# A STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE CEREBROVASCULAR ACCIDENTS

*Dissertation submitted to*

**THE TAMILNADU Dr.MGR MEDICAL UNIVERSITY**

*In partial fulfillment of the regulations*
*For the award of the degree of*

**M.D.BRANCH-1**

**GENERAL MEDICINE**


**GOVERNMENT STANLEY MEDICAL COLLEGE AND HOSPITAL**
**CHENNAI, INDIA**

**MARCH 2010**

# CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE CEREBROVASCULAR ACCIDENTS"** submitted by Dr.M.HEMA to The TamilNadu Dr.M.G.R. Medical University Chennai is in partial fulfillment of the requirement of the award of M.D DEGREE BRANCH I (General medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

**Signature of Professor & HOD** **Signature of Dean**

# DECLARATION

I hereby declare that the dissertation title **"A STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE CEREBROVASCULAR ACCIDENTS"** was done by me at Stanley medical college and hospital during the year 2008-2009, under the guidance and supervision of Prof. MAGESH KUMAR, M.D. Professor of Therapeutics, Department of Internal Medicine.

This dissertation is submitted to the Tamilnadu Dr.M.G.R Medical university towards the partial fulfillment of requirement for the award of M.D. DEGREE Branch-1 in General Medicine.

Place : Chennai

Date :

**Dr. M. HEMA**

# ACKNOWLEDGEMENTS

I wish to express my gratitude to Dr.A.PRIYA, M.S., D.O., Dean Government Stanley hospital for allowing me to avail the facilities needed for the study.

With a deep sense of indebtedness and reverence, I thank Prof.S.RAMASAMY, M.D, Professor and the head of the Department of Internal Medicine, Stanley medical college, for permitting me to do this study and for his encouragement.

I am extremely thankful to my unit chief PROF. S. MAGESHKUMAR, M.D for his valuable guidance and constant help.

I would like to express my sincere gratitude to my Assistant Professors, Dr.Samuel Dinesh, M.D. and Dr.Madhavan,M.D., and to the Medical Registrar, Dr.Vasumathi, for their generous help and guidance.

# CONTENTS

# INTRODUCTION

Cushing first described hemodynamic changes after acute intracerebral hemorrhage. [1] Cardiac abnormalities were described with various CNS diseases including seizures, trauma, ischemic stroke, ICH and less commonly tumors, electroconvulsive therapy and meningitis. [2] More recently emotion and stress induced cardiomyopathy has been described. [3-5]

The anatomy and physiology of pathways involved in brain-heart interaction have been elucidated in both animal and human studies. The ability to reproduce the arrhythmia by activation of the sympathetic nervous system suggested a neurogenic mechanism. [6, 7]

The medulla has been described as the principal site of vagal parasympathetic and sympathetic areas involved in cardiac control. [8, 9]

In addition both anatomical and physiological evidences implicate the hypothalamus in cardiac control. [10] Electrical stimulation experiments suggests a posteriorly located area of cardiovascular sympathetic control and anterior parasympathetic control region. [11]

Beattie an colleagues first described cardiac arrhythmias after hypothalamic stimulation.[12] Arrhythmias from hypothalamic stimulation were subsequently confirmed in other animal models. [13, 14] Areas of cerebral cortex with connection to autonomic nervous can also elicit cardiac response. The autonomic – emotional interaction with cardiovascular function have been linked to central nucleus of amygdale. [15, 16]

Stimulation of orbito frontal, cingulated and temporal regions can elicit a cardiac response. [17-19] Majority of evidence suggests that the insular cortex has a pivotal role in integrating autonomic response and is strongly associated with adverse cardiac events after neurological injury.

## ISCHEMIC AND HEMORRAGIC STROKE

Cardiac abnormalities occur in 60 to 70 percent of patients after stroke. [20] The most common disturbance include ECG abnormalities, cardiac arrhythmias and myocardial injury and dysfunction distinguishing cardiac abnormalities directly caused by stroke. It however remains difficult because the prevalence of preexisting cardiac disease is high particularly among patients with ischemic stroke. [21, 22] More importantly, cardiac disturbances are most common cause of death

stroke accounting for up to 6 percent of unexpected death during the first month. [23]

## THE INSULAR CORTEX

The insular cortex has widespread connectivity with other areas of brain that are involved in autonomic control. [24] Oppenheimer and colleagues first identified the insular cortex in rats as a site from which lethal cardiac arrhythmias and myocardial damage could be produced, resembling changes seen in patients after stroke and sudden death in patients with epilepsy. These micro stimulation experiments in the rat posterior insular cortex produced stereotyped ECG changes from progressive atrio-ventricular block leading to complete heart block interventricular block, QT interval prolongation's, ST segment depression, ventricular ectopy and finally death in asystole.

The ECG changes were directly correlated with pathological changes in the myocardium called myocytolysis that were thought to be induced by excess activation of intrinsic cardiac sympathetic nerves. [25] Rat model of cerebral infarction was associated with increased renal sympathetic nerve activity and elevated nor epinephrine levels. [29]

Stroke involving the insular cortex in humans abolished the nocturnal decline in blood pressure, prolonged the corrected QT interval, increased ventricular tachyarrhythmia's, and resulted in higher plasma nor epinephrine levels. [29] Clinical observational studies among stroke patients also suggest a loss of parasympathetic tone[30], loss of nocturnal vagal dominance[31], and increased sympathetic tone. [32]

This evidence collectively support the belief that stroke can alter cardiovascular tone by directly damaging the insular cortex or other interrelated areas shifting the balance towards a predominance of sympathetic activation.

Elevated baseline heart rate and blood pressure were noted after right-sided injury and significantly increased baroreflex sensitivity was obtained after left sided injury. [33]

An observational study of stroke patients indicated an increase incidence of sudden death among patients with right insular strokes. [37]

Right middle cerebral artery strokes were associated with increased incidence of supraventicular tacyarrythmias. [38]

Left parieto insular stroke was associated with an increased incidence of new onset atrial fibrillation. [40]

In a prospective study of patients with stroke and transient ischemic attacks left insular stroke were independent risk factor for adverse cardiac outcome in patients without heart disease. In analysis of North American symptomatic end arterectomy trial (NASCET), long term risk of sudden death was significantly increased in patients with left brain infarction. [42]

These observations highlight the complex interaction of insular cortex and other autonomic centers of brain. The relative balance of excitatory and inhibitors pathways involved in cardiovascular control can vary with the state of arousal as well as with involvement of adjacent inhibitory pathways in the fronto parietal cortex. Evidence suggests that in human, stroke isolate to the left anterior insula or the right fronto- parietal cortex sparing the insula will have similar effects on cardiovascular outcomes. [43]

## THE ELECTROCARDIOGRAM

ECG abnormalities are common occurring, in 92 percent of patients with acute stroke. [44] In 1947, Byer and colleagues first

described marked QT prolongation with large T and U waves on the ECG of four patients with stroke. [45] Subsequently Burch and colleagues described an ECG pattern after stroke consisting of large inverted T waves, prolonged QT intervals and large septal U waves that has become distinctive of cerebral vascular injury. [45]

In the 17 abnormal ECGs reported in their study, the abnormalities were most frequently observed after SAH, followed by ICH and ischemic lesion. There was significantly increased incidence of ST depression, prolongation of QT interval, T wave inversion, ventricular premature beats among stroke patients compared to age and sex matched controls.

**QT PROLONGATION**

The most common stroke related ECG abnormality is QT prolongation. A myocardial repolarization abnormality associated with increased risk of a characteristic life threatening cardiac arrythmias. [49]Among stroke patients prolonged QT interval is frequently observed after hemorrhagic stroke occurring in 45 to 71 percent of patients with SAH or ICH compared to 38 percent of those with ischemic strokes. [44, 47, 51, 52]

Ventricular tacyarrythmia including sudden death are often preceded by QT prolongation in patients with SAH. [51, 53] QT prolongation accompanied by U waves and T wave changes, often correlates with elevated systolic blood pressure. [44]

## REPOLARIZATION ABNORMALITIES

The similarities between ECG changes due to acute myocardial ischemia and infarction and those associated with stroke are most striking with the repolarization abnormalities involving the ST segment leading many investigators to hypothesis coexisting cardiac disease as the primary cause.

ST segment changes occur in[22, 23, 24, 25] percent of patients with ischemic stroke which is complicated by increased prevalence of cardiac disease in the subgroup of stroke patients. [48, 54]

New T wave abnormalities appear in approximately 15 percent of the patients with acute stroke, even in the absence of electrolyte disturbance or primary ischemic heart diseases. [48] Inverted or flat T waves has also been reported in upto 55 percent of patients with SAH, the stroke with lowest prevalence of coexistent cardiac diseases. [55, 56]

More direct evidence comes from an autopsy study of five patients with ECG changes who died of SAH none of the of the autopsied patients had evidence of epicardial coronary disease. [57]

Kono and colleagues performed detailed cardiac assessments on 12 patients with acute SAH and ST elevation in the ECG although patients were found to have apical wall motion abnormalities on the echocardiogram, there was no evidence of coronary artery stenosis or coronary artery vasospasm on angiography. [58]

These findings, along with the observation that stroke induced ECG are evanescent, resolving over a period of days to months with little residuum, argue against myocardial ischemia or infarction as the only cause of repolarization changes on ECG.

