

A study of electrocardiographic changes in acute cerebrovascular accidents

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Abstract

Background: Cardiac abnormalities occur in majority of patients after cerebrovascular accidents, accounting for unexpected deaths during the first month. The most common disturbances include electrocardiogram (ECG) abnormalities, cardiac arrhythmias, and myocardial injury and dysfunction.

Objectives: The present study aims to determine the electrocardiographic changes observed in different types of stroke, their prevalence, and to establish the prognostic significance of the ECG changes.

Material and Methods: This prospective study was carried out in a superspeciality hospital during 2 years which comprised of 100 patients. Patients were categorized based on computerized tomography findings into cerebral infarction, intracerebral hemorrhage, and subarachnoid hemorrhage. ECG changes are interpreted with rate, rhythm, and abnormalities and conclusions were derived.

Results: Stroke was most common in 5th and 6th decade. Cerebral infarction formed the largest group. Males had higher preponderance. Hypertension was the most common risk factor. In total, 74% had electrocardiographic abnormality. ECG changes are more common among cerebral hemorrhage and subarachnoid hemorrhage. Most common ECG abnormality was prolonged QTc interval. Overall immediate mortality was 23%. It was high in cerebral hemorrhage. Mortality was high in patients with abnormal ECG, mostly with prolonged QTc and with T-wave inversion.

Conclusion: Patients with cerebrovascular accidents often have abnormal ECG in the absence of known organic heart disease or electrolyte imbalance. These ECG changes are more common in hemorrhagic than ischemic stroke. The mortality in these patients did not relate to the ECG changes seen but was dependent on the type of cerebrovascular accident and the level of consciousness on admission.

KEY WORDS: Cerebrovascular accidents; electrocardiographic changes; mortality

Introduction

Cerebrovascular accidents (CVAs) or stroke is defined as "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 h, or longer or leading to death, with no apparent cause other than

of vascular origin".^[1] A typical ischemic stroke presents with the abrupt onset of a focal neurological deficit and is characterized clinically by mode of onset and subsequent course in it. Three major classes of strokes are now recognized, i.e., the ischemic variety with cerebral infarction or ischemia, the hemorrhagic variety intracerebral with ruptured aneurysm in the young, and the hypertensive cerebral hemorrhage in the elderly group and subarachnoid hemorrhage, and rare type lacunar strokes are deep, small cerebral infarcts.^[2]

The symptoms and signs of an ischemic stroke vary depending on the location of the occlusion and the extent of spaced collateral flow.^[3,4] Virtually any symptom of brain dysfunction may occur. However, the abrupt onset of a hemiparesis/hemiplegia in an individual in atherosclerotic age groups is hallmark. Either anterior (carotid) or posterior (vertebro-basilar) circulation may be involved.^[5]

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Cushing first described hemodynamic changes after acute cerebral hemorrhage.^[6] Subsequent clinical observations began to identify the importance of the brain–heart interaction in patients with cerebral lesions. Cardiac abnormalities were described with various central nervous system diseases including seizures, trauma, ischemic stroke, and intracerebral hemorrhage and less commonly with tumors, electroconvulsive therapy, and meningitis.^[7] In recent times, an emotion- and stress-induced cardiomyopathy has been described.^[8,9]

The medulla has been described as the principal site of vagal parasympathetic and sympathetic areas involved in cardiac control.^[10] In addition, both anatomical and physiological evidence exist to implicate the hypothalamus in cardiac control.^[11] Electrical stimulation experiments suggest a posteriorly located area of cardiovascular sympathetic control and an anterior parasympathetic control region. Beattie and colleagues first described cardiac arrhythmias after hypothalamic stimulation.^[13]

Cardiac abnormalities occur in 60–70% of patients after stroke.^[14] The most common disturbances include electrocardiogram (ECG) abnormalities, cardiac arrhythmias, and myocardial injury and dysfunction. Distinguishing cardiac abnormalities directly caused by stroke, however, remains difficult because the prevalence of preexisting cardiac disease is high, particularly among patients with ischemic stroke.^[15] Cardiac disturbances are the most common cause of death after stroke, accounting for up to 6% of unexpected deaths during the first month.^[16] Clinical observational studies among stroke patients also suggest a loss of cardiac parasympathetic tone,^[17] loss of nocturnal vagal dominance,^[18] and increased sympathetic tone.^[19]

