THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU.

DISSERTATION

ON

"SIGNIFICANCE OF ACTIVITY AND TIME OF OCCURANCE

IN STROKE AND EVALUATION OF RISK FACTORS"

SUBMITTED FOR M.D. DEGREE EXAMINATION

BRANCH I

(GENERAL MEDICINE)

EXAMINATION

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TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

CERTIFICATE

This is to certify that this dissertation entitled **"SIGNIFICANCE OF ACTIVITY AND TIME OF OCCURANCE IN STROKE AND EVALUATION OF RISK FACTORS"** is the bonafide record work done by **Dr. S.JEFF REDLEENE,** submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in APRIL 2013.

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Certificate of Approval

This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. S.JEFF REDLEENE, a **POSTGRADUATE IN GENERAL MEDICINE** in the Department of **GENERAL MEDICINE**, of Tirunelveli Medical College /Hospital, Tirunelveli titled **"SIGNIFICANCE OF ACTIVITY AND TIME OF OCCURANCE IN STROKE AND EVALUATION OF RISK FACTORS"** registered by the IEC as 114/G.M/IEC/2011 dated. 12.8.2011. The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.

Issued on this Date

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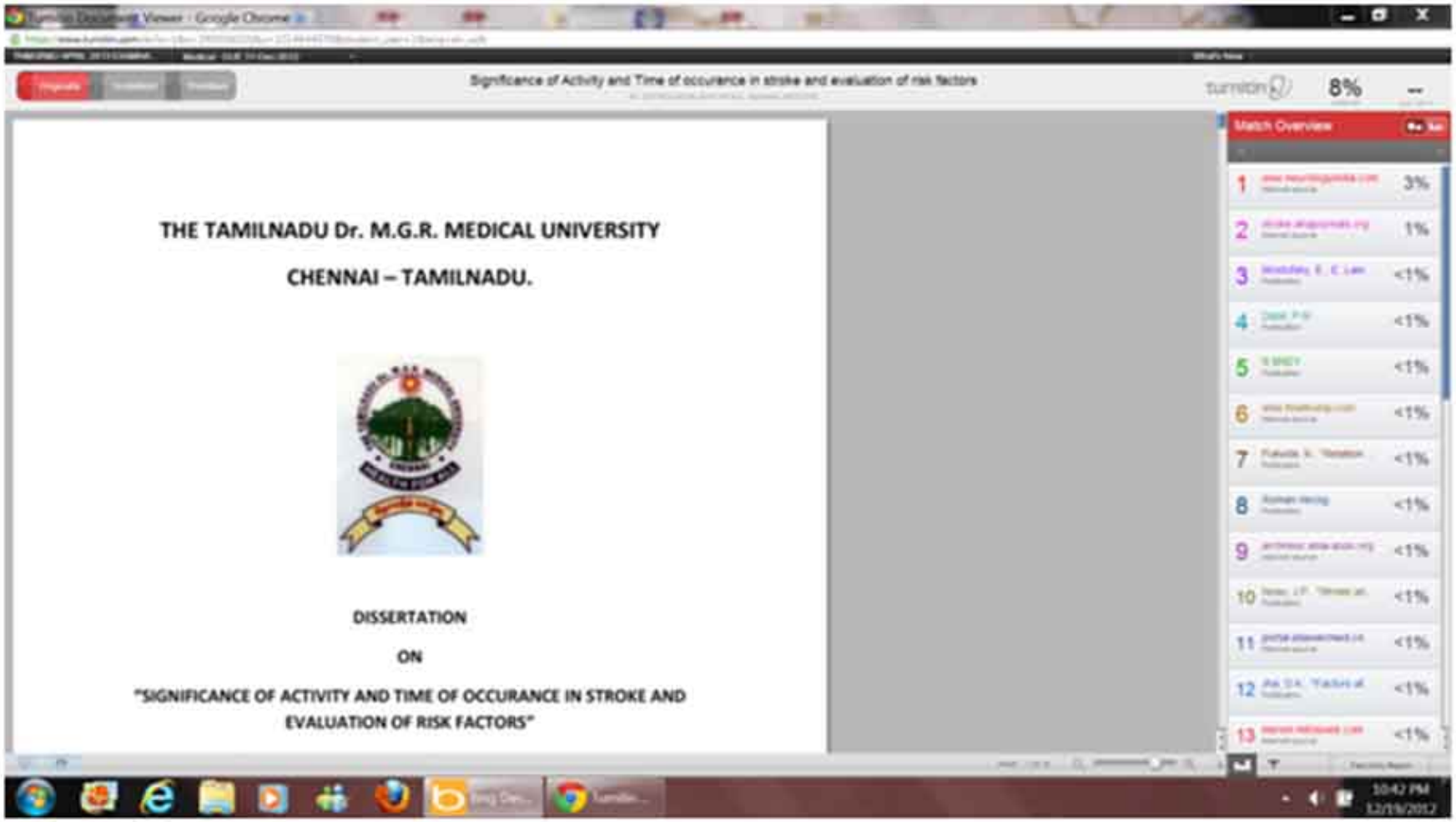
Significance of Activity and Time of occurance in stroke and evaluation of risk factors
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DECLARATION

I**, DR. S.JEFF REDLEENE** , solemnly declare that dissertation titled **"SIGNIFICANCE OF ACTIVITY AND TIME OF OCCURANCE IN STROKE AND EVALUATION OF RISK FACTORS"** is a bona fide work done by me at Govt. Tirunelveli Medical College and Hospital from September 2011 To November 2012 under the guidance and supervision of my unit chief **PROF.Dr.S.ALAGESAN.M.D., D.M (NEURO)** , Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch-I) in GENERAL MEDICINE.**

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CONTENTS

Introduction

INTRODUCTION

Stroke is one of the most common neurological disorders in clinical practice and causing death in the developing countries as well as developed countries.[1]Stroke is an acute neurological injury occurring as a result of vascular pathological processes which manifest either as brain infarction or hemorrhage. Risk factors do not explain the timing and activity in the occurrence of stroke. But they are helpful to prevent the onset of stroke by reducing the risk factors.

There are studies which reveal that physical activity has role in decreasing the Coronary events but only few for stroke. The present study has been designed to know whether time and activity has any significance in onset of the stroke and to evaluate the risk factors like alcoholism, smoking, Diabetes mellitus, CAHD, Systemic Hypertension, Dyslipidemia triggering the stroke.

Aim of the study

AIM OF THE STUDY

- To find the Significance of Time in the occurrence of the stroke .
- To find the Significance of activity in the occurrence of stroke.
- To evaluate the Risk factors associated with the stroke

Review of literature

REVIEW OF LITERATURE

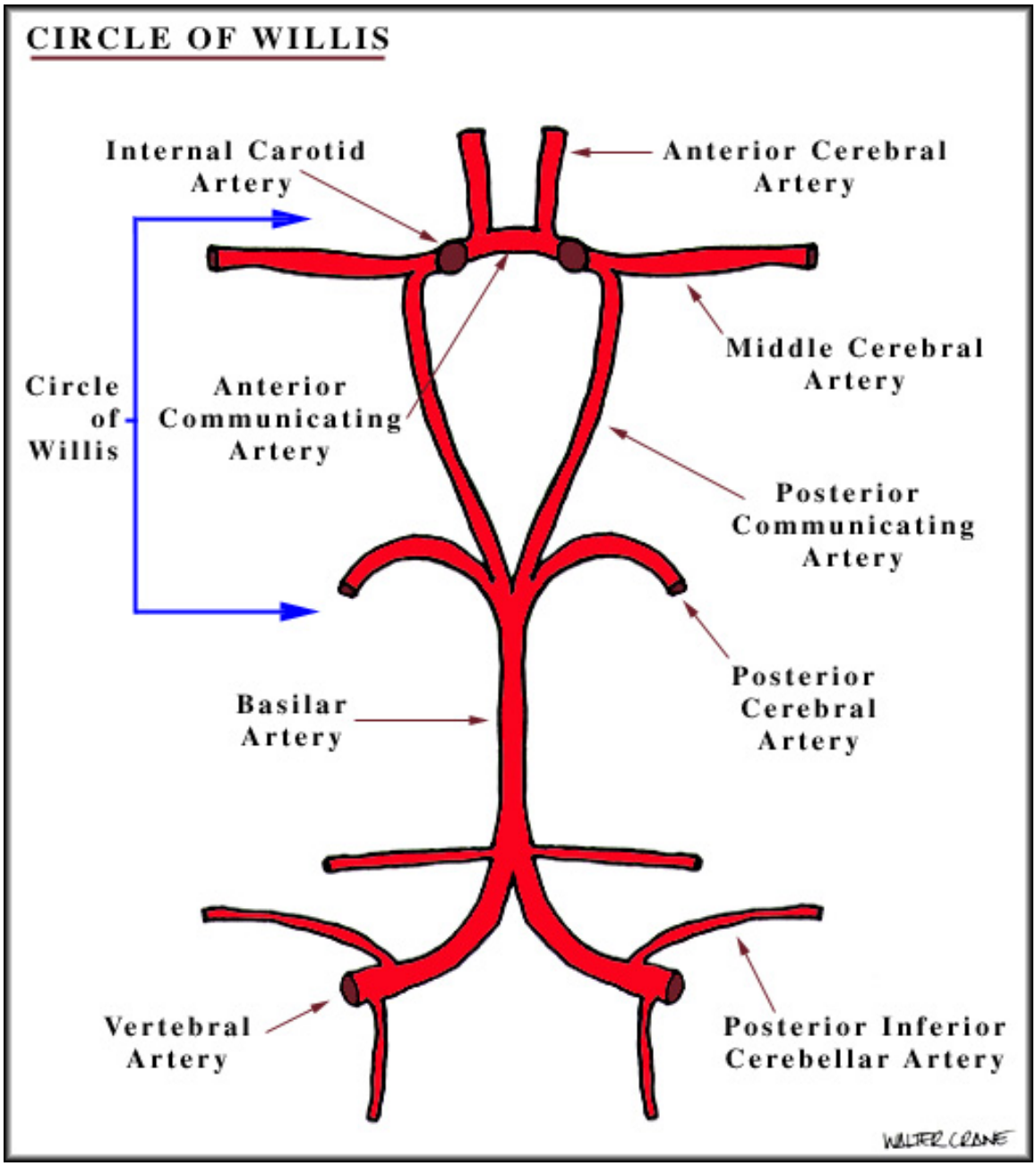
The normal function of the brain is dependent upon a relatively constant supply of the glucose and oxygen, which is derived from the blood which perfuses it i.e. 55 to 70 ml of blood per 100 gram of brain per minute. The principal source of the energy is almostexclusively due to the oxidation of the glucose. When the blood flow to the brain is critically reduced below 15 ml per 100 g per min, the resulting ischaemia which is associated with the hypoxia, when sufficiently prolonged, may cause death of neurons and glia resulting in the cerebral infarction. The blood supply to the cerebral cortex is via the anterior, middle and posterior cerebral arteries.

The mean arterial blood pressure, cerebrovascular tissue resistance, local metabolic products i.e.pH, PaO2, PaCO2, etc, when together with several known and unknown factors, helps to maintain the critical threshold of blood flow for the metabolism of energy. And also the blood flow varies in different areas of the brain and a self regulatory mechanism i.e. the auto-regulation determines the regional flow to meet local metabolic needs.

In the regions of the cerebral ischaemia, there is loss of the autoregulation and the microvasculature becomes non-reactive to the pressure changes, to the vasoactive agents and also to the other forms of the stimuli. The cerebral vasculature in these ischaemic zones becomes

permeable to the proteins and the fluid leaking in the vicinity resulting in the extracellular cerebral oedema. Such vascular events can also lead to the local haemoconcentration and the vascular stasis.

There are some mechanism for protecting the brain from the ischemia. One such way is by the several collateral pathways which exists. The vertebral arteries and the four major extracranial arteries forms a good calibre, low resistance anastomosis which is the circle of Willis. In addition to it, extracranial anastomosis exists between the cervical branches of the ipsilateral external carotid, subclavian and vertebral arteries. These arterial anastomosis helps to maintain the cerebral blood supply even with the occlusion of major arteries and various cerbellar arteries. These post-Willisian anastomosis further protect cerebral tissue from the effects of occlusion of single cortical branches.


CIRCLE OF WILLIS
Internal Carotid Artery
Anterior Cerebral Artery
Middle Cerebral Artery
Circle of Willis
Anterior Communicating Artery
Posterior Communicating Artery
Posterior Cerebral Artery
Basilar Artery
Vertebral Artery
Posterior Inferior Cerebellar Artery
WALTER CRANE

In the presence of generalised arterial disease like atherosclerosis, congenital variations and also in the presence of multiple skipped stenotic lesions, these collateral pathways may prove inadequate and they will predispose to the cerebral ischaemia or infarction.

When for any reasons like, such as cardiac arrest or prolonged hypotension (systolic BP below 70 mmHg), the brain tissue will be significantly deprived of its nutrition for more than three minutes, cerebral infarction will occur. Such infarcts will either be pale (ischaemic infarction in thrombosis) or it may show petechial haemorrhages in the cortical mantle (haemorrhagic infarction in embolism). The sylvian region is commonly involved in middle cerebral artery occlusion (mid-field infarct), whereas with internal carotid artery lesions, the cerebral infarction is mostly located in the distal water territory (end-field infarction).

A mid-field infarct is usually produced by the occlusion of a small penetrating vessel, and if located in the territory of a major anatomical pathway, it may prove catastrophic. An end-field infarct resulting from occlusion of a major vessel in the neck with good collateral circulation may be asymptomatic. Obstruction to venous return can also result in haemorrhagic infarction (e.g. cerebral venous thrombosis).

Acutely infarcted brain tissue is always soft and swollen, it frequently herniates downwards and it may compress the vital centres

within the brainstem, ending in a fatal outcome. Like infarction elsewhere in the body, cerebral infarct also heals by fibrosis. It is frequently replaced by a firm scar or a cystic cavity.

Cerebral infarction is mainly attributed to the partial or total occlusion of its regional microvasculature by thromboembolism. Cerebral atheroma is by far the most common underlying intimal vascular pathology. Thrombosis associated with arteritis like syphilitic, tuberculous, etc. is not an uncommon aetiology for the strokes in the young patients in India.

Recent studies have shown that there is a dense central core, surrounded by a less dense zone of ischaemia which is called as penumbra, and the neuronal death occurs in this focus unless perfusion is quickly restored to it. On the other hand, when the cells in the zone of penumbra remain viable for at least three hours i.e. the therapeutic window, then it can be salvaged by reperfusion with or without neuroprotective agents. Major factors which will enhance the nerve cell injury are, an increase in the intracellular cytosolic calcium concentration from the failure of ionic pump functions or leaks, and the changes in Na+/K+ gradients, acidosis, the release of free radicals, and some other unknown factors, which in turn disrupt the blood-brain barrier (BBB) and also the microvascular function. Here, energy depletion from brain hypoxia is one of the key events that fails to maintain normal

concentrations of cellular adenosine triphosphate (ATP), which leads to the delay in resynthesis of macromolecular proteins, which is essential for the cell structure. Such energy failures will also induce proteolysis and lipolysis in addition to production of arachidonic acid and platelet activating factors, free radicals, etc., thus resulting in further neuronal damage i.e. the ischaemic cascade. Thus, the severity of cerebral infarct is not the mere result of ischaemia from occluded vessels but the end-result of several highly complex ischaemia-modifying factors.

Internal carotid artery syndrome can occur in the thrombotic stroke. The common site for thrombotic occlusion is the cervical portion of the internal carotid artery near the carotid sinus. Almost 1/3rd of the occlusive lesions are located in this segment. Most of these lesions can be silent or asymptomatic because enough blood reach the affected territory via the external carotid ophthalmic anastomosis or from the superficial and deep cervical anastomoses or from the opposite carotid artery through the anterior segment of the circle of Willis.

In the patients with symptomatic carotid thrombosis, warning symptoms usually will occur before the main insult in majority of the patients. These patients present with the symptoms of brief episodes of confusion, difficulty with speech which can be aphasia or dysarthria or dyslexia and even sensory paraesthesia, with or without motor weakness of the opposite side. Ipsilateral (same side) transient monocular blindness

(amaurosis fugax), fleeting or semipermanent, alternating with or can be accompanied by a contralateral hemiplegia or asensory deficit, is the pathognomonic of carotid artery syndrome.