## Q WAVES AND U WAVES

New Q waves similar in morphology to those observed in acute myocardial infarction are also common after acute stroke, reported in approximately 10 percent of patients with acute stroke. [47, 52]

Q waves may be transient or proceed the evolutionary changes in myocardial infarction. [47, 52] Cardiac evaluation may be necessary in

patients with q waves and ST segment changes, if they are older than 65 years with coronary risk factors such as diabetes mellitus. [59]

New U waves occur in isolation or with T waves and QT abnormalities in approximately 13 to 15 percent of the patients with acute ischemic stroke and SAH. [44, 60] Isolated U waves were equally distributed between ischemic and hemorrhagic strokes, but the combination of U waves and QT prolongation was more common with hemorrhagic strokes. [44] There is no relationship between presence of U waves and stroke mortality, suggesting that this ECG change did not require any treatment.

## CARDIAC INJURY AND DYSFUNCTION

Evidence from autopsy series in both ischemic and hemorrhagic stroke indicate that cardiac dysfunction can occur in the absence of underlying CAD. [57, 70, 71] Subendocardial hemorrhages were initially described in patients dying after acute strokes and seizures. Further studies suggested that these pathological changes were secondary to excessive sympathetic stimulation. [72-75] Offerhaus and Van Gool showed convincingly that following intracranial hemorrhage, there was an increase in cardiac tissue catecholamines. [76] A broader description of

these catecholamine-induced subendocardial lesions included scattered foci of swollen myocytes surrounded by infiltrating monocytes, interstitial hemorrhages, and myofibrillar degeneration. [77] Collectively, the characteristic pathological changes have been called contraction band necrosis, coagulative myocytolysis, or myofibrillar degeneration. In patients with CAD, myocardial necrosis typically follows as vascular distribution. [78] Neurogenic myocardial injury can be visible within minutes of onset, with appreciable differences observed on a cellular level. In myocytolysis, mononuclear infiltration predominates, with early calcification, and myocardial cells in a hyper-contracted state with contraction bands. [78]

Further evidence of a neurogenic mechanism of cardiac injury comes from studies of cardiac function after SAH, which typically affects younger patients without a history of coexistent cardiac disease. Global or regional left ventricular systolic dysfunction on echocardiogram has been described after SAH with an approximate incidence of 10 to 28 percent. [58, 79, 80] The severity of neurological injury is strongly associated with the presence of left ventricular dysfunction. [81] Similarly, diastolic dysfunction is also common after SAH, is associated with the severity of neurological injury, and may be the cause

of pulmonary edema seen in these patients. [82] The onset of left ventricular dysfunction occurs early in the course of SAH. [83, 84] In the largest study to date, a regional wall motion abnormality was most likely to be present within the first 2 days. [84] The prevalence then declined during days 3 to 8 after hemorrhage. In this same study, the authors demonstrated complete or partial resolution of left ventricular dysfunction in the majority of patients during the acute hospitalization. Cardiac dysfunction appears to be reversible in most cases and normalizes over time. [85-88]

There is a well-demonstrated, unique, apical-sparing pattern of regional wall motion abnormality that differentiates SAH patients from those with the typical patterns seen in CAD. The most frequently affected segments are the basal and mid-ventricular portions of the anteroseptal and anterior walls and the mid-ventricular portions of the inferoseptal and anterolateral walls. [84, 89] A retrospective study of SAH patients demonstrated reversibility and both global and regional left ventricular dysfunction, most commonly affecting the anterior and anteroseptal walls that do not involve the apex. [90] Younger age and anterior aneurysm position were independent predictors of this pattern. [91] This apical-sparing pattern of left ventricular dysfunction argues

against an obstruction or vasospasm of coronary arteries against an obstruction or vasospasm of coronary arteries and provides indirect evidence of neurally mediated mechanism of injury.

Experimental and clinical studies have addressed a neurogenic catecholamine-mediated mechanism of injury or "catecholamine hypothesis" of cardiac dysfunction. [92]

# REVIEW OF LITERATURE

Abnormalities of ECG are extremely useful in the recognition of heart disease. But they may also occur in extra cardiac condition. ECG abnormalities described in neurological disease are among the most striking deviation from the normal.

First account of ECG changes which consisted of Upright T waves prolonged QTC in patients with subarachnoid hemorrhage was published in 1947.

Not until 1953 however when Levine reported on ECG changes attributed to myocardial infarct in patients with SAH whose heart at autopsy was normal

Subsequently Burch and colleagues[101] described abnormal T waves, prominent U waves, long **QTC** in patients with CVA.

Crop and Manning[102] analysed ECG in 29 patients with SAH. Flat or negative T wave were noted in 15patients, prolonged in QTc 14, ischemic ST segment in 11patients.

Schuster[103] had observed QTC and bradycardia as characteristic of SAH

Frentz and Gorsmen[104] observed depression of ST segment is the most common abnormality in 11 out of 15 patients with intracerebral hemorrhage.

Kreus and his coworkers[105] observed high frequency of ECG in CNS lesions. 25 out of 35 patients with SAH had ECG abnormalities.

Miller and Abildskov, [106] most of 50 patients had non specific ST-T wave abnormalities. They observed high incidence of notched T waves.

Litcher and schaub[107] in a series of 418 patients with various cardiovascular disorder found that only 13 or 3. 1 showed ECG abnormalities.

Levine[108] in 1953 referred to cascading T wave which become replaced by RS-T segment in patients with rupture of aneurysm of circle of wills.

Burch, Mayer, Abildskov(1954) found 10 abnormal ECG in patients with CVA admitted to manning hospital in 1950.

Commonest abnormalities were prolonged QT interval, T waves of increased amplitude and duration which were negative and some large U wave.

Wasserman et al [109](1956) described prolongation of stand deep wide T waves and inverted U waves.

Crop and Manning (1960) and Hugenholtz [110] (1962) stressed the absence of any clinical evidence of coronary infarction and normality of the heart and coronary arteries at autopsy. Thus there is marked difference in the reported frequency of ECG abnormalities in patients with CNS lesion.

## CEREBRO VASCULAR DISEASE –A REVIEW [(i)]

Cerebrovascular disease include some of the most common devastating disorders ischemic stroke, hemorrhagic stroke, cerebrovascular anomalies such as intra cranial aneurysm and AV malformation.

They cause 2000000 deaths each year in the developed countries such as USA and major cause of disability.

**STROKE** - Abrupt onset of neurological deficit that is attributable to focal vascular cause. Clinical manifestation are highly variable because of complex anatomy of brain and its vasculature cerebrovascular disease is caused by one of the several pathological process involving blood vessel of the brain.

The process may be

1) Intrinsic to vessel as in atherosclerosis, inflammation, arterial dissection, venous thrombosis etc.

2) Originate remotely as occurs when embolus from heart lodge in an vessel.

3) Result from decreased perfusion pressure or increased blood viscosity with inadequate cerebral blood flow.

4) Result from rupture of a vessel in the subarachnoid space or intracerebral tissue mimics of stroke, seizures, intracranial tumor, migraine and metabolic encephalopathy.

**ISCHEMIC STROKE**

Acute occlusion of intra cranial vessel causes reduction in blood flow to the brain region it supplies a fall in cerebral blood flow to zero causes brain tissue to die within 4-10 min.

- 16-18 ml/100g blood flow of tissue causes infarction in an hour
- 20ml/100g causes ischemia with out infarction
- focal cerebral infarction occurs via

    a) Necrotic pathway cellular cytoskeleton breaks down due to energy failure

    b) Apoptotic pathway-cells become programmed to die

**CAUSES**

1) Thrombosis
2) Embolic occlusion
3) Cardio-embolic
4) Hyper coagulable disorders
5) Venous sinus thrombosis
6) Vasculitis

**HEMORRHAGIC STROKE**

Accounts for 10% of all stroke associated with 50% case of fatality rate. Hypertension, trauma, cerebral amyloid angiopathy cause major of the hemorrhage. Aneurysmal subarachnoid hemorrhage, hypertension intracranial hemorrhage are the important cause.

Common site are basalganglia, (putamen), thalamus cerebellum and pons. In non hypertensive patient hemorrhagic disorders, neoplasm, vascular malformation are the causes.

The hemorrhage may be small or large, clot may form and compress adjacent tissue causes herniation and death blood may dissect into ventricular space which increase morbidity and cause hydrocephalus.

## SUB ARACHNOID HAEMORRHAGE

The most common cause of SAH is rupture of saccular aneurysm other causes are bleeding from AVM, extension into the subarachnoid space from primary intracerebral haemorrage.

## BERRY ANEURYSM

Most common cause of SAH occur at bifurcation of large to medium sized intracranial arteries into subarachnoid space in basal cistern into parenchyma of adjacent brain.

85% occurs in anterior circulation 20%multiple aneurysm at minor sites bilaterally.

Three common sites: terminal internal carotid artery, middle cerebral artery bifurcation, top of basilar artery caused due to thinning and weakening of internal elastic laminae and media

Cardiac arrhythmias can be detected in almost all patients during the first few hours after SAH; in approximately 20% of cases, the arrhythmias can be severe or life-threatening. Ventricular arrhythmias are a potential cause of sudden death after SAH. Di Pasquale and coworkers noted torsades de pontes in 3. 8% of 132 patients with SAH

who underwent Holter monitoring. Increased QT dispersion is a common electro-cardiographic finding after SAH.

