

Quantification of Cardiac Ventricular Repolarization and its Spatial Dispersion through the Surface Electrocardiogram

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SUMMARY

The ventricular repolarization dispersion (VRD) is determined basically by the heterogeneity of the action potentials in different myocardial regions. Usually the heart responds to certain physiopathological states by producing a VRD increase, which may lead to a malignant ventricular arrhythmia and/or sudden death. For 25 years, the VRD has been quantified with several indexes obtained by computational ECG processing, in order to identify patients with high cardiovascular risk. These indexes are based on the detection of T wave changes in duration or morphology in presence of heart diseases. A revision of the spatial dispersion indexes and their potential as supporting tool for the diagnosis of cardiac risk is presented in this work.

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Key words >

T-wave Morphology - QT Interval Dispersion - ECG Decomposition - Risk Factors - Ventricular Repolarization Dispersion - Sudden Death

Abbreviations >

T_{PT}	Total angle principal component-to-T	V_G	Ventricular gradient
T_{CRT}	Total R T cosine	AMI	Acute myocardial infarction
MVA	Malignant ventricular arrhythmia	PVS	Premature ventricular stimulation
T_{WL}	T wave loop	SD	Sudden death
DCM	Dilated cardiomyopathy	AP	Action potential
ICD	Implantable cardioverter defibrillator	MAP	Monophasic action potential
HCM	Hypertrophic cardiomyopathy	RMS	Root-mean-square
R_c	Repolarization complexity	VR	Ventricular repolarization
QT_o	QT interval dispersion	LQTS	Long QT syndrome
QT_{cd}	Corrected QT interval dispersion	T_{MD}	T wave duration
APD	Action potential duration	T_{PE}	T-wave peak to T-wave end interval
VRD	Ventricular repolarization dispersion	T_{WR}	T-wave residuum
SVD	Singular value decomposition	3D	Three-dimensional
ECG	Electrocardiogram	VCG	Vectocardiogram

BACKGROUND

Electrocardiography was one of the first medical diagnostic applications of digital computers. (1) Automated analysis of ECG data is an active area of ongoing research, as a means of improving diagnosis and prediction of heart diseases. (2)

Quantification of ventricular repolarization dispersion (VRD) is one of the goals of the digital analysis of ECG data, as increased VRD has been associated with risk of malignant ventricular arrhythmias (MVAs) and sudden death (SD). (3)

There is abundant experimental and clinical evidence of the association between an increased dispersion of action potential duration (APD) and the incidence of MVAs and/or SD. The underlying mechanisms are reentrant circuits, increased automatism, influence of the autonomous nervous system and substrates associated with heart diseases. These anomalies are seen in situations of ischemia, (4) hypothermia, (5) electrolyte imbalance, (6) long QT syndrome (LQTS), (7), premature beats (8) or effects of the autonomous nervous system. (9) For example, in congenital LQTS, cardiac SD is the first

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manifestation of the disease in 10% of asymptomatic patients. (10)

Cardiac SD is defined as the sudden loss of heart function and may occur preceded by or in the absence of prodromes. A recent study performed in our country (11) in 642 021 subjects reported 1274 deaths, suggesting that coronary artery disease a risk factor for SD.

In patients at high risk of MVAs and/or SD, the current therapeutic options are antiarrhythmic drugs, implantable cardioverter defibrillator (ICD) or the combination of both. (12) Antiarrhythmic therapy should be monitored to prevent arrhythmias associated with acquired LQTS. (13)

For this reason it is important to identify patients at risk and to assess the cost-effectiveness of treatment interventions, combining the criteria used for treatment decision making with a thorough analysis of the ECG.

Currently there are experimental and clinical studies that validated the digital analysis of the ECG as a support tool to assess cardiac risk. (2)

The analysis of the VRD through ECG records can detect local or global modifications of the electrical gradient under pathological conditions at risk of for VMAs and/or SD.