The patients with acute carotid occlusion present in the similar fashion as the patients with the middle cerebral syndrome. On examination of these patients , some features like the feeble pulsations of the internal carotid artery or superficial temporal artery, dilated pupil and poorly pulsating retinal vessels with or without optic atrophy on the side of the suspected carotid lesion, and ocular or cervical bruits on the ipsilateral side, may suggest the correct diagnosis. Carotid Doppler sonography and angiography are helpful in the diagnosis and also to find the degree of stenosis and also to determine the collateral flow.

The patients with an old or silent occlusive carotid artery lesion on one side, with a new lesion on the oppostie side may prove catastrophic. Here, the patients present with the physical finding of bilateral hemiplegia (quadriplegia) with coma which can even be mistaken for basilar artery syndrome.

Major Risk factors for stroke are

- Old age
- Obesity
- Hypertension
- Smoking
- Diabetes mellitus
- Atrial fibrillation
- Heart disease
- Dyslipidemia
- Hyperfibrinogenemia
- Alcohol
- Coagulopathy
- Contraceptive pill
- Markers of arterial atheroma (TIA, Angina, Claudication, Bruit)

  Old age is very important risk factor . As age advances the risk of stroke increases . Male Hypertensive old patients are more vulnerable than female patients. All the risk factors usually accelerates the atherosclerosis and then causing thrombosis and stroke.

Normal BP	**<120mmHg/<80mmHg**
Pre Hypertension	120-139mmHg/80-89mmHg
Stage I hypertension	140-159mmHg/90-99mmHg
Stage II hypertension	>160/>100mmHg
Isolated hypertension	>140mmHg / <90 mmHg

Hypertension is a modifiable risk factor in stroke. It usually accelerates atherosclerosis and causes stroke. Isolated systolic hypertension is much more dangerous risk factor. BP control helps to prevent the recurrent stroke. Blood pressure treatment, resulting in a reduction in SBP of 10-12 mm Hg and *5-6* mm Hg diastolic, is associated with a 38% reduction in stroke incidence.[2] When hypertension is controlled it reduces the atherosclerosis progression thus reducing the progression rate of stenosis of major vessels.

Diabetes mellitus which is Fasting blood sugar >126 mg% and PP>200mg% or RBS>200 mg% with symptoms of diabetes is also an important risk factor in stroke. Diabetes can cause acute complications, microvascular complications and macrovascular complications. Macrovascular complications are stroke, coronary artery disease and peripheral artery disease. When diabetes is present with hypertension, it increases the more chances of Coronary artery disease, by accelerating the atherosclerosis and thus it increases the chances of stroke. So diabetes

and hypertension must be controlled. Increased insulin levels are associated with atherosclerosis. When the microvascular complications of diabetes are present like retinopathy, neuropathy and nephropathy, pt is more susceptible for stroke.

Dyslipidemia are important risk factor in stroke. They can be elevated TGL levels, cholesterol levels, LDL levels or decreased HDL level. Dyslipidemia accelerates the atherosclerosis causing stroke. Statins has been well established to reduce the ischemic stroke. LIPID study showed reduction in stroke .

Atrial fibrillation can be due to lone atrial fibrillation, hyperthyroidism, valvular heart disease, alcohol, cardiomyopathy. Atrial fibrillation is an important risk factor in stroke .As the age advances the AF rate increases. It causes embolic stroke. AF increases the mortality in patients. Patient will need both rate and rhythm control. For hemodynamically stable AF patients, drugs are needed and for unstable patient, a DC shock is usually needed. Anticoagulants are also needed in AF patients to maintain a INR ratio of 2 – 3. When patients with no structural heart disease present with AF, it is called as lone AF.

Smoking increases the atherosclerosis, it usually depletes the antioxidants like proteases. Thus it will prevent the protective mechanism action against preventing atherosclerosis and thus increasing the risk of stroke. Cigar or pipe smoking is very dangerous, Beedi and also cigarette

smoking are dangerous. Smoking also causes the spasm of the coronary vessels. Smoking can cause hypoxia. Smoking also increases the risk of many malignancies including lung cancer. Patient has to counseled about the ill effects of smoking and advised to quit the smoking. Some patients who are not able to quit smoking due to irresistible desire to smoke can be give nicotine replacement therapies.Patient can also be given antianxiety drugs like buprioprion.

Alcohol is usually protective when taken in mild quantity as it increases the good cholesterol HDL. When taken in moderate and large quantities it is harmful as it increases the LDL and decreases the HDL thus favouring the atherosclerosis. It can cause atrial fibrillation and can cause stroke. It can cause hypertension and even hemorrhagic stroke. So alcohol consumption should be avoided. Heavy drinkers should be given counseling for quitting alcohol. They can be treated with antianxiety drugs. Alcoholic anonyms are present in various parts of the country which helps the patients to quit from alcohol.

Carotid artery stenosis when greater than 75 % is an important risk factor in stroke. TIAs are important risk factor in stroke. TIA is one in which focal neurological defecit usually resolves within 24 hours.

TIA can be of 3 types

1. Low flow TIAs – atherosclerotic lesion at ICA, MCA or at junction of vertebral with basilar arteries
2. Embolic TIAs – usually single.
3. Lacunar TIAs – due to lipohyalinosis in response to atheroma or hypertension.

Recurrent stroke have high mortality rate.

Some unusual causes of stroke are Marfan, marantic endocarditis, moyamoya disease, MELAS, HIV, Collagen vascular disease, arterial dissection, syphilis, fabry disease, scleroderma, arterial dissection, drugs, hanging, cervical radiation.

Hematological causes of stroke are

- Polycythemia
- Thrombocythemia
- TTP
- Sickle cell disease
- PNH
- Lupus anticoagulant
- Protein C deficiency
- Protein S deficiency

- Antithrombin III deficiency
- Leukemia
- Homocysteinemias

Elevated Homocysteine is associated with increased risk for stroke.

Homocysteine metabolism usually requires vitamin B6, folic acid and vitamin B12. Increased homocysteine levels are seen in patients with deficiency of folic acid, vitamin B6 and B12. It has been found to increase the cardiovascular risk and thus increasing the stroke. Its level can be reduced by giving vitamins B6, B12 and folic acid.

Hematological factors should be looked in young stroke patients which is defined as patients with stroke onset before the age of 40 years. Protein C deficiency, protein S deficiency, anti thrombin III mutation have increased risk for thrombosis thus increasing the risk of stroke.

Antiphospholid antibody syndrome are associated with increased risk of stroke.

Antiphospholipid antibodies (aPL) are acquired antibodies present in circulation which can be either a). lupus anticoagulant (LAC) and b). anticardiolipin antibodies (ACA).

The antibodies may belong to the IgG or IgM classes.LAC is detected by coagulation assays. LAC leads to prolongation of PTT which is the screening test

The clinical manifestations are

Major manifestations :	Venous thrombosis
	Arterial thrombosis
	Recurrent foetal loss manifesting as second or first trimester abortions or intrauterine foetal death and thrombocytopenia.
Minor features:	Coomb's positive haemolytic
	anaemia, skin lesions such as
	livedoreticularis.
	Neurological lesions : TIAs and strokes
	Heart valve lesions : Verrucous
	endocarditis, coronary artery
	disease and catastrophic APS.

Anti phospholipid antibody syndrome can be treated with antiplatelet drugs, immunosuppressants and even plasmapheresis is tried in many patients.

In TTP, usually in majority of the patients there is no aggravating cause. It is due to the deficiency or antibodies to ADAMTS13. It can be inherited (Upshaw Shulman) or acquired abnormality due to HIV, pregnancy, cancer, antiplatelet drugs.The condition includes by renal dysfunction, microangiopathic hemolytic anemia, fever, central nervous system abnormalities, and thrombocytopenia.

Jaundice can be seen. Females are more commonly affected. The neurological features can includecoma, convulsions,headache, confusion, delirium and focal neurological deficits. The presence of coombs negative hemolytic anemia, fragmented RBCs in peripheral smear, thrombocytopenia confirms the diagnosis. It is usually treated by corticosteroids, platelet aggregator inhibitors, exchange Transfusion.

Von will brand is the most common hereditary bleeding disorder. It is characterized by prolonged bleeding time, decreased factor VIII level and reduced level of VWF. It is associated with increased risk for stroke. Desmopressin increases the VWF level and it is used for treatment of VWF. Other treatment includes cryoprecipitate, platelet transfusions and Fresh frozen plasma.

The term polycythaemia denotes increase in the total number and volume of RBCS. Polycythaemiacan be either absolute or relative. It is absolute when either No. of RBCs is increased or when plasma volume alone is decreased. Primary polycythaemia vera is an absolute increase in RBC mass due to neoplastic proliferation of erythroid elements. The erythroid proliferation is not due to increase in erythropoietin levels although the erythroid tissue still retains its capacity to respond to erythropoietin. Secondary polycythaemia often results from increased erythropoietin activity caused by hypoxia like high altitude, lung disorder, smoking, cyanotic congenital heart disease, carbon monoxide toxicity, sleep apnoea syndrome or abnormal and inappropriate secretion of erythropoietin by neoplasm of kidney, liver, lung, uterus and cerebellum. Drugs can also lead to mild or moderate increase in erythrocyte numbers.Haemoconcentration by any cause can leads to relative increase in erythrocyte numbers and volume in a given unit of blood. This is termed relative polycythaemia.

In this condition there is no rise in number or volume of total erythrocytes. Polycythemia is a well established risk factor in stroke. Polycythemia causes hyperviscosity then reducing the blood flow to the brain and thus they cause more chance of stasis of blood and thrombosis and thus causing stroke. They can cause ischemic stroke and cerebro vascular thrombosis. Most common neurological manifestations are

headache followed by vertigo followed by TIA followed by ischemic stroke. Anticoagulants are usually prescribed for it .

Leukemia usually causes stroke by meningeal spread. Patients may present with headache, vomiting, vertigo, nausea, hydrocephalus, encephalopathy, cranial neuropathy, radiculopathy, seizures or myelopathy. Leukemia can also cause SOL (space occupying lesion). It involves cerebral hemisphere more than cerebellum, brainstem and spinal cord which can also be involved. Symptoms are due to the site of SOL and depend on the rate of growth of SOL.AML can manifest in the central nervous system as a extramedullary manifestation which is called as Chloroma (granulocytic sarcoma).

Chronic lymphocytic leukemia can involve the brain by the development of NHL which is called as Richter syndrome. .

Leukemia can cause ischemic CVA and Hemorrhagic CVA. It can cause vasculitis and which can cause stroke. Hairy cell leukemia has been reported with CVA. Drugs used in leukemia can also cause CVA.

Posterior reversible encephalopathy syndrome is a form of Hypertensive encephalopathy seen in leukemia. It is usually reversible but sometimes it can lead to deformity. Leukemia patients are more prone for CNS infections.

Estrogen has been both cardio and neuroprotective. But its role in prevention of stroke in postmenopausal women is controversial. Some studies reveal it increases risk of stroke in post menopausal woman. Some studies reveal it does not decrease the risk of stroke. Raloxifene and tamoxifen increases the risk of CVA . So these drugs cannot be used as prophylaxis for prevention of stroke.

Oral contraceptives have been associated with increased risk of CVA and CVT. Pregnancy is also a risk factor of CVT. When OCP usage is associated with hematological abnormalities which can cause thrombosis then the risk of CVA increases.

Depression can also cause stroke. Depression has also been related to increased cardiovascular events. Treatment includes antidepressants.

Diet which includes increased salt intake will cause hypertension and then it will increase the risk for stroke. So diet with low salt i.e. low sodium and high potassium should be given. DASH diet (dietary approach to stop hypertension) includes low salt, high potassium, calcium, magnesium, low nonfat diary products, fruits and vegetables.

This diet has shown to reduce the hypertension and decrease the risk of stroke and also seen to cause weight loss. Physical inactivity can lead to obesity, metabolic syndrome, HT and can increase the risk of stroke. Physical activity is advised to decreased all the risk factors. It should include exercise 30 minutes atleast 5 days a week. Physical activity has lot of beneficial effects.

Obesity is defined as BMI more than 30. Obesity has increased risk of stroke by increasing other diseases like diabetes. So weight control is advised.

Regular physical activity and healthy life style and balanced diet is advised. For moribund obesity there are bariatic surgeries available. Cerebral embolism is the local occlusion of an intracranial artery by a circulating fragment, which may be composed of broken bits of thrombi, platelet-fibrin masses, cholesterol debris, tumour cells, fat globules, gas bubbles, bacteria and parasites or other foreign bodies. Atrial fibrillation is the commonest cause. Then atheroma of the great arteries is the next cause. Atrial fibrillation causes stroke by sudden dislodge of the thrombus in the cardiac chamber or vegetations in the cardiac chamber. Thrombosed pulmonary veins in lung abscess, empyema and purulent lung infections are also causes. Sometimes Recurrent embolic stroke can occur due to SABE, NBTE and atrial myxoma. Most commonly they

involve middle cerebral artery. They can cause RIND which is defined as neurological defecit which completely resolves within a period of 1 – 3 weeks. Usually embolic stroke are sudden in onset with maximum defecit at onset because enough time is not there for protective mechanisms to protect the brain from ischemia. When this embolus occlusion breaks away, circulation is restored in the infarct zone and patient recovers.

General physical examination reveals murmur, arrhythmia, hypotension and patient can complain of syncope, palpitation, angina, dyspnoea. Ophthalmoscope examination can show may embolic segment in the retinal vessels .Pt may present with fever.

There are no warning signs or symptoms before the onset of stroke. The stroke is usually complete at onset developing within seconds or minutes but sometime patient can have stuttering onset too. The clinical feature vary with the site of involvement in brain, and the syndromes occur according to the sites of involvement in brain and thus they can resemble like thrombotic stroke.Massive infarct can present even as coma.

Bad prognosis in Embolic stroke are

- coma at the onset of the stroke
- fever
- intercurrent infections
- cardiac disease
- HT
- DM
- Advancing age
- Embolism in the basilar artery

Nearly 30 per cent of all patients with first embolic stroke die from the first attack and almost 80 per cent of coma patients succumb to the stroke.Patients in embolic stroke surviving the acute insult, recovery occurs. Recurrence is common in embolic stroke and 40 % die from the second attack.

Laboratory findings are similar to that of the thrombotic stroke. In septic infarct, WBC count is increased and in haemorrhagic stroke, there is blood stained CSF . Atrial fibrillation, atrial flutter, arrhythmia , ECG abnormalities, cardiomyopathy can be seen. Angiographic studies reveal the occlusion in cerebral arteries which are usually migratory and when repeated the studies few days later, occlusion is not seen which reveals the vanishing emboli. 2-D echo or transoesophagealechocardiography

helps to find the intracardiac and valvular abnormalities present in the heart and detailed cardiovascular evaluation by newer imaging techniques like MRI, PET scan can be useful.

Embolic stroke can be diagnosed by :

- clinical history of sudden onset of complete stroke within few minutes
- absence of warning symptoms
- presence of potential source of embolism
- evidence of previous or concurrent episode of embolism to other parts of the body
- clear CSF

Nursing care and medical care are the mainstay of the therapy. There is no role for vasodilators. Anticoagulants are of little help. In Massive infarction or in presence of Hypertension, anticoagulants are not given to the patient. Long-term anticoagulant treatment is given to patients with irregular tachyarrythmia to prevent recurrent embolism. Treat the cause of embolic stroke. Active rehabilitation should be given to the patient. Antiarrythmics should be given according to the arrhythmia present in the patient. Surgical treatment can be rewarding in atrial fibrillation and in removal of embolus from the cardiac chamber. Infective endocarditis should be treated with the antibiotics according to

the organism present and duration of the therapy should be as advised according to organism and its resistance.

In Atrial fibrillation, the heart rate is irregularly irregular and atrial fibrillation is due to uncoordinated activation of the atria occurying irregularly. Many of the rapid atrial impulses are blocked by the A-V node. Some impulses however pass through and activate the ventricles. Concealed conduction may occur in addition.

