VIA MEDICA
REVIEW ARTICLE
Cardiology Journal
2008, Vol. 15, No. 5, pp. 402–407

ISSN 1897–5593

Atrial conduction and atrial fibrillation: What can we learn from surface ECG?

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Abstract

Despite the advancements in pharmacological and non-pharmacological management of atrial fibrillation (AF) observed during last decades, available treatment modalities and predictors of their success are still far from optimal. Understanding of pathophysiological mechanisms underlying AF and assessment of atrial electrophysiological properties using easily available non-invasive diagnostic tools such as surface ECG are essential for further improvement of patient-tailored treatment strategies. P-wave duration is generally accepted as the most reliable non-invasive marker of atrial conduction and its prolongation has been associated with history of AF. However, patients with paroxysmal AF without structural heart disease may not have any impressive P-wave prolongation thus suggesting that the global conduction slowing is not an obligatory requirement for development of AF. In these settings, the morphology of P-wave becomes an important source of information concerning propagation of atrial activation. One of the most common morphologies, i.e. biphasic configuration of P-waves in right precordial leads has been considered a marker of left atrial enlargement but, seen in patients with structurally normal hearts, appears to be linked to an interatrial conduction defect. Recent advances in endocardial mapping technologies have linked certain P-wave morphologies with interatrial conduction patterns that may have clinical implications for invasive treatment of AF patients. The value of P-wave morphology extends beyond cardiac arrhythmias associated with atrial conduction delay and can be used for prediction of clinical outcome of wide range of cardiovascular disorders such as survival after myocardial infarction or the risk of stroke. (Cardiol J 2008; 15: 402–407)

Key words: P-wave, electrocardiography, interatrial conduction, atrial fibrillation

Introduction

Atrial fibrillation (AF) remains the most common cardiac arrhythmia demanding treatment, and the number of patients affected worldwide is increasing along with the ageing of the population in the industrial world. Despite the advances in pharmacological and non-pharmacological management of AF, observed during recent decades, we are still far from being satisfied with the available treatment modalities and predictors of their success. Understanding of pathophysiological mechanisms underlying AF and assessment of atrial electrophysiological properties using easily available non-invasive diagnostic tools such as surface ECG are essential for further improvement of patient-tailored treatment strategies.

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Received: 21.07.2008 Accepted: 28.08.2008

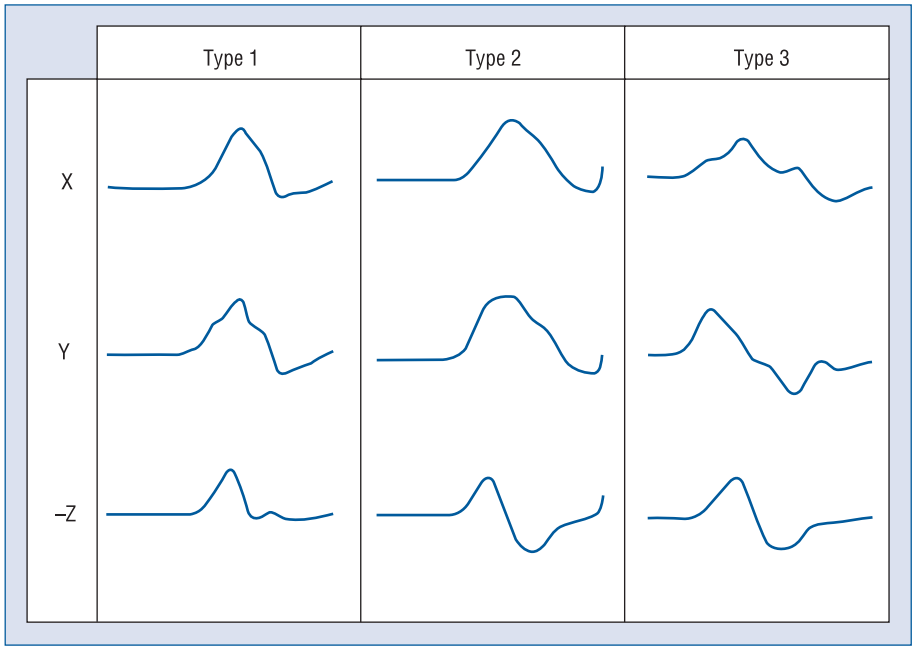

Figure 1. Common orthogonal P-wave morphologies. Type 1: Upright P-waves in all orthogonal leads — common in healthy subjects below 50 years of age; Type 2: Upright P-waves in leads X and Y and biphasic in lead Z — common in patients with paroxysmal atrial fibrillation, left atrial enlargement but may also be seen in healthy older than 50; Type 3: Upright in X but biphasic in leads Y and Z — advanced interatrial block, often associated with prolongation of P-wave >120 ms. This P-wave morphology is uncommon in healthy subjects.