Changes resembling those seen in acute myocardial ischemia can be noted in 25% to 80% of patients. In fact, many people with SAH have secondary myocardial ischemia and left ventricular dysfunction. An elevation of the cardiac isoenzyme creatine kinase can be detected. Sub endocardial areas of focal ischemic necrosis are found among patients who died of SAH even those without prior history of coronary artery disease. The reduction of cardiac output after severe SAH might increase the risk of cerebral ischemia secondary to vasospasm.

The frequency of electrocardiographic changes can be predicted from (1) the severity of bleeding detected on CT and (2) the patient's neurologic status. The changes are most common among seriously ill patients with diffuse subarachnoid blood, intraventricular hemorrhage, or a large intracerebral hematomas. The presence of a large clot in the right sylvian cistern and fissure is associated with electrocardiographic changes.

**ELECTROCARDIOGRAPHIC ABNORMALITIES AFTER SUBARACHNOID HEMORRHAGE**

Prominent P waves

Prolonged/shortened PR interval

Broad/inverted/flattened T waves

Prolonged/shortened QT interval

Elevation/depression ST segment

Prominent/inverted U waves

Pathological Q waves

S in $V_1$ and R in $V_5$ combined> 35mm

Rhythm disturbances

## ARTERIOVENOUS MALFORMATION

Congenital shunts between arterial and venous systems consist tangle if abnormal vessels across the cortical surface or deep within brain substance occur in part of cerebral hemisphere, brainstem, spinal cord largest one are most frequently in posterior half of hemisphere causes stroke by

1) Cerebral hemorrhage

2) Steal blood away from adjacent brain tissue or to increase venous pressure significantly to produce venous ischemia most often in territory of MCA.

## ARTERIAL SUPPLY OF BRAIN

Supplied by branches of internal carotid and vertebral arteries each ICA gives of two major branches. They are anterior and middle cerebral arteries. The two vertebral arteries ascend and on the lower border of pons they unite to form basilar artery. The basilar artery lies in midline ventral to pons, at the lower border of pons. It bifurcates into two posterior cerebral artery. The internal carotid and vertebro- basilar system are connected by posterior communicating arteries. The two

anterior cerebral arteries are connected by anterior communicating artery as a result, arterial anastomosis is formed in relation to base of brain

## CEREBRAL BLOOD FLOW

Cerebral **blood flow**, or **CBF**, is the blood supply to the brain in a given time. In an adult, CBF is typically 750 millitres per minute or 15% of the cardiac output. This equates to 50 to 54 millilitres of blood per 100 grams of brain tissue per minute. CBF is tightly regulated to meet the brain's metabolic demands Too much blood (a condition known as hyperemia) can raise intracranial pressure (ICP), which can compress and damage delicate brain tissue. Too little blood flow (ischemia) results if blood flow to the brain is below 18 to 20 ml per 100 g per minute, and tissue death occurs if flow dips below 8 to 10 ml per 100 g per minute.

In brain tissue, a biochemical cascade known as the ischemic cascade is triggered when the tissue becomes ischemic, potentially resulting in damage to and death of brain cells. Medical professionals must take steps to maintain proper CBF in patients who have conditions like shock, stroke, and traumatic brain injury.

Cerebral blood flow is determined by a number of factors, such as viscosity of blood, how dilated blood vessels are, and the net pressure of the flow of blood into the brain, known as cerebral perfusion pressure, which is determined by the body's blood pressure and intracranial pressure.

Cerebral blood vessels are able to change the flow of blood through them by altering their diameters in a process called autoregulation; they constrict when systemic blood pressure is raised and dilate when it is lowered Arterioles also constrict and dilate in response to different chemical concentrations. For example, they dilate in response to higher levels of carbon dioxide in the blood

CBF is equal to the cerebral perfusion pressure (CPP) divided by the cerebrovascular resistance (CVR)

$$CBF = CPP / CVR$$

## NORMAL ECG OVERVIEW[II]

### P WAVE-

Normal P wave is best evaluated as P wave form in lead 11, P wave form in lead v1, frontal plane P wave axis. P wave in lead 11 is pyramidal in shape Duration of P wave - 0. 08 to 0. 10 sec in lead 11.

The maximum amplitude is 2. 5 mm in lead 11. P wave in lead v1 is biphasic having an initial positivity and a terminal negativity. The amplitude of initial positive deflection is 1. 5mm. Duration is 0. 05 sec and does not exceed 0. 08sec.

Terminal negative deflection should not exceed 1mm in depth and 0. 03 sec in duration.

### TALL P WAVES

P Congenitale

COPD

Congenital heart diseases

### T WAVE

Deflection is produced by ventricular repolarization usually upright in left orientated leads. T wave in lead v6 > v1.

Tall T waves are seen in lead v2-v4. Inverted T waves in lead v1 to v3 in adulthood constitute persistent juvenile pattern.

Amplitude of T wave > 5 mm is considered significant.

**TALL PEAKED T WAVES**

1) Acute sub endocardial ischemia or infarction
2) Hyperkalemia
3) Resolving inferior infarction

**INVERTED T WAVES**

Coronary heart disease

Pericardial effusion

Myxoedema

**U WAVES**

Normal U wave is small rounded deflection 1mm that follows T wave usually has polarity as T wave. An abnormal increase in u wave amplitude is most common due to drugs or hypokalemia. Very prominent U waves are marker of increased susceptibility to torsades de pointes type of ventricular tachycardia.

## Q WAVES

A pathological Q wave is its duration which exceeds 0. 04 sec or depth of q wave 25% or more of the QRS complex provided R wave exceeds 5 mm.

Q waves are normally seen in lead avR and avL in vertical heart and lead 111 with horizontal heart any Q wave in lead 2 is abnormal

## ST SEGMENT

Represents greater part of ventricular repolarization it leaves the baseline immediately after its origin from end of QRS. ST segment usually merges smoothly and imperceptibly with proximal limb of T wave when it deviates 1mm above or below the isoelectric line in either limb lead or precordial lead is abnormal.

## ST SEGMENT ELEVATION

Infarction

Pericarditis

Hypercalaemia

Hyperkalemia

Hypothermia

**ST SEGMENT DEPRESSION**

Myocardial ischemia

Subendocardial infarcts

Digoxin and

Left and right ventricular hypertrophy

**QT INTERVAL**

The interval from beginning of QRS complex to end of T wave it represent the total duration of ventricular activity. This is sum of ventricular depolarization and repolarization.

**PROLONGED QTC**

Sleep

Hypocalcaemia,

Acute myocardial infarction,

Procainamide effect,

Cerebral injury,

Hypothermia,

Jervell-lange neilson syndrome

Romono-ward syndrome

**SHORTENED QTC**

Digitalis effect

Hypercalaemia

Hyperthermia

Vagalstimulation

**MEASUREMENT OF QT INTERVAL**:

May at time present some difficulty because it may be difficult to determine the exact beginning and end of interval. The beginning of QRS complex is best determined in lead1, 11, avL, v5, v6.

When QT interval is measured from a lead where U is prominent the dip or notch between T and U wave is taken as the end of T wave.

QT interval shortens with tacycardia and lengthens with bradycardia. This is QT shortens with diminution of R-R INTERVAL and lengthens with increase of R-R interval.

## CORRECTION OF QT INTERVAL

Corrected QT interval is known as the QTC interval various formulae have been proposed for correction of QT interval. the most frequently used it the bazett.

BAZETS FORMULAE : QTc =QT/√(RR ) QTc may be regarded as K constant K=QT/√RR.

Normal value for k is 0. 39 to 0. 04 sec. Normal range is 0. 35 to 0.43 sec.

The value of QTC corresponds to QT duration at a heart rate of 60/m.

# AIM OF THE STUDY

To study the incidence and pattern of ECG changes in patient with cerebrovascular accidents.

To study the association of ECG changes with electrolytes abnormalities and

To assess the relation of ECG changes in acute cerebrovasular accident to the location of cerebral lesion.

# MATERIAL AND METHODS

## STUDY POPULATION

The study was conducted at Government Stanley Hospital during the year 2008 to 2009 on all patients admitted to medical ward with acute cerebrovascular accidents. Study population consisted of 50 patients. This is an observational study.

## INCLUSION CRITERIA

All patients with acute cerebrovascular accidents.

## EXLUSION CRITERIA

Patients with underlying heart diseases.

Patients on drugs.

Previously diagnosed patients with electrolyte abnormalities.

Patient with hepatic or renal diseases.

# METHODOLOGY

All patients with acute cerebrovascular accidents were studied.

They were assessed with serum electrolytes, X ray and blood urea and sugar 12 lead ECG was taken and monitored on the day of admission. CT scan was taken within 24-48 hrs.

Screening ECHO was done on all patients with ECG changes.

Patients showing cardiomegaly on X ray were excluded from the study.

Patients previously diagnosed to have electrolyte abnormalities were also excluded from the study.

Patients were categorized based on the CT finding as cerebral infarction, cerebral hemorrhage and sub- arachnoid hemorrhage.

ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude and morphology and QT interval was calculated. QTC interval was calculated based on Bazetts formulae.

# RESULTS OF THE STUDY

A detailed analysis of the ECG of all the patients was done.