The difference in the duration and shape of the action potential (AP) in the anterior, posterior and inferior wall of the left ventricle and between both ventricles contributes to regional inhomogeneity in ventricular repolarization (VR). In addition, the difference in recovery times of endocardial cells, M cells and epicardial cells generates transmural inhomogeneity. In consequence, regional and transmural dispersion characterize repolarization as a "spatial" phenomenon. Even more, VR depends on heart rate and on beat-to-beat-changes presenting "temporal" characteristics.

This review deals with the principal "spatial indexes" of VRD based on the quantification of "intervals" and "morphology" of VR.

COMPUTATIONAL ECG PREPROCESSING

ECG data is stored in the computer; baseline noise (breathing movements and/or patient movements) electrical interference (50 Hz) and muscular contraction are removed by digital filtration.

An algorithm detects the QRS complex. Finally, a delineator algorithm (14) detects the onset, peak and end of P waves, QRS complex and T waves beat-by-beat within a "window" defined by the previous QRS complex.

The analysis of "repolarization intervals" (section 3) is performed after measuring the QT interval, T-wave onset, T-wave end and then the signals extending from the T-wave onset to the T-wave end. Segmentation of repolarization is used to analyze "T wave morphology" (section 4) in fixed or variable windows

(ST-T complex or T wave) depending on the RR interval. Repolarization windows are calculated as the earliest and latest reliable T-wave onset and end between all leads. (15)

SPATIAL DISPERSION INDEXES ASSOCIATED WITH REPOLARIZATION INTERVALS

This section described markers of spatial heterogeneity associated to T wave intervals and their usefulness to stratify cardiac risk.

QT interval dispersion

QT interval dispersion (QT_D) is the difference between the maximum and minimum durations of the QT interval in the ECG. Originally, QT_D was determined in multilead recording systems (16) and thereafter in the standard 12-lead ECG. (17)

Day et al. (18) proposed that if each lead of the ECG recorded regional activity, QT_D might estimate the local dispersion of the myocardium, associating the dispersion reflected in the ECG with cell dispersion. Based on this hypothesis, they quantified increased VRD in patients with myocardial infarction who were treated with antiarrhythmic drugs. (19) Then, they compared the QT_D in hearts with normal sinus rhythm and with controlled ventricular stimulation, and concluded that the QT_D reflected regional differences in ventricular recovery time (20) (Table 1). Estimation of QT_D is simple and constitutes a non-invasive marker of MVA.

Higham et al. (21) compared VRD during sinus rhythm and ventricular pacing and found a great correlation between VRD measured with monophasic action potentials (MAPs) and with QT_D . Zabel et al. (22) recorded MAP and standard ECG using rabbit hearts and observed that QT_D correlated well with the dispersion of ventricular recovery times and with the APD (Table 1). Thereafter, they confirmed these results in human beings in ECG records performed 24 hours after recording MAPs; (23) they observed a simultaneous increase in QT_D and a differential increase in the duration of epicardial MAPs.

Bender et al. (24) studied the rate-corrected QT interval dispersion (QT_{CD}), defined as the difference between the maximum and minimum durations of the rate-corrected QT interval during acute myocardial infarction. They demonstrated the presence of favorable outcomes with amiodarone therapy as they found an absence of modification of the QT_{CD} in patients treated with high doses of the antiarrhythmic drug during the acute phase of acute myocardial infarction.

Other authors described QT_D as a marker of risk for arrhythmias due to LQTS, (7) for proarrhythmic effect of class III antiarrhythmic drugs, (25) in patients with acute myocardial infarction (26), hypertrophy (27) and *torsade de pointes*. (28)

Table 1. Results of some indexes quantifying ventricular repolarization dispersion.

