Editorial

P wave duration and dispersion as a useful conventional electrocardiographic marker for atrial fibrillation prediction

Volume 8 Issue 3 - 2017

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Received: February 24, 2017 | **Published:** March 08, 2017

Editorial

The increase in prevalence of atrial fibrillation (AF) is related with aging, and it has been reported to be associated with degeneration of the atrial muscle in pathological studies in elderly people.[1–3] It has been shown that there is clear evidence in the human atrial myocardium of age-related electrical uncoupling of the side-to-side connections between bundles. This is histologically related to the proliferation of extensive collagenous tissue septa in intracellular spaces.[4–6] In pathological studies, it was demonstrated that these age-induced changes include a reduction in the number of myocardial cells within the sinus node, a generalized loss of atrial myocardial fibers, as well as an increase in fibrosis which leads to an apparent loss of myocardial fiber continuity.[1–6]

Patients with diseased atrial tissue with progressive fibro-degenerative changes may develop abnormal electrophysiological alterations.[7–11] Connective tissue surrounding atrial myocardial cells represents sites where electrical coupling between adjacent cells is altered.[1–3] Therefore, the micro-architecture and anisotropic characteristics may play an important role in reentry by causing inhomogeneous and discontinuous propagation of the impulse in the atrium.[3] The P wave of the electrocardiogram may show alterations that can be associated with atrial arrhythmias and AF. P-wave dispersion (PWD) is considered a noninvasive electrocardiographic (ECG) marker for atrial remodeling and predictor for AF.[12–15] PWD reflects disturbances of intra-atrial and inter-atrial conduction, and it is defined as the difference between the wider and the narrower P-wave duration recorded from the 12 ECG leads at a paper speed of 50mm/s. The correct measurement of PWD is derived by subtracting the minimum P-wave duration from the maximum in any of the 12 standard surface ECG leads in supine position following 15min of rest and room temperature and lighting kept constant.[13] The onset of the P-wave is defined as the point of first detectable upward or downward slope from the isoelectric line for positive or negative waveforms, respectively. Return to the isoelectric line is considered as the end of the P-wave. PWD can be calculated by manual measurements with hand-held calipers or computerized methods. Manual measurement with hand-held calipers is performed by increasing the ECG rate to 50mm/s and the voltage to 1mV/cm, accompanied by the use of magnification. The normal value of PWD was found to be 29 ± 9ms, and values greater than 40ms indicate the presence of heterogeneous electrical activity in different regions of the atrium that might cause AF to develop.[13] It has been shown that increased P-wave duration and PWD reflect prolongation of intra-atrial and inter-atrial conduction time and the inhomogeneous atrial propagation of sinus impulses.[12–14]

The analysis of the P-wave with the 12 standard surface ECG leads in the stratification of patient suffering from AF is a recognized universal approach. It is well accepted that not only the P-wave duration, but also the P-wave morphology and dispersion have the potential to give information about the anatomical substrate predisposing to AF.[13–16] It has been demonstrated that a P wave maximum duration value of 106ms separated patients with paroxysmal AF from control subjects with a sensitivity of 83%, a specificity of 72%, and a positive predictive accuracy of 79%. In addition, a PWD value greater than 36ms separated AF patients from control subjects with a sensitivity of 77%, a specificity of 82%, and a positive predictive accuracy of 85%.[13] PWD has shown to have a significant correlation with maximum P-wave duration ($r=0.702$, $p<0.001$) and a weak, although significant association with age ($r=0.270$, $p<0.001$).[13] In addition, PWD was shown to be a significant predictor of frequent symptomatic AF paroxysms.[14] It was also found to have a significantly positive correlation with maximum P-wave duration ($p<0.001$) and negatively with minimum P-wave duration ($p=0.06$). Extensive clinical evaluation of P-wave dispersion has been performed in the assessment of the risk for atrial fibrillation in patients without organic heart disease, in patients with arterial hypertension, in patients with coronary artery disease, in patients undergoing coronary artery bypass surgery, in patients with congenital heart diseases, as well as in other groups of patients suffering from various cardiac or non-cardiac diseases.[17–28] Consequently, PWD can be helpful in discriminating patients with different kinds of diseases whom are prone to develop paroxysmal AF in the course of their lives.[29–37]

We have previously found that patients with a predisposition to develop AF have significantly higher incidence of atrial conduction defects, and abnormally prolonged and fractionated atrial endocardial electrograms.[7–11] At the time of the atrial endocardial catheter mapping

during sinus rhythm, we have found that an abnormally prolonged and fractionated right atrial electrogram may reflect inhomogeneous local electrical activity related to a delayed and non-uniform anisotropic conduction through diseased atrial muscle, and were closely related to the vulnerability of the atrial muscle in patients with paroxysmal AF.[9–11] Indeed, we demonstrated that the greater the extent of the compromised atrial muscle, the greater the likelihood that paroxysmal AF would develop.[9] Qualitative and quantitative analysis of atrial endocardial electrograms recorded during sinus rhythm should be an important analysis in evaluating local atrial electrophysiological abnormalities, and acquire particular relevance in the study of patients with paroxysmal AF. In the evaluation of patients with altered P wave morphology and dispersion in the electrocardiogram, it is very important to keep in mind that patients who have a great susceptibility to develop AF possess abnormally prolonged and fractionated atrial endocardial electrograms, a significantly longer P wave duration, a significantly longer intra-atrial and inter-atrial conduction time of sinus impulses; and a significantly greater sinus node dysfunction and higher incidence of induction of sustained atrial fibrillation.

Conclusion

PWD reflects prolonged, inhomogeneous and anisotropic distribution of connections between myocardial fibers resulting in discontinuous anisotropic propagation of sinus impulses, as well as, inhomogeneous and discontinuous atrial conduction. PWD is considered as a sensitive and specific ECG predictor of paroxysmal AF.

Acknowledgments

None.

Conflicts of interest

Author declares there is no conflicts opf interest.

Funding

None.

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Citation: Centurion OA, Aquino N, Torales J, et al. P wave duration and dispersion as a useful conventional electrocardiographic marker for atrial fibrillation prediction. *J Cardiol Curr Res*. 2017;8(3):1–6. DOI: 10.15406/jccr.2017.08.00285