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EVALUATION OF ELECTRODE IMPEDANCE, SENSING, AND PACING THRESHOLD CHANGES AT 12 HOURS AFTER PACEMAKER IMPLANTATION

Serdar Karalük[1], Hilal Öztürk[2], Mustafa Balkaya[3], Kıvanç Yalın[4], Devrim Saribal[1]

[1]İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, Department of Biophysics, Istanbul, Türkiye.
[2]Karadeniz Technical University, Faculty of Medicine, Department of Biophysics, Trabzon, Türkiye.
[3]Cornell University, Weill Cornell Medicine, Department of Urology, New York, USA.
[4]İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, Department of Cardiology, Istanbul, Türkiye.

ORCID iD: Serdar Karalük: 0009-0005-1601-4647; Hilal Öztürk: 0000-0003-0079-5184; Mustafa Balkaya: 0000-0002-1893-8721; Kıvanç Yalın: 0000-0001-5602-0711; Devrim Saribal: 0000-0003-3301-3708.

*** Corresponding Author:** Devrim Saribal **e-mail:** devrim.saribal@iuc.edu.tr

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Abstract

Objective: The aim of this study was to evaluate the early changes in electrode impedance, sensing, and pacing threshold values within the first 12 hours after pacemaker implantation.

Methods: A total of 50 patients who underwent device implantation between June and December 2024 were included in this prospective study. Lead impedance, sensing amplitude, and pacing threshold were measured immediately after implantation and reassessed at 12 hours post-implantation. Changes between the two time points were statistically analyzed.

Results: Lead impedance values significantly decreased within the first 12 hours following implantation ($p<0.05$). In contrast, sensing amplitudes and pacing thresholds remained relatively stable, and no statistically significant changes were observed ($p>0.05$). Both atrial and ventricular leads demonstrated similar patterns in impedance reduction without compromising sensing or pacing efficacy.

Conclusion: This study demonstrates that a significant early decrease in lead impedance occurs within the first 12 hours after pacemaker implantation, while sensing and pacing thresholds remain stable. These findings confirm the short-term reliability of modern pacing and defibrillation leads and highlight the importance of early monitoring of lead parameters following device implantation.

Keywords: *Pacemaker, electrode impedance, sensing threshold, pacing threshold.*

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Introduction

Pacemakers are life-saving cardiac implantable electronic devices used to treat bradyarrhythmias and reduce sudden cardiac death.[1-3] Pacemakers maintain adequate heart rates in sinus node dysfunction or atrioventricular block, while implantable cardioverter-defibrillators (ICDs) provide both pacing support and high-voltage shock therapy for malignant tachyarrhythmias.[4,5] Both systems are externally programmable, and patients undergo regular device checks where key lead parameters are measured to ensure optimal function and early detection of problems.[5]

Key lead/device metrics are lead impedance, sensing amplitude, and capture (pacing) threshold, assessed at implant and follow-up as markers of integrity and performance.[5,6] Impedance reflects total circuit resistance (conductor, electrode–tissue interface, device connection); values within a few hundred ohms suggest stability, while abrupt falls imply insulation failure and marked rises imply conductor break/poor contact.6 Sensing amplitude is the intrinsic P-/R-wave voltage; adequate signals (e.g., >2 mV atrial, >5 mV ventricular) ensure reliable sensing and prevent under/oversensing.[3,5] Capture threshold is the minimum output that consistently depolarizes myocardium; lower thresholds conserve battery and maintain safety margins, whereas high thresholds risk loss of capture.[7,8] Thus, normal impedance, robust intrinsic signals, and low stable thresholds are essential for long-term device reliability.[6,8]

Despite careful placement and modern design, the immediate post-implantation period can show dynamic shifts in lead parameters. Within hours to days, acute tissue injury and edema at the electrode–myocardial interface may raise capture threshold and reduce P/R-wave amplitudes, changes that usually improve as the trauma resolves.[9,10] Impedance often starts higher and then declines as the electrode equilibrates with blood/interstitial fluids and polarization dissipates.[10] This early fall is typically benign and reflects contact stabilization; by contrast, abrupt deviations from the expected trend may signal complications and justify close early monitoring.[6]

