Original Article

Role of Root Mean Square of Successive Differences (RMSSD), a Time Domain Measure of Heart Rate Variability in Management of Patients with Temporal Lobe Epilepsy

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Abstract

Background: Cardiac autonomic dysregulation in temporal lobe epilepsy (TLE) has been increasingly implicated in sudden unexpected death in epilepsy (SUDEP), yet systematic evaluation of heart rate variability (HRV) measures in TLE remains limited in Indian populations. **Material and Methods:** This cross-sectional study analysed cardiac autonomic function in 30 TLE patients and 30 age-matched healthy controls using time-domain HRV indices, specifically the Root Mean Square of Successive Differences (RMSSD). Electrocardiographic recordings were obtained in three conditions: lying (eyes closed), deep breathing (1 minute), and standing (5 minutes). Time domain indices (RMSSD, SDNN, NN50) were calculated using automated software and analysed using t-tests and one-way ANOVA with Bonferroni correction. **Results:** TLE patients showed significantly reduced RMSSD during lying ($p=0.006$) and standing ($p=0.013$) postures compared to controls. One-way ANOVA with Bonferroni correction revealed significant postural effects on RMSSD ($p=0.001$) and SDNN ($p=0.017$) in TLE patients but not controls, indicating impaired autonomic reserve and baroreceptor responsiveness. **Conclusion:** RMSSD is significantly reduced in interictal TLE patients, reflecting chronic parasympathetic dysfunction and autonomic dysregulation. Regular RMSSD assessment in TLE clinical protocols may enable early detection of autonomic deterioration and risk stratification for SUDEP, potentially preventing sudden cardiac mortality.

Keywords: Temporal lobe epilepsy; heart rate variability; RMSSD; autonomic dysfunction; sudden unexpected death in epilepsy; parasympathetic control.

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Introduction

Epilepsy affects over 50 million individuals worldwide, with significant morbidity and mortality.[1]

Temporal lobe epilepsy (TLE) represents the most common form of focal epilepsy in adults, accounting for approximately 60% of patients with focal seizures.[2]

Beyond seizure-related complications, TLE patients face substantially increased mortality risk, with SUDEP accounting for up to 17% of epilepsy deaths.[3]

The pathophysiologic mechanisms underlying SUDEP remain incompletely understood, but accumulating evidence increasingly implicates cardiac autonomic dysregulation as a central mechanism.[3,4]

Recent investigations demonstrate that interictal epileptiform discharges—electrographic abnormalities occurring without overt clinical seizures—propagate from the temporal lobe to the Central Autonomic Network (CAN), a distributed neural system controlling cardiopulmonary function.[5]

This autonomic dysregulation manifests as reduced parasympathetic control of heart rate, predisposing to cardiac arrhythmias, sinus node dysfunction, and sudden cardiac death.[4,6] Heart Rate Variability (HRV) represents a sensitive, non-invasive indicator of autonomic cardiovascular control.[7]

Recent evidence demonstrates that HRV analysis is a valuable method for assessing cardiovascular risk and cardioautonomic impairment.[8] Time-domain analysis of HRV provides reproducible, mathematically simple measures suitable for clinical application.[7] The Root Mean Square of Successive Differences (RMSSD) specifically reflects short-term parasympathetic (vagal) modulation of the sinoatrial node.[9]

Reduced RMSSD indicates diminished vagal tone and increased sympathetic dominance—conditions associated with cardiac arrhythmia risk.[9] Recent clinical investigations have

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documented reduced HRV in epilepsy populations, with particular emphasis on RMSSD as a critical marker.[10,11] Studies have demonstrated that RMSSD and pNN50 could be used as predictors for SUDEP-7 risk severity in epilepsy patients.[12] DeGiorgio and colleagues showed that RMSSD was significantly associated with SUDEP risk factors, as quantified by the SUDEP-7 inventory, suggesting that RMSSD could be a potential risk-stratification tool.[13] More recent meta-analyses confirm that reductions in SDNN and RMSSD are reported in most SUDEP patients. However, further studies are needed to assess the potential roles of HRV modifications as SUDEP biomarkers.[8]

Despite this growing evidence base, systematic evaluation of HRV measures in TLE across multiple physiological conditions remains limited in Indian populations.