Conduction velocity in atrial fibrillation genesis

The presence of triggers, shortened refractoriness and delayed conduction are important prerequisites for re-entry underlying AF [1]. The likelihood that a trigger can initiate a arrhythmia that could sustain itself requires an arrhythmic substrate in the atria and depends on the length of the re-entry wavelet defined as a product of the local effective refractory period and the conduction velocity. Slowing conduction, resulting in shorter wavelength, increases the number of wavelets that could coexist in the given atrial dimensions thus increasing the likelihood that AF would sustain itself. This mechanism underlying AF has been confirmed in animal experiments and clinical studies reviewed recently [2].

P-wave duration is generally accepted as the most reliable non-invasive marker of atrial conduction and its prolongation has been associated with history of AF [3], the development of the arrhythmia after bypass surgery [4] and deterioration of paroxysmal AF into the permanent form of the arrhythmia [5]. In 1985, Bayes de Luna et al. [6] described an advanced interatrial block with retrograde activation of the left atrium associated with histories of atrial tachyarrhythmias, that was later confirmed by others [5, 7]. This kind of interatrial block is seen on the surface ECG as a wide (> 120 ms) and biphasic P-wave in the inferior leads (Fig. 1, Type 3) [8]. While the prevalence of this advanced block is relatively low, the partial interatrial block defined as a P-wave longer than 120 ms with possible notched morphology is a much more common finding and in a recent report was documented in about 40% of hospitalized patients [9].

Atrial fibrosis, either age-related [10] or caused by coexisting conditions such as heart failure or hypertension [2], may result in a slowing of atrial conduction observed as a prolongation of P-wave on the surface ECG. However, the P-wave prolongation *per se* may be regarded only as a marker of general slowing of atrial conduction secondary to extended structural abnormalities of atrial walls and interatrial pathways. In reality, patients with paroxysmal AF without structural heart disease may not have any impressive P-wave prolongation thus suggesting that global conduction slowing is not an obligatory requirement for the development of AF [11–13].

P-wave morphology

In the absence of significant P-wave prolongation in patients without structural heart disease, the

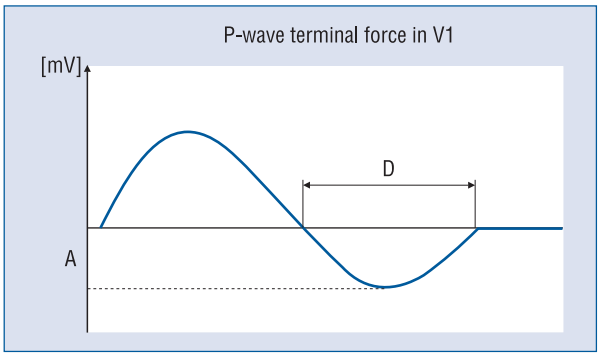

Figure 2. P-wave terminal force defined as the product of the duration (D) and amplitude (A) of the terminal phase of the P-wave in lead V1; PTF > 0.04 mV × ms has been found to be specific but less sensitive ECG marker of left atrial enlargement [25].

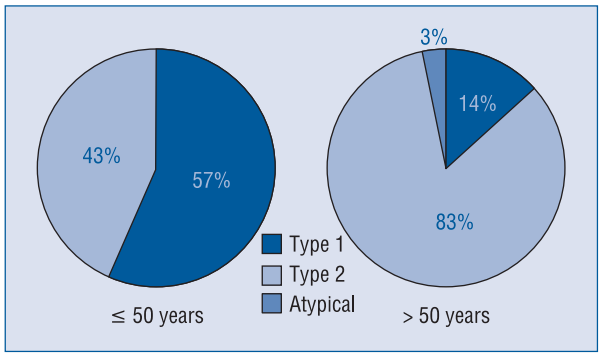

Figure 3. Distribution of common P-wave morphologies in healthy subjects. Younger subjects (20–49 years old) have nearly equal representation of Type 1 and Type 2 P-waves while middle-aged and seniors (50–80 years old) have predominantly Type 2 morphology.

morphology of P-wave becomes an important source of information concerning propagation of atrial activation.

