How to temporarily pace a pacemaker-dependent patient after lead extraction for device infection?

We read with interest the work by Pecha et al.\(^1\) recently published in the Journal. The authors aimed to evaluate a new option of temporary pacing (TP) in pacemaker-(PM)-dependent patients undergoing lead extraction (LE) for a cardiac implantable electronic device (CIED) infection, that was used to delay re-implantation and improve safety and freedom from re-infection in follow-up.

An active-fixation lead (AFL) was ipsilaterally implanted and connected to an externalized PM, in this way keeping the infected extraction side for TP and preserving the site of definitive device re-implantation. They did not observe infection recurrences, nor lead dislocation after a mean follow-up time of 21.1 months in spite of the long duration of TP (median 12.7 days, range 6–24).

Similar good results were found in another recent experience,\(^2\) in which the authors used an AFL for TP after LE, choosing immediately the contralateral side (internal jugular vein) to change the site of pacing since the early post-extraction phases.

In both experiences, the prolongation of anti-biotic therapy (AT) using a reliable TP was considered the cornerstone for a proper healing. Although taking opposite directions, both strategies had a clear rationale. The first choice (TP from the ipsilateral, rather than contralateral side) is preferred in our institution, but we do not have the answer about the best strategy. We do not even know if one strategy fits the other one.

Secondly, if the AFL has been implanted after LE, did the authors use the LE sheath as a channel to introduce the guidewire or did they perform a new subclavian puncture? In terms of re-infection risk, the second approach is certainly safer than using the ‘retaining guidewire technique’, especially in the case of septic thromboflebitis, which is often very difficult to diagnose.

Then, a median of AT duration of 16.5 days (range 6–42) and a median of duration of TP of 12.7 days (range 6–24) is reported: we wonder if the AT was started only at the moment of LE. In our opinion, one of the cornerstones for infection eradication is a proper duration of the pre-operative AT. Separate data about AT duration in the setting of local and systemic infection would be interesting. Moreover, did they start with cephalosporin even in cases of sepsis and endocarditis (particularly numerous in this cohort)? Which kind of cephalosporin did they use? The question is of interest because it is known that some broad-spectrum cephalosporins (as those they report to have used), are less effective in staphylococcal infections, that represent particularly numerous in this cohort.

In conclusion, the best way to manage TP after LE for CIED infection in PM-dependent patients remains a subject of interesting debate and research, given the high risk of re-infection of this kind of patients.

Conflict of interest: none declared.

References

Lack of efficacy of radiofrequency catheter ablation in Andersen–Tawil syndrome: are we targeting the right spot?

Andersen–Tawil syndrome (ATS) is associated with a high arrhythmic burden as demonstrated recently by Delannoy et al.\(^1\) in this Journal. Almost all subjects (91%) had very frequent ventricular arrhythmias (> 10 000 premature ventricular contractions (PVCs)/day) including episodes of bigeminy and non-sustained ventricular tachycardia. As in other hereditary sudden cardiac death syndromes, it seems appropriate to eliminate these arrhythmias by means of radiofrequency catheter ablation (RFCA). To our knowledge, there is no publication of a successful RFCA in ATS. Delannoy et al.\(^1\) reported that RFCA was unsuccessful in the five patients in which it was attempted. In our Institution, during the follow-up of a large family with a genetically confirmed diagnosis of ATS,\(^2\) one of them became symptomatic (syncope), and an implantable cardioverter-defibrillator (ICD) was implanted in 2005 due to inducibility of ventricular fibrillation in an electrophysiological study. Intracavitary electrogams from the ICD showed frequent PVCs initiating the episode of ventricular fibrillation. Radiofrequency catheter ablation was attempted in 2006 and 2007 due to appropriate discharges of the ICD. In the first procedure, two ectopic sites in the left ventricle were targeted (anterosetal and anterolateral) guided by electroanatomical mapping, also, ablation of both Purkinje bundles was added. In the following year, she was submitted to a second procedure after a recurrent appropriate discharge of the ICD. In this time, RFCA was delivered in the postero septal and apical zones, without elimination of the PVCs.

These apparently large areas of arrhythmogenicity could be the basis for the suggestion expressed by Dr Wilde\(^3\) in an accompanying letter.

LETTERS TO THE EDITOR
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References


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References

1. Neuzner J, Dietze T, Bobzin M, Paliege R, Gradaus R. In cryoballoon pulmonary vein isolation there is no correlation between biomarker release indicating myocardial necrosis and cumulative freezing time, they found no linear correlation between biomarker release and cumulative freezing time, so they object that our biomarker index calculation is questionable.

First of all, some methodological differences have to be acknowledged. In our study, creatine kinase (CK), troponin T (TnT), and lactate dehydrogenase (LDH) were measured at three different time points. In contrast, Neuzner et al. measured TnI and CK at one time point (24 h after ablation). The probability to identify the peak value of biomarker release decreases with a single measurement and therefore, biomarker release kinetics will have a greater impact on data. In addition, we observed the CK peak 12 h after ablation. Their measurement 24 h post-ablation may have missed the peak biomarker release.

In humans, biomarker release is a surrogate of myocardial ablation injury. Neuzner et al. found no correlation for biomarker releases between a range of 24–48 min freezing time. As discussed in our manuscript, Wojcik et al. reported a linear correlation of 28 mm first-generation cryoballoon-induced CK peaks for longer ablation times (median 74, Q1–Q3: 64–86 min). To the best of our knowledge, no human data are available for shorter freezing time. In a canine model, Andrade et al. demonstrated similar histological lesion in dogs treated with either 120 or 240 s with regards to transmurality, but not quantifying the total myocardium injury. To clarify whether a linear correlation between biomarker and freezing time exists, studies investigating progressively increasing ablation times are needed (dose–response curves). They should range from a few seconds to minutes of freeze, to cover also the extreme part of the hypothesized correlation line. Obviously, such a study cannot be performed in human, reducing its clinical implications.

In this context, we think that our suggested biomarker index is a representative index of an increased efficacy in lesion formation. It should not be interpreted as an absolute value, but needs to be seen in the context of direct comparison of two cryoballoon generations. Neuzner et al. concluded that the reported differences in ablation efficacy between the two generations of cryoballoons might not be caused by differences in overall induced myocardial necrosis. We agree, but a similar myocardial necrosis can now be reached with less applications and with a shorter freezing time. In other words: with a higher efficacy.