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Simulation of Mechanical Environment in Active Lead Fixation: Effect of Fixation Helix Size

The risk of myocardial penetration due to active-fixation screw-in type pacing leads has been reported to increase as the helix electrodes become smaller. In order to understand the contributing factors for lead penetration, we conducted finite element analyses of acute myocardial micro-damage induced by a pacemaker lead screw-in helix electrode. We compared the propensity for myocardial micro-damage of seven lead designs including a baseline model, three modified designs with various helix wire cross-sectional diameters, and three modified designs with different helix diameters. The comparisons show that electrodes with a smaller helix wire diameter cause more severe micro-damage to the myocardium in the early stage. The damage severity, represented by the volume of failed elements, is roughly the same in the middle stage, whereas in the later stage the larger helix wire diameter generally causes more severe damage. The onset of myocardial damage is not significantly affected by the helix diameter. As the helix diameter increases, however, the extent of myocardial damage increases accordingly. The present findings identified several of the major risk factors for myocardial damage whose consideration for lead use and design might improve acute and chronic lead performance. [DOI: 10.1115/1.4004288]

Keywords: pacing lead, defibrillation lead, cardiac penetration, finite element analysis

1 Introduction

The active fixation of pacemaker and implantable cardioverter-defibrillator (ICD) leads, such as screw-in leads in the right atrium and right ventricle, has gained more popularity due to the low dis-

lodgement rate, versatility in electrode positioning, and isodiametricity [1]. Despite the performance improvement of screw-in leads, serious mechanical complications such as cardiac penetration have been reported. Penetration might produce pericardial pain or no symptoms at all. Additionally, it might cause intermittent or complete failure of cardiac pacing and result in diaphragmatic stimulation. If penetration does occur, it is usually at the time of implantation or within the first 24 h [2]. The reported rate of lead penetration in either the right atrium or the right ventricle is 0.1% to 0.8% for pacemakers and 0.6% to 5.2% in ICDs [1,3,4]. The risk of myocardial penetration due to active-fixation

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Fig. 1 Geometric and FE models of the myocardium and the helix electrode: (a) the myocardium with the electrode screwed in; (b) the helix diameter, D , and helix wire diameter, d , of the electrode; (c) the FE mesh of the myocardium; and (d) the interface between the center and outer cylindrical meshes

screw-in type leads has been reported to increase as the active fixation helixes are reduced in size with progressively smaller lead designs [5,6].

In order to reduce the incidence of penetration with new lead designs, a thorough understanding of the mechanism of penetration from a mechanics perspective is inevitably needed. In this regard, both experimental mechanical testing, *ex vivo* and *in vivo*, and computational modeling play essential roles in optimizing lead performance. Although lead penetration has gained some attention from the biomechanics community in recent years [7–9], research efforts in this direction are still scarce. In the current work, we focused on finite element analysis in order to investigate the mechanisms of acute myocardial micro-damage induced by screw-in leads using various helical electrode designs of different sizes. The findings herein might shed light on the underlying factors in the increased penetration incidences with smaller leads, and they might provide insight for new lead designs.

2 Methods

2.1 Geometric and Finite Element Models. Figure 1(b) illustrates the geometric definition of the helix electrode. D is defined as the helix diameter of the electrode, that is, the diameter of the center line helix as it is projected onto the x - y plane. d is the helix wire cross-sectional diameter, and H is the pitch (the distance in the z -direction of one full turn) of the center line of the electrode. Seven electrodes were modeled, i.e., the baseline model with a helix wire diameter of 0.3 mm and helix diameter of 0.7 mm, three modified designs with smaller helix diameters (i.e., $d = 0.15$, 0.2, and 0.25 mm, respectively) and a constant helix diameter, and three modified designs with larger helix diameters (i.e., $D = 0.8$, 0.9, and 1.0 mm, respectively) and a constant helix wire diameter. All designs had the same pitch, $H = 1.0$ mm. Table 1 lists the geometric parameters for the seven lead designs.

For simplicity, the myocardial wall was modeled as a cylinder with a diameter of 2.4 mm and a thickness of 2.0 mm (see Fig. 1(a)). The geometric surface model of each helix electrode was created and then meshed with hexahedral solid elements using TRUEGRID (XYZ Scientific Applications, Inc., Livermore, CA). In this study, we assumed an already screwed-in electrode and considered the postimplantation interaction between the electrode and the myocardium without considering the screw-in process. The lateral surface of the myocardium was clamped, and the electrode was given a prescribed displacement (i.e., $d_z = 1.5$ mm) into the myocardium along its center axial direction, which was the global z -direction. The LS-DYNA explicit formulation (Livermore Software Technology Corporation, Livermore, CA) was utilized to simulate the process of moving the electrode against the myocardial wall. The explicit solution scheme is based on time integration using the central difference method. It is usually utilized to solve short-duration transient problems. However, it also can be used to solve quasi-static problems as long as the kinetic energy is very small relative to the internal energy ($<10\%$). The usage of the explicit scheme in the current work is the latter. The total duration was 0.1 s. It should be noted that this time is not the actual time scale of lead penetration.