Autor and year	Index (units)	Condition Nº 1	Condition Nº 2	Condition Nº 3	Comparison	Reference
Daand, et al. (1992)	QT_D (ms)	22 ± 2 (9 NSB)	80 ± 4 ^[E] (9 PVS)	23 ± 6 (9 NSB)	SST vs Nº 2 CI = 300 ms	20
		18 ± 2 (9 NSB)	87 ± 6 ^[E] (9 PVS)	18 ± 2 (9 NSB)	SST vs Nº 2 CI = 350 ms	
Zabel, et al. (1995)	Correlation with its respective p value (non-dimensional)	0,59 (p < 0,001) (50 B and 52 DS)	–	–	QT_D vs recovery time disp.	22
		0,61 (p < 0,001) (50 B and 52 DS)	–	–	QT_D vs APD disp.	
		0,81 (p < 0,0001) (50 B and 52 DS)	–	–	T_{PE} vs APD disp.	
Badilini, et al. (1997)	QT_D (ms)	33,3 (25 N)	61,4 (17 LQTS)	62,7 ^[NS] (30 AMI)	SST LQTS vs AMI	57
	T_{WL} (non-dimensional)	LQTS > AMI ^[A] (flat T_{WL}) (17 LQTS and 30 AMI)	AMI > LQTS ^[A] (rounded T_{WL}) (17 LQTS and 30 AMI)	–		
Priori, et al. (1997)	QT_D (ms)	35 ± 9 (40 N)	80 ± 42 ^[D] (36 LQTS)	–	SST	55
	R_C (%) (λ_2/λ_1)	13 ± 3 (40 N)	34 ± 12 ^[D] (36 LQTS)	–	SST	
Lee, et al. (1998)	QT_D (ms)	41 ± 18 (12l) (N 129)	40 ± 20 ^{NS} (12xyz) (N 129)	–	SST	29
MacFarlane, et al. (1998)	QT_D (ms)	29,1 ± 10,2 (12l) (N 1.220)	27,5 ± 10,8 ^{NS} (12xyz) (N 1.220)	–	SST	30
Kors, et al. (1999)	QT_D (ms)	54,2 ± 27,1 (for narrow T_{WL}) (382 N + 838 DD)	69,5 ± 33,5 ^[B] (for rounded T_{WL}) (382 N + 838 DD)	–	SST	31
Acar, et al. (1999)	T_{CRT} (non-dimensional)	0,52 ± 0,29 (76 N)	-0,35 ± 0,52 ^[F] (63 HCM)	–	SST	52
	R_C (%) (λ_2/λ_1)	15,56 ± 6,16 (76 N)	23,56 ± 10,85 ^[F] (63 HCM)	–	SST	
	Complexity (%) (λ_2 /total energy)	4,82 ± 2,37 (76 N)	7,76 ± 4,23 ^[F] (63 HCM)	–	SST	
Malik, et al. (2000)	QT_D (ms)	33,6 ± 18,3 (78 N)	47,0 ± 19,3 ^[A] (68 HCM)	57,5 ± 25,3 ^[A] (81 AMI)	SST HCM and AMI vs N	41
	T_{WR} (%)	0,029 ± 0,031 (78 N)	0,067 ± 0,067 ^[A] (68 HCM)	0,112 ± 0,154 ^[A] (81 AMI)		
	Correlation with its respective p value (non-dimensional)	-0,0446 (p = NS) (78 N)	0,2805 (p = NS) (68 HCM)	0,0771 (p = NS) (68 HCM)	QT_D vs T_{WR}	
Fuller, et al. (2000)	Correlation (non-dimensional)	0,91 (epicardial RMS) (52 records)	0,84 (total ECG RMS) (52 records)	0,81 (optimal ECG RMS) (52 records)	recovery time vs T_{WD}	33
		0,46 (epicardial RMS) (52 records)	0,47 (total ECG RMS) (52 records)	0,11 (optimal ECG RMS) (52 records)	recovery time vs QT_D	

