"A STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN CEREBROVASCULAR ACCIDENTS"

By

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ABSTRACT

BACKGROUND:

The interrelationship of cerebrovascular disease and cardiovascular disease has been repeatedly emphasized. Decompensation in one system may adversely affect the other, whether or not the patient has recognized disease of both systems. Role of heart as a cause of stroke has received much attention. The more interesting possibility that neurologic disease could result in cardiac rhythm disturbances or structural damage, has solely been the subject of quaint conjuncture or isolated case reports in the past years. However as the heart has an important autonomic innervation, it is not beyond the bounds of reason that acute disturbances of the central nervous system could result in disordered cardiac function. Thus cerebrovascular accidents may be associated with abnormal ECG even in patients without heart disease

METHODS:

All 36 CVA cases which fulfilled the criteria were studied with respect to ECG changes. 12 Lead electrocardiogram was taken for all cases as soon as possible after the admission (within 24 hours). Subsequent to the ECG taken at the time of admission, 12 lead ECGs were repeated at 24hrs and Another ECG was taken at discharge.

RESULTS:

It was observed that 88.89% patients (n=36) had ECG changes. All Patients (100%) with cerebral hemorrhage, SAH and CVT had ECG changes whereas it was only 82.6%in cerebral thrombosis cases. ECG changes like ST-depression (16.7%), T-

wave invertion (52.8%) ,QT-c prolongation (19.5%), sinus tachycardia (11.1%), sinus brady cardia (2.7%), U –waves(8.4%) and arrhythmias (5.6%) were noted. Mortality was more (37.5%) among cases with ECG changes. One other important thing which we noticed in our study is that (58.33%) of patients had hypertriglyceridemia and it was noted that among patients with hypertriglyceridemia and ECG changes (41.67%) mortality was seen which indicated a bad prognosis.

CONCLUSION:

Different ECG changes are seen in CVA cases with out any primary cardiac disease and these ECG changes can be predictor of prognosis.

KEY WORDS: CVA, ECG changes, Prognosis.

LIST OF ABBRIVIATION

A	ECG on 1st Day
ADP	Adenosine Di Phosphate
AF	Atrial Fibrillation
ATP	Adenosine Tri Phosphate
AV Malformation	Arterio Venous Malformation
AV node	Arterio Ventricular Node
B	ECG at 24hrs Day
C	ECG at discharge
CK Values	Creatinine Kinase
CPK	Creatine Phospho Kinase
CSF	Cerebro Spinal Fluid
CT Scan	Computed Tomography Scan
CVA	Cerebro Vascular Accidents
CVT	Cortical Venous Thrombosis
ECG	Electro Cardiography
F	Female
HR	Heart Rate
IP No.	In Patient Number
K^+	Potassium
LBBB	Left Bundle Branch Block
Lt	Left
M	Male
MCA	Middle Cerebral Artery

Min.	Minute
Mv	Millivoltes
N	Normal
Na^+	Sodium
NINDS	National Institute for Neurological Diseases and
NTS	Nucleus Tractus Solitarius
PCA	Posterior Cerebral Artery
Q T_c inter	Q T_c interval
RBBB	Right Bundle Branch Block
Rhy	Rhythm
Rt	Right
SA Node	Sino Atrial Node
SAH	Sub Arachnoid Hemorrhage
Sec.	Seconds
ST ↓	ST-segment depression
STEMI	ST Segment Elevation Myocardial Infarction
	Stroke
T ↓	T-wave invertion
TIA	Transient Ischemic Attack
VDRL	Veneral Disease Research Laboratory
VPB	Ventricular Premature Beats
VPC's	Ventricular Premature Complexes
TRIG	Triglycerides

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INTRODUCTION

The term cerebrovascular accident or stroke includes any neurological dysfunction of sudden onset secondary to interruption in blood flow or by hemorrhage in to or around brain other than trauma. It produces deficits in neurological function that relate to specific part(s) of brain that are affected. Stroke is a major cause of long term disability. It is the third leading cause of death after heart disease and cancer in developed countries, with an average incidence of approximately 1per1000 population. It is the leading cause of morbidity and mortality worldwide. In India stroke accounts for 20% of all neurological admissions and 4% of all hospital admissions.

The interrelationship of cerebrovascular disease and cardiovascular disease has been repeatedly emphasized. Decompensation in one system may adversely affect the other, whether or not the patient has recognized disease of both systems.[1]

Role of heart as a cause of stroke has received much attention. The more interesting possibility that neurologic disease could result in cardiac rhythm disturbances or structural damage, has solely been the subject of quaint conjuncture or isolated case reports in the past years. However as the heart has an important autonomic innervation, it is not beyond the bounds of reason that acute disturbances of the central nervous system could result in disordered cardiac function. Thus cerebrovascular accidents may be associated with abnormal ECG even in patients without heart disease.

There are numerous reports demonstrating the fact that primary neurologic abnormalities may produce ECG changes without any myocardial lesion. ECG changes affecting T wave, U wave, S-T segment, Q-T interval and various arrhythmias have been reported. These electrocardiographic changes have been

predominantly reported in patients with subarachnoid haemorrhage, but also with other forms of intracranial bleeding and cerebral thrombosis. These electrocardiographic changes may resemble those of myocardial ischaemia and sometimes acute myocardial infarction [2] and misinterpretation has led to delay in operative treatment for subarachnoid haemorrhage. Operation on an intracranial aneurysm may be postponed or an unduly grave prognosis given if it is wrongly believed that ECG indicates a fresh myocardial infarct.

There are evidences suggesting, that patients who had ECG changes fallowing cerebrovascular accidents had poor prognosis compared to those who did not show ECG changes .Approximately 2 to 6% of all stroke patients die from cardiac causes in the first three months after ischemic stroke.[3]

The possible explanations for these ECG changes following stroke are the activation of cerebrogenic autonomic nervous system, neurohumoral dysregulation and elevated levels of catecholamines. Other causes that can produce ECG abnormalities like electrolyte imbalance, renal, cardiac, metabolic, respiratory disease must also be considered.

In view of the above speculations, present study was undertaken to identify the electrocardiographic changes produced primarily due to cerebrovascular accidents. The literature regarding the incidence and pathogenesis of electrocardiographic changes in cerebrovascular accidents has been reviewed.

AIMS OF THE STUDY

Cerebrovascular accidents have been associated with variety of cardiovascular abnormalities. Many studies have shown casual relationship between cerebrovascular accidents and cardiovascular abnormalities. Observations of these studies have led to the hypothesis that cerebrovascular accidents may increase the sympathetic activity with the resultant myocardial cell necrosis and electrocardiographic abnormalities.

Since acute stroke has been shown to be associated with cardiac arrhythmias and myocardial cell necrosis producing electrocardiographic changes mimicking those of ischaemic heart disease, their recognition is important as they may pose diagnostic problems. So the present study aims at the following aspects:

1. To study the incidence of ECG changes in patients with acute stroke who are not suffering from any primary cardiac diseases.
2. To study the nature of ECG changes seen in patients with acute stroke and the types of ECG changes observed in different types of stroke.
3. To find out whether there is any prognostic significance for these abnormal ECG findings in patients with acute stroke.

REVIEW OF LITERATURE

The effect of brain injury on heart was demonstrated in experimental animals in the 1930s.Cardiac changes in patients with acute stroke were first reported in four patients in 1947.[4,5] These four cases were described, with at least two patients suffering a subarachnoid hemorrhage, ECG patterns suggestive of an acute myocardial ischaemia predominantly involving left ventricular endocardium were identified. Subsequently Burch et al studied the phenomenon in a more systematic fashion[6]. Seventeen abnormal ECGs were separated from recordings obtained from stroke patients. The predominant diagnosis was intracerebral bleed or subarachnoid haemorrhage. A triad of changes were illustrated comprising prolongation of Q-T interval, T wave of increased amplitude and duration, abnormal U waves especially in septal leads. Although the study sample was small, changes were most frequently observed after subarachnoid haemorrhage, followed by intracerebral bleed and then by ischaemic stroke. Subsequent controlled studies indicated that acute haemorrhagic stroke whether intracerebral or subarachnoid, is associated with ECG changes in 61% - 71% of cases. Such changes were observed in 5%-17% of ischaemic strokes.[1,7,8]

Ischaemic heart and cerebrovascular disease frequently co-exist in the same patient, moreover they share similar risk factors. Any ECG changes following stroke could therefore be caused by exacerbation of coincident coronary artery disease.

A study compared recordings from 100 consecutive acute stroke patients taken within 3 days of admission with those from age/sex matched controls. S-T depression and prolongation of Q-T interval were seven times more frequent in patients after cerebrovascular accidents than those in control patients. T-wave inversion and ventricular premature beats were likewise 4-times more common in

the acute stroke group.[1] Later a study specifically addressed the problem of associated cardiac disease as a cause of the ECG changes following stroke. ECGs of acute ischaemic stroke or intracerebral haemorrhage patients with no history of heart disease and normal recent recording were analysed for changes occurring after their cerebrovascular events. 44% showed either a recent onset ischaemic pattern ECG or a cardiac arrhythmia[9].

In a further attempt to control for the effects of concomitant coronary artery disease, a study in 1979 compared the ECGs of 53 acute stroke patients taken within 24 hours of admission, with tracings taken an average of 4 months earlier. A control group comprised 63 age/sex matched patients admitted for reasons other than stroke or a cardiac cause whose previous tracings were also available. Abnormal prolongation of Q-T interval not seen in the previous recordings were observed in 32% of stroke group and 2% of controls. New T-wave inversion was apparent in 15% of the stroke group and abnormal *U* waves in 13%, neither appeared as a new feature in the admission ECGs of control group. These differences were highly significant . [10]

In a study conducted from 2000-2002 over 279 patient's suffering from Acute ischaemic stroke found prolonged Q-Tc in 36% , ST depression in 24.5%, atrial fibrillation in 19.9% and T wave inversion in 17.8%.[11]

Direct evidence confirms that ECG changes can occur even in the presence of a normal coronary arteries and in the absence of acute ischaemic changes. In 1960, a study proved this in this study of 29 stroke patients. Similar proof was also gathered by subsequent studies in 1962,[7] 1979[10] and in 1987[12].

Echocardiograms have shown transient abnormalities in left ventricular wall motion in patients with subarachnoid hemorrhage. A study compared two groups of patients with SAH , one group with one group with out ST- segment elevation. Left

ventricular wall motion was significantly decreased in group with with ST- segment elevation compared to group with out ST- segment elevation .[13]

A study in 2004 over 97patients found atrial fibrillation in 27% of stroke or transient ischaemic attacks and ECG changes occurred in 56%. Atrial fibrillation accounted for a quarter of ECG rhythms in elderly acute stroke or transient ischaemic attack patients .[14]

The ECG effects induced by acute stroke are often evanescent resolving with little residuam over a period of days to months. This argues against myocardial infarction as a cause and changes that can be expected to produce a persistent ECG deficit in the majority of cases.[7,15,16]

Cardiac complications such as arrhythmias and ischemic heart damage are related to an impaired prognosis during the acute phase of stroke. Although the pathogenesis of these complications is still incompletely understood, they are obviously associated with central autonomic cardiovascular dysregulation involving both the sympathetic and the parasympathetic nervous system.[17]

A study done on patiens with subarachnoid hemorrhage showed ECG findings with high amplitude R waves in 19% of subjects, ST depression in 15%, T wave abnormalities in 32%, U wave greater than 1mm amplitudein 47% and prolonged Q-Tc interval in 23%.[13]

Also found arrhythmias like sinus bradycardia, sinus tachycardia, wandering atrial pacemaker and atrial fibrillation. Occasionally premature atrial, junctional, ventricular complexes, ventricular tachycardia and atrioventricular blocks have been detected. [13]

A study in 1974 demonstrated 39% incidence of new arrhythmias in the admission ECGs of acute ischeamic stroke and intracerebral haemorrhage patients, not known to have previous heart disease, [9] a similar study, found a 25% of incidence of new arrhythmias after acute stroke admissions of all types. Of these cardiac arrhythmias, the most common was atrial fibrillation that occurred with a frequency of 9%.[10] A study also demonstrated that atrial fibrillation was the most common arrhythmias (21%) and ventricular arrhythmias occurred at a frequency of 13%.[1]

A study done on 127 patients in 2007 with acute ischemic stroke demonstrated an association between frequent atrial premature beats (APB_S) in 24hr ECG recordings and an increased incidence of paroxysmal atrial fibrillation in patients with ischemic stroke.[19]

A study was done in 2004 over 12months in 222patients, of them 162 were of ischaemic stroke and 60 patients had hemorrhagic stroke. ECG changes were noted in 65% of ischemic stroke patients and 57%in hemorrhagic stroke patients. Atrial fibrillation was more frequent in ischemic stroke(34%) than in hemorrhagic stroke(13%).[20]

A study demonstrated a statistically significant increase in the occurrence of ventricular and atrial premature beats and atrial fibrillation following hemispheric, as opposed to brainstem stroke, in a study of intracerebral haemorrhage, left frontal hematomas were associated with prolongation of the Q-T interval and T wave abnormalmes[10]. Sinus bradycardia and premature ventricular beats accompanied temporoparietal haematomas whereas sinus tachycardia was more common after thalamic or basal ganglia haemorrhages. Brainstem haemorrhages were more likely to be accompanied by atrial fibrillation on premature atrial beats. [21]

The study of cerebrogenic cardiac arrhythmias in l990 say that repolarisation changes comprising of Q-T prolongation, T-wave flattening and inversion, S-T segment alterations are most commonly seen after subarachnoid haemorrhage and intracerebral bleed, but may occur in other neurological conditions. The effects likely are mediated by sympathetic nervous system. Cerebral arrhythmogenesis may underlie sudden death in both normal and epileptic populations. [22] Experimental evidence suggest that insula has a cardiac chronotropic organisation and may be involved in the genesis of arrhythmias seen in epilepsy and after stroke.[23,24]

Recent studies suggest that cerebrovascular diseases also cause a prognostically unfavorable suppression of heart rate variability similar to that observed in coronary artery disease ,and the sympathetically and parasympathetically mediated components of heart rate variability both are diminished as a consequence of acute stroke.[25]

Circadian fluctuation of heart rate variability is abolished in the acute phase of ischemic stroke and returns during the subsequent six months. This reversible abolition, which reflects both sympathetic and parasympathetic autonomic dysfunction, may be caused by an infarction located either in the hemispheric or brainstem level of the brain.

The loss of the relative vagal nocturnal dominance may contribute to the incidence of cardiac arrhythmias and other known cardiovascular complications commonly found in the acute phase of stroke.

The article "Cardiac consequences of stroke" in Neurologic clinics 1992[26] says stroke whether ischaemic or haemorrhagic induces cardiac damage by nonspecific mechanisms. Recent experimental evidence indicates that insular cortex plays a principal role in strokes related cardiac damage.[27] It also opined that patients

with insular stroke and those showing acute electrocardiographic changes might be especially vulnerable to stroke extension and should be monitored closely.

A study conducted in 846 of ischemic stroke patients in 2007 stated that the ECG variables highly predictive of serious cardiac adverse events were prolonged QT_C and presence of ventricular premature beats (VPB_S) on base line ECG in stroke patients.[3]

A study in 1994, states about neurogenic cardiac effects of cerebrovascular accidents. ECG changes and cardiac arrhythmias frequently encountered after stroke are not solely explicable by concomitant ischaemic heart disease. Excessive sympathoadrenal tone is contributory. Specifically it is now believed that augmentation of intracardiac sympathetic nerve activity occurs producing cardiac myocyte damage and depolarising ionic shifts resulting in ECG repolarisation changes and arrhythmogensis. Experimental and clinical evidence now implicate the insular cortex and its subcortical connections in the generation of cardiac arrhythmias under stress and following stroke. Lateralisation studies indicate that destruction of areas adjacent to the right insular cortex or evolving non cardioactive zones within this region have especially marked cardiac effects. This very likely contributes to the cardiac mortality, which is the principal long term cause of death in stroke patients. [28]

Recent studies have shown that acute stroke is associated with impairment of cardiac autonomic balance and increased incidence of arrhythmias . Abnormalities of cardiovascular autonomic control may also retain prognostic relevance, as reduced heart rate variability (HRV) and impaired cardiac baroreceptor sensitivity have both been associated with adverse clinical outcome after stroke.[29]

Further more , all stroke-related autonomic abnormalities appear more relevant in patients with right-sided hemispheric infarctions, with concurrent involvement of

right insula implying further derangement of cardiovascular function and even an increase of the odds of death with in three months of acute stroke.[29]

In a study done over 192 patients to know the ”prognostic importance of ECG changes in stroke” in 2004, one month mortality was analysed. Observation showed ischemia like ECG changes in 79% of stroke patients, long QTc in 26% and arrhythmia in 44%. In the above said group early mortality rate was 27% ST segment changes and abnormal U wave were univariate predictors of early mortality.[30]

The ECG changes are frequently seen in selected patients with ischemic stroke. Regardless of origin,ST-segment change can be a predictor of early mortality.[30]

A study included 87 patients as study group in 2003 found 62.1%of patients with cerebral infarct had ECG changes and six month mortality rate among patients with ECG changes was 38.9% suggesting that cardiac evaluation in patients with acute stroke is of prognostic importance.[4]

NEURAL CONTROL OF THE HEART

The annual cardiac mortality after stroke (5 to 10%) represents the single most common cause of death on long-term follow-up. Accumulating evidence suggests that this mortality may not be entirely explained by concomitant coronary artery disease: cerebral injury may directly contribute to the generation of cardiac dysfunction. Acute stroke has been associated with a pattern of increased serum cardiac enzymes and ECG repolarisation changes that are not typical of ischemia. The reported association between cardiac arrhythmia and rise in serum cardiac enzyme levels at stroke onset may indicate an acute myocardial lesion related to brain injury in some cases, rather than the converse phenomenon.[31]

A particular form of cardiac necrosis termed myocytolysis has been identified in patients who have died after stroke; these changes can be seen with normal coronary arteries and are focused on intracardiac nerves rather than blood vessels, indicating a possible neural origin. The nature of the nerves involved is unclear but plasma norepinephrine may be elevated after stroke and associated with functional cardiac alteration in experimental models indicating a sympathetic neural association. In this regard, patients with stroke with higher mean plasma norepinephrine concentration show higher CK values, suggesting cardiac damage from activated sympathetic tone.[31]

In an experimental stroke model, cats with myocytolysis had a significant elevation of plasma norepinephrine compared to cats with out myocardial damage. These data indicate that stroke related cardiac damage likely arises from a shift of autonomic function toward augmented cardiac sympathetic tone.[31]

Neurogenic myocardial stunning has been described after acute ischemic stroke and subarachnoid hemorrhage

The concept of an interrelationship between mind and heart date back to antiquity. The heart was thought to be the source of consciousness in ancient Greece and Egypt and the seat of human emotions in several other cultures. In more modern times, neural input to the cardiovascular system by the autonomic nervous system has been well recognized, but studies have largely dealt with the independent function of the nervous system and the cardiovascular system. Despite the heart's ability to generate a normal rhythm and to maintain cardiac output in the absence of innervation, tight coupling of neural and cardiovascular activity plays a significant role in balancing cardiovascular responses to stimuli in health and disease. Neural input to the heart, however, is not always salutary to cardiac function. Central and

peripheral neural disturbances not only can induce cardiac abnormalities in anatomically healthy hearts, but also contribute to cardiac dysfunction in the presence of identifiable structural abnormalities. The scope of these interactions may have far reaching implications.

Underlying central nervous system disease has been implicated as one cause of cardiac disorders, but the clinical relevance and underlying basic mechanisms of the influence have been studied only recently. This article outlines the anatomic basis of interactions between brain and heart and discusses relevant clinical observations in the context of neural control of the heart.

A study in 2007 concluded that irrespective of the side of ischemia, post-acute stroke patients showed a parasympathetic cardiac deficit .Additionally, sympathetic cardiovascular modulation was increased in patients after right-sided stroke. Post-acute stroke patients might be at an increased risk for cardiac arrhythmia after unopposed sympathetic stimulation.[32]

NEUROANATOMY OF CARDIOVASCULAR REGULATION

Cardiac Afferents:

Both the vagus (parasympathetic) and the sympathetic nerves carry afferent nerve fibers from the heart. Mechanoreceptor and chemoreceptor endings of these fibers are located in the atria, ventricles, coronary vessels, and pericardium. Vagal fibers largely arising from cells in the nodose ganglia pass caudally to the heart, where they terminate in these specialized nerve endings and rostrally to their termination on the cells in the nucleus tractus solitari (NTS) m the medulla oblongata, Other afferents that serve to regulate cardiac rate are part of arterial baroreceptor and chemoreceptor reflexes. Those from the aortic arch are carried in the vagus or aortic

depressor nerves, whereas those from the carotid sinus (baroreceptors) and carotid body (chemoreceptors) are carried in the carotid sinus and glossopharyngeal nerves. The cells of origin of afferents from the carotid sinus and body are in the petrosal ganglion, and similar to other visceral afferents, their central projections terminate in the NTS. Similar to cardiac vagal afferents, the cells of origin of aortic baroreceptors lie in the nodose ganglion and project to NTS.