A number of observations were made, which are given in Table 1-11

**INCIDENCE OF ABNORMAL ECG'S IN THE STUDY GROUP**

**TABLE-1**

| Study Group | No of Cases | Abnormal Cases | Percentage |
|---|---|---|---|
| Cerebral Infarction | 27 | 19 | 70 |
| Cerebral Hemorrhage | 20 | 17 | 89 |
| Subarachnoid Hemorrhage | 3 | 3 | 100 |
| **Total** | **50** | **39** | **78** |

78% of all stroke patients had some form of ECG changes

89% of patients with hemorrhages had abnormal ECG changes,

70 % of patients with infarct had changes.

100 % of patients with SAH had changes.

## TABLE-2

## THE INCIDENCE OF ST SEGMENT CHANGES

## IN THE STUDY GROUP

| Study group | Total No Cases | ST Segment Elevation | ST Depression Segment | Percentage With ST Segment Changes |
|---|---|---|---|---|
| Cerebral Infarction | 27 | 1(3%) | 9(33. 3%) | 10(37%) |
| Cerebral Hemorrhage | 20 | 10(50%) | 2(10%) | 12(60%) |
| Subarachnoid Hemorrhage | 3 | 1(33. 3%) | 0 | 1(33. 3%) |

ST segment changes were most commonly noted after cerebral hemorrhage.

33% of patients with infarction had ST depression.

ST elevation was found in 50% of patients with ICH.

## TABLE-3

## INCIDENCE OF T WAVE CHANGES IN THE STUDY POPULATION

| Study Group | Total No of Cases | Tall T Waves | T Wave Inversion | Percentage with T Waves Changes |
|---|---|---|---|---|
| Cerebral Infarction | 27 | 2 (7%) | 6(22. 2%) | 8(30%) |
| Cerebral Hemorrhage | 20 | 7(35%) | 2(10%) | 9(45%) |
| Subarachnoid Hemorrhage | 3 | 1(33. 3%) | 0 | 1(33. 3%) |

T wave changes were present in 45% 0f patients with ICH. 30% of patient with infarct had T wave changes.

## TABLE-5

## INCIDENCE OF PROLONGED QTC INTERVAL IN THE STUDY GROUP

| Study Group | Total No of Cases | Prolonged QTC | Percentage |
|---|---|---|---|
| Cerebral Infarction | 27 | 8 | 29. 6% |
| Cerebral Hemorrhage | 20 | 10 | 50% |
| Subarachnoid Hemorrhage | 3 | 1 | 33% |

50% of patients with ICH had QTC prolongation. 29. 6% of patients with infarct have QTC prolongation. 33% of patients with SAH have ECG changes.

## TABLE-6

**INCIDENCE OF RHYTHM DISTURBANCES IN THE STUDY GROUP**

| Study Group | Total No of Cases | Sinus Tachycardia | Sinus Bradycardia | Percentage with Rhythm Disturbances |
|---|---|---|---|---|
| Cerebral Infarction | 27 | 4(15%) | 0 | 4(15%) |
| Cerebral Hemorrhage | 20 | 4(20%) | 4(20%) | 8 (40%) |
| Subarachnoid Hemorrhage | 3 | 3 (100%) | 0 | 3(100%) |

Rhythm disturbance were present in 15% of patients with infarct. 40% of patients with ICH have changes of which 20% had sinus tachycardia and 20% had sinus bradycardia. 100% of patients with SAH have ECG changes.

## TABLE-7

## INCIDENCE OF PATHOLOGICAL Q WAVES

## IN THE STUDY GROUP

| Study Group | Total No of Cases | Q Wave | Percentage |
|---|---|---|---|
| Cerebral infarction | 27 | 2 | 7% |
| Cerebral hemorrhage | 20 | 0 | 0 |
| Subarachnoid Hemorrhage | 3 | 0 | 0 |

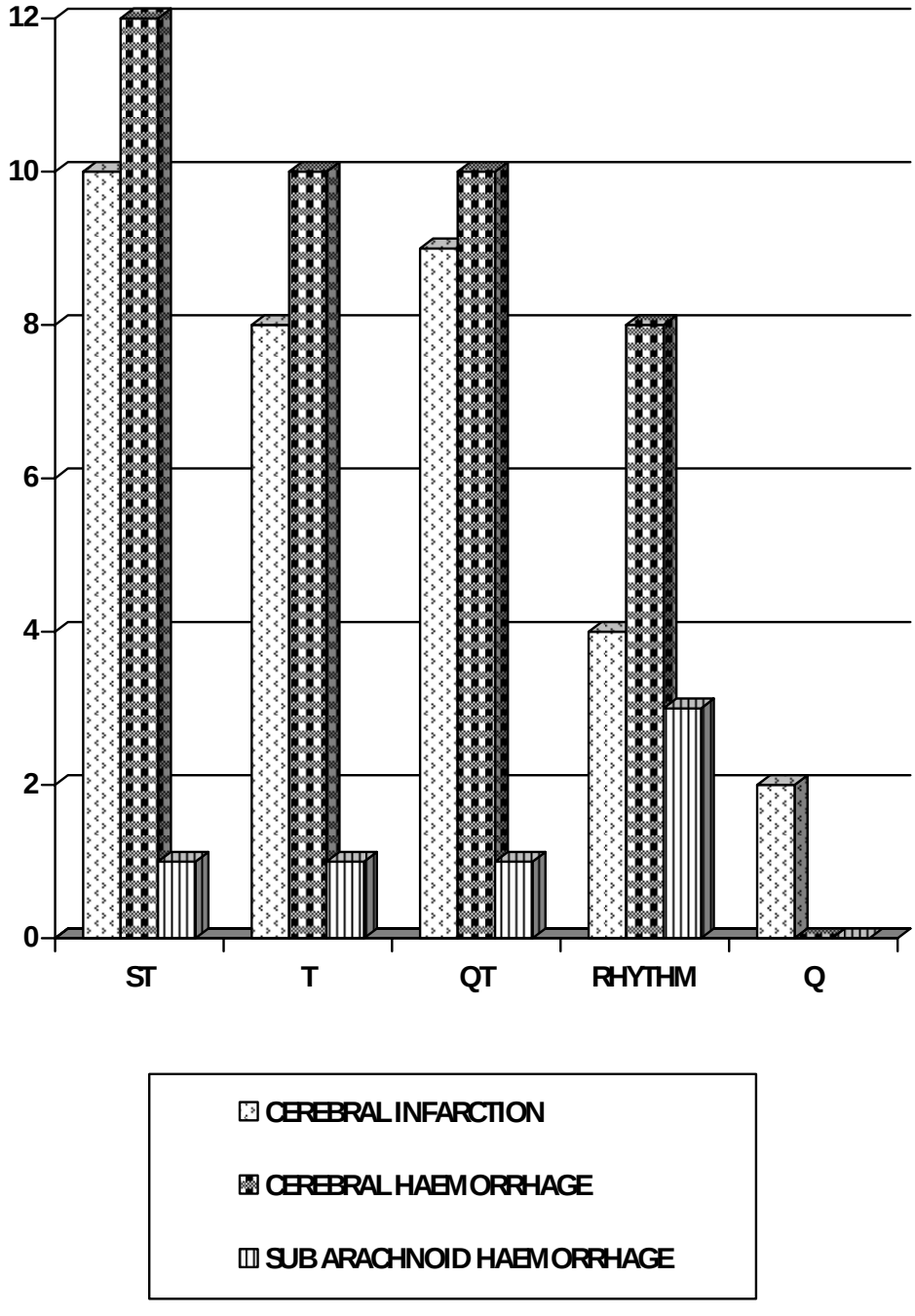
In our study only 7% of patients have pathological Q waves. Mostly associated infarct. None of the patients with ICH in our study have Q wave changes.

## TABLE -8

## INCIDENCE OF ABNORMAL U WAVES

| Study Group | Total No of Cases | U Wave | Percentage |
|---|---|---|---|
| Cerebral infarction | 27 | 0 | 0 |
| Cerebral hemorrhage | 20 | 0 | 0 |
| Subarachnoid Hemorrhage | 3 | 0 | 0 |

None of the patients in our study group have abnormal U waves.


12
10
8
6
4
2
0
ST
T
QT
RHYTHM
Q
CEREBRAL INFARCTION
CEREBRAL HAEMORRHAGE
SUB ARACHNOID HAEMORRHAGE

## TABLE-9

## INCIDENCE OF ELECTROLYTE ABNORMALITIES IN THE STUDY GROUP

| Study Group | Total No Patients | Electrolyte Abnormalities | Percentage |
|---|---|---|---|
| Cerebral infarction | 27 | 0 | 0 |
| Cerebral hemorrhage | 20 | 0 | 0 |
| Subarachnoid Hemorrhage | 3 | 0 | 0 |

None of the patients in our study group has electrolyte abnormalities.

From the above table it is evident ECG changes observed in cerebrovascular lesion were not associated with electrolyte abnormalities.