Atrial fibrillation may be paroxysmal or persistent. In lone atrial fibrillation, no organic cause can be detected. Atrial fibrillation can also occur in tachybrady syndrome, which occurs in sick sinus disease. Chronic AF is usually seen in the tropics in patients with RHD, especially mitral stenosis, IHD, thyrotoxicosis, cardiomyopathy, chronic obstructive lung disease, ASD and post-operatively

Causes of atrial fibrillation are

1. mitral stenosis due to rheumatic etiology
2. thyrotoxicosis
3. IHD
4. SN dysfunction
5. Cardiomyopathy
6. Alcohol
7. Myocarditis
8. HT

9. Pericarditis

10. CHD

The ventricular response is very fast and it can be stopped by digitalis. Verapamil and diltiazem act as a brake in AV node and thus help to decrease the rate. DC shock can be given in hemodynamically unstable AF. Normal sinus rhythm may be restored with quinidine,. Procainamide can also be given but usually large doses may be necessary. Propranolol and verapamil are given when the ventricular rate is very rapid. In resistant patients amiodarone may be tried.

Long-term anticoagulation have to be given to the patient as it will help in preventing the thromboembolic episodes. Before the elective conversion of AF, the patient have to be administered anticoagulants for some period of time.

Haemorrhagic strokes can occur due to the following reasons

1. HT cerebral haemorrhage
2. Ruptured cerebral aneurysm which can be saccular, mycotic, etc.
3. Ruptured angioma which can be either arterial, venous or mixed
4. Trauma
5. Blood dyscrasias like purpura, hyper viscosities syndrome, bleeding diathesis, leukemias
6. due to anticoagulants of when the pt is put on anticoagulant therapy
7. Brain tumours which can cause bleeding

8. other causes like arteritis, bleeding in haemorrhagic infarct.

9.unknown cause

In Hypertensive hemorrhagic CVA, Spontaneous intracerebral bleeding occur from smallblood vessels like arterioles, capillaries or venules in severely hypertensive individuals.

Haemorrhagic stroke occurs in almost 20 % of all strokes and aincidence increases as the age advances and most of the individuals in hypertensive patients from 55 to 75 years of ageare usually of the primary intracerebral haemorrhage type. Males and females are usually equally affected. Hypertension can be primary or secondary.

Site of haemorrhages are usually supratentorial in location in the cerebral hemispheres of about 4/5th, the remainder 1/5this in the posterior fossa in the brain. The order of frequency of ICH according to the location are as follows,

- putamen and internal capsule, often extending into the central white matter
- thalamic-subthalamic region
- cerebellum
- pons .

Hypertensive CVA are usually not seen in the cerebral white matter. The source of haemorrhage is not usually seen can be which can be, from small vessels. The blood whichflows under high arterial tension,usually dissects the brain parenchyma,it causes the physical disruption of the brain tissue, the bleeding source is either not seen or destroyed. Finally it will find its way into the ventricular system and subarachnoid space and sothe CSF becomes bloody. Signs of raised ICT can also be seen in the patients. Usually haematomatas which are large, seen in the supratentorial location, displaces the midline structures,and also it can interfere with the venous drainage in the midbrain. The prognosis in the brainstem haemorrhages which causes obstruction to the venous drainage system is usually lethal.

Patients with hematoma which are small in size have a better prognosis, egthalamic hematoma of size < 2.5 cms.

Usually these hematomas can be slowly resorped over a period of 8 to 24 weeks and then they can be finally replaced by a chocolate-coloured cystic cavity, or cleft lined with as troglial scar tissue and also haemosiderin-laden macrophages.

The hypertensive individuals can present withplethora; Usually they present with a blood pressure higher than 160 mmHg SBPand 100 mmHg DBP. Other signs revealing hypertensive retinopathy can be seen in the patients, while papilloedema can be seen in the patients who

present with malignant hypertension. Concentric LVH is not found routinely in the patients. In patients presenting with features of secondary hypertension, signs of the cause of the disease can be seen.

Usually there is no prodromal symptoms in these patients. Sometimes when the patient is carrying out some physical activity, the patient suddenly develops the headache and sooner usually within 30 minutes, patient develops the signs of focal neurological deficit. After sometime (few hours), the headache disappears rapidly. Finally the patients presents with areflexia, flaccidity, sensory paralysis or amotor paralysis or combination of both with or without coma.

In the patients who develop putamen and capsular haemorrhage, patient strong deviation of conjugate lateral gaze, and the head to the side of the lesion in brain, with contralateral side hemiplegia. Some patients develop few signs ofthe middle cerebral artery syndrome.

Patients who present with the same side (ipsilateral) dilated pupil, with contralateral hemiplegia, is suggestive of cerebral edema with herniation and the prognosis is usually very bad and in many cases death.

The patients who develop thalamic haemorrhage, the signs of sensory deficit usually predominate than the motor paralysis (hemiplegia). In these patients usually eyes reveal several abnormal findings. These can be either a paralysis of vertical gaze, or a downward deviation of the eyes or even absence of convergence with intact lateral

gaze or a skew deviation of the eyes or a small non-reactive pupil on the side of the haematoma or even a retraction nystagmus. Patients can be aphasic, and may reveal many signs such as agnosia, homonymous hemianopia, hallucinosis and mutism. The patient is usually alert or lethargic and survival from the lesion is common.

The patients who have pontine haemorrhages, coma is the initial presentation itself and signs of paraplegia or quadriplegia (bilateral sensory-motor deficit) can be seen. During the initial stages, patient can present with paralysis of lateral rectus muscle (lateral movements – abduction), these movements can be even absent to caloric stimulation, but as the time progresses the eyes finally do not show any movement in any direction (fixed gaze). Patients show pin-point pupils which have very sluggish reaction to the bright source of light. When the patients have haematoma in this site, it is always fatal.

Patient with cerebellar haemorrhage, the initial presentation is usually an intense occipital pain, vertigo and they vomit quite often. Patients are usuallyalert at the onset of the stroke, but they are unable to maintain an upright balance in sitting or standing or walking. The unusual feature of these patients are lack of the ataxic signs in the limbs and syndromes are usually never seen. The patient can present with a quadriplegia when the haematomais compressing the pons, apontine syndrome can be seen in these settings. The patients can present with the

paralysis of lateral gaze to the side of the lesion in the brain or a forced lateral gaze deviation to the opposite side of lesion in the brain. The patients have usually sparing of their vertical gaze. Patients have small pupils which are usually reactive to light until late in the disease. Neck retraction can also be seen in these patients.

The laboratory findings in the ICH is sanguinous CSF which confirms the diagnosis. The fluid is uniformly blood-stained and it does not clot on standing and the supernatant fluid shows xanthochromia. The leucocyte count (WBC) of the fluid may be elevated, RBCS appear within 2 hours and usually they disappear in about 4 weeks. Indirect van den Bergh test may be positive due to the conversion of oxyHb to bilirubin in the first 72 hours.

Patients who undergo a traumatic tap can easily be differentiated from ICH, the CSFobtained from traumatic tap is usually not completely sanguinous and it clots and never shows xanthochromia. The WBC:RBC ratio (leucocyte : red cell ratio) in the CSF remains unchanged.

X-ray Skull, EEGand cerebral angiography are not useful in the diagnosis of the infratentorial haematoma, but signs of midline displacement in supratentorial lesions can be seen in them. EEG usually reveals high voltage, focal or diffuse slow waves in these patients. Patients who undergoes radio-isotopic brain scan showsabnormal concentrations at the site of the haematoma. CT scan is very helpful in

diagnosing, it can reveal the anatomical location, even the dimension of the cerebral haematoma. CT scan can also show vascular malformation and helpful in excluding other lesions in the brain which mimics ganglionic haemorrhage. CT scan is very helpful in defining the approachwhen surgical evacuation of the cerebellar haematoma is done.But in almost 80% of cases, the diagnosis can be straightforward by examination of the CSF, though caution should be exercised because the raised intracranial pressure which is seen with haemorrhage can cause coning (brainstem herniation into the foramen magnum). MRI is definitely superior to CT scan in aiding in diagnosis. In patients with arteiovenous malformation, Angiography is done to rule out middle cerebral artery aneurysm, and before surgical intervention.

The ICH is diagnosed by

- patient presents at the onset with headache, vomiting and nuchal rigidity
- patient do not reveal any prodromal symptoms
- stroke develops rapidly and no stuttering course is seen
- patient can present with lethargy which can progress to coma
- presence of fresh blood in the CSF in HT individualrevealing the diagnosis of hypertensive intracerebral haemorrhage.

Intracerebral bleed can occur from the ruptured congenital aneurysm, or an angioma in persons with high blood pressure which can

only be diagnosed cerebral angiogram. Appropriate blood tests should be done to rule out any bleeding disorders such as purpura or leukaemia.

In patients with pontine haemorrhage and large dissecting haematomas the prognosis are usually lethal. Death which occurs in later course of the illness may be due to bleeding peptic ulcer of neurogenic origin. The patients who have small occult haematomas like thalamic haemorrhage and slit haemorrhages, the neurological deficit remains severe and unaltered.

Indicators of grave prognosis in ICH are

- Fever
- intercurrent infections
- severe hypertension
- advanced age
- uncontrolled diabetes mellitus
- bleeding diathesis

Patients with ICH should be given a general nursing and medical care. Patient are usually are treated with parenteral hypotensive agents which are needed for rapid lowering of the high blood pressure. During the parenteral Antihypertensive therapy, continuous monitoring of blood pressure is mandatory. The role of hypothermia needs to be evaluated. When there is evidence of cerebral edema, steroids or oral glycerol therapy is indicated, the role of beneficial effects of dexamethasone in the treatment of primary intracerebral haemorrhages is controversial.

The clinical impression in favour of using aminocaproic acid (20 g IV 8 hourly) is gaining support, though there are no supportive trials.

Patients with supratentorial or cerebellar haematoma, surgical evacuation should be done to prevent temporal lobe herniation or even the compression of the pons, surgical evacuation is usually a life saving measure and it should not be delayed. In cerebellar haematoma, surgery should be performed before the brainstem is compressed resulting in decreased level of consciousness, but clot evacuation may be life-saving even in comatose subjects if compression has not been prolonged.

Moyamoya disease, which is not diagnosed clinically as it is an angiographic diagnosis which is characterised by an unusual combination of progressive occlusive lesion at the intracranial carotid bifurcation with an extensive or profuse abnormal anastomotic network of arterioles and capillaries with dilated main arteries at the base of the

brain. The characteristic angiographic appearance in these patients is usually described as puff of cigarette smoke drifting in the air. These patients have carotid occlusion which is a non-inflammatory, and it has subintimal fibroplasia near arterial cushion. Moyamoya disease are reported in children and young adults among Japanese and other Asians. These patients can present with variety of clinical manifestations like headache, convulsions and SAH . Till now there is no specific treatment.

Fibromuscular dysplasiacan also be cause of ICH and it is a segmental, non-atheromatous, non-inflammatory angiopathy of unknown cause.

FMD can involve either the intima, media and sometimes even the adventitia of renal, aorto-iliac, splenic and internal carotid arteries where vascular bruits are present. FMD reveals characteristic angiographic appearance, irregular beading stenotic areas alternate with pouch dilatation of aneurysmal segments.It is seen in young or middle-aged women, on oral contraceptive pills, and it may present with migraine type headache or TIA. Treatment involves the surgical dilatation of the narrowed segments which are helpful in reducing the symptoms. Binswanger's subcortical arteriosclerotic encephalopathy, leukoaraiosis, Winiwarter-Buerger disease are some rare causes of stroke.

Different risk factors have been attributed for strokes in the young individuals, hypertension and diabetes mellitus being the most common among them. Elevated haematocrit levels (PCV) and disturbed equilibrium in coagulation profile and fibrinolysis have been highly attributed in the aetiopathology of nonembolic cerebral infarction in the young individuals. Patients with stroke, and the possibility of subacute central nervous system infections like tuberculosis, syphilis, arteritis and autoimmune angiitis as risk factors needs to be evaluated. Ischaemic cerebrovascular complications of pregnancy and puerperium are commonly seen throughout India, most of the cases are from cerebral venous thrombosis occurs during the perinatal period.

Spontaneous bleeding in the subarachnoid space is known as subarachnoid haemorrhage (SAH).SAH usually occurs due to ruptured artery or vein which is called as primary SAH or when it occurs from an intracerebral haematoma which dissects through cerebral tissue into the ventricles, it is called as secondary SAH. Subarachnoid bleeding occurs usually from rupture of a congenital cerebral aneurysm

Important causes of SAH are

- Ruptured aneurysm which can be a
  - congenital
  - mycotic
- Arteriovenous malformation
- Intracerebral haemorrhage with extension to subarachnoid space
- Haemorrhagic cerebral infarction
- Rupture of atherosclerotic vessel
- Haemorrhage in a tumour
- Bleeding disorders, leukaemia, sickle cell anaemia
- Clotting disorders
- Vasculitis
- Infection (TB, syphilis, bacterial)
- Moyamoya disease

Most of the cerebral aneurysms are found in the anterior half of the circle of Willis. Location or cerebral aneurysm occurring most frequently are

- anterior communicating anterior cerebral junction
- posterior communicating-internal carotid junction
- middle cerebral bifurcation
- intracranial carotid bifurcation
- vertebro-basilar or basilar bifurcation
- distal anterior cerebral territory

A literature which reveals autopsy surveys say that only half the aneurysms show evidence of bleeding, whereas an equal number is unruptured. A high incidence of PKD and COAhave been seen in patients with cerebral aneurysm.

Usually the media is defective in the branches and bifurcation of the cerebral arteries, so, cerebral aneurysms frequently occur in these sites. The commonest point of tear is seen in the dome of the aneurysm because it is the weakest point of the saccule. These can rupture during the episodic raise of blood pressure to alarming heights in normal individuals, during violent bouts of coughing or sneezing or heavy physical exercise or coitus or emotional excitement, and these incidents can occur before a rupture. In the patients in whomthe aneurysm bleeds

both within the brain substance and also in the subarachniod space, the prognosis is always grave.

General physical examination of these patientsusually shows no abnormality. The blood pressure of these patients are usually within the normal range. Some patients may reveal a huge polycystic kidney on abdominal examination, while some can show the physical signs of coarctation of the aorta

Some patients with an unruptured aneurysm, which is large and firm enough to press on neighbouring structures, can present with visual defects, ocular palsies, hypopituitarism or obstructive hydrocephalus. These patients are usually misdiagnosed as brain tumour at initial presentation because the clinical syndrome appears insidiously, until the angiography is done which reveals aneurysm.

While doing physical activity, a "snap" may be heard within the head of the patient and the patient may complain a violent headache with associated vomiting and sustain a sudden fall on the floor. In 10 per cent of cases, seizures are seen at the onset. The symptoms and signs of an aneurysm depends on its site, the amount of the bleeding and the time of occurrence of Subarachnoid haemorrage. In about 8 per cent of cases sudden death from a massive bleed occurs, but in majority of cases i.e. about 80 per cent cases awake from the initial coma and they still remain confused, disoriented and amnesic for about a week. Intermittent

headaches and lethargy are known to occur during this period in these patients.

In an anterior communicating rupture, the presentation may be transient weakness of both the legs, akinetic mutism with pyramidal signs. In a posterior communicating rupture, an ipsilateral third nerve palsy is seen. Dilatation of the ipsilateral pupil is an early sign of posterior communicating rupture. In the rupture of internal carotid and middle cerebral artery aneurysms, a hemiplegic syndrome as described under cerebral thrombosis appears. Rapid recovery from a dense neurological deficit is highly suggestive of a reflex vasospasm with ischaemia of that territory.

The common clinical findings in these patients usually includes neck rigidity, fever, transient hypertension, pre-retinal haemorrhages, restless state with confusion, amnesia with bilateral Babinski's signs.

In patients with a massive intraventricular bleed like a basilar aneurysm, there are signs like extensor rigidity, irregular respirations and bradycardia that accompany the deep coma. Usually death is an almost inevitable outcome in these patients.

