SENSIBILITY ANALYSIS OF THE ARRUDA LOCALIZATION METHOD

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Abstract: This paper presents an analysis of the Arruda accessory pathway localization method (for patients suffering from Wolff-Parkinson-White syndrome) with suggestions to increase the overall performance. The Arruda method was tested on a total of 121 patients, and an 90% localization performance was reached. This was considered almost as performing result as the highest published (90%) by L. Boersma in 2002. After a deeper analysis of each decision point of Arruda localization method we considered that the lead AVF is not as relevant as other used leads (I, II, III, V1). The overall performance (90%) was slightly lower then the correct decision rate (91,67%) at the weakest decision element (AVF+) of the method. The vectorial space constructed from the most used leads (II, V1, AVF) is not orthogonal which can be a reason for weaker rate in case of AVF.

Introduction

A. Description of the Wolff-Parkinson-White syndrome

The Wolff-Parkinson-White syndrome is characterized by an accessory pathway (by-pass tract) between the atria and ventricles that conducts in parallel with the atrioventricular (AV) node - His bundle, but faster [1], [2]. 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 can cause a catastrophically rapid ventricular response with degeneration to ventricular fibrillation (VF).

Electrocardiographically the WPW syndrome can be characterized by a specific pattern in sinus rhythm, paroxysms of re-entry tachycardia (the incidence in the young adult population is about 10% and growing up with age to 30%) and more rarely by paroxysm of atrial fibrillation (20–30% of patients with the syndrome) or atrial flutter [3], [4].

In the case of WPW syndrome, the electrocardiogram (ECG) tracing is a mixture of the electrical activities [5] 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) [6]. 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 causes secondary abnormalities in ventricular repolarization such as: ST segment displacement (elevation or depression), T wave shape distortion and abnormal U wave appearance. The accessory AV connection's conduction capacity variances can cause alternating WPW pattern, concertina effect, and episodic conduction. Changes may occur hour by hour or day by day.

An adequate analysis of this phenomenon is necessary, because 0.1-0.2% of the population suffer from WPW syndrome [7], [8]. When the accessory connection's refractory period is too short, the patient's life is in danger due to a possible VF. Unfortunately the exact risk for developing VF during high ventricular rates is unknown [9].

In consequence of the accessory connection's cells small mass, their electrical properties cannot be seen on an ordinary ECG measurement (with maximum 12-bit resolution).

B. WPW syndrome analyzer methods

Usually the WPW analysis is focused to develop and validate an AP localization method [10]. A number of investigations have correlated ECG patterns and algorithms for detecting the localization of the AP [11]–[16]. Some study has been focused on the localization, realized through three-dimensional (3D)-heart reconstruction by the inverse solution of the ECG [17]–[22].

Several approaches have been explored to handle the problem of multiple solutions by using equivalent cardiac generators (such as equivalent dipole [23] and multipole), heart surface isochrones [17]–[18], or epicardial potential [19]–[22]. The high sensitivity of solutions to the different disturbances forced the investigators to explore regularization techniques [19]–[21]. These methods allow a significant progress, but the different uncertainty elements of the processing limit the potentially beneficial ECG inverse solutions from becoming a routine clinical tool at present.

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In this paper we present a sensibility analysis of the Arruda's stepwise method [16], and a decomposition algorithm to increase the performance of AP localization. Our main purpose is to decipher the location of the ventricular insertion.

Materials And Methods

A. The starting data

This study starts from results of paper M. Arruda, J. McClelland, X. Wang, K. Beckman, et al, "Development and Validation of an ECG Algorithm for Identifying Accessory Pathway Ablation Site in Wolff-Parkinson-White Syndrome" [16]. In Arruda's paper the study population considered of 256 consecutive patients referred for RF catheter ablation of a manifest accessory atrioventricular pathway. Subjects wth more than one anterogradely conducting AP were excluded from the retrospective phase of study. There were 157 men and 99 women (mean age 32±15 years, range to 78). The algorithm to predict AP location was developed by correlating the preablation ECG with the successful RF ablation site in 135 consecutive patients with a single antergradely conducting AP. The method was then tested prospectively in 121 consecutive patients undergoing RF catheter ablation to assess its accuracy in predicting the successful ablation site.

B. The ARRUDA localization method

Our first task in WPW syndrome analysis was to determine the location and nature of the accessory connection. As the standard 12-lead ECG recordings held most of the desired information, we could locate the AP from our measurements.

