

# Additional factors underlying pacing-induced cardiomyopathy in patients who underwent right ventricular pacing and His bundle pacing

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DOI: 10.33963/v.kp.97243

**Received:**

August 15, 2023

**Accepted:**

August 28, 2023

**Early publication date:**

September 3, 2023

I have read with great interest the article comparing the impact of right ventricular pacing (RVP) and His bundle pacing (HBP) on cardiac fibrosis and systolic function. Mizner et al. [1] reported that HBP is related to better systolic function, compared to RVP, and to increases in fibrosis markers in patients whose ejection fraction was reduced by more than 5% during follow-up.

Although the most common cause of advanced atrioventricular (AV) blocks is idiopathic fibrosis of the conduction system caused by aging; cardiomyopathies and drug toxicities also may result in advanced AV blocks [2]. Sarcoidosis and amyloidosis are well-known diseases related to cardiac involvement and advanced conduction system disorders. Both sarcoidosis and amyloidosis tend to progress despite the current optimal treatment [3]. Therefore, it would be valuable if the study population had been screened for such cardiomyopathies because the reduction in ejection fraction and increased fibrosis markers might have indicated the progression of the underlying disease rather than pacing-related cardiomyopathy.

Current evidence from heart failure treatment points out that some medications including renin-angiotensin-aldosterone system inhibitors and SGLT2 inhibitors have beneficial effects on cardiac remodeling [4]. A significant proportion of the study population had comorbidities such as hypertension, diabetes, and coronary artery disease, so most of these patients might have been under treatment with the abovementioned drugs. The use of these drugs might have affected the results including changes in ejection fraction and fibrosis markers.

Programming the cardiac implantable electronic device is crucial because it may affect the pacing rates. Heart rate decreases during the night [5] and if the heart rate reduces below the limit, the pacemaker intercedes, resulting in increased pacing rates. Beta-blockers also decrease heart rates and may cause increased ventricular pacing burden. Therefore, I think it is important to take into account the baseline-set lower heart rate limit and the use of beta blockers in assessing the burden of ventricular pacing.

To conclude, of course, pacing-induced cardiomyopathy may develop in patients with high ventricular pacing burden, however, the impact of used medications and possible underlying cardiomyopathies should not be overlooked.

## Article information

**Conflict of interest:** None declared.

**Funding:** Founded by National Science Center of Poland — grant ID# 2017/27/B/NZ5/02944.

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