Table 1. Continuation

Autor and year	Index (units)	Condition N° 1	Condition N° 2	Condition N° 3	Comparison	Reference
Zabel, et al. (2002)	T_{WR} (%)	0,33 ± 0,57 (520 A)	0,43 ± 0,62 ^[C] (252 D)	–	SST	42
	Absolute T_{WR} (mV.ms)	85.601 ± 144.844 (520 A)	111.91 3± 164.700 ^[C] – (252 D)	–		
	T_{CRT} (non-dimensional)	-0,11 ± 0,65 (520 V)	-0,23 ± 0,60 ^[A] (252 F)	–		
	R_C (%)	0,23 ± 0,14 (520 A)	0,25 ± 0,13 ^[A] (252 D)	–		
Arini, et al. (2008)	T_{WD} (ms)	95,2 ± 7,9 (10 C)	118,5 ± 15,7 ^[B] (10 PVS)	–	SST	34
		78,0 ± 10,3 (10 C)	133,6 ± 29,6 ^[B] (10 DS)	–		
	T_{CRT} (°)	137 ± 65 (10 C)	129 ± 61 ^[NS] (10 PVS)	–	SST	
		41 ± 17 (10 C)	73 ± 42 ^[A] (10 DS)	–		

Values are expressed as mean ±SD. **Condition:** B (basal), C (control), HCM (hypertrophic cardiomyopathy), DD (diverse diseases), DS (d-sotalol administration), PVS (premature ventricular stimulation), D (dead), AMI, (acute myocardial infarction), NSB (normal sinus beat), N (normal), RMS (root-mean-square), LQTS (long QT syndrome), A (alive), 12 I (standard 12 leads), 12 xyz (synthesized 12 leads from XYZ). **Statistically significant values;** ^[A] $p < 0.01$, ^[B] $p < 0.001$, ^[C] $p < 0.0005$, ^[D] $p < 0.0001$, ^[E] $p < 0.00001$, ^[F] $p < 0.000001$, ^[NS] Non significant. **Comparison:** SST (statistical significance test), CI (coupling interval).

However, QT_D generates controversies. Lee et al. (29) and MacFarlane et al. (30) (Table 1) demonstrated independently that QT_D calculated using the synthesized 12 leads from the orthogonal XYZ leads (which did not show the effects of regional heterogeneity) had the same magnitude than the QT_D measured using a standard 12-lead ECG. By the same time, Kors et al. (31) (Table 1) found a high correlation between the QT_D and the T-wave loop (TW_L), demonstrating that QT_D might be a three dimensional (3D) attribute of TW_L morphology rather than an effect of the local VRD (Figure 1). If all the information of repolarization is in the 3D TW_L , the QT_D might be due to the different projections of the heart vector on the different lead axes. In consequence, TW_L projections on the different lead axes present differences in QT intervals, reflecting as QT_D due to loss of information and not to the effect of real VRD. There are technical limitations to define the end of the T wave, such as algorithms that overestimate or underestimate its measurement, the existence of U-wave and low amplitude waves (32) (Figure 2).

T wave duration

Once T wave onset and T wave end have been determined, (14) T wave duration is calculated (T_{WD}). Increased T_{WD} indicates a differential shortening or

lengthening of the AP in some myocardium areas, reflecting ventricular heterogeneity.