Modern lead design reduces acute post-implant changes. Steroid-eluting electrodes blunt myocardial inflammation and limit early threshold rise.[8] Compared with earlier designs, they show lower acute/subacute capture thresholds and more stable early performance.[8-10] Consequently, many patients exhibit little or no threshold increase after implantation, with capture maintained without reprogramming.[10] Some variability still occurs in the first hours as the lead beds in. Even with newer technologies, transient shifts are reported—for example, a leadless pacemaker showed a day-1 rise in impedance and threshold that normalized over subsequent days.[10] Thus, the early post-implant period remains critical for verifying lead function, regardless of device or technology.[1]

Device interrogation within the first 24 hours after pacemaker implantation is standard to verify acceptable lead function and to detect early complications (e.g., dislodgement, perforation) reflected by parameter changes.[11] Early recognition of significant shifts in impedance, sensing, or capture threshold enables timely intervention and prevents adverse events.[11]

Accordingly, we systematically evaluated early (~12 h) changes in lead impedance, sensing amplitude, and capture threshold after pacemaker implantation.

The aim of this study was to quantify the changes in electrode impedance, sensed signal amplitude, and pacing threshold values approximately 12 hours after implantation, compared to the immediate post- implant measurements, in patients with newly implanted pacemakers.

Methods

Study Design and Population

This single-center prospective observational study was conducted between June and December 2024 at İstanbul University–Cerrahpaşa, Cerrahpaşa Faculty of Medicine, Department of Cardiology (Istanbul, Türkiye). Ethical approval was obtained from the institutional clinical research ethics committee (E-83045809-604.01-1083169), and written informed consent was obtained from all participants prior to enrollment. All implantation procedures were performed in a dedicated angiography and catheterization laboratory under standard clinical practice. 50 patients were included in the study.

Implant Indications and Clinical Presentation

Pacemaker implantations followed contemporary ACC/AHA/HRS and ESC criteria and were verified from the medical record by two electrophysiologists.

High-grade atrioventricular block (Mobitz II or complete) (n=27, 73%)

Typical symptoms included syncope/presyncope, dizziness, and fatigue/exertional intolerance. Objective findings comprised ECG-documented high-grade AV block (Mobitz II or complete; in some cases wide-QRS), intermittent or persistent AV conduction loss on monitoring, and exercise-provoked AV block in selected patients. These presentations align with Class I indications.

Sinus node dysfunction/chronotropic incompetence (n=10, 27%)—including tachy-brady syndrome and AF with slow ventricular response or post–AV node ablation

Common symptoms were fatigue, reduced exercise capacity, daytime somnolence, palpitations, and dyspnea. Objective findings included inappropriate resting bradycardia (e.g., <50 bpm), sinus pauses ≥3 s, and chronotropic incompetence (failure to achieve age-appropriate heart rate on formal/informal exercise testing); in tachy-brady cases, recurrent symptomatic AF and rate-control difficulties were observed. These presentations also meet Class I criteria.

Where applicable, pre-implant echocardiography was reviewed to document left ventricular ejection fraction and overall cardiac substrate. This classification is provided for clinical context only; no inferential analyses by indication were planned.

Inclusion criteria were adult patients (age ≥18 years) undergoing de novo pacemaker implantation. Patients with device revisions, lead replacements, or known coagulation disorders were excluded.

Device Type and Lead Positioning

Device type (single vs. dual chamber) and lead positions were determined per standard clinical practice by the attending electrophysiologist using fluoroscopic landmarks. In pacemaker implants, the right atrial (RA) lead was targeted to the appendage or lateral wall, and the right ventricular (RV) lead to the apex or septum; active-fixation, steroid-eluting leads were used. Lead sites (RA/RV and specific location) were recorded prospectively and were used to define subgroups for an exploratory analysis of early (~12 h) changes in impedance, sensing amplitude, and capture threshold between RV apex vs. septum.

Device Implantation Procedure

All procedures were performed under local anesthesia using standard transvenous implantation techniques. Leads were positioned under fluoroscopic guidance in the right atrium,

right ventricle, or both, depending on the type of device and the clinical indication. Final lead positioning was confirmed through intraoperative electrical measurements and fluoroscopic imaging prior to completing the implantation.[12-14]

A standardized approach was adopted in all cases by using the same brand and model of cardiac pacing leads. All atrial and ventricular pacing/defibrillation leads used in the study were bipolar, equipped with electrically active helices, and featured extendable and retractable active-fixation mechanisms.[15,16] Two specific active-fixation lead models were utilized: model 6935M-62 for ventricular defibrillation, and models 4076–52 and 5076–58 for atrial and ventricular pacing, respectively, all manufactured by Medtronic Inc. (Minneapolis, Minnesota).