## TABLE-10

## LOCATION AND TYPE OF CEREBRO VASCULAR LESION

## WITH NO OF PATIENTS

| Cerebral Leshion | Total No of Patients | No of Patients with Hemorrhage | No on Patients with Infarct |
|---|---|---|---|
| Basal Ganglia | 1 | NIL | 1 |
| Thalamus | 5 | 2 | 3 |
| Capsuloganglion | 31 | 13 | 18 |
| Frontal | 3 | 1 | 2 |
| Temporo-Parietal | 7 | 3 | 4 |
| Parietal | 2 | 2 | 0 |
| Occipital | 1 | 1 | 0 |

## TABLE-11

## PERCENT AND NO OF PATIENTS WITH SPECIFIC ECG CHANGES

| Cerebral Lesion | Rhythm Disturbance | ST Segment Changes | QTC Prolngaton | T Wave Changes |
|---|---|---|---|---|
| Basal Ganglia | 1(100) | 1(100) | 1(100) | NIL |
| Thalamus | 2(40) | 1(20) | 2(40) | 2(40) |
| Capsuloganglion | 6(31) | 11(36) | 6(31) | 11(36) |
| Frontal | 2(66) | 1(33) | 2(66) | 2(66) |
| Parietal | 2(100) | 1(50) | NIL | NIL |
| Temporo-Parietal | 2(28) | 2(28) | 2(28) | 1(14) |
| Occipital | 1(100) | NIL | NIL | NIL |

# DISCUSSION

The study was carried out in medicine ward. Study population consisted of 50 cases of which 27 cases were cerebral infarction and 20 cases were cerebral hemorrhage 3 cases were SAH. For all these patients admitted to the medical ward base line RFT AND ELECTROLYTES were taken and analysed.

Patients with previous abnormalities were excluded from the study. 12 lead ECG taken for all the patients admitted and were monitored. ct scan was taken within 24-48 hrs and analysed and patients were categorized as cerebral infarct and intracerebral hemorrhage and SAH. In our study considerable no of patients had ECG changes.

The most common abnormality noted was ST segment changes in patient with cerebral hemorrhage. 60 percent of patients had the above changes. Of which 50% had st segment elevation and 10% had ST segment depression. This findings consistent with study of Frentz and Gorsmen who reported an incidence of 71 % with ICH and 15% with infarction and also study of Lindgren Et Al who showed ST segment depression in lateral leads.

QT*C* prolongation was the next common abnormality noted in our study. 50 percent of patients with intracerebral hemorrhage had QTc prolongation.

This is consistent with study of Arruda and Lacerda[111] which showed 67% of patients with ICH and also study of Keller and Williams [112]in patients with stroke

The next common abnormality noted was tall T waves, which was observed in 40% 0f patients with intracerebral hemorrhage. This was observed in the study of Cruickshank et al, [113] who observed Tall T waves, short PR interval in their study on CVA.

T wave inversion was observed in 10% of patients with intracerebral hemorrhage and 20% patients with cerebral infarction.

This is consistent with study of Hugenholtz consisting of extremely inverted and wide T wave, prominent U waves and prolongation of QTc interval in their study.

Rhythm disturbance was observed in 15% 0f patients with cerebral infarction and 40% of patients with ICH AND 100% of patients with SAH.

Sinus bradycardia was most commonly observed in patients with ICH. This is observed in study of stober and associates described sinus bradycardia in 23% of patients.

Q wave was noted in 7% of patients with cerebral infarction. This is in correlation with study of Chou et al[114] and Crop and Manning et al.

We could not find tall P waves or rhythm disturbances like ventricular arrhythmias AF, or U waves during the study.

Among the 50 patients admitted with CVA, out of 27 patients with infarct 22 patient were discharged with improvement. 5 patients died during the course in hospital.

Among the 20 patients of ICH, 2 patients died within 6 hrs due to massive bleed. 2 patients died on the $2^{ND}$ day of admission.

None of the patients with ECG abnormalities had altered electrolytes values showing that these ECG changes were not associated with electrolyte disturbance and their pathogenesis is different.

We also tried to correlate the ECG changes with any specific area of cerebral lesion.

Regarding the relationship between the locations of CVA lesions and ECG abnormalities, Frentz and Gormsen, and Kreus Et Al. briefly noted that ECG changes appeared to bear no relationship to arteriographic findings.

Recently, however Yamour Et Al. [115], using the computerized tomographic (CT) scan, suggested that frontal lobe hemorrhages were associated especially with the ECG abnormalities of corrected QT interval (QTC) prolongation and neurogenic T waves. However in our study there no specific correlation of ECG changes with site of cerebral lesion.

All these patients with ECG abnormalities, a screening echo were performed to rule out cardiac abnormalities associated.

Out of 27 patients with cerebral infarction only 3 had regional wall motion abnormalities. 5Bpatients out of 20 patients with ICH had regional wall motion abnormalities.

These were predominantly involving the basal and midventricular portion of anteroseptal and anterior walls and ventricular portion of inferoseptal and antero lateral walls differentiating from the CAD which involved apical portion.

We also tried to follow up these patients to see the reversal of the ECG changes. We could only follow 10 patients, whose ECG changes were completely reverted. Other 40 patient didn't turn up for follow up study.

All the patients (100%) with SAH had ECG abnormalities. Among the 3 patients noted in our study 1 had berry aneurysm for which clipping was done. Rest of the 2 patients died during hospital stay.

Sites which contribute to regulation of the cardiovascular function are known to be the anterior half of the cerebral cortex which includes the top of the frontal lobe, the motor and premotor cortex, and anterior part of the temporal lobe, hypothalamus, the limbic system, and the cerebellar hemisphere. The intimate functional connections between the hypothalamus, and posterior orbital and anterior insula and those between the hypothalamus and peripheral sympathetic nerves have also been demonstrated.

These findings suggest that the structures related to cardiovascular function are widely distributed within the central nervous system. Therefore, it is likely that CVA lesions not only in the frontal lobe, but also in the temporo-parietal lobe and basal ganglia can destroy or irritate such widely spread neurons or pathways regulating the cardiovascular system, resulting in ECG changes

# CONCLUSION

Of all the CVA patients assessed in this study, ECG changes of all forms were noted in as high as 78% of the patients.

ECG changes were more commonly associated with intracerebral hemorrhage compared with cerebral infarction.

ST segment changes were the most common abnormality noted in our study. It was most commonly associated with ICH.

Next common abnormality noted was QTc prolongation which was noted in 50% of patient. Most common in patients with ICH.

T wave changes were noted in 45% of patients. Most commonly observed in patients with ICH. Tall T wave was noted mostly in ICH.

Rhythm disturbance were noted in 40% percent of patients with ICH. Sinus bradycardia was the common abnormality associated with ICH.

7% percent of the patients with cerebral infarction had pathological Q waves.

None of the patients in our study had associated electrolyte abnormalities.

These ECG changes were not associated with any particular site of cerebral lesion.

Cardiac disturbance are diverse and frequent in the setting of acute neurological injury. More importantly the presence of cardiac abnormalities has significant impact on clinical management and affects cardiac and neurological outcome.

Understanding that these ECG changes which are occurring in patients with CVA is important because it may lead to erroneous judgment of assigning these patients as CAD. These patients should be evaluated for cardiac injury and treated only if necessary.

# REFERENCES


1. Cushing H : The blood pressure reaction of acute cerebral compression illustrated by cases of intracranial hemorrhage, Am J Med Sci 125:1017, 1903.

2. Banki NM, Zaroff JG : Neurogenic cardiac injury. Curr Treat Options Cardiovasc Med 5:451, 2003.

3. Ako J, Sudhir K, Farougue HM, et al: Transient left ventricular dysfunction under severe stress: brain-heart relationship revisted. Am J Med 119:10, 2006.

4. Bybee KA, Kara T, Prasad A, et al : Systemtic review : transient left ventricular apical ballooning : a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med 141:858, 2004

5. Wittstein IS, Thiemann DR, Lima JA, et al: Neuro-humoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 352:539, 2005.

6. Levy A: The exciting causes of ventricular fibrillation in animals under chloroform anaesthesia. Heart 4:319, 1913.

7. Levy A: Further remarks on ventricular extrasystoles and fibrillation under chloroform. Heart 7:105, 1919.

8. Housley GD, Martin-Body RL, Dawson NJ, Sinclair JD: Brain stem projections of the glossopharyngeal nerve and its carotid sinus branch in the rat. Neuroscience 22:237, 1987.

9. Kalia M, Mesulam MM: Brain stem projections of sensory and motor components of the vagus complex in the cat: II. Laryngeal, tracheobronchial, pulmonary, cardiac, and gastrointestinal branches. J Comp Neurol 193:467, 1980.

10. Calaresu FR, Ciriello J: Projections to the hypothalamus from buffer nerves and nucleus tractus solitarius in the cat. Am J Physiol 239:R130, 1980.

11. Melville KI, Blum B, Shister HE, Silver MD : cardiac ischemic changes and arrhythmias induced by hypothalamic stimulation. Am J Cardiol 12:781, 1963.

12. Beattie J, Brow G, Long C: Physiology and anatomical evidence for the existence of nerve tracts connecting the hypothalamus with spinal smpathetic centers. Proc R Soc Lond (Biol) 106:253, 1930.

13. Hockmann CH, Mauck HP Jr, Hoff EC : ECG changes resulting from cerebral stimulatin. II A spectrum of ventricular arrhythmias of sympathetic origin. Am heart J 71:695, 1966

14. Weinberg SJ, Fuster JM: Electrocardiographic changes prodced by localized hypothalamic stimulations Ann Intern Med 53:332, 1960

15. Price J, Russchen F, Amaral D : The limbic region II : the amygdaloid complex p. 389. In Bjorklund A, Hokfelt T, Swanson LW (eds) : Handbook of Chemical Neuroanatomy, Vol 5. Elsevier, Amsterdam, 1987.