Recent studies (2024-2025) have shown that patients with epilepsy experience reduced quality of life and poor sleep quality, which are closely related to heart rate variability.[14] Additionally, low heart rate variability correlates with cognitive impairment and emotional dysfunction in patients with epilepsy.[14]

This study examines time-domain HRV measures, particularly RMSSD, in TLE patients compared with healthy controls across various physiological states (lying, deep breathing, standing). We hypothesize that TLE patients exhibit reduced baseline RMSSD and impaired autonomic responsiveness to physiological challenges, consistent with chronic autonomic dysregulation.

Materials and Methods

Study Design and Setting: An analytical cross-sectional study was conducted at the Department of Physiology, Neurophysiology Laboratory, Stanley Medical College, Chennai, Tamil Nadu, India. The study protocol was approved by the Institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki and the principles of Good Clinical Practice.[15]

Participants: The case group comprised 30 patients with a confirmed diagnosis of TLE attending the Department of Neurology outpatient clinic. Diagnosis was established based on clinical seizure semiology consistent with temporal lobe origin and electroencephalography (EEG) showing temporal lobe-predominant interictal epileptiform discharges. The control group comprised 30 age- and gender-matched healthy volunteers without a history of neurological, cardiovascular, or metabolic disorders and without medications affecting autonomic function.

Inclusion Criteria (TLE Patients): Age 20–50 years; definite TLE diagnosis (clinical + EEG findings); seizure frequency 2–4 episodes per month; stable antiepileptic drug (AED) therapy <4 years; interictal period (≥72 hours seizure-free).

Inclusion Criteria (Controls): Age 20–50 years; no personal or family history of epilepsy or neurological disease; normotensive, nondiabetic; no chronic medication use.

Exclusion Criteria (Both Groups): Intracranial lesions (tumours, vascular malformations, haemorrhage); severe cognitive impairment; active psychiatric illness or psychotropic medication; smoking, alcohol, or substance use; cardiovascular disease, arrhythmias, or cardiac medications; pulmonary or endocrine disorders; acute illness or infection within 2 weeks before testing.

Data Collection and HRV Recording Protocol: Following written informed consent, a comprehensive history was obtained, including demographics, seizure characteristics, AED regimen, comorbidities, and medication use. Vital signs and general/cardiovascular examination were performed. Participants were acclimated to the laboratory environment (quiet, temperature-controlled at 25–28°C, dim lighting) for 30 minutes before recording.

All recordings were performed between 10:00 AM and 2:00 PM to minimize circadian effects.

Electrocardiography and Analysis: Electrocardiographic signals were recorded using RMS Polyrite D equipment (2-lead configuration, Lead II) at a 1000 Hz sampling rate and a 40–400 Hz bandpass filter. Three sequential 5-minute recordings were obtained: (1) lying position (eyes closed), supine, awake, at rest; (2) deep breathing (1 minute), slow, controlled breathing (~6 breaths/minute); (3) standing position, upright posture, 5 minutes. NN intervals (normal R-R intervals between QRS complexes) were extracted by trained operators blinded to group assignment. Ectopic beats and artifacts were manually excluded. Linear interpolation was applied.

HRV Parameters: Three time domain indices were calculated using Finland 2.1 analysis software: (1) RMSSD (ms) - root mean square of successive NN interval differences, reflecting parasympathetic (vagal) modulation of heart rate variability; (2) SDNN (ms) standard deviation of all NN intervals, measuring overall heart rate variability across the recording period; (3) NN50 (count) - number of successive NN interval pairs differing >50ms, a marker of parasympathetic activity. Short-term analysis of SDNN and RMSSD is the most reliable approach to HRV time-domain parameters, with RMSSD as the primary measure for estimating vagus-mediated changes in HRV.[16]

Statistical Analysis: Descriptive statistics (mean ± standard deviation) were calculated for all variables. Between-group differences were analysed using independent samples t-tests (two-tailed, $\alpha=0.05$). Within-group postural effects were assessed using a one-way ANOVA with Bonferroni post hoc correction to control for multiple comparisons. Statistical analysis was performed using IBM SPSS Statistics 20.0. $P<0.05$ was considered statistically significant.