In contrast, afferent C and A-delta fibers in cardiac sympathetic nerves arise from cells in the dorsal root ganglia at T1-T5 levels and enter the spinal cord through the upper 4-5 thoracic roots. These fibers, which are activated by cardiac ischaemia, irritation, or stretch, terminate in the dorsal funiculus on the same cells that receive somatic afferent input from the T1-T5 dermatomal levels. These recent observations likely define the anatomic substrate for referred pain during cardiac ischaemia. Afferents in cardiac nociceptive pathways interact with other central mechanisms for cardiac regulation, but the manner and extent of the interactions have not been elucidated.

Fibers ascending from the thoracic spinal cord towards the- thalamus also project to NTS and other cardiovascular nuclei. The NTS, which also receives inputs from other viscera through vagus and glossopharyngeal nerves, integrates multiple visceral inputs including those from the cardiovascular system.

Axonal projections from NTS terminate on preganglionic parasympathetic neurons in the dorsal motor nuclei of vagus and nuclei ambiguus, on preganglionic sympathetic neurons in the intermediolateral columns and on cells in the ventrolateral medulla and raphe nuclei that regulate the cardiovascular system by modulating activity of the sympathetic cells. Preganglionic cells in the intermediolateral column also receive inputs from the parabrachial nucleus, hypothalamus, and cells of the A5

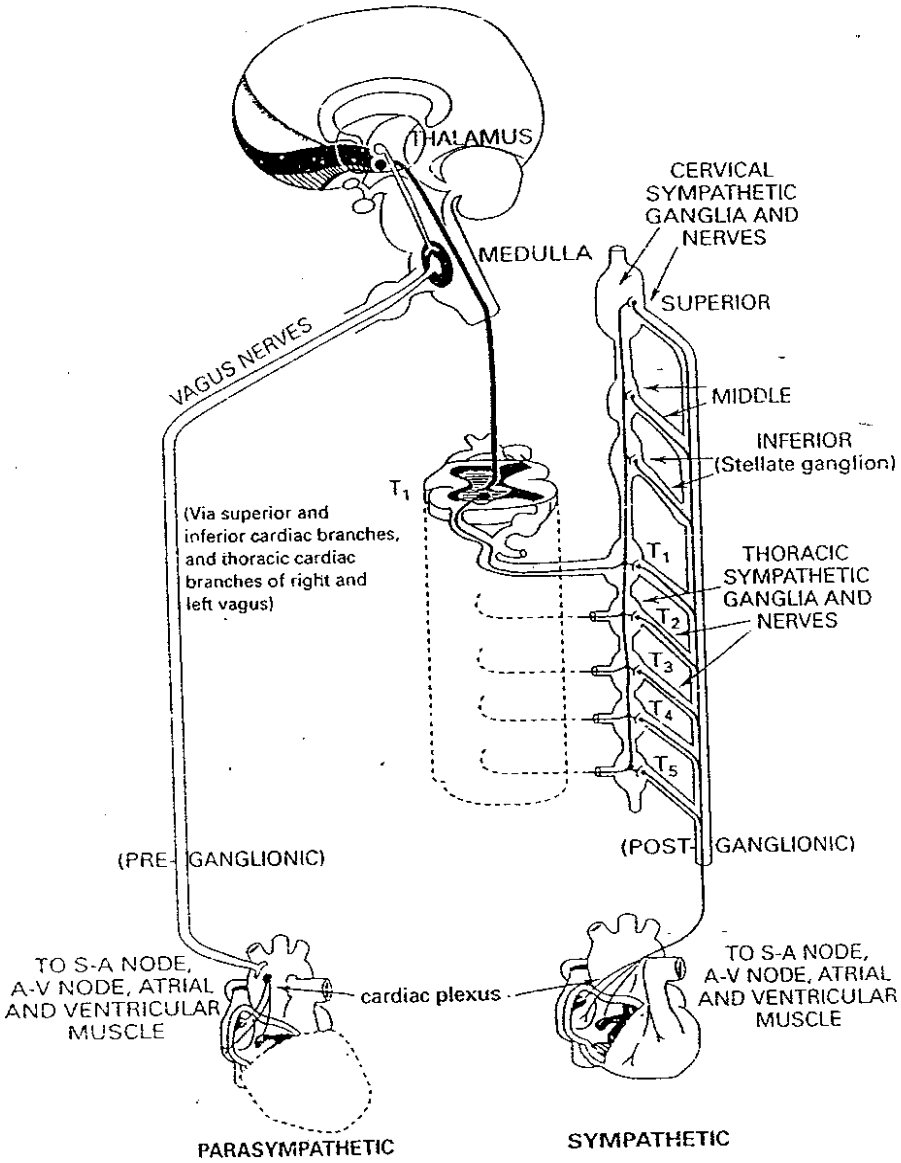
group. The cerebral cortex projects to NTS both directly[24] and by way of the limbic system, hypothalamus,[33] or parabrachial nuclei.[34] The parabrachial nuclei therefore are reciprocally connected to NTS as well as other brainstem nuclei that project to the spinal cord.[35,36,37]

Cardiac Efferents

Preganglionic parasympathetic fibers originate in the nuclei ambiguus and pass via ipsilateral cardiac vagal nerves to the heart, where they terminate on neurons of the intracardiac ganglia.Efferent preganglionic sympathetic fibers arise in the upper 4-5 thoracic segments of the spinal cord. Although these fibers synapse in the upper thoracic or lower cervical ganglia, the principal site of this synapse in humans is unknown.[38]Preganglionic vagal and postganghonic sympathetic fibers combine at the base of the heart to form the cardiacplexus.[39]

After penetrating the heart, however, sympathetic and parasympathetic fibers are distributed differently. Sympathetic fibers are more uniformly disputed in contrast to parasympathetic fibers that are concentrated in the sinoatrial (SA) and the atrioventricular(AV)node,[40,41,42]where fibers from the right and left vagus overlap.

Right sympathetic neves predominantly innervate the SA node, whereas those on the left innervate the AV node. In addition, sympathetic fibers pass through the ventricular sub epicardium, whereas cardiac vagal fibers lie deep m the myocardium and adjacent to the endocardium.[43,44] Because of the different locations of these nerves, it is possible for sympathetic fibers to be spared when myocardial ischaemia does not extend to the sub epicardium. As is discussed later, such a relative augmentation of sympathetic input may predispose to cardiac arrhythmias.[43,44]



A simplified, diagrammatic representation of the efferent autonomic innervations of the heart. The parasympathetic and sympathetic nerves to the heart , many of which closely accompany each other in and through the various cardiac and coronary plexuses ,have been separated for illustrative purpose.

Diencephalic Sites and Cardiac Control

Substantial evidence exists to implicate the hypothalamus in cardiac control. Both anatomically and physiologically, cardiovascular afferent activity has been demonstrated for this region. Anatomically, there is a marked input to this region from cortical and brainstem areas intimately involved in cardiovascular control.

A variety of cardiovascular responses have been elicited on electrical stimulation of the hypothalamus. In anesthetized cats bradycardia and pressor responses were specially dialed on stimulation of the anterior hypothalamus, and the tachycardia and pressor responses were most likely to be produced by stimulation of the lateral and posterior hypothalamus[45]. However, the nature of the response to electrical stimulation may be state-dependent.

In general terms, the results of such electrical stimulation experiments suggest a division of the hypothalamus into a posteriorly located area of cardiovascular sympathetic control and an anterior parasympathetic control region. Different results occur with chemical stimulation (in which case cell bodies alone are stimulated rather than a mixture of these and fibers of passage with electrical stimulation). A study done[46], using the excitatory amino acid L-Glutamate, demonstrated bradycardia and depressor sites within the posterior hypothalamus, chiefly within the periventricular zone caudal to the posterior hypothalamic nucleus. Glutamate stimulation of the posterior hypothalamic nucleus produced minimal bradycardia and depressor effects. Stimulation of the dorsal hypothalamic area resulted in similar responses. A similar study[47] microinjected DL-homocysteic acid into various hypothalamic regions and observed the cardiovascular response. Depressor responses accompanied by bradycardia were elicited from the pre-optic area, the lateral hypothalamus, and the

anterior hypothalamus. Increases in heart rate and blood pressure occurred from the paraventricular nucleus and an area ventral to this along the wall of the third ventricle. Cardiac arrhythmias can be elicited on stimulation of the hypothalamus. These were first [48] demonstrated in paralyzed, ventilated cats that ventricular tachycardia was frequently elicited on stimulation of the posterior hypothalamus. This was preceded by prolongation of the Q-T interval in the electrocardiogram and marked increased in the size of the T wave. Shortening of the P-R interval was also observed as well as AV dissociation. Several ectopic ventricular pacemakers may be induced by such stimulation. In similar experiments involving stimulation of the ventromedial hypothalamus, [49] showed that incremental intensity of stimulation increased the severity of the rhythm from sinus tachycardia to ventricular ectopy, then to ventricular tachycardia, and ultimately to ventricular fibrillation. On termination of stimulation, this sequence was reversed, and the animal returned to sinus rhythm. Bilateral vagotomy had no effect on the responses. However, intravenous propranolol in the vagotomized animal abolished the effect of hypothalamic stimulation on heart rate and rhythm. This suggested that, under these circumstances, the arrhythmias were of sympathetic origin.

Other diencephalic areas that have been investigated with respect to cardiovascular changes include the thalamus and the zona incerta. The principal area from which cardiovascular response was elicitable was the mediodorsal nucleus, which has connectivity with the prefrontal cortex. Similar responses were obtained from the thalamic midline nuclei and the parafascicular region.

Microinjection of. L-glutamate into the zona incerta in rats produced depressor response and a bradycardia that could be incompletely blocked atropine.[50] The zona incerta is situated in the diencephalon and is bounded" ventrally by the cerebral

peduncle, dorsally by the medial lemniscus, laterally by the thalamus, and medially by the lateral hypothalamic area. Many of these injections also involved the subthalamic nucleus, the ventroposterior thalamic complex, and part of the lateral hypothalamic area, so the specificity of the response to the zona incerta remains somewhat in question.

The Amygdala and Cardiac Control

Recent evidence has indicated an important role for the amygdala in the control of cardiovascular function, especially with respect to autonomic-emotional integration. The amygdala is composed of numerous subnuclei, of which the central nucleus appears to play a major role in the elaboration of autonomic responses. There are profuse reciprocal connections to this region from brainstem and cortical regions involved in autonomic afferent input to it has been demonstrated in the cat. Studies [51] showed that the discharges of neurons in this region were related to the cardiac cycle and that their spontaneous firing rate could be changed by stimulation of the aortic depressor nerve and the carotid sinus nerve. Similarly, in humans undergoing surgery for epilepsy, amygdaloid cells have been identified which showed correlations in their firing rate with the cardiac cycle.

It is well known from animals and clinical studies that cerebrovascular diseases can alter Cardiovascular and autonomic function. Stroke has been shown to produce changes in autonomic function, increase the incidence of cardiac arrhythmias, cause myocardial damage, and raise plasma catecholamine levels..The most important consequence of these changes is an increased susceptibility to sudden death. In patients with acute stroke, the incidence of sudden death as a result of arrhythmic causes has been reported to be 6%.In clinical; and experimental studies, the most

important control sites of the autonomic function are found to be the insular cortex, amygdala and lateral hypothalamus.[52.]

Studies have shown that right hemisphere infarction is associated with a greater number of supraventricular tachycardia, and they speculated that a decrease in cardiac parasympathetic activity in right sided infarction may cause the probable reciprocal rise in the sympathetic tone.[52]

Some studies have demonstrated that changes in heart rate could be produced in squirrel monkeys by stimulation of the amygdala. Both tachycardic and bradycardic responses were encountered, and it was concluded that each had separate representation and projection pathways[53]. A study conducted induced cardiac arrhythmias by stimulation of the feline medial amygdaloid nuclei. Initially, before the arrhythmias became established, stimulation was associated with prolongation of the Q-T interval, S-T segment changes, and T wave amplitude increases. These ECG changes persisted on cessation of the stimulus leading to sequential bradycardia, eventually followed by idioventricular escape rhythms and ventricular fibrillation[54]. Others have shown that ventricular fibrillation induced by coronary ischaemia can be aborted by bilateral amygdaloid cooling, but not by similar cooling applied to adjacent areas or by unilateral cryoprobe application.

Cortical Areas and Cardiac Control

Cardiovascular changes were elicited on stimulation of the cortex as early as 1875, when it was demonstrated that surface stimulation of the motor cortex resulted in tachycardia accompanied by, but independent of, changes in arterial blood pressure. Cortical areas from which changes in heart rate have been elicited include

the sigmoid cortex, the frontal lobe and especially the medial agranular regions, the temporal pole and the cingulate gyrus

In the humans stimulation of the rostral cingulate gyrus(frontal lobe), both rise and fall in heart rate and blood pressure were observed, with no evidence of chronotropic organization. Similar responses were obtained from the anterior temporal lobe and uncus. In the rabbit, cardiovascular changes also have been elicited from the medial frontal cortex. The characteristic response to such midline frontal stimulation was bradycardia associated with a decline in blood pressure. The most marked responses were obtained on stimulation of the orbitofrontal cortex. The anterior midline cortex (orbitofrontal, prelimbic, and infralimbic regions) projects to other cerebral areas involved in autonomic control, which could serve as a substrate for these responses[55].

In addition to alteration of cardiac chronotropicity, ECG changes indicative of repolarization abnormalities have been reported on stimulation of the feline cerebral cortex. Similarly evoked Q waves, alterations in size and polarity of the T wave and of the QRS complex, the elevation or depression of the S-T segment on stimulation of the sigmoid gyrus ,the anterior sylvian and ectosylvian gyri have been observed. [56]
The few reports of cardiac arrhythmias induced by cortical stimulation include investigations which noted the dictation of both atrial and ventricular ectopic beats from the cat subiculum, the cingulate gyrus, and the temporal pole. A study was able to induce ventricular fibrillation after a delay of some 6 hours on stimulation of the feline hippocampus; the current strength was large and the mechanism may well have involved kindled seizure activity. [54]

The infrequent dictation of cardiac responses from the cerebral cortex contrasts with the relative ease with which cardiac arrhythmias and ECG changes can be provoked by stimulation of the hypothalamus and certain brainstem areas.

The insular cortex and stroke

The insular cortex is defined as that part of the cerebrum which overlies the claustrum. In primates this region lies buried beneath the frontoparietal and temporal opercula. Anatomical evidence indicates widespread connectivity between the insular cortex and other areas of the brain (such as the lateral hypothalamic area, parabrachial nucleus, and the nucleus of the solitary tract) which are known to be involved in autonomic control. This would strongly suggest a role for the insula in cardiovascular function.

The insular cortex has been implicated in the control of cardiac autonomic function in humans and animals. Insular cortex stimulation in rats results in ECG repolarisation changes and cardiac arrhythmias associated with elevation of plasma norepinephrine levels and myocardial damage.[31]

It is found that patients with insular infarction had a significant nocturnal blood pressure "rise" and higher norepinephrine levels than those with strokes in other cortical locations, suggesting increased sympathetic activity.[31]

Insular cortex infarction is associated QT_C prolongation and an increased incidence of ventricular arrhythmia.[31]

In animal models of stroke, myocytolysis can only be demonstrated on cardiac examination if the insular cortex is involved in the infarcted region. More recently, infarction involving the insula in an animal model of stroke was associated with

increased renal sympathetic nerve activity, prolongation of the Q-T interval (which is a frequent ECG accompaniment of acute stroke), and elevated norepinephrine levels. The insular cortex is a frequent site of seizure activity. Abnormal efferent traffic from this region to brainstem sites involved in autonomic control, such as may occur during a seizure, may be arrhythmogenic.

It may therefore be that stroke alters cardiovascular tone either by directly damaging the insular cortex or by damaging interrelated adjacent areas, thereby shifting the balance toward a predominance of sympathetic activation and leading to cardiac arrhythmias.

STROKE AND CARDIAC PATHOLOGY

The circumstantial evidence from the three controlled studies implies that although ischaemic coronary artery disease may contribute to the production of acute ECG changes, it is unlikely to be the only operative mechanism. However, none of the studies has been sufficiently well controlled to resolve this issue. More direct information has been gathered from autopsy studies. A high incidence of ECG changes has been noted following subarachnoid haemorrhage, and patients with such haemorrhage are often young and would not be expected to have significant ischaemic heart disease.A study identified 29 patients who had developed ECG changes following subarachnoid haemorrhage; 8 patients died and 5 were autopsied. None showed evidence of coronary artery disease or myocardial ischaemia. Similar findings were noted by others. [2] In a study 8 of the 37 patients who died were autopsied. All of these had elevated circulating creatine kinase (CK) levels and had died from an intracerebral haemorrhage. No evidence of an acute ischaemic cardiac event was found. Again, in a series of patients with acute cerebral infarcts confirmed by autopsy,

none of the patients who died and who had ECG abnormalities showed evidence of coronary artery occlusion. [10] Others also reported a patient who died following cerebral infarction from carotid occlusion without autopsy evidence of coronary artery stenosis. [57]

A study in 1933, described scattering subendocardial haemorrhages in patients dying during epileptic seizures. Subsequently these were observed in patients dying within a few days of onset of a stroke. Others [58] noted that the cardiac pathology (termed myocytolysis) following stroke encompassed a wider range of changes than just subendocardial haemorrhage. These included scattered foci of swollen myocytes surrounded by infiltrating monocytes, interstitial haemorrhages and myofibrillary degeneration. Moreover, the lesions were centered on intracardiac nerves rather than blood vessels, suggesting that humoral or ischaemic factors were unlikely to play a causal role.

Changes in myocardial formazan granule deposition have been observed in the tetrazolium-treated hearts of patients dying after acute cerebrovascular events. These precede histological evidence of myocytolysis. Studies done later[59]showed such changes in 89 percent of patients dying following subarachnoid haemorrhage, in 71 percent of those dying after an intracerebral haemorrhage and in 52 percent after ischaemic strokes. Similar abnormalities were seen in only 26 percent of hearts from control patients dying of other causes.

Some patients who died of subarachnoid hemorrhage were found to have subendocardial hemorrhages and focal areas of myocardial cell injury and myocytolysis.[13]

The mechanism of myocytolysis probably related to excessive sympathoadrenal activation. Indeed, plasma catecholamine levels following stroke are significantly

elevated compared with control groups. Myocytolysis may be induced by catecholamine infusion in animals or humans and is associated with pheochromocytoma. Similar changes are induced by stimulation of the canine left stellate ganglion. Moreover, the ECG changes following stroke may be mimicked by norepinephrine or epinephrine infusion. Both the ECG changes and the myocardial damage following subarachnoid haemorrhage can be abolished by phentolamine and propanolol[60]. It is therefore likely that sympathoadrenal tone can be elevated by stroke producing myocardial damage evidenced by ECG and CK changes[61].

Evidence from a number of studies indicate that patients with subarachnoid hemorrhage are at high risk for malignant ventricular arrhythmias, including ventricular tachycardia, torsades de pointes and ventricular fibrillation ,particularly if the corrected QTc interval is prolonged. A decrease in cardiac out put due to alteration in cardiac rate associated with subarachnoid hemorrhage ,such as sinus bradycardia, sinus tachycardia, or rapid atrial fibrillation, also can adversly affect patients clinical status.[13]

AUTONOMIC INFLUENCES ON CARDIAC FUNCTION

Both parasympathetic and sympathetic nerves have chronotropic effects on the heart. The parasympathetics decrease heart rate by reducing discharge of the SA nodal pacemaker, whereas sympathetics exert the opposite effect. Similarly both exert opposite inotropic effects, with the parasympathetics reducing and the sympathetic increasing contractility of atrial and ventricular myocardium. AV nodal conduction and His-Purkinje ventricular conduction are also affected by both parasympathetics (decreased) and sympathetics (increased).

The overall physiologic response to activation of sympathetics and parasympathetics also differs as a result of the different latencies of onset and termination of response. Sympathetic responses are both of slower onset (1to 3 seconds latency) and longer duration man those mediated by the vagus (latencies to onset of 200msec and to maximal effect of 400msec).[40,41] The different durations of the two responses depend on inactivation of the transmitter released from each. Cardiac vagal responses are mediated by acetylcholine, whose concentration is rapidly (2.5seconds) reduced enzymatically by acetylcholinesterase.[62] In contrast, termination of sympathetic responses depends on slower inactivation of norepinephrine through reuptake and diffusion.

The level of activity of cardiac parasympathetics and sympathetics is not fixed. Instead activity of each is reflexly modulated in response to stimuli from arterial and cardiopulmonary mechanoreceptors. In humans, sympathetic and parasympathetic outflow to the SA node is primarily influenced by arterial baroreceptors.. Interestingly, despite this dominant role for the baroreflex in the control of heart rate by the SA node, total interruption of the barorecerptor reflex does not lead to an erratic heart rate, even though similar interruption of the arterial barorerflex in both humans and other vertebrates leads to chronic lability of arterial pressure that responds excessively to environmental stimuli.[63,64]

In addition to the powerful effects of autonomic activity on cardiac conduction and contractility, the nervous system also may contribute to control ot coronary blood flow.[65] A central pathway running from the lateral hypothalamus to neurons in the peri acqueductal gray matter and thence to the rostral ventrolateral medulla that modulates cardiac sympathetic activity has been shown to be involved in modulation of coronary blood flow in several species. This central pathway and the sympathetic

responses to its activation may play an important role in integrating appropriate coronary blood flow response to behaviour and to activation of the baroreflex.[66]

CLINICAL OBSERVATIONS:

Cardiac function can be disturbed by interruption of its innervation. With total removal of the influences of cardiac nerves, the heart beats approximately 90 times per minute and does not alter that rate either spontaneously or in response to stimuli.[67] Incomplete denervation, as may occur with autonomic neuropathies, may also cause fixed tachycardia.[68]

Even with intact innervation, disturbances of the balance between sympathetic and parasympathetic influences on the heart can result in cardiac electrical instability and abnormalities of cardiac function. Clinically relevant cardiac alterations in the setting of primary neurologic disorders include electrocardiographic signs of ischaemia and arrhythmias.