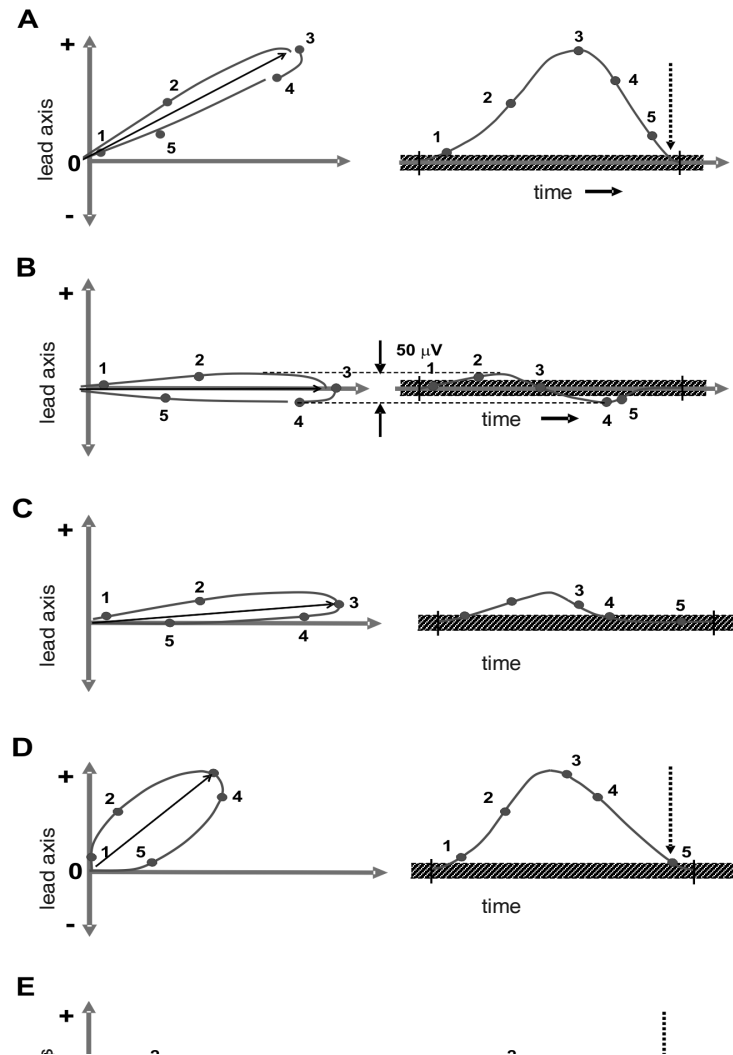
Fuller et al. (33) (Table 1) used isolated-perfused canine hearts and assessed VRD by changing temperature, cycle length and the activation sequence. They measured T_{WD} from the root-mean-square (RMS) curve obtained from 64 epicardial electrograms, 192 ECG surface leads, 6 standard precordial ECG leads, and 6 optimal leads. They found that recovery times from epicardial potentials strongly correlated with T_{WD} computed from the RMS series but, conversely, the correlation was low between the recovery times from epicardial potentials and QT_D from surface ECG leads, standard precordial leads and optimal leads.

Another research study performed on an isolated rabbit heart model measured T_{WD} from multilead ECG recordings during control dispersion (34). A T wave obtained from multilead recordings showed that T_{WD} quantified VRD better than QT_D , T_{WR} and T wave amplitude. The T_{WD} was a good marker of risk for increased VRD induced by supplying d-Sotalol (DS) or by premature ventricular stimulation (PVS).

T wave-peak -to-end duration

T wave-peak -to-end duration (T_{PE}), calculated from T-wave peak position to T-wave end, is a marker of transmural VRD. In a model of myocardial wedge

Fig. 1. Projection of T loops on hypothetical lead axis yields T waves depicted. Shaded zone represents noise band. End of T wave detected by an algorithm is marked by a downwards dashed arrow and does not coincide with end of repolarization. See the T wave obtained in all the charts after modifying the angle and the shape of the T_{WL} . Narrow, elongated T_{WL} at oblique angle with lead axis results in well-discernible T wave in that lead with well-defined end, almost coinciding with real end of repolarization (**A**). If same T loop of case **A** is rotated to be perpendicular (**B**) or almost perpendicular (**C**) to lead axis, low ST-T amplitudes result. In cases **B** and **C**, when peak-to-peak amplitude sinks to <50 mV, lead is excluded from further analysis. Cases **D**, **E** and **F** correspond to wide loops. Case **D** shows the influence of wide T loop and angle giving rise to T wave that ends before overall end of repolarization, while case **F** shows the maximal influence of a wide T loop with its terminal part perpendicular to lead axis.



preparation (35) the peak of the T wave represents the end of the repolarization in the epicardium, the end of the T wave reflects the end of repolarization in M cells and the descending limb of the T wave is associated with the repolarization in the endocardium. T_{PE} is a measurement of transmural dispersion; however, it is difficult to associate with the

standard ECG, as T_{PE} is a concept derived from the ECG of the wedge preparation. However, some studies have quantified the transmural dispersion (36) from the ECG. In addition, T_{PE} may replace measurement of T_{WD} during ischemia, as measurement of T wave onset is unstable when the ST-segment is modified. (15)