Among acute stroke patients, prolonged QT interval is more frequently observed after hemorrhagic strokes, occurring in 45–71% of patients with subarachnoid hemorrhage (SAH) or intracerebral hemorrhage (ICH) compared to 38% of those with ischemic strokes.^[20–22] ST segment changes (including ST elevations) occur in 22–35% of patients with ischemic stroke, which is complicated by the increased prevalence of cardiac disease.^[23] Inverted or flat T waves have also been reported in up to 55% of patients with SAH, the stroke subgroup with the lowest prevalence of coexistent cardiac disease.^[24] New Q waves similar in morphology to those observed in acute myocardial infarction are also common after acute stroke, reported in approximately 10% of patients with acute ischemic or hemorrhagic stroke.^[21,23] New U waves occur in isolation or with T waves and QT abnormalities in approximately 13–15% of patients with acute ischemic stroke and SAH.^[25] Most arrhythmias occur within the first week after stroke, occurring in 25–40% of patients with ischemic stroke or ICH, and 98% of patients with SAH.^[26–28]

Material and Methods

This is a hospital-based prospective study carried out in a superspeciality hospital during a period of 2 years which

comprised of 100 patients. All patients with acute cerebrovascular accidents admitted in the medical ward within 72 h of onset of stroke were considered. After admission a detailed history regarding the temporal profile of the stroke including history of risk factors like hypertension, diabetes mellitus, smoking, history of ischemic heart disease, and rheumatic heart disease were obtained. Patients were categorized based on Computerized Tomography findings into cerebral infarction, cerebral hemorrhage and subarachnoid hemorrhage. ECG changes are interpreted with rate, rhythm, ST-segment, QRS complex, T-wave amplitude and morphology, and QTc interval were calculated. Data were tabulated and subjected to statistical analysis.

Results

In total, 100 patients with the diagnosis of cerebrovascular accidents admitted in neurointensive care unit during the period of 2 years were studied and analyzed with regard to ECG changes. P-value greater than 0.05 was considered as not significant. The incidence of stroke was more common in 5th and 6th decade (57%) and there was slight male (54%) preponderance compared to females (46%) making male–female ratio of 1.1:1 and with the increase in age incidence of stroke is more among females when compared to males (Table 1). The incidence of cerebral infarction was more common (64%) compared to cerebral hemorrhage (29%) and SAH (7%) and the incidence of stroke was little more common among males (Table 2) Out of total 54 males, 64.8% had ischemic stroke and out of 46 females, 63% had hemorrhagic stroke. The difference of 1.8% is found to be not significant (Table 3). Hypertension (HTN) was the most common risk factor which was present in 35% of the stroke cases, followed by diabetes mellitus (DM) (16%), smoking (15%), combination of HTN and DM (10%), and previous history of stroke is seen in 7%, and hyperlipidemia in 9%. Briefly, 26 cases had a normal ECG (26%) while 74 cases (74%) had some abnormality in the ECG.

ECG abnormalities were more common in patients of stroke. ECG abnormalities were more among cases of hemorrhagic stroke, i.e., cerebral hemorrhage (23 of 29 cases, i.e., 79.31%) and SAH (5 out of 7 cases, i.e., 71.4%), and in cerebral infarction, it was 71.87% (i.e., 46 of 64 cases),

Table 1: Age and sex distribution in stroke patients

| Age group | Male | Female | Percentage |
|-----------|------|--------|------------|
| 30–39 | 3 | 1 | 4 |
| 40–49 | 13 | 5 | 18 |
| 50–59 | 16 | 13 | 29 |
| 60–69 | 13 | 15 | 28 |
| 70–79 | 6 | 8 | 14 |
| 80–90 | 3 | 4 | 7 |
| | 54 | 46 | 100 |

Table 2: Incidence of infarct and hemorrhage with reference to sex

| Type of stroke | Male | | Female | | Total | Percent |
|-------------------------|------|-------|--------|-------|-------|---------|
| | No | % | No | % | | |
| Cerebral infarction | 35 | 64.81 | 29 | 63.04 | 64 | 64 |
| Cerebral hemorrhage | 15 | 27.77 | 14 | 30.43 | 29 | 29 |
| Subarachnoid hemorrhage | 4 | 7.40 | 3 | 6.52 | 7 | 7 |
| Total | 54 | | 46 | | 100 | 100 |