ANATOMY AND PHYSIOLOGY OF CEREBRAL CIRCULATION

A knowledge of the anatomy and physiology of the cerebral circulation is essential to understand the changes brought about by cerebrovascular accidents.
Since most strokes are the result of cerebral infarction it is important to be familiar with the anatomy of both the extra and intracranial arterial supply and the w ay in which it is affected by atheroma.

The brain has a particularly rich blood supply which is derived from four main arteries: the left and right internal carotid and vertebral arteries. The internal carotid artery supplies the eye through the ophthalmic artery and then divides into the anterior and middle cerebral arteries which supply the anterior 2/3rd of the cerebral

hemisphere. The vertebral arteries unite to from the basilar artery, whose branches supply the brainstem and the cerebellum and then divides into the two posterior cerebral arteries supplying the posterior l/3rd of the cerebral hemisphere. The carotid arterial systems are interconnected by the anterior communicating artery and are linked to the vertebrobasilar system by the posterior communicating arteries so forming the Circle of Willis at the base of the brain.

In many individuals part of this arterial ring are hypoplastic and less than half are of the standard pattern. Nonetheless, it can form unless it is affected by disease, an excellent collateral channel for blood supply to the brain if one or more of the four main extra cranial arteries become occluded. Other potential collateral channels exists and may become functionally important if the major arteries are occluded. Branches of the internal carotid artery and external carotid artery anastomose with each other around and within the orbit, leptomeningeal anastomoses exist between the cortical branches, there are meningeal anastomoses since the dura is supplied by the internal carotid artery, external carotid artery and vertebral arteries.

Cerebral blood flow is auto-regulated to ensure a constant supply of blood to the brain between a mean systemic blood pressure of 60 to 160 mm of Hg [69] Within this range a rise in blood pressure will be met by intracranial vasoconstriction and fall by vasodilatation. However outside these limits cerebral blood flow will follow perfusion pressure and therefore result either in ischaemia if the systemic blood pressure falls or vasogenic oedema and ultimately hypertensive encephalopathy if systemic blood pressure rises. These limits within which auto- regulation is effective are set higher in patients with hypertension who will, therefore, experience ischaemia at a higher level of systemic blood pressure than in normotensive individuals. When the brain is damaged as a result of stroke and other diseases focal areas may no longer

autoregulate normally and blood flow will follow fluctuations in systemic blood pressure and the normal reactivity to carbondioxide is often impaired as well. These effects are extremely unpredictable and presumably depend on the age of the lesion and exactly where flow is being measured in relation to that lesion.

The various other factors which have been described to influence the cerebral circulation include alterations in cardiac output, haemotocrit and changes in the arteries distal to the brain like subclavian and descending aorta and various drugs like acetazolamide, theophylline etc.

The venous return iron, brain is accomplished by numerous thin walled veins which ultimately drain into venous sinuses lying between the two layers of duramater. Then they open directly into internal jugular vein. They communicate with meningeal veins and by emissary veins with veins of scalp.

BLOOD BRAIN BARRIER:

Over 50 years ago it was first demonstrated that when acidic dyes such as Trypan blue are injected into living animals all tissues except brain and spinal cord are stained. To explain this, the existence of blood brain barrier was postulated. All the vessels in central nervous system are surrounded by a thin covering formed by the endothelium of the capillaries and their basement membrane, external to which is extravascular space of nervous system. The blood barrier consists of

1. Endothelial lining cells
2. Basement membrane
3. Perivascular processes of astrocytes.

Acute cerebral lesions whether due to trauma, inflammation or infarction increase the permeability of the barrier and thus alter extra and intravascular

concentration of protein, water, electrolytes. Increased levels of catecholamines in the plasma of patients with cerebrovascular accidents could be due to altered blood brain barrier allowing catecholamines to escape into the circulation from brain substance. [70]

PATHOGENESIS AND PATHOLOGY OF STROKE

Cerebrovascular disease is caused by one of several pathologic processes involving the blood vessels of the brain. The process may.

1) Be intrinsic to the vessels, as in atherosclerosis, lipohyalinosis, inflammation, myloid deposition, arterial dissection, developmental malformation, aneurysmal dilatation or venous thrombosis.
2) Originate remotely, as occurs when an embolus from the heart or extracranial circulation lodges in an intracranial vessel.
3) Result from decreased perfusion pressure or increased blood viscosity with inadequate cerebral blood flow or
4) Result from rupture of a vessel in the subarachnoid space or intracerebral tissue.

A 'Stroke' is the acute neurologic injury occurring as a result of one of these pathologic processes. Other secondary symptoms may accompany stroke and vascular disease including pressure on cranial nerves from an aneurysm, .vascular headache or increased intracranial pressure with a venous thrombosis.

Normal brain function requires continuous supply of oxygenated blood. Cardiac arrest results in unconsciousness within 10 seconds, in animal experiments, total cessation of blood flow produces irreversible cerebral infarction within three minutes. Reduced blood flow may interfere with brain function but brain can remain viable for more prolonged periods. For example, patients who suffer cerebral embolism or cerebrovascular vasospasm following subarachnoid haemorrhage often

recover partially or completely, suggesting that focal areas of brain can remain functionless and ischaemic for hours, even days, yet recover us has led to the notion of an ischaemic zone that surrounds an infarct. These include excitotoxins released by damaged neurons, cerebral edema and alterations local blood flow.

An infarcted brain is pale initially, within hours to days the gray matter becomes congested with engorged dilated blood vessels and minute petechial haemorrhages. When an embolus blocking a major vessel migrates, lyses or disperses within hours, recirculation into the ischaemic area causes haemorrhagic infarction and may aggravate subsequent edema formation after the blood brain barrier has been disrupted. A primary intracerabral haemorrhage, on the other hand, damages the brain by directly injuring tissue at the site of the haemorrhage and by compressing the surrounding tissue. After an ischaemic stroke, intracerebral haemorrhage or transient episode of cerebral ischaemia has occurred, the prelude to therapy is a precise diagnosis. The evaluation of a stroke must include clear definitions of the character and location of the lesion, the vascular pathologic process producing the symptoms and the anatomy of spared collateral circulation to the ischaemic area. The brain repairs itself only by forming fibrogliotic scar tissue at the site of an infarction or haemorrhage, therefore therapeutic efforts after a stroke can only hope to minimize secondary loss.

Except for the elimination of risk factors, all aspects of therapy remain controversial. Because proof of efficacy is lacking for many therapies, current therapy is largely empirical and based on the physician's knowledge of the risks associated with various diagnostic procedures and therapeutic initiatives.

CLASSIFICATION AND DIAGNOSIS OF STROKE

A detailed classification comprising all the problems involved in cerebrovascular disease is not yet available. In 1958 an Adhoc Committee of National Advisory Council of National Institute of Neurological Diseases and Stroke published a "Classification and Outline of cerebrovascular diseases" which remains a useful basis of classification today. The principle types of cerebrovascular diseases were classified as follows:

1) Cerebral infarction
2) Transient cerebral ischaemia without infarction
3) Intracranial haemorrhage
4) Vascular malformations and developmental abnormalities
5) Inflammatory diseases of arteries
6) Vascular diseases without changes in the brain
7) Hypertensive encephalopathy
8) Dural sinus and cerebral venous thrombosis
9) Strokes of undetermined origin.

General classification given by Dalal P.M. (1999)is as follows[71];

1. Ischaemic strokes

A. With cerebral infarction

1. Cerebral thrombosis with or without atherosclerosis
2. Cerebral embolism (congenital heart disease and acquired valvular disease, cardiomyopathy, myocardial infarction, endocarditis, prolapsed mitral valve, etc)
3. Lacunar strokes (deep, small cerebral infarcts)

4. Cerebral venous thrombosis and cortical thromobophlebitis
5. Arteritis (syphilitic, tuberculous, rheumatic, Takayasu's disease, collagen diseases, etc.)
6. Blood diseases (polycythaemia, sickle cell anaemia, thrombotic states, hyperproteinaemia, etc.)
7. arterial hypotension and anoxic encephalopathy
8. dissecting aneurysms of brachio-cephalic vessels
9. Angiographic complications
10. Infarction of undetermined cause

B. With cerebral ischaemia

1. Transient ischaemic attacks (platelet-fibrin microemboli associated with atheroma, etc.)
2. Arterial hypotension or haemodynamic crisis (e.g. massive gastrointestinal bleeding)
3. With cardiac arrhythmias (atrial fibrillation, complete heart block, sick sinus syndrome, etc.)
4. With migraine
5. Local embolism from athero-plaque and paradoxical embolism
6. Undetermined source

C. Idiopathic and rare types (drugs and oral contraceptives, disseminated intravascular coagulopathy, consumption coagulopathy, cerebral malaria, Bahcet's syndrome, congophilic angiopathy, homocystinuria, hyperviscosity syndrome, paraproteinaemia, etc) .

2. Haemorrhagic strokes

1. Hypertensive cerebral haemorrhage
2. Ruptured aneurysm (saccular, mycotic, etc.)
3. Ruptured angioma (arterial, venous or mixed)
4. Trauma
5. Blood dyscrasias (leukaemia, purpura, hyperviscosity syndrome, other bleeding diathesis)
6. Complication of anticoagulant therapy
7. Bleeding in brain tumours
8. Miscellaneous causes (arteritis, bleeding in haemorrhagic infarct, etc.)
9. From undetermined source

3. Strokes of undetermined origin

1. Moya moya disease
2. Fibromuscular dysplasia
3. Binswanger's subcortical arteriosclerotic encephalopathy
4. Winiwater - Buerger disease (thromboangitis obliterans cerebral form)
5. Aortic arch syndrome (non-inflammatory)
6. Leukoaraiosis

Diagnosis of cerebrovascular diseases:

There are two separate aspects of the problem of differential diagnosis

1. Vascular disease must be distinguished from other neurologic illness
2. Different kinds of vascular disease must be separated from one another.

Differentiation from other neurologic disease*:*

The diagnosis of vascular lesion rests essentially on recognition of stroke syndrome i.e. sudden non convulsive focal neurological deficit. In its more severe form patient become hemiplegic or comatose.

The three criteria by which the stroke is identified are:

1. Temporal profile of the clinical syndrome
2. Evidence of focal brain disease
3. Clinical setting

Neurologic deficit in cerebrovascular stroke develops suddenly and later in the illness, if death does not occur, stabilization and some degree of recovery takes place After differentiating from other neurologic diseases, the type of stroke should be diagnosed with high degree of accuracy. Since each one requires a special approach therapy and management, correct diagnosis is advantagenous to the patient.

The criteria for diagnosis are as follows. Differentiation of cerebral thrombosis, embolism, hypertensive intracerebral haemorrhage, ruptured sacular aneurysm and angioma is made on the following points.[72]

A.The atherosclerotic thrombosis:

1. History of prodromal transient ischaemic attacks.
2. An intermittent stepwise evolution of neurologic deficit with recovery or improvement between worsenings rather than a steady progression.
3. Relative preservation of consciousness unless the upper part of basilar territory is infarcted.
4. Normal CSF except for modest elevation of protein and occasional pleocytosis with massive infarctions.

5. Evidence of atherosclerosis elsewhere, especially in the coronary, peripheral vessels and aorta.
6. Onset during sleep or shortly after arising or during period of hypotension.
7. Advanced age of the patient and the presence of disorders usually associated with atherosclerosis (hypertension, diabetes, xanthomatosis)
8. Headache of moderate severity (either as a prodromal warning of accompanying the stroke).
9. Carotid bruit in the neck indicating carotid stenosis
10. Occlusion of internal cartotid artery in the neck as determined by palpation auscultation, ophthalmodynamometry.

A. Arteritis:

1. Evidence of arteritis elsewhere
2. In any young individuals who manifest neither with hypertension, nor with signs of cardiovascular disease
3. In individuals with an infection which could affect the meningeal vessels (syphilis, tuberculosis etc.)

B. Venous Thrombosis:

Should be considered when focal neurological signs develop in the period following parturition or an operation in the course of meningeal infection, ear or sinus suppuration and in patients with cachexia, congenital heart disease, polycythemia or sickle cell disease.

D. Cerebral Embolism:

1. Abrupt development of completed stroke within a few seconds to minutes
2. Absence of prodromal TIAs
3. Source of emboli usually in the heart, i.e. atrial fibrillation or other arrhythmia, myocardial infarction, subacute bacterial endocarditis, mitral stenosis, valvotomy or prosthetic valve and marantic endocarditis associated with carcinoma
4. Evidence of recent embolism in other organs (spleen, kidney, extremities, G.I. tract or lungs)
5. Evidence of recent involvement of several regions of the brain in different cerebrovascular territories
6. A clear CSF except in small proportion of cases with extensive haemorrhagic infarction.
7. Rapid improvement (many embolic strokes produce persistent deficits, but it is not uncommon for an extensive focal deficit to reverse itself in minutes, hours or days)
8. Relative preservation of consciousness in the presence of extensive neurological deficit, unless the upper part of the basilar territory is involved or massive cerebral oedema has occurred with temporal lobe tentorial herination.
9. Occurrence at an age when atherosclerosis is usually not a factor and the absence of hypertension, diabetes, arteritis or infection.
10. Localized headache of moderate severity.

C. Hypertensive cerebral haemorrhage:

1. Presence of hypertension
2. Absence of prodromal phenomenon
3. Frequent, but not invariable occurrence of headache.
4. Gradual development of neurologic deficit over a period of 10 minutes upto several hours (some times the onset is more abrupt)
5. Grossly bloody CSF (this is also variable since rarely the haemorrhage does not extend to ventricular system and this does not reach CSF)
6. Deepening stupor or coma. Generally speaking, a patient with an extensive paralysis due to haemorrhage will be stuporous and a hemiplegic stroke which leaves the patient alert and the mind relatively clear, proves in nearly all instances to be due to an infarct.
7. Onset during working hours rather than sleep.
8. Neck rigidity, except when deep coma supervenes

D. Ruptured saccular aneurysms:

1. Sudden onset of severe headache
2. Brief or prolonged loss of consciousness at onset (in the most severecases coma persists and patient dies within few hours)
3. Grossly bloody CSF under increased pressure
4. Relative alertness (after initial episode) and absence of focal neurologic signs (except for 3^{rd} and 6^{th} nerve palsy)
5. Preretinal (subhyaloid) haemorrhage (these suggest ruptured aneurysm or A.V. malformation although they can occur in massive intracerebral haemorrhage and after trauma)

6. Stiff neck on forward flexion, Kerning's sign
7. Transient weakness, numbness, aphasia or seizure at the onset.
8. Usually an absence of warning attacks although there may be a history of one or more transient episodes of headache
9. Onset during exertion, sexual intercourse etc.
10. Absence of hypertension in many cases
11. Co-arctation of aorta and polycystic diseases of kidneys may be present.

If CSF is bloody and patient retains mental clarity or is only mildly confused, aneurysm or cerebellar haemorrhage is the likely diagnosis.

Intracerebral haemorrhage should be differentiated from a vascular malformation by fallowing criteria.

1. Stroke in young patient with bloody CSF in the absence of hypertension
2. Antecedent epilepsy often with postictal paralysis
3. Presence of cervical or cranial bruit.
4. Repeated subarachnoid haemorrhages
5. Calcification in the region of lesion in the skull films
6. Lateralising neurologic signs which are more frequent with aneurysm

CARDIOVASCULAR CHANGES SECONDARY TO STROKE [73]

Cardiac abnormality predominantly arrhythmias have been observed in various central nervous system diseases. ECG changes observed in cerebrovascular accidents resemble those of myocardial ischaemia or even infarction. They have been observed in the absence of primary cardiac disease and in some cases who survived with such

changes. They reverted back to normal. The commonly observed ECG changes include:

1. Q-T interval prolongation
2. Prominent U wave
3. Ischaemic pattern with S-T segment depression and T-wave inversion
4. Various arrhythmias like:
    - Sinus arrhythmia
    - Sinus bradycardia
    - Sinus tachycardia
    - Supraventricular tachycardia
    - Atrial fibrillation
    - Atrial flutter
    - Atrial premature complexes
    - Paroxysmal atrial tachycardia
    - VentricularPrematureComplexes
    - Ventricular Tachycardia
    - Ventricular fibrillation
    - Conduction disturbances like first, second and third degree AV blocks
    - Rarely other arrhythmias

Relation of ECG changes to the site of CNS lesions:

There is little information in human subjects regarding a relationship between specific stroke location and disturbances in cardiac rhythm, conduction and repolarisation, but some experimental evidence is available. The left insular cortex of rat contains a site of cardiac chronotropic representation.[23,74] Because of this observation, it is postulated that there might be a difference in cardiac expression between right and left sided lesions[26]. There is evidence that asymmetries in brain function influence the heart through ipsilateral pathways and there is a reported association between right hemispheric strokes and tachyarrhythmias of supraventricular origin and left hemispheric strokes and arrhythmias of ventricular origin. More recent reports call attention to the cardiovascular effects of human insular cortex stimulation, and there is a reported association between a neurosurgical intervention in the region of the left insular cortex, premature ventricular complexes and prolonged Q-T interval.

Causation of ECG changes in cerebrovascular accidents[75]

Several mechanisms have been postulated for the genesis of ECG changes in acute CVA. They are as follows [75]

1. Hypertension or other haemodynamic factors
2. Electrolyte imbalance: mainly low levels of total body K^+.
3. Irritation of Area 13 on orbital surface of the frontal lobes. Vagus nerve is thought to have a cortical representation in this area.
4. Intense sympathetic tone may be the cause of ischaemic changes.
5. Scattered myocardial damage or myocardial myocytolysis.
6. Sub-endocardial petechial haemorrhages.

Cerebrovascular spasm is a well-recognised accompaniment of subarachnoid haemorrhage.[76,77] Spasm affecting small hypothalamic blood vessels would lead to lesion in these areas and cause an abnormal hypothalamic response with increased circulating steroid levels via the pituitary-adrenal axis. Corticosteroids are also thought to play a role in potentiating the cardiotoxic effects of catecholamines. Increased sympathetic tone has been supported by the presence of high plasma nor-epinephrine concentrations with stroke patients. There is recent experimental evidence that the cardiac arrhythmia produced by experimental cerebral ischaemia could be prevented by autonomic blockade.[48,49]

In a study conducted in I979[78] a serial analysis of CPK was done in patients with acute CVA, CPK levels were elevated in 5% of the cases and ECG changes in most of the patients in this group were suggestive of myocardial ischaemia. CPK-MB isoenzyme levels were assayed in 25% of these cases and were found to be elevated in 17% of the cases. These observations suggest that clinically undetected myocardial lesions occurs in association with acute cerebrovascular lesion and might be responsible for ECG changes observed. Similar study was done in the past in 1969[79] .

Site of Cerebral lesion of ECG changes:

The site of cerebral infarction appeared to be a factor in the genesis of arrhythmias. Patients with hemispheric infarction had more severe arrhythmias as than those with lesions in brainstem[21]

A study[70] opined that primary neurologic effect of release of neurotransmitter substances into systemic circulation through the damaged blood brain barrier in the

hemispheric lesions may cause increased incidence of ECG changes as compared to brainstem lesions

ELECTROCARDIOGAPHIC COMPLEXES AND THEIR ELECTRO-PHYSIOLOGICAL IMPORTANCE

Recognition of normal electrocardiogram is the most important aspect of understanding the electrocardiogram. The electrocardiogram is recognized as being within or beyond normal limits by the normality or otherwise of the shape and dimensions of its various deflections, their frequency and their relationships in time with the deflections preceding and succeeding them. This introduction to the subject considers only morphological normality or abnormality.[80,81]

Electrophysiology of heart:

The electrocardiogram (E.C.G) is the graphic description of the electrical activity of the heart recorded from the body surface by electrodes positioned to reflect activity from a variety of spatial perspectives.

The following are the factors involved in the genesis of the electrocardiogram.

1. Initiation in impulse formation in the primary pacemaker i.e. S.A. node
2. Transmission of impulse through the specialized conduction system of the heart.
3. Activation (depolarisation) of atrial and ventricular myocardium.
4. Recovery (repolarisation) of all the above areas.

Intracellular potentials:

Most of the cardiac cells maintain a membrane resting potential (MRP) of -90 mv, the inside of the cell which is negative with respect to outside. The major factor that determines the MRP is gradient of postassium (K) across the cell membrane. Intracellular concentration of K^+ is 30 to 35 times higher than the extracellular concentration. On the other hand, an opposite gradient exists for sodium ions (Na^+). The extracellular concentration of Na^+ is about 10 to 15 times higher than intracellular concentration. At the onset of depolarisation of cardiac muscle cell, there is abrupt change in the permeability of cell membrane to sodium. Sodium ions (and calcium ions to a lesser extent) enter the cell and result in sharp rise of intracellular potential to positivity (+20mv). This is designed as "***Phase - O***" and represents the fast inward current.