Other indicators of spatial dispersion

The amplitude and symmetry of the T wave, and the relationship between its areas have been proposed to be arrhythmogenic markers. During ischemia, changes in the symmetry and amplitude of the T wave (37) were consistent with the computational model (38) that simulated ischemia. Other studies found changes in the amplitude, area and symmetry of the T wave related to exercise stress test, (33), antiarrhythmic drugs and PVS, (40) compared to controls.

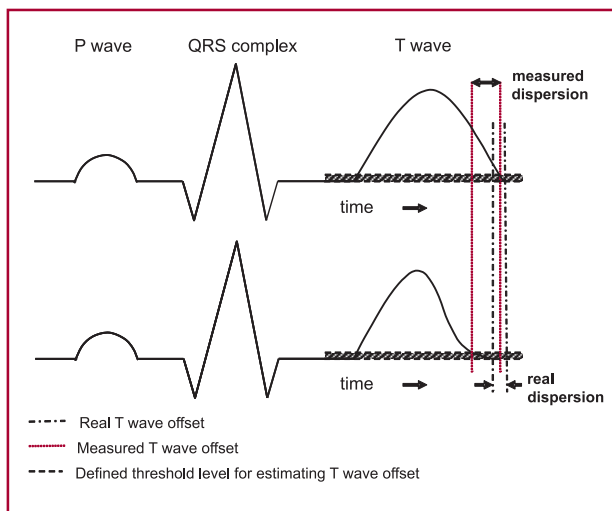


Fig. 2. QT interval dispersion between two hypothetical electrocardiographic leads. Two hypothetical T waves of the same amplitude have different offset when the heart vector becomes perpendicular to the axis of one of the leads. This results in "real dispersion" (vertical dash-dot-dash line). Different proportion of the final part of the two T waves is below the threshold level defined with an automatic threshold method (horizontal dashed line). This leads to the "measured dispersion" (vertical dotted line), which is different from the "real dispersion".

SPATIAL DISPERSION INDEXES ASSOCIATED WITH T WAVE SHAPE

The Singular Value Decomposition (SVD) is a mathematical transformation based on the correlation between signals obtained in this case from the standard ECG. In general, SVD is applied to the eight ECG leads reciprocally independent (I, II, V_1 - V_6) and the information is reconstructed in an optimal orthogonal space of eight pseudo-leads (S_1 ... S_8) as illustrated in Figure 3. In that space, S_1 will contain the maximal energy or eigenvalue (λ_1) in that direction, S_2 will contain the maximal energy (λ_2) perpendicular to S_1 , S_3 will contain the maximal λ_3 perpendicular to the first two pseudo-leads and so on. The components S_1 S_2 S_3 define the dipolar cardiac electrical vector component containing around 98% of the total energy ($\lambda_1\lambda_2\lambda_3$), while the components S_4 ... S_8 contains the remaining 2% of energy which cannot be represented through the dipolar model (λ_4 ... λ_8).

T wave residuum

T wave residuum (T_{WR}) estimates the relative energy of the non-dipolar components related to the total energy. The dipolar component is the electrical 3D representation, while the non-dipolar component is related to the local heterogeneity of the myocardium that is not represented in the cardiac electrical vector. One study (41) was designed to compare QT_D with T_{WR} in patients with hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM) and acute myocardial infarction (AMI), and in normal subjects. The T_{WR} was useful to discriminate between normal and abnormal subjects, and there was a low correlation between QT_D and non-dipolar components (Table 1). A retrospective study (42) with 10 years of follow up performed in dead and live patients with heart disease reported that T_{WR} and absolute T wave residuum (unnormalized T-wave morphology dispersion) were good predictors of mortality (Table 1). In addition, in a model of animal heart, the T_{WR} increased during PVS. (43)