Table 3: Test for association between sex and type of stroke

| Sex | Ischemic stroke | Hemorrhagic stroke | Total |
|--------|-----------------|--------------------|------------|
| Male | 35 (64.8%) | 19 (35.2%) | 54 (100%) |
| Female | 29 (63%) | 17 (37%) | 46 (100%) |
| Total | 64 (64%) | 36 (36%) | 100 (100%) |

Chi-square test value = 0.03.

Table 4: Test for association between electrocardiographic abnormalities and type of stroke

| ECG changes | Ischemic stroke | Hemorrhagic stroke | Total |
|-------------|-----------------|--------------------|------------|
| Abnormal | 46 (62.1%) | 28 (37.9%) | 74 (100%) |
| Normal | 18 (69.2%) | 8 (30.8%) | 26 (100%) |
| Total | 64 (64%) | 36 (36%) | 100 (100%) |

Chi-square test value = 0.42 (p -value >0.05 not significant).

Table 5: ECG findings: showing specific ECG changes in different types of stroke

| ECG changes | Ischemic | | Cerebral hemorrhage | | SAH | | Stroke overall |
|-----------------------|----------|-------|---------------------|-------|-------|----|----------------|
| | 46 | | 23 | | 5 | | 74 |
| | Cases | % | Cases | % | Cases | % | Percentage |
| QT prolongation | 13 | 28.26 | 8 | 34.78 | 2 | 40 | 31.08 |
| T wave inversion | 11 | 23.91 | 6 | 26.08 | 1 | 20 | 24 |
| ST segment depression | 9 | 19.56 | 5 | 21.73 | 1 | 20 | 20 |
| U waves | 3 | 6.52 | 2 | 8.69 | 0 | 0 | 6 |
| Tachycardia | 7 | 15.21 | 2 | 8.69 | 1 | 20 | 13 |
| Bradycardia | 2 | 4.34 | 0 | 0 | 0 | 0 | 2 |
| Other arrhythmias | 1 | 2.17 | 0 | 0 | 0 | 0 | 1 |

though not much difference was noted. Among 74 patients with abnormal ECG changes, 62.1% had ischemic stroke and among 26 patients with normal ECG, 69.2% had hemorrhagic stroke. The difference of 1.8% is found to be not significant. (Table 4) The most common ECG abnormality associated with stroke was prolonged QTc interval (31.08%, i.e., 23 of 74 cases) followed by T wave inversion (24.32% i.e., 18 cases), ST segment changes (20.27%, i.e., 15 cases), sinus tachycardia (13.51%, i.e., 5 cases), U-waves (6.75%, i.e., 10 cases), bradycardia (2.70%, i.e., 2 cases), and other arrhythmias (1.35%, i.e., 1 case). QTc was prolonged in 31.08% of patients. It occurred more frequently in patients with SAH (34.78%, i.e., 2 out of 5 cases) and cerebral hemorrhage (40%, i.e., 8 out of 23 cases) and less frequently in cerebral infarction. In total, 24.32% of patients showed T wave changes and in some cases where ECG was repeated after a few days T wave reverted to normal. T wave changes were more frequent in cerebral hemorrhage. ST segment depression was seen in 20.27% of patients. ST segment depression

was more frequent in cerebral hemorrhage (5 out of 23 patients, i.e., 21.73%) and subarachnoid hemorrhage (1 out of 5 patients, i.e., 20%). Sinus tachycardia is seen in 13% of the patients, more frequent in SAH (1 out of 5 cases) cerebral hemorrhage (i.e., 2 out of 23 cases). U waves were seen in 6% of cases. They occurred more frequently in patients with cerebral hemorrhage (2 out of 23 patients, i.e., 8.69%) and in cerebral infarction (3 out of 46 cases, i.e., 6.52%). Bradycardia is seen in 2% of patients, more frequent in cerebral infarction (4.34%, i.e., 2 out of 46 cases). Among other arrhythmias, they are seen in 1% of stroke patients (i.e., 1 out of 74 cases) (Table 5).

