BRIEF REPORT

Mechanisms of damage related to ICD and pacemaker lead interaction

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Increasing acceptance of left bundle branch area pacing for cardiac resynchronization therapy raises the possibility for multiple leads in the right ventricle. The interaction between 2 leads is one of the few mechanisms that may cause damage to both insulation and conductors and could ultimately cause loss of therapy.[1,2] The general guidance is to avoid contact between leads, which can be challenging. However, there is limited information about the time progression of damage and factors that may influence which lead is damaged. Previous engineering reports have focused on wear from pulse generator material and from internal cable conductor components.[3–5] Studies reporting mechanisms of lead-lead interaction causing insulation and conductor damage are lacking. Although there are published reports of the lead bending conditions associated with lead fracture, there are no reports of in vivo lead-lead interaction conditions, namely, relative sliding distance and contact force. Therefore, this study presents results from a series of bench tests aimed at reproducing and accelerating insulation and conductor damage due to lead-lead interaction.

The lead samples were Medtronic SelectSecure 3830 (Medtronic, Dublin, Ireland) (pacing lead, polyurethane insulated) and Sprint Quattro 6947 (Medtronic) (implantable cardioverter-defibrillator [ICD] lead, polyurethane insulated). Testing was motivated by the observations of a case where the pacing lead was observed to have substantial damage after interacting with the defibrillation lead for 21 months, or approximately 55 million heartbeats.[2] The pacing lead in that case was returned to the manufacturer for analysis and wear scar characterization, providing a basis for comparison with in vitro testing. The in vitro tests were developed to apply repetitive sliding motion to the leads, assuming that a single heartbeat corresponds to a single sliding cycle.

Because the in vivo loading conditions were unknown, initial test conditions were selected to target the lowest, yet controllable load on available in vitro equipment (test 1, Rtec Universal Tribometer; Online Supplemental Figure 1 and Online Supplemental Video 1). Leads were mounted perpendicular to each other in opposing fixtures, 1 to a load cell on a vertical stage and 1 to a horizontally reciprocating table with a water bath. Applied contact force was nominally 3 N, controlled by the equipment software. The sliding distance was 10 mm/cycle, estimated from the wear scar on the returned pacing lead. The sliding direction was for the pacing lead to move in the axial direction of the defibrillation lead, across the windings of the defibrillation electrode. The cyclic frequency was 4 cycles/s, selected to accelerate the test (4× relative to 60 beats/min) as much as possible within the equipment capability. Visual inspection of both lead samples was performed approximately daily, and the test stopped when gross conductor fracture or insulation breach occurred to either lead. One sample was tested.

In the test 1 configuration, fracture of the defibrillation electrode conductor and breach of the ICD lead insulation was observed after approximately 1 million cycles (equating to 9 days in vivo). At this time point, the outer polyurethane insulation of the pacing lead decreased in thickness but not entirely breached. The damage to the ICD lead was likely similar to the scenario presented by Sato et al,[1] in which the shock coil was completely fractured if that case had been interrupted at an earlier stage (Figure 1, top right). However, the number of in vitro sliding cycles was likely much lower than the number required in vivo for a clinical presentation of lead malfunction, usually tens to hundreds of million, equivalent to months or years.

Based on the results of test 1, subsequent test conditions were developed using novel equipment (test 2; Online Supplemental Figure 2 and Online Supplemental Video 2). The aim of this test was to produce more wear on the pacing lead, match the number of contact cycles closer to the Mahajan report, and to increase throughput. This required reducing the contact force and increasing the frequency of contact events. In test 2, custom fixturing was developed to mount 4 lead segments with defibrillation electrodes to a cylinder attached to a rotary motor operating at 27 rpm. The apparatus produced 108 contact events/s, each approximately 10 mm, in a single sliding direction (vs bidirectional sliding in test 1).

KEYWORDS Cardiac lead; Insulation damage; Conductor fracture; Lead-lead interaction; Left bundle area pacing; Conduction fracture (Heart Rhythm O2 2023;4:820–822)

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KEY FINDINGS

- In vitro lead-lead interaction was able to produce damage to either or both pace-sense and defibrillator leads, including complete fracture of the conductor cables.
- Adequate distance needs to be maintained in the right ventricle to avoid lead-lead interaction when using the left bundle area pacing lead and defibrillator lead for cardiac resynchronization therapy defibrillator therapy.
- If one lead is damaged, there is an increased likelihood of damage to the other lead.