Table 1 Geometric parameters of the seven designs of the helix electrode: the baseline model (Design 1), and the modified designs with various helix diameters and helix wire diameters

Design ID	1	2	3	4	5	6	7
Helix diameter D (mm)	0.7	0.7	0.7	0.7	0.8	0.9	1.0
Helix wire diameter d (mm)	0.3	0.25	0.2	0.15	0.3	0.3	0.3

2.2 Contact Algorithm. The contact between the helix electrode and the myocardium was modeled using a 3D penalty-type contact algorithm with erosion (*CONTACT_ERODING_SURFACE_TO_SURFACE_ID) in LS-DYNA. This algorithm allows the contact surface to be updated as the initial element facets erode, which causes the original contact surface to vanish. The soft constraint penalty formulation was applied to this contact due to the large difference in stiffness between the electrode and the myocardium. In LS-DYNA, the “soft” formulation is implemented to treat the contact between bodies with dissimilar material properties [10]. The cardiac tissue was defined as the master entity, and the surface of the helix electrode was designated as the slave entity.

Due to the complexity of the meshing scheme at the center of the myocardial region, meshes at the interfaces between the big cylinder and the small cylinder did not match (see Figs. 1(c) and 1(d)). Therefore, a constraint-based tied contact (*CONTACT_TIED_SURFACE_TO_SURFACE_ID) was adopted at these interfaces, which allows these two regions to act as one continuous material.

2.3 Failure Criteria. Because there is no built-in failure option in the constitutive model for myocardium (*MAT_HEART_TISSUE), the micro-damage of the myocardium was implemented by using the *MAT_ADD_EROSION option in LS-DYNA. The failure criterion was based on the maximum principal stress; i.e., when the principal Cauchy stress in an element exceeded its highest allowable value ($\sigma_{\max}$), the element was deleted. The failure stress $\sigma_{\max}$ was set to $20 \text{ g/mm}^2 \approx 196 \text{ kPa}$ [11].

2.4 Constitutive Models. The helix electrode is typically made of platinum alloy, which is much stiffer than heart tissue. Consequently, the deformation during lead implantation and myocardial micro-damage (or penetration) is negligible. Therefore, it is reasonable to model the electrode as a rigid material. The density of the electrode was set to $20,000 \text{ kg/m}^3$, the Young’s modulus was 168 GPa , and the Poisson’s ratio was 0.3 . The Young’s modulus and Poisson’s ratio should be used for rigid materials in order to determine the sliding interface parameters if the rigid body interacts with a contact definition [12].

The passive mechanical behavior of the myocardium was modeled as a nearly incompressible transversely isotropic hyperelastic strain energy function proposed by Guccione et al. [13], namely,

$$W = \frac{C}{2} (\exp[Q] - 1) \quad (1a)$$

$$Q = b_f E_{11}^2 + b_t (E_{22}^2 + E_{33}^2 + E_{23}^2 + E_{32}^2) + b_{fs} (E_{12}^2 + E_{21}^2 + E_{13}^2 + E_{31}^2) \quad (1b)$$

where C is a stresslike material constant; b_f , b_t , and b_{fs} are dimensionless material constants; E_{11} is the Green–Lagrange strain in the fiber direction; E_{22} is the strain in the cross-fiber in-plane direction; E_{33} is the strain in the out-of-plane direction; and the rest are shear components of the strain tensor. The following material constants were adopted from Wenk et al. [14] for diastolic myocardial passive elastic properties, i.e., $C = 0.88 \text{ kPa}$, $b_f = 18.5$, $b_t = 3.58$, and $b_{fs} = 1.63$. The bulk modulus was set to 10 MPa . The fiber direction was assumed to align with the x -direction, which is tangent to the plane of the myocardial block.

The incompressibility condition requires the decoupling of the strain energy function W into dilatational and deviatoric parts, namely,

$$W(\mathbf{C}) = U(J) + \bar{W}(\bar{\mathbf{C}}) \quad (2)$$

where $\mathbf{C} = \mathbf{F}^T \mathbf{F}$ is the right Cauchy–Green deformation tensor, $\mathbf{F}$ is the deformation gradient, J is the determinant of $\mathbf{F}$, $\bar{\mathbf{C}}$ is the deviatoric part of $\mathbf{C}$, $\bar{W}(\bar{\mathbf{C}})$ is the deviatoric contribution of W , and $U(J)$ is the dilatational contribution.

