Coronary Heart Disease Work Up - 2015

Dr.S.Shanmugam, M.D., PGDHSc (Echocardiography),
P.G.P in Diabetology (Johns Hopkins University, U.S.A),
Diagnostic, Preventive and Echo Cardiologist & Diabetologist,
Researcher in New Methodologies in Medicine,
Geeyes Diabetes and Hypertension Research Centre,
Research Centre for Undiagnosed & Rare Diseases,
Thirunelveli,Tamil Nadu, India.

WORLD HEART DAY - 2015

29th September

HEALTHY HEART CHOICES FOR

EVERY ONE, EVERY WHERE

HEART CHOICES NOT HARD CHOICES

Dedicated to

Thiru. R.Sundara Raman,

B.Com., C.A., (Late)

My Friend and An Important Soul in

Bringing the Dream Project of Tirunelveli

Getwell Hospitals in 1994.

Golden Jubilee Celebrating

Tirunelveli Medical College

Where I have finished my M.B.B.S

as well as M.D (Gen. Medicine).

Acknowledgement

I thank all my family members for sparing their time of mine.

Coronary Heart Disease Work Up - 2015

Dr.S.Shanmugam, M.D., PGDHSc (Echocardiography),
P.G.P in Diabetology
(The Johns Hopkins University School of Medicine, U.S.A),

Thirunelveli

GEEYES TRUST
Thirunelveli
2015

Coronary Heart Disease work up - 2015

S.Shanmugam

Second Edition of Coronary Heart Disease Work up : September, 2015.

Publisher : **GEEYES TRUST**
1, Bharathi Street, S.T.C.Road,
N,G.O.'A'.Colony,
Thirunelveli - 627 007,
India.

Typeset and Printed at : **G.P.R. Offset Printers,**
159/23/1, Kurichi Road,
Melakulavanikarpuram,
Thirunelveli - 627 005.
Mob: 9443453271
Ph : 2573281 (0462)

PRICE : ₹ 50/-

CORONARY HEART DISEASE WORK UP - 2015

Dr.S.Shanmugam, M.D., PGDHSc (Echocardiography),
P.G.P in Diabetology
(The Johns Hopkins University School of Medicine, U.S.A),
Diagnostic, Preventive and Echo Cardiologist & Diabetologist,
Researcher in New Methodologies in Medicine,
Geeyes Diabetes and Hypertension Research Centre,
Research Centre for Undiagnosed & Rare Diseases,
Thirunelveli,Tamil Nadu, India.
Cell: 9843009655
Email: shanbhatir@gmail.com

Introduction:

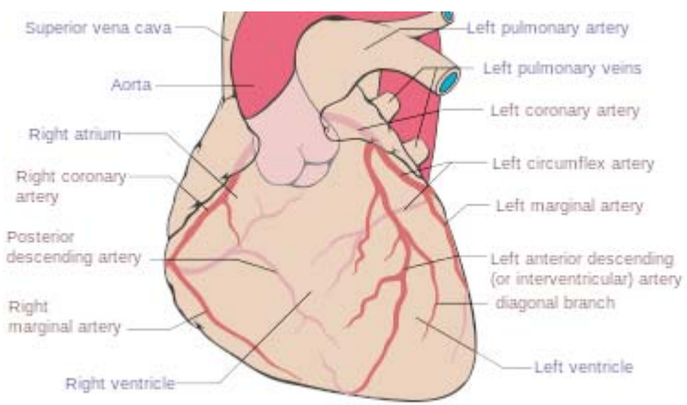
Coronary Heart Disease or Coronary Artery Disease is a spectrum of diseases affecting the anatomy, physiology and the metabolism of coronary arteries and ultimately that of the myocardium. There is a paucity of reliable contemporary data on the prevalence of coronary artery disease (CAD) and risk factors in Indians. The prevalence of CAD in India is getting into a speedy pathway is a known fact as per the statistics available. Four people die of heart attack every minute in India and the age group is mainly between 30 and 50.Twenty-five per cent of heart attack deaths occur in people less than 40.Nine hundred people under 30 die due to heart disease in India every day. Coronary artery disease and coronary risk factors are two or three times higher among the urban compared with the rural subjects, which may be due to greater sedentary behaviour and alcohol intake among urban. It is high time, every right thinking medical personnel should come forward to make the prevention of CAD as the main aim of practice for the sake of humanity to survive with better people.

The real problem today related to CAD in India is, we as Physicians and Cardiologists are following the protocol created by Western literature and in spite of lot of follow up studies in India, going through the literature available, there is no specific India Centric Cardiac Work Up Methodology available even from bigger centres mainly concentrating on this important and dangerous disease of the present century. The aim of this short and practical write up is to create a Cardiac work up for Indian patients related to CAD or CAHD.

Being in the field of medicine for more than thirty nine years (1977 as a house surgeon), my experiences with the so called heart attack in vernacular language or CAD in technical language, as a House Surgeon, Family Physician, Post Graduate in General Medicine(1980-1982), Physician, ICU in charge Physician of a corporate hospital, Preventive Cardiologist and Diagnostic Cardiologist- Echo Cardiologist and the main area of my concentration namely as a Diabetologist, I have gained knowledge related to prevention, assessment and guiding at the present juncture using the modern methodology namely mapping the coronary arteries using conventional Coronary Angiography(CAG) or CT Coronary Angiography and deciding about the PTCA or CABG or medical management. As an important care giver after these procedures namely PTCA or CABG to prevent further deterioration in the long run, which is very much lacking in concept in India, I have also dwelt into the realm of so called rare causes of CAD especially in young people namely **“Myocardial bridging”** as a cause of CAD and also **“Spongiform cardiomyopathy”** always mistaken as Ischaemic Cardiomyopathy and are also diagnosed by me independently and have made me to think about sharing my hard earned knowledge for the development of the science of coronary arteries ,can be christened as **Coronology** (I hope this is the first of its kind nomenclature in medical literature)and not chronology

and also for the benefit of patients. That is the reason for rewriting this small booklet on Coronary heart disease work up -2015. This will enhance the India centric approach among the Physicians, Cardiologists and Cardiothoracic Surgeons for creating in the long run a protocol for our Indian patients and this is the aim of rewriting this book and also my wish.

