

## Comparison of electrocardiographic and vectorcardiographic planes on a set of left ventricular hypertrophy patients

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**Abstract**—Most common electrocardiographic diagnostic criteria for left ventricular hypertrophy (LVH) are based on depolarization information. However, reports support that LVH also alters repolarization. Two measures relate depolarization/repolarization: the angle between the QRS-complex and the T-wave in a plane; the planar QRST ( $QRST^p$ ) and the vector obtained from the cross sum of the depolarization and repolarization vectors ( $RT^\alpha$  and  $RT^m$ ). We compared the performance of these measures as hypertrophy markers in two sets of planes: the ECG frontal plane (FP) versus the VCG frontal plane (XYP) and the ECG horizontal plane (HP) versus the VCG horizontal plane (XZP). The horizontal views picked up a significant increase of the  $QRST^p$  (HP controls vs LVH:  $40.18 \pm 41.20^\circ$  vs  $66.50 \pm 51.65^\circ$ ,  $p < 0.05$ ; XZP controls vs LVH:  $43.87 \pm 39.76^\circ$  vs  $66.35 \pm 38.30^\circ$ ,  $p < 0.05$ ) and a consistent behaviour in the frontal views (XYP controls vs LVH:  $17.71 \pm 37.23^\circ$  vs  $35.60 \pm 47.98^\circ$ ,  $p < 0.005$ ). On the other hand, the angle of the equivalent RT vector significantly increased in the HP (HP controls vs LVH:  $24.28 \pm 26.50^\circ$  vs  $33.53 \pm 22.42^\circ$ ,  $p < 0.05$ ). In conclusion, the angular information in their two forms ( $QRST^p$  and  $RT^\alpha$ ) relating depolarization and repolarization was the most informative parameter and should be regarded for the construction of more sensitive electrocardiographic LVH indexes.

**Keywords**—hypertrophy, VCG, ECG, LVH-induced electrophysiological remodelling, QRST-angle

### I. INTRODUCTION

LVH indexes based on electrocardiogram fell into disuse due to low sensitivity. Opposite to echocardiography or nuclear magnetic resonance, the ECG does not measure left ventricular mass in a straightforward way, but describes the electrophysiological remodeling in a hypertrophied heart [1, 2]. It is well known that repolarization is also modified with LVH [3] although no attempt to include this information in electrocardiographic indexes was registered up to date, focusing all efforts on different features of the depolarization phase [4, 5, 6]. On the other hand, there has been a longstanding disagree-

ment as to whether the ECG or VCG are more informative, with reports finding the VCG greater [7, 8], similar [6] or poorer [9] than the ECG to diagnose LVH.

Herein, we have combined depolarization and repolarization information in the form of the angle formed between the QRS-complex and the T-wave in a plane; the planar QRST ( $QRST^p$ ) and the equivalent RT vector obtained from the vector sum of the depolarization and repolarization vectors in a 2D space ( $RT^\alpha$  and  $RT^m$ , expressed as angle and modulus, respectively). The diagnostic utility of these parameters was compared along four planes: the horizontal and frontal planes in the ECG (HP and FP) and VCG (XZP and XYP).

### II. MATERIALS AND METHODS

#### A. Population

A total of 58 subjects without intraventricular conduction disturbances were retrospectively studied. Two groups were balanced for age characteristics: the LVH group (31 subjects, mean age  $68.5 \pm 12.3$  years old) and the control group (27 subjects, mean age  $60.6 \pm 13.2$  years old). The hypertrophy group included patients with left ventricular mass indexes greater than  $125 \text{ g/m}^2$  in males and greater than  $110 \text{ g/m}^2$  in females, as calculated by Deveuraux's formula from echocardiography on M-mode [10]. These patients lacked a coronary artery disease history. Healthy subjects, without clinical or echocardiographic evidence of cardiovascular disease, comprised the control group. Besides echocardiographic data, 5-minute 12-lead ECG recordings acquired at 400 Hz were taken from all the subjects. Patients were recruited in the medical institution "Instituto de Investigaciones Médicas, Dr. Alfredo Lanari" of the University of Buenos Aires and in all cases informed consent was signed.

#### B. ECG Preprocessing

Signal preprocessing was applied to the 12 standard ECG leads, implementing QRS-detection and normal beat selection according to the method given in reference [11]. The

QRS-complexes and T-waves were located and delineated using the wavelet transform based method described in [12] and baseline wandering attenuation was treated by cubic spline. Noisy beats were rejected when differences in mean isoelectric level with respect to adjacent beats were larger than 300  $\mu\text{V}$ .

VCG was synthesized by means of the Kors Matrix [13]. The XZP was obtained from the VCG signals while the HP was derived from the standard ECG signals. More specifically, the precordial leads  $V_6$  and  $V_{1-2}$  were used, being the latter an average between leads  $V_1$  and  $V_2$ .