For all electrical measurements, a Medtronic pacing system analyzer (model 2090) was employed. Bipolar intracardiac electrograms (EGMs) were recorded at a sweep speed of 25 mm/s at three time points: before lead fixation, during active fixation, and approximately 30 minutes after fixation, prior to connecting the leads to the pulse generator.

Threshold, impedance, and sensing values were documented immediately after device implantation and again at 12 hours post-implantation. The baseline pacing threshold was defined as the lowest voltage that consistently achieved five consecutive myocardial captures at a pulse width of 0.4 milliseconds.

Measurement of Lead Parameters

Lead parameters evaluated in this study included atrial lead impedance, ventricular lead impedance, sensing amplitudes, and pacing thresholds. All measurements were obtained using the device programmer following pacemaker implantation.

Measurements were performed at two predefined time points:

- Baseline measurement (M0 hour):

Recorded immediately after implantation, within 30 minutes post-procedure.

- Follow-up measurement (M12 hours):

Recorded 12 hours after implantation, without repositioning the patients or modifying device settings.

At each time point, impedance measurements were repeated three times consecutively for both the atrial and ventricular leads in order to minimize random measurement variability and device-related fluctuations. These repeated measurements were performed without any change in patient position or measurement settings.

The individual impedance measurements were classified according to the time point and repetition number as follows:

- Baseline (0 hour) measurements:
  Measurement 1 (M0-1)
  Measurement 2 (M0-2)
  Measurement 3 (M0-3)
- 12-hour measurements:
  Measurement 1 (M12-1)
  Measurement 2 (M12-2)
  Measurement 3 (M12-3)

In addition to impedance measurements, the following parameters were assessed at each time point:

- Sensing Amplitude:

Measured in millivolts (mV) during intrinsic cardiac activity.

- Pacing Threshold:

Determined in volts (V) using the standard threshold test with a pulse width of 0.4 milliseconds.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics Version 31.0 (IBM Corp., Armonk, NY, USA). Lead parameters, including atrial and ventricular lead impedance, sensing amplitudes, and pacing thresholds, were obtained from the same individuals at two predefined time points: baseline (M0 hour) and 12 hours after implantation (M12 hours). At each time point, measurements were repeated three times under identical conditions, and the arithmetic mean of the three repeated measurements was calculated for each subject and used as the representative value for statistical analysis.

An a priori power analysis was conducted using G*Power software, which indicated that a sample size of 50 patients was sufficient to detect within-subject differences with a 95% confidence level.

The normality of continuous variables was assessed using the Shapiro–Wilk test. Since the data showed a normal distribution, continuous variables are presented as mean±standard deviation (SD). Comparisons between baseline and 12-hour measurements were performed using the paired samples t-test, as measurements were obtained from the same subjects at two time points. A p-value of less than 0.05 was considered statistically significant for all analyses.

Results

Patient Demographics

A total of 50 patients underwent device implantation and were included in the study. The population was predominantly older, with 31 patients (62%) over 65 years of age, 18 patients (36%) between 46–65 years, and only 1 patient (2%) under 45. The mean age fell in the older adult range. There were 27 male patients (54%) and 23 female patients (46%).

These baseline characteristics were similar across the groups and provided a foundation for comparing post-implant measurements.

Lead Impedance Measurements

Both atrial and ventricular lead impedances demonstrated a significant acute decline from the time of implantation to 12 hours post-implantation.

As summarized in Table 1, the mean atrial lead impedance immediately after implantation (M0 hour) was 672.9±135.9Ω (median 676.2Ω; range 419.3–1016.3Ω). By 12 hours after implantation (M12 hours), the mean atrial impedance had decreased to 537.9±85.5Ω (median 523.0Ω; range 379.0–754.7Ω). This reduction in atrial lead impedance was statistically significant based on paired samples analysis (p<0.001).

Table 1. Comparison of mean atrial impedance measurements at baseline (M0 hour) and 12 hours (M12 hours)(Ω).