We preferred to solve this localization with Arruda's stepwise method [16] instead of the Fitzpatrick algorithm [11]. The clinically tested and well-known Arruda method had used only five leads (I, II, III, aVF, V1) from the 12-lead ECG recordings. However this localization method could reach 90% recognition rate, some modification in this place identification algorithm could be benefic. Starting form the stepwise method of Arruda, we had to determine its performance and eventually to propose some modifications.

The possible AP locations were divided into three major regions, which were further divided thereafter, as follows:

- Septal accessory pathways: anteroseptal tricuspid annulus and right anterior paraseptal (AS/RAPS), mid-septal tricuspid annulus (MSTA), posteroseptal tricuspid annulus (PSTA), posteroseptal mitral annulus (PSMA), subepicardial posteroseptal (SEC);

- Right free-wall accessory pathways: right anterior (RA), right anterolateral (RAL), right lateral (RL), right posterolateral (RPL), right posterior (RP);

- Left free-wall accessory pathways: left anterolateral (LAL), left lateral (LL), left posterolateral (LPL), left posterior (LF).
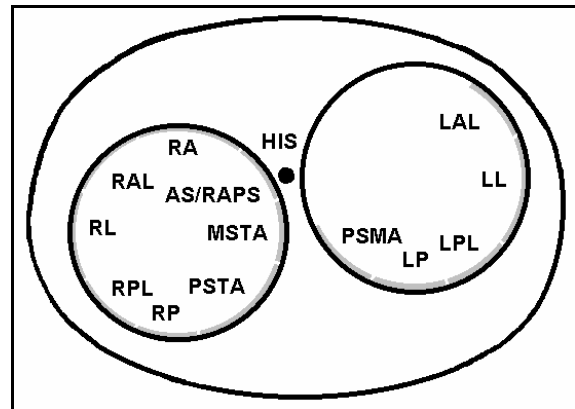

Figure 1: Schematic representation of the heart as viewed in the left anterior oblique projection.

These major and minor locations were illustrated in Figure 1, indicating also the place of the His bundle (HIS). The starting points of our WPW analysis were the study of delta wave and QRS complex mixture. We had to analyze the amplitude relations of the R, S and delta (Δ) waves in order to determine the AP location. The onset of the delta wave in each lead was measured from the onset of the earliest delta wave in any of the 12 leads. After 20 ms the displacement of the delta wave in each lead was classified as positive (+), negative (-) or isoelectric (0).
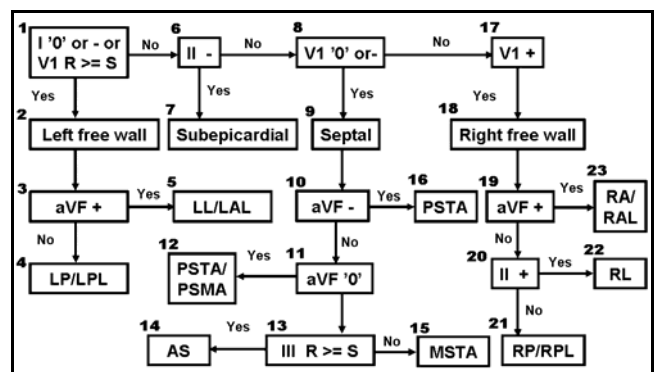

Figure 2: Stepwise Arruda method for determination of AP location

Several attempts have been made to correlate electrocardiographic findings with anatomic locations of accessory AV pathways in patients suffering from WPW syndrome. ECG criteria based upon surgical dissection of accessory pathways have shown accuracy in identifying AP location. The ARRUDA method differs from prior algorithms in its combined use of the resting ECG, utilization of only five ECG leads (I, II, III, V1, aVF), and by prospective validation of the algorithm. The relationship between the predicted location (based upon the ECG algorithm) and the actual

location (based upon ablation site) is shown in Table 1. These predictions were analyzed in order to determine the strong and week points of Arruda's method. From decision tree represented by Figure 2 we could evaluate each step.

Our analysis started from Table 1 and evaluates each step (1-23) of Arruda's stepwise method. Then the used leads were represented vectorial to evaluate the sensibility of each decision. Finally all decision were evaluated, a new decision tree were constructed and evaluated with signal samples shown in M. Arruda's paper [16].