Laboratory diagnosis of subarachnoid hemorrhage is done by the examination of CSF examination which reveals uniformly sanguinous CSF under raised pressure and it contains nearly a million RBCs per

cumm. CSF examination also reveals the presence of pleocytosis and xanthochromia in the patients.

Cerebral angiography or DSA plays an important role in the diagnosis of SAH. It helps to outlines the aneurysm and reveals coincident vasospasm or a subdural clot. Serial CT scan should be taken. These serial CT s has an important role in diagnosis and helps to tell about the prognosis of the patient. Serial CT scan not only reveals the subarachnoid haemorrhage, it also shows the aneurysm when taken after administration of the contrast. More than 75 per cent of patientsreveals SAH bleed on a noncontrast CT scan which was done within the first 48 hours of rupture. An MRI scan is superior to CT scan in visualization of the aneurysm. In patients with SAH, when ECG was taken it revealed the abnormalities which are highly suggestive of myocardial ischaemia, when there is no myocardial damage. Hyponatraemia is also seen due to the inappropriate secretion of anti-diuretic hormone with volume expansion or because of unknown factors which can cause salt and water loss with subsequent volume depletion.

About 8 % of the individuals die in the first few hours, and 40 % die within the next six months due to recurrence.

Those patients who survive the attack, and who are confused and amnesic state,for more than two weeks,indicates a bad prognosis and probably it reveals the irreversible damage and intellectual impairment;

and epilepsy can also be seen in these patients. Those patients who survive the acute insult show residual disability and are unable to resume full work.

When there is SAH due to rupture of aneurysm or angioma, these patients present with excruciating headache and a brief period of unconsciousness (collapse) which is usually followed by a lucid interval, or a restless state with confusion and disorientation, there is paucity of localizing signs, and presence of nuchal rigidity in a normotensive individual. Clinically, a close DD is etiologies which causes sudden rise of intracranial pressure. Another DD is meningitis when the bleed is small. Cerebral angiographic findings are helpful in arriving at the diagnosis, but the associated vasospasm may decrease or even prevent the visualisation of the malformation aneurysm.

Treatment of SAH includes absolute bed rest, head end slightly elevated, for a period of six weeks. During the acute phase of the illness, physical straininglike coughing, sneezing or straining during bowel movementhave to be avoided. Patients can be prescribed laxatives. Other treatment includes general nursing care and medical care.The blood pressure should always be controlled to target levels. Mild sedatives or even analgesics can be given for severe headache. Aspirin is inappropriate but acetaminophen or meperedine and phenobarbitone are given. Heavy sedation has to be prevented as it can cause difficulty in

the initial assessment or delayed neurological deficit. Patients can be prescribed Chlorpromazine as it sedates the patient and is helpful in controlling the nausea and vomiting. Sometimes the seizures can itself elevate the blood pressure, and so patients are prescribed anti-convulsants as prophylaxis measure. The prime objective of the therapy is the prevention and treatment of vasospasm. Hypervolaemic therapy is useful in increasing the Cerebral Blood Flow during vasospasm.

Gamma aminocaproic acid and related derivatives like tranexamic acid are used in the treatment of SAH and they are of important value to reduce the incidence of rebleeding, but some recent studies have shown increased risk of cerebral infarction.

Surgical interventions should be carried out for patients who have aneurysms in the accessible territories. The treatment of symptomatic vasospasm includes the use of calcium-channel blockers like nimodipine (0.7 mg/kg followed by 0.35 mg/kg every 4 hours for 21 days). A conservative medical line of treatment should be carried out only when the patient has when the aneurysm is in an inaccessible territory or aneurysm cannot be visualised or when in presence of systemic diseases which are contraindications to surgery. When the patient has severe vasospasm, surgery should be postponed. But when the patient presents with a subdural bleed or clot, its immediate evacuation is often life-saving.

AHA and ASA have published guidelines for prevention of strokes which are helpful in reducing the risk factors associated with stroke.[3] Hypertension is an independent risk factor for stroke.[4]Life style modifications include physical activity, obesity management, decreasing the salt intake. In the meta-analysis of 23 randomized trials, where the patients were put on antihypertensive therapy, and comparision was made with patients not put on anymedication. The results were the risk of the stroke reduced by 32% in patients with antihypertensives.[5] When the BP was reducd, SBP <140, DBP<90, the risk of cardiovascular events decreased. In patients with CKD or DM the target BP is still lower i.e. < 130/80 mmHg.[(4)]

Cigarette smoking has direct link with stroke as it increases the risk of both ischemic CVA and SAH, the risk of ischemic stroke doubles and risk of SAH increases 2- 4 fold.[(6,7,8,9,10,11)]Smoking increases the incidence of Hemorrhagic CVA in young individuals. When smoking is quitted after few years, the risk of vascular events in smokers and non smokers are almost similar.[(12,13,14,15)]

Diabetes increases the risk of stroke.[(16)]Statins are helpful in reducing the risk of stroke. Role of aspirin in diabetics are yet to be established.[(17,18,19)]

Increased level of total cholesterol increases the risk of ischemic stroke, revealed in various epidemiological studies.[20,21,22,23]The National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) have outlined the targets which are helpful to prevent the stroke.

CHAD S2 score is helpful in treating atrial fibrillation patients.[24,25,26,27]ACTIVE A and ACTIVE W has revealed that warfarin at an adjusted dose is superior in the prevention of stroke in patients with AF when compared to aspirin and clopidogrel, and when clopidogrel with aspirin is used it is superior to aspirin used alone.[28]Regular monitoring of INR is necessary in the patients with warfarin as it can itself cause ICH. Adjusted-dose warfarin with a target of INR 2-3,helps preventing the stroke in patients with AF, and it helps to decrease the mortality and severity of stroke. Aspirin is given to patients with low risk to prevent stroke. As the LV rejection fraction decreases the risk of stroke keeps on increasing.

A healthy lifestyle includes avoiding smoking, maintaining adequate body mass index, involving in regular physical activity, adequate intake of vegetables and one study has been linked to decreased incidence of total, ischemic, and hemorrhagic stroke when patient follows healthy lifestyle.

Materials And Methods

MATERIALS AND METHODS

Selection criterion:

All CVA patients irrespective of their age with CT Findings of infarct and hemorrhage.

Total no. of patients under study- 100

Period of Study:

All the patients admitted as inpatients in Tirunelveli Medical College Hospital, during the period of September2011 – November 2012 were included in thisstudy.

Geographic distribution:

Geographic distribution of the patients were predominantly from rural areas of Tirunelveli, Tenkasi, Tuticorin Districts.

Exclusion criterion:

- CVA patients without CT scan
- Sub arachnoid hemorrhage

Limitation of the Study:

- MRI could not be done in all patients because of its its cost factor.
- Echo done only in patients where it was necessary

Selection and study of this patients were done as mentioned in the proforma.

All the patients underwent a thorough and detailed history regarding onset, activity during stroke and general and neurological examinations.

Neurological examination was done with reference to motor, sensory, autonomic and higher functional disturbances.

Power is graded according to MRC grading system from 0 to 5.Sensory system examinations were done for somatic, special andcortical sensation.

Higher functions testing were done with minimental scale.

Clinical assessment of nature of lesion

Embolic stroke

- Patients can present with abrupt onset with maximum defecit at onset and fast recovery
- When patient presents with signs of arrhythmias in ECG or clinically like AF,SN dysfunction
- When patient has organic cardiac murmurs
- When patients presents with signs of heart failure
- When the patient has a recent h/o MI
- When echo reveals vegetations which can be due to systemic lupus erythematosus , neoplasia, or which can be marantic endocarditis
- The presence of unequal pulses or BP in limbs suggests the careful evaluation of Carotid or vertebrobasilar dissections.

- Ischemic infarct in the CT scan with embologenic cardiac lesion proved by Echocardiogram

Hemorrhagic stroke

Haemorrhagic stroke is suspected clinically by the presence of intense throbbing headache, projectile vomiting, seizures, altered sensorium, Neck stiffness, dissociated eye movements, Papilloedema in a known hypertensive patients.CT brain revealing hemorrhage (hyperdense).

Thrombotic stroke

- Patient presenting with family h/o thrombotic stroke, DM, HT, TIA, dyslipidemia.
- Patient may present with abrupt onset but they evolve somewhat more slowly over a period of several minutes or hours and occasionally days; in the later case, the stroke usually progresses in a step ladder pattern (in a series of steps).
- Clinical features vary according to the site of involvement
- CT brain does not reveal any bleed or hemorrhage

Time of occurance were divided by the basis:

- Morning 6 AM –2 PM
- Noon 2 pm – 10 pm
- Night 10pm – 6 am

Activity during the occurrence of stroke were classified as

1. **Severe activity**which included hard physical straining like sports activity, bicycle riding, sexual intercourse, doing hard physical work in sunlight, sporting etc.
2. **Moderate activity** includesgrooming, walking, shopping, less strain physical activities.
3. **Mild activities** like sitting, brushing, conversation, watching Telivision, doing benchwork, reading, writing.
4. **Sleep.** It also includes when the patient says that when he woke up he found himself hemiplegic and before sleep he was absolutely normal.
5. After careful clinical examination of the patients all the patients were submitted to the following investigations.

I Basic lab investigations:

a. Urine analysis.
b. Blood-Sugar, Urea, Creatinine.
c. Serum electrolytes.
d. Lipid profile.

II ECG

12 lead ECG was taken atleast once in all those patients. The ECG was taken soon after admission and subsequently thereafter if needed.

Rhythms strips were also taken when required.

Rate, Rhythm, PR interval, QRS axis, QTC interval, ST segment, U waves, Ischaemic Changes, chamber hypertrophy were analysed and recorded.

III ECHO

The ECHO was done in 18 patients who showed ischaemic and other changes in ECG. Special attention was given for any valvular dysfunction, reduced LVEF, and any regional wall motion abnormalities.

IV CT Scan Brain

CT Scan Brain was done in all patients included in this study within 24 – 48 hrs of admission. It was repeated after 48 hrs when the initial CT Scan was negative and also repeated when the patient suddenly detoriated or if there is no expected recovery.

The Imagining of brain was done to

- To diagnose CVA.
- To document the presence of haemorrhage or infarct.
- To locate the site of lesion, size of lesion, extent of brain damage and surrounding edema.

Results

RESULTS AND OBSERVATIONS

Statistical analysis

The continuous variables among the stroke subjects were analysed and interpreted according to their stroke type by ANOVA (Analysis of Variance) and the difference were interpreted by Bonferroni post hoc test. The fasting BGL and PPBGL was compared by Student' paired 't' test. The Categorical variables were analysed and interpreted by $\chi 2$ (Chi-square) test. The above statistical procedures were performed by statistical package IBM SPSS statistics 20. The P values less than 0.05 ($P<0.05$) were considered as significant in two – tail test.

I. Age and stroke type distribution

The total number of patients included in the study was 100 during the period of 2011 – 2012 in Tirunelveli Medical College and Hospital. The Age and type of stroke distributions were compiled in tabular columns as follows:

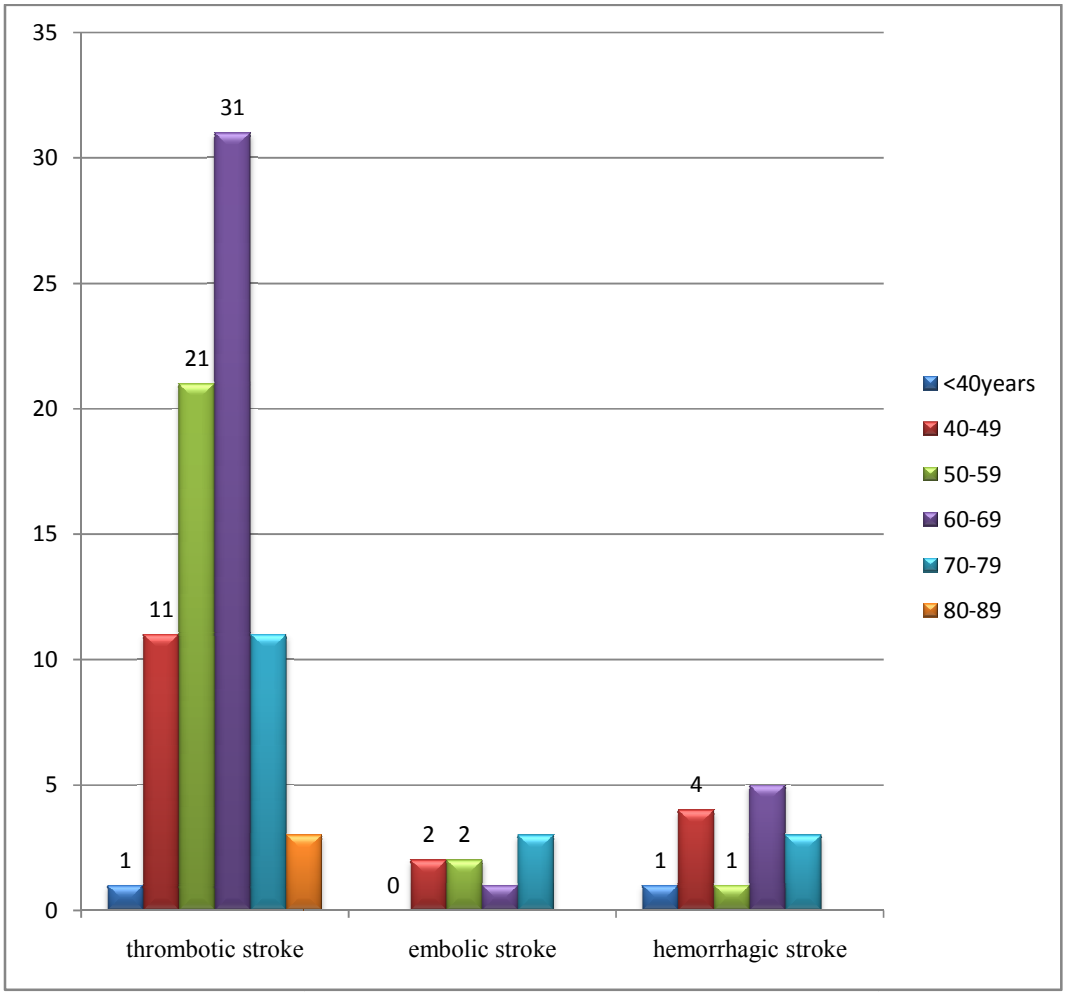
Table .1

Age and stroke type distribution

Age (years)	Embolic		Hemorrhagic		Thrombotic		Total
	No	%	No	%	No	%	
<40	0	0.0	1	7.1	1	7.1	2
40-49	2	25.0	4	28.6	11	14.1	17
50-59	2	25.0	1	7.1	21	26.9	24
60-69	1	12.5	5	35.7	31	39.7	37
70-79	3	37.5	3	21.4	11	14.1	17
80-89	0	0.0	0	0.0	3	3.8	3
Total	8	100.0	14	100.0	78	100.0	100
Mean ± SD	61.1± 14.2 Range (40to 79)		56.6 ±13.0 Range (35to 76)		59.9±10.9 Range (21to 88)		59.6±11.4
ANOVA 'F'	0.590						-
Significance	P>0.05						

The above table -1describes the type of stroke with age of incidence. The mean ages of Embolic, Hemorrhagic and Thrombotic were 61.1± 14.2 years, 56.6 ±13.0 years and 59.9±10.9 years respectively. The difference between the types were not statistically significant (P>0.05).

Our study reveals that mean age of onset of embolic stroke, hemorrhagic and thrombotic stroke 61.1, 56.6 and 59.9 respectively.



II. Age and sex distribution

The total number of patients included in the study was 100 during the period of 2011 – 2012 in Tirunelveli Medical College and Hospital. 69 were male and 31 were female patients.