I thank all the patients for their confidence reposed on the very dangerous and life threatening illness to be treated during the heydays of Physicians as practical Cardiologists and now in a new avatar as a guiding Cardiologist for CAG done patients, the need for PTCA or CABG or medical management after assessing CAG personally.This work up will not discuss about Acute Myocardial Infarction and its management. Emergency PTCA or CABG also does not come into this discussion for obvious reasons known to the readers.



Coronary circulation is the circulation of blood in the blood vessels supplying the heart muscle (myocardium). The vessels that deliver oxygen-rich blood to the myocardium are known as **coronary arteries**. The vessels that remove the deoxygenated blood from the heart muscle are known as cardiac veins. These

include the great cardiac vein, the middle cardiac vein, the small cardiac vein and the anterior cardiac veins.

As the left and right coronary arteries run on the surface of the heart, they can be called as epicardial coronary arteries. These arteries, when healthy, are capable of auto regulation to maintain coronary blood flow at levels appropriate to the needs of the heart muscle. These relatively narrow vessels are commonly affected by atherosclerosis and can become blocked, causing angina or a heart attack. The coronary arteries that run deep within the myocardium are referred to as subendocardial.

The coronary arteries are classified as "end circulation", since they represent the only source of blood supply to the myocardium; there is very little redundant blood supply, which is why blockage of these vessels can be so critical.

The two coronary arteries originate from the left side of the heart at the beginning (root) of the aorta, just after the aorta exits the left ventricle. The left coronary artery originates from the left aortic sinus, while the right coronary artery originates from the right aortic sinus. No artery arises from the posterior aortic sinus. The left main coronary divides into **left anterior descending (LAD)** and **Left circumflex (LCx)** branches.

Coronary artery dominance

The artery that supplies the posterior descending artery (PDA) determines the coronary dominance. If the posterior descending artery is supplied by the right coronary artery (RCA), then the coronary circulation can be classified as "right-dominant". If the posterior descending artery is supplied by the circumflex artery (CX), a branch of the left artery, then the coronary circulation can be classified as "left-dominant". If the posterior descending

artery is supplied by both the right coronary artery and the circumflex artery, then the coronary circulation can be classified as "co-dominant".

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

The basic facts: The CAD is nowadays easily noted and treated at the rural level itself is a good development in the field of social and therapeutic cardiology. The city based Cardiologists and Cardiothoracic Surgeons' main job is to handle the cases of CAD with the recent armamentarium available namely angiography followed by angioplasty with stent namely PTCA or CABG. The work up of CAD patients in India is not perfect with the knowledge available at present is the observation which made me to pen this booklet. Because of this, the recurrent rate of symptoms and also mortality among post PTCA and CABG is possible and is a reality. To prevent such incidents, there are good number of scientific information available and need to be applied in practice is the theme of this booklet.

The important points missed during work up of CAD in India during follow up of cases of PTCA or CABG are identified during the work up as noted below. The missing links in the work up will be discussed then and there for the benefit of the reader.

A. Biochemical and clinical pathology investigations:

1. **Triglycerides:** Level of serum lipid profile is not given much importance even by higher centres specializing in this art. Mainly

the triglycerides which invariably is higher in cases of severe form of CAD is not given importance as in the case of total cholesterol. So, most of the time,the prescription is not containing drug for reducing or controlling the triglycerides. In my experience triglycerides increase, produces more damaging and dangerous type of CAD and also affecting major arteries of the body including the formation of Carotid plaques, producing block to the blood supply to brain even in non diabetics. Dyslipidemia is defined as any of the following:

- Serum total cholesterol ≥ 200 mg/dL.
- Serum LDL cholesterol ≥ 130 mg/dL.
- Serum HDL cholesterol < 40 mg/dL in men and <50 mg/dL in women.
- Serum triglycerides ≥ 150 mg/dL.
- Ratio of cholesterol to HDL cholesterol = 3.72 - 4.9. In my experience if it is above 5, there is a predilection of AMI at the earliest.
- Ratio of LDL cholesterol to HDL cholesterol =1.0 - 3.55

2. **HDL Cholesterol:** Most of our CAD patients are having normal level of lipids except low HDL was noted by me in 1995 itself and now it is a known fact in Indian scenario. With no drugs available for increasing HDL and only exercise being the option, a follow up vigilance is not done in these patients.

3. **Lipoprotein 'A'** and fibrinogen are not tested routinely in patients in India. Even in reangiography group, it is not done. My experience is that Lipoprotein' A' can be one of the reasons for reocclusion or new lesion in CAD. So, by estimating it and treating it with available nicotinic acid, a positive outcome can be expected. As an example, I have come across a lady of 42 years who developed AMI in her forties and treated with single stent. Later within two years, she developed two more occlusive vessels and done PTCA once again.

While this patient was worked up, she had a fourfold increase in Lipoprotein A and now after treating her increased Lipoprotein A, she is better and the stent patency is confirmed by CT Coronary Angiography recently after two years and no new lesions noted in other arteries.