Table 1: Location and detection rate of the accessory pathway

Ablation Site	Number	RA/RAL	RL	RP/RPL	AS/RASP	MSTA	PSTA	PSMA	LP/LPL	LL/LAL	SEC	Sens. (%)	Spec. (%)
RA/RAL	17	17										100	97
RL	13	1	11		1							85	100
RP/RPL	9	1		8								89	100
AS/RASP	4	1			3							75	99
MSTA	5					5						100	98
PSTA	22					2	18		2			82	100
PSMA	1							1				100	99
LP/LPL	4						1		3			75	96
LL/LAL	32								3	29		91	100
SEC	14										14	100	100
All	121											90	99

Results

We considered important to represent the relationship between the predicted and actual (based upon ablation site) distribution of AP location (see in Table I). Ablation sites are represented in vertical and the predicted locations in horizontal direction.

Table 2: Accuracy of each decision point for all accessory pathways

Place no.	Ablation site	Number	Failed	Decision performance
4	LP/LPL	4	1	1; 4/3/1 3; 4/4/0
5	LL/LAL	32	3	1; 32/32/0 3; 32/29/3
7	Subepicardial (SEC)	14	0	1; 14/14/0 6; 14/14/0
12	PSMA	1	0	1; 1/1/0 6; 1/1/0 8; 1/1/0 10; 1/1/0 11; 1/1/0
14	AS/RAPS	4	1	1; 4/4/0 6; 4/4/0 8; 4/3/1 10; 4/4/0 11; 4/4/0 13; 3/3/0
15	MSTA	5	0	1; 5/5/0 6; 5/5/0 8; 5/5/0 10; 5/5/0 11; 5/5/0 13; 5/5/0
16	PSTA/PSMA	22	4	1; 22/20/2 6; 20/20/0 8; 20/20/0 10; 0/18/2
21	RP/RPL	9	1	1; 9/9/0 6; 9/9/0 8; 9/9/0 19; 9/8/1
				20; 8/8/0
22	RL	13	2	1; 13/13/0 6; 13/13/0 8; 13/13/0 19; 3/11/2 20; 11/11/0
23	RA/RAL	17	0	1; 17/17/0 6; 17/17/0 8; 17/17/0 19; 7/17/0

Table 2 represents the performance of each decision point from Arruda's stepwise algorithm. From this data the decision performance was determined and represented in Table 3.

Table 3. Global accuracy of each decision point

Place no.	Decision number	Failed decisions	Performance
1	121	3	97,52%
3	36	3	91,67%
6	83	0	100,00%
8	69	2	97,10%
10	30	2	93,33%
11	10	0	100,00%
13	8	0	100,00%
19	39	3	92,31%
20	19	0	100,00%

Discussion

After we had analyzed the Arruda AP localization algorithm, we observed that most times the estimation errors (places 3, 10, 19) were correlated with aVF sign test. In the graphical representation of the localization algorithm (see in Figure 3) the sensitive spots (all of them represent aVF+) were encircled.
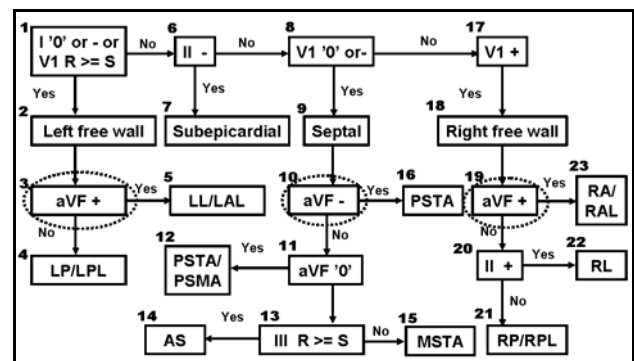

Figure 3: The most sensitive spots in stepwise Arruda AP localization algorithm

In our study about 75% of the prediction errors were caused by a week aVF sign decision (see in Table 1). The localization made by simple comparison shows lower recognition percentage in case of RL, PSTA, and LL/LAL locations. In case of PSTA the $R \geq S$ relation in V_1 and aVF- could happen, so these restrictions do not imply in all cases LP/LPL.

These considerations were tested on our database, and must be used with care due to the followings:

- Our database was too small to guarantee a solid statistical confirmation;

- The selected patients were not 100% representative (due to the small number);

- We used only few recordings from one patient, so the WPW syndrome could manifest in other way (it could change its behavior hourly).