The Age, Sex distributions were compiled in tabular columns as follows:

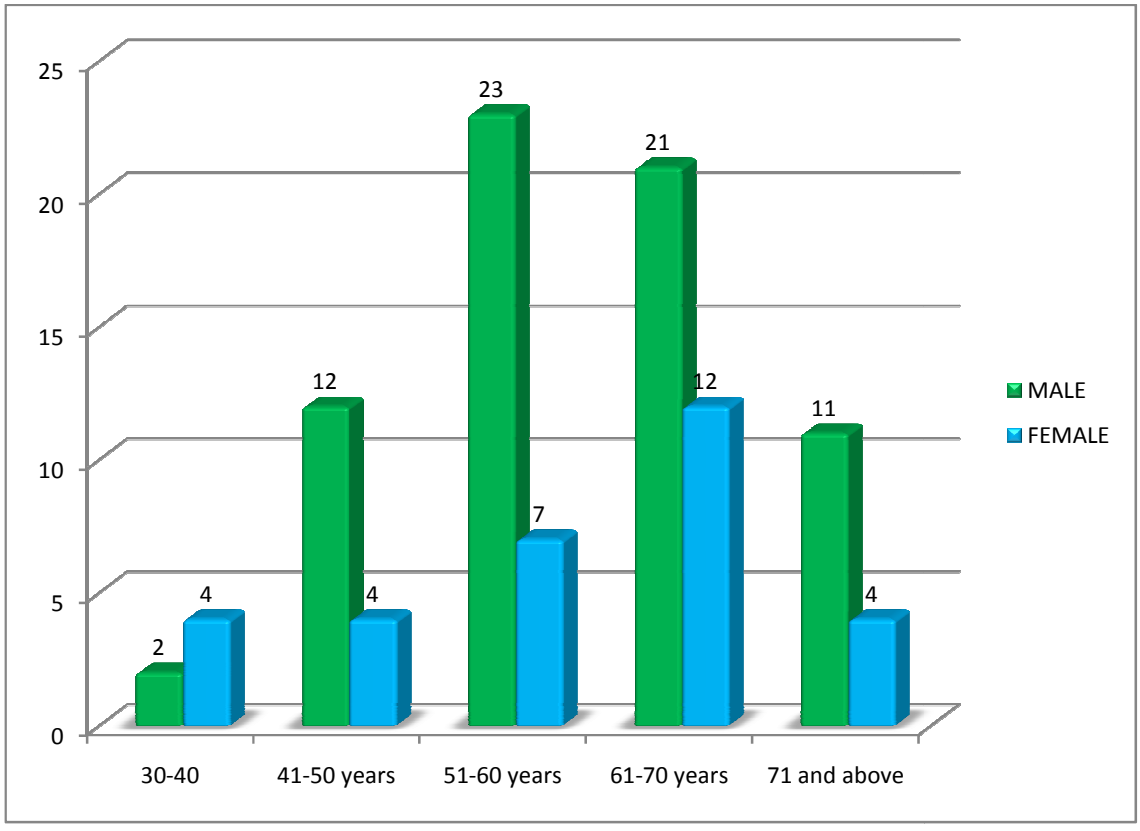
Table. 2

Age and sex distribution

Sex	30 - 40 years	41- 50 years	51- 60 years	61 - 70 years	71 years and above
Male	2	12	23	21	11
Female	4	4	7	12	4
Total	6	16	30	33	15

Total male patients were 69 and female patients were 31 in this study. Youngest patient was a male 35 year old and the oldest was a female and the patient was 88 years old. Oldest male patient in our study was 82 years old.

Between 30 – 40 years, there were 2 male and 4 female patients; Between 41- 50 years age group there were 12 male and 4 female patients; Between 51- 60 years age group there were 23 male and 7 female patients; Between 61 -70 years age group there were 21 male and 12 female patients; Last but not least there were 11 male patients and4 female patients in the above 71 years category.



III. Gender wise description of stroke

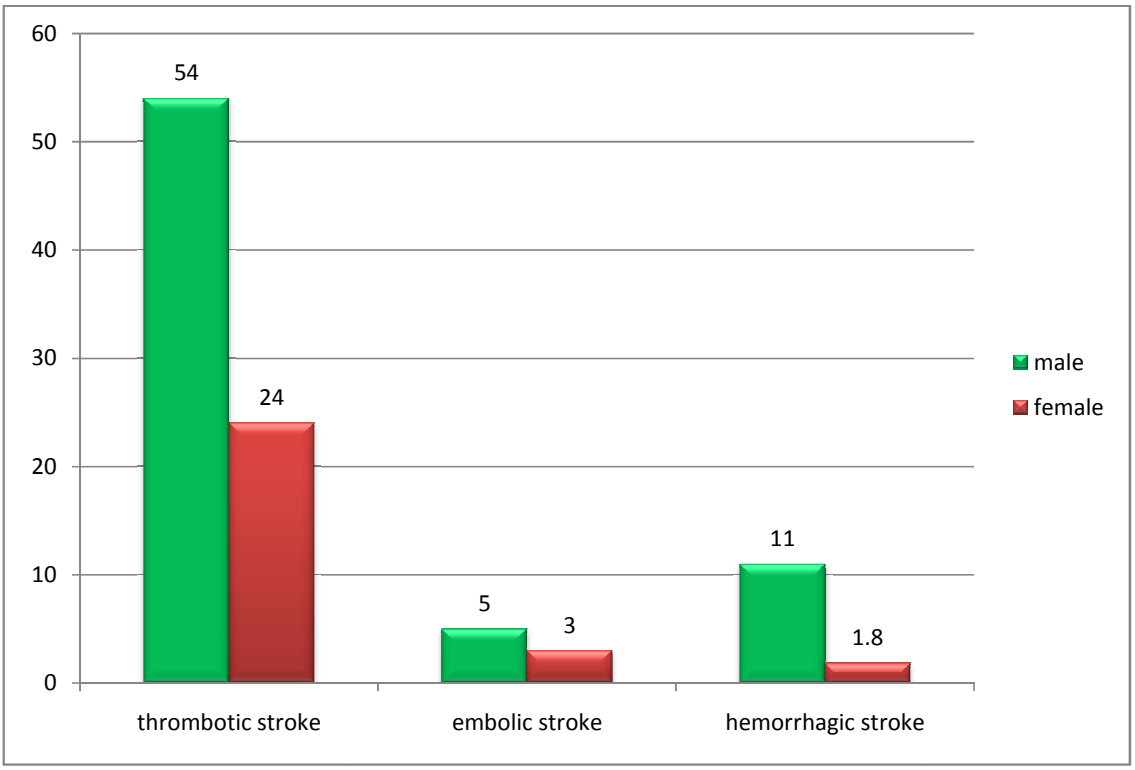
Table - 3.

Genders wise description of stroke.

Stroke Type	Genders			χ^2	df	Signi.
	Male	Female	Total			
Embolic	5	3	8	0.726	2	P>0.05
Hemorrhagic	11	3	14			
Thrombotic	54	24	78			
Total	70	30	100			

The gender wise comparison of stroke was shown in the above table. There was no significant association between the genders in the incidences of stroke (P>0.05).

8 patients had embolic stroke, 14 patients had hemorrhagic stroke and 78 had thrombotic stroke. Out of 8 embolic stroke patients, 5 were male patients and 3 were female patients. Out of14 hemorrhagic stroke patients, 11 were male patients and 3 were female patients. Out of 78 thrombotic stroke patients, 54 were male patients and 24 were female patients.



IV. Significance of risk factors:

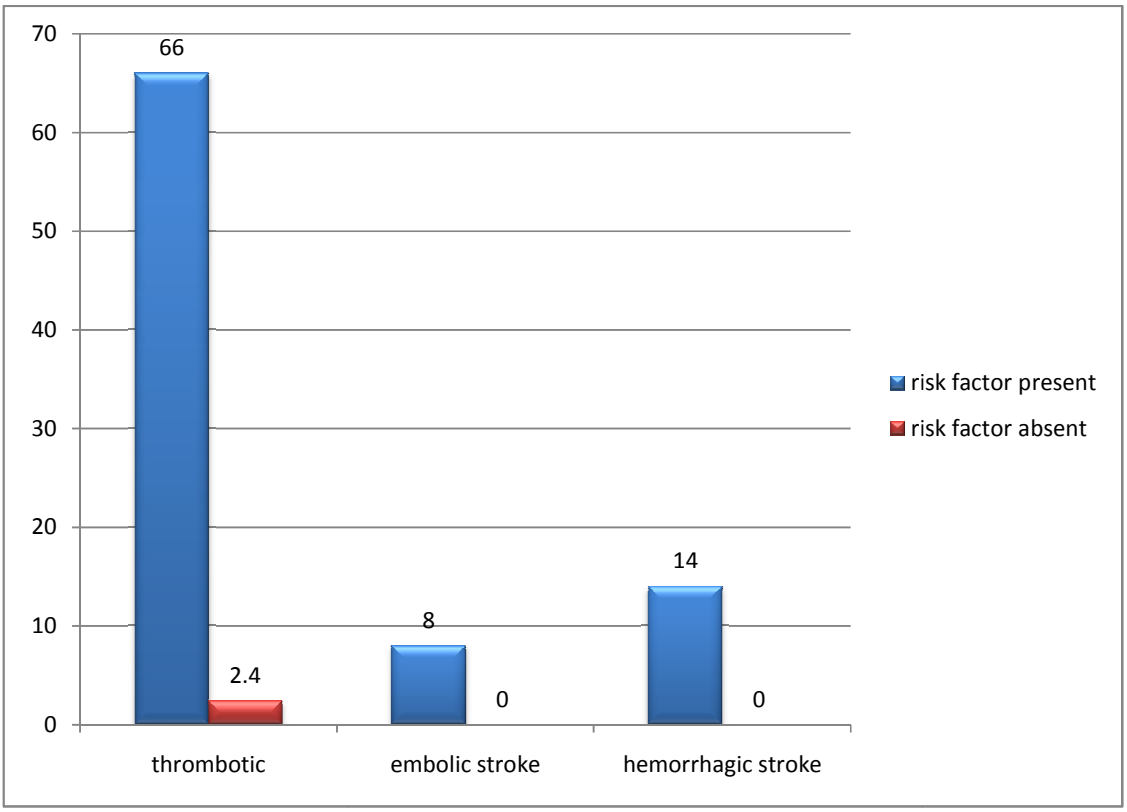
The type of strokes such as Embolic, Hemorrhagic and Thrombotic were evaluated according to any of the risk factors such as Diabetic, Hypertension, CAHD, Dyslipidemia, Smoking and Alcohol present in the patient.

Table - 4

Comparison of Risk factors with incidence of stroke.

Stroke Type	Risk factor			χ^2	df	Signi.
	Yes	No	Total			
Embolic	8	0	8	6.191	2	P<0.05
Hemorrhagic	14	0	14			
Thrombotic	66	12	78			
Total	88	22	100			

The comparison of presence of risk factors with incidence of stroke was shown in the above table. There is significant association between the presence of risk factors with incidences of stroke (P>0.05).



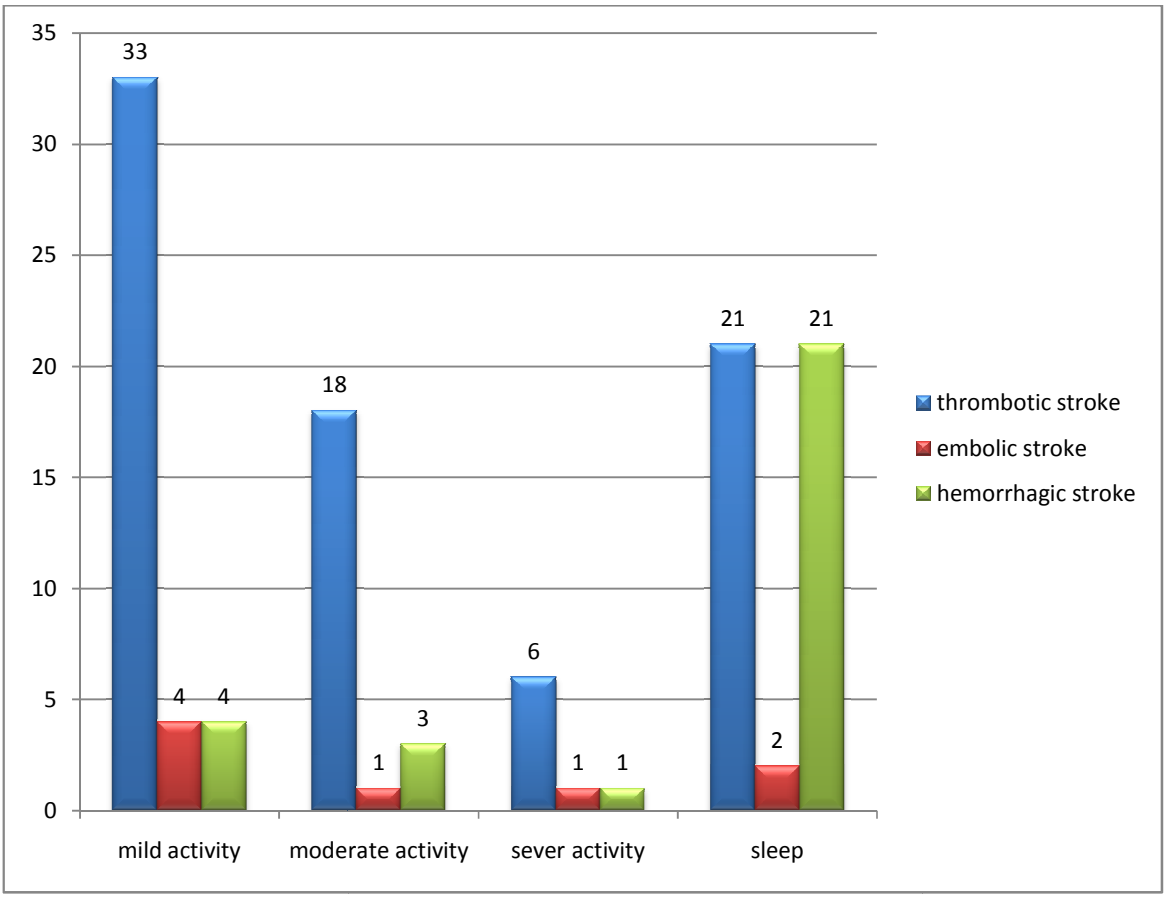
V. Activity with stroke.

Table-5.

Comparison between activities with incidence of stroke

Activities	Stroke Type				χ^2	df	Signi.
	Embolic	Hemorrhagic	Thrombotic	Total			
Mild	4	4	33	41	2.401	6	P>0.05
Moderate	1	3	18	22			
Severe	1	1	6	8			
Sleeping	2	6	21	29			
Total	8	14	78	100			

The activities were compared with incidence of strokes in table-5. The results revealed that there was no significant association between the Activities and incidences of strokes (P<0.05).



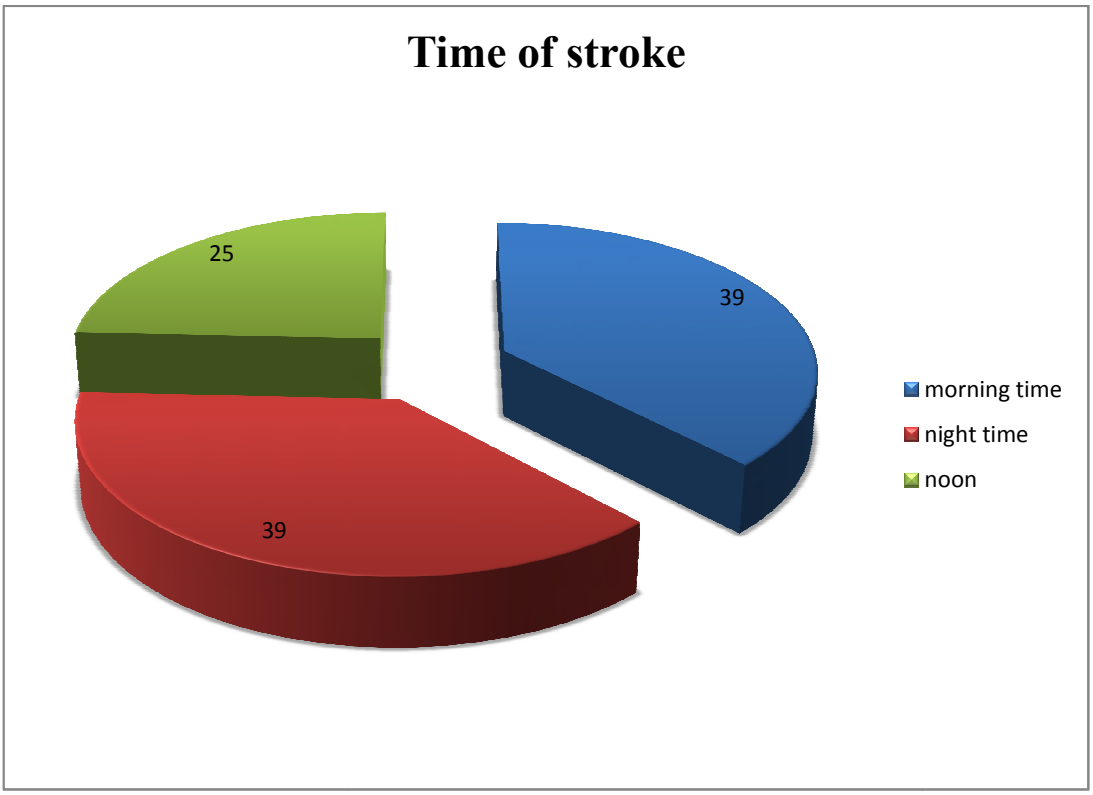
VI. Significance of stroke incidence between activity with time

Table - 6.