Following depolarisation, there is relatively slow and gradual return of intracellular potentials to MRP (Phase-4). This is repolarisation and is divided into three phases.

Phase - 1*:* An initial rapid return of intracellular potential to 0 mv. This results mainly due to abrupt closing of sodium channels. Chloride ions entering the cell may also contribute to this phase.

Phase - 2: *A* plateau phase or repolarisation owing to slow entrance phase or repolarisation owing to slow entrance of calcium into the cell.

Phase - 3: This represents the slow gradual return of intracellular potential to MRP. It results from extrusion of K^+ out of the cells, which re-establishes normal negative resting potential.

However, the cell is left with an excess of Na^+ and deficient if K^+. To restore the original ion concentration, a cell membrane sodium - potassium pump mechanism becomes effective. The energy required for this pump is derived from conversion of ATP to ADP. This pump removes Na^+ from the cell and permits K^+ influx.

The summation of all phase - O potential of atrial myocardial cell results in P wave of E.C.G. All phase - O potentials of ventricular muscle cell produce the QRS complex. Phase - 2 correlates with ST segment and Phase-3 with T wave of ECG. The duration of action potential is longer in Purkinje fiber than in any other site. This is due to the prolongation of Phase-2 and Phase-3 and results in U wave of ECG.

P-Waves: The deflection produced by atrial depolarisation. The limb lead which normally best shows the P wave is lead II. In this lead normal P wave does not exceed 0.11sec, and its height does not exceed 2.5mm.

P waves are usually upright in lead II, AVF and from V_4 to V_6. Upright or biphasic P waves may occur in V_1 and V_2. If the P waves are biphasic the negative component of the P wave must have a smaller area than the positive component.

QRS Complexes*:* QRS complexes results from depolarisation of right and left ventricles. Any Q wave present in lead I, II or AVF must not exceed one quarter the height of the ensuing R wave and must not equal or exceed 0.04 sec. Q waves present in AVL should satisfy the same criteria as those in leads I, II or AVF unless the frontal plane of the QRS axis is more positive than +60 degree in which case large Q waves in AVL are acceptable. The electrical axis of the heart must not lie outside the limits of-30° to+110.°

The QRS complex in V_1 typically shows a small initial positive wave followed by a large negative wave and in V_6 a small initial negative wave followed by a large positive wave. In general, the size of the initial positive R wave increases progressively from V_1 -V_6. However, certain normal variations are possible. The direction of the initial part of the QRS is positive in V_1-V_3 but negative in V_4-V_6 i.e. V_1-V_3 show initial R waves and V_4-V_6 initial Q waves. Leads showing a RS complex are being primarily influenced by right ventricular myocardium and leads showing a QR complex by left ventricular myocardium. The transition zone between the right and left ventricular precordial leads is seen to be between V_2-V_4.

When the transition zone falls outside this region the heart is said to be rotated. If the transition zone occurs further to the left of the precordial leads (e.g. $V_5$and V_6) then the heart is said to be clockwise rotated. Conversely, if the transition zone is moved to the right in the precordial leads the heart is said to be counter clockwise rotated. Clockwise and counter clockwise rotation refer to a normal state of variability between one subject and another and are not in themselves indicative of abnormality.

The dimensions of the individual wave making up each part of the precordial QRS complexes are of crucial importance in determining normality or otherwise.

1) The amplitude of QRS complex will depend upon following factors:
    a. The electrical force which is generated by the ventricular myocardium
    b. The distance of the sensing electrode from the ventricles. Thus precordial leads reflect large amplitude QRS deflection than the frontal plane leads.
    c. Body build: Thin persons having larger deflection than an obese person
    d. And also on the direction of the frontal plane QRS axis.

Thus it is clear that several variables make it difficult to ascribe obsolute values to QRS amplitude.

2) Maximum duration: the total QRS duration in any one of the precordial leads must not exceed 0.10 sec.
   a) Q wave criteria:
   b) Precordial Q wave must not equal or exceed 0.04 sec.
   c) Precordial Q wave must not exceed a depth greater than a quarter of the height of the R wave in the same lead.
3) Ventricular activation toe: in leads facing the left ventricle QRS complexes, must not exceed 0.04 sec.

S-T segment: It must not deviate by more than 1 mm above or 0.5mm below the isoelectric line in either limb lead or precordial lead. The isoelecrtic line is that vertical position of the E.C.G. recording when no part of the heart is be.ng depolarised or repolarised i.e. the interval between the end of one T wave and beginning of the next P wave i.e. T-P interval.

T Waves: In general, the T waves and QRS complexes in the limb leads are concordant, i.e. when the QRS complexes are negative, the T waves are negative. A normal T wave will always be negative in AVR and positive in I and II, T waves can be positive or negative in AVL and III without necessarily indicating abnormality.

Normal adults usually have upright T waves in V_1 and V_2. But if there is a T wave inversion in V_2 with an upright T wave in V_1 it is considered abnormal. T wave inversion in V_4, V_5 or V_6 is always abnormal. The normal T wave is always upright in the left oriented leads and is always upright in the right oriented leads m the adults.

There are no strict criteria for the size of the T waves. T wave is lead V_6 is usually of the greater amplitude than the T wave is lead V_1. A T wave amplitude is lead V_1, which is equal to or greater than T wave amplitude of lead V_6 constitutes a pointer to the potential presence of cardiac disease. Tall or relatively tall T waves are normally seen in mid precordial leads (V_2 - V_4).

U Wave: It is the deflection seen following the T wave. Exact cause is unknown. Currently thought to be slow repolarisation of ventricular (Purkinje) conductive system. U wave is positive in leads in which QRS complex is positive. Inversion of U wave in the precordial leads is abnormal and is usually due to either coronary heart diseases or hypertensive heart diseases.

QRS-T angle: The mean frontal plane QRS axis and the mean frontal plane T wave axis are usually similarly directed i.e. they are close to each other, the angle between them is consequently narrow and does not normally exceed 60 degree.

In the presence of myocardial disease, the T wave axis tends to deviate from the ischaemic region whereas the QRS axis usually remains normally directed or may even deviate in the opposite direction. The angle between the QRS and T wave axis therefore widens and it is usually a sign of myocardial disease when it exceeds 60 degree in the adult.

P-R interval: Interval from the beginning of P wave to the beginning of QRS complex normal range 0.12 to 0.20 sec.

Q-T Interval: The Q-T interval is measured from the beginning of the QRS complex, to the end of the T wave. It represents the combined phase of depolarization and repolarization.

The Q-T interval varies with age, sex and heart rate. It lengthens with bradycardia and shortens with tachycardia. The valid Q-T interval is therefore derived by correcting for the variation in rate. The corrected Q-T interval is known as the Q-Tc, for which absolute values have been established. It must be emphasized, however, that measurement of the Q-T interval is technically difficult and often erroneous. This is due to difficulty in determining the exact end point of , the T wave, since the T wave is often obscured by the U wave. The U wave is usually isoelectric in lead AVL and it is therefore best to measure the Q-Tc interval in this lead. The criteria now used for normal and abnormal Q-Tc intervals are thus only approximate. As a general rule, with heart rate of 60 - 80 beats per minute the Q-Tc interval does not normally exceed 0.39 sec. At normal heart rates between 60 to 80 beats per minute it should not exceed half the R-R interval. The normal Q-Tc should not exceed 0.42 sec in men and 0.43 sec in women.[81]

A prolonged Q-Tc interval may be associated with[26] Diffuse myocardial disease or myocardial infarction.

a) Acute carditis.
b) Head injury or cerebrovascular accident.
c) Hypocalcaemia.
d) Quinidine or procainamide effect
e) Tricyclic and tetracyclic antidepressant drugs
f) Hypothermia

g) Hypertrophic cardiomyopathy

h) A-V blocks

i) Jervell-Lange-Nielsen syndrome

j) Romano-ward syndrome

A shortened Q-Tc interval may be associated with either digitalis effect, hypercalcaemia, hyperthermia, vagal stimulation.

METHODOLOGY

The material for the present study comprised of thirty six patients, admitted to various medical units of R L JALLAPPA Hospital, attached to SRI DEVRAJ URS Medical College, KOLAR, during DECEMBER 2008-MAY 2010.

The cases of head injury and those who did not survive up to 2 days after admission were excluded from the study.

Individuals with known cardiac diseases and those under treatment with cardiovascular drugs have been particularly excluded from the study. Patients with hepatic or renal disorders which are known to induce circulatory, metabolic and electrolyte imbalances also have been excluded from this study.

Detailed clinical history was recorded with particular reference to prodromal symptoms, mode of onset, evolution of neurological disease. Symptoms of valvular heart disease, hypertensive ,diabetes and ischaemic heart disease was particularly enquired into.

Thorough physical examination with special emphasis to nervous system and cardiovascular system was done as per the proforma.

The diagnosis of cerebrovascular accident was made by recognizing "Stroke Syndrome." The three criteria by which the stroke was identified were[72]

1) Temporal profile of the clinical syndrome
2) Evidence of focal brain disease
3) Clinical setting

Different types of cerebrovascular accidents were diagnosed on the following criteria.

1) Cerebral thrombosis was diagnosed when:

a) An intermittent stepwise evolution of neurologic deficit occurred.

b) History of prodromal transient ischaemic attacks were present.

c) Neurologic deficit occurred in elderly patient with evidence of atherosclerosis elsewhere

d) The onset was during sleep, shortly after arising or during period of hypotension.

2) Cortical venous thrombosis (CVT) with infarction is considered when focal neurologic signs developed in the period following parturition, or an operation, in the course of meningeal infection, ear or sinus suppuration.

3) The diagnosis of cerebral embolism was made in cases:

a) Of abrupt development of completed stroke, within a few seconds or minutes, without prodromal symptoms.

b) With a source of emboli usually in the heart or with evidence of recent emboli in other organs.

c) With the evidence of recent involvement of several regions of brain in different cerebrovascular territories and in those with rapid improvement.

4) Intracerebral haemorrhage was diagnosed when patient suffered sudden onset of headache with progressive lateralizing signs, progressive deterioration of consciousness

5) The diagnosis of subarachnoid haemorrhage was made:

a) When an abrupt onset of headache with signs of meningeal irritation occurred in the absence of focal neurologic signs.

b) When the onset was during exertion each patient was assigned to one of the following types of cerebrovascular accidents depending on these criteria:

1) Cerebral thrombosis
2) Cerebral haemorrhage
3) Subarachnoid haemorrhage (SAH)
4) Cerebral embolism
5) Cortical venous thrombosis (CVT)

12 Lead electrocardiogram was taken for all cases as soon as possible after the admission (within 24 hours). This was studied in detail as per the proforma.

In case of ECG abnormality, every attempt was made to find out the cause, including the electrolyte imbalance. Serum electrolytes (sodium, potassium and calcium) estimations were done by taking a sample of blood immediately.

Subsequent to the ECG taken at admission, 12 lead ECGs were repeated at 24hrs and at the time of discharge and follow up of individual patient was done. In analyzing these ECGs, the following criteria were considered to recognize the abnormalities.

I. Normal sinus rhythm: criteria

1. P-wave of sinus origin.
2. Constant and normal P-R interval
3. Constant P wave configuration in a given lead.
4. Rate between 60 and 100 beats/min.
5. Constant P-P interval.

II. Sinus arrhythmia:

This was recognized by normal P-QRS-T complexes, with alternating periods of gradually lengthening and shortening of P-P intervals.

III. Sinus tachycardia:

This was diagnosed when all the criteria for sinus rhythm are fulfiled except that the rate is more than l00/min. (Usually between 101-160/min).

IV. Sinus bradycardia:

This is diagnosed when all criteria for sinus rhythm are fulfilled except that the rate is less than 60/min. (Usually between 45-59/min).

V. Q waves

Q waves were considered significant, if they were greater than 0. 04 sec. in duration and 1/4th of R wave for the lead.

VI. Left ventricular hypertrophy (L VH) was considered when:

1. R wave in V_5 or V_6 more than 27 mm
2. The sum of R wave in V_5 or V_6 and S wave in V_1 is more than 35mm.
3. R wave in L_1 more than 15 mm.
4. R wave in lead AVL more than VAT over .05 sec in V_5 and V_6 1l mm
5. Secondary ST-T changes.

VII. Right ventricular hypertrophy (R VH) was considered when:

1. Right axis deviation.
2. R wave greater than S wave in V_1 or R wave greater than 5mm in V_1
3. RS or r S complexes in left oriented leads
4. RS complex in transition zone
5. Clockwise electrical rotation.
6. S-T depression and T inversion in leads to V_1 - V_3

VIII. R. B.B .B. was identified when:

1. RsR' complexes in V_1-V_2.
2. Slurred and bizarre S wave in V_5-V_6
3. QRS interval 0.12 sec. or more.
4. S-T depression and T wave inversion in V_1- V_3.

IX. L.B.B.B. was identified when:

1. Tall and notched R wave (rsR^1 or RsR^1 complexes) in V_5V_6
2. Widened, notched QS complexes or r S complexes in V_1 and V_2
3. QRS interval 0.12 s or more
4. VAT 0.09 sec. or more in V_5 - V_6
5. S-T depression and T wave inversion in V_4 - V_6

X. S-T segment:

This is considered abnormal when there is elevation or depression of more than l mm from the isoelectric line.

XI. T wave abnormality was considered when there was:

1. Inversion of T waves in which it should have been upright (I, II, V_3-V_6)
2. Abnormally tall T waves of more than 5mm m standard lead and 10mm in any precordial lead.

XII. U wave was taken as significant when:

1) Exaggeration of U wave voltage was noted. (> 2mm)
2) When appeared in leads in which it was not normally seen (other than V_3 V_4)

XIII. Q-T interval:

This was measured and was corrected with the heart rate (Q-Tc) using the Normogram .It was taken as prolonged when Q-Tc was more than 0.43 seconds.

All patients were subjected to investigations like serum electrolytes, urine analysis, blood sugar, blood urea, serum cholesterol, serological evidence for syphilis (VDRL).

During the hospital stay all the cases were followed up regularly upto discharge and repeat ECGs were taken on 24hrs and 72hrs to see for any change in the initial ECG status. All patients were managed with general and specific measures according to the type of stroke. Follow up ECG was taken at the time of discharge.

The only prognostic criteria considered was mortality among patients within study period.

RESULTS

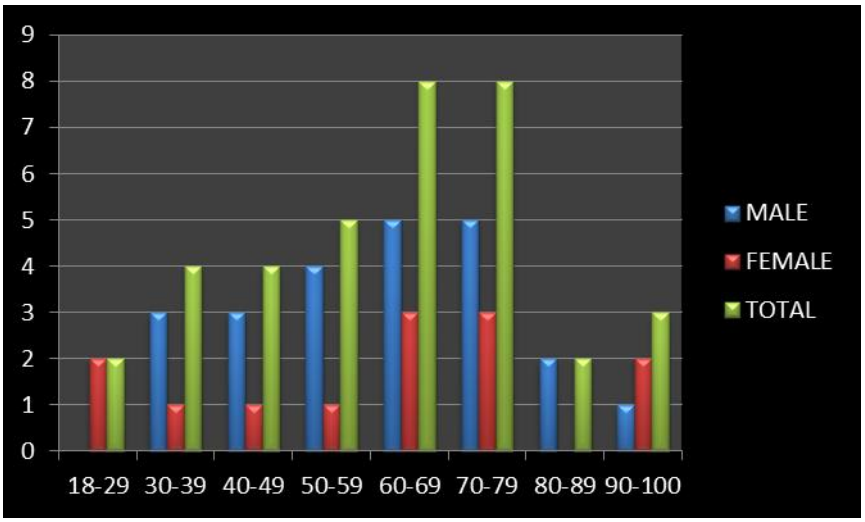
Thirty six patients admitted with acute stroke were studied in detail and following observations were noted:

Age and Sex distribution

Table A-I showing Age and Sex Distribution

AGE GROUP	MALE	FEMALE	TOTAL	Percentage
18-29	0	2	2	5.6
30-39	3	1	4	11.11
40-49	3	1	4	11.11
50-59	4	1	5	13.89
60-69	5	3	8	22.22
70-79	5	3	8	22.22
80-89	2	0	2	5.56
90-100	1	2	3	8.33
	23	13	36	100

Table A-l shows the age and sex distribution of the stroke patients. As evident from this table, strokes are common in middle aged and elderly. Incidence of stroke below 40 years is 16.7% and in the age group above 40 years is 83.3%. The youngest patient in this study was of 21 year old i.e. a case of CVT. and the oldest was 96 years old. Male and female ratio in this study group is 3:2.



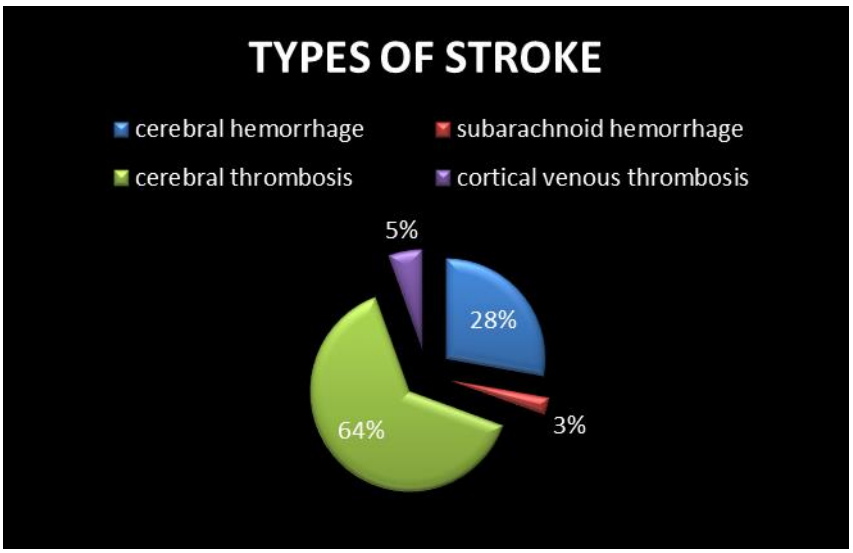
Types of stroke

C T scan was used for all patients to identify the type of stroke. Table A-II shows the incidence of different types of stroke.

Table A- II Incidence of different types of stroke

Type of stroke	No. of Patients	Percentage
Cerebral haemorrhage	10	27.778%
Subarachnoid haemorrhage	1	2.778%
Cerebral thrombosis	23	63.889%
Cortical venous thrombosis	2	5.556.%
Total	**36**	**100%**

As evident from table A-II cerebral thrombosis contributed highest number of cases i.e. 63.889% fallowed by intra cerebral hemorrhage i.e 27.78% subarachnoid haemorrhage 2.77% and CVT accounted small percentage of cases i.e 5.56%. Our study did not include cerebral embolism.

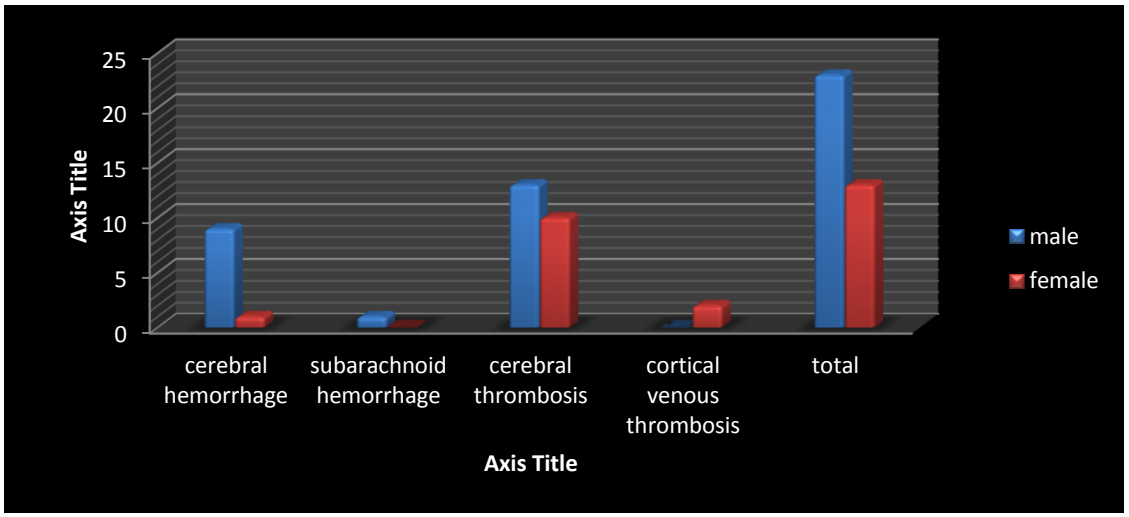


Sex based distribution of different types of stroke

Table A-III shows sex based distribution of stroke

Type of stroke	Males	Percentage	Females	Percentage	Total
Cerebral thrombosis	13	56.52%	10	76.93%	23
Cerebral Hemorrhage	9	39.10%	1	7.69%	10
SAH	1	4.34%	0		01
CVT	0		2	15.38%	02
Total	23		13		36

As evident from the table A-III total incidence of stroke in males exceed that of females. Cerebral thrombosis and intra cerebral hemorrhage is more in males as compared to females, where as SAH is confined to males and CVT is exclusively confined to female group.