Pacing lead samples were mounted perpendicular to the axis of the cylinder holding the defibrillation coils, using suspended weights to provide tension on the pacing lead. The amount of interference between the pacing lead and defibrillation electrode coils provided contact force. Peak normal force for each contact event ranged from 0.03 to 0.06 N, selected to reduce the wear rate by at least an order of magnitude from that in test 1. Rotation of the cylinder produced motion of the defibrillation electrode along the axis of the pacing lead, parallel to the windings of the defibrillation electrode. The 4 defibrillation electrode samples each contacted the pacing lead once per revolution. The apparatus was suspended in a room temperature water bath. The test stations were replicated to provide capacity for 6 samples at a time. Visual inspections were performed approximately daily, and the test stopped when gross conductor fracture or insulation breach occurred to the pacing lead.

In the test 2 configuration, initial breach of the insulation was observed as early as 15 million cycles (n = 2 of 6) and major insulation breach along with flattening of the outer conductor coils was observed after 30 million cycles (n = 6 of 6). Complete fracture and substantial damage to the pacing lead was observed after 40 million cycles (n = 4 of 6), with accompanying damage initiated to the ICD defibrillation electrode coils. The damage to the pacing lead was similar to the findings of Mahajan et al[2] (Figure 1, bottom right; Online Supplemental Figure 3).

This study found that in vitro lead-lead interaction was able to produce damage to either or both leads, including complete fracture of pace-sense and defibrillation conductors. Different lead motion patterns could damage either the pacing lead or the ICD lead. This study highlights the importance of returned product analysis to determine the root cause of malfunctions. Limitations of this study include the following. These experiments were conducted to demonstrate the feasibility of reproducing clinical failure mechanisms and were not powered for hypothesis testing. The results are specific to the interaction between the outer insulation of a polyurethane pacing lead and the defibrillation electrode of an ICD lead. Electrical parameters were not measured, so the time course of clinical electrical signals related to progressive lead damage is unknown. The results presented here may not apply when other materials are in contact. Actual in vivo loading conditions for lead-lead contact have not been measured; therefore, the in vitro acceleration factor is unknown. These experiments used linear motion in a single direction. In vivo motion is 3-dimensional and may include relative rotational components. Although the actual conditions are unknown, the similarity between in vitro and clinical damage provides insight into potential ranges for in vivo forces, an approach discussed by Placette et al.[5] Experiments in a water bath do not reproduce the fluid and biochemical environment in vivo, which could result in unknown changes to the wear mechanisms. Finally, only 2 lead models were studied; other leads may respond differently.

In conclusion, (1) if one lead is known to be damaged, there is an increased likelihood of damage to the other lead; and (2) with increasing adoption of left bundle branch area pacing for

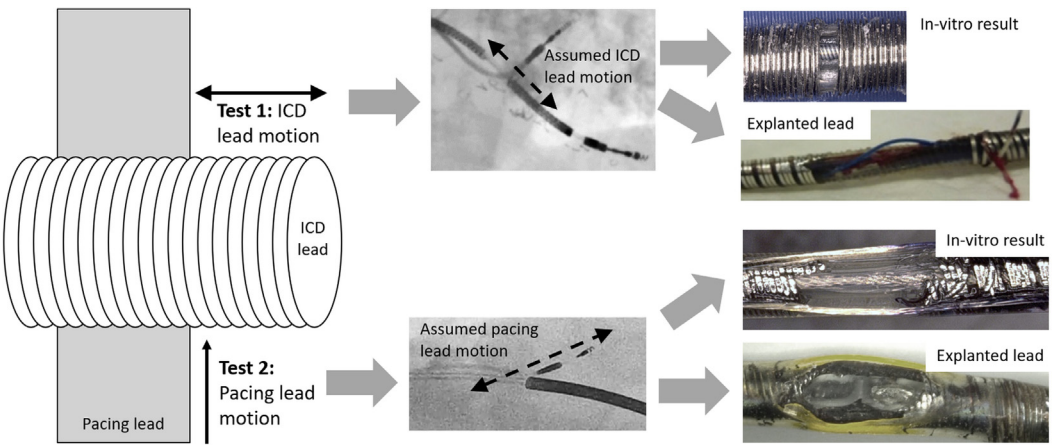

Figure 1 Bench test configuration with the implantable cardioverter-defibrillator (ICD) lead (test 1; **top**) or pacing lead (test 2; **bottom**) in motion (**left**), in vivo lead interaction and assumed motion (**middle**), and in vitro and explanted lead results (**right**). Radiographic and explanted lead images were modified from references 1 and 2, used with permission.

cardiac resynchronization therapy defibrillator, it is important to avoid lead-lead interaction by maintaining an adequate distance between the leads and confirming it in left anterior oblique and right anterior oblique projections at the time of implantation. Further research is necessary to learn more about the in vivo mechanisms of lead-lead interaction and the need for lead design/optimization.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hroo.2023.11.009.

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