Overall immediate mortality was 23%. It was high in cerebral hemorrhage (37.93%, i.e., 11 of 29 cases) followed by subarachnoid hemorrhage (42.85%, i.e., 3 of 7 cases) and cerebral infarction (14.06%, i.e., 9 of 64 cases). Among 74 patients with abnormal ECG changes, 25.7% died and among 26 patients with normal ECG, 15.4% died. The difference of 10.3% is found to be not significant (Table 6).

Mortality was higher in patients with prolonged QTc [SAH (50%) and cerebral hemorrhage (44.44%)] and with T-wave inversion. Mortality was high in cases of infarct (25%) compared to hemorrhage (22.22%) with ST-segment depression (Table 7). Mortality was higher in patients of stroke with QTc prolongation (42.10%), followed by T-wave inversion (26.31%), and ST-Segment depression (21.05%), but none of them were statistically significant (Table 8). The highest mortality was seen in comatose patients (8 of 13 cases, i.e., 61.53%), followed by stuporous (9 of 17, i.e., 52.19%), lethargic (4 of 22, i.e., 18.18%) and least in alert patients (2 out of 48 i.e. 4.16%).

Discussion

In 1947, Byer et al^[29] described large and upright T waves and prolonged QT intervals in a patient with subarachnoid hemorrhage. In 1953, Levine reported inverted, deep T waves and ST segment elevations in a patient with a ruptured aneurysm of the circle of Willis, while the next year Burch et al^[30,31] described a new electrocardiographical pattern consisting of a prolonged QT interval, large and often inverted T waves and large U waves in patients with cerebrovascular accidents. Since then, more reports concerning ECG alterations in stroke patients appeared in the literature.

Table 6: Test for association between electrocardiographic abnormalities and mortality in stroke patients

| ECG changes | Dead | Alive | Total |
|-------------|------------|------------|------------|
| Abnormal | 19 (25.7%) | 55 (74.3%) | 74 (100%) |
| Normal | 4 (15.4%) | 22 (84.6%) | 26 (100%) |
| Total | 23 (23%) | 77 (77%) | 100 (100%) |

Chi-square test value Yates corrected = 0.64 (p -value > 0.05 not significant).

Table 7: Mortality in stroke types and its co-relation with ECG changes

| ECG changes | Cerebral infarction (N = 46) | | | | Cerebral hemorrhage (N = 23) | | | | SAH (N = 5) | | | |
|-----------------------|------------------------------|-------|----------|------|------------------------------|-------|----------|-------|-------------|-------|----------|----|
| | Alive (38) | | Dead (8) | | Alive (14) | | Dead (9) | | Alive (3) | | Dead (2) | |
| | N | % | N | % | N | % | N | % | N | % | N | % |
| QTc prolongation | 10 | 26.31 | 3 | 37.5 | 4 | 28.57 | 4 | 44.44 | 1 | 33.33 | 1 | 50 |
| T wave inversion | 9 | 23.68 | 2 | 25 | 4 | 28.57 | 2 | 22.22 | 0 | 0 | 1 | 50 |
| ST segment depression | 7 | 18.42 | 2 | 25 | 3 | 21.42 | 2 | 22.22 | 1 | 33.33 | 0 | 0 |
| U Waves | 2 | 5.26 | 1 | 12.5 | 1 | 7.14 | 1 | 11.11 | 0 | 0 | 0 | 0 |
| Sinus tachycardia | 7 | 18.42 | 0 | 0 | 2 | 14.28 | 0 | 0 | 1 | 33.33 | 0 | 0 |
| Sinus bradycardia | 2 | 5.26 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Arrhythmias | 1 | 2.63 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Chi-square value = 11.48, degrees of freedom = 30, p -value < 0.05 not significant.

