

# Modification of Arruda's Accessory Pathway Localization Method to Improve the Performance of WPW Syndrome Interventions

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## INTRODUCTION

### Description of the Wolff-Parkinson-White Syndrome

The Wolff-Parkinson-White (WPW) syndrome is characterized by an accessory pathway (by-pass tract) between the atria and ventricles that conducts parallel with the atrioventricular (AV) node-His bundle, but faster (Wolff, Parkinson, & White, 1930; Yee, Klein, & Guiraudon, 1995). An accessory AV connection can conduct in both directions. The presence of these by-pass tracts may predispose to atria-ventricular reentrant tachycardia. Moreover, in the setting of atrial fibrillation, the WPW syndrome may cause a catastrophically rapid ventricular response with degeneration to ventricular fibrillation (VF).

Electrocardiographically, the WPW syndrome can be characterized by a specific sinus rhythm pattern. Its other specific features are paroxysms of re-entry tachycardia (the incidence in the young adult population is about 10% and growing up with age to 30%), more rarely paroxysm of atrial fibrillation (20-30% of patients with

the syndrome), or atrial flutter (Guize, Soria, Chaouat et al., 1985; Wellens, Fare, & Bar, 1987).

In the case of WPW syndrome, the electrocardiogram (ECG) tracing is a mixture of the electrical activities caused by the accessory AV connection and normal AV conduction system. The fast impulse conduction produces an initial deflection in the QRS complex (delta wave).

The length of this delta wave is determined by the difference between the accessory AV connection and normal AV conduction times. The modified ventricular activation may cause secondary abnormalities in the ventricular repolarization such as: ST segment displacement (elevation or depression), T wave shape distortion, and abnormal U wave appearance. The conduction capacity variances (changes may occur hour by hour or day by day) of the accessory AV connection may result in alternating WPW pattern (complete, partial, or missing pre-excitation, concertina effect).

An adequate analysis of this phenomenon is important, since 1-2% of the population suffer from WPW syndrome (Wellens, Brugada, Penn et al., 1990). When the refractory period of the accessory connection be-

comes too short, the patient's life is endangered by a possible VF. Unfortunately, the exact risk for developing VF during high ventricular rates is unknown.

**WPW Syndrome Analysis**

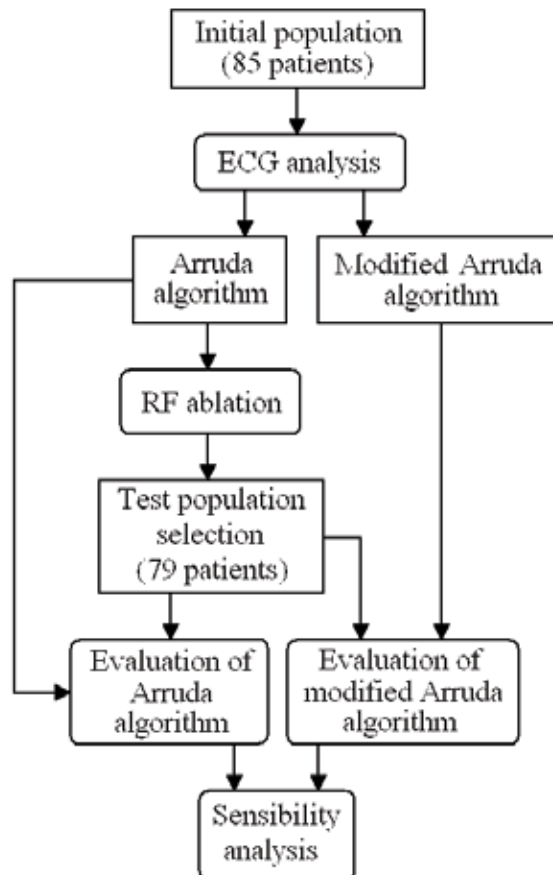
Usually the WPW analysis is focused to develop and validate an accessory pathway (AcP) localization method (Rosenbaum, Hecht, Wilson, & Johnston, 1945). A number of investigations have correlated ECG patterns and processing algorithms for detecting the place of the AcP (Arruda, McClelland, Wang, Beckman et al., 1998; Fitzpatrick, Gonzales, Lesh et al., 1994; Reddy & Schamroth, 1987), while other studies have been focused on the localization, realized through three-dimensional (3D)-heart reconstruction by the inverse solution of the ECG (Huiskamp & Greensite, 1997).

Unfortunately, the inverse problem, in contrast to the forward approximation methods, does not possess a mathematically unique solution. Another not easily by-passable difficulty is rooted in its ill-posed nature, whereby the obtained inverse solution could be unstable and may oscillate widely with the slightest perturbation.

Several approaches have been explored to handle the problem of multiple solutions by using equivalent cardiac generators, such as equivalent dipole (De Guise et al., 1985) and multipole, heart surface isochrones (Huiskamp & Greensite, 1997), or epicardial potential (Guanglin & Bin, 2001; Shahidi, Savard, & Nadeau, 1994). The high sensitivity of solutions to the different disturbances forced the investigators to explore various regularization techniques (Shahidi et al., 1994).

These methods allow a significant progress, but the diverse uncertainty elements of the processing limit

*Figure 1. Flow sheet of the research*



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