	M	YES,32YRS	YES	NO	YES,2YRS	26.9	0.9984	22.4	7	35.68	130	80	104	50	48	TVD	22
48	M	NO	YES	YES,5YRS	NO	25.46	0.987	21.2	5.888889	4.56	120	60	108	47	62	NORMAL	0
46	F	NO	NO	NO	YES,5YRS	25.3	0.88	21.6	5.684211	2.35	136	70	104	43	56	SVD	16
45	F	NO	YES	YES,3YRS	YES,4YRS	26.3	0.98	21.6	6.687307	4.89	124	86	85	37	43	SVD	24
42	M	yes 20 yrs	YES	NO	NO	24.5	0.96	21.8	6.392962	9.89	130	90	96	34	38	DVD	34
43	F	NO	YES	YES 2YRS	YES 5YRS	26.9	1.02	23	7.055215	19.50	120	80	95	34	53	TVD	54

48	M	YES 15 YRS	YES	NO	YES 10YRS	25.6	1.15	23.2	7.182663	28.46	110	70	92	36	42	TVD	58
47	M	YES,40 YRS	YES	YES 6 YRS	NO	25.6	0.99	22.1	5.580808	12.90	130	80	95	33	45	SVD	14
49	M	YES,15 YRS	YES	NO	NO	29.01745	0.9524	27	6.75	11.26	120	80	120	32	45	SVD	48
50	M	YES,10YRS	YES	NO	NO	21.51694	0.924	16	4.571429	12.90	140	90	105	34	42	DVD	92
50	M	YES,20YRS	YES	NO	NO	24.53896	0.9535	20	5.405405	4.23	140	80	110	32	55	NORMAL	
45	F	NO	YES	YES,3YRS	YES,6 MO	27.84	0.975	21.4	6.114286	4.86	110	70	98	42	60	NON OBS	2
41	F	NO	YES	NO	NO	25.453	0.8954	21	6.034483	5.60	120	80	85	45	60	NORMAL	0
62	F	NO	YES	YES,6YRS	YES,6YRS	32.89474	1	22.2	6.434783	8.60	110	80	105	33	60	SVD	24
40	M	NO	YES	NO	YES,3YRS	27.43484	0.955	22.9	7.02454	25.68	120	90	80	41	33	TVD	68
40	M	NO	NO	YES 5YRS	YES,3YRS	28.42	0.9245	21.3	5.916667	2.89	160	90	96	36	35	NORMAL	0
48	F	NO	YES	YES,10YRS	NO	27.11111	0.966	21	3.230769	20.42	150	70	89	50	60	DVD	21
46	F	NO	YES	YES,10YRS	YES,5YRS	25.6	0.896	21	6.422018	5.46	150	100	90	40	60	NON OBS	4
45	F	NO	YES	YES,3YRS	YES,5YRS	22.3	0.9314	21	6.086957	6.87	120	80	92	37	64	NON OBS	2
43	M	yes,3YRS	YES	YES,1YR	YES,1YR	21.54	0.96	21.5	5.657895	5.68	126	84	86	45	60	NON OBS	2.5
49	M	NO	NO	NO	NO	22	0.844	18	4.5	12.45	130	80	87	43	50	NON OBS	1
47	M	YES,50YRS	YES	NO	NO	26.02617	0.9	22	6.790123	26.50	120	70	99	38	54	DVD	84
44	M	YES,20YRS	YES	NO	NO	23.4	0.945	21.6	6.315789	6.48	120	70	99	45	45	SVD	12
43	M	YES,10YRS	YES	NO	NO	20.95717	0.85	21	6	6.50	110	70	87	42	50	NORMAL	0
49	M	NO	NO	NO	NO	23.11111	0.825	21.8	5.589744	5.98	120	80	95	39	45	SVD	6
48	M	YES,30YRS	YES	NO	NO	24.97704	0.9125	22.4	6.327684	5.47	120	80	110	46	45	SVD	24
50	F	NO	YES	NO	YES,3YRS	37.195	0.967	21	6	8.95	190	110	104	40	41	SVD	38