The interest in P-wave morphology in this setting dates back to the mid 1960s when Morris et al. [14] introduced the concept of a P-wave negative terminal force (PTF) seen as a pronounced negative deflection in the terminal portion of the P-wave in the right precordial leads in patients with known mitral or aortic valve disease (Fig. 2) that has long been regarded as a marker of left atrial enlargement. Shortly thereafter, Robitaile et al. [15] suggested that abnormal PTF is associated with the presence of paroxysmal AF. Ten years later, Josephson et al. [16] suggested that biphasic P-waves in the right precordial leads may represent an interatrial conduction defect, not necessarily linked to left atrial enlargement.

The variability of the normal P-wave morphology has been appreciated. The P-wave vector in the frontal plane may vary from –50 to +60 degrees, reflecting right-to-left propagation of the wave front originating in the sinus node located at the junction of the superior vena cava and right atrium. In the precordial leads, P-waves are usually positive with only exception: lead V1. In this lead, P-waves may be upright, biphasic (+/–) or negative [17]. Observations of abnormal PTF in V1 in apparently healthy men 40–60 years old have also been reported [18]. Our group has studied P-wave morphology in 120 healthy men and women aged 20 to 80 years and observed that although biphasic P-waves in the Z-lead are observed in healthy subjects, this particular morphological pattern was observed predominantly in subjects over 50 years of age, in whom any other P-wave morphologies were unusual [19] (Fig. 3). However, even in this healthy cohort several atypical P-wave morphologies.

What makes P-waves appear to be biphasic or monophasic in right precordial leads? Using electro-anatomical mapping of the left atrium during sinus rhythm, our group proposed an explanation of the origin of P-wave morphology in subjects without structural heart disease [20]. Upright P-wave in the right precordial leads appears to be associated with conduction, not only via Bachmann's bundle but also via other interatrial connections in the vicinity of the right pulmonary veins on the back side of the heart, thus resulting in posterior-to-anterior propagation of sinus beat in both right and left atria (Fig. 4A). Several endocardial mapping studies have demonstrated that conduction via these posterior connections, not via Bachmann's bundle, may dominate during sinus rhythm [21, 22]. Biphasic P-waves in these leads, on the other hand, are associated with conduction via Bachmann's bundle only resulting in the anterior-to-posterior direction of left atrial activation, i.e. the terminal part of the P-wave (Fig. 4B). It is therefore plausible to suggest that posterior interatrial connections, known to be generally thinner and less developed compared to the Bachmann's bundle [23, 24], are therefore more affected by ageing and leave only Bachmann's bundle as a major interatrial route in middle-aged and senior patients, leading to biphasic P-waves in the right precordial leads in the surface ECG.

One should bear in mind, however, that these considerations are only applicable for subjects without considerable left atrial enlargement. Increased size of the atrial chamber itself may affect the vector of propagation of sinus beat resulting in the

