

Additional factors underlying pacing-induced cardiomyopathy in patients who underwent right ventricular pacing and His bundle pacing. Author's reply

Jan Mizner¹, Petr Waldauf², Domenico Grieco³, Hana Linkova¹, Oana Ionita¹, Pugazhendhi Vijayaraman⁴, Robert Petr¹, Radka Raková¹, Jana Vesela¹, Petr Stros¹, Dalibor Herman¹, Pavel Osmancik¹, Karol Curila¹

¹Cardiocenter, 3rd Faculty of Medicine, Charles University and University Hospital Kralovske Vinohrady, Prague, Czech Republic

²Department of Anesthesia and Intensive Care, Charles University and University Hospital Kralovske Vinohrady, Prague, Czech Republic

³Department of Cardiology, Policlinico Casilino of Rome, Rome, Italy

⁴Geisinger Heart Institute, Wilkes Barre, Pennsylvania, United States

Correspondence to:

Karol Curila, MD, PhD, MSc,
Cardiocenter, 3rd Faculty of
Medicine,
Charles University,
Srobarova 50, 100 34 Prague,
Czech Republic,
phone: +420 2 6716 2621,
e-mail: karol.curila@fnkv.cz

Copyright by the Author(s), 2023

DOI: 10.33963/v.kp.97882

Received:

September 23, 2023

Accepted:

September 23, 2023

Early publication date:

October 17, 2023

Thanks to Dr. Şener for their comments. We agree that concomitant diseases such as cardiac sarcoidosis (CS) and amyloidosis (CA) might influence the clinical course in patients with bradycardia. The clinical spectrum of transthyretin cardiac amyloidosis (ATTR-CA) symptoms includes advanced conduction disorders requiring pacemaker implantation. It had been documented that 3%–13% of ATTR-CA patients had pacemakers implanted before they were diagnosed with CA [1]. However, in the European population, the prevalence of ATTR-CA in pacemaker patients was very low (only 2%) [2]. Cardiac sarcoidosis (CS) also leads to advanced symptomatic atrioventricular blocks, and according to Kandolin et al. [3], it was the first symptom in up to 44% of patients with diagnosed CS. However, the prevalence of CS remains very low in the European population, where it is a rare condition. We initiate further diagnostic steps only if other risk factors are present [3]. Therefore, we believe that the low prevalence of these diseases and the randomized study design should not affect the differences in the left ventricular ejection fraction between studied groups. On the other hand, we agree that due to the pathophysiology of the diseases, scanning for CS and CA could be helpful while measuring the markers of collagen metabolism.

Pharmacological treatments, such as SGLT2 inhibitors and others, may influence left ventricular ejection fraction and myocardial

fibrosis. We have not provided their numbers, but SGLT2 inhibitors were not as available during the study period as they are now. Again, the randomized nature of the project should minimize their effect on the results.

We also agree that it is possible to adjust the pacemaker programming to avoid ventricular pacing. On the other hand, it is known that sacrificing atrioventricular synchrony at the cost of AV delay prolongation promotes atrial fibrillation and may worsen patients' clinical course. In light of this information and our experience, we are convinced that right ventricular pacing is fundamentally inappropriate treatment for bradycardia. It increases ventricular dyssynchrony, while His bundle pacing and left bundle branch pacing do not [4]. The pacemaker's primary purpose should be to keep an adequate heart rate and preserve the atrioventricular and ventricular synchrony as close to the physiological state as possible. Moreover, it should also follow other physiological needs of the human organism: not only a decline in the basal heart rate during rest or sleep but also a change in the heart rate in concordance with breathing [5].

Article information

Conflict of interest: None declared.

Funding: None.

Open access: This article is available in open access under Creative Commons Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, which allows downloading and sharing articles with others as long as they credit the authors and the

publisher, but without permission to change them in any way or use them commercially. For commercial use, please contact the journal office at kardiologiapolska@ptkardio.pl.

REFERENCES

1. Rapezzi C, Merlini G, Quarta CC, et al. Systemic cardiac amyloidoses: disease profiles and clinical courses of the 3 main types. *Circulation*. 2009; 120(13): 1203–1212, doi: [10.1161/CIRCULATIONAHA.108.843334](https://doi.org/10.1161/CIRCULATIONAHA.108.843334), indexed in Pubmed: 19752327.
2. López-Sainz Á, de Haro-Del Moral FJ, Dominguez F, et al. Prevalence of cardiac amyloidosis among elderly patients with systolic heart failure or conduction disorders. *Amyloid*. 2019; 26(3): 156–163, doi: [10.1080/13506129.2019.1625322](https://doi.org/10.1080/13506129.2019.1625322), indexed in Pubmed: 31210553.
3. Kandolin R, Lehtonen J, Airaksinen J, et al. Cardiac sarcoidosis: epidemiology, characteristics, and outcome over 25 years in a nationwide study. *Circulation*. 2015; 131(7): 624–632, doi: [10.1161/CIRCULATIONAHA.114.011522](https://doi.org/10.1161/CIRCULATIONAHA.114.011522), indexed in Pubmed: 25527698.
4. Vijayaraman P, Chelu MG, Curila K, et al. Cardiac conduction system pacing: A comprehensive update. *JACC Clin Electrophysiol*. 2023, doi: [10.1016/j.jacep.2023.06.005](https://doi.org/10.1016/j.jacep.2023.06.005), indexed in Pubmed: 37589646.
5. Shanks J, Abukar Y, Lever NA, et al. Reverse re-modelling chronic heart failure by reinstating heart rate variability. *Basic Res Cardiol*. 2022; 117(1): 4, doi: [10.1007/s00395-022-00911-0](https://doi.org/10.1007/s00395-022-00911-0), indexed in Pubmed: 35103864.