Table 8: Mortality in stroke patients and its correlation with ECG changes

| Type of ECG changes | Stroke patients | | | |
|-----------------------|-----------------|-------|-----------|-------|
| | Alive (55) | | Dead (19) | |
| | No | % | No | % |
| QTc prolongation | 15 | 27.27 | 8 | 42.10 |
| T-wave inversion | 13 | 23.63 | 5 | 26.31 |
| ST segment depression | 11 | 20 | 4 | 21.05 |
| U waves | 03 | 5.45 | 2 | 10.52 |
| Sinus tachycardia | 10 | 18.18 | 0 | 0 |
| Sinus bradycardia | 2 | 3.63 | 0 | 0 |
| Arrhythmias | 1 | 1.81 | 0 | 0 |

Chi-square value = 6.077. Degree of freedom = 6, p -value = < 0.05 not significant.

The present study which dealt primarily with ischemic stroke (cerebral infarct), cerebral hemorrhagic stroke, and SAH demonstrates that electrocardiographical abnormalities occur very frequently in these conditions. Among the 100 patients, 54 were males and 46 were females (sex ratio was M:F – 1.1:1). The cases of stroke were more common in the 5th and 6th decade, making 61%, which is comparable to Bozluoclay et al study in which 47.1% were females and 52.9% males.^[32] Similar findings were noted by Garg.^[33] The mean age was 65. Cerebral infarction formed the largest group in the present study comprising 64% of cases. The next common type of stroke was cerebral hemorrhage comprising 29% of cases and subarachnoid hemorrhage (7%) which is comparable to Daniele et al study where 78% had ischemic stroke and 21% hemorrhagic stroke.^[34] Stroke is one of the leading causes of death in many countries.

Although there was a lack of unanimity, several factors have been reported to increase the risk of stroke. Reports from different countries have implicated different factors associated with high risk of stroke. Shaper et al in 1991 concluded that, hypertension; cigarette smoking and pre-existing IHD were found to be the major risk factors.^[35,36] Both diabetes and metabolic syndrome are recognized to increase the risk of ischemic stroke in men and women.^[37] In the present study, hypertension was present in majority of the cases, i.e., 35%, which is comparable with that found in the studies of Foulkes et al in which hypertension was the most frequently reported cardiovascular disease affecting 38–75% of patients in each stroke diagnostic subtype, and diabetes mellitus was present in 16% of the patients in present study, which is comparable to Foulkes et al study where incidence of diabetes is 21%.^[38] In a study conducted by Whisnant et al Hypertension accounts for 35–50% of stroke risk^[39] which is comparable to the present study, i.e., 35%. In the present study, ECG changes are seen in 74% of the patients, which is comparable to Daniele et al study in which ECG abnormalities are seen in 75% of the patients^[34] and Goldstein et al study where ECG changes are seen in 92% of the stroke patients.^[37] In a study conducted by Goldstein et al (92%), the most common ECG abnormalities seen are: QT prolongation (45%), followed by T wave inversion (29%), ST-Segment depression (27%), U waves (28%), tachycardia (28%), and other arrhythmias (7%). Similar observations were noted in the present study with QT prolongation (31.08%), followed by T wave inversion (24.32%), ST-segment depression (20.27%), U waves (6%), tachycardia (13.51%), and other arrhythmias (1.35%). Similar findings were also observed by Golwalkar et al.^[40] Overall immediate mortality was 23%. It was high in SAH (43%) followed by cerebral hemorrhage (38%), and cerebral infarction (14%) which can be compared to Goldstein et al study where overall mortality in ICH, SAH, and infarction are 56%, 54%, and 14%, respectively. Overall immediate mortality was 23% which is comparable to Goldstein et al study (25%).^[36] In the present study, it was observed that mortality is high in stuporous and comatose patients, i.e., 52.19% and 61.53%, respectively, and less in alert patients, i.e., 4.16% which is comparable

to Goldstein et al^[36] study where mortality in stuporous and comatose patients was 47% and 75%, respectively, and in alert patients it was 4%.

An attempt was made to correlate the mortality in these patients with the ECG changes. It was found that the mortality in these patients did not relate to the ECG changes seen, but was dependent on the level of consciousness on admission, the type of cerebrovascular accident, the extent of the lesion and other co-existent disease. The occurrence of cardiac abnormalities in the acute phase of stroke as evidenced by the appearance of new ECG alterations or aggravation of pre-existing ones may raise the possibility that the cardiac pathology pre-existed or even served as a predisposing factor for the appearance of the stroke, as it is known that stroke and heart disease share common vascular etiology in the older age groups, however in present study patients with prior history of heart disease and who are on cardiac drugs are not included. Further ECG changes become manifest or aggravated due to extracerebral causes such as electrolyte imbalance, loss of fluid, shock and respiratory disturbances which may accompany the cerebrovascular event. However, in our patients, who were closely watched in an ICU, the ECG abnormalities occurred in the absence of such disturbances, leaving the impression that in most cases the ECG changes were induced by the brain lesion itself. The present study demonstrates that by continuous ECG monitoring cardiac disturbances, especially the rhythm and conduction abnormalities can be detected and treated immediately.