Atrial Impedance (Ω)	n	Mean±SD (Ω)	P value
M0 hour	50	673±136	<0.001*
M12 hours	50	538±85	<0.001*

SD: Standard deviation; *Statistically significant
M0 hour: Recorded immediately after implantation, within 30 minutes post-procedure.
M12 hours: Recorded 12 hours after implantation.

Similarly, the ventricular lead impedance showed a significant reduction over the same period (Table 2). The mean ventricular impedance immediately after implantation (M0 hour) was 782.7±207.6Ω (median 771.0Ω; range 418.0–1184.3Ω), which decreased to 632.5±152.1Ω (median 644.3Ω; range 382.7–963.7Ω) at 12 hours post-implantation (M12 hours).

This reduction was statistically significant ($p<0.001$). Consistent with the atrial impedance findings, ventricular impedance values exhibited an approximate 20-25% decline within the first 12 hours following implantation.

Table 2. Comparison of mean ventricular impedance measurements at baseline (M0 hour) and 12 hours (M12 hours)(Ω).

Ventriculer impedance (Ω)	n	Mean±SD (Ω)	P value
M0 hour	50	783±208	<0.001*
M12 hours	50	632±152	<0.001*

SD: Standard deviation; *Statistically significant
M0 hour: Recorded immediately after implantation, within 30 minutes post-procedure.
M12 hours: Recorded 12 hours after implantation.

No patients exhibited an out-of-range or rising impedance; all individual leads followed the overall pattern of impedance decrease. These findings indicate an expected acute drop in lead impedance between the immediate and 12-hour measurements for both pacemaker leads.

Pacing Threshold Measurements

In contrast to impedance, the pacing capture thresholds remained stable from implantation to 12 hours, with no significant differences observed. The atrial pacing threshold was 0.58±0.32 V (median 0.50 V; range 0.25–1.00 V) immediately after device implantation, and 0.48±0.26V (median 0.44 V; range 0.25–1.00 V) at the 12-hour follow-up. This slight decrease in atrial threshold was not statistically significant ($p=0.510$)($p>0.05$).

Similarly, the ventricular pacing threshold showed a minor change from 0.52±0.23 V (median 0.50 V) at implantation to 0.42±0.17V (median 0.44 V) at 12 hours, which was not significant ($p=0.347$)($p>0.05$). In both chambers, the median capture threshold remained at 0.5 V or below at both time points, and the range of threshold values (0.25–1.0 V) did not change. The minimal changes in atrial and ventricular threshold values over time. These results suggest that pacing thresholds were stable within the first 12 hours post-implant, with no evidence of threshold rise (acute capture failure) in any patient.

Sensing Values

Sensing amplitudes for both atrial and ventricular leads remained essentially unchanged between the immediate post-implant measurement and the 12-hour follow-up. The atrial sensing (P-wave) amplitude was stable, with most values falling in the 2.0-4.0 mV range at both time points. The median P-wave sensing amplitude remained around 2.8 mV, and no significant shift in the distribution of atrial sensing values was observed from baseline to 12 hours ($p>0.05$). Similarly, ventricular sensing (R-wave amplitude) remained high and showed no significant difference over time. The majority of ventricular leads had R-wave amplitudes in the 8–15 mV range initially, and these amplitudes persisted in the same range at 12 hours. In fact, no deterioration in sensing was noted: all leads maintained adequate sensing margins, and no lead demonstrated a drop to an unsafe sensing level. Statistical comparison confirmed that there was no significant change in sensing amplitudes between the immediate and 12-hour measurements ($p>0.05$ for both P-wave and R-wave comparisons). Overall, these findings indicate that sensing function was stable in the early post-implant period, with reliable detection of P-waves and R-waves maintained in all patients.

Device Type and Lead Positioning

Pacemaker: Among 37 pacemaker recipients, 31 (83.8%) received dual-chamber (DDDR) systems and 6 (16.2%) single-chamber (VVIR) systems. In dual-chamber systems, the RA lead was positioned in the appendage in 26 patients (83.9%) and on the lateral wall in 5 (16.1%). Across all pacemaker cases, the RV lead was positioned at the apex in 29 (78.4%) and at the septum in 8 (21.6%).