16. Reis DJ, Oliphant MC: Bradycardia and tachycardia following electrical stimulation of the amygdaloid region in monkey. J Neurophysiol 27L893, 1964

17. Pool JL, Ransohoff J : Autonomic effects on stimulating rostral portion of cingulated gyri in man. J Neurophysiol 12:385, 1949.

18. Buchanan SL, Valentine J, Powell DA : Autonomic responses are elicited by electrical stimulation of the medical but not lateral frontal cortex in rabbits. Behav Brain Res 18:51, 1985

19. Manning J, Cotton MD : Mechanism of cardiac arrhythmias induced by diencephalic stimulation. Am J Physiol 203:1120, 1962.

20. Hachinski VC : The clinical problem of brain and heart. Stroke 24:I1, 1993.

21. Love BB, Grover-McKay M, Biller J, et al: Coronary artery disease and cardiac events with asymptomatic symptomatic and symptomatic cerebrovascular disease. Stroke 23:939, 1992.

22. Wilterdink JL, Furie KL, Eastron JD : Cardiac evaluation of stroke patients. Neurology 51:S23, 1998.

23. Silver FL, Norris JW, Lewis AJ, Hachinski VC : Early Mortality following stroke: a prospective review. Stroke 15:492, 1984.

24. Cechetto DF, Saper CB: Evidence for a viscerotopic sensory representation in the cortex and thalamus in the rat. J Comp Neurol 262:27, 1987.

25. Oppenheimer SM, Wilson JX, Guirdaudon C, Cechetto DF: Insular cortex stimulation produces lethal cardiac arrhythmias: a mechanism of sudden death? Brain Res 550:115, 1991.

26. Hachinski VC, Oppenheimer SM, Wilson JX, et al: Asymmetry of sympathetic consequences of experimental stroke. Arch Neurol 49:697, 1992.

27. Hachinski VC, Smith KE, Silver MD, et al: Acute myocardial and plasma catecholamine changes in experimental stroke. Stroke 17:387, 1986.

28. Cechetto DF, Wilson JX, Smith KE, et al: Autonomic and myocardial changes in middle cerebral artery occlusion: stroke models in the rat. Brain Res 502:296, 1989.

29. Sander D, Klingelhofer J: Changes of circadian blood pressure patterns after hemodynamic and thromboembolic brain infarction. Stroke 25:1730, 1994.

30. Barron SA, Rogovski Z, Hemli J: Autonomic consequences of cerebral hemisphere infarction. Stroke 25:113, 1994.

31. Korpelainen JT, Sotaniemi KA, Kuikuri HV, Myllyla VV: Circadian rhythm of heart rate variability is reversibly abolished in ischemic stroke. Stroke 28:2150, 1997.

32. Strittmatter M, Meyer S, Fischer C, et al: Location dependent patterns in cardio-autonomic dysfunction in ischaemic stroke. Eur Neurol 50:30, 2003.

33. Zhang ZH, Rashba S, Oppenheimer SM : insular cortex lesions alter baroreceptor sensitivity in the urethane anesthetized rat. Brain Res 813:73, 1998.

34. Oppenheimer SM, Gelb A, Girvin JP, Hachinski VC: Cardiovascular effects of human insular cortex stimulation. Neurology 42:1727, 1992.

35. Chapman WP, Livingston KE, Poppen JL : Effect upon blood pressure of electrical stimulation of tips of temporal lobes in man. J Neruophysiol 13:65, 1950.

36. Zamrini EY, Meador KJ, Loring DW, et al: Unilateral cerebral inactivation produces differential left/right heart rate responses. Neurology 40:1408, 1990

37. Tokgozoglu SL, Batur MK, Topuoglu MA, et al: Effects of stroke localization on cardiac autonomic balance and sudden death. Stroke 30:1307, 1999.

38. Lane RD, Wallace JD, Petrosky PP, et al: Supraventricular tachycardia in patients with right hemisphere strokes. Stroke 23:362, 1992.

39. Oppenheimer SM, Kedem G, Martin WM : Left-insular cortex lesions perturb cardiac autonomic tone in humans. Clin Auton Res 6:131, 1996.

40. Vingerhoets F, Bogousslavsky J, Regli F, Van Melle G: Atrial fibrillation after acute stroke. Stroke 24:26, 1993.

41. Laowattana S, Zeger SL, Lima JA, et al: Left insular stroke is associated with adverse cardiac outcome. Neurology 66:477, 2006.

42. Algra A, Gates PC, Fox AJ, et al: Side of brain infarction and long-term risk of sudden death in patients with symptomatic carotid disease. Stroke 34: 2871, 2003.

43. Oppenheimer S: Cerebrogenic cardiac arrhythmias: cortical laterlization and clinical significance. Clin Auton Res 16:6, 2006

44. Goldstein DS : The electrocardiogram in stroke : relationship to pathophysiological type and comparison with prior tracings. Stroke 10:253, 1979.

45. Byer E, Ashman R, Toth LA: Electrocardiograms with large, upright T waves and QT intervals. Am Heart J 33:796, 1947

46. Burch GE, Meyers R, Abildskov JA : A new electrocardiographic pattern observed in cerebrovascular accidents. Circulation 9:719, 1954.

47. Dimnant J, Grob D: Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents. Stroke 8:448, 1977.

48. Lavy S, Yaar I, Melamed E, Stern S: The effect of acute stroke on cardiac functions as observed in an intensive stroke care unit. Stroke 5:775, 1974.

49. Moss AJ: Long QT syndrome. JAMA 289:2041, 2003

50. Jackman WM, Friday KJ, Anderson JL, et al: The long QT syndromes: a critical review, new clinical observations and a unifying hypothesis. Prog Cardiovasc Dis 31:115, 1988.

51. Oppenheimer SM, Cechetto DF, Hachinski VC: Cerebrogenic cardiac arrhythmias. Cerebral electrocardiographic influences and their role in sudden death. Arch Neurol 47:513, 1990

52. Yamour BJ, Sridharan MR, Rice JF, et al: Electrocardiographic changes in cerebrovascular hemorrhage. Am Heart J 99: 294, 1980.

53. Di Pasquale G, Pinelli G, Andreoli A, et al: Torsade de pointes and ventricular flutter-fibrillation following spontaneous cerebral subarachnoid hemorrhage. Int J Cardiol 18:163, 1988.

54. Valeriano J, Elson J: Electrocardiographic changes in central nervous system disease. Neurol Clin 11:257, 1993.

55. Solenski NJ, Haley EC Jr, Kassell NF, et al : Medical complications of aneurismal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the Multicenter Cooperative Aneurysm study. Crit Care Med 23:1007, 1995.

56. Sommargren CE, Warner R, Zaroff JG, et al: Electrocardiographic abnormalities in patients with subarachnoid hemorrhage and normal adults: a comparison study, J Electrocardiol 37, suppl 1:42, 2004.

57. Cropp G, Manning G: Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. Circulation 22:25, 1960.

58. Kono T, Morita H, Kuroiwa T, et al : Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. J Am Coll Cardiol 24:636, 1994.

59. Rem JA, Hachinski VC, Boughner DR, et al: Value of cardiac monitoring and echocardiography in TIA and stroke patients. Stroke 16:950, 1985.

60. Sommargren CE, Zaroff JG, Banki N, et al: Electrocardiographic repolarizaiton abnormalities in subarachnoid hemorrhage. J Electrocardiol 35 suppl: 257, 2002.

61. Andreoli A, di pasquale G, Pinelli G, et al: Subarachnoid hemorrhage: frequency and severity of cardiac arrhythmias. Stroke 18:558, 1987.

62. Cheung RT, Hachinski V: Cardiac effects of stroke. Current Treat Options Cardiovasc Med 6:199, 2004.

63. Norris JW, Froggatt GM, hachinski VC: Cardiac arrhythmias in acute stroke. Stroke 9:392, 1978.

64. Stober T, Anstatt T, Sen S, et al: Cardiac arrhythmias in subarachnoid haemorrhage. Acta Neurochir (Wien) 93:37, 1988.

65. Di Pasquale G, Lusa AM, Manini GL, et al: Cardiac arrhythmias associated with subarachnoid hemorrhage. Prospective study with dynamic electrocardiography. G Ital Cardio 14:323, 1984.

66. Di Pasquale G, Pinelli G, Andreoli A, et al: Holter detection of cardiac arrhythmias in intracranial subarachnoid hemorrhage. Am J Cardiol 59:596, 1987.

67. Carruth JE, Silverman ME: Torsade de pointe atypical ventricular tachycardia complicating subarachnoid hemorrhage. Chest 78:886, 1980.

68. Soffer J, Dreifus LS, Michelson EL: Polymorphous ventricular tachycardia associated with normal and long Q-T intervals. Am J Cardiol 49:2021, 1982.

69. Wong KY, Mac Walter RS, Douglas D, et al: Long QTc predicts future cardiac death in stroke survivors. Heart 89:337, 2003.