The second Piola–Kirchhoff stress tensor $\mathbf{S}$ is given by

$$\mathbf{S} = pJ\mathbf{C}^{-1} + 2J^{-2/3} \text{Dev} \left(\frac{\partial \bar{W}}{\partial \bar{\mathbf{C}}} \right) \quad (3a)$$

where p is the hydrostatic pressure acting as a Lagrange multiplier to enforce the incompressibility condition, and $\text{Dev}(\bullet)$ is the deviatoric projection operator given by

$$\text{Dev}(\bullet) = (\bullet) - \frac{1}{3} [(\bullet) : \mathbf{C}] \mathbf{C}^{-1} \quad (3b)$$

3 Results

Figure 2 shows a cutaway view of the undeformed reference configuration, a micro-damaged configuration, and the penetrated configuration for the case of baseline model ($D = 0.7 \text{ mm}$ and $d = 0.3 \text{ mm}$). The micro-damage process was similar for the other cases, which are not shown for brevity. Myocardial micro-damage was found to occur mostly at the front surface of the helix tip, especially at the initialization of the micro-damage, as indicated by the failure process of the elements. After the elements failed, the eroding-type contact allowed the contact interface between the

Fig. 2 Cutaway view of the (a) undeformed reference configuration, (b) micro-damaged configuration, and (c) penetrated configuration for the case of baseline model, i.e., $D = 0.7 \text{ mm}$ and $d = 0.3 \text{ mm}$

Fig. 3 Effect of the helix wire diameter d on ((a) and (b)) the failed volume of the myocardium and ((c) and (d)) the contact force between the electrode and the myocardium, keeping the helix diameter the same, i.e., $D = 0.7$ mm. The figures on the left have a truncated abscissa for the sake of clarity.

electrode and the myocardium to be updated so the new elements at the contact surface could bear load. The extent of myocardial micro-damage can be well described by the volume of the failed elements. Figure 3(a) shows the effect of the helix wire diameter on the failed volume at a constant helix diameter ($D = 0.7$ mm). The abscissa, denoting time, is truncated at 0.04 s for clarity of comparison. Abrupt increases in the failed volume represent the rapid accumulation of myocardial micro-damage induced by the movement of the electrode against the myocardium. The micro-damage initialization time generally decreased with a decrease in the helix wire diameter.

By observing the failed volume history in Fig. 3(b), it was found that the myocardial micro-damage process could be divided into three stages with a different damage severity for each helix wire diameter. In the early stage, where the micro-damage was initialized, electrodes with a smaller helix wire diameter caused earlier and more severe micro-damage to the myocardium, as mentioned earlier. The middle stage was characterized by similar micro-damage severities. In the later stage, a larger helix wire diameter generally caused more severe myocardial micro-damage.

Figures 3(c) and 3(d) show the temporal change of the contact force between the electrode and the myocardium for the four cases with various helix wire diameters. The contact force did not vary significantly for the four lead designs before myocardial micro-damage was initialized. Due to the difference in the contact area, a different stress was induced in the elements at the lead tip. Therefore, the electrode with a smaller helix wire diameter caused

relatively earlier micro-damage initialization. Once the elements failed in front of the electrode tip, there was not sufficient external force to sustain the current deformation of the myocardium. Thus, the elastic deformation of the myocardium had a trend to recover toward its reference configuration. The interaction between the myocardium and the electrode is visually observed through the helical (rotational) motion of the electrode about its center axis until the tip comes in contact with previously unengaged myocardium, at which point the contact force drops to its local minimum and then increases gradually again. In Fig. 3(d), this phenomenon is clearly demonstrated by the drastic decrease of the contact force for the four designs. A typical distribution of the first (maximum) principal Almansi strain is shown in Fig. 4. The highest strain was about 30%, near the micro-damaged region around the electrode.

Figure 5 shows the effect of helix diameter on the failed volume at a constant helix wire diameter ($d = 0.3$ mm). The initialization of myocardial micro-damage was not affected significantly by the helix diameter. The micro-damage extent at the end of the simulation, however, was relatively higher for the larger helix diameter. This was due to the relatively higher energy dissipated by the failure of elements for larger electrodes.

4 Discussion

Clinical studies have reported that the risk of myocardial penetration due to active-fixation screw-in type leads has increased as the helical electrodes have become smaller [5,6]. In order to better

Fig. 4 Contour of the first (maximum) principal Almansi strain in the penetrated myocardium

understand the underlying mechanisms, experimental and numerical studies from a biomechanics perspective are inevitably necessary, based on realistic geometric features of the helical electrode, the material properties of the myocardium, and the interaction between the electrode and the myocardium. In this study, we conducted finite element analyses in order to understand the realistic interaction between the helix electrode of screw-in leads and the myocardium, so as to identify the effect of the helix wire diameter and helix diameter of the electrode on the propensity for myocardial micro-damage. In particular, we adopted a realistic constitutive model for the myocardium and utilized an erosion-type contact algorithm.

