Rev Port Cardiol. 2020;39(7):415–416

Revista Portuguesa de Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

LETTER TO THE EDITOR

Atrial fibrillation: Relationship with smoking and risk identification on the electrocardiogram☆

Fibrilhação auricular: relação com o tabagismo e identificação de risco no ECG

Atrial fibrillation (AF) is the most common sustained arrhythmia in clinical practice and is a significant cause of morbidity and mortality. The prevalence of this arrhythmia has increased in recent decades, and is predicted to rise exponentially in the coming years with the aging of the population, reaching the scale of a veritable epidemic.

Recent years have seen extensive research into AF, with a systematic search for clinical situations and signs on common diagnostic exams that indicate a greater likelihood of its occurrence.

As there appear to be few references to this subject in the Portuguese medical literature, we decided to review international publications on two of the most interesting aspects of the condition: the relationship between smoking and AF, and the possibility of predicting the risk of this arrhythmia based on changes in the P wave on the electrocardiogram (ECG).

Smoking and atrial fibrillation

Various prospective cohort studies have compared the occurrence of AF in smokers and non-smokers.

Analysis of data from cohorts of the Framingham Heart Study (FHS),[1] beginning in 1968 and including 4764 patients followed for 10 years, shows that smoking increased the risk of AF by 40% in women but had no significant effect in men, and was not used in the FHS risk prediction model for risk of incident AF.

In a substudy of the Atherosclerosis Risk in Communities (ARIC) study (1987-2002)[2] including 15 329 individuals over a mean follow-up of 13.1 years, multivariate analysis showed that the hazard ratio (HR) for AF was 2.05 among current smokers, representing a more than two-fold increase in risk, and 1.58 for ever smokers, compared to never smokers. A dose-response association was also seen, with a proportionally higher risk of AF among heavy smokers.

As part of the Rotterdam Study, an analysis of the link between cigarette smoking and risk of AF published in 2008[3] in 5668 subjects with a median follow-up of 7.2 years showed that current smokers and former smokers had increased risks of AF as compared to never smokers (relative risk [RR] 1.51, 95% confidence interval [CI] 1.07-2.12; and RR 1.49, 95% CI 1.14-1.97, respectively). There was no difference between the sexes.

An analysis of 11 047 participants of the REGARDS Study (2003-2007)[4] identified AF in 9.5% of smokers and 7.8% of non-smokers ($p<0.001$), a 15% increase in risk, in a 10-year follow-up. The association was stronger in younger participants, in those with prior cardiovascular disease, and in blacks.

Although a few studies have shown conflicting results,[5] there is general agreement that the evidence for an association between smoking and AF is strong. However, the pathogenesis of this link has yet to be fully established. Various potential mechanisms have been postulated; it is accepted that the association is strongly influenced by cardiovascular risk factors, but other arrhythmogenic mechanisms are certainly involved, including increased oxidative stress, inflammation, the sympathomimetic effects of nicotine, disruption of potassium channels and dysregulation of microDNA leading to fibrosis.[5]

Indices of atrial fibrillation risk based on P-wave alterations

Recent advances in high-definition signal acquisition and the development of automatic electrocardiographic systems have facilitated the use of the ECG to identify pathological markers, including indices of risk for AF.

Several studies have analyzed P-wave duration and dispersion, some using high-definition signal-averaged electrocardiography. Most variables are measured automatically by specialized software and the results are verified by a cardiologist.

P-wave duration is the time taken by the electrical impulse to propagate from the sinus node throughout the atria and is usually defined as the highest value on the 12-lead ECG. This parameter is an indicator of electrophysiological and structural remodeling of the atrium.

In a study by Perez et al.[6] of 42 751 patients followed for a mean of 5.3 years, new-onset AF was detected in 1050

☆ Please cite this article as: Bonhorst D. Fibrilhação auricular: relação com o tabagismo e identificação de risco no ECG. Rev Port Cardiol. 2020;39:415–416.

patients (0.45%/year). After correcting for age and gender, maximum P-wave duration >120 ms was a predictor of AF, with HR of 1.6 (95% CI 1.3-1.8, p<0.0001).

This index was also assessed in the FHS[1] and ARIC[2] studies, in which the results were similar; these and smaller studies confirm that P-wave duration predicts AF in patients both with and without heart disease.

By contrast, other studies have shown a relationship between AF and short P waves. In the Copenhagen ECG Study,[7] both shorter and longer P-wave duration presented a higher risk of AF than normal duration (100-105 ms).

Another index that has been the subject of research is P-wave voltage in lead I (PVL1). Lower PVL1 was associated with AF recurrence after catheter ablation in a study by Park et al.,[8] in which PVL1 was significantly lower in patients with recurrent AF in multivariate Cox analysis (HR 2.163, 95% CI 1.307-3.581, p=0.003). These authors found that in patients with low P-wave amplitude, interatrial conduction via Bachmann's bundle was reduced (interatrial block), an abnormality that could be related to atrial electrical remodeling and fibrosis due to reduced blood perfusion in this region.

A study by Alexander et al.[9] in 322 patients with non-ST-elevation myocardial infarction concluded that reduced PVL1 and interatrial block were associated with AF. Participants who developed AF within one year had significantly lower PVL1 (p=0.007) and longer P-wave duration (126±20 vs. 119±17 ms, p=0.022) than those who did not develop AF. The authors suggest that PVL1 and interatrial block may be associated with the same pathological process, namely atrial fibrosis, leading to increased P-wave duration and reduced P-wave voltage.

P-wave dispersion (PWD) is the difference between the longest and the shortest P-wave duration. A PWD>80 ms is associated with AF, and a standard deviation of P-wave duration across 12 leads >35 ms is also a risk factor for this arrhythmia.[10]

Other P-wave indices that have been studied as predictors of AF include interatrial block (defined as P-wave duration ≥120 ms with or without inversion of the terminal part of the wave), P-wave terminal force (product of the negative part of the P wave in lead V1 in μV and its duration in ms), notching, P pulmonale, and alterations in the P-wave axis.[10]

Conflicts of interest

The author has no conflicts of interest to declare.

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