Conclusion

Several attempts had been made to correlate ECG findings with anatomic locations of accessory AV pathways in patients with WPW syndrome. Most of them use different leads, based on empirical methods. When we took a closer look at the Arruda method we observed that the most used leads (V_1, II, AVF) cannot form an orthogonal coordinate system. Instead of the AVF lead we would prefer to use the AVL lead, which is perpendicular to lead II. Unfortunately we had no possibility to realize a test with the registrations used in case of Arruda's method testing. The about 89% recognition rate we considered quite good, compared with the highest published value of 90% [24]-[28]. These considerations could be useful to create a better non-invasive localization method.

References

[1] L. WOLFF, J. PARKINSON, P. WHITE, (1930): 'Bundle branch block with short PR interval in healthy young people prone to paroxysmal tachycardia.', *Am Heart J* , vol 5, pp. 685–704,1930.

[2] R. YEE, G. J. KLEIN, G. M. GUIRAUDON, (1995): 'The Wolff-Parkinson-White syndrome. ', In: D. P. Zipes, J. Jalife, eds. *Cardiac electrophysiology. From cell to bedside.* Philadelphia: WB Saunders Co, pp.1199–1214,1995.

[3] L. GUIZE, R. SORIA, J. C. CHAOUAT, ET AL., (1985): 'Prevalence and course of Wolff-Parkinson-White syndrome in population of 138,048 subjects.', *Ann Med Interne (Paris)* ,136, pp. 474– 489, 1985.

[4] H. J. J. WELLENS, J. FARE, F. W. BAR, (1987): 'The Wolff-Parkinson-White syndrome.', In: Mandel WJ, ed. *Cardiac arrhythmias. Their mechanisms, diagnosis and management.* Philadelphia: JP Lippincott, pp. 274–296,1987.

[5] S. M. SZILÁGYI, Z. BENYÓ, L. DÁVID, (2003): 'Heart Model Based ECG Signal Processing', *Modelling and Control in Biomedical Systems 2003, Proceedings volume from the 5th IFAC Symposium Melbourne (2003),* pp. 213-217, 2003.

[6] S. M. SZILÁGYI, Z. BENYÓ, L. DÁVID, (2003): 'WPW Syndrome Identification And Classification Using ECG Analysis', *World Congress on Medical Physics and Biomedical Engineering, Sydney,* *Australia (2003),* 4423.pdf, 2003.

[7] H. J. J. WELLENS, P. BRUGADA, O.C. PENN, ET AL., (1990): 'Pre-excitation syndromes: clinical presentation, course and therapy. ', In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology: from cell to bedside.* Philadelphia: WB Saunders, pp. 691–702, 1990.

[8] M. JOSEPHSON, 'PREEXCITATION SYNDROMES', IN: JOSEPHSON M, ED., (1993): *Clinical cardiac electrophysiology.* Philadelphia: Lea & Febiger, pp. 311–416,1993.

[9] J. A. GOUDEVENOS, C. S. KATSOURAS, G. GRAEKAS, O. ARGIRI, V. GIOGIAKAS, D. A. SIDERIS, (2000): 'Ventricular pre-excitation in the general population: a study on the mode of presentation and clinical course', *Heart* ,83,pp. 29-34,2000.

[10] F. F. ROSENBAUM, H. H. HECHT, F. N. WILSON, F. D. JOHNSTON, (1945): 'The potential variation of the thorax and the esophagus in anomalous antrioventricular excitation (Wolff–Parkinson–White syndrome)', *Am. Heart* J., 29, pp. 281–326, 1945.

[11] A. P. FITZPATRICK, R. G. GONZALES, M. D. LESH, ET AL., (1994): 'New algorithm for the localisation of accessory atrioventricular connections using a baseline electrocardiogram'. *J Am Coll Card*iol, 23, pp. 107–116, 1994.

[12] G. V. REDDY, L. SCHAMROTH, (1987): 'The localization of bypass tracts in the Wolff-Parkinson-White syndrome from the surface electrocardiogram', *Am Heart J*, 113:984, 1987.

[13] M. ARRUDA, X. WANG, J. MCCLELLAND, ET AL, (1992): 'ECG algorithm for predicting radiofrequency ablation site in posteroseptal accessory pathways' (Abstract), *PACE*, 15(Pt II): 535, 1992.