4. **Fibrinogen:** I have got the opportunity to estimate the same from 1994 onwards in Thirunelveli. There was a drug available namely Bezafibrate and is not available at present. Even in USA, they are not testing fibrinogen because of the non availability of a drug for treating the same. I am using a medicinal plant powder drug from Siddha System of Medicine which acts well to reduce Fibrinogen. Recently, a patient who had high Fibrinogen level was treated using this drug and her stent is patent was made out using CT Coronary Angiography after four years of PTCA, with no other lesion in the arteries.

My another important observation related to fibrinogen is that in Indian patients even the level of 300 mg ,especially in diabetic patients produces more atherosclerotic events in the form of CAD or PVD.Normal value quoted is 200 to 400mg/100 ml.

Fibrinogen plays an important role in viscosity, and therefore blood flow, and also appears to play an important role in thrombus development and lysability. Fibrinogen is a protein that plays a key role in blood clotting. Fibrinogen is a sticky, fibrous coagulant in the blood that appears to significantly increase the risk of experiencing one of the leading causes of death and disability - stroke. High fibrinogen level is significantly associated with CAD and carotid atherosclerosis. The plasma level of fibrinogen is associated with the risk of ischaemic heart disease (IHD) and the severity of atherosclerosis. Patients with CAD tended to have higher fibrinogen levels than those without the disease. Of the patients whose fibrinogen levels fell within the two highest quartiles (>331 mg/dL), about 75% of men and 50% of women were diagnosed with

clinical CAD. A previous history of heart attack in the group with CAD was also associated with significantly higher average levels of fibrinogen. Fibrinogen was a strong and significant independent predictor of death from all causes in both men and women. The percent mortality rate jumped by over seven-fold in those with the highest fibrinogen levels, compared to those with the lowest levels. Fibrinogen's association with increased mortality is probably directly related to its ability to promote thromboses or clots, by causing platelets to clump inside blood vessels. This is one of the main mechanisms underlying ischemia and heart attack. Exercise, quitting smoking and certain medications have been shown to lower fibrinogen in the short term. Certain antioxidants are capable of dramatically reducing blood levels of lipid peroxides and oxidized lipoproteins.

5. **Platelets** play an important role in cardiovascular disease both in the pathogenesis of atherosclerosis and in the development of acute thrombotic events. Their importance in coronary disease and in acute coronary syndromes is indirectly confirmed by the benefit of antiplatelet agents (particularly aspirin, clopidogrel and the glycoprotein IIb/IIIa inhibitors) in these disorders.

In our observation, patients who have got increased platelet count in a routine investigation programme, have got more complicated CAD and in CAG, lesions of significance are noted.

The other biochemical markers like Uric acid, Homocystine and High Sensitive C-Reactive protein (high-sensitivity CRP (hs-CRP) are not estimated and included in analysis because this work up itself has given proper direction for prediction.

Five blood markers of CAD- Dr.S.Shanmugam's Formula of Prediction of CAD

Analysing one thousand CAGs investigated patients in a hospital, an important formula for the prediction of CAD which needs intervention in the form of PTCA or CABG in a particular

patient is created. I call this as "Dr.S.Shanmugam's Formula of Prediction of CAD". The following tests are mandatory in every patient to conclude and get into action.

1. Serum Lipid profile revealing two factors namely increase in **Triglycerides** and Low **HDL cholesterol** as factors of importance.
2. Increase in **Platelet count** more than 2.5 Lakhs.
3. Increased **Lipoprotein A** level.
4. Increased **Fibrinogen** level.

As per the formula if three factors out of these five factors are positive, then the CAD severity is high and will have to decide about CAG followed by PTCA or CABG.

B.Investigations pertaining to Cardiac work up:

1. ECG: This is a basic work up tool in the CAD analysis. It is one of the most important tools and is the oldest and most reliable tool for prediction and confirmation of CAD spectrum in bedside itself. It also gives us the data related to which portion of the heart is affected namely inferior, anteroseptal, lateral and basal of left ventricular infarction or right ventricular infarction including atrial infarctions. The electrical disturbances due to CAD can also be studied in ECG which may sometime produces adverse results due to life threatening arrhythmias. The atrial and ventricular enlargements and electrolyte imbalances due to treatment with diuretics especially potassium level changes and digoxin effect or toxicity can be gained out of this simple tool.

The present knowledge about ECG findings after doing CAG is that even in a case of severe occlusion in LMCA(Left main Coronary artery) namely 90% block , there may not be any ECG changes including the changes expected in aVR lead related to

LMCA. So, with the present protocol, ECG helps to rule out present changes especially AMI and predictive value for development of AMI is not specific.

2. X Ray Chest: Role of X-ray Chest in Cardiology- forgotten investigation

The development of investigations in cardiology started with X-Ray Chest (1895) and then ECG (1901). This is apart from clinical assessment of the said case. There was a time, after these two investigations, cardiac catheterization and Cine angiography were used till such time the non invasive Echocardiography comes to the centre stage of cardiac investigation with the further development of TEE and stress ECHO and so on. For every investigation, there is always some role play in the diagnosis of a disease or group of diseases.

Unfortunately nowadays most of the cardiologists believe that there is no need for taking X-Ray Chest in a case of cardiac complaint. The important information which can be derived out of taking X-Ray Chest before taking Echo is not well understood in Indian context nowadays is the observation made by me as a Diagnostic Cardiologist. The following information will be got only out of X-Ray Chest namely

1. The massiveness of Cardiomegaly can be appreciated as a wholesome only in X-Ray Chest which helps to report the Echo findings after verification- Which is not usually done and produces below average reporting in Echo most of the time. To decide which chambers of the heart are enlarged can be cross verified with the X-Ray findings- an added advantage for better diagnosis.
2. It will help to decide about the possible Pericardial Effusion effectively in Echo.
3. Most important thing is the status of the aorta and any aortic

dilatation and the etiology depending on the calcification in the aortic knob helps to decide the possible atherosclerosis as a cause.