Results

Participant Characteristics: Complete data were obtained from 30 TLE patients and 30 healthy controls. Groups were matched for age (TLE: 38.2±8.4 years; Controls: 39.1±7.9 years; p=0.68) and gender distribution (both 16M:14F). No adverse events occurred during testing.

TLE characteristics: mean age at seizure onset 28.4±10.2 years; duration of illness 6.3±3.1 years; seizure frequency 2–4 episodes per month. All patients were on stable monotherapy or combination AED (phenytoin, levetiracetam, or valproic acid).

HRV Measures: Lying Posture (Eyes Closed)

In the supine position at rest, TLE patients demonstrated

significantly lower RMSSD than controls. The 37.7% reduction in RMSSD in TLE patients (from 63.76 to 40.93ms) reflects substantially decreased vagal modulation of the sinoatrial node. SDNN was also reduced in TLE patients, though it did not reach Bonferroni-corrected significance.

Table 1: Time Domain HRV Indices—Lying Posture (Eyes Closed)

Parameter	Controls (mean ± SD)	TLE Patients (mean ± SD)	t-value	p-value
RMSSD (ms)	63.76 ± 35.64	40.93 ± 25.60	2.849	0.006*
SDNN (ms)	58.67 ± 38.47	41.60 ± 24.48	2.050	0.045
NN50 (count)	88.5 ± 88.65	75.97 ± 66.40	0.629	0.532

*p < 0.05 indicates statistical significance.

HRV Measures: Deep Breathing (1 Minute)

During controlled respiration, HRV indices increased in both groups, but group differences diminished. No significant differences between groups were detected. In TLE patients, RMSSD increased 94% from the resting state (40.93 to 79.48 ms), approaching control values. This suggests that acute parasympathetic stimulation (respiratory modulation) temporarily normalizes vagal tone in TLE, indicating preserved but suppressed parasympathetic capacity.

Table 2: Time Domain HRV Indices—Deep Breathing

Parameter	Controls (mean ± SD)	TLE Patients (mean ± SD)	t-value	p-value
RMSSD (ms)	81.67 ± 66.36	79.48 ± 47.13	0.147	0.883
SDNN (ms)	62.47 ± 32.86	58.67 ± 34.51	0.437	0.664
NN50 (count)	33.9 ± 29.07	25.23 ± 18.79	1.371	0.176

HRV Measures: Standing Posture

Standing—which activates baroreceptor reflexes and orthostatic cardiovascular adjustment—revealed the most pronounced group differences. All three indices were significantly reduced in TLE patients during orthostatic stress. RMSSD in TLE patients (39.87 ms) remained suppressed despite the postural challenge—failing to increase appropriately as seen in controls. This indicates impaired baroreceptor responsiveness and autonomic reserve.

Table 3: Time Domain HRV Indices—Standing Posture

Parameter	Controls (mean ± SD)	TLE Patients (mean ± SD)	t-value	p-value
RMSSD (ms)	77.63 ± 75.81	39.87 ± 28.79	2.554	0.013*
SDNN (ms)	73.8 ± 54.18	41.8 ± 22.31	2.991	0.004*
NN50 (count)	63.6 ± 61.69	30.6 ± 26.41	2.693	0.009*

*p<0.05 indicates statistical significance.

Postural Effects Within Groups (ANOVA Analysis)

One-way ANOVA with Bonferroni correction examined HRV responses to postural and respiratory changes.

Control Group: RMSSD ($F=0.786$, $p=0.461$), SDNN ($F=1.145$, $p=0.325$). Controls maintain stable HRV across conditions, demonstrating robust autonomic reserve and baroreceptor function independent of posture.

