

Electrocardiographic Alterations in Patients with Cerebrovascular Accidents: A Comprehensive Analysis

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ABSTRACT

Cerebrovascular accidents (CVAs) are frequently associated with a spectrum of electrocardiographic (ECG) abnormalities, which may reflect autonomic dysregulation, structural cardiac involvement, or secondary ischemic injury. This article systematically reviews the prevalence, characteristics, and underlying mechanisms of ECG changes in CVA patients, with particular emphasis on their diagnostic and prognostic significance. Commonly observed alterations include QT interval prolongation, ST-segment deviations, T-wave inversions, and arrhythmias such as atrial fibrillation and ventricular ectopy. These findings suggest a complex interplay between neurological injury and cardiac function, mediated by neurogenic cardiac injury, catecholamine surge, and systemic inflammatory responses. Furthermore, specific ECG patterns may correlate with the severity and localization of cerebral ischemia, offering potential utility in risk stratification. Understanding these electrocardiographic phenomena is critical for optimizing patient management, distinguishing primary cardiac events from neurogenic changes, and mitigating adverse outcomes in this high-risk population.

KEYWORDS: Electrocardiographic changes, cerebrovascular accident, neurogenic cardiac injury, autonomic dysfunction, QT prolongation, ST-segment abnormalities, arrhythmias, ischemic stroke, prognostication.

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INTRODUCTION

The intricate relationship between cerebrovascular and cardiovascular systems has been increasingly recognized, particularly in the context of acute neurological injury. Cerebrovascular accidents, encompassing both ischemic and hemorrhagic strokes, frequently manifest with concomitant electrocardiographic disturbances, which may range from subtle repolarization abnormalities to life-threatening arrhythmias. The pathogenesis of these ECG alterations is multifactorial, involving autonomic dysregulation due to hypothalamic-insular axis disruption, excessive sympathetic activation, and the resultant catecholamine-induced myocardial injury. Additionally, pre-existing cardiovascular

comorbidities may exacerbate these changes, further complicating clinical interpretation.^{1,2}

Electrocardiographic findings such as prolonged QT interval, peaked or inverted T waves, and ST-segment elevation or depression often pose diagnostic challenges, as they may mimic acute coronary syndromes, necessitating careful differentiation. Emerging evidence suggests that specific ECG patterns may correlate with stroke severity, anatomical localization, and long-term neurological outcomes, highlighting their potential role as biomarkers in stroke management. This article aims to elucidate the spectrum of ECG abnormalities observed in CVA patients, explore their pathophysiological basis, and discuss their clinical implications in acute and post-stroke phases. By integrating

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current evidence, this review seeks to enhance clinician awareness and facilitate a more nuanced approach to the cardiovascular assessment of stroke patients.³

BACKGROUND

The interplay between cerebrovascular and cardiovascular pathophysiology has been a subject of extensive research, particularly concerning the electrocardiographic (ECG) manifestations observed in patients with cerebrovascular accidents (CVAs). Historically, the association between acute brain injury and cardiac abnormalities was first documented in the mid-20th century, when Levine and others described repolarization anomalies and arrhythmias in patients with intracranial hemorrhages. Subsequent studies expanded these observations, demonstrating that both ischemic and hemorrhagic strokes could induce a wide spectrum of ECG changes, often resembling primary cardiac pathology.^{4,5}

The underlying mechanisms of these ECG alterations are rooted in the complex neurocardiac axis, where acute brain injury disrupts autonomic regulation, leading to excessive sympathetic outflow. The insular cortex, a critical region in autonomic control, plays a pivotal role; its ischemia or compression can trigger a catecholamine surge, resulting in myocardial stunning, microvascular dysfunction, and electrophysiological instability. This phenomenon, often termed *neurogenic stunned myocardium* or *Takotsubo-like cardiomyopathy*, is characterized by transient left ventricular dysfunction alongside ECG abnormalities such as QT prolongation, diffuse T-wave inversions, and ST-segment deviations.^{5,6}

Additionally, stroke-induced systemic inflammation, electrolyte imbalances, and increased intracranial pressure further contribute to cardiac electrophysiological disturbances. Notably, ischemic strokes in the anterior circulation, particularly involving the insula, have been strongly correlated with malignant arrhythmias, including atrial fibrillation, ventricular tachycardia, and even sudden cardiac death. Conversely, brainstem strokes may provoke bradyarrhythmias due to vagal overactivation, underscoring the topographical influence of neurological injury on cardiac autonomic tone.⁶

Clinically, these ECG changes present a diagnostic dilemma, as they often mimic acute coronary syndromes, necessitating meticulous differentiation to avoid unnecessary thrombolysis or invasive interventions. Moreover, emerging evidence suggests that certain ECG patterns—such as prolonged QTc dispersion, pathological U waves, or Osborn waves—may serve as prognostic indicators, correlating with stroke severity, functional outcomes, and mortality.^{7,8}