Conclusion

In conclusion, patients with cerebrovascular accidents often have abnormal ECG in the absence of known organic heart disease or electrolyte imbalance. These ECG changes are more common in intracerebral hemorrhage and subarachnoid hemorrhage than in infarcts. The common ECG changes are prolonged QTc, T wave inversion, ST segment depression and tachycardia. The mortality in these patients did not relate to the ECG changes seen but was dependent on the type of cerebrovascular accident and the level of consciousness on admission. ECG abnormalities in stroke patients do not have any prognostic significance.

References

1. WHO MONICA Project Principal Investigators. The World Health Organization MONICA Project (Monitoring trends and determinants in cardiovascular disease): A major international collaboration. *J Clin Epidemiol* 1988;41(2):105–14.
2. Dalal PM. Ischemic cerebrovascular diseases. In: *API Textbook Of Medicine*, Munjal YP (Ed.). 9th ed. New Delhi: Jaypee, 2012. pp. 1401–10.
3. Romero JR, Pikula A, Nguyen TN, Nien YL, Norbash A, Babikian VL. Cerebral collateral circulation in carotid artery disease. *Curr Cardiol Rev* 2009;5(4):279–88.

4. Na DG, Sohn C-H, Kim EY. Imaging-based management of acute ischemic stroke patients: Current neuroradiological perspectives. *Korean J Radiol.* 2015;16(2):372–90.
5. Wade S Smith, Joey D English, S Claiborne Jhonston. *Cerebrovascular diseases. Harrison's principles of Internal Medicine.* 18th ed. New Delhi: Jaypee; 2012. pp 3271–89.
6. Cushing H. The blood pressure reaction of acute cerebral compression illustrated by cases of intracranial hemorrhage. *Am J Med Sci* 1903;125:1017.
7. Banki NM, Zaroff JG. Neurogenic cardiac injury. *Curr Treat Options Cardiovasc Med* 2003;5:451.
8. Wittstein IS, Thiemann DR, Lima JA. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med* 2005;352:539.
9. Ako J, Sudhir K, Farouque HM. Transient left ventricular dysfunction under severe stress: brain–heart relationship revisited. *Am J Med* 2006;119:10.
10. Kalia M, Mesulam MM. Brain stem projections of sensory and motor components of the vagus complex in the cat: II. Laryngeal, tracheobronchial, pulmonary, cardiac, and gastrointestinal branches. *J Comp Neurol* 1980;193(2):467–508.
11. Calaresu FR, Ciriello J. Projections to the hypothalamus from buffer nerves and nucleus tractus solitarius in the cat. *Am J Physiol* 1980;239:R130.
12. Melville KI, Blum B, Shister HE, Silver MD. Cardiac ischemic changes and arrhythmias induced by hypothalamic stimulation. *Am J Cardiol* 1963;12:781–91.
13. Beattie J, Brow G, Long C. Physiology and anatomical evidence for the existence of nerve tracts connecting the hypothalamus with spinal sympathetic centres. *Proc R Soc Lond (Biol)* 1930;106:253.
14. Hachinski VC. The clinical problem of brain and heart. *Stroke* 1993; 24:11.
15. Wilterdink JL, Furie KL, Easton JD. Cardiac evaluation of stroke patients. *Neurology* 1998; 51:S23–6.
16. Reeves MJ, Bushnell CD, Howard G, Gargano JW, Duncan PW, Lynch G, et al. Sex differences in stroke: Epidemiology, clinical presentation, medical care, and outcomes. *Lancet Neurol* 2008;7(10):915–26.
17. Barron SA, Rogovski Z, Hemli J. Autonomic consequences of cerebral hemisphere infarction. *Stroke* 1994;25:113–6.
18. Korpelainen JT, Sotaniemi KA, Huikuri HV, Myllylä VV. Circadian rhythm of heart rate variability is reversibly abolished in ischemic stroke. *Stroke* 1997;28:2150–4.