4. The lung field findings namely the picture of acute pulmonary edema can be assessed by the X-Ray Chest than the Echo alone. Because in Echo if the EF is decided as low, usually the diagnosis is one of dilated or ischemic cardiomyopathy but here are conditions in which the EF is low but the heart functions are not that much affected as in the case of Non-compaction cardiomyopathy or spongiform cardiomyopathy.
5. The presence of pleural effusion after the procedures like PTCA and also CABG are well brought out only by X-Ray Chest. The Cardiothoracic Surgeons fortunately do regular X-Rays after surgery.
6. The pulmonary artery dilatation is well thought of only in X-Ray Chest and CT scan thorax or CT Angiography helps in confirming the diagnosis.

These are Cardiology related findings which make it mandatory taking of X-ray Chest in a case of cardiac complaint especially in CAD work up.

3. Echocardiography: The introduction of Echocardiography with colour doppler has revolutionized the practice of cardiology and CAD work up. It helps to decide about the regions of affection, cardiac function in the form of EF, thrombosis in any chambers and also any other affection like pericardial effusion due to massive infarction. It helps even to decide in an ECG negative case, the possibility of infarction in the bedside itself. When there is hypokinetic segment in Echo, it almost decides the CAD severity and there is no need for TMT test to assess the severity and ideal is to go for CAG investigation in a recent case of CAD. The Dobutamine stress Echo is not routinely done in most of the centres.

4. TMT: Tread mill test was considered as a golden test for ruling out CAD. But now with the arrival of CAG and also CT coronary angiography, the value of TMT in predicting CAD is still retained but with limitations. Negative result does not preclude CAD. Most of the time in the elderly people, because of the affection of knee joints due to Osteo arthritis, the test cannot be done.

5. Coronary Angiography (CAG): It is the golden test at present. The details expected related to CAG are

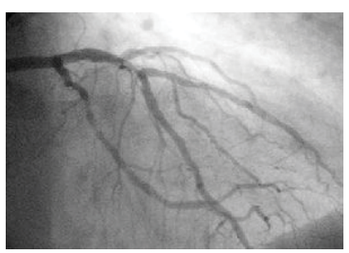
a. It should bring out the anatomy of all the three coronaries.
b. It should tell any calcification is present in fluoroscopic screening.
c. The functional aspect of the heart is to be reported-LV function.
d. The abnormal anatomy of the coronaries like ALCAPA (Anomalous origin of the left coronary artery arising from the pulmonary artery) is to be noted.
e. The milking effect in the case of myocardial bridging should be noted especially in cases of young people who developed AMI and also when there is a positive finding in Echocardiography.
f. For the purpose of doing CABG, LIMA (Left Inferior Mammary Artery) anatomy should be brought out in every CAG.
g. The renal arteries should be studied as a routine to identify any defect in the arteries especially in severe hypertension associated with CAD.
h. If possible and suspected, the aortic anatomy to rule out the aortic aneurism.
i. The carotids may be possible to study if needed.

Unfortunately, most of the time the CAG done in our centres need more improvement in clarity and also with the above

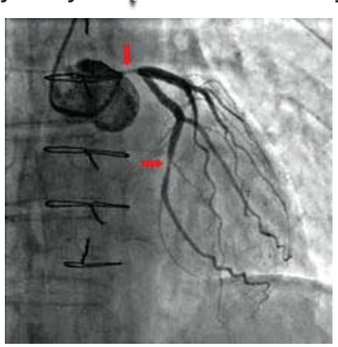
information availability is the observation of the author after perusing 1000 CAGs in a span of four years with various centres doing CAG for the patients.

The important observation, I have made is related to collaterals in CAG. If there is a method by which the collaterals helping as natural CABG is worked out , lot of cases of CABG as well as PTCA can be avoided in more than 30 % of cases of CAD. The end circulation concept needs revision in the wake of collaterals from one artery to another. If a methodology is created which can quantify the occlusion effectively with the available CAG and not with Fractional flow reserve (FFR) CAG which is not available in all the centres, it will be a good decision making approach.

A coronary angiogram showing the left coronary circulation.



Coronary angiography of a critical sub-occlusion of the common **trunk of the left coronary artery and the circumflex artery. (See arrows)**



6. CT Coronary Angiography (CTCA): An important tool in the cardiac work up of CAD, it is not accepted widely by Cardiologists especially in India. The fact should be understood that if CAG is like a video of an individual to be assessed for marriage, the CT Coronary angiography is like a photo. Both have got important roles to play to assess. The same is applicable in CAD work up. Some centres routinely do both. But it is really useful in certain areas of work up is my finding.

1. In ALCAPA it is the best investigation for correct diagnosis.
2. In Myocardial bridging especially when suspected milking effect is noted in CAG.
3. In Spongiform Cardiomyopathy when Cardiac MRI is not available it helps to diagnose the case.
4. Calcium score (CACS) helps to predict the CAD and is routinely used in Western centres of Cardiology.

A computerized tomography (CT) coronary angiogram is an imaging test that looks at the arteries that supply heart with blood. CT angiograms do not use a catheter threaded through blood vessels to heart. CT coronary angiograms are noninvasive and an option for people who have only a moderate risk of coronary artery disease. This test uses a special type of computerized tomography to check for calcium in coronary arteries, which can be a risk factor for coronary artery disease. Calcium score above 400 is considered as predictive of severe CAD.