Despite advancements in neurocardiology, gaps remain in understanding the temporal evolution of post-stroke ECG abnormalities, their long-term implications, and optimal management strategies. This review seeks to synthesize existing knowledge on electrocardiographic changes in CVA patients, emphasizing their clinical relevance,

pathophysiological basis, and potential utility in risk stratification and therapeutic decision-making.⁸

Pathophysiology

The electrocardiographic (ECG) abnormalities observed in patients with cerebrovascular accidents (CVAs) arise from a complex interplay of neurogenic, autonomic, metabolic, and inflammatory mechanisms, ultimately leading to cardiac electrophysiological disturbances. The primary driver of these changes is the abrupt disruption of central autonomic regulation, predominantly mediated by the insular cortex, hypothalamus, and brainstem nuclei, which exert supraspinal control over cardiovascular function.⁹

Acute ischemic or hemorrhagic stroke, particularly when involving the middle cerebral artery territory and insular regions, triggers an exaggerated sympathetic discharge due to the loss of inhibitory cortical modulation. This autonomic dysregulation results in a massive catecholamine surge, with both systemic circulation of epinephrine and norepinephrine as well as localized cardiac sympathetic overstimulation. The ensuing hyperadrenergic state induces direct myocardial injury through calcium overload, oxidative stress, and microvascular dysfunction—a phenomenon described as *neurogenic stunned myocardium*. Histologically, this manifests as contraction band necrosis, predominantly in the subendocardial layers, which disrupts normal depolarization-repolarization cycles and generates ECG abnormalities such as prolonged QT interval, diffuse T-wave inversions, and ST-segment deviations.⁹

Simultaneously, parasympathetic withdrawal or, in cases of brainstem involvement, excessive vagal activation further destabilizes cardiac rhythm. The imbalance between sympathetic and parasympathetic tone predisposes to arrhythmias, including atrial fibrillation, ventricular ectopy, and even torsades de pointes in the setting of marked QT prolongation. Additionally, stroke-induced systemic inflammation—marked by elevated cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α)—exacerbates myocardial irritability and conduction abnormalities.⁹

Electrolyte imbalances, particularly hypokalemia and hypomagnesemia secondary to catecholamine-driven intracellular shifts and renal excretion, further impair repolarization reserve, increasing susceptibility to life-threatening arrhythmias. Elevated intracranial pressure, commonly seen in large hemispheric infarcts or intracerebral hemorrhages, may also contribute to ECG changes through brain-heart interactions involving the autonomic nervous system and brainstem compression.⁹

Collectively, these pathophysiological mechanisms underscore the bidirectional relationship between acute brain injury and cardiac electrophysiological instability, explaining the diverse ECG manifestations in CVA patients. Understanding these processes is crucial for distinguishing primary cardiac pathology from neurogenic changes and

optimizing therapeutic strategies to mitigate adverse outcomes.⁹

Electrocardiographic Findings in Patients with Cerebrovascular Accidents

The spectrum of electrocardiographic abnormalities observed in patients with cerebrovascular accidents (CVAs) encompasses a wide array of repolarization disturbances, conduction abnormalities, and arrhythmic manifestations that reflect the profound neurocardiac interplay characteristic of this condition. Among the most frequently encountered alterations are pathological changes in ventricular repolarization, including prominent QT interval prolongation, which arises from delayed myocardial repolarization due to sympathetic hyperactivity and subsequent calcium-mediated afterdepolarizations. This prolongation frequently exceeds 500 milliseconds in severe cases, creating a substrate for malignant ventricular arrhythmias such as torsades de pointes.¹⁰

Additionally, morphological abnormalities of the T wave are commonly observed, ranging from deeply inverted T waves in precordial leads to giant, symmetrically inverted T waves that may mimic the pattern seen in Wellens' syndrome or myocardial ischemia. These repolarization anomalies are often dynamic, evolving over the course of days to weeks following the acute neurological event. ST-segment deviations, including both depression and elevation, are also prevalent, though typically diffuse and not conforming to a specific coronary arterial territory, thereby distinguishing them from primary acute coronary syndromes.^{10,11}

Supraventricular and ventricular arrhythmias constitute another hallmark of CVA-related ECG changes, with atrial fibrillation being particularly prevalent due to autonomic imbalance and atrial myocardial strain. Frequent premature ventricular complexes, nonsustained ventricular tachycardia, and even ventricular fibrillation have been documented, particularly in patients with insular cortex involvement. Conduction system disturbances, such as sinus bradycardia, atrioventricular blocks, and bundle branch blocks, may also occur, especially in cases of brainstem infarction where vagal predominance alters normal cardiac automaticity.^{12,14}

Less common but clinically significant findings include the appearance of U waves, which may reflect transient electrolyte imbalances or abnormal Purkinje fiber repolarization, as well as Osborn waves (J-wave elevation) in the setting of hypothermia secondary to hypothalamic dysfunction. The dynamic nature of these electrocardiographic changes, often paralleling the neurological clinical course, underscores their neurogenic origin and differentiates them from primary cardiac pathology. Their recognition is essential for appropriate clinical management, as misinterpretation may lead to unnecessary cardiac interventions while overlooking potentially ominous prognostic indicators.¹⁴