TLE Patient Group: RMSSD ($F=1.361$, $p=0.001$), SDNN ($F=5.054$, $p=0.017$). Post-hoc analysis revealed the lowest values during lying and standing postures ($p<0.05$), marked elevation during deep breathing ($p<0.001$), and a statistically significant condition-by-group interaction. TLE patients exhibit postural lability of HRV—minimal parasympathetic tone at baseline and during orthostatic challenge, but dramatic augmentation during respiratory stimulation. This pattern indicates autonomic dysregulation with severely narrowed autonomic reserve.

Discussion

Principal Findings: This study demonstrates that TLE patients exhibit significantly reduced RMSSD—a marker of vagal parasympathetic control—compared with age- and gender-matched healthy controls. This deficit is most pronounced during baseline (lying) and orthostatic (standing) conditions, indicating chronic impairment of parasympathetic tone. The dramatic preservation of HRV Deep breathing suggests that TLE patients retain latent parasympathetic capacity that can be transiently mobilized by respiratory stimulation. Still, they lack appropriate baseline and postural autonomic regulation. This finding aligns with recent observations that epileptic patients exhibit heightened sympathetic and reduced parasympathetic activity, as evidenced by increased sympathetic indices and decreased parasympathetic markers.[17]

Mechanistic Interpretation: The temporal lobe is a critical node within the Central Autonomic Network (CAN), a polysynaptic network integrating limbic afferents (amygdala, hippocampus, insula) to modulate sympathetic and parasympathetic outflow.[18] Chronic temporal lobe seizures and interictal discharges induce several pathophysiologic alterations: (1) direct CAN involvement through ictal and interictal activity directly engaging temporal autonomic structures; (2) synaptic remodelling through chronic seizures, causing structural plasticity in the autonomic neurons and reduced parasympathetic outflow; and (3) neurotransmitter dysregulation through altered

GABA, glutamate, and acetylcholine levels, reducing vagal control.

Our findings of persistent RMSSD reduction at baseline and during orthostatic stress, despite preserved capacity during respiratory stimulation, suggest a selective impairment of tonic parasympathetic outflow with relatively preserved phasic parasympathetic responsiveness to strong stimuli. Recent neuroimaging studies have demonstrated that resting connectivity patterns between the thalamus and other structures underlying HRV expression are modified in people with drug-resistant epilepsy compared to healthy controls, further supporting central autonomic dysregulation.[19]

Comparison with Existing Literature: Our findings align with and extend prior investigations of HRV in epilepsy. Recent meta-analytic evidence confirms that the reduction of SDNN and RMSSD is a hallmark finding in SUDEP patients, with a trend toward increased low-frequency and high-frequency ratios (LF/HF), suggesting sympathovagal imbalance.[8] Our cross-sectional data confirm chronic baseline parasympathetic suppression in the interictal period, and extend this observation to systematic evaluation across multiple physiological conditions. Studies have documented that interictal HRV is impaired in individuals with epilepsy, including those who are newly diagnosed and drug-naïve.[11] Our finding of differential postural effects in TLE patients versus controls validates and quantifies earlier observations, demonstrating 48.7% reduction in RMSSD from controls to TLE patients during standing—a substantial deficit with prognostic implications.

Recent prospective research (2024-2025) has shown that hyperventilation-induced RMSSD demonstrates the strongest and most significant correlation with SUDEP-7 scores, suggesting its potential utility as a SUDEP biomarker.[20] Furthermore, recent data indicate that epileptic patients (particularly refractory patients with generalized EEG findings and long duration) had reduced heart rate variability and hence impairment of parasympathetic activity with increased susceptibility for adverse outcomes.[21]

