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P-wave dispersion in different clinical situations: Expanding list with resembling mechanisms

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Dear Editor,

We would like to thank the authors for the letter regarding with our manuscript entitled "*P-wave dispersion: What we know till now?*" previously published in your distinguished journal.[1] In that paper, we summarized the measurement methods, current use in different clinical situations, strengths and limitations of the of P-wave dispersion (Pd).[1]

Pd is a non-invasive electrocardiographic (ECG) marker for atrial remodeling and predictor for atrial fibrillation (AF).[1–3] Pd is simply calculated as the difference between the widest and the narrowest P-wave duration recorded from the 12-lead ECG.[1–4] In the letter entitled "*The role of P wave dispersion in Dystrophic and Thalassemic Cardiomyopathy*," the authors shared their notable experiences with Pd in different clinical situations. Among these studies, Russo et al.[5] evaluated Pd in beta Thalassemia major (β-TM) patients with preserved systolic and diastolic functions. Pd was measured from 12-lead ECG. Cardiac iron levels were measured by cardiac magnetic resonance T2 star (CMR T2*) imaging. This study showed a significant increase of Pd in β-TM patients with conserved systolic and diastolic cardiac functions. Furthermore, there was a significant correlation between Pd and CMR T2* values.[5]

In another study, Russo et al.[6] investigated the role of maximum P-wave duration (P max) and Pd, calculated through a manually performed measurement with the use of computer software from all 12-lead ECG, as predictors of AF in β-TM patients during one-year follow-up. In this study, β-TM patients presented increased P-max and Pd. A cut-off value of 35.5 ms for PD had a sensitivity of 90% and a specificity of 85% in identifying β-TM patients at risk for AF.[6]

Paroxysmal AF frequently occurs in Emery-Dreifuss muscular dystrophy (EDMD).[7] Russo et al.[7] evaluated the P-wave duration and Pd in patients with EDMD with preserved systolic and diastolic cardiac function. P-wave dispersion measured by using 12-lead ECG. There was a significant increase of maximum P-wave duration and Pd in patients with EDMD. In another study, Russo et al.[8] disclosed that myotonic dystrophy type 1 (MD1) patients had higher maximum P-wave duration and Pd, compared to age- and sex-matched healthy controls. P max and Pd were higher in MD1 patients subgroup with AF compared to MD1 patients without arrhythmia.[8]

These studies,[5–8] as summarized above, are examples for the expanding use of Pd in various clinical situations. Increased P-wave duration and Pd reflect prolongation of intraatrial and interatrial conduction time with lack of a well-coordinated conduction system within the atrial muscles.[1,2] Despite different clinical situations can alter Pd, main mechanistic logic behind them were similar. Increased Pd reflects inhomogeneous, asynchronic and discontinuous propagation of sinus impulses mainly between the left and right atria, interstitial/extracellular fibroblast activation and collagen deposition with fibrosis in atrial tissue, insufficient blood supply, significant not isotropic myoelectric activity, and thin wall thickness and consequent expansion tendency all well-known electrophysiological characteristics in patients with atrial arrhythmias and especially paroxysmal AF.[1,2]

As a conclusion, Pd has been applied in a wide range of clinical conditions. P-wave duration and Pd reflect prolongation of intraatrial and interatrial conduction time and the inhomogeneous propagation of sinus impulses which are well-known electrophysiologic characteristics in patients with atrial arrhythmias and especially paroxysmal AF.[1,2,4] Pd has been used in the assessment of the risk for AF in patients without apparent heart disease, coronary artery disease, hypertension, valvular heart diseases, heart failure, congenital heart diseases, rheumatologic diseases and various clinical conditions with expanding list.[1,2] Prediction of the AF

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recurrence might guide the physician to determine the antiarrhythmic therapeutic strategies.

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