### C. Cardiac vectors

Cardiac vectors were measured in all planes following the same methodology as the one used here for the HP. Segmentation of the QRS-complex and T-wave was accomplished for each  $i_{th}$  beat using a single lead criterion, where the respective QRS and T onsets were taken at the earliest reliable QRS and T, either for  $V_6$  or for  $V_{1-2}$ . The offsets, in a symmetric way, were accepted respectively as the latest reliable QRS-complex and T-wave offsets for the same leads, as in the previous case. On these segmented waves, the QRS-loop and the T-wave loop for each  $i_{th}$  beat were constructed and both cardiac vectors computed at the loop samples  $n \in W_i^{\text{QRS}}$  and  $n \in W_i^{\text{T}}$ , representing the QRS-complex and T-wave windows respectively. The sample  $n_{\text{max}}^{\text{QRS}}(i)$  and  $n_{\text{max}}^{\text{T}}(i)$  at which the respective QRS-complex and T-wave cardiac vectors resulted maximum were computed. Thereafter, the angle and modulus of the QRS dominant vector for the  $i_{th}$  beat,  $R_H^\alpha(i)$  and  $R_H^m(i)$  respectively, were defined as,

$$R_H^\alpha(i) = \text{atan} \left( \frac{V_{1-2}(n_{\text{max}}^{\text{QRS}}(i))}{V_6(n_{\text{max}}^{\text{QRS}}(i))} \right) \quad (1)$$

$$R_H^m(i) = \sqrt{V_6(n_{\text{max}}^{\text{QRS}}(i))^2 + V_{1-2}(n_{\text{max}}^{\text{QRS}}(i))^2} \quad (2)$$

where,

$$n_{\text{max}}^{\text{QRS}}(i) = \arg \max_n \left[ \sqrt{V_6(n)^2 + V_{1-2}(n)^2} \right] \quad (3)$$

where  $n \in W_i^{\text{QRS}}$

Equivalently, and extending the above definitions for every plane under study, the angle and modulus of the main depolarization vectors for the  $i_{th}$  beat,  $R_F^\alpha(i)$ ,  $R_{XZ}^\alpha(i)$ ,  $R_{XY}^\alpha(i)$  and  $R_F^m(i)$ ,  $R_{XZ}^m(i)$ ,  $R_{XY}^m(i)$  respectively, as well as the angle and modulus of the T-wave maximum cardiac vectors for the

$i_{th}$  beat,  $T_F^\alpha(i)$ ,  $T_{XZ}^\alpha(i)$ ,  $T_{XY}^\alpha(i)$  and  $T_F^m(i)$ ,  $T_{XZ}^m(i)$ ,  $T_{XY}^m(i)$  were defined as in (1)-(3).

The  $QRST^p$  was then calculated as shown here for the HP, extending it to every plane,

$$QRST_H^p(i) = \text{abs}(R_H^\alpha(i) - T_H^\alpha(i)); \quad (4)$$

The equivalent RT vector components for the  $i_{th}$  beat  $RT_H^\alpha(i)$  and  $RT_H^m(i)$  were computed as the following vector sum in the HP, extending it to every plane,

$$RT_H^\alpha(i) = \text{atan} \left( \frac{R_y(i) + T_y(i)}{R_x(i) + T_x(i)} \right) \quad (5)$$

$$RT_H^m(i) = \sqrt{(R_x(i) + T_x(i))^2 + (R_y(i) + T_y(i))^2} \quad (6)$$

### D. Statistical Analyses

All data were expressed as Mean $\pm$ SD. The D'Agostino-Pearson normality test was applied to quantify the discrepancy between the distribution of the indexes and an ideal Gaussian distribution. In order to determine the statistical power of each marker to discriminate health from hypertrophy, a non-parametric two-tailed Mann-Whitney test was applied between controls and LVH patients. When  $p$ -value was  $<0.05$ , differences were considered statistically significant.

## III. RESULTS

Table 1 summarizes the results for every plane under study, together with the significance levels. Note that the horizontal planes described a significant increase of the planar QRST while the frontal planes picked up a significant decrease of the same parameter. This parameter, the planar QRST, provided statistical significance in almost every plane (except for the FP). On the other hand, the modulus of the equivalent RT vector failed to produce statistical significance in any plane and the angle of the former significantly decreased in the horizontal plane of the ECG (HP).

In order to assess the diagnostic utility of these parameters, their ROC curves with the respective area under the curve (AUC) values were obtained. Figure 1 shows the AUC values for every index under study in both the ECG and VCG space.

The ROC curves for the four LVH indexes with AUC values greater than 0.70 together with their ECG(VCG) counterparts are displayed in Figure 2. The optimal cut-off point in the ROC curves were computed as the point nearest to the top left-hand corner. This selection maximizes the sensitivity

Table 1: Mean±SD values for every parameter in Control and LVH groups together with statistical significance  $p$ .

