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The role of P-wave dispersion in dystrophic and thalassemic cardiomyopathy

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Dear Editor,

We read with great interest the recent review entitled "*P-wave dispersion: What we know till now?*" by Okutucu et al.[1] in Journal of the Royal Society of Medicine Cardiovascular Disease. In their well-designed paper, the authors summarized the current use, measurement methods, strength points and limitations of the P-wave dispersion (PD) evaluation.

PD, defined as the difference between the maximum and minimum PD on standard 12-lead electrocardiogram (ECG), is considered a non-invasive indicator of intra-atrial conduction heterogeneity of sinus impulses, and it seems to be associated with an increased risk of atrial fibrillation (AF) in a broad range of clinical settings including cardiovascular and non-cardiovascular diseases.[2,3]

Okutucu et al. reported an extensive clinical evaluation of PD in the assessment of the AF risk in patients with arterial hypertension, coronary artery disease, valvulopathy, heart failure, congenital heart diseases, and who suffering from various cardiac or non-cardiac disorders, as well as in subjects without apparent heart disease. We suggest the authors to include in their future reviews a more detailed analysis about the clinical utility of PD in other clinical conditions, such as β-thalassemia major (β-TM), dystrophic cardiomyopathy, obesity and obesity hypoventilation syndrome, which may predispose to early AF.[4–12] According to our previous findings, β-TM patients with conserved systolic and diastolic cardiac functions showed an increased PD which well correlated to the myocardial iron deposit assessed by CMR T2* imaging,[4] and it seems to be a useful electrocardiographic marker for identifying the β-TM high-risk patients for AF onset.[5]

Our recent studies showed that myotonic dystrophy type 1 (MD1) and Emery-Dreifuss muscular dystrophy patients presented increased maximum P wave duration (P max) and PD values,[6–8] compared to age- and sex-matched healthy controls. P max and PD were statistically significantly increased in MD1 patients subgroup with AF compared to MD1 patients with no arrhythmias.[6] These results suggested the hypothesis that atrial fibrosis degeneration and fatty infiltration pattern may be responsible for intra-atrial conduction heterogeneity producing substrate for reentry which predispose to the onset and the perpetuation of AF in MD1.[8]

Furthermore, Okutucu et al. reported the utility of PD and atrial electromechanical delay to detect a subgroup of patients with atrial septal aneurysm (ASA) at high risk for paroxysmal supraventricular arrhythmias (SVAs), assessed by a short-time ECG Holter monitoring.[9] We suggest the authors to consider our recent study which evaluated the relationship between ASA and SVAs onset through a long-period external loop recorder (ELR) monitoring in a large young healthy ASA population[10]; our analysis confirms the results of the one by the authors, but it overcomes its limitations evaluating the SVAs occurrence in ASA patients without interatrial shunt, monitored through 30-day ELR during a four-year follow-up.

In conclusion, the 12-lead resting ECG remains the most frequently used examination in the evaluation of patients for cardiovascular disease and, because of its relatively low cost, it has the greatest potential to be used as a screening tool in these patients. According to the data in literature, PD may be considered a simple electrocardiographic parameter for AF risk assessment in many clinical conditions and it should be implemented in our daily clinical practice. For patients with increased PD, we suggest to perform a careful cardiac monitoring with seriate ECG Holter recordings or ELR and a periodical evaluation of device stored electrograms to early detect AF onset and to evaluate the opportunity of prophylactic anticoagulation or antiarrhythmic treatment or non-pharmacologic approaches for stroke prevention.

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