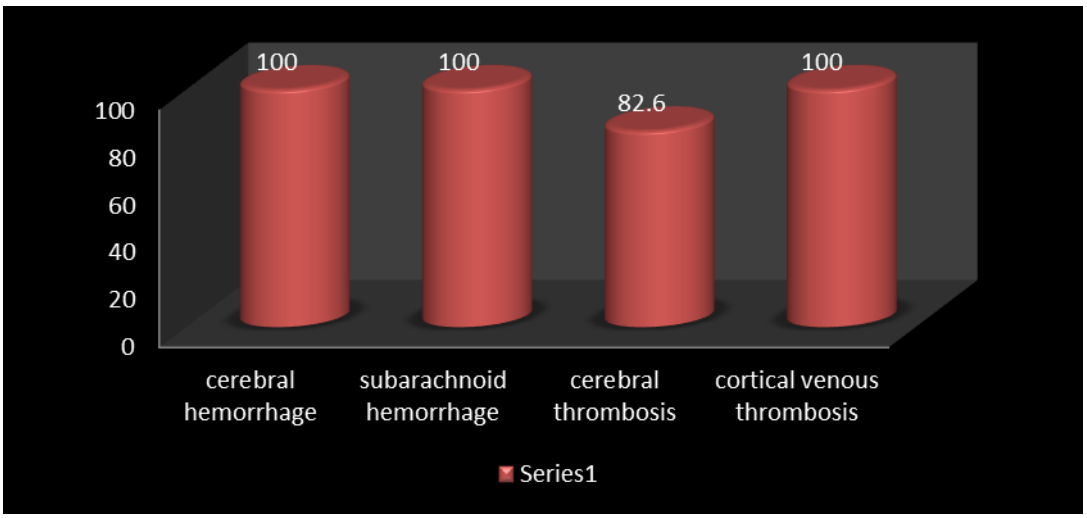
Incidence of ECG changes

Table A-IV - Incidence of abnormal ECG

Sl. No.	Type of Stroke	Total Number of Cases	Cases with abnormal ECG	Percentage
1	Cerebral haemorrhage	10	10	100
2	Subarachnoid haemorrhage	1	1	100
3	Cerebral thrombosis	23	19	82.60
4	Cortical venous thrombosis	2	2	100
	Total	**36**	**32**	**88.89**

The table A-IV shows the incidence of abnormal ECGs in patient with stroke. Out of the thirty six cases studied thirty two showed ECG abnormalities (88.89%). Remaining four cases (11.11%) did not show any changes in ECG.

All the haemorrhagic strokes i.e. both intracerebral haemorrhage and subarachnoid haemonhage, showed some ECG changes. Out of the 23 cases of cerebral thrombosis, only 19 (88.89%) showed ECG changes. Both cases of CVT showed ECG changes.

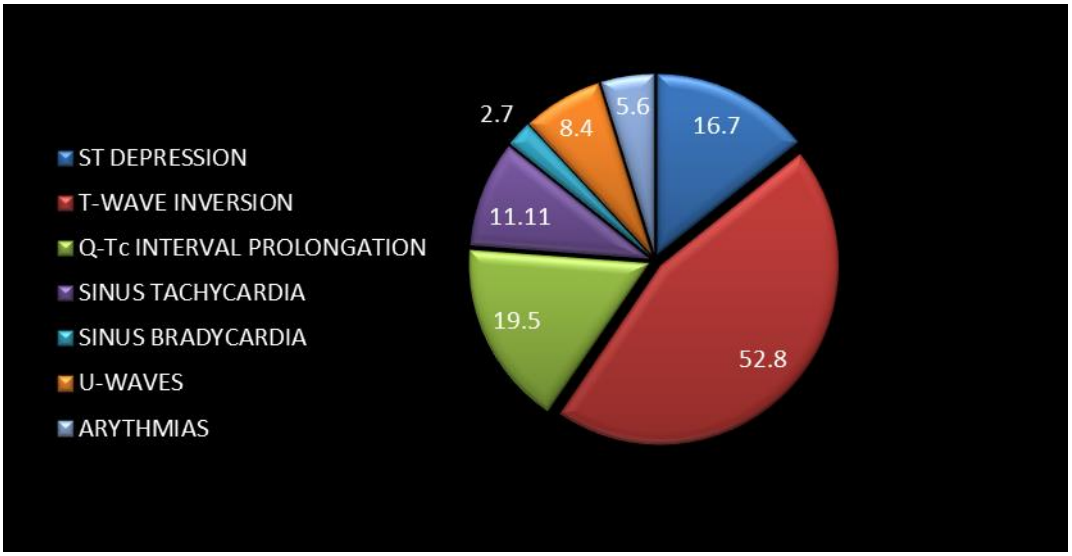


Types of ECG changes observed:

Table A-V Different types of ECG changes

Sl. No.	Types of ECG Changes	Number of Patients	Percentage
1	S-T segment depression	6	16.7
2	T wave inversion	19	52.8
3	Q-T_c interval prolongation	7	19.5
4	Sinus tachycardia (rate >100/min) sinus bradycardia (rate < 60/min) U waves Arrhythmias (atrial and ventricular)	4	11.1
5	Sinus bradycardia (rate V60/min)	1	2.7
6	U waves	3	8.4
7	Arrhythmias(atrial and ventricular)	2	5.6

As evident from the table A-V, which shows the types of ECG changes, S-T depression was seen in 6 cases (16.7%). T wave inversion was seen in 19 cases (52.8%), Q-T_c prolongation was seen in 7 cases (19.5%). Arrhythmias in the form of atrial fibrillation is seen in two cases was seen accounting for two cases in total (5.6%). Sinus tachycardia in 4 cases (11%), sinus bradycardia in 1 cases (2.7%), prominent U waves were seen in 3 cases (8.4%)



Specific ECG changes in different types of stroke

Table A-VI Types of ECG changes in different types of stroke

Sl. No.	Types of ECG Changes	Cerebral thrombosis	Cerebral haemorrhage	Subarachnoid haemorrhage	Cortical venous thrombosis	Total	%
1	S-T segment depression	4	2	-	-	6	16.7
2	T-wave inversion	19		-	1	19	52.8
3	Q-T_c interval prolongation	2	5	-	-	7	19.5
4	Sinus tachycardia (rate>100/min)	4		-	-	4	11.1
5	Sinus bradycardia (rate <60/min)	1	-	-	-	1	2.7
6	U waves	-	2	1	-	3	8.4
7	Arrhythmias (atrial and ventricular)	-	2	-	-	2	5.6

The table A-VI shows the incidence of specific ECG change in different types of stroke.

1) **S-T depression:** S-T depression accounted for 16.7% of the cases **(**total of 6 cases) out of which 2 cases are intracerebral haemorrhage. Remaining four cases of S-T depression was of cerebral thrombosis group. S-T segment, depression was not seen in CVT cases.

2) **T wave inversion:** A total of ninteen cases showed T wave inversion (52.8%). T inversion was noted in fourtien cases of cerebral thrombosis mainly. Five cases of

intracerebral haemorrhage showed T inversion. No T inversion was noted in CVT and SAH.

3) **Q-Tc interval prolongation:** Q-Tc interval prolongation was seen in a total of 7 cases (19.5%). Five cases of intracerebral haemorrhage showed Q-Tc interval prolongation and 2 cases of cerebral thrombosis showed Q-Tc prolongation.
4) **Arrhythmias:** Arrhythmias like Atrial fibillation noted in 2 cases, both were noted in intra cerebral thrombosis cases and both cases expired.
5) **Sinus tachycardia:** Sinus tachycardia was seen in 4 cases, one each in cerebral haemorrhage, CVT and 2 cases of Cerebral thrombosis.
6) **Sinus bradycardia:** This was noted in one case of Cerebral thrombosis . None of the other types of CVA showed this change.
7) **U waves:** Significant U waves were seen in 3 cases. 2 casesof intra cerebral hemorrhage, and in a case of SAH.

ECG changes resembling STEMI were not noted in any of the cases.

a) As subsequent ECGs were taken after 24hrs, and at discharge, ECGs which were normal on the first day did not develop any new changes in any of the cases.
b) ECGs which showed arrhythmias like atrial fibrillation patient expired after 3 days.
c) ECGs which showed repolarisation changes ST-T changes, U waves, Q –Tc prolongation persisted till the time of discharge and fallow up.
d) Sinus tachycardia and sinus bradycardia reverted to normal rate during the fallow up.

12 patients out of 36 patients expired after the CVA event. This was confirmed by record section and over phone. Mortality was taken as the prognostic indicator and fallowing conclusion were drawn;

Total no of patients in the study	**Patients with ECG changes**	**Patients with out ECG changes**	**No of patients who expired in the study group**
36	32	4	12

Table A-VII Correlation of mortality with respect to ECG changes

No of patients who expired and had ECG changes	12	37.5%
No of patients who expired and did not have any ECG changes	0	0%

As quoted in table A-VII it was noted that out of thirtysix patients twelve patients expired and all of them had ECG changes and patient did not have any ECG changes had no mortality. Its also noted that patients with ECG changes had bad prognosis(mortality 37.5%) as compared to patients with out ECG changes.

Table A-VIII Mortality with respect to type of stroke

Type of stroke	**No of patients expired**	**Percentage**
Cerebral hemorrhage	7	70%
Subarachnoid hemorrhage	0	0%
Cerebral thrombosis	5	21.8%
Cortical venous thrombosis	_	
Total	**12**	

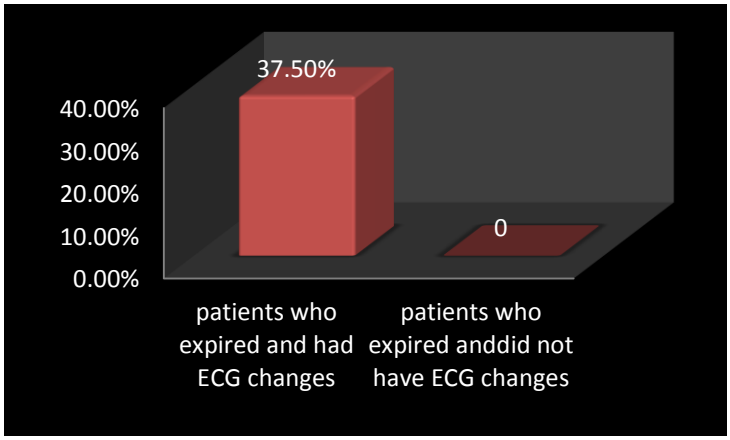
As evident from table A-VIII it was noted that patients with intracerebral haemorrhage had 70% and it was only 21.8% in cerebral thrombosis. where as SAH and CVT had no mortality.

Table A-IX Specific ECG changes in Patients who expired

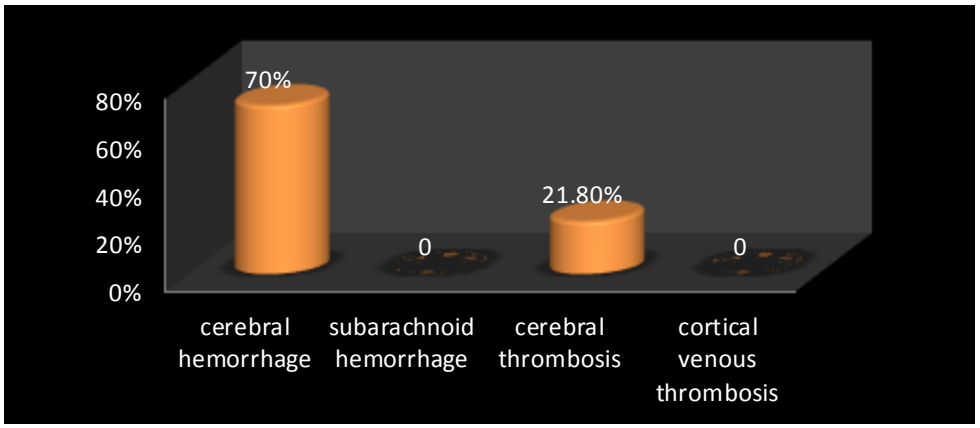
Sl. No.	Types of ECG Changes	Cerebral thrombosis	Cerebral haemorrhage	Subarachnoid haemorrage	Cortical venous thrombosis	Total	%
1	S-T segment depression	2	1	-	-	3	50
2	T-wave inversion	2	3	-	-	5	26.3
3	Q-T_c interval prolongation	1	3	-	-	4	57.14
4	Sinus tachycardia (rate>100/min)	-	-	-	-	-	-
5	Sinus bradycardia (rate <60/min)	1	-	-	-	1	100
6	U waves	-	-	-	-	-	-
7	Arrhythmias (atrial and ventricular)	2	-	-	-	2	100

As evident from the table A-IX which show relation between type of ECG changes and its relation to mortality, it was noted that ATRIAL FIBRILLATION and sinus bradycardia had highest value(100%) for predilection of bad prognosis(mortality), where as Q-Tc prolongation and abnormal U waves had 57.14%, S-T segment depression had 50% and T-wave invertion had least i.e 26.3% predilection for bad prognosis (mortality).Other type of ECG changes did not infer about prognosis. Combined ST-T changes contributed for the highest rate of mortality(76.3%).

GRAPH SHOWIN MORTALITY RATE



GRAPH SHOWING MORTALITY AMONG DIFFERENT TYPES OF STROKE



GRAPH SHOWING MORTALITY RATES IN DIFFERENT TYPES OF ECG CHANGES

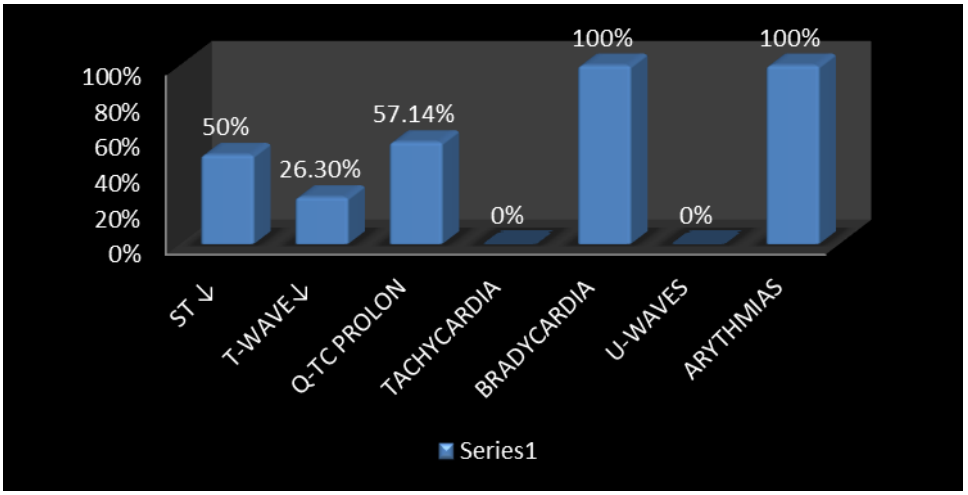


Table A-X HYPERTRIGLYCERIDEMIA AND STROKE

No of patients who had HYPERTRIGLYCERIDEMIA, Triglyceride levels >160mg/dl.	21	58.33%
No of patients who had normal lipid levels.ie triglyceride levels <160mg/dl.	15	41.67%

As quoted in table A-X it was noted that out of thirtysix patients, twenty one patients(58.33%) had hypertriglyceridemia and fifteen patients had normal lipid levels. Out of 21 patients 7 were females and 14 were males.

Table A-XI HYPERTRIGLYCERIDEMIA AND TYPE OF STROKE

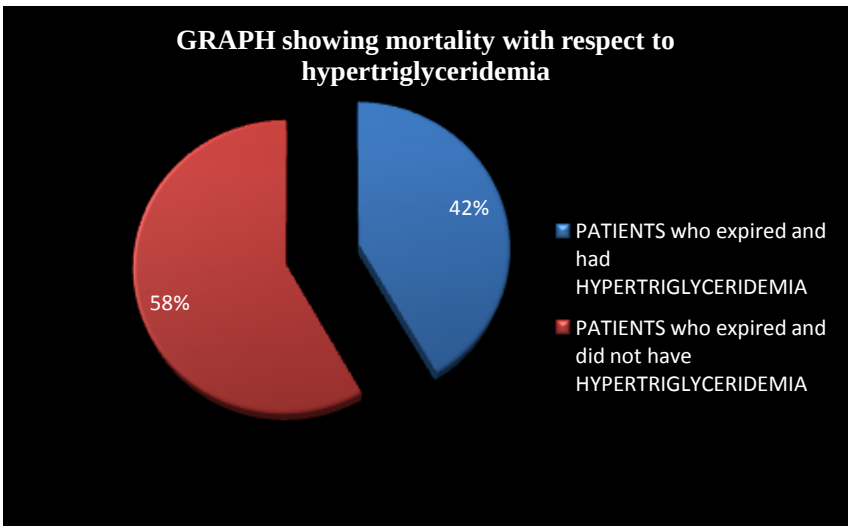
	FEMALE	MALE	TOTAL
cerebral hemorrhage	0	3	3
subarachnoid hemorrhage	0	0	0
cerebral thrombosis	7	11	18
cortical venous thrombosis	0	0	0
	7	14	21

As quoted in table A-XI it was noted that out of 21 patients of hypertryglceridemia, seven females and 11 males had cerebral thrombosis, 3 male patients had cerebral hemorrhage.

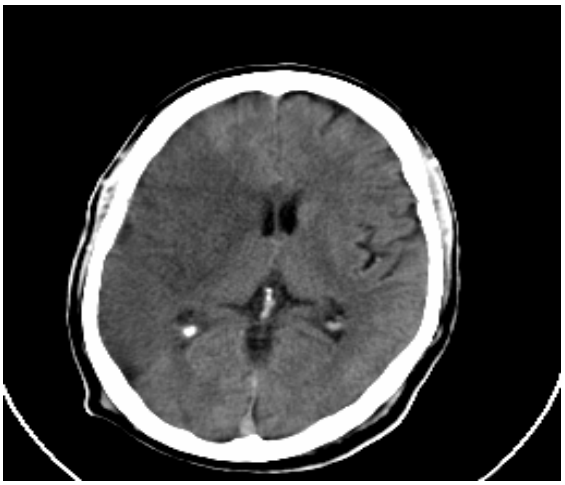
Table A-XII MORTALITY WITH RESPECT TO HYPERTRIGLYCERIDEMIA.

No of patients who expired and had HYPERTRIGLYCERIDEMIA.	5	41.67%
No of patients who expired and did not have HYPERTRIGLYCERICEMIA.	7	58.33%

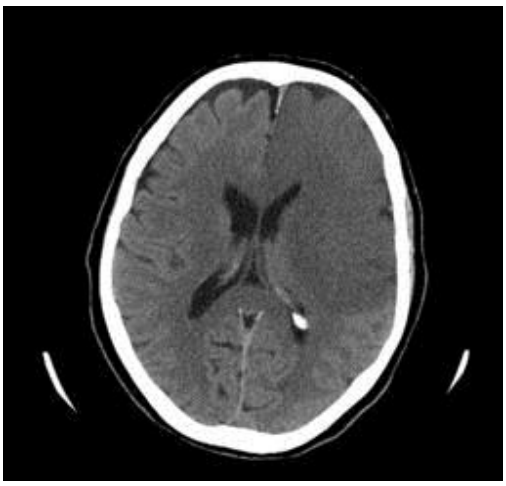
As quoted in table-XII it was noted out of twenty one patients who had hypertriglyceridemia five patients expired and out of five patients three had cerebral hemorrhage and all three were males, two had cerebral thrombosis and both were females. Out of twelve patients expired five patients had hypertriglyceridemia.(41.67%), It is also noted that patients with hypertriglyceridemia and ECG changes had bad prognosis(mortality 41.67%).