In patients with RV leads at the apex or septum, lead impedance decreased similarly over ~12 hours after implantation; capture thresholds and sensing (P/R-wave amplitudes) remained stable in both groups. For RA leads placed in the appendage or on the lateral wall, the same impedance decline was observed; sensing remained adequate and no clinically meaningful rise in thresholds occurred. Exploratory comparisons (RV apex vs. septum; RA appendage vs. lateral wall) showed no statistically or clinically meaningful differences. No out-of-range (very low/high) impedance values were observed and no reprogramming was required.

ICD: Among 13 ICD recipients, 5 (38.5%) received dual-chamber (DDDR-ICD) and 8 (61.5%) single-chamber (VVIR-ICD) systems. In dual-chamber ICDs, the RA lead location was the appendage in 4 patients (80%) and the lateral wall in 1 (20%). The RV lead position was the apex in 7 (53.8%) and the septum in 6 (46.2%).

In patients with RV leads at the apex or septum, impedance likewise declined early, with stable thresholds and sensing. In dual-chamber ICDs, RA leads at the appendage or lateral wall showed a similar pattern. Because of the small sample size and device-specific algorithms, no site-stratified comparative analysis was planned; findings are reported descriptively.

Discussion

This study assessed early (~12 h) changes in pacemaker lead parameters. Impedance declined as expected, while capture thresholds and sensing amplitudes remained stable. These results support early lead integrity, routine surveillance without reprogramming, and may facilitate safe early discharge.

Within 12 hours, the stability of pacing thresholds and sensing amplitudes indicates unchanged myocardial capture requirements and preserved sensing. Unchanged thresholds suggest no lead displacement or edema at the lead–tissue interface, supporting effective stimulation and expected battery longevity—especially critical for pacemaker-dependent patients. Prior work shows thresholds are typically stable in the first 24 hours, with rises appearing days later due to inflammatory/fibrotic changes (e.g., increase at 1–2 weeks).[17]

In this study, the absence of a 12-hour change in capture threshold suggests a well-controlled acute inflammatory response—likely aided by steroid-eluting leads—and provides an early "safety window" that supports expedited discharge. Contemporary practice aligns with this: most pacemaker recipients can be discharged the same day or next day, with <1% difference in early complication rates compared with overnight stays.[18] Early impedance decline is physiologic and was consistent across our cohort, reflecting known bipolar lead behavior. Mechanistically, screw fixation increases the lead-myocardium contact area, while surrounding blood/interstitial fluid completes the circuit, reducing resistance; similar immediate reductions after active fixation have been reported.[19]

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discharge; contemporary series show <1% difference in early complications between same-day and overnight strategies.[18] As expected for bipolar leads, impedance declined uniformly in the early postoperative period. Mechanistically, active fixation enlarges the lead–myocardium contact area and conductive fluids complete the circuit, lowering resistance; immediate ~50% post-fixation drops have been reported.[19] Overall, in the absence of other issues, the short-term risk of loss of capture or sensing appears low.

In this study, the absence of a 12-hour change in capture threshold suggests a well-controlled acute inflammatory response, likely aided by steroid-eluting leads. Early stability of capture and sensing provides a safety margin that supports expedited discharge; most pacemaker recipients can be discharged the same day or the next, with <1% absolute difference in early complication rates compared with overnight observation.[18] Accordingly, in patients with normally functioning devices and stable lead parameters, prolonged hospitalization solely to prevent early lead failure is seldom necessary.[18]

Early stability implies low short-term risk of loss of capture/sensing. The early impedance drop is expected for bipolar, active-fixation leads and reflects greater contact area plus conductive fluids; ~50% immediate reductions have been reported.[19]

Early impedance decline is expected: as the lead tip embeds in myocardium and insulating microbubbles dissipate, intracardiac conductivity rises, producing a peri-implant drop of ~20-25% reported for bipolar leads.[6] Our data capture this early phase within 12 hours; values remained within normal limits and showed no insulation or conductivity issues. Similar immediate decreases with active-fixation screws have been described, followed by stabilization during acute follow-up.[19] Bipolar, active-fixation, steroid-eluting leads likely underpin the stable thresholds and consistent sensing we observed: bipolar geometry localizes current and reduces artifact, while the screw helix resists early displacement (typically ~1-3%) and supports accurate sensing.[20] We noted no acute displacements or under/oversensing, implying minimal micro-dislodgement and durable lead–tissue contact. Steroid elution further tempers the acute inflammatory response, helping prevent early threshold rises, consistent with prior reports.[19]