The ideal cardiac work up for CAD should include CT Coronary Angiography followed by CAG.The Anatomy will be well brought out by CTCA and Physiology by CAG.The exercise will be cost effective considering the best decision making in the long run.

7. Thallium stress test is a nuclear imaging test that shows how well blood flows into the heart during exercise and at rest. A **radioisotope** (nuclear material) is administered intravenously. It settles into the heart muscle and pinpoints spots that are abnormal. The thallium stress test can show:

1. The size of the heart chambers

2. Ventricular function

3. Myocardial perfusion

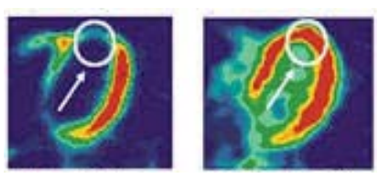
4. Scarring of the heart muscle from previous infarction.

This is used to decide whether CABG or PTCA will be useful in case if it is needed.

8. MRI Scan is nowadays used in some centres to assess the scarring of the cardiac muscles before deciding about the usefulness of CABG or PTCA.

9.Cardiac PET Study

A PET study is a diagnostic test used to evaluate blood flow to the heart. During the test, a small amount of radioactive tracer is injected into a vein. A special camera, called a gamma camera, detects the radiation released by the tracer to produce computer images of the heart. Combined with a medication, the test can help determine if there is adequate blood flow to the heart during activity versus at rest. The medication simulates exercise for patients unable to exercise on a treadmill or stationary cycle.



No Metabolism

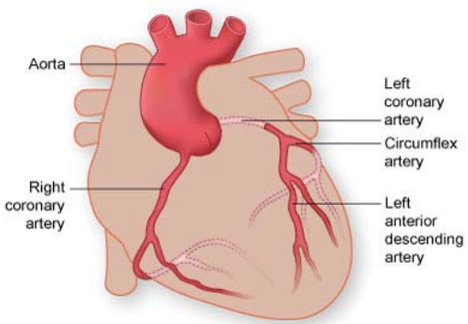
Metabolic activity

The test is also used to evaluate the amount of damage to heart after a heart attack and also evaluate the effectiveness of cardiac treatment plan.

These steps help to decide about the Cardiac work up effectively and scrupulously in a given case of CAD. Following that CABG or PTCA counselling is needed depending on the necessity.

Let us see the rare causes of CAD presentation which are not as uncommon as seen by me in a series of one thousand cases of CAG review.

1. Myocardial Bridge



A myocardial bridge is a band of heart muscle that lies on top of a coronary artery, instead of underneath it. With a myocardial bridge, part of a coronary artery dips into and underneath the heart muscle and then comes back out again. The band of muscle that lies on top of the coronary artery is called a “bridge,” and this is how the condition gets its name. For the most part, a myocardial bridge is harmless. Patients with a myocardial bridge will have had it since birth, and most patients live their whole lives without ever knowing they have the condition. However, some patients can develop myocardial ischemia because of a myocardial bridge. Myocardial ischemia means that the heart muscle is not getting enough oxygen-rich blood. When the heart “squeezes” (contracts) during the heartbeat, the bridge of heart muscle can “tighten down” on the artery, pinching it and decreasing the blood flow. Luckily, most of the blood flow to the heart happens during the “rest” phase of the heartbeat, not during the “squeezing” phase. Even so, the

tightening of the bridge on the artery can decrease blood flow to the extent that myocardial ischemia develops, especially during exercise or when the heart is beating quickly. Up to one-third of patients with a myocardial bridge do not have any symptoms, even if their hearts are not getting enough blood supply. If patients do have symptoms, they are most likely caused by the myocardial ischemia. Symptoms may include

- Chest pain
- Tightness in the chest or a feeling of pressure or heaviness on the chest
- Pain in the left arm or jaw
- Shortness of breath
- Fatigue

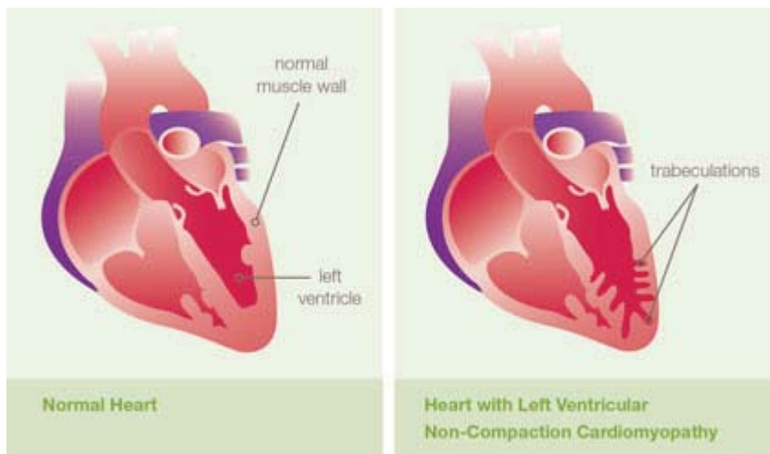
It is best diagnosed by CAG by identifying "milking effect" or CT Coronary Angiography. In most patients, a myocardial bridge is not treated if it is not causing any symptoms. In patients with symptoms, medicines such as beta-blockers and calcium channel blockers are usually the first line of treatment. In rare cases, patients need surgery to relieve their symptoms. Surgery involves removing the bridge that is pressing on the coronary artery.