19. Strittmatter M, Meyer S, Fischer C. Location-dependent patterns in cardio-autonomic dysfunction in ischaemic stroke. *Eur Neurol* 2003;50(1):30–8.
20. Dimant J, Grob D. Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents. *Stroke* 1977;8:448–55.
21. Yamour BJ, Sridharan MR, Rice JF. Electrocardiographic changes in cerebrovascular hemorrhage. *Am Heart J* 1980; 99(3):294–300.
22. Amin OSM, Myckan HA, Hussein EH. QTc interval prolongation and stroke: Any differences between ischemic and hemorrhagic strokes?. *Cukurova Med J.* 2014;39(1):75–82.
23. Di Pasquale G, Pinelli G, Andreoli A. Torsade de pointes and ventricular flutter-fibrillation following spontaneous cerebral subarachnoid hemorrhage. *Int J Cardiol* 1988;18:163.
24. Solenski NJ, Haley Jr EC, Kassell NF. Medical complications of aneurysmal subarachnoid hemorrhage: A report of the multicenter, cooperative aneurysm study. Participants of the Multicenter Cooperative Aneurysm Study. *Crit Care Med* 1995;23:1007.
25. Sommargren CE, Warner R, Zaroff JG. Electro-cardiographic abnormalities in patients with subarachnoid hemorrhage and normal adults: A comparison study. *J Electrocardiol* 2004; 37(Suppl 1):42–3.
26. Sommargren CE, Zaroff JG, Banki N. Electro-cardiographic re-polarization abnormalities in subarachnoid hemorrhage. *J Electrocardiol* 2002; 35(4):257–62.
27. Andreoli A, di Pasquale G, Pinelli G. Subarachnoid hemorrhage: Frequency and severity of cardiac arrhythmias. *Stroke* 1987; 18(3):558–64.
28. Cheung RT, Hachinski V. Cardiac effects of stroke. *Curr Treat Options Cardiovasc Med* 2004;6(3):199–207.
29. Byer E, Ashman R, Toth LA. Electrocardiograms with large upright T waves and long QT intervals. *Am Heart J* 1947;33:796–806
30. Levine HD. Nonspecificity of the electrocardiogram associated with coronary artery disease. *Am J Med* 1953;15:344–55.
31. Burch CE, Meyers R, Abildskov JA. A new electrocardiographic pattern observed in cerebrovascular accidents. *Circulation* 1954;9:719–23.
32. Bozluolcay M, Ince B, Celik Y, Harmanci H, Ilerigelen B, Pelin Z. Electrocardiographic findings and prognosis in ischemic stroke. *Neurol India* 2003;51(4):500–2.
33. Garg Y. ECG changes in patients of cerebrovascular diseases. *Int Med J* 2015; 2(4):256–9.
34. Daniele O. Stroke and cardiac arrhythmias. *J Stroke Cerebrovascular Dis* 2002;11(1):28–33
35. Sharper AG, Phillips AN, Pocock SJ. Risk factors of stroke in middle aged British men. *BMJ* 1991;302:1111–5.
36. Sandhya M, Satyanarayana U, Joyarani D. Study of ECG changes and left ventricular diastolic dysfunction as hemodynamic markers of myocardial stress in chronic smokers. *Int J Res Med Sci.* 2015;3(3):588–92.
37. Goldstein LB, Adams R, Alberts MJ. Primary prevention of ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council. *Circulation* 2006;113:e873–923.
38. Foulkes MA, Wolf PA, Price TR. The stroke database: Design, methods & baseline characteristics. *Stroke* 1988;19:547–54.
39. Whisnant JP. Effectiveness versus efficacy of treatment of hypertension for stroke prevention. *Neurology* 1996;(2):301–7.
40. Golwalkar JK, Dond PT, Rayate VS, Mugadlimath AB. Study of electrocardiographic patterns in clinically normal persons above sixty years of age. *International Journal of Advances in Medicine.* 2015;2(3):211–7.

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