Parameter	Controls	LVH	$p$
$RT_H^\alpha$	24.28±26.50°	33.53±22.42°	0.047
$RT_H^m$	2.07±0.57 mV	2.02±1.16 mV	NS
$QRST_H^p$	40.18±41.20°	66.50±51.65°	0.049
$RT_{XZ}^\alpha$	10.40±29.40°	21.03±34.28°	NS
$RT_{XZ}^m$	1.73±0.46 mV	1.81±0.89 mV	NS
$QRST_{XZ}^p$	43.87±39.76°	66.35±38.30°	0.012
$RT_F^\alpha$	48.16±46.64°	45.84±35.63°	NS
$RT_F^m$	1.58±0.65 mV	1.90±0.81 mV	NS
$QRST_F^p$	23.63±52.25°	28.49±47.55°	NS
$RT_{XY}^\alpha$	21.91±34.82°	20.73±11.65°	NS
$RT_{XY}^m$	1.88±0.50 mV	1.79±0.78 mV	NS
$QRST_{XY}^p$	17.71±37.23°	35.60±47.98°	0.002

and specificity sum, when it is assumed that the 'cost' of a false negative result is the same as that of a false positive one [14]. Characterization of the cut-off points are also shown in the paired format (Sensitivity; Specificity).

#### IV. DISCUSSION

Three parameters ( $RT^\alpha$ ,  $RT^m$  and  $QRST^p$ ) combining features from the QRS-complex and the T-wave in both horizontal and frontal planes of the ECG and VCG were presented. We have found the angular parameters ( $QRST^p$  and  $RT_F^\alpha$ ) and not the amplitude parameters to better separate controls from LVH patients. This finding may be explained in relation to the geometric aspects of the electrophysiological process. That is, while the two main vectors of depolarization and repolarization can modify both its magnitude and direction as a result of hypertrophy causing significant changes in their respective angles, these same changes may have little impact on the modulus after vectorial sum. Moreover, other reports in the literature support this observation for 3D spaces [15]. Although it has been well described that LVH alters both depolarization and repolarization phases [16, 3], no electrocardiographic index has taken into account the simultaneous changes induced by hypertrophy in the depolarization and repolarization phases and their interactions, to the best of our knowledge. ECG describes the complex electrophysiological modifications at cellular level that affect simultaneously depolarization and repolarization rather than assessing directly left ventricular mass. Moreover, this electrophysiological remodelling, consisting of a ventricular conduction delay [17] and a prolongation of the action potential duration [18] acts in

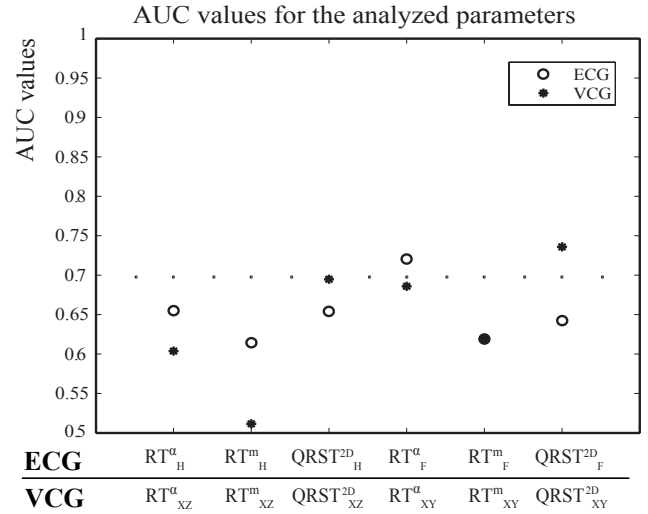


Fig. 1: AUC values for every pair of parameters in the ECG/VCG spaces. Notice that three out of twelve parameters presented AUC values  $\geq 0.70$ . The latter were:  $RT_F^\alpha$ ,  $QRST_H^p$  and  $QRST_F^p$ .

an opposite manner in the repolarization phase, classified into primary and secondary repolarization changes by Bacharova et al. [3] and depending the final amplitude, axis and loop morphology of the T-wave on a balance between these two changes, making it even harder to obtain clear ECG/VCG patterns when studied in an isolated fashion. The main hypothesis applied herein was that cardiac vectors compensate for this particular fluctuations and show a more robust behavior to the LVH-induced changes when studied simultaneously.

#### V. CONCLUSION

The planar QRST angles provided parameters with better classification performance than the equivalent vectors RT. Also, angles, and not moduli, presented greater AUC values, suggesting that the interplay between depolarization and repolarization is better described by angular rather than amplitude information. Finally, no clear superiority of the VCG over the ECG, could be addressed, encouraging the search of plain, direct LVH markers based on depolarization/repolarization relationships in the ECG.

#### CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

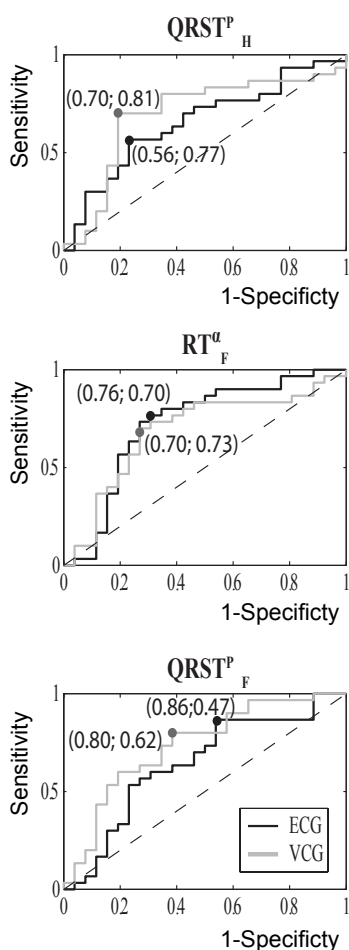


Fig. 2: ROC curves for the pair of parameters in the ECG (black) and the VCG (grey) with AUC values  $\geq 0.70$ . Cut off points are displayed (dots) together with the numeric pairs (Sensitivity; Specificity) corresponding to the cut off values.

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## REFERENCES

1. Bacharova L.. What is recommended and what remains open in the American Heart Association. Recommendations for the standarization and interpretation of the electrocardiogram. Part V: electrocardiogram changes associated with cardiac chamber hypertrophy. *Journal of Electrocardiology*. 2009;42:388-91.
2. Bacharova L.. Electrocardiography - Left ventricular mass discrepancies in left ventricular hypertrophy: electrocardiography imperfection or beyond perfection? *Journal of Electrocardiology*. 2009;42:593:96.

3. Bacharova L, Szathmary V, Mateasik A.. Secondary and primary repolarization changes in left ventricular hypertrophy: a model study. *J. of Electrocardiology*. 2010;43(6):624-33.
4. Sokolow M, Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar and limb leads. *Am. Heart J.* 1949;37:161-86.
5. Casale P, Devereux R, Kligfield P, et al. Electrocardiographic detection of left ventricular hypertrophy: development and prospective validation of improved criteria. *J Am Coll Cardiol.* 1985;6:572-80.
6. Romhilt D, Greenfield J, Estes E. Vectorcardiographic diagnosis of left ventricular hypertrophy *Circulation*. 1968;XXXVII:15-19.
7. Bristow J, Porter G, H Griswold. Observations with the Frank system of vectorcardiography in left ventricular hypertrophy *Am Heart J.* 1961;62:621.
8. Perez Riera AR, Uchida AH, Filho CF, et al. Significance of vectorcardiogram in the cardiological diagnosis of the 21st century. *Clin Cardiol.* 2007;30(7):319-23.
9. Borun E, Chapman J, Massey F. Electrocardiographic data recorded with Frank leads *Am. J. Cardiol.* 1966;18:656.
10. Devereux R B, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation*. 1977;55:613-8.
11. Moody G. B., Mark R. G.. Development and evaluation of a 2 lead ECG analysis program *Computers in Cardiology*. 1982:39-44.
12. Mendieta J. Algoritmo para el delineado de senales electrocardiograficas en un modelo animal empleando tecnicas avanzadas de procesamiento de senales Master's thesis Facultad de Ingenieria, Univ. de Buenos Aires (FI-UBA) 2012.
13. Kors J. A., Van Herpen G., Sittig A. C., Van Bommel J. H.. Reconstruction of the Frank vectorcardiogram from standard electrocardiographic leads: diagnostic comparison of different methods *European Heart Journal*. 1990;11:1083-1092.
14. Altman D.. *Practical Statistics for Medical Research*. 26 Boundary Row, London: Chapman Hall 1991.
15. Zabel Markus, Malik Marek. *Morphological Assessment of T Wave Patterns*:350-357. Blackwell Publishing 2007.
16. Hart George. Cellular electrophysiology in cardiac hypertrophy and failure *Cardiovascular Research*. 1994;28:933-946.
17. Cooklin M., Wallis W. R. J., Sheridan D. J., Fry C. H.. Changes in Cell-to-Cell Electrical Coupling Associated With Left Ventricular Hypertrophy *Circulation Research*. 1997;80:765-771.
18. McIntosh MA, Cobbe SM, Kane KA, Rankin AC. Action Potential Prolongation and Potassium Currents in Left-Ventricular Myocytes Isolated from Hypertrophied Rabbit Hearts *Journal of Molecular and Cellular Cardiology*. 1998;30:43 - 53.

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