2. Non-compaction cardiomyopathy (NCC), also called as spongiform cardiomyopathy, is a rare congenital cardiomyopathy that affects both children and adults. It results from the failure of myocardial development during embryogenesis. Non compaction cardiomyopathy was first identified as an isolated condition in 1984 by Engberding and Benber. They reported on a 33 year old female presenting with exertional dyspnea and palpitations. Investigations concluded persistence of myocardial sinusoids (now termed non compaction).Common symptoms associated with a reduced pumping performance of the heart includes:

- Breathlessness
- Fatigue
- Swelling of the ankles
- Limited physical capacity and exercise intolerance

Due to its recent establishment as a diagnosis, and it being unclassified as a cardiomyopathy according to the WHO, it is not fully understood how common the condition is. Some reports suggest that it is in the order of 0.12 cases per 100,000.

This can be diagnosed with Echo followed by Cardiac MRI. CT Coronary Angiography helps in the diagnosis and I have diagnosed two cases in our work up using CT Coronary angiography after CAG being reported as normal.



Treatment schedule in long term follow up after PTCA or CABG or medical management:

The basic points to be remembered while prescribing in the above situations are not regularized in the world scenario. I have got a five points charter for prescription in these cases with scientific base.

1. Antiplate drugs namely Clopidogrel and or Aspirin in adequate doses depending on the situation along with H_2 receptor antagonist or PPI to prevent long term gastric problems. Newer version of antiplatelets like Prasugrel and Ticagrelor can be used depending on the necessity.
2. Depending on the heart rate, the rate limiting drug namely beta blocker preferably Metoprolol 25 to 50 mg twice daily only. No thrice daily as seen in number of prescriptions which are not the accepted schedule. Nebivolol can also be used as a once daily dosage. Ivabradine (Coralan) is nowadays used for the same purpose. In case of increase in heart rate needed , amlodepine can be used for associated hypertension.
3. The ACE inhibitor or ARB for cardiac remodeling as per the necessity like Ramipril or Perindopril (Coversyl).
4. The anti cholesterol and or antitriglyceride drugs commonly atorvastatin or resuvastatin and fenofibrate. The role of nicotinic acid in reducing Lipoprotein A is to be utilized whenever needed. No specific drug available for Fibrinogen reduction except a research drug from Siddha medicine which I am working out and found it useful in my patients and need a larger trial.
5. Nicorandil, Trimetazidine, nitrate drugs, Ranolazine and anti failure and anti arrhythmic drugs depending on the situation.

By following this five pronged strategy, most of the time patient's cardiac status can be well maintained. This is apart from

treatment for Diabetes Mellitus and Hypertension and any other systemic diseases, the patient may be suffering.

Post CABG and PTCA follow up clinic: Ideal way of follow up of post CABG and PTCA patients are yet to be established in Indian Cardiac centres including corporate hospitals is my observation. The follow up can be done by Physician trained in Cardiology or Cardiologist, so that the time factor for meeting Cardiothoracic Surgeon in Indian scenario can be avoided and also good counseling for regular activities, bowel movements to avoid strain to the heart and also family life resumption etc.The patients, instead of being given pamphlets in English regarding follow up instruction or in vernacular language, DVD or VCD to highlight the follow up in vernacular language will help even the uneducated and village people.

Preventive methodology:

The following factors are playing a major role to play in the development of CAD and effective steps related to modifying their role play helps in the prevention of CAD. The factors are not elaborated and only enumerated due to the basic topic being cardiac work up. This is apart from the factors described in detail above.

Ten Commandments for prevention of Myocardial Infarction:

1. Smoking
2. Alcohol
3. High cholesterol levels, High LDL Cholesterol level (High Triglycerides level and low HDL cholesterol level)
4. Diabetes Mellitus
5. Hypertension
6. Obesity
7. Unhealthy food habits-eating high fatty and salty foods
8. Physical inactivity
9. Mental Stress
10. Genetics-Family History

All these factors need modification for avoiding CAD.

Advice to patients:

Cessation of smoking and abstinence from alcohol is a must for avoiding CAD and also after CABG or PTCA. Persons with these two habits developing AMI between 30 to 40 years is my regular observation. Control of Diabetes, Hypertension and dyslipaedemia can be done with the armamentarium of excellent drugs available today. Obesity is to be prevented among children and to be treated vigorously in adults. Avoid unhealthy food habits and do physical activity in the form of walking for 30 minutes at least five days a week. Mental stress can be reduced by proper planning of daily activities as well as doing pranayama, meditation and if possible yogasanas. Genetics cannot be modified presently but can be conquered in the case of CAD by following other commandments. Have a heart attack free life by following these things regularly.

The use of Palm oil for cooking purposes in India especially people who are not doing manual work, the risk of CAD is more, is my observation. The same way people who eat the meat of cows are also more prone even in younger age group and that too in women is a notable information for the prevention of CAD in Indian Context.

Conclusion:

A book can be written on the same and I have made it as simple and practical for easy reading and follow up by every medical practitioner irrespective of their qualification to do the best for the dreaded and dangerous disease of the century not to play havoc with the life of human race. A seminar module to highlight this information to the Medical practitioners and Physicians is available and can be utilized by those who are interested.

Newer Research Findings in 2015:

1. Right Coronary Artery and development of AMI

The three coronary arteries are important for the blood supply to the heart. LMCA is the life line of heart circulation. If it is affected, the possibility of sudden cardiac arrest and the sudden death is a well known phenomenon in cardiology. While going through more than 1000 coronary angiograms, it has been noted that whenever there is a small RCA, there is a definite clinical presentation of CAHD in the form of AMI- non fatal or fatal depending on the affection of the other coronary arteries. When the RCA is normal in size or bigger, in spite of some block in the other vessels, the clinical presentation will be minimal. The deciding factor for the clinical presentation in most of the patients with CAHD is related to RCA size as well as pathology, is the new finding, I have noted while going through patients' CAG. So, my hypothesis is whenever RCA is smaller or diseased , there should be effective ways of preventing AMI in that particular patient. This has got a predictive value related to AMI. It is new information in Cardiology and especially related to AMI.