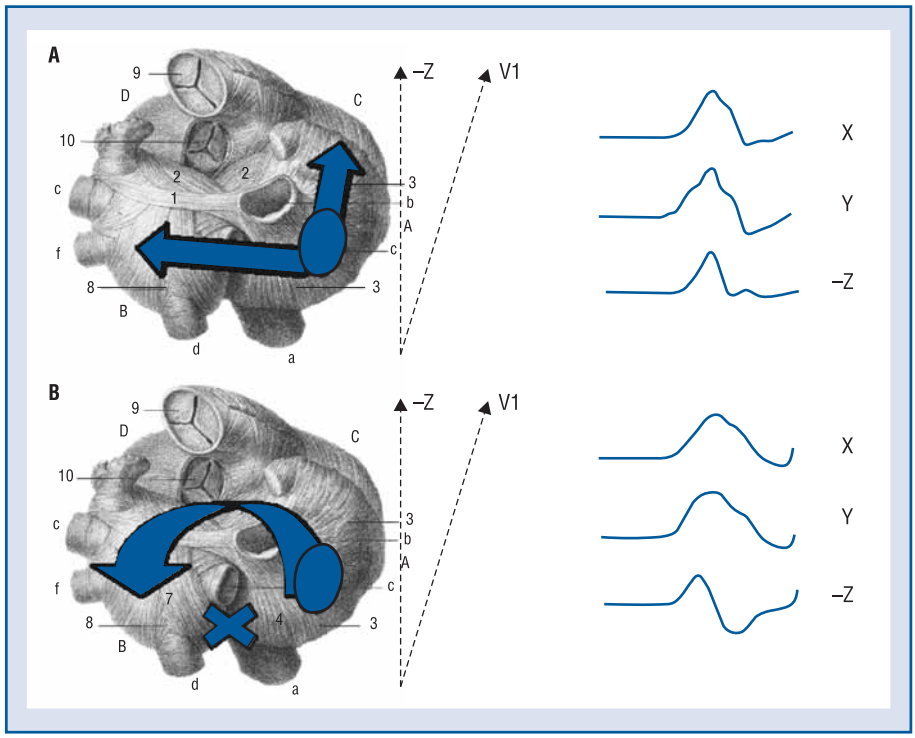

Figure 4. Schematic representation of the genesis of biphasic P-waves in leads V1 and Z in patients without structural heart disease. Interatrial conduction over the posterior interatrial connections result in the posterior-to-anterior propagation of exitation in the left atrium and positive or isoelectric P-waves in V1/lead Z (Type 1, panel **A**). Interatrial conduction over Bachmann's bundle only without contribution from posterior interatrial connections result in the anterior-to-posterior activation of the left atrium and biphasic P-wave in V1/lead Z [26].

abnormal PTF in lead V1 as shown recently in a study using cardiac magnetic resonance imaging [25]. The resulting P-wave morphology is therefore a complex interplay of geometrical and electrophysiological considerations, both affecting the way the sinus beat travels across the atria.

Local disturbance of interatrial conduction and atrial fibrillation

Apart from the rare finding of advanced interatrial block with retrograde left atrial activation associated with atrial arrhythmias and AF in particular, the morphology of P-waves has mostly been neglected in the context of this arrhythmia. In the limited material of patients with lone AF, we reported the presence of significant differences in P-wave morphology between patients with lone paroxysmal AF and healthy subjects not associated with any substantial P-wave prolongation [11], thus suggesting that the local rather than global slowing of interatrial conduction may be related to AF mechanisms in this patient cohort. The differences were mostly confined to the orthogonal lead Z, corresponding to the V1–V2 chest leads where we observed biphasic P-waves in the majority of AF patients. In a recent study, similar findings were obtained using the same unfiltered P-wave signal-averaged ECG technique in a cohort of patients with hypertrophic obstructive cardiomyopathy known for their higher risk of developing AF [26].

However, it is important to acknowledge that solid data linking histological substrates to deteriorated atrial conduction and higher propensity of AF are still lacking. Diffuse structural abnormalities such as vacuolar degeneration, inflammatory changes and fibrosis have been reported in patients with lone AF [27]. Critically located lesions have been shown by Becker et al. [10], who described the presence of an extensive fibro-fatty replacement of Bachmann's bundle in patients with a history of AF. Our group has not been able to document differences either in the structure or location of interatrial connections associated with a history of AF in the largest anatomical study to date on 84 human hearts [28].

Clinical implications of P-wave morphology

The diagnostic potential of P-wave morphology for the delineation of interatrial conduction and

identification of interatrial routes participating in the propagation of sinus beats indicates its possible use for guiding the placement of the atrial lead for optimal atrial resynchronization. Several studies have addressed the effect of atrial septal pacing for the prevention of paroxysmal AF by placing the atrial lead in the vicinity of Bachmann's bundle[29], the postero-inferior interatrial connections [30–32] or both [33]. While some studies have demonstrated the clinical benefit of atrial septal pacing [29, 31, 32], the largest one, by Padeletti et al. [33], did not show any decrease in AF burden. The selection of patients that would benefit from this treatment is therefore still poorly defined, and the selection of a pacing technique is not individually tailored. However, these trials demonstrated that septal electrode placement is safe and it is likely that in patients with indication for atrial pacing the septal positioning of the electrode guided by P-wave morphology would improve the resynchronisation effect and possibly improve rhythm control in patients with paroxysmal AF.

The clinical implications of P-wave morphology analysis extend beyond its use as a marker of left atrial enlargement and show it to be more than yet another characteristic of interatrial conduction during sinus rhythm. In a registry-based analysis of discharge ECGs of 641 patients who survived the acute phase of myocardial infarction, abnormal PTF, observed in 11% of the patients, was associated with a nearly two-fold increase in 5-year mortality [34]. In other, smaller studies, abnormal PTF was found to be a predictor of poor prognosis during acute phase of myocardial infarction [35, 36] and at 4-year follow-up after myocardial infarction [37], and the risk of stroke [38]. These data demonstrate that regardless of the mechanisms underlying the genesis of the P-wave morphology, its value extends beyond cardiac arrhythmias associated with atrial conduction delay and can be used for the prediction of the clinical outcome of a wide range of cardiovascular disorders.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

The study was supported by the Swedish Heart-Lung Fundation.

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