70. Fentz V, Gormsen J Electrocardiographic pattern in patients with cerebrovascular accidents. Circulation 25:22, 1962.

71. Shuster S: The electrocardiogram in subarachnoid haemorrhage. Br Heart J 22:316, 1960.

72. Burch GE, Colcolough H, Giles T: Intracranial lesions and the heart. Am heart J 80:574, 1970.

73. Burch GE, Sun sc, Colcolugh HL, et al: Acute myocardial lesions; following experimentally-induced intracranial hemorrhage in mice: a histological and histochemical study. Arc Pathol 84:517, 1967.

74. Connor RC: Myocardial damage secondary to brain lesions. Am heart J 78:145, 1969.

75. Groover ME Jr, Stout C: Neurogenic myocardial necrosis. Angiology 16:180, 1965.

76. Offerhaus L, Van Gool J: Electrocardiographic changes and tissue catecholamines in experimental subarachinoid haemorrhage. Cardiovasc Res 3:433, 1969.

77. Greenhoot JH, Reichenbach DD: Cardiac injury and subarachnoid hemorrhage. A clinical, pathological and physiological correlation. J Neurosurg 30:521, 1969

78. Samuels M: Electrocardiographic manifestations of neurologic disease. Semin Neurol 4:453, 1984

79. Kuroiwa T, Morita H, Tanabe H, et al: Significance of ST segment elevation in eloectrocardiograms in patients with ruptured cerebral aneurysms. Acta Neurochir 133:141, 1995.

80. Zaroff J, Aronson S, Lee B, et al: the relationship between immediate outcome after cardiac surgery, homogenous cardioplegia delivery, and ejection fraction. Chest 106:38, 1994.

81. Kothavale A, Banki NM, Kopelnik A, et al: Predictors of left ventricular regional wall motion abnormalities after subarachnoid hemorrhage. Neurocrit Care 4:199, 2006.

82. Kopelnik A, Fisher L, Miss JC, et al; prevalence and implications of diastolic dysfunction after subarachnoid hemorrhage. Neurocrit Care 3:132, 2005.

83. Zaroff JG, Rordorf GA, Titus JS, et al: Regional myocardial perfusion after experimental subarachnoid hemorrhage. Stroke 31:1136, 2000.

84. Banki N, Kopelnik A, Tung P, et al: Prospective analysis of prevalence, distribution and rate of recovery of left ventricular systolic dysfunction in patients with sub arachnoid hemorrhage. J Neurosurg 105:15, 2006.

85. Donaldson JW, Pritz MB : Myocardial stunning secondary to aneurismal subarachnoid hemorrhage. Surg Neurol 55:12, 2001.

86. Handlin LR Kindred LH, Beauchamp GD, et al: Reversible left ventricular dysfunction after subarachnoid hemorrhage. Am Heart J 126:235, 1993.

87. Wells C, Cujec B, Johnson D, et al: Reversibility of servere left ventricular dysfunction in patients with subarachnoid hemorrhage. Am Heart J 129:409, 1995

88. Banki N, Parmley W, Foster E, et al; Reversibility of left ventricular systolic dysfunction in humans with subarachnoid hemorrhage. Circulation 104:493, 2001.

89. Banki NM, Kopelnik A, Dae MW, et al: Acute neurocardiogenic injury after subarachnoid hemorrhage. Circulation 112:3314, 2005

90. Zaroff JG, Rordorf GA, Ogilvy CS, et al: Regional patterns of left ventricular systolic dysfunction after subarachnoid hemorrhage: evidence for neurally mediated cardiac injury. J Am Soc Echocardiogr 13:774, 2000.

91. Khush K, Kopelnik A, Tung P, et al; Age and aneurysm position predict patterns of left ventricular dysfunction after subarachnoid hemorrhage. J Am Soc Echocardiogr 18:168, 2005.

92. Allman FD, Herold W, Bosso FJ, et al: Time-dependent changes in norephinephrine-induced left ventricular dysfunction and histopathologic condition. J Heart Lung Transplant 17:991, 1998.

93. Lang SA, Maron MB, Bosso FJ, et al: Temporal changes in left ventricular function after massive sympathetic nervous system activation. Can J physiol Pharmacol 72:693, 1994

94. Mertes P, Carteaux J, Jobin Y, et al: Estimation of myocardial interstitial norepinephrine relase after brain death using cardiac microdialysis. Transplantation 57:371, 1994.

95. Yeh T Jr, Wechsler AS, Graham LJ, et al: Acute brain death alters left ventricular myocardial gene expression. J Thorac Cardiovasc Surg 117:365, 1999

96. Zaroff J, Pawlikowska L, Miss J, et al: Adrenoceptor polymorphisms and the risk of cardiac injury and dysfunction after subarachnoid hemorrhage. Stroke 37:1680, 2006.

97. Norris JW, Hachinski VC, Myers MG, et al: Serum cardiac enzymes in stroke. Stroke 10:548, 1979.

98. Horowitz MB, Willet D, Keffer J: The use of cardiac troponin-I (cTnI) to determine the incidence of myocardial ischemia and injury in patients with aneurismal and presumed aneurismal subarachnoid hemorrhage. Acta Neurochir 140:87, 1998.

99. Parekh N, Venkatesh B, Cross D, et al: Cardiac troponin I predicts myocardial dysfunction in aneurismal subarachnoid hemorrhage. J Am Coll Cardiol 36: 1328, 2000

100. Tung P, Kopelnik A, Banki N, et al: predictors of Neuro-cardigenic injury after subarachnoid hemorrhage. Stroke 35:548, 2004

(i) Harrison principles of internal medicine -17th edition

(ii) Leo Schamroth, An Introduction to Electrocardiography-7 edition

101. Burch Ge, Myers R, Abildskov: A new electrographic pattern, observed in cerebrovascular accidents. circulation9;719, 1954

102. Cropp GJ Manning GW;Electrographic changes simulating ischemia and infarction associated spontaneous intracranial hemorrhages, Circulation25;22, 1962

103. Schuster-ECG in subarachnoid hemorrhage. British Heart journal22;316, 1960

104. Frentz v, GormsenJ;Electrocardiographic pattern with cerebrovascular accidents, circulation 25;22, 1962

105. Kreus Ke, Kemilla SJ, Takala JK;Electrocardiographic changes in cerebrovascular accidents, Actamed Scand 185;327, 1969

106. Miller and Abildskov, Angiology vol, 22, no, 11, 616-621

107. Litchen and Schaub, Angiology vol, 22, no11, 616-621

108. Lewine –American journal of medicine 15;344, 1953

109. Wasserman F;Chouquette G;CassenelliR;1956, American Journal of Med. sci, 231, 502

110. Hugenholtz;ECG changes after radical neck dissection. American Heart Journal 47;43, 19

111. Arrudo and Lacerda; ECG findings in acite cerebrovascular hemorrhage-Arq neuropsiquiatr 50(3) 269-74, 1992 september

112. Keller and Williams; Cardiac dysarrhythmias associated with CNS dysfunction; Journal of Neuroscience 25© 349-55;1993

113. Cruickshnok, Neil Dcoyer and J. Price ECG changes and their prognostic significance in CVA –Journal of Neurology-37;735-759;1974

114. Chou TC; ECG changes in intracranial hemorrhage. J. Electrocardiology2;193, 1969

115. Yamour BJ, Sridharan MR, Rice JF, et alElectrocardiographic changes in cerebrovascular hemorrhage. Am Heart J 99;294, 1980

