An 80-year-old male, with a history of coronary artery disease (coronary artery bypass graft surgery) and bradycardia syndrome was referred for a recurring pacemaker pocket infection of his dual chamber pacemaker system. The infected system was explanted and the leads extracted using laser, followed by antibiotic treatment. As the patient only required infrequent pacing, the choice for a single chamber pacing system was made. In January 2013, a leadless pacemaker (LP; Nanostim, St. Jude Medical, MN) was successfully implanted in the right ventricular (RV) apex. Four months postimplant, a transesophageal echocardiographic examination reconfirmed correct deployment of the LP in the RV apex, and the distal end of the LP moving freely during myocardial contraction (Figure 1A and 1B; Data Supplement), touching the lateral RV wall but not interfering with the RV septum (Movie in the Data Supplement). The LP did not interfere with the RV outflow tract, pulmonary valve or tricuspid valve, and no mobile structures were identified on the LP. The patient was monitored in the pacemaker outpatient clinic until he died because of an irreversible cholangiocarcinoma 19 months postimplant, in August 2014. The LP performance was stable throughout the entire follow-up, no device-related adverse events were observed and there was no occurrence of pacemaker syndrome. The patient consented before his death for autopsy of the heart and lungs, and a postmortem computed tomographic examination reconfirmed the position of the LP in the RV apex, and the distal end of the LP moving freely during myocardial contraction (Figure 1A and 1B; Data Supplement). A three-dimensional reconstruction of the computed tomographic scan is shown in Figure 2A and 2B. A three-dimensional reconstruction of the computed tomographic scan is shown in Figure 2A and Data Supplement.

Autopsy
At autopsy, the heart showed hypertrophy and slight dilatation of both ventricles (heart weight: 605 g). The coronary artery bypass grafts appeared calcified but patent. The tricuspid valve showed retraction and thickening of leaflet edges at multiple sites potentially caused by previous implantation and removal of the conventional pacemaker leads (Figure 3A). The LP was located near the apex of the RV between trabeculae, adjacent to a layer of endocardial thickening, but not penetrating the myocardium (Figure 3A and 3C). The LP was covered by a thin capsule (<1 mm thick), which was firm near the base of the LP and fragile at the top (Figure 3B). Adjacent to the LP, the septal and free wall myocardium showed several focal fibroelastic patches, interpreted as lesions caused by the friction of consecutive pacemaker leads and LP touching the free RV wall.

Microscopic analysis revealed that the LP was covered for ≈60% in a thin fibrous capsule containing many α-smooth muscle actin immunostainable myofibroblasts. There was no endothelial lining present, which was confirmed by immunohistochemical analysis (CD34−), and no inflammation or foreign body reaction was observed. The distal part of the LP was covered by a fragile, thin layer composed of fibrin/platelet thrombus (Figure 3D and 3E). Throughout the thin capsule and the thrombus material large amounts of iron depositions were observed, suggesting that the proximal fibrocellular part of the capsule in fact represents organized/stabilized thrombus material. The lungs (left, 375 g and right, 305 g) showed no evidence of thromboembolism.

Discussion
Although (sub)acute LP retrieval has shown to be feasible, the occurrence of pacemaker encapsulation in humans and the possibility of long-term retrieval after years of implantation remains unknown. We report the first postmortem histological evaluation of an intermediate-term LP implant that showed partial, ongoing myofibrocellular encapsulation of the LP. Further research is required to assess long-term LP encapsulation and its impact on LP retrievability. Full encapsulation of the device might be a factor that complicates device retrieval. At the same time, encapsulation may lower the risk for device infection in the presence of bacteremia, in comparison with conventional transvenous pacemaker systems.

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Disclosures

None.

References


Key Words: autopsy ■ heart ■ pacemaker ■ pathology
Figure 3. Postmortem pathohistological examination of heart and leadless pacemaker (LP) A, Gross anatomy of the right ventricle showing the LP near the apex between the trabeculae. B, LP with thin capsule, fibrous at the base and fibrin/platelet material at the top. The left-sided part was attached to the endocardium. C, Fibrous endocardial thickening of right ventricle at the site of implantation of LP. Myocardium (*) is intact. D and E, Cross-sectional view of the capsule. The firm part of the capsule is fibrocellular, and contains many actin positive myofibroblasts. There is a gradual transition to the fragile part, which contains only blood elements (D, hematoxylin and eosin stain; E, smooth muscle actin-1 immunostain).
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Data Supplement (unedited) at:
http://circep.ahajournals.org/content/suppl/2016/02/04/CIRCEP.115.003101.DC1
SUPPLEMENTAL MATERIAL

Movie 1A. Transesophageal echocardiogram (TEE) shows leadless pacemaker in right ventricular apex.

Movie 1B. Transgastric view of leadless pacemaker in relation to the tricuspid valve.

Movie 2. Post-mortem computed tomography scan with three dimensional (3D) reconstruction of leadless pacemaker implanted in right ventricular apex.