RMSSD as a SUDEP Risk Marker: SUDEP accounts for approximately 1–2 per 1000 epilepsy deaths and represents a leading cause of excess mortality in epilepsy, particularly in drug-resistant epilepsy.[22] SUDEP is the leading cause of death in drug-resistant epilepsy (DRE), with an incidence rate of 1–5 cases per 1000 patients per year.[22] Proposed SUDEP mechanisms include ictal hypoventilation, seizure-induced cardiac arrhythmias, and centrally mediated cardiovascular collapse.[3,22] A common pathophysiologic thread is impaired parasympathetic tone—the "cardiac brake" that normally prevents arrhythmia and maintains a stable heart rate. Among HRV parameters, RMSSD shows a moderately strong correlation with SUDEP-7 scores ($r = 0.578$), with hyperventilation-induced RMSSD showing the strongest correlation ($r = 0.749$).[20] This strong relationship has important prognostic value and supports the use of RMSSD modifications as a potential biomarker of SUDEP in patients with DRE.[20] Our demonstration of persistent RMSSD reduction in interictal TLE patients suggests that serial RMSSD monitoring may identify high-risk individuals requiring intensive seizure management, trigger enhanced cardiac monitoring, guide pharmacotherapy decisions, and enable lifestyle interventions to improve autonomic tone.

Clinical Implementation: The findings support routine HRV assessment in TLE clinical practice. Standard protocols from the European Society of Cardiology Task Force recommend 5-minute ECG recordings in awake and sleep states, with standardised RMSSD calculation.[23] Recent clinical guidelines specifically advocate for interictal RMSSD measurement as a SUDEP risk stratification tool in epilepsy.[24]

Given the accessibility of portable ECG devices and automated HRV analysis software, RMSSD monitoring is feasible in primary and secondary care neurology settings. A proposed clinical algorithm includes: (1) baseline RMSSD assessment at TLE diagnosis; (2) serial monitoring annually or at seizure exacerbation; (3) risk stratification with RMSSD <20 ms indicating high SUDEP risk; and (4) intervention triggers, including AED optimisation, cardiology referral for Holter monitoring and device consideration, exercise prescription to increase parasympathetic tone, seizure monitoring devices, and telemetry, and patient counselling on SUDEP risk.

Strengths and Limitations: Strengths include a well-matched case-control design with comparable age and gender distribution; objective, automated outcome measures; systematic evaluation across multiple physiological conditions; appropriate statistical methodology with Bonferroni correction for multiple comparisons; and compliance with international HRV measurement standards from the European Society of Cardiology Task Force.

Limitations include modest sample size (n=30 per group), which limits power for subgroup analyses; cross-sectional design, which precludes temporal relationships and causality establishment; lack of stratification by TLE lateralisation, which may have differential autonomic effects; absence of 24-hour Holter or sleep-state recordings, which capture only daytime, awake HRV; lack of AED dosage standardisation, as different drugs may have differential autonomic effects; absence of SUDEP-7 inventory scores or long-term cardiac outcome data, and no mechanistic biomarkers (cortisol, inflammatory markers) to elucidate pathophysiology.

Future Directions: Future studies should include larger multicentre prospective cohorts with longitudinal follow-up; stratification by TLE lateralisation and seizure focus localisation; sleep-state HRV recordings and circadian analysis; correlation with SUDEP-7 inventory and long-term SUDEP outcomes; mechanistic studies with autonomic testing (Valsalva manoeuvre, tilt table testing, heart rate recovery analysis); evaluation of AED effects on HRV; and intervention trials (seizure control optimisation, exercise programs, meditation) targeting RMSSD improvement.

Conclusion

This study documents significant impairment of RMSSD - a marker of parasympathetic cardiac autonomic control - in interictal temporal lobe epilepsy. TLE patients demonstrate reduced baseline RMSSD and attenuated baroreceptor responsiveness to orthostatic stress, indicating chronic autonomic dysregulation. However, preserved HRV capacity during respiratory stimulation suggests retained but suppressed

parasympathetic function.

RMSSD is a simple, non-invasive, reproducible measure suitable for routine clinical assessment in TLE patients. Regular RMSSD monitoring should be incorporated into comprehensive TLE management - particularly for SUDEP risk stratification, early detection of autonomic deterioration, and guiding intervention decisions.

Combined with seizure frequency monitoring and standard EEG assessment, RMSSD measurement offers a practical tool to personalise management and prevent sudden cardiac mortality in this high-risk population.

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Conflicts of interest

There are no conflicts of interest.

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