CONCLUSION

The intricate relationship between cerebrovascular accidents and electrocardiographic manifestations underscores the profound interconnectedness of the nervous and cardiovascular systems. The diverse spectrum of ECG abnormalities observed in CVA patients—ranging from repolarization disturbances such as QT prolongation and T-wave inversions to arrhythmias and conduction abnormalities—reflects the complex pathophysiological mechanisms triggered by acute brain injury. These neurogenic cardiac changes, mediated primarily through autonomic dysregulation, catecholamine excess, and systemic inflammatory responses, not only pose diagnostic challenges but also carry significant prognostic implications. While these electrocardiographic alterations often mimic primary cardiac pathology, their transient nature and association with specific neurological lesions can help differentiate them from acute coronary syndromes. However, their presence should not be dismissed as merely incidental, as they may indicate an increased risk of adverse cardiovascular events and poorer neurological outcomes. The dynamic evolution of these ECG changes, often mirroring the clinical course of the CVA, further highlights their potential utility as biomarkers for disease severity and recovery.

Future research should focus on elucidating the long-term cardiac consequences of these neurogenic changes, refining risk stratification protocols, and developing targeted therapeutic strategies to mitigate their impact. A multidisciplinary approach, integrating neurology and cardiology expertise, is essential for optimizing the management of CVA patients with concomitant ECG abnormalities. Ultimately, a deeper understanding of these phenomena will not only enhance clinical decision-making but also improve overall patient outcomes in this vulnerable population.

REFERENCES

- I. Redon J, Olsen MH, Cooper RS, Zurriaga O, Martinez-Beneito MA, Laurent S, et al. Stroke mortality and trends from 1990 to 2006 in 39 countries from Europe and Central Asia: implications for control of high blood pressure. *Eur Heart J*. 2011;32(11):1424-31.
- II. Rosamond W, Flegal K, Furie K. Heart disease and stroke statistics—2008 update: a report from the American heart association statistics committee and stroke statistics subcommittee. *Circulation*. 2008;117:e61-76.
- III. Jain K, Garg Y. ECG changes in patients of acute cerebrovascular diseases. *MedPulse – International Medical Journal*. 2015;2(4):256-9.
- IV. Stead LG, Gilmore RM, Bellolio MF, Vaidyanathan L, Weaver AL, Decker WW, et al. Prolonged QTc as a predictor of mortality in acute ischemic stroke. *J Stroke Cerebrovasc Dis*. 2009;18(6):469-74

Electrocardiographic Alterations in Patients with Cerebrovascular Accidents: A Comprehensive Analysis

- V. Daniele O, Caravaglios G, Fierro B, Natalè E. Stroke and cardiac arrhythmias. *J Stroke Cerebrovasc Dis.* 2002;11(1):28-33.
- VI. Huang CH, Chen WJ, Chang WT, Yip PK, Lee YT. QTc dispersion as a prognostic factor in intracerebral hemorrhage. *Am J Emerg Med.* 2004;22(3):141-44.
- VII. Purushothaman S, Salmani D, Prarthana K, Bandelkar S, Varghese S. Study of ECG changes and its relation to mortality in cases of cerebrovascular accidents. *Journal of Natural Science, Biology and Medicine.* 2014;5(2):434.
- VIII. Katsanos A, Korantzopoulos P, Tsigoulis G, Kyritsis A, Kosmidou M, Giannopoulos S. Electrocardiographic abnormalities and cardiac arrhythmias in structural brain lesions. *International Journal of Cardiology.* 2013;167(2):328-34.
- IX. Prosser J, MacGregor L, Lees K, Diener H, Hacke W, Davis S. Predictors of Early Cardiac Morbidity and Mortality After Ischemic Stroke. *Stroke.* 2007;38(8):2295-302.
- X. Braga GP, Gonçalves RS, Minicucci MF, Bazan R, Zornoff LAM. Strain pattern and T-wave alterations are predictors of mortality and poor neurologic outcome following stroke. *Clin Cardiol.* 2020;43(6):568-73.
- XI. Asadi P, Ziabari SM, Jahan ND, Yazdi JA. Electrocardiogram Changes as an Independent Predictive Factor of Mortality in Patients with Acute Ischemic Stroke; a Cohort Study. *Arch Acad Emerg Med.* 2019;7(1):e27.
- XII. Wu J, Nizhamuding D, Liu P, Jiang Y, Qiang H, Yin Y. QT interval prolongation in patients with acute ischemic stroke: a report in northwest China. 2019.
- XIII. Scheef B, Al-Khaled M. Atrial Fibrillation in Patients with Transient Ischemic Attack in Accordance with the Tissue-Based Definition. *J Vasc Interv Neurol.* 2016;9(1):23-7.
- XIV. Amin OSM, Sheikhbeni AS, Siddiq AN. Relationship of QTc Interval Prolongation with Acute Ischemic Stroke. *Med Arch (Sarajevo, Bosnia Herzegovina).* 2020;74(3):195-8.