[14] M. ARRUDA, X. WANG, J. MCCLELLAND, ET AL, (1993): 'ECG algorithm for predicting sites of successful radiofrequency ablation of accessory pathways' (Abstract), *PACE*, 16(Pt II): 865, 1993.

[15] M. ARRUDA, X. WANG, J. MCCLELLAND, ET AL, (1994): 'Negative delta wave in lead II identifies posteroseptal accessory pathways requiring ablation in venous branches of the coronary sinus' (Abstract), *J Am Coll Cardiol* (Suppl), 224A, 1994.

[16] M. ARRUDA, J. MCCLELLAND, X. WANG, K. BECKMAN, ET AL, (1998): 'Development and Validation of an ECG Algorithm for Identifying Accessory Pathway Ablation Site in Wolff-Parkinson-White Syndrome', *J Cardiovascular Electrophysiology*, Vol. 9, pp. 2-12, Jan 1998.

[17] J. J. M. CUPPEN AND A. VAN OOSTEROM, (1984): 'Model studies with inversely calculated isochrones of ventricular depolarization,' *IEEE Trans. Biomed. Eng.*, vol. BME-31, pp. 652–659, 1984.

[18] G. HUISKAMP AND F. GREENSITE, (1997): 'A new method for myocardial activation imaging,' *IEEE*

Trans. Biomed. Eng., vol. 44, pp. 433–446, June 1997.

[19] A. V. SHAHIDI, P. SAVARD, AND R. NADEAU, (1994): 'Forward and inverse problems of electrocardiography: Modeling and recovery of epicardial potentials in humans', *IEEE Trans. Biomed. Eng.*, vol. 41, pp. 249–256, Mar. 1994.

[20] P. R. JOHNSTON AND R. M. GULRAJANI, (1997): 'A new method for regularization parameter determination in the inverse problem of electrocardiography', *IEEE Trans. Biomed. Eng.*, vol. 44, pp. 19–39, Jan. 1997.

[21] H. S. OSTER, B. TACCARDI, R. L. LUX, P. R. ERSHLER, AND Y. RUDY, (1997): 'Noninvasive electrocardiographic imaging: Reconstruction of epicardial potentials, electrograms, and isochrones and localization of single and multiple electrocardiac events', *Circulation*, vol. 96, pp. 1012–1024, 1997.

[22] L. GUANGLIN, H. BIN, (2001): 'Localization of the Site of Origin of Cardiac Activation by Means of a Heart-Model-Based Electrocardiographic Imaging Approach', *IEEE Trans. Biomed. Eng.*, vol. 48, pp. 660–669, 2001.

[23] J. DE GUISE, R. M. GULRAJANI, P. SAVARD, R. GUARDO, AND F. A. ROBERGE, (1985): 'Inverse recovery of two moving dipoles from simulated surface potential distributions on a realistic human torso model', *IEEE Trans. Biomed. Eng.*, vol. BME-32, pp. 126–135, 1985.

[24] L. BOERSMA, E. G. MORAN, L. MONT, J. BRUGADA, (2002): 'Accessory pathway localization by QRS polarity in children with Wolff-Parkinson-White syndrome.', *Journal Cardiovasc Electrophysiol*. 2002 Dec;13(12), pp. 1222-1226, 2002.

[25] S. M. SZILÁGYI, (2000): 'Heart Model Based Computerized ECG diagnosis', *World Congress on Medical Physics and Biomedical Engineering Chicago, USA*, July 23-28, 2000.

[26] B. BENYÓ, B. ASZTALOS, (2000): 'Detection of Pathologic Alterations of the Heartwall Based on Ultrasound and Echocardiographic Pictures', *ORKI Medical and Hospital Engineering*, vol. XXXVIII. No. 2. pp. 36-40, 2000.

[27] L. SZILÁGYI, (1999): 'Wavelet-Transform-Based QRS Complex Detection in On-Line Holter Systems', *Proceedings of the 21th IEEE Conference on Engineering in Medicine and Biology,* Atlanta, October 13-16, 1999, pp. 271, 1999.

[28] S. M. SZILÁGYI, Z. BENYÓ, L. DÁVID, (2003): 'Iterative ECG Filtering for Better Malfunction Recognition and Diagnosis', *Modelling and Control in Biomedical Systems 2003, Proceedings volume from the 5th IFAC Symposium Melbourne (2003)*, pp. 295-300, 2003.