2. Markers of CAHD-Dr.S.Shanmugam's Theory of predictability of CAHD

In the biochemical prediction of CAHD and its severity, there are two formulas have been found out as new. Apart from the formula of five which is described in this book already, the first formula - there is another combination noted as the experience in CAHD grows, namely the combination of increase in Total

cholesterol, Triglycerides and LDL cholesterol when combined, the chances of high risk CAHD is noted in all the patients dealt. The second formula is already there namely low HDL level. What is surprising is, that may be the only finding and usually when the level of HDL is lower than 30 and the CAHD severity is more in spite of every other parameter of predictive value is normal.

So, we have got three formulas namely

1. Dr.S.Shanmugam's formula of prediction of CAD using five blood markers namely 1.increase in Triglycerides 2. Low HDL Cholesterol 3. Increase in platelets 4. Increase in Lipoprotein A and 5. Increase in Fibrinogen- Pancha predictability
2. Increase in Total cholesterol, Triglycerides and LDL cholesterol- Triad predictability
3. Low HDL level- Unique predictability.

Any one of the above three criteria will predict the severity of CAD. So, by doing the above blood markers one can predict the possibility of severity of CAD and in turn developing AMI-severe or moderate or mild. This will be useful even in cases of TMT negative chest pain.

3. Dissection as a cause of CAHD is rare but is noted with only four patients out of the one thousand CAG studies analyzed. Only males were noted and not females as suggested in the literature.

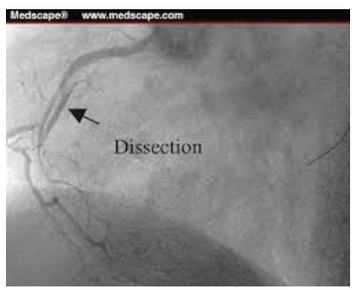
A **coronary artery dissection** (also known as **spontaneous coronary artery dissection** or **SCAD**) is a rare, sometimes fatal traumatic condition, with eighty percent of cases affecting women. The coronary artery develops a tear, causing blood to flow between the layers which forces them apart. Early studies of the disease placed mortality rates at around 70% but more recent data indicate this figure may be closer to 18%. The symptoms are often very similar to those of myocardial infarction (heart attack), with the most common being persistent chest pain.

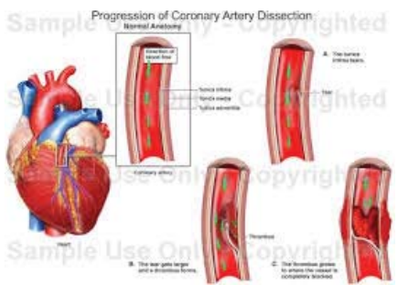
There is evidence to suggest that a major cause of spontaneous coronary artery dissection (SCAD) is related to female hormone levels, as most cases appear to arise in pre-menopausal women, although there is evidence that the condition can have various triggers. Other underlying conditions such as hypertension, recent delivery of a baby, fibromuscular dysplasia and connective-tissue disorders (e.g.,Marfan syndrome and Ehlers-Danlos syndrome) may occasionally result in SCAD. There is also a possibility that vigorous exercise can be a trigger. However, many cases have no obvious cause. Sometimes while doing PTCA, patient may develop dissection which was noted in one of our cases.

Coronary artery dissection results from a tear in the inner layer of the artery, the tunica intima. This allows blood to penetrate and cause an intramural hematoma in the central layer, the tunica media, and a restriction in the size of the lumen, resulting in reduced blood flow which in turn causes myocardial infarction and can later cause sudden cardiac death.

A selective coronary angiogram is the most common method to diagnose the condition, although it is sometimes not recognised until after death. Intravascular ultrasound (IVUS) is also used as it is able to more easily differentiate the condition from atherosclerotic disease.

Treatment is varied depending upon the nature of the case. In severe cases, coronary artery bypass surgery is performed to redirect blood flow around the affected area and thrombolytic drug therapy are less invasive options for less severe cases.





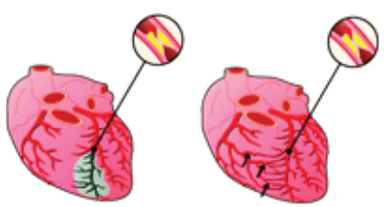
4. In Myocardial bridging, because of the shearing effect due to pressing in the particular area, there is a possibility of development of thrombosis in that area and so AMI can develop. The mechanism of development of AMI in myocardial bridging is not out of pressing but out of shearing effect is made out by me.

In a case of 17 year old boy, initially he presented with VPCs. He was worked out with TMT also. No positive finding. But patient developed full blown AMI within a month. Whether TMT has increased the shearing effect in the vessel in question is to be answered.

5. So, in young people presenting with VPCs, think about the possibility of myocardial bridging. By doing CT Angiography it is possible to help the patient earlier without going for AMI as in the case above quoted.

6. Role of collaterals- Natural CABG

The decision regarding the treatment whether urgent PTCA or CABG is required or the procedures are required later or not at all can be made by observing an important component in CAG namely development of collaterals. This particular so called natural bypass surgery equivalent is not taken at the moment by any cardiologist or cardiothoracic surgeon throughout the world. After analyzing cases when there is a total cut off of the vessels, the patient has not developed any urgent symptomatology because of the development of collaterals noted in CAG is the observation which will be very much useful in developing countries like India where the resource is limited and also when there is no possibility of going for the procedures because of lack of money or physical restriction or viability question related to myocardium. This observation can be used to develop drugs which will help to develop natural collaterals to help the people who cannot undergo CABG.