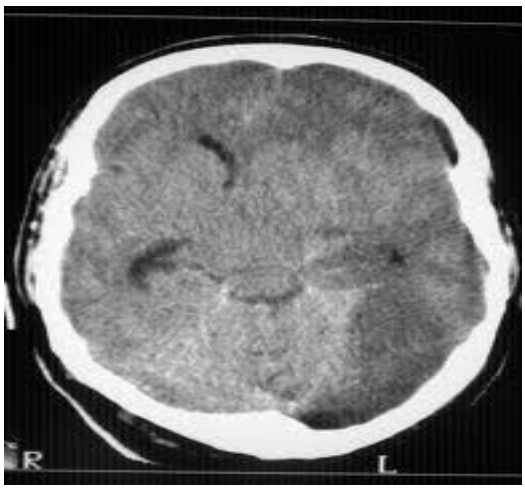
01-CT-SCAN BRAIN OF PATIENT-8A IN MASTER CHART SHOWING RT-MCA INFARCT



02-CT-SCAN BRAIN OF PATIENT-12A, SHOWING LT-MCA INFARCT

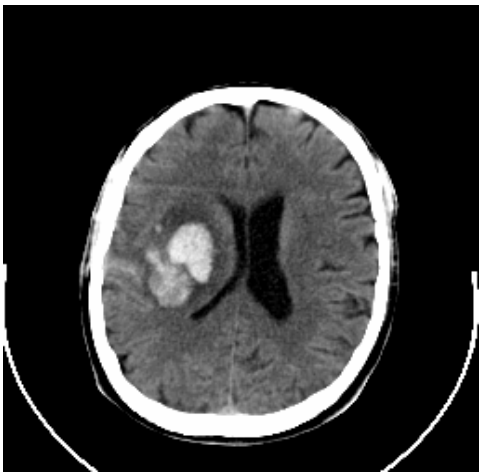


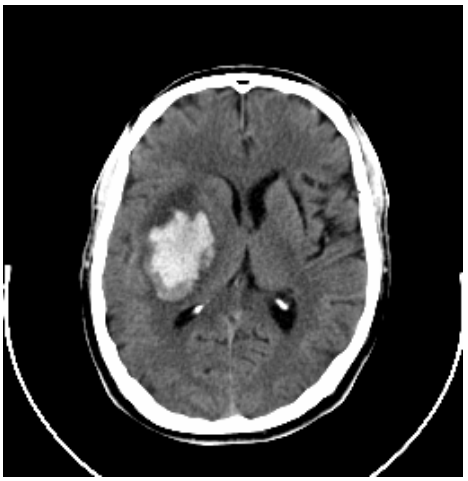
03-CT-SCAN BRAIN OF PATIENT-13A, SHOWING LT-ACA AND LT-PCA INFARCT



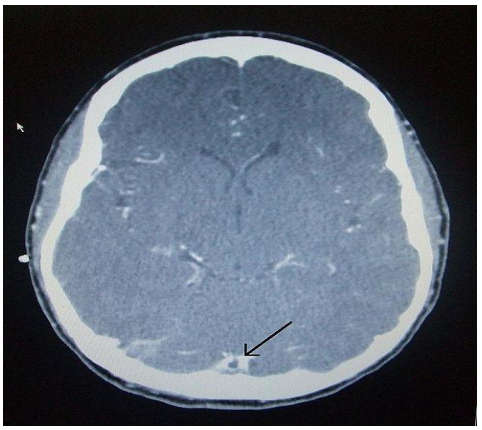
04-CT-SCAN BRAIN OF A PATIENT-11A IN MASTER CHART SHOWING

INTRACEREBRAL RT-CAPSULOGANGLIONIC HAEMORRHAGE

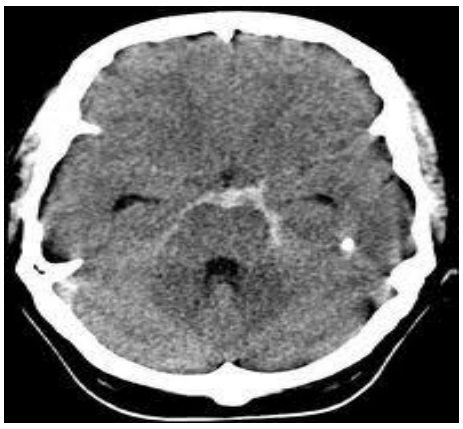




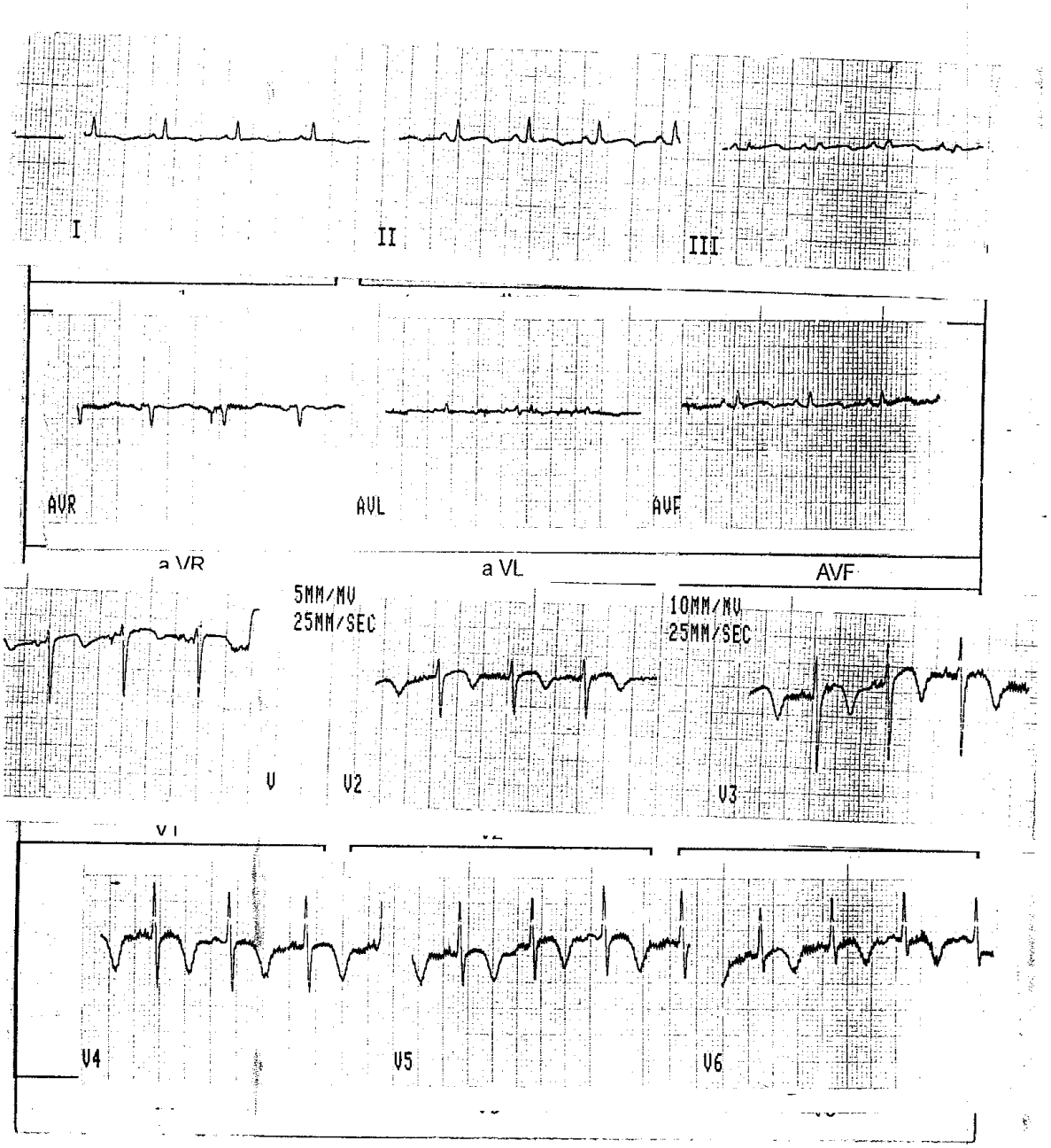
05-CT-SCAN BRAIN OF PATIENT- 31A IN MASTER CHART SHOWING CVT



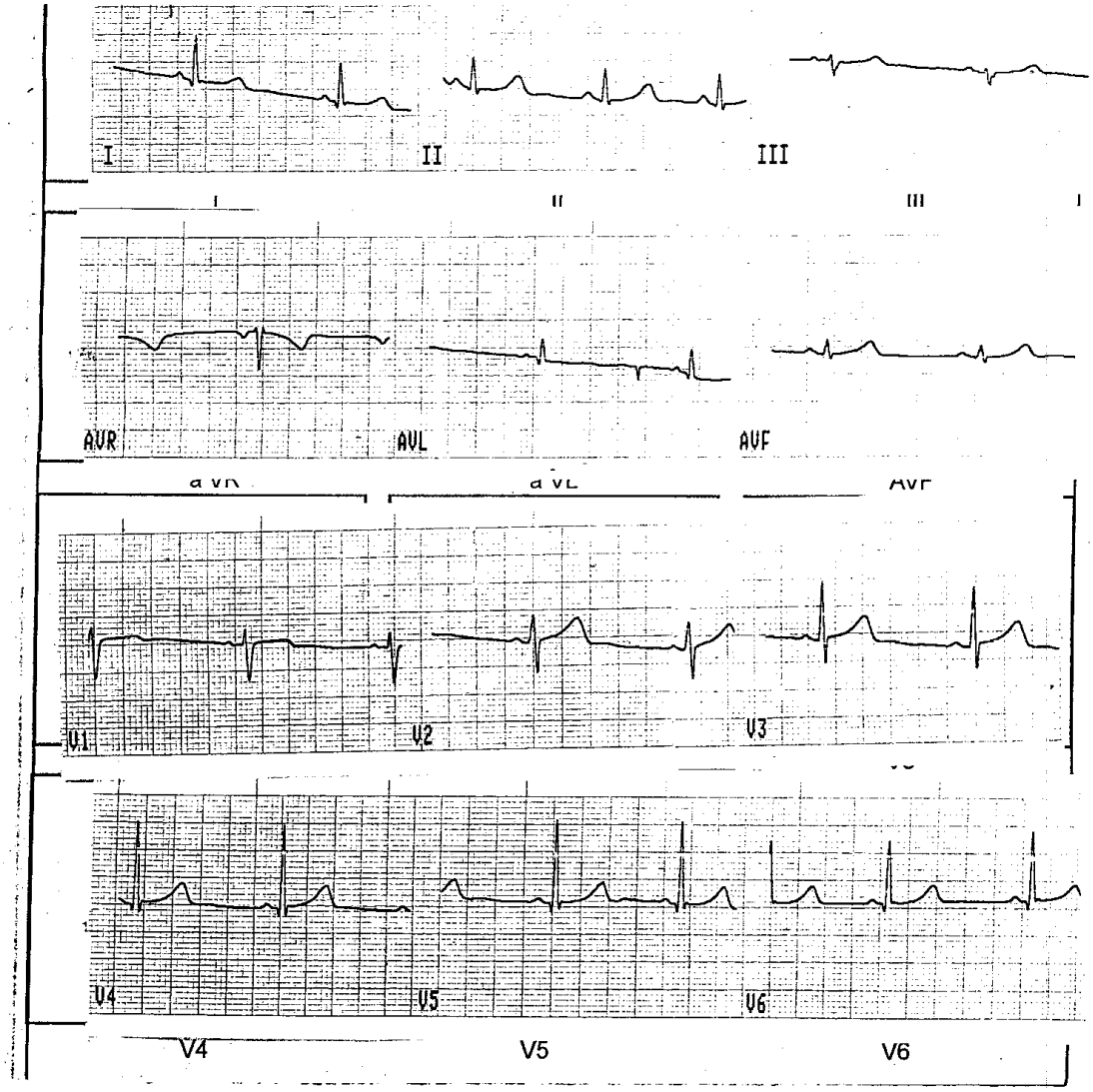
06-CT-SCAN BRAIN PATIENT-27A IN MASTER CHART SHOWING SAH



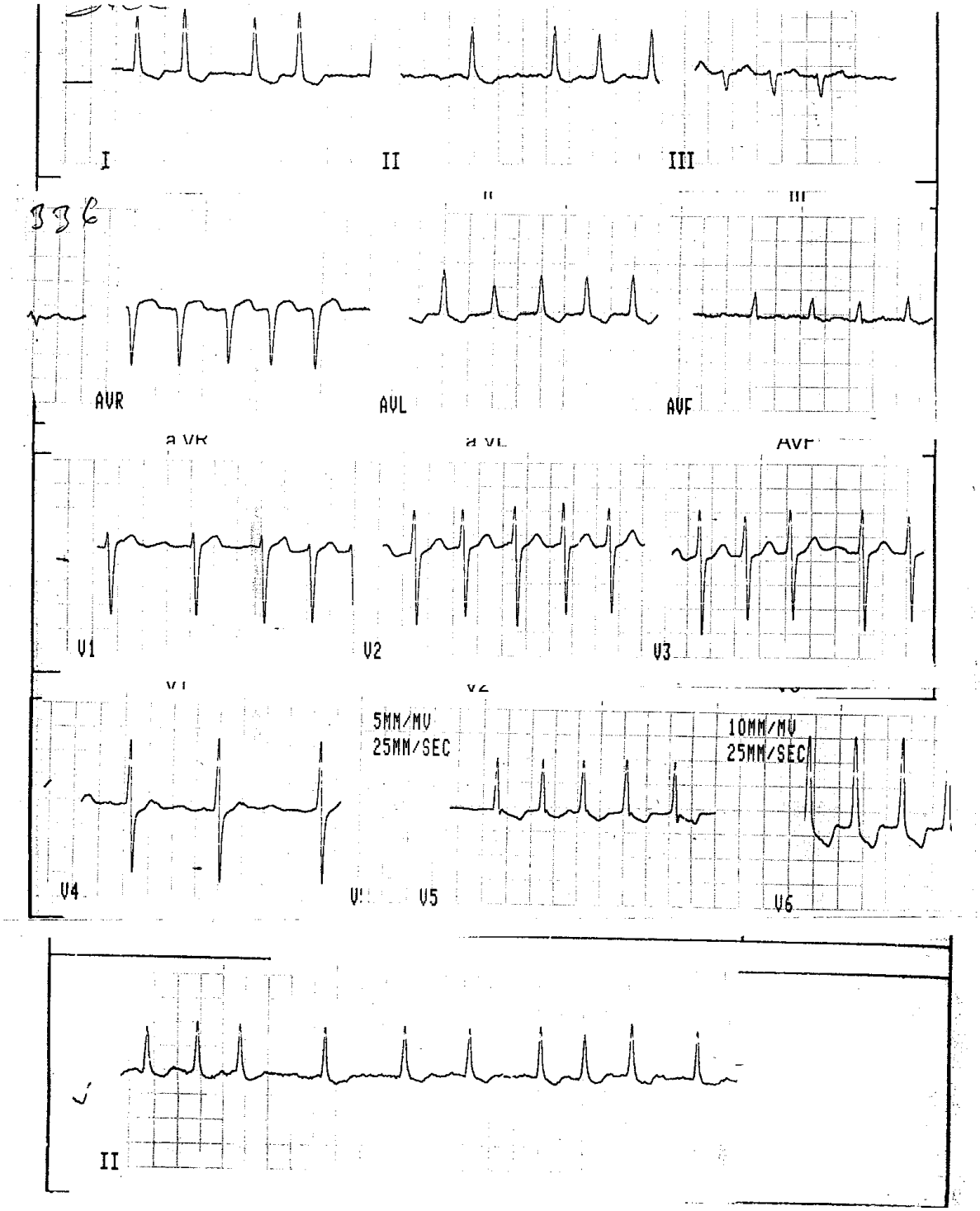
07-ECG OF A PATIENT WITH CVT-31A IN MASTER CHART SHOWIN T-WAVE INVERSION FROM V1-V6



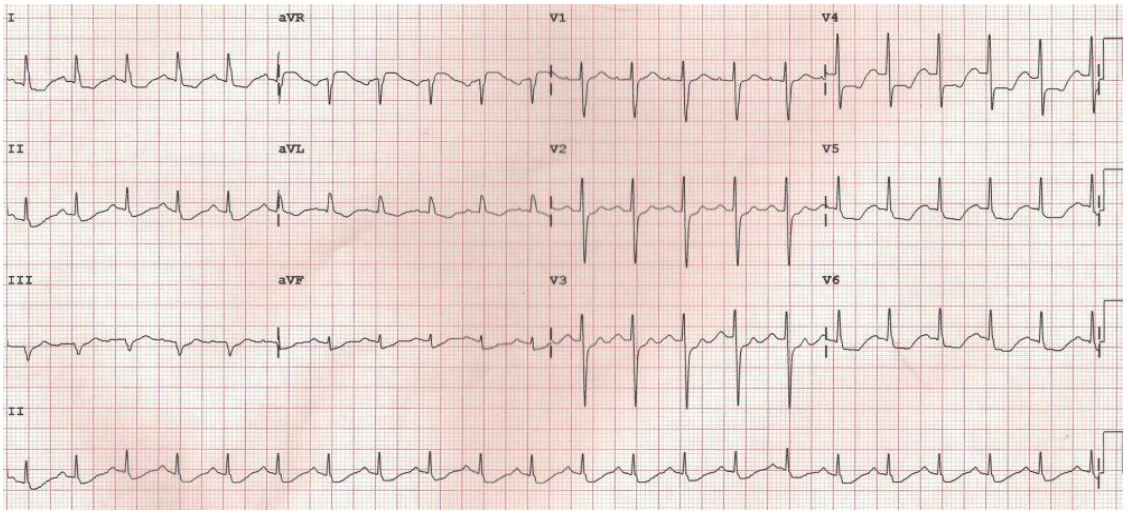
08-ECG OF PATIENT WITH CEREBRAL INFARCT-22A IN MASTER CHART SHOWIN QTc-INTERVAL PROLONGATION



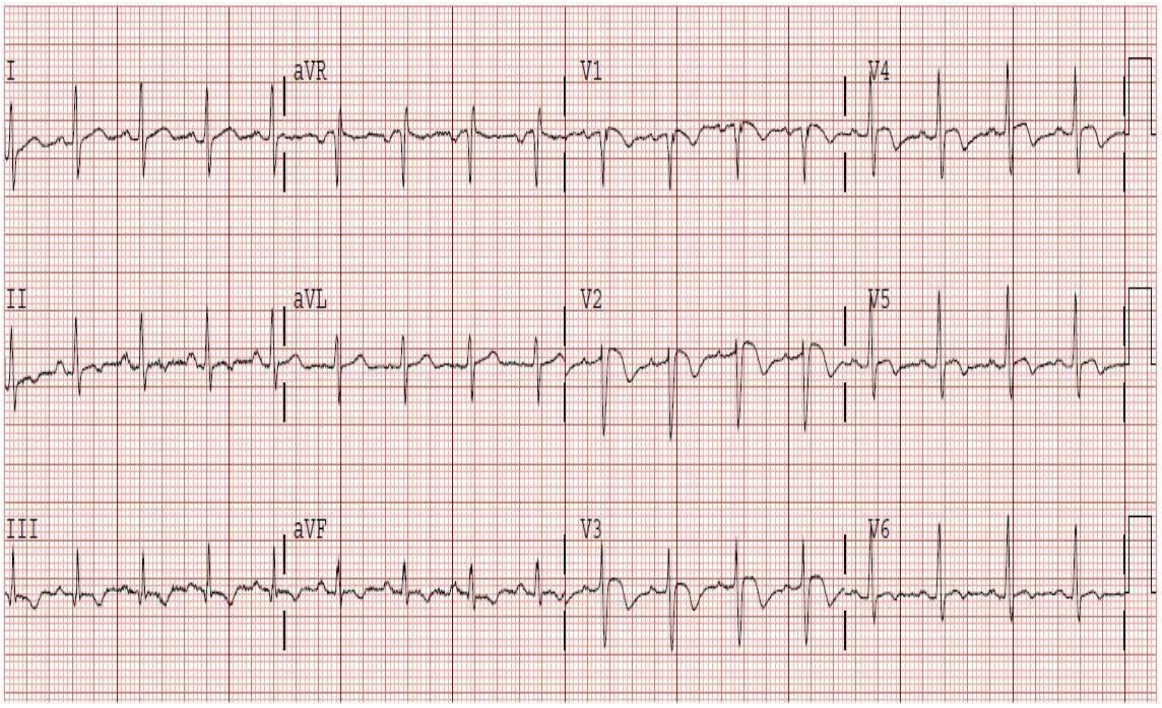
09-ECG OF PATIENT WITH B/L FRONTAL LOBE INFARCTS-23A IN MASTER CHART SHOWIN AF



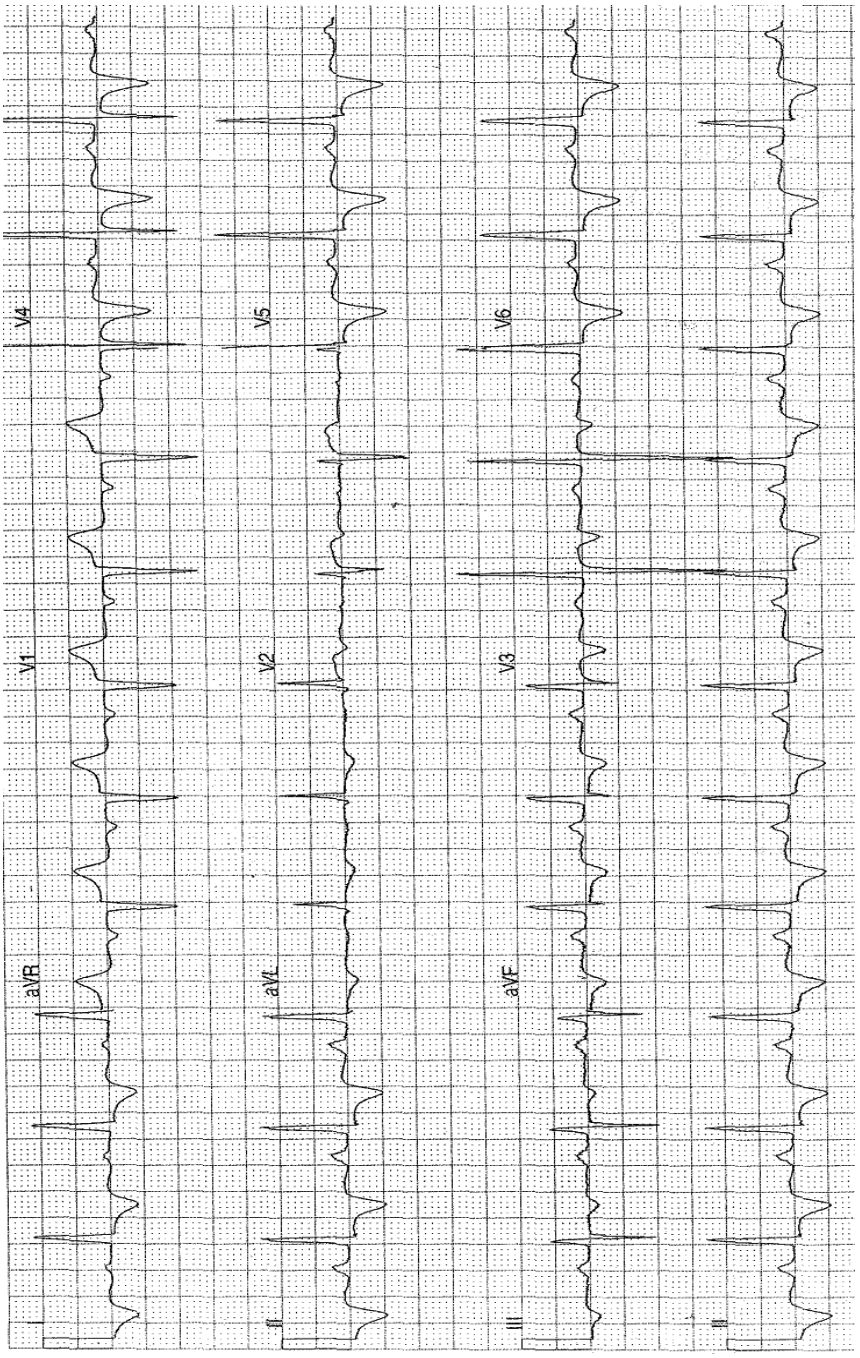
10-ECG OF A PATIENT WITH LT-THALAMIC HAEMORRHAGE-6A IN MASTER CHART SHOWING ST-↓ AND T-↓IN Ⅰ,AVL AND V4-V6



11-ECG OF A PATIENT WITH RT-MCA INFARCT-4A IN MASTER CHART SHOWING T↓ IN Ⅱ,Ⅲ,AVF AND V1-V6



12-ECG OF A PATIENT WITH CEREBELLAR AND PONTINE HEAMORRHAGE-21A IN MASTER CHART SHOWING ST-↓ AND T↓ IN ALL THE LEADS EXCEPT V1 AND V2



DISCUSSION

A close relationship between cerebrovascular accidents and cardiovascular diseases is a well known fact. Cardiovascular diseases may be coexisting or a causative condition for stroke. There are numerous reports demonstrating the fact that primary neurologic abnormalities like cerebrovascular accidents can produce ECG changes affecting S-T segment, T waves, Q-T interval, U waves. A high incidence of cardiac arrhythmias have also been demonstrated in acute cerebrovascular lesions. Some of the ECG changes resemble myocardial infarction.