Comparison of stroke incidence between Activities with Time.

Activities	Time			χ^2_{paired}	df	Signi.
	Night	Other time	Total			
Sleeping	25	4	29	4.500	1	P<0.05
Others activities	14	57	71			
Total	39	61	100			

The incidence of strokes during night at sleep was significantly more than the incidence of other activities and other Times (P<0.05)



VII. Description of the level of biochemical parameters:

The biochemical parameters such as Fasting Blood Glucose, PP Blood Glucose, Cholesterol, and TGL.

Table-7

Comparison blood glucose level of Fasting and PP.

Variable	Fasting		PP		Increase		Paired 't'	df	Signi
	Mean	SD	Mean	SD	Mean	SD			
BGL	120.16	39.7	176.3	65.4	56.1	38.2	14.704	99	P<0.001

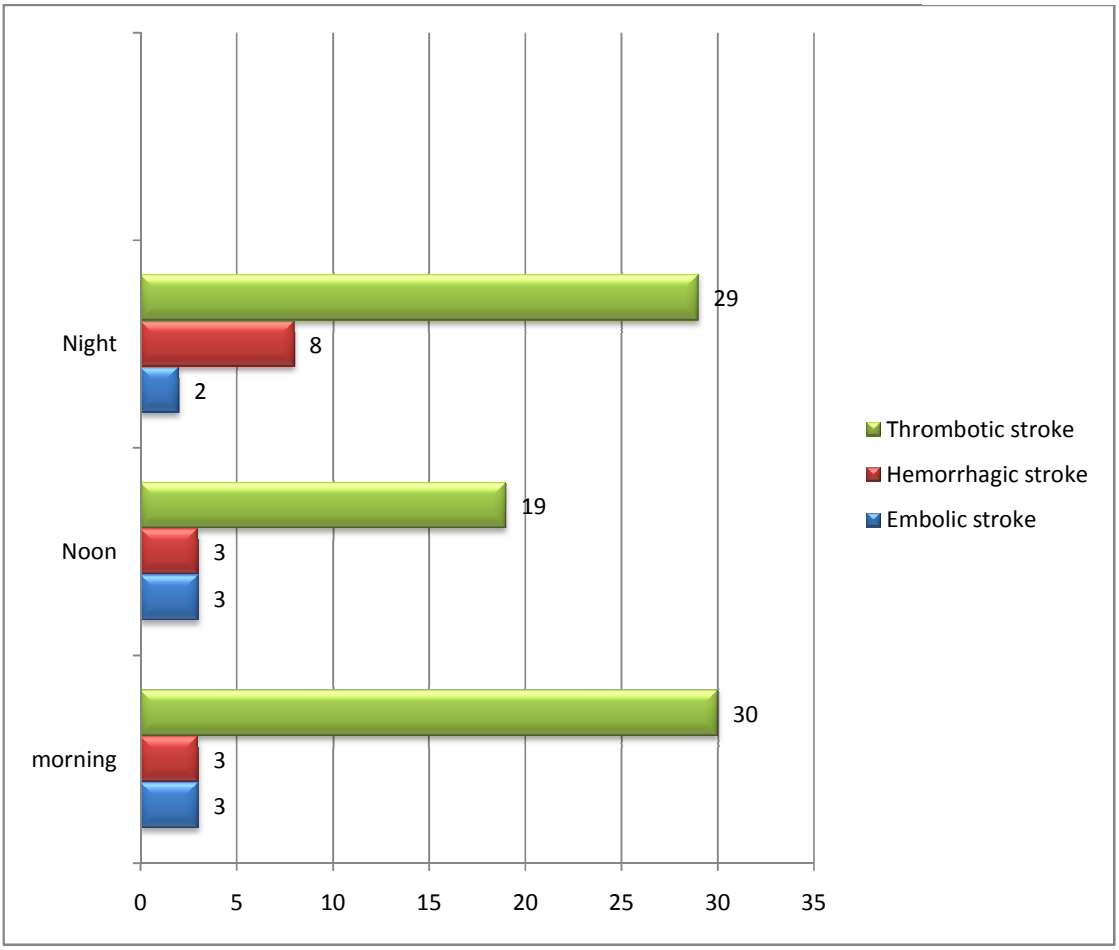
The Fasting BGL and PPBGL were compared in the above table. The mean Fasting BGL was 120.16± 39.7 and mean PPBGL was 176.3±65.4 . The increased BGL 56.1±38.2 was statistically very highly significant (P<0.001).

VIII. Time of stroke with type of stroke

	Morning	**Noon**	**Night**
Embolic	3	3	2
Hemorrhagic	3	3	8
Thrombotic	30	19	29

- 36 patients had stroke in the morning. Out of which 3 were embolic, 3 were hemorrhagic and 30 were thrombotic.
- 25 patients had stroke in the Noon. Out of which 3 were embolic, 3 were hemorrhagic and 19 were Thrombotic.

- 39 patients had stroke in the night. Out of 2 were embolic, 8 were hemorrhagic and 29 were thrombotic.
- So in this study 78 patients had thrombotic stroke,14 had hemorrhagic stroke and 8 had embolic stroke.



IX. Evaluation of risk factors

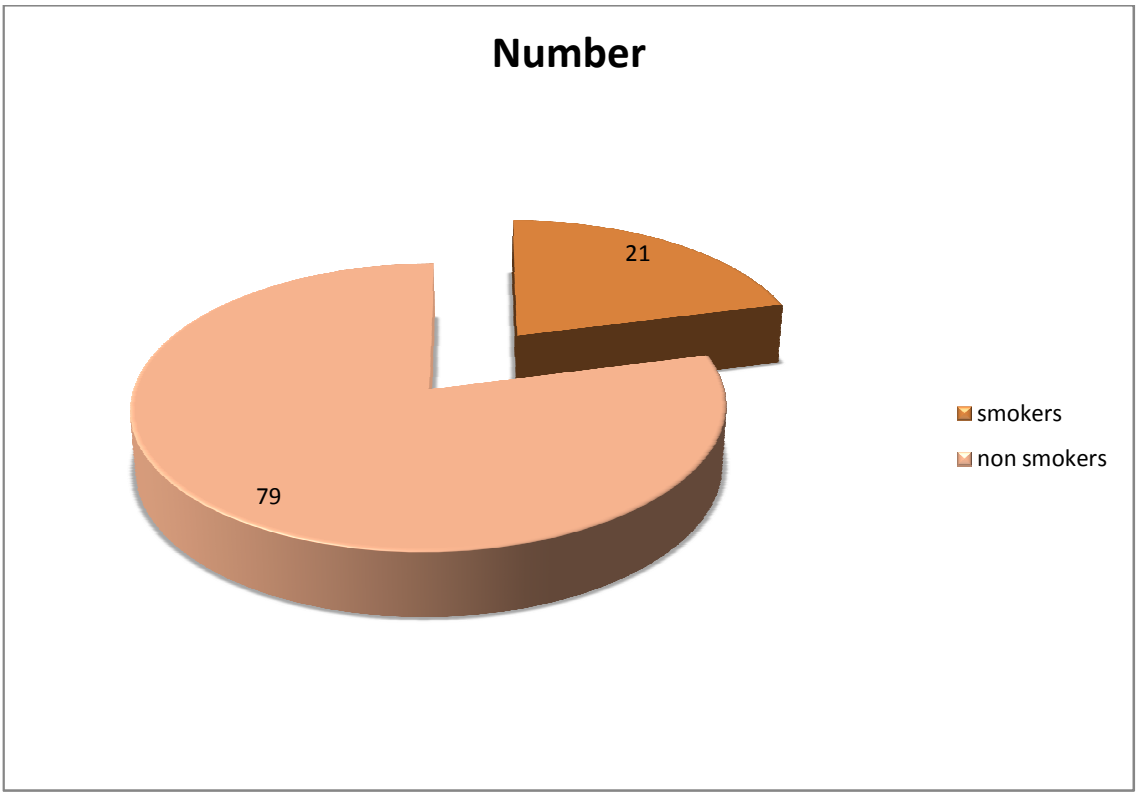
Risk factors were studied invidually as observational study.

a. Smokers Vs Non smokers

Table. a

smokers	21
Non smokers	79

21 patients were smokers and 79 were non smokers.

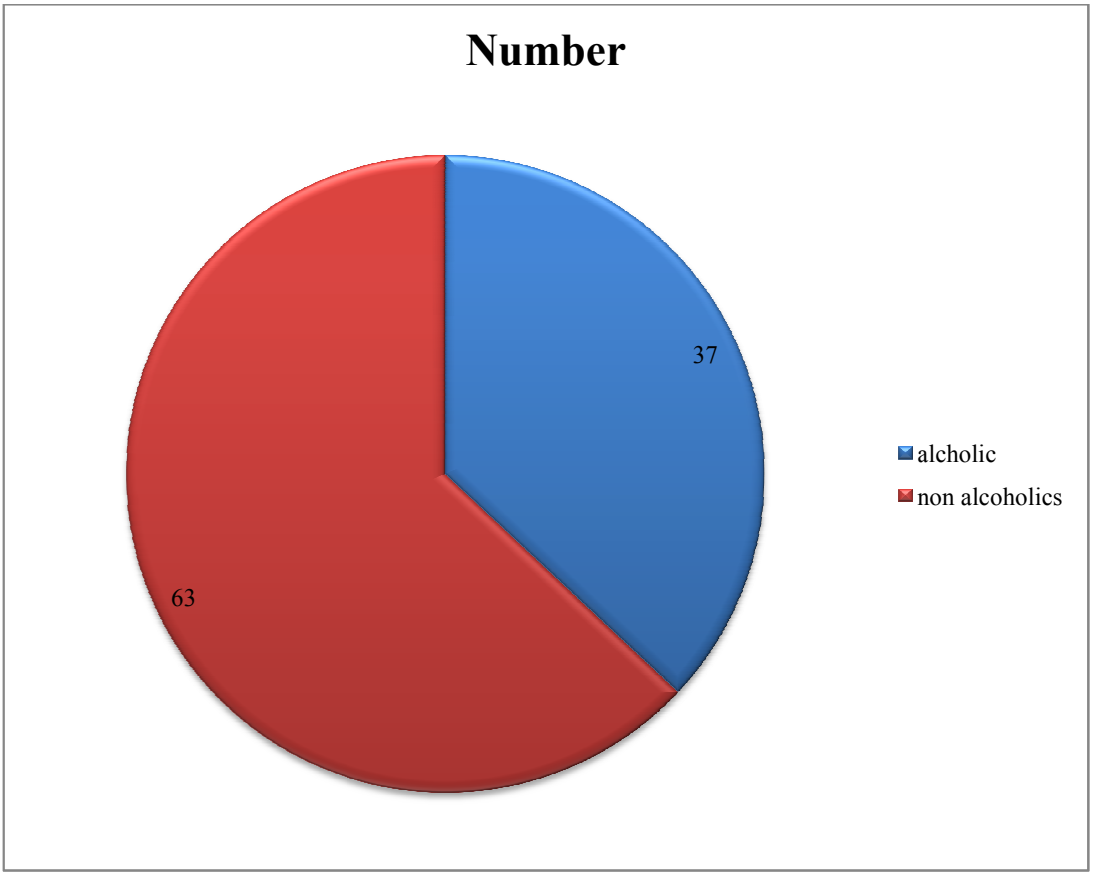


b. Alcoholics

Table. b

Alcholics	37
Non alcoholics	63

37 patients were alcoholics, while 63 were non alcoholics.

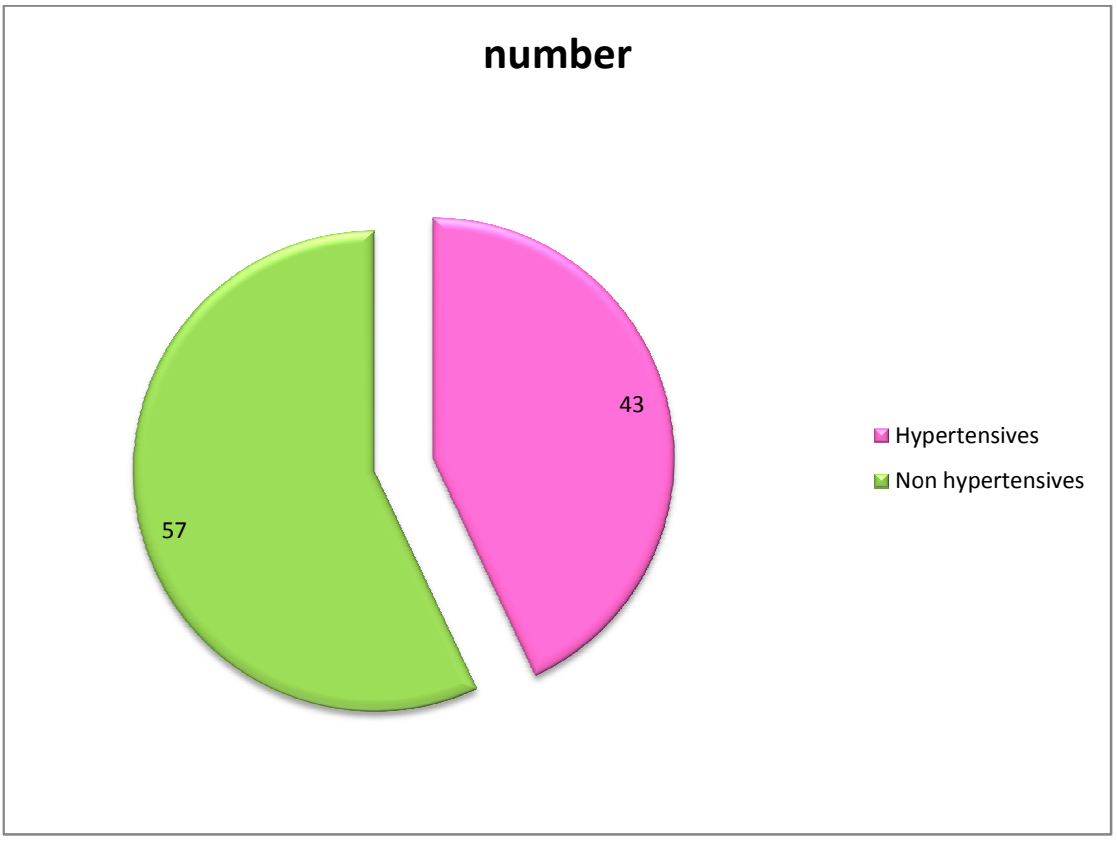


C) Hypertensives

Table. c

Hypertensives	43
Non hypertensives	57

Out of 100 patients, 43 were hypertensives and 57 were non hypertensives.