In response to endurance exercise training (such as running, bicycling, swimming, and hiking), blood flow is increased, which leads to a conversion from capillaries into collaterals.

Dr.S.Shanmugam's contribution to Cardiology

Special mention:

Teaching stethoscope: Using two sets of earpieces, a stethoscope for teaching purpose was created in 1983 and published in JAPI in December 1986 as "Teaching Stethoscope- Revisited". Presented as an award session paper in Dr.D.P.Basu award for Cardiology-APICON 87 at Madurai. The first person in the world to coin the word **"Teaching Stethoscope".**

Desk Top Echocardiography- a book under preparation.

International trends in Hypertension –Commentator on articles –France (2000&2001)-one among four Indians, only one from South India.

Travel grant : 15th International Society of Hypertension meeting, Melbourne, Australia [1994].

19th Annual Conference of Japanese Society of Hypertension-Japan -1999.

Mitral Valve Prolapse Syndrome:

Association with Hypertension-Sixth European Congress on Hypertension, Milan, Italy-1993-abstract published; Sudden death among athletes- 2000 Pre-Olympic Medical congress - Australia-2000-paper accepted for presentation.

Carotid Hypertension: a new form of hypertension - Fifth European Congress on hypertension - 1992 - paper sent.

International Conferences attended : World Congress on Coronary Heart diseases- Mumbai-1990- Ildamen (Oxyfederine) –Poor man's streptokinase;MSD Award of International society of Hypertension [1994] - Member of International Society of Hypertension-one among the five Indians;15th Scientific Meeting of International Society of Hypertension at Melbourne, Australia- March 1994;9th International Conference on Hypertension in Pregnancy-Sydney, Australia-March 1994-Eclampsia- a theory;19th Annual Conference of Japanese Society of Hypertension-Japan -1999-paper accepted for presentation.

Author of "Heart Attack prevention" in Tamil for public-2004.

Research in PTCA or CABG prevention:

Using a siddha drug, a trial is on related to avoiding CABG or PTCA and also to prevent further progression of atherosclerosis in CABG or PTCA undergone patients who are TMT positive.

Dr·S·Shanmugam's contribution

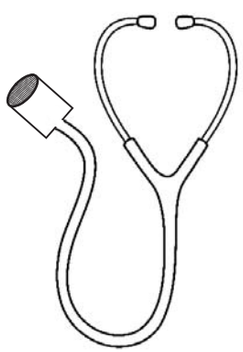
On July 22, 1997 ,with patent no:5,650,598 an United States Patent,in which Dr.S.Shanmugam's article was mentioned in the nonpatent citations as " Shanmugam.S.Teaching Stethoscope revisited.J.A.P.I;34.892.1986.This is a recognition for this research at the international level.

ARTERIAL STETHOSCOPE

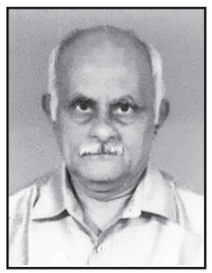
(NEURO DIABETOLOGY STETHOSCOPE)

New Invention? Discovery?

On October 2, 2015, a new idea of developing an auscultatory method of arteries specially carotid arteries auscultation which is commonly used in neurology practice as well as auscultation of peripheral arteries which are very much relevant in Diabetology practice has been developed. The instrument is ready and will be applied for patent. It is the first of its kind in medical auscultation. This will help to identify the narrowing and or occlusion of the arteries at the bed side and will make the investigation using Doppler study better.



Dr.S.Shanmugam, M.D., PGDHSc (Echocardiography),
P.G.P in Diabetology (Johns Hopkins University, U.S.A),
Diagnostic, Preventive and Echo Cardiologist & Diabetologist,
Researcher in New Methodologies in Medicine,
Geeyes Diabetes and Hypertension Research Centre,
Research Centre for Undiagnosed & Rare Diseases,
Thirunelveli,Tamil Nadu, India.



Dear Doctor,

As a learned person it is your duty to look after your life's longevity by taking care of your heart's health. You would have come across info on even a Cardiothoracic Surgeon had sudden death due to LMCA block at the age of 63 years recently. This sort of events can be predicted earlier so that anybody can avoid such situation which will be a loss to the family as well as society.

To have prediction of heart related events especially Acute myocardial infarction, I have developed a theory which is described in this Coronary Heart Disease Workup-2015 and will be useful to you personally as well as to your near and dear and patients. Use these proven formulas and make your heart healthy. If you need more details on the same please contact me.

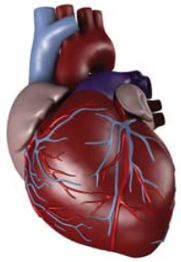
Have a long happy life with healthy heart.

Best wishes

Dr.S.Shanmugam,M.D

Research centre
for undiagnosed and rare diseases.

Post PTCA and Post CABG
follow up clinic.



Dr.S.Shanmugam, M.D., PGDHSc (Echocardiography),
P.G.P in Diabetology (Johns Hopkins University, U.S.A),
Diagnostic, Preventive and Echo Cardiologist & Diabetologist,
Researcher in New Methodologies in Medicine,
Geeyes Diabetes and Hypertension Research Centre,
Research Centre for Undiagnosed & Rare Diseases,
1, Bharathi Street, S.T.C.Road,
N,G.O.'A'.Colony,
Thirunelveli - 627 007,
Tamil Nadu, India.
Cell: 9843009655
Email: shanbhatir@gmail.com