39	M	NO	YES	YES,1YR	NO	23.5	0.945	21.86	6.726154	9.56	100	70	83	46	45	SVD	8
38	M	NO	YES	YES,6MON	YES,18YRS	27.73438	0.9	21.3	6.085714	8.52	140	90	88	48	40	SVD	12
46	M	YES,10YRS	YES	NO	YES,2YRS	30.40529	1.047	22.9	5.871795	4.86	140	90	108	50	48	SVD	6
48	M	YES,25YRS	YES	NO	YES,12YRS	24.87	0.92	21	5.949008	3.46	146	80	94	47	48	SVD	50
47	M	NO	YES	YES,5YRS	NO	25.21736	0.9765	21.8	6.987179	65.45	120	80	87	43	40	TVD	17
49	M	YES,5YRS	YES	NO	NO	19.60716	0.879	20.8	5.730028	10.50	140	80	105	48	40	SVD	12
50	M	YES,32YRS	YES	NO	YES,2YRS	22.71897	0.984	22.32	7.2	42.53	130	80	104	50	48	TVD	22
50	M	NO	YES	YES,5YRS	NO	26.03749	0.947	21.5	5.512821	2.86	120	60	108	47	62	NORMAL	0
46	F	NO	NO	NO	YES,5YRS	25.3	0.9756	21.7	5.740741	4.86	136	70	104	43	56	SVD	16
42	F	NO	YES	YES,3YRS	YES,4YRS	26.3	0.9845	21.6	5.684211	4.56	124	86	85	37	43	SVD	24
47	M	YES,25YRS	YES	NO	YES,10YRS	26.5	0.968	22.8	7.102804	15.98	150	80	91	46	42	TVD	52
47	M	NO	YES	YES,5YRS	NO	25.21736	0.9965	24	7.5	38.54	120	80	123	43	40	TVD	17
38	M	YES,5YRS	YES	NO	NO	19.60716	0.85	22.1	6.820988	4.85	140	80	105	48	40	SVD	10
53	M	YES,32YRS	YES	NO	YES,2YRS	27.3	0.94	22.6	7.040498	45.60	130	80	114	45	48	TVD	22
47	M	NO	YES	YES,5YRS	NO	25.988	0.9523	22	5.789474	18.1	120	60	108	47	62	NORMAL	0
53	F	NO	NO	NO	YES,5YRS	25.3	0.965	22	6.567164	18.87	136	70	104	43	56	SVD	18
47	F	NO	YES	YES,6YRS	NO	24.2	0.9785	21.4	6.858974	13.4	124	86	85	37	43	SVD	26
48	F	NO	YES	NO	YES,4.5yR	22.6	0.9863	21.5	6.697819	23.45	180	74	114	38	43	SVD	42
49	M	NO	YES	YES,6MON	YES,18YRS	22.1	0.9	21	6	8.52	140	90	88	48	40	SVD	12

KEY TO MASTER CHART

S – SEX

M – MALE

F – FEMALE

NV – NON VEGETARIAN DIET

HT – HYPERTENSION

DM – DIABETES

BMI – BODY MASS INDEX

SAD – SAGITTAL ABDOMINAL DIAMETER

TSFT – TRICEPS SKIN FOLD THICKNESS

WHR – WAIST HIP RATIO

HOMO – SERUM HOMOCYSTEINE LEVEL

SBP – SYSTOLIC BLOOD PRESSURE

DBP – DIASTOLIC BLOOD PRESSURE

LDL – LOW DENSITY LIPOPROTEIN

HDL – HIGH DENSITY LIPOPROTEIN

CAG – CORONARY ANGIOGRAM

EF – LEFT VENTRICULAR EJECTION FRACTION

GEN - MODIFIED GENSINI SCORE

SVD – SINGLE VESSEL DISEASE

DVD – DOUBLE VESSEL DISEASE

TVD – TRIBLE VESSEL DISEASE

NON OBS – NON OBSTRUCTIVE CAD

UA – UNSTABLE ANGINA