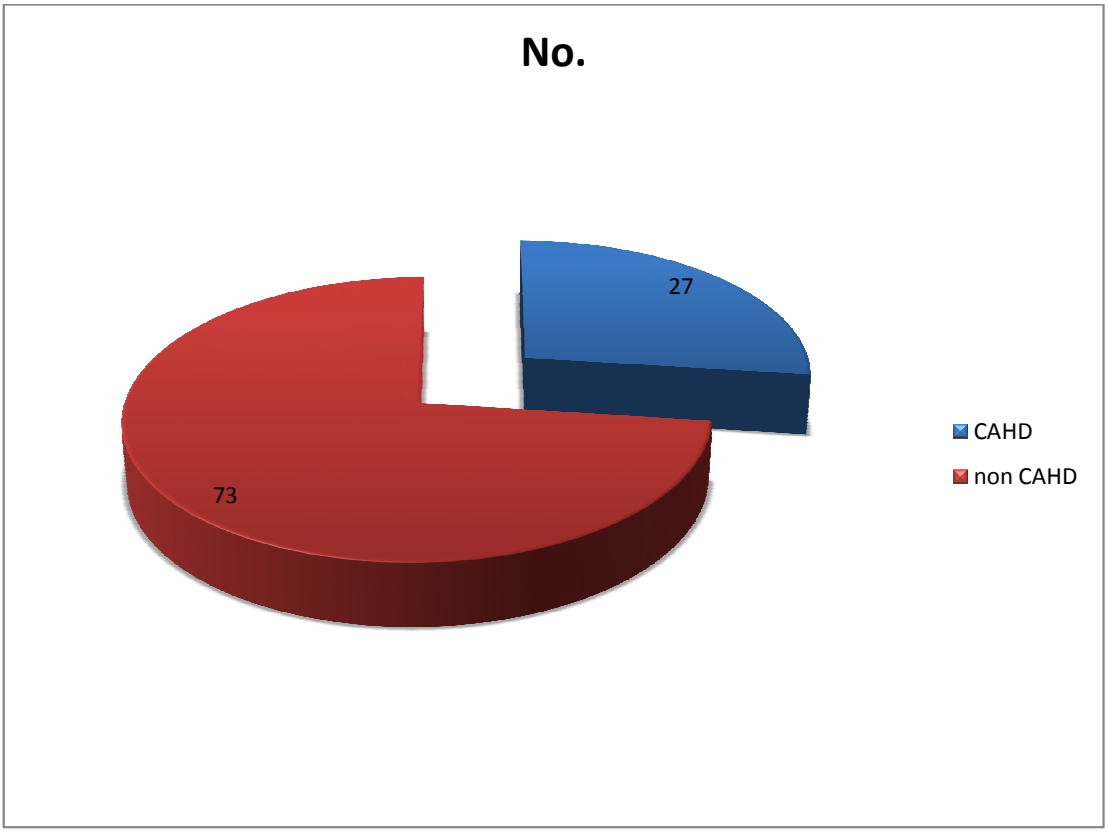


d)CAHD VS NON CAHD

Table. d

CAHD	27
No H/o CAHD	73

Out of 100 patients 27 patients had CAHD.

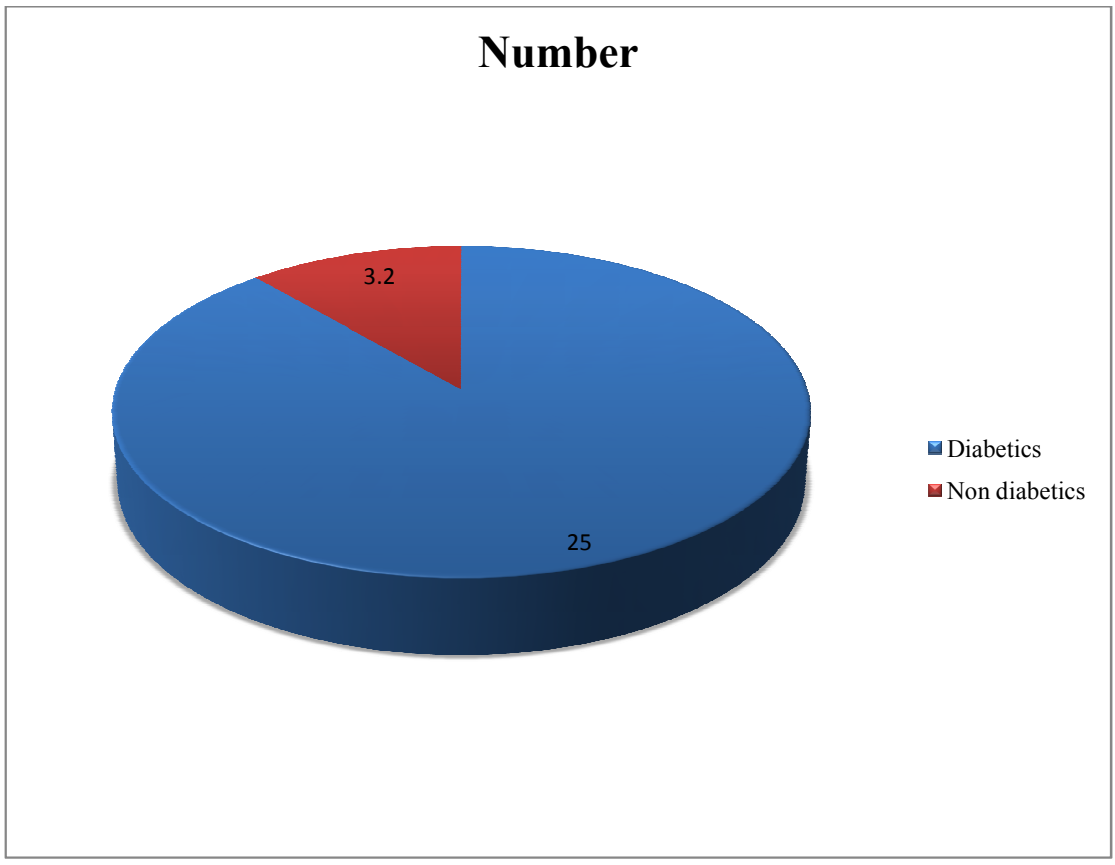


e.Diabetics vs Non Diabetes

Table. e

Diabetes mellitus	25
No H/o DM	75

Out of 100 patients, 25 were diabetic and 75 were non diabetic.

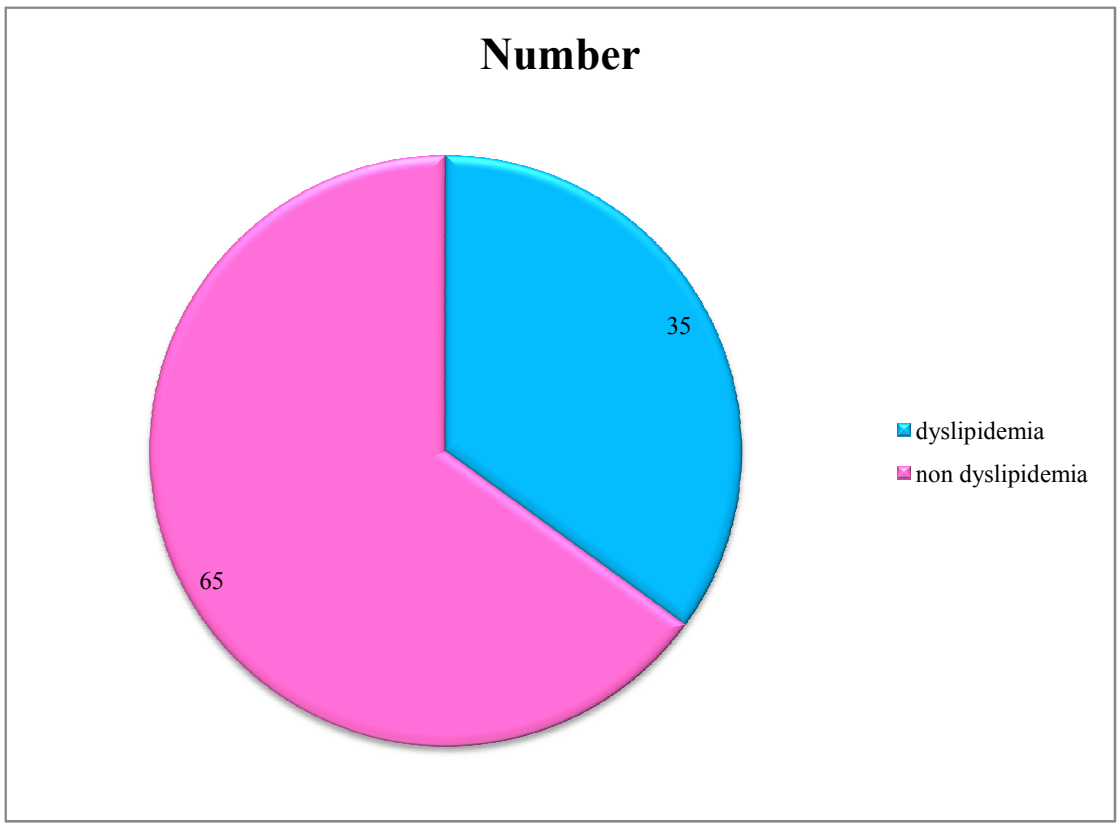


f) **Dyslipidemia**

Table. f

Dyslipidemia	35
Non dyslipidemia	65

Out of 100 patients, 35 are dyslipidemia and 65 are non-dyslipidemia.



Discussion

DISCUSSION

All the 100 patients selected for this study were thoroughly examined and the diagnosis of CVA was made clinically and CT Scan was taken in all the patients and detailed history regarding the risk factors, time of onset of stroke and the activity at the time of stroke were obtained.

Totally 100 stroke patients were studied, out of which 78 had thrombotic stroke, 14 had hemorrhagic stroke and 8 had embolic stroke.

Out of 78 thrombotic stroke patients, 30 had stroke in morning, 19 had stroke in the noon and 29 had stroke in the night .Out of 14 hemorrhagic stroke patients, 3 had stroke in morning, 3 in the noon and 8 in the night.

Out of 8 embolic stroke patients, 3 had stroke in the morning, 3 had stroke in the afternoon and 2 in the night.

Thirty-one publications[29-43] were reviewed and the data from the meta analysis of these world's thirty one published studies on the circadian timing of stroke onset indicates that, the risk of onset of acute stroke is increased during the early morning hours. These data are consistent across the various subtypes of stroke like ischemic stroke, hemorrhagic stroke and even the transient ischemic attacks, that the excess risk during the 6 AM to noon time period is significantly higher than would be expected by chance. Similarly, there is a significantly

lower risk of stroke during the nighttime hours i.e from midnight to 6 AM for each stroke subtype when it was compared with the normalized risk for the other 18 hours of the day. There are few reports that division of the numbers of strokes according to each of the 24hours of the day, and it is possible that these categorization of stroke frequencies into the arbitrary time periods for the meta-analysis is incorrect. Previous studies regarding time of onset of stroke.

Study	**Year**	**n**	**Population**	**Peak onset**
Hossmann	1971	131	Germany	1 am to 5 am
Marshall	1977	707	England	Midnight-6:00 AM
Agnoli et al	1975	256	France	6:00 AM-2:00 PM
Jovidic	1983	85	Yugoslovakia	8:00 AM-11:00 AM
Our study	2012-2013	100	India	10 PM to 6 AM

As summarized, results of four studies have been published. Our studies correlates with the studies of Marshall and Hossman whose studies reveal that the peak of onset of stroke is midnight to 6AM and 1AM to 5 AM respectively.

Our study and several others show that the onset of stroke occurs with the hypothesis that onset of the stroke onset coincides with low blood pressure, which is well known to occur early in the morning and when most people are a sleep.A fall in blood pressure will not lead to the fall incerebral blood flow (CBF) when the autoregulation is intact. The auto regulation is lost in the patients with cerebrovascular disease for a few days after an acute cerebrovascular episode. So there is evidence of impaired auto regulation in patients with cerebrovascular disease. It can also be possible that due to the impairment of vasomotor reflexes, which usually develops in the older age group people, the nocturnal fall in blood pressure is more profound in these age groups. It is also thought that blood pressure may sometimes fall below the lower limit for auto regulation, and thus leading to the fall in cerebral blood flow.

The onset of intracerebral hemorrhage and subarachnoid hemorrhage are more obviously related to blood pressure. In view to the several well known circadian rhythms in the humans, it is not surprising that there are several parameters associated with the events that may lead to the stroke onset, which has shown to fluctuate with a predictable

periodicity. The 24 hour variation in the cortisol secretion is well known and this secretion rhythm is not only passively driven by environmental events but, it is the product of an endogenous 'clock' within the organism.

Our study reveals that out of 100 stroke patients, 41 had stroke during mild activity, 22 had stroke during moderate activity, 8 had stroke during vigorous activity and 29 had stroke during sleep. In a study where 200 patients of ischemic stroke were studied, Koton et al. [(44)] found an approximately 2-fold increased risk of ischemic stroke within 2 hours of physical activity that did not reach nominal statistical significance. That study did not assess whether the risk is varied by habitual physical activity

Our study also reveals that the onset of stroke with activity is not statistically significant. Our study also does not assess whether this risk could be varied by habitual physical activity.

Risk factors were evaluated in our study. 43%were hypertensives. 35 % of the patients had dyslipidemia.25% of the patients were diabetics. 27% had the h/o CAHD. 37% of the patients were alcoholics. 21% of the patients were smokers.

Conclusion

CONCLUSIONS

1. Our study coincides with the results of studies of Marshall and Hossman which says that the onset of stroke is more during night.
2. Onset of stroke during night is observed in all types of stroke which is significant in our study.
3. The onset of the stroke is not associated with severity of the activity.
4. Most of the stroke has occurred during mild activity and sleep.
5. Risk factors evaluated in our study were
    - 43% - hypertension
    - 37% - alcoholism
    - 35% - dyslipidemia
    - 27 % - CAHD
    - 25% - Diabetes mellitus
    - 21 % - smokers

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Proforma

PROFORMA

NAME OF THE PATIENT:

AGE:

SEX:

IP NO:

ADDRESS:

OCCUPATION:

TIME OF OCCURANCE OF STROKE: MORNING / NOON / NIGHT

ACTIVITY AT THE TIME OF STROKE:

SLEEP/MILD/MODERATE/SEVERE

CHIEF COMPLAINTS:

HISTORY OF PRESENTING COMPLAINTS:

PAST H/O: HT/ DM / TIA/ PTB/ BA/ COPD/ CKD/ CAHD/ RHD/ LIVER DISEASE/ SEIZURES/ CVA

PERSONAL H/O: SMOKING / ALCOHOLISM

GENERAL EXAMINATION:

VITALS: BP- , PR- , RESPIRATORY

RATE AND PATTERN-

CLINICAL EXAMINATION-

CENTRAL NERVOUS SYSTEM-

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

ABDOMEN:

SPINE AND CRANIUM:

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

Blood sugar- , urea- , se creatinine-

F and PPBS-

Serum electrolytes: sodium - , potassium –

Blood hemogram- , TC- , DC-

, ESR-

Urine albumin- , sugar- , deposits-

Total cholesterol- , TGL –

ECG-

Chest xray-

CT brain –

MRI brain –

ECHO-

FINAL DIAGNOSIS-

Master chart

S.No	Ip. No	Name	Age	Sex	Past h/o	Personal h/o	Time	Activity at stroke	Clinical diagnosis	CT findings	F&PP bsugar in mg%	Urea Creat In Mg%	Tcholes/ TGL in mg%	ECG	Cxray	Echo	Others
1	16389	Velsamy	47	M	DM/ CAHD	Alcoholic/ smoker	noon	mild	Thrombotic stroke	Lt MCA &PCA infarct	83/120	38/ 0.7	240/204	Tinversion V5,V6	cardiomegaly	RWMA mod LV sys dysfn, grade 2 diastolic dysfn	
2	16299	Vella pandi	65	M	-	Smoker/ alcoholic	noon	severe	Embolic stroke	Lt MCA infarct	89/ 115	21/ 0.9	180/ 118	Atrial fibrillation	normal	-	
3	25711	Kavitha	40	F	HT	-	night	sleep	Thrombotic	normal	110 /145	22/ 1	170/ 130	LVH strain, AL ischemia	cardiomegaly	-	160/90 mmHG
4	14102	Palavesam	72	F	TIA/ Hyper thyr	-	morning	mild	embolic	RT MCA infarct	112/ 135	33/ 1.1	190/ 184	AF	normal	-	-
5	25791	Natarajan	65	m	CAHD	alcholic	morning	mild	hemorrhagic	Hemorr In lt Thalamus Inter Capsul region	111/ 143	24/ 1.1	180/142	NSTEMI	normal	-	160/100mm hg
6	35104	madasamy	72	m	-	alcholic	noon	moderate	thrombotic	Infarct Lt mca	98/119	29/ 1.4	170/ 114	Normal	normal		
7	35809	veeravan	67	M	HT	-	night	mild	thrombotic	Infarct in RT MCA	94/ 116	39/ 1.5	154/ 113	normal	normal		
8.	35161	senthil	35	M	-	alcoholi	night	sleep	Hemorrhagic stroke	Hemorrhageinrt ventricle	131/ 292	3/1.2	289/ 212	n	n		
9.	33124	kathirvel	60	m	-	Smoke/alc	night	sleep	thrombotic	Infarct in ltparieto Occipital region	97/ 160	40/ 1.4	180/ 146	n	n		
10.	35187	mohamed	76	m	HT	-	noon	moderate	Hemorrhagic stroke	Left Frontal Hemorr hage	118/ 207	40/ 1.5	165/ 130	n	n		
11.	24186	annathai	65	f	HT/ TIA		morning	moderate	thrombotic	Rt MCA infarct	113/ 193	32/ 1.3	294/ 212	n	n		
12	65836	sarvanan	70	m	HT/	alc	noon	mild	Hemorrhagic stroke	Hemorrhage in Lt fronto	108/ 178	39/ 1.4	190/ 134	n	n		200/ 100

										Parietal area							mmHG
13	67165	ram	70	m	HT/ DM	Alcoholic smoker	night	moderate	thrombotic	Infarct LT mca	180/ 298	35/ 1.4	312/ 243	LVH strain	n		160/90 mmhG
14	60600	Booda than	60	m	HT	Alcoholic smoker	night	sleep	hemorrhagic	Hemorrhage in rtmca	112/ 166	40/ 1.5	198/ 115	LVH	cardiomegal y		200/110
15	40355	sitaram	50	M		alcoholic	morning	mild	thrombotic	Multiple infarct in rt cerebral hemisphere	97/ 160	35/ 1.4	188/ 115	n	n		
16	30355	thiruvengadam	45	m	-	alcoholic	noon	mild	Thrombotic	Multiple infarct in rt Cerebral hemisphere	112/ 145	40/ 1.4	190/ 144	n	n		
17.	59550	armugam	59	m	HT	Alcoholic smoker	night	sleep	thrombotic	Rtmca infarct	122/ 149	39/1	189/ 137	LVH	Cardiomegal y		
18	37705	avudayammal	80	f	Ht/ DM		night	sleep	thrombotic	Lt mca	189/ 222	33/ 1.5	312/ 276	Inferior wall ischemia	n		
19	68466	nagarajan	62	m	HT/ DM	smoker	morning	mild	thrombotic	Rtmca	120/177	28/0.5	288/ 212	n	n		
20	38466	thirumani	63	m	HT	alcoholic	noon	mild	thrombotic	Rtmca	110/167	30/1	198/ 146	LVH	n		
21	75836	sankaran	66	m	HT/ DM	Alcoholic smoker	night	sleep	hemorrhagic	Hemorrhage in ltmca	190/ 248	33/ 1.2	292/ 226	LVH	n		
22	66222	meenakshi	66	f	HT/ DM		noon	sleep	thrombotic	Rtmca	140/ 201	34/ 1.3	288/ 205	n	n		
23	213234	sundar	48	m	-	alcoholic	morning	mild	embolic	Rtmca infarct	112/ 144	33/ 1.3	188/ 134	Atrial fibrillation	n		
24	24442	rani	40	f	CAD		noon	sleep	embolic	Lt mca infarct	121/ 177	39/ 1.5	198/ 113	Atrial fibrillation	n	Severe MS/ LAE+/pht	
25.	214232	kani	45	f	-		morning		thrombotic	Rtmca infarct	120/ 165	37/ 1.4	188/ 112	n	n	n	
26	234543	shyam	48	m	-	alcohlic	noon	severe	thrombotic	rtmca infarct	112/ 198	40/ 1	323/246	n	n		
27	213423	ibrahim	50	m	-	-	noon	mild	thrombotic	Lt mca infarct	121/ 199	39/ 1.4	345/ 243	n	n		
28	312021	kavitha	70	f		DM	morning	severe	hemorrhagic	Hemorrhage in Rtparieto occipital area	214/344	40/ 1.6	332/ 286	n	n		200/ 110mm Hg
29	213423	arun	60	m	-	-	night	sleep	hemorrhagic	Hemorrhage in Rt frontal area	210/ 334	40/1.5	312/ 268	n	n		180/130 mmhg
30	214234	rahim	73	m	-	HT	night	mild	Thrombotic	Lt mca infarct	112/ 154	20/0.7	169/ 112	LVH	n		

31	214341	mahima	60	f	-	DM	noon	moderate	thrombotic	Rtmca infarct	212/ 300	33/ 1	241/ 195	n	n		
32	312293	ravi	77	m	-	DM	morning	moderate	thrombotic	Lt mca infarct	232/ 336	40 1	212/ 189	n	n		
33	49015	savier	56	m	Smoker alcholic	CAHD	noon	moderate	thrombotic	Lt mca infarct	112/ 145	34 1.4	298/ 212	Old asmi	n		
34	25836	rasammal	70	f		HT/DM	night	sleep	thrombotic	Lt mca infarct	240/523	40/1	312/ 200	n	n		
35	37165	ponnusamy	82	m	alcoholic		noon	moderate	thrombotic	Lt mca infarct	121/ 192	33/ 1.3	170/ 143	LVH strain,infer ior wall ischemia	n		
36	58430	mariamal	60	f		HT/DM	night	sleep	thrombotic	rtmca infarct	189/ 280	32/ 1.4	243/ 179	n	n		
37	55836	rajendran	69	m		HT/DM	night	sleep	thrombotic	ltmca infarct	199/ 290	33/1.3	256/ 180	n	n		
38	68430	shanmugavadi vu	58	F		HT	morning	MILD	thrombotic	Rtmca infarct	110/ 140	32/ 1.3	298/ 234	n	n		
39	35891	natarajan	41	m	Smoker/ alch		noon	moderate	hemorrhagic	Rt ICH with intraventricularext	121/ 198	29/ 1.5	187/ 150	n	n		
40	58447	selvi	67	f		HT/DM	night	sleep	thrombotic	Rtmca infarct	180/ 240	33/ 1.4	198/ 144	n	n		
41	42490	sudali	60	f		HT	morning	mild	thrombotic	Lt mca infarct	111/ 144	39/ 1.2	162/ 136	n	n		
42	43953	utthandu	50	m			morning	mild	thrombotic	Rtmca infarct	112/ 155	28/ 1.2	156/ 111	n	n		
43	43903	nellaippan	70	m			morning	mild	thrombotic	Lt mca infarct	113/ 144	33/ 1.4	166/ 122	n	n		
44	42653	durai	75	m	alcoholic		mornigng	mild	thrombotic	Lt mca infarct	103/ 124	33/ 1.4	228/ 198	n	n		
45	41467	vadivelu	21	m	Smoker/ alch		night	mild	thrombotic	Lt mca infarct	103/ 149	33/ 1.5	160/ 122	n	n		
46	42708	iyanal	65	f			noon	moderate	thrombotic	Lt mca infarct	103/ 155	33/ 1.5	232/ 217	Lateral wall ischemia	n		
47	42705	parvathi	88	f			noon	mild	thrombotic	Lt mca infarct	113/ 144	33/ 1.4	166/ 122	n	n		
48	42687	sornavadivu	75	f		HT/DM	morning	mild	thrombotic	Lt mca infarct	78/ 135	38/ 1.4	145/ 122	n	n		
49	43997	perumal	74	m			night	sleep	thrombotic	High parietal infarct	85/114	34/ 1.3	154/ 122	n	n		
50	63162	hariharan	56	m		CAHD	night	sleep	thrombotic	Rtmca infarct	88/	38/	150/	n	n		

											113	1.4	120				
51	54361	kandasamy	67	M		CAHD	night	sleep	thrombotic	Lt mca infarct	91/ 112	40/ 1.4	152/ 122	n	n		
52	41572	viswanathan	58	m		CAHD	night	sleep	thrombotic	Lt mca INFARCT	90/125	33/0.9	160/130	N	N		
53	64105	selvarajan	61	m	Smoking	CAHD/	morning	moderate	thrombotic	Lt mca infarct	80/140	30/0.8	140/110	n	n		
54	53521	murugesan	73	m	Smoking/Alcoholism	CAHD/	orning	mild	thrombotic	Lt mca infarct	89/133	33/1.0	150/113	n	n		
55	36431	nakulan	53	m		HT	night	Sleep	thrombotic	Rtmca infarct	82/150	32/0.8	170/110	n	n		
56	35413	vijayaraghavan	48	m		HT/DM	morning	moderate	thrombotic	Lt mca infarct	97/160	33/0.9	156/134	n	n		
57	33514	manoharan	55	m		CAHD/DM	night	mild	thrombotic	rtmca infarct	110/180	31/1.0	160/126	n	n		
58	38156	dhinakaran	68	m	Alcoholic	CAHD/	morning	mild	thromotic	Lt mc infarct	85/140	42/0.8	150/113	n	n		
59	32501	gunasekaran	61	m		HT	morning	mild	thrombotic	rtmca infarct	84/150	50/0.8	170/123	n	n		
60	32506	gunasankaran	62	m	alcholic	HT	morning	mild	thrombotic	Rtmca infarct	85/150	40/0.9	143/112	n	n		
61	73721	Saraswatiammal	61	f		HT/DM	night	sleep	thrombotic	Lt mca infarct	99/180	36/1.5	180/154	n	n		
62	41575	murugan	60	m		HT/Alcohol	morning	severe	thrombotic	Lt mca infarct	85/150	31/0.8	173/124	n	n		
63	32568	manimaran	62	m		CAHD/DM	morning	moderate	thrombotic	Lt mca infarct	96/164	32/0.8	200/173	n	n		
64	35512	dharmalingam	65	M		CAHD	night	mild	thrombotic	normal	89/112	39/1.4	165/112	n	n		
65	63125	kartik	61	m	alcoholic	CAHD	night	sleep	thrombotic	normal	81/141	40/.8	152/ 112	n	n		
66	64332	gunasekar	59	m		CAHD/DM	night	moderate	embolic	Rtmca infarct	82/112	34/1.3	154/112	Atrial fibrillation	cardiomegaly		
67	46122	gopalan	72	m	alcoholic	CAHD/DM	morning	mild	thrombotic	Lt mca infarct	181/241	40/.8	212/ 112	Old awmi	n		
68	49586	kalidasan	52	m	Smoking/alcohol		night	sleep	thrombotic	normal	89/112	39/1.4	165/112	n	n		
69	43758	vasanth	56	n	Smoking/alcohol	CAHD	morning	moderate	thrombotic	normal	81/141	40/.8	152/ 112	Old IWMI	n		
70	51523	meenakshi	55	f		CAHD	night	sleep	thrombotic	Lt mca infarct	96/164	32/0.8	200/173	Old AWMI	n		
71	41532	ravikumar	60	m		HT	night	mild	thrombotic	Rtmca infarct	88/ 118	40/1.5	177/145	LVH	n		
72	61108	Kumara shankar	65	m	alcoholism	CAHD	morning	moderate	thrombotic	Lt mca infarct	111/141	40/1.4	150/112	N	n		
73	25438	kuppusamy	53	m	alcoholism	CAHD	morning	moderate	thrombotic	Lt mca infarct	114/145	40/1.4	140/102	N	n		
74	53254	mujibeer	59	M		DM/HT/CAHD	morning	moderate	thrombotic	Rtmca infarct	180/ 243	38/ 1.6	212/ 188	Old awmi	n		
75	26858	ramalingam	53	M	Smoking/alcoholism	CAHD	night	sleep	thrombotic	Lt mcainfarct	111/178	40/1.3	156/112	Old IWMI	n		
76.	21615	sivasubramanian	59	M		CAHD	noon	severe	thrombotic	Rtmca infarct	112/163	39/1.2	154/117	Old AWMI	n		

77	35462	dhanabalan	56	M		CAHD	morning	severe	thrombotic	Lt mca infarct	110/154	40/1.2	150/124	Old IWMI	n		
78	28017	Abdul	79	M	smoking	CAHD	noon	mild	embolic	Rtmca infarct	112/145	41/1.5	152/112	Atrial fibrillation /IWMI	cardiomegaly		
79	28481	sundar	76	m	Smoking/alcoholism	CAHD	morning	mild	embolic	Rt MCA with PCA, watershed zone infarct	212/312	40/.7	170/134	Multiple VPCs	cardiomegaly	Mild LV systolic dysfn, large LV apical clot	
80	28292	thangavel	53	m		CKD	night	sleep	hemorrhagic	Lt ICH	70/112	138/7.6	212/190	normal	n		
81	29860	lakshmi	65	f		HT	noon	mild	thrombotic	normal	72/119	21/0.6	150/88	n	n		
82	65332	muthu	55	m	alcoholism		night	severe	thrombotic	Lt mca infarct	112/145	41/1.5	152/112	n	n		
83	35421	ramnath	45	m	alcoholism	-	noon	mild	thrombotic	normal	72/119	23/0.6	150/89	n	n		
84	45231	kannan	47	m	smoking	HT	morning	mild	thrombotic	Rtmca infarct	112/145	41/1.5	152/112	n	n		
85	87768	ramakrishnan	63	m		HT	morning	severe	thrombotic	Lt mca infarct	108/145	42/1	152/115	n	n		
86	25480	esaki	60	M	Smoking/alcohol	HT	morning	mild	thrombotic	Rtmca infarct	112/150	36/1.2	144/112	Inferior wall ischemia	n		
87	25521	anwar	60	M	Smoking/alcohol	HT/CKD	noon	mild	thrombotic	Rt MCA with PCA, watershed zone infarct	116/166	110/7	198/186	Lvh strain	n		
88	23539	karupaiya	64	m		HT/ckd/dm	night	mild	hemorrhagic	Hemorrhage in rt parietal lobe	121/189	96/4	196/158	LVH strain	n		
89	29195	sudalai	45	m	Smoking/alcohol	HT	night	sleep	hemorrhagic	RT ICH with dilated ventricle with midline shift	121/154	44/1.3	176/110	LVH	n		
90	45841	kalaiselvi	47	f		HT	morning	moderate	hemorrhagic	RT ICH	121/154	44/1.3	176/110	Sinus bradycardia	n		
91	58430	vadivukani	58	f		HT	morning	mild	thrombotic	Rtmca infarct	112/145	23/.5	298/232	n	n		
92	56232	valli	65	f		DM/HT	noon	sleep	thrombotic	Rtmca infarct	200/288	23/.6	288/254	n	n		
93	39105	kavya	56	f			night	moderate	thrombotic	Lt mca infarct	112/188	24/0.7	144/115	n	n		
94	47615	bhavana	50	f		CAHD	night	sleep	embolic	Rtaca infarct	102/165	32/.8	150/112	AF	n	Lv systolic dysfunction,LV clot	

95	48625	ramnya	40	f		HT	night	mild	hemorrhagic	ICH with intraventricular extension	82/178	28/0.7	164/125	n	n		
96	37619	suganya	66	f			noon	sleep	thrombotic	Rtaca infarct	92/150	28/1	144/115	n	n		
97	27915	muthu	62	f			night	moderate	thrombotic	Lt mca infarct	91/148	24/1.7	286/215	n	n		
98	17676	praba	42	f			noon	moderate	thrombotic	Rtmca infarct	112/188	24/1.1	294/245	n	n		
99	17915	vasantha	40	f		DM	night	mild	thrombotic	Lt mca infarct	151/208	24/0.6	299/202	n	n		
100	17421	ravi	40	M	smoker	HT	night	mild	thrombotic	Rtmca infarct	102/135	23/1	122/144	n	n		