In our study, capture thresholds did not increase in the early period; if anything, 12-hour values trended slightly lower (not significant). Steroid-eluting leads largely abolish the early "threshold peak," with Rhoden et al. showing no acute rise in steroid-releasing ventricular electrodes.[21] Thresholds remain low and stable acutely/subacutely, with only minor long-term increases—unlike nonsteroidal leads, where thresholds commonly double within 1-2 weeks before stabilizing.[17,21]

Early thresholds showed no abrupt change, suggesting that steroid-eluting electrodes help preserve myocardial excitability. Clinically, this permits programming lower output settings without compromising safety, conserving energy and prolonging battery life.

This pattern markedly lowers the risk of acute loss of capture; since steroid-eluting electrodes became standard, acute pacemaker failure from threshold rise has declined substantially.[22]

In our cohort, modern leads provided reliable support within the first 12 hours, consistent with reports of early impedance decline and stable, low thresholds. For example, in 40 patients with active-fixation leads, Canabal et al. noted an immediate impedance drop and a modest threshold improvement by 48 h (ventricular thresholds from 0.86 V at implant to 0.48 V at 48 h), after which values stabilized—attributed to steroid release and microtrauma healing.[19] Our 12-hour data capture this early phase: thresholds remained within safe limits and sensing amplitudes did not deteriorate; in the same study, R-waves were stable while P-waves decreased slightly (3.6→2.3 mV).[19] At 12 hours, neither atrial nor ventricular sensing declined. Minor early fluctuations in P- or R-wave amplitudes may occur—due to slight lead movement or autonomic tone—but are typically not clinically meaningful. Notably, De Buitleir et al. showed that with screwed tips, R-wave amplitude can acutely rise within minutes and peak around 20 minutes when the tip is stable and tissue contact is established.[7] Although our intraoperative measurements were not minute-by-minute, the absence of any decrease through 12 hours indicates that post-fixation changes equilibrated and stable sensing was achieved.

Overall, the first 12 hours after implantation are characterized by an expected fall in lead impedance alongside stable capture thresholds and sensing amplitudes, consistent with preserved lead integrity and function. Bipolar, active-fixation, steroid-eluting leads likely contribute by preventing acute threshold rises and maintaining secure myocardial contact. Clinically, these findings support safe early discharge when no other complications are present and device settings are appropriate.

Early post-implant measurements thus appear reliable for predicting short-term lead performance and can reassure both clinicians and patients. Larger, multicenter cohorts could refine post-implant evaluation and discharge protocols and assess whether discharge after only a few hours is safe in selected low-risk patients.

Conclusion

In conclusion, this study demonstrates that lead impedance decreases significantly within the first 12 hours after pacemaker implantation, while pacing thresholds and sensing amplitudes remain stable. These findings confirm the early reliability of modern pacing systems and support the feasibility of safe early discharge strategies following implantation.

Limitations

Firstly, as a single-center investigation, external validity is limited; the work would be strengthened by multicenter enrollment across diverse practice settings to better support generalizability. Secondly, the modest sample size (n=50) constrains precision and subgroup power; a prospectively powered, larger cohort with pre-specified strata would narrow confidence intervals and enable detection of clinically relevant effects. Thirdly, lead position and clinical indication were neither standardized nor stratified (e.g., RV septum vs. apex; detailed atrial location; high-grade AV block vs. sinus node dysfunction), factors that can influence acute electrical parameters; future studies should adopt explicit positioning criteria and indication-based stratification/interaction testing to isolate time-dependent effects more confidently. Finally, only short-term (12-hour) follow-up with two acute time points was performed; extending follow-up to days–weeks with repeated-measures analyses and adding patient-centered endpoints would more directly link early parameter dynamics to sustained lead performance and clinical outcomes.

Conflict of Interest

The authors declare no conflicts of interest.

Compliance of Ethical Statement

Approval for the study was obtained from the Clinical Research Ethics Committee of İstanbul University-Cerrahpaşa (Approval No: E-83045809-604.01-1083169, Date: 03.09.2024). Written informed consent was obtained from all participants and/or their legal guardians prior to study enrollment.

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Author Contributions
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