The simulation results showed that the helix wire diameter plays a predominant role in determining the onset of myocardial micro-damage; i.e., the smaller the helix wire diameter, the earlier the onset of myocardial micro-damage. Moreover, myocardial

micro-damage was found to occur mostly at the front surface of the helix tip, especially at the initialization of the micro-damage, which renders a helical motion of the electrode. The eroding-type contact algorithm allowed the contact interface between the electrode and the myocardium to be updated after elements failed so that the new elements at the contact surface could bear load. The effect of the helix diameter on the initialization of myocardial micro-damage, however, was not as significant. Nevertheless, the micro-damage extent was higher with larger helix diameters, because more energy needs to be dissipated by the failed elements.

In order to model the process of the micro-damage of the myocardium, experimental data of the material strength or rupture strain of the myocardium are inevitably necessary in order to determine whether the elements will fail according to failure criteria based on stress or strain. Unfortunately, these data are currently scarce. To the best of our knowledge, the most complete experimental study on the failure properties of the myocardium is provided by Przyklenk et al. [11]. The authors investigated the effect of myocyte necrosis on the strength, strain, and stiffness of isolated myocardial strips using canine models. No significant differences between normal and necrotic myocardium in terms of tensile strength and strain at rupture were found. They reported a mean tensile strength of 21.3 ± 3.3 and 21.3 ± 3.2 g/mm² for the endocardium and the epicardium, respectively, which is significantly greater than those of the midmyocardium and subepicardium (4.0 ± 0.3 and 5.0 ± 0.5 g/mm²). However, the strain at rupture did not vary significantly among the myocardial layers. A mean rupture strain of 0.46 ± 0.02 for epicardium and 0.53 ± 0.03 for endocardium was measured. In the current numerical study, we adopted their data (~ 20 g/mm²) on endocardium and epicardium for myocardial strength. Data on the myocardial strength of healthy and diseased hearts are extremely valuable in complementing the understanding of myocardial damage mechanisms.

Experimental and numerical studies of lead penetration have only recently been investigated. Investigations into lead penetration have been focused mainly on clinical case studies and trials. From a mechanics point of view, Gasser et al. [7] studied the penetration property of cardiac tissue by penetrating a cylindrical rod into the tissue. This work provided valuable data for lead

Fig. 5 Effect of the helix diameter D on the failed volume of the myocardium. The helix wire diameter was kept constant, i.e., $d = 0.3$ mm.

penetration. Zhang et al. [8] conducted in vivo experiments to measure the screw-in torque and pull-out force for various lead designs and various implant locations. Fang et al. [9] conducted numerical simulations of the screw-in process of the helical electrode using the finite element method and found that the best angle to screw in at is perpendicular to the myocardium surface. They, however, did not take into account the anisotropy of the myocardium.

The current work has the following limitations. First, the failure properties of the myocardium in and across the cardiac fiber direction were not discriminated, as the failure mechanism of myocardium is not completely understood. Generally speaking, the weak bond between the cardiac fibers might cause the myocardium to fail in the mode of fiber splitting rather than fiber dissection if a blunt object penetrates into the myocardium. Further experimental studies, however, are needed in order to determine whether splitting or dissection is the primary failure mode when myocardium is subjected to penetration by sharp objects such as lead screws. In order to simulate the splitting mode failure of myocardium, a fracture model based on cohesive theories [15] should be utilized.

Moreover, due to limited computational capability, the elements in the myocardium were controlled at a relatively large size in order to reduce the computational time. The outcome of this compromise was that the penetration process exhibited a jumping pattern, which was regulated by the erosion algorithm. One could, however, utilize a higher mesh density in order to realize a smoother penetration simulation that is closer to reality. We believe that the primary findings will still be valid if a denser mesh is used.

Although it was found that smaller leads are more prone to penetration, there are other factors that might dictate acute myocardial penetration, e.g., the geometric and structural properties of the lead body, the mechanical environment of the lead implantation including the exertion of screw-in force and torque, the periodic motion of the heart, pre-existing damages induced during the screw-in process, the implantation location, and the mechanical properties of the myocardium. Therefore, in order to better clarify the acute penetration mechanism, combined experimental and computational studies on the various aforementioned factors are needed.

In summary, we found that smaller leads might be more vulnerable to penetration by considering a realistic interaction between the helical electrode and the myocardium. This finding might help clinicians choose more appropriate leads in order to reduce the possibility of lead penetration postimplantation. Moreover, these findings might provide insight for lead engineers in terms of new lead designs. Needless to say, it is necessary to conduct more complete experimental studies on the failure mechanisms of the

myocardium and to integrate the outcomes with numerical simulations in order to model electrode–myocardium interactions and predict lead penetration in a more realistic fashion.

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