# PROFORMA

NAME

AGE

SEX

OCCUPATION

RESIDENTIAL ADDRESS

**HISTORY**

ONSET OF WEAKNESS

PREVIOUS H/O OF CARDIAC DRUGS

PREVIOUS H/O OF ELECTROLYTE ABNORMALITIES

H/O LIVER DISEASES

**GENERAL EXAMINATION**

**CNS EXAMINATION**

**INVESTIGATION**

URINE ROUTINE

BLOOD UREA, SUGAR

Sr CREATINE, ELECTROLYTES

ECG

CT BRAIN

ECHOCARDIOGRAM

| S. No. | Name, age | Diagnosis | Rate | Rhythm | P Wave | P-R Interval | QRS complex | ST Segment | T- Wave | U-Wave | QT Interval | QT-C Interval | Na, K |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | Anjali, 55 | Left frontalinfarct | 130 | Sinus Tachycardia | N | N | N | N | N | N | 0.36 | 0.50 | 130 3.5 |
| 2. | Muthu, 60 | Right thalamicinfarct | 90 | Sinus | N | N | N | Depressed V4V5V6 | Inverted V4v5v6 | N | 0.32 | 038 | 135 4.0 |
| 3. | Manickam, 45 | Left Temporoparitalinfarct | 72 | Sinus | N | N | N | N | N | N | 0.38 | 0.42 | 140 3.6 |
| 4. | Venkaiyan 50 | Left capsuloganglionic infarct | 80 | Sinus | N | N | N | Depressed AVL, V5, V6 | N | N | 0.32 | 0.36 | 142 3.8 |
| 5. | Vendavaram 65 | LeftThalamicinfarct | 120 | Sinus Tachycardia | N | N | N | N | N | N | 0.36 | 0.46 | 133 4.0 |
| 6. | Mani, 65 | Right Frontalinfarct | 78 | Sinus | N | N | N | N | N | N | 0.42 | 0.47 | 136 3.5 |
| 7. | Ambujam, 54 | Rightcapsulo ganglionicinfarct | 150 | Sinus Tachycardia | N | N | N | N | Tall T V2V3 | N | 0.32 | 0.52 | 136 3.4 |
| 8. | Palani, 70 | Right Capsulo Ganglionicinfarct | 63 | Sinus | N | N | N | Depressed V2-V4 | Inverted V2-V4 | N | 0.38 | 0.43 | 142 3.8 |
| 9. | Gangan, 67 | Left Capsulo Ganglionicinfarct | 100 | Sinus | N | N | N | N | Inverted V1V2V3 | N | 0.40 | 0.50 | 136 3.6 |
| 10. | Rangan, 50 | Left basalgangliainfarct | 90 | Sinus | N | N | N | Depressed 1, AVL, V6 | Inverted 1, AVL V6 | N | 0.38 | 0.42 | 144 3.7 |
| 11. | Saroja, 62 | Left temporopareitalinfarct | 72 | Sinus | N | N | N | N | Tall V1-V3 | N | 0.38 | 0.42 | 133 3.4 |
| 12. | Kalaiammal, 48 | Left Capsulo Ganglionicinfarct | 80 | Sinus | N | N | N | N | N | N | 0.44 | 0.46 | 138 3.7 |
| 13. | Peter, 58 | Right temporoparietalinfarct | 90 | Sinus | N | N | N | N | N | N | 0.32 | 0.38 | 132 3.4 |
| 14. | Abdul kadar, 80 | Right Capsulo Ganglionicinfarct | 100 | Sinus | N | N | N | N | N | N | 0.44 | 0.51 | 142 3.8 |

| S. No. | Name, age | Diagnosis | Rate | Rhythm | P Wave | P-R Interval | QRS complex | ST Segment | T- Wave | U-Wave | QT Interval | QT-C Interval | Na, K |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 15. | Nanmullai, 56 | Left capsulogaglionicinfarct | 80 | Sinus | N | N | N | Depressed II, III. AVF | Inverted V4, v5 | N | 0.32 | 0.36 | 135 3.6 |
| 16. | Clara, 70 | Left Capsulo Ganglionicinfarct | 70 | Sinus | N | N | N | Elevated V1, V2 | N | N | 0.36 | 0.36 | 136 3.5 |
| 17. | Murugan, 62 | Right capsuloganglionic infarct | 70 | Sinus | N | N | N | Depressed III, V5, V6 | Inverted v4, v6 | N | 0.36 | 0.40 | 137 3.5 |
| 18. | Rosy, 58 | Left Capsulo Ganglionicinfarct | 72 | Sinus | N | N | N | N | N | N | 0.40 | o.44 | 138 3.4 |
| 19. | Muniyan, 70 | Right Capsulo Ganglionicinfarct | 90 | Sinus | N | N | N | N | N | N | 0.36 | 0.44 | 137 3.6 |
| 20. | Govindhan, 84 | Left Capsulo Ganglionicinfarct | 120 | Sinus Tachycardia | N | N | Q in II, III, AVF | N | N | N | 0.36 | 0.46 | 133 3.6 |
| 21. | Duraikannu, 50 | Right Capsulo Ganglionicinfarct | 80 | Sinus | N | N | N | Depressed V1-V4 | N | N | 0.32 | 0.36 | 133 3.5 |
| 22. | Thulukanam, 58 | Left, temporopareitalinfarct | 100 | Sinus | N | N | N | Depressed V4-V6 | N | N | 0.38 | 0.47 | 134 3.6 |
| 23. | Kathejabee, 53 | Right Capsulo Ganglionicinfarct | 70 | N | N | N | N | N | N | N | o.36 | 0.40 | 135 3.6 |
| 24. | Malar, 55 | Left Capsulo Ganglionicinfarct | 82 | N | N | N | N | N | N | N | 0.32 | 0.38 | 135 3.4 |
| 25. | Venkatesan, 58 | Right thalamicinfarct | 90 | N | N | N | N | N | N | N | 0.36 | 0.44 | 136 3.4 |
| 26. | Rangan, 60 | Left Capsulo-ganglionicinfarct | 70 | N | N | N | N | N | N | N | 0.40 | 0.40 | 135 3.6 |
| 27. | Kamaluddin, 64 | Left Capsulo Ganglionicinfarct | 90 | N | N | N | N | Depressed V4-V6 | N | N | 0.32 | 0.38 | 140 3.5 |
| 28. | Rajammal, 57 | Left frontalbleed | 118 | Sinus tachycardia | N | N | N | N | N | N | 0.38 | 0.46 | 133 3.6 |

| S. No. | Name, age | Diagnosis | Rate | Rhythm | P Wave | P-R Interval | QRS complex | ST Segment | T- Wave | U-Wave | QT Interval | QT-C Interval | Na, K |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 29. | Kasi, 62 | Right Capsuloganglionic Bleed | 130 | Sinus Tachycardia | N | N | N | Elevated V1-V4 | Tall T V1-V4 | N | 0.36 | 0.50 | 134 3.6 |
| 30. | Arul, 55 | left thalamicbleed | 40 | Sinus Bradycardia | N | N | N | N | Tall V1-V2 | N | 0.60 | 0.50 | 136 4.0 |
| 31. | Madurai, 60 | Left Capsuloganglionic Bleed | 58 | Sinus Bradycardia | N | N | N | Elevated V1-V2 | Tall V1-V3 | N | 0.42 | 0.42 | 132 3.5 |
| 32. | Kailasam, 57 | Right temporopareitalbleed | 130 | Sinus Tachycardia | N | N | N | Depressed V5, V6 | Inverted v4-v6 | N | 0.36 | 0.50 | 133 3.5 |
| 33. | Mannar.67 | Right frontalbleed | 42 | Sinus Bradycardia | N | N | N | N | Tall V2-V3 | N | 0.60 | 0.52 | 130 3.6 |
| 34. | Kalaiyan, 50 | Right capsuloganlionic Bleed | 72 | Sinus | N | N | N | N | N | N | 0.36 | 0.38 | 140 3.5 |
| 35. | Mumtaj, 60 | left capuloganglionic Bleed | 40 | Sinus Bradycardia | N | N | N | Elevated V4, V5, V6 | Tall V4, V5, V6 | N | 0.60 | 0.50 | 135 3.6 |
| 36. | Ekambaram, 65 | Left Capsuloganglionic Bleed | 75 | Sinus | N | N | N | Elevated 11, 111, V6 | Tall 11, 111.V6 | N | 0.39 | 0.44 | 136 3.6 |
| 37. | Madurai, 55 | Left Capsuloganglionic Bleed | 80 | Sinus | N | N | N | Elevated V4-V6 | N | N | 0.44 | 0.46 | 140 3.5 |
| 38. | Raman, 67 | LeftpareitalBleed withmidline shift | 72 | Sinus | N | N | N | Elevated V5, V6 | Tall V5-V6 | N | 0.38 | 0.42 | 132 3.4 |
| 39. | Ravathoor, 55 | Left capsuleganglionbleed | 100 | Sinus | N | N | N | Depressed 1, AVL, V6 | Inverted 1, AVL, V6 | N | 0.38 | 0.47 | 133 3.6 |
| 40. | Mathew, 45 | Right thalamicbleed | 80 | Sinus | N | N | N | N | N | N | 0.44 | 0.46 | 136 3.5 |
| 41. | Nagappan, 65 | Left Capsulo ganglionicbleed | 75 | Sinus | N | N | N | Elevated V4, V5, V6 | Tall V4, V5, V6 | N | 0.39 | 0.44 | 135 3.6 |
| 42. | Basha, 70 | Left occipital bleed | 100 | Sinus | N | N | N | N | N | N | 0.36 | 0.46 | 140 3.6 |

| S. No. | Name, age | Diagnosis | Rate | Rhythm | P Wave | P-R Interval | QRS complex | ST Segment | T- Wave | U-Wave | QT Interval | QT-C Interval | Na, K |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 43. | Sekar, 60 | Right Capsulo ganglionic bleed | 55 | Sinus Bradycardia | N | N | N | Elevated V1-V4 | N | N | 0.41 | 0.39 | 133 3.5 |
| 44. | Kosalai, 58 | Leftcapsulo Ganglionbleed | 150 | Sinus Tachycardia | N | N | N | Elevated V1-V4 | N | N | 0.39 | 0.62 | 134 3.4 |
| 45. | Kannaiyan, 60 | Right capuloganglionicbleed | 60 | N | N | N | N | N | N | N | 0.39 | 0.39 | 133 3.5 |
| 46. | Senguthan, 58 | Left temporopareitalbleed | 75 | Sinus | N | N | N | Elevated V2V3V4 | N | N | 0.35 | 0.39 | 135 3.6 |
| 47. | Rani 58 | Left capuloganglionicbleed | 60 | Sinus | N | N | N | N | N | N | 0.39 | 0.39 | 140 3.7 |
| 48. | Poronam 64 | PrimarySAH | 150 | Sinus Tachycardia | N | N | N | Elevated V1-V4 | Tall V1-V4 | N | 0.39 | 0.62 | 136 3.6 |
| 49. | Ponnammal, 65 | SecondarySAH | 100 | Sinus | N | N | N | N | N | N | 0.30 | 0.39 | 134 3.6 |
| 50. | Mohammed Raffi, 70 | Secondary SAH | 75 | Sinus | N | N | N | N | N | N | 0.35 | 0.39 | 133 3.5 |