In clinical practice, these changes may be misinterpreted as changes of myocardial ischaemia or myocardial infarction, thereby affecting the assessment of immediate prognosis and thus delaying the definite surgical procedures being undertaken in some of these stroke patients.

Table B-I shows the sex-wise incidence of stroke in present study as compared to the Framingham study.[87]

Table B-I: Incidence of different types of strokes in males and females

Sl. No.	Types of stroke	Framingham Study		Present study	
		Male	Female	Male	Female
1	Cerebral infarction	44.3%	43.7%	56.52%	76.92%
2	Intracerebral haemorrhage	5.7%	4.5%	39.13%	7.69%
3	Subarachnoid haemorrhage	6%	7.2%	4.34%	_
4	Cerebral embolism	18.5%	22.9%	_	_
5	Others including CVT	2.2%	2.1%	—	15.38%

This table shows that cerebral infarction and intracerebral haemorrhages are more common in males. Both studies show similar observations. In case of cerebral

embolism and subarachnoid haemorrhage females have higher incidence according to Framingham study. In our study, cerebral infarction and CVT are more common in females, intracerebral haemorrhage and subarachnoid haemorrhage cases were more in males. Our study did not have cerebral embolism patients. Incidence of cerebral embolism in Framingham study is 18.5% in males and 22.9% in females. As our study group was small, studies with large number of patients may be required to demonstrate the actual male and female sex incidence of stroke.

As for the age incidence, our study shows that maximum stroke cases were seen between 60-79 years of age. Incidence is less below 40 years of age.

Incidence increases after 40 years. Now because of the strict control of hypertension and other risk factors, incidence of stroke is less in the middle age group i.e. 40-60 years and incidence increases thereafter.

Table B-II shows the comparison of different types of stroke between National Institute for Neurological Diseases and Stroke (NINDS).[90] Stroke data bank statistics and present study.

Table B-II: Incidence of different types of stroke

SI. No.	Type of stroke	NINDS study	Present study
1.	Cerebral infarction	70%	63.8%
2.	Intracerebral haemorrhage	13%	27.8%
3.	Subarachnoid haemorrhage	13%	2.78%
4.	Others (CVT)	3%	5.6%

According to table B-II, incidence of cerebral thrombosis in the present study is less as compared to NINDS data. Incidence of intracerebral haemorrhage is more compared to NINDS data. Incidence of intracerebral haemorrhage is 27.8% as

compared to 13% of the NINDS data. Subarachnoid haemorrhages are less as compared. As evident from studies, haemorrhagic strokes are more common in Indians compared to the Western population. CVT was more common in our study.

In various control studies, a comparison of ECG changes and arrhythmias seen in stroke patients, were made with the ECG finding of age and sex matched controls. The differences in the ECG findings were statistically significant and these ECG changes were attributed to cerebral cause.

In the present study, 32 out of 36 cases showed ECG changes, while in the remaining 4 cases ECG changes were not seen. ECG changes of the present study, as compared to the previous 2 studies done by Mathur K.S [61] and Goldstein D.S[10] are as follows.

In the study conducted by Mathur K. S[61] the ECG changes which were attributable to stroke were 36.4%.

Table B-III: Incidence of ECG changes in present study as compared to Mathur's study

Study	Total number of cases studied	Number with ECG changes	Percentage
Mathur's study	140	51	36.4%
Present study	36	32	88.89%

In Mathur's study, out of 140 cases ECGs were abnormal in 84 cases (60%). The abnormalities in 33 cases (i.e. 23.5%) could be explained on the basis of associated cardiac disease i.e. ischaemic heart disease 13 cases, hypertension 17 cases and rheumatic heart disease 3 cases, while those in the remaining 51 cases (36.4%) were attributed to cerebrovascular accidents. In the present study, ECG changes were

seen in 88.89% of the cases.(our study did not include patients with diabetes, hypertension and ischemic heart disease)

Incidence of ECG changes in different types of stroke in the present study and the study done by Mathur are shown in table B-IV.

Table B-IV - Comparison of incidence of *ECG* changes in various types of strokes between present study and Mathur's study

SI. No.	Types of stroke	Mathur's study	Present study
1	Cerebral haemorrhage	71.1%	100%
2	Subarachnoid haemorrhage	83.3%	100%
3	Cerebral infarction	32.3%	52.78%

The table B-IV shows that study done by Mathur shows an incidence of ECG changes 71.1% in cerebral haemorrhage and 32.3% in cerebral infarction. Present study shows ECG changes in all the cases of intracerebral haemorrhages and subarachnoid haemorrhages studied. 52.78% cases of cerebral thrombosis showed ECG changes in the present study. Mathur's study did not include CVT. In the present study, both cases of CVT showed ECG changes. Although overall incidence of ECG changes in the two studies do not tally, the incidence of changes in different types of stroke closely resemble as shown in table B-V.

Comparison of different types of ECG changes of the present study and study done by Mathur are shown in table B-V.

Table B-V: Different types of ECG changes in present study as compared to Mathur's study

Sl. No.	Types of ECG changes	Mathur's Study	Present Study
1	S-T segment depression	31%	16.7%
2	T wave inversion	49%	52.8%
3	Q-T_c interval prolongation	54.9%	19.5%
4	Ventricular premature complexes	3.92%	-
5	Atrial fibrillation	1.96%	5.6%
6	Sinus tachycardia	3.92%	11.11%
7	Sinus bradycardia	31.3%	2.7%
8	U- waves	--	8.4%

Different types of ECG changes as observed in Goldstein's study are as follows (Table B-VI)

Table B-VI: Comparison of different types of ECG changes between present study and Goldstein's Study

Sl. No.	Types of ECG changes	Goldstein's Study	Present Study
1	S-T segment depression	21%	16.7%
2	Q-T_c interval prolongation	32%	19.5%
3	Sinus tachycardia	28%	11.11%
4	Atrial fibrillation	31%	5.6%
5	VPC's	8%	-
6	U- waves	13%	8.4%

This table (Table B-VI) shows Q-Tc prolongation as the most common ECG change (32%), followed by S-T depression (21%).In our study S-T depression were of

16.7% and Q-Tc prolongation accounted for19.5%.Arrhythmias were seen frequently in Goldstein's study. He demonstrated an incidence of 31% of atrial fibrillation and 8% of VPCs. Our study showed only 5.6% of atrial fibrillation.

S-T depression and T wave abnormalities

S-T depression and T wave inversion were among the commonest changes observed in stroke. In the present study S-T depression was seen in 6 cases and T wave inversion in 19 cases. S-T depression was seen in 4 cases of cerebral thrombosis and 2 cases of intracerebral haemorrhage. T- wave inversion was seen in 5 case of cerebral haemorrhage, 12 cases of cerebral thrombosis and 2 cases of CVT. Thus ST-T changes were seen in all types of stroke except for SAH in the present study.

Table B-VII shows the percentage incidence of ST-T changes observed in the present study as compared to Mathur's and Goldstein's study (Table B-VII)

Table B-VII: Percentage of ST-T changes

Study	Percentage
Mathur's study	49%
Goldstein's study	35%
Present study	69.4%

Thus all the 3 studies, show an approximately equal incidence of ST-T changes. ST-T changes were seen more frequently in ischaemic strokes than haemorrhagic in the present study. These ST-T changes which appeared during stroke perisisted till the end of follow up period. Increased sympathetic activity has been proposed as the cause for these ST-T changes

Q-Tc prolongation: In the present study Q-Tc prolongation was also the common ECG changes observed. Out of 32 cases with ECG changes, 7 cases showed Q-Tc prolongation. 5 cases were intracerebral haemorrhage and 2 were cerebral thrombosis. Abnormal serum electrolytes and other causes of Q-Tc prolongation have been ruled out.

Sinus tachycardia: In our study, sinus tachycardia was seen in 11.11% of cases as compared to 31.3% in Mathur's and 28% in Goldstein's study. In the present study, all the cases of sinus tachycardia became normal during the follow up. Sympathetic overactivity has been postulated as a cause of these sinus tachycardia.

Sinus bradycardia: In our study 1 case of sinus bradycardia were seen (2.7%). Mathur's study showed 3.92% incidence, Goldstein's study did not show any cases with bradycardia.

U-*waves:* In the present study, 8.4% showed prominent U waves. In Mathur's study, U waves were not seen in any of the cases. In Goldste.n's study, an incidence of 13% of U waves were observed. These U waves also persisted till the end of follow up period.

Arrhythmias: Atrial fibrillation was seen in 5.6%cases in our study, whereas Goldstein's study showed an incidence of 31%, Mathur's study did not show a significant incidence of atrial fibrillation. A study in 1981, showed a 50% of incidence of arrhythmias in the form of VPCs and atrial fibrillation. Other similer studies in 1975 and 1976 proved that propronolol is beneficial in treating these

neurogenic cardiac arrhythmias.[91,76] Arrhythmias are likely to contribute to the sudden unexpected mortality seen following stroke, as well as to the stroke extension due to arrhythmia induced hypotension. In 1992 a study suggested that patients with the acute stroke should receive continuous cardiac monitoring for 3days. In our study both the cases with arrhythmia expired after 3days.[26]

ECG changes and type of stroke:

In the present study, few ECG changes characterized particular type of stroke.

Cerebral haemorrhage: Intracerebral haemorrhages showed all types of changes, i.e. ST-T changes, Q-Tc prolongation, arrhythmias, sinus tachycardia, exept for sinus bradycardia and abnormal U-waves, commonest ECG change was Q-Tc prolongation Other changes frequently seen were ST-T changes. Mortality was highest among patients with cerebral haemorrhage, was more in patients showing ST-segment depression fallowed by Q-Tc prolongation and T wave invertion.

Subarachnoid haemorrhage: One case of subarachnoid haemorrhage showed abnormal U wave.

Cerebral thrombosis: T-wave invertions and S-T depression were frequently seen with cerebral thrombosis. One case of cerebral thrombosis showed Sinus tachycardia. Where as Q-Tc prolongation ,sinus bradycardia and U waves were not seen. One patient who showed T-wave invertion had bad prognosis.

Cortical venous thrombosis: Both cases of CVT had ECG changes in the form of sinus T wave inversion.

In our study all the ECG changes other than arrhythmias and sinus bradycardia, sinus tachycardia persisted during the follow up period of 30 days. In a study conducted ECG changes persisted for a period up to 6-10 weeks.Similarly in another study conducted in 1986, the ECG changes persisted upto 6 weeks after the stroke.[75] So further follow up study may be required to demonstrate these findings.

In an attempt done to find the prognostic importance of ECG changes in stroke patients our study found that patients who had persistent ECG changes had bad prognosis compared to patients with normal ECG.

Table B-VIII shows comparison between present study and a study done by Bozluoclay et al in 2003.[4] and Dogan et al in 2004.[30]

Dogan's study		**Bozluolclay's study**		**Present study**	
Patients with ECG changes	**Patients mortality**	**Patients with ECG changes**	**Patients mortality**	**Patients with ECG changes**	**Patients mortality**
79%	27%	62.1%	38.9%	88.89%	33.34%

According to table B-VIII mortality among patients with ECG changes is almost similar in all three studies.

As compared to study by Dogan in 2004 ST-segment changes,abnormal U – waves were univariate predictors of mortality.[30]

In our study the types of ECG changes which predicted mortality were ST-T changes, prolonged Q-Tc and arrythmias.

CONCLUSION

Following conclusions are drawn after completing this study.

1) Strokes occur more frequently after the age of 40 years .
2) Hemorrhagic and thrombotic strokes are more common in males Compared to females and CVT was exclusive to females.
3) ECG abnormalities were seen in 88.89% of the patients. 11.11% did not show any abnormalities.
4) Haemorrhagic strokes showed higher incidence of ECG abnormalities as compared to non-haemorrhagic strokes.
5) ST-T changes and Q-Tc prolongation were the frequently seen abnormalities. Arrhythmias were seen in 5.6% of cases, Sinus tachycardia in 11.11% and sinus bradycardia in 2.7% of the cases. U waves were seen in 8.4% of the cases. S-T depression was seen in 16.7% and T wave inversion was seen 52.8% cases.
6) Some characteristic relationship was noted between type of ECG change and the type of stroke. Cerebral thrombosis showed all types of ECG changes i.e. ST-T changes, Q-T prolongation, arrhythmias, sinus tachycaidia, sinus bradycardia except for U waves. Subarachnoid haemorrhage showed abnormal U- wave . In intracerebral heamorrhage Q-Tc interval prolongation ,T-wave invertion, ST-Tchanges was frequently seen. other ECG changes like sinus tachycardia and S-T depression were also seen .
7) As this study was done on a small number of patients, no conclusion could be drawn between the location of stroke and ECG changes
8) It was found that patients who had ECG changes had bad prognosis(mortality) compared to patients with out ECG changes .

9) Among the patients died large number (6) was contributed by patients with intra cerebral hemorrhage compared to thrombotic stroke(1).

10) Most common ECG changes among patients expired were ST-T changes and Q-Tc prolongation.

11) Hence it is important to monitor cardiac activity in all patients with stroke.

12) It was found that of the 36 patients, twenty one patients(58.33%) had hypertriglyceridemia and fifteen patients had normal lipid levels. Out of 21 patients 7 were females and 14 were males. It was noted that out of 21 patients of hypertryglceridemia, 7 females and 11 males had cerebral thrombosis, 3 male patients had cerebral hemorrhage.

13) Out of twelve patients expired five patients had hypertriglyceridemia.(41.67%), It is also noted that patients with hypertriglyceridemia and ECG changes had bad prognosis(mortality 41.67%).

14) Hence it is also important to check for lipid levels in all patients of stroke.

SUMMARY

This study confirms the fact that cerebrovascular accidents frequently produce ECG changes affecting S-T segments, T waves, Q-T interval, U waves and arrhythmias (both atrial and ventricular). Thus this study confirms the role of ECGs as a part of routine investigations of patients admitted with stroke. Knowledge of these ECG changes will prevent unnecessary delay in operative treatment in some of these patients, which would otherwise be postponed thinking that these ECG changes are due to myocardial ischaemia.

All patients with stroke should be observed for arrhythmias, as they occur frequently with cerebrovascular diseases. They may contribute to the sudden unexpected death of such patients. Hence appearance of arrhythmias following stroke has prognostic significance. Such patients have to be monitored till the arrhythmias disappear and they may be treated with antiarrhythmic drugs like beta-blockers (propranolol).

Routine prophylaxis for cardiac arrhythmias is not advocated for cerbral infarction or intracerebral haemorrhage at this time. This situation may change in the near future, once investigations have revealed which ECG features, stroke locations, and shifts in cardiac autonomic tone are strongly predictive of the occurrence of malignant cardiac arrhythmias. In the interim, ventricular tachyarrhythmias or supraventricular arrhythmias which compromise cardiac output should be treated with standard antiarrhythmic therapy once they occur.

Patients whose Q-Tc interval is prolonged by such treatment or by anticonvulsants should be carefully monitored. Patients with sinus tachycardia and a normal or elevated blood pressure after subarachnoid haemorrhage should be treated with prophylactic beta-blockade, as their cardiovascular sympathetic tone is probably

elevated and the incidence of malignant ventricular arrhythmias is high in this condition.

Present study was conducted on 36 patients. Cases with known cardiovascular diseases, metabolic, renal, and hepatic disorders were not included in the study. This study showed an incidence of 88.89% ECG changes which could be attributable to stroke. This study approximately coincides with some of the similar previous study reports, except that the incidence of arrhythmias in the present study is much less. This may be because we have not done Holter monitoring for any of the cases, so some of the paroxysmal arrhythmias may have been missed.

One other important thing which we noticed in our study is that 58.33% of patients had hypertriglyceridemia and it was noted that among patients with hypertriglyceridemia and ECG changes 41.67% mortality was seen which indicated a bad prognosis.

Other observation is that some of the ECG changes that appeared during stroke persisted till discharge and follow up. So further work up may be needed like coronary angiography, post mortem studies in cases of death, to prove that these changes are not due to myocardial ischaemia.

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PROFORMA

PRELIMINARY DATA OF THE PATIENT:

Name	:	DO. A.	:
Age	:	D.O.D.	:
Address	:	0. P.No.	:
		Unit	:
		Hospital: R L J H Rc	

PRESENTING COMPLAINTS:

			Right	Left
Weakness	:	UL		
		LL		
		Face		
Onset	:	Sudden / Gradual / Stepwise progression		
		During activity / at rest / during sleep.		

Prodromal Symptoms

Headache / Giddiness / Vertigo / Vomiting.

Yes / No

Seizures:

Local / Generalized

Frequency / Parts involved.

State of Consciousness:

Duration of altered state

Onset : Sudden / Gradual

Deteriorating / Recovering

Speech:

INTELLIGENCE AND MEMORY DISTURBANCES

BOWEL AND BLADDER SYMPTOMS: TYPE OF DYSFUNCTION

HISTORY OF ANY CRANIAL NERVE INVOLVEMENT

HISTORY OF SENSORY DISTURBANCES

RECOVERY : Rapid / Gradual

HISTORY OF HEAD INJURY : Yes/No

HISTORY OF : Hypertension / Diabetes

HISTORY OF : Palpitation / Chest Pain

HISTORY OF : Dyspnea on Exertion / Orthopnea / P.N.D.

HISTORY OF FEVER :

HISTORY OF RECENT EVIDENCE OF ANY THROMBOEMBOLISM:

HISTORY OF BLEEDING DIATHESIS:

PAST HISTORY

- TIA
- Seizures
- Headache
- Migraine
- Trauma to Head
- Rheumatic fever
- Syphilis

FAMILY HISTORY

- Diabetes mellitus
- Hypertension
- Stroke
- Ischaemic heart disease
- Sudden death
- Bleeding disorders.

PERSONAL HISTORY:

- Diet : Veg. / Non Veg. / Mixed.
- Appetite :
- Weight :
- Bowel & Bladder Habits :
- Habits : Smoking / Alcohol / Tobacco.

MENSTRUAL HISTORY:

TREATMENT HISTORY:

GENERAL PHYSICAL EXAMINATION:

- Built and Nourishment:
- Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy.
- Pulse:
- B.P. :
- Temperature :
- Respiratory Rate:
- Signs of dehydration:

CNS EXAMIANTION

HIGHER MENTAL FUNCTIONS

- Level of consciousness.
- Orientation to time, place, person.
- Memory
- Intelligence
- Speech
- Delusions / Hallucinations.

CRANIAL NERVE EXAMINATION

1. Olfactory - Sense of smell
2. Optic nerve - Acuity of vision
   - Field of vision
   - Colour vision
   - Fundus examination.
3. Occulomotor, Trochlear and Abducent nerves:
   - Ocular movements

- Pupillary size and reaction to light and accomodation
- Ptosis

4. Trigeminal nerve :

- Sensory :

- Motor :
- Jaw Jerk :

FACIAL NERVE

- Motor :
- Taste in the anterior 2/3rd of tongue :

VESTIBULOCOCHLEAR NERVE

- Acuity of hearing
- Rinnes test
- Weber test
- Caloric test

GLOSSOPHARYNGEAL & VAGUS NERVE

- Position of vuvla
- Palatal movement
- Pharyngeal reflex (gag reflex)

- **ACCESSORY NERVE**
- Sternocleidomastoid
- Trapezius

HYPOGLASSAL NERVE

- Position of tongue
- Wasting / Fasciculation.

MOTOR SYSTEM	Right	Left
Bulk & Nutrition	UL	UL
	LL	LL
Tone	UL	UL
	LL	LL

Power	Right	Left
- Shoulder		
- Elbow		
- Wrist		
- Hand grip		

- Hip
- Knee
- Ankle
- Toes

DEEP TENDON REFLEX:	Right	Left
- Biceps jerk		
- Triceps jerk		
- Supinator jerk		
- Knee jerk		
- Ankle jerk		

SUPERFECIAL REFLEXES:	Right	Left
- Corneal		
- Conjunctival		
- Abdominal		
- Cremasteric		

SENSORY SYSTEM:	Right	Left
- Touch : Fine / Deep		
- Pain		
- Temperature: Hot / Cold		
- Position sense		
- Vibration sense		
- Cortical sensation	Steriognosis	
	Tactile localization	
	Sensory inattention	

CEREBELLAR SYSTEM:

- Finger nose test, Knee Heal test
- Rapid alternating movements
- Rebound phenomenon.

Gait:

INVOLUNTARTY MOVEMENTS:

SIGNS OF MENINGEAL IRRITATION:

SKULL & SPINE :

CARDIOVASCULAR SYSTEM EXAMINATION:

Inspection:

Palpation:

Percussion:

Auscultation:

RESPIRATORY SYSTEM:

ABDOMINAL EXAMINATION:

INVESTIGATIONS:

- Routine : Hb%, TC, DC, ESR.
- Urine : Albumin Sugar Microscopy
- F.B.S :
- Urea :
- Creatinine :
- Cholesterol :
- Serum Sodium :
- Serum Potassium :
- Serum Calcium :

CT SCAN BRAIN:

ECG	At admission	At 24hrs	At discharge
Rate			
Rhythm			
P Wave			
P-R interval			
QRS complex			
ST Segment			
T Wave			
U Wave			
Q-Tc interval			
Arrhythmias			

MASTER CHART

Sl number	NAME	FILE NUMBER	AGE	SEX	FINAL DIAGNOSIS	BLOOD INVESTIGATIONS	LIPID PROFILE, TRIG-(mg/dl) N-<165mg/dl.	HEART RATE	RHYTHM	AXIS	P-WAVE CONFIGARATION	AMPLITUDE	DURATION	PR-INTERVAL	CONFIGURATION	QRS-DURATION	CONFIGARATION	ST-SEGMENT	T-WAVE	U-WAVE	QTC-INTERVAL	REMARKS
1-A	RAMAKKA	522922	75 YR	F	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-200	40	N	60	N	0.01	0.08	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.36	SINUS BRADYCARDIA with T-WAVE↓V1-V6
1-B								58	N	60	N	0.01	0.08	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.36	SINUS BRADYCARDIA with T-WAVE↓V1-V6
1-C								64	N	60	N	0.01	0.08	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.36	SINUS BRADYCARDIA with T-WAVE↓V1-V6. Pt expired after 1 month.
2-A	BASHA KHAN	523035	70 YR	M	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-185	80	N	90	N	0.01	0.08	0.12	N	0.06	N	ST↓ Ⅰ, AVL, V1-V6	T-WAVE↓V1-V6	N	0.34	ST↓ Ⅰ ,AVL, V1-V6 and T-WAVE↓V1-V6
2-B								84	N	90	N	0.01	0.08	0.12	N	0.06	N	ST↓ Ⅰ, AVL, V1-V6	T-WAVE↓V1-V6	N	0.34	ST↓ Ⅰ ,AVL, V1-V6 and T-WAVE↓V1-V6
2-C								82	N	90	N	0.01	0.08	0.12	N	0.06	N	ST↓ Ⅰ, AVL, V1-V6	T-WAVE↓V1-V6	N	0.34	ST↓ Ⅰ ,AVL, V1-V6 and T-WAVE↓V1-V6 and
3-A	DODDAAKKAMMA	535542	90 YR	F	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-1000	128	N	30	N	0.01	0.04	0.06	N	0.08	N	N	T-WAVE↓ALL THE LEADS	N	0.30	T-WAVE↓ IN ALL THE LEADS
3-B								98	N	30	N	0.01	0.04	0.06	N	0.08	N	N	T-WAVE↓ALL THE LEADS	N	0.32	T-WAVE↓ALL THE LEADS
3-C								84	N	30	N	0.01	0.04	0.06	N	0.08	N	N	N	N	0.34	N

4-A	THIMMARAY APPA	561035	55 YR	M	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-190	80	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ Ⅱ Ⅲ,AVF and V1-V6	N	0.36	T-WAVE↓ Ⅱ Ⅲ ,AVF and V1-V6
4-B								82	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ Ⅱ Ⅲ,AVF and V1-V6	N	0.36	T-WAVE↓ Ⅱ Ⅲ ,AVF and V1-V6
4-C								80	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ Ⅱ Ⅲ,AVF and V1-V6	N	0.36	T-WAVE↓ Ⅱ Ⅲ ,AVF and V1-V6
5-A	ABDUL KHUDUS	485128	70 YR	M	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-188	120	N	30	N	0.01	0.06	0.06	N	0.06	N	ST-↓V1-V6.	T-WAVE↓ IN ALL THE LEADS	N	0.34	SINUS TACHYCARDIA WITH ST-↓V1-V6 and T-WAVE↓IN ALL THE LEADS
5-B								98	N	30	N	0.01	0.06	0.06	N	0.06	N	ST-↓V1-V6.	T-WAVE↓ IN ALL THE LEADS	N	0.34	ST-↓V1-V6 and T-WAVE↓IN ALL THE LEADS
5-C								90	N	30	N	0.01	0.06	0.08	N	0.06	N	N	T-WAVE↓ IN ALL THE LEADS	N	0.36	T-WAVE↓ IN ALL THE LEADS
6-A	KANNAPPA	570886	65 YR	M	LT-THALAMIC BLEED	N	HYPERTRIGLYCERIDEMIA, TRIG-190	120	N	60	N	0.01	0.06	0.08	N	0.06	N	ST-↓ Ⅰ ,AVL	T-WAVE↓ Ⅰ , AVL and V4-V6	N	0.34	SINUS TACHYCARDIA, ST-↓ Ⅰ ,AVL WITH T-WAVE↓ Ⅰ ,AVL and V4-V6
6-B								118	N	60	N	0.01	0.06	0.08	N	0.06	N	ST-↓ Ⅰ ,AVL	T-WAVE↓ Ⅰ , AVL and V4-V6	N	0.34	SINUS TACHYCARDIA, ST-↓ Ⅰ ,AVL WITH T-WAVE↓ Ⅰ ,AVL and V4-V6

6-C								120	N	60	N	0.01	0.06	0.08	N	0.06	N	ST-↓ Ⅰ,AVL	T-WAVE↓ Ⅰ, AVL and V1-V6	N	0.34	SINUS TACHYCARDIA, ST-↓ Ⅰ,AVL WITH T-WAVE↓ Ⅰ,AVL and V4-V6,Pt expired on 3rd day
7-A	MUNIYAMMA	572730	70 YR	F	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-180	80	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4
7-B								78	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4
7-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4
8-A	THIMAKKA	551966	96 YR	F	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-340	92	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓Ⅱ, Ⅲ,AVF	T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6	N	0.36	ST-↓Ⅱ,Ⅲ,AVF AND T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6
8-B								88	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓Ⅱ, Ⅲ,AVF	T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6	N	0.36	ST-↓Ⅱ,Ⅲ,AVF AND T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6
8-C								92	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓Ⅱ, Ⅲ,AVF	T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6	N	0.36	ST-↓Ⅱ,Ⅲ,AVF AND T-WAVE↓Ⅱ, Ⅲ,AVF AND V1-V6, PATIENT EXPIRED AFTER 15DAYS.

9-A	RAVI.G	495592	30 YR	M	B/LPARIETAL LOBE INFARCT WITH BASILAR ARTERY THROMBOSIS	N	N	90	AF	90						0.06	N	N	N	N	0.38	ATRIAL FIBRILLATION
9-B								92	AF	90						0.06	N	N	N	N	0.38	ATRIAL FIBRILLATION
9-C								94	AF	90						0.06	N	N	N	N	0.38	ATRIAL FIBRILLATION, PT EXPIRED AFTER 9 DAYS
10-A	VENKATASWAMY	478106	65 YR	M	LT-INTERNAL CAPSULE BLEED	N	N	82	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	U-WAVE IN V1-V4	0.36	U-WAVE IN V1-V4
10-B								80	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	U-WAVE IN V1-V4	0.36	U-WAVE IN V1-V4
10-C								78	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	U-WAVE IN V1-V4	0.36	U-WAVE IN V1-V4
11-A	MUNIVENKATAPPA	485542	65 YR	M	RT-CAPSULOGANGLIONIC HEMATOMA	N	HYPERTRIGLYCERIDEMIA, TRIG-190	72	N	60	N	0.01	0.06	0.18	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.42	PROLONGED QT-INTERVAL WITH T-WAVE↓ Ⅰ,AVL AND V1-V6
11-B								70	N	60	N	0.01	0.06	0.18	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.42	PROLONGED QT-INTERVAL WITH T-WAVE↓ Ⅰ,AVL AND V1-V6

11-C								72	N	60	N	0.01	0.06	0.18	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.42	PROLONGED QT-INTERVAL WITH T-WAVE↓ Ⅰ,AVL AND V1-V6, PATIENT EXPIRED AFTER 20 DAYS.
12-A	GIRIJAMBHA	501608	63 YR	F	LT-MCA INFARCT	N	N	80	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	N	0.36	N
12-B								84	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	N	0.36	N
12-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
13-A	NAREMMA	508565	75 YR	F	LT-ACA AND LT-PCA INFARCT	N	N	92	N	60	N	0.01	0.06	0.14	N	0.08	N	N	T-WAVE↓1-V6	N	0.48	QT-INTERVAL PROLONGED WITH T-WAVE↓V1-V6
13-B								90	N	60	N	0.01	0.06	0.14	N	0.08	N	N	T-WAVE↓1-V6	N	0.48	QT-INTERVAL PROLONGED WITH T-WAVE↓V1-V6
13-C								90	N	60	N	0.01	0.06	0.14	N	0.08	N	N	T-WAVE↓1-V6	N	0.48	QT-INTERVAL PROLONGED WITH T-WAVE↓V1-V6, PATIENT EXPIRED AFTER 15DAYS.
14-A	NARAYANAPPA	523252	50 YR	M	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-300	110	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.36	SINUS TACHYCARDIA WITH T-WAVE↓ Ⅰ,AVL AND V1-V6

14-B								90	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.36	T-WAVE↓ Ⅰ,AVL AND V1-V6
14-C								92	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.36	T-WAVE↓ Ⅰ,AVL AND V1-V6
15-A	BUDAN SAB	519539	70 YR	M	LT- PCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-185	92	N	90	N	0.01	0.06	0.12	N	0.08	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.38	T-WAVE↓ Ⅰ,AVL AND V1-V6
15-B								90	N	90	N	0.01	0.06	0.12	N	0.08	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.38	T-WAVE↓ Ⅰ,AVL AND V1-V6
15-C								88	N	90	N	0.01	0.06	0.12	N	0.08	N	N	T-WAVE↓ Ⅰ, AVL AND V1-V6	N	0.38	T-WAVE↓ Ⅰ,AVL AND V1-V6
16-A	PAPAMMA	494834	38 YR	F	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-255	72	N	60	N	0.01	0.06	0.14	N	0.08	N	N	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6	N	0.36	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6
16-B								76	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6	N	0.36	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6
16-C								74	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6	N	0.36	T-WAVE↓ Ⅱ, Ⅲ,AVF AND V4-V6
17-A	RATHNAMMA	436224	45 YR	F	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-189	82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
17-B								80	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
17-C								84	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
18-A	BHASKAR	440621	43 YR	M	LT-EXTERNAL CAPSULE BLEED	N	N	92	N	90	N	0.01	0.06	0.14	N	0.06	N	ST-↓V1-V6	T-WAVE↓V1-V6	N	0.36	ST-↓V1-V6 AND T-WAVE↓V1-V6

18-B								90	N	90	N	0.01	0.06	0.14	N	0.06	N	ST-↓V1-V6	T-WAVE↓V1-V6	N	0.36	ST-↓V1-V6 AND T-WAVE↓V1-V6
18-C								90	N	90	N	0.01	0.06	0.14	N	0.06	N	ST-↓V1-V6	T-WAVE↓V1-V6	N	0.36	ST-↓V1-V6 AND T-WAVE↓V1-V6
19-A	SAMPPANGIRAMAIAH G L	986666	93 YR	M	LT-THALAMIC BLEED	N	N	84`	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.52	QT-INTERVAL PROLONGED AND U WAVES IN V1-V6
19-B								84	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.52	QT-INTERVAL PROLONGED AND U WAVES IN V1-V6
19-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.52	QT-INTERVAL PROLONGED AND U WAVES IN V1-V6, PATIENT EXPIRED AFTER 2 WEEKS
20-A	VENKATESHAPPA	507690	45 YR	M	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-178	90	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVES IN V1-V6	N	0.36	T-WAVES IN V1-V6
20-B								92	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVES IN V1-V6	N	0.36	T-WAVES IN V1-V6
20-C								88	N	90	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVES IN V1-V6	N	0.36	T-WAVES IN V1-V6
21-A	ANJINAPPA	477355	60 YR	M	CEREBELLAR AND PONTINE INFARCT	N	N	82	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓ IN ALL THE LEADS EXCEPT V1,V2	T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2	N	0.36	ST-↓ IN ALL THE LEADS AND T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2

21-B								84	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓ IN ALL THE LEADS EXCEPT V1, V2	T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2	N	0.36	ST-↓ IN ALL THE LEADS AND T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2
21-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	ST-↓ IN ALL THE LEADS EXCEPT V1,V2	T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2	N	0.36	ST-↓ IN ALL THE LEADS AND T-WAVE↓IN ALL THE LEADS, EXCEPT V1,V2
22-A	CHANGALAR AYAPPA	467875	70 YR	M	RT-INTRACEREBRAL BLEED WITH VENTRICULAR EXTENTION	N	N	84	N	60	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.54	QT-INTERVAL PROLONGED
22-B					R EXTENTION			82	N	60	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.54	QT-INTERVAL PROLONGED
22-C								82	N	60	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.54	QT-INTERVAL PROLONGED, PATIENT EXPIRED AFTER 1 MONTH.
23-A	KANTHAMMA		65 YR	F	B/L FRONTOTEMPORAL INFARCTS	N	N	90	AF	90						0.06	N	N	N	N	0.36	ATRIAL FIBRILLATION
23-B								92	AF	90						0.06	N		N	N	0.36	ATRIAL FIBRILLATION
23-C								90	AF	90						0.06	N	N	N	N	0.36	ATRIAL FIBRILLATION. PT DIED AFTER 3 DAYS

24-A	LAKSHMAMMA	491270	65 YR	F	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA,TRIG-337	92	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
24-B								90	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
24-C								92	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	N
25-A	MUNIVENKATA REDDY	495890	75 YR	F	RT-PARIETAL LOBE INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-190	80	N	60	N	0.01	0.08	0.14	N	0.06	N	N	N	N	0.48	QT-INTERVAL PROLONGED
25-B								82	N	60	N	0.01	0.08	0.14	N	0.06	N	N	N	N	0.46	QT-INTERVAL PROLONGED
25-C								80	N	60	N	0.01	0.08	0.14	N	0.06	N	N	N	N	0.38	N
26-A	MUNIVENKATAPPA	485542	65 YR	M	RT-INTERNAL CAPSULE BLEED	N	N	88	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6	N	0.36	T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6
26-B								90	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6			T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6
26-C								90	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6			T-WAVE↓Ⅱ, Ⅲ, AVF AND V1-V6
27-A	RAMESH	556796	31 YR	M	SUB ARCHANOID HEMORRAGE	N	N	78	N	30	N	0.01	0.08	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.38	U-WAVES IN V1-V6
27-B								80	N	30	N	0.01	0.08	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.38	U-WAVES IN V1-V6
27-C								80	N	30	N	0.01	0.08	0.12	N	0.06	N	N	N	U-WAVES IN V1-V6	0.38	U-WAVES IN V1-V6
28-A	CHAMARAIAH N	477419	38 YR	M	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-182	86	N	90	N	0.01	0.06	0.14	N	0.08	N	N	T-WAVE↓Ⅰ, AVL AND V1-V4	N	O.36	T-WAVE↓Ⅰ, AVL AND V1-V4
28-B								82	N	90	N	0.01	0.06	0.12	N	0.08	N	N	T-WAVE↓Ⅰ, AVL AND V1-V4	N	0.36	T-WAVE↓Ⅰ, AVL AND V1-V4

28-C								84	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅰ, AVL AND V1-V4	N	0.36	T-WAVE↓Ⅰ, AVL AND V1-V4
29-A	AHAMED BASHA	498641	55 YR	M	RT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-182	90	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6	N	0.38	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6
29-B								92	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6	N	0.38	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6
29-C								90	N	60	N	0.01	0.06	0.14	N	0.06	N	N	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6	N	0.40	T-WAVE↓Ⅱ, Ⅲ, AVF ANS V3-V6
30-A	CHANGAPPA	501933	45 YR	M	LT-MCA INFARCT	N	HYPERTRIGLYCERIDEMIA, TRIG-300	86	N	90	N	0.01	0.08	0.12	N	0.06	N	N	N	N	0.36	N
30-B								82	N	90	N	0.01	0.06	0.12	N	0.08	N	N	N	N	0.38	N
30-C								84	N	90	N	0.01	0.08	0.12	N	0.06	N	N	N	N	0.36	N
31-A	SHRUTHI	432160	24 YR	F	CVT	N	N	82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ IN V1-V6	N	0.38	T-WAVE↓ IN V1-V6
31-B								86	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ IN V1-V6	N	0.36	T-WAVE↓ IN V1-V6
31-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓ IN V1-V6	N	0.38	T-WAVE↓ IN V1-V6
32-A	HASINA BEGAM	567780	21 YR	F	CVT	N	N	120	N	90	N	0.01	0.06	0.12	N	O.O6	N	N	N	N	0.36	SINUS TACHYCARDIA
32-B								112	N	90	N	0.01	0.06	0.12	N	0.06	N	N	N	N	0.36	SINUS TACHYCARDIA
32-C								90	N	90	N	O.O1	0.08	0.12	N	0.06	N	N	N	N	0.36	N
33-A	GOPALAPPA	486750	82 YR	M	LT-MCA HEMORRAGE	N	N	88	N	90	N	0.02	0.08	0.14	N	0.06	N	N	N	N	0.52	QT-INTERVAL PROLONGED
33-B								90	N	90	N	0.02	0.08	0.14	N	0.06	N	N	N	N	0.54	QT-INTERVAL PROLONGED
33-C								88	N	90	N	0.02	0.08	0.14	N	0.06	N	N	N	N	0.52	QT-INTERVAL PROLONGED, PATIENT EXPIRED AFTER 3 WEEKS.

34-A	RATHNAMMA	478920	58 YR	F	RT-MCA HEMORRAGE	N	N	82	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.38	T-WAVE↓V1-V6
34-B								80	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.38	T-WAVE↓V1-V6
34-C								82	N	60	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V6	N	0.36	T-WAVE↓V1-V6, PATIENT EXPIRED AFTER 2 WEEKS.
35-A	MUNISHAMAPPA	575720	85 YR	M	RT-PUTAMEN BLEED	N	HYPERTRIGLYCERIDEMIA, TRIG-260	78	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	N	0.54	QT-INTERVAL PROLONGED
35-B								80	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	N	0.52	QT-INTERVAL PROLONGED
35-C								82	N	90	N	0.01	0.06	0.14	N	0.06	N	N	N	N	0.50	QT-INTERVAL PROLONGED, PATIENT EXPIRED AFTER 3WEEKS.
36-A	NARASIMHAPPA	575508	55 YR	M	RT-PCA INFARCT	N	N	86	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4
36-B								84	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4
36-C								82	N	90	N	0.01	0.06	0.12	N	0.06	N	N	T-WAVE↓V1-V4	N	0.36	T-WAVE↓V1-V4.