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

We read with interest the work by Pecha et al. 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, 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 antibiotic 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 contralateral side (internal jugular vein) to change the site of pacing since the early post-extraction phases.

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
Editorial, that the arrhythmogenic substrate could be present throughout both ventricles thus explaining the lack of efficacy of RFCA. An alternative consideration is that we are not targeting the right spot. Delannoy et al. states that ‘initial attempts to map ventricular ectopy in patients with ATS reveal that ectopic beats originate from different parts of left Purkinje network’. It could be interesting to study the possibility of uncommon ventricular substrates, like the posterior papillary muscles, recently described as a distinct clinical entity. These could be considered in future attempts of RFCA of ectopic beats in this group of patients because of the similarities in the morphology of the PVCs.

Conflict of interest: none declared.

References

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doi:10.1093/europace/euu083
Published online 13 June 2014

In cryoballoon pulmonary vein isolation there is no correlation between biomarker release indicating myocardial necrosis and cumulative freezing time

We read with interest the article by Bordignon et al.¹ presenting a comparative analysis of biomarker release in pulmonary vein isolation with two different cryoballoon generations. We believe, however, that the rationale of the presented data processing in this article is questionable. The authors created a biomarker index by dividing the values of biomarker release (hs troponin T and creatine kinase (CK)) by cumulative freezing time. Using this method of data processing a difference in biomarker release between the two tested cryoballoon generations was detectable, otherwise no difference in measurable induced myocardial necrosis was found. The method of data processing is questionable because in the reported range of cumulative ablation times the biomarker release is not correlated with the ablation duration.

In our analysis in 58 consecutive patients undergoing pulmonary vein isolation for paroxysmal atrial fibrillation with a 28 mm second-generation cryoballoon, we found a median cumulative freezing duration of 34 min (range 24–48 min), the mean Troponin I values, 24 h after ablation, were 6.6 ± 4.2 μg/L and the mean CK values were 328 ± 134 U/L. The correlation coefficients for Troponin I and CK release and cumulative freezing time were r = −0.057 (P = 0.3382) and r = −0.118 (P = 0.1952), respectively.

In the reported range of cumulative freezing durations, there is no correlation between biomarker release and ablation duration in cryoballoon pulmonary vein isolation. Therefore, the two tested generations of cryoballoon showed a comparable amount of measurable myocardial necrosis. The reported differences in ablation efficacy between the two generations of cryoballoons might not be caused by differences in overall induced myocardial necrosis.

Conflict of interest: none declared.

Reference

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doi:10.1093/europace/euu132
Published online 13 June 2014

Author reply
We thank Neuzner et al. for the interest in our work.¹ They also investigated biomarker release in cryoballoon AF ablation. In their experience, 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,² 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, Wojcek et al.¹ 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.⁶ demonstrated similar histological lesion in dogs treated with either 120 or 240 s with regards to transmuralinity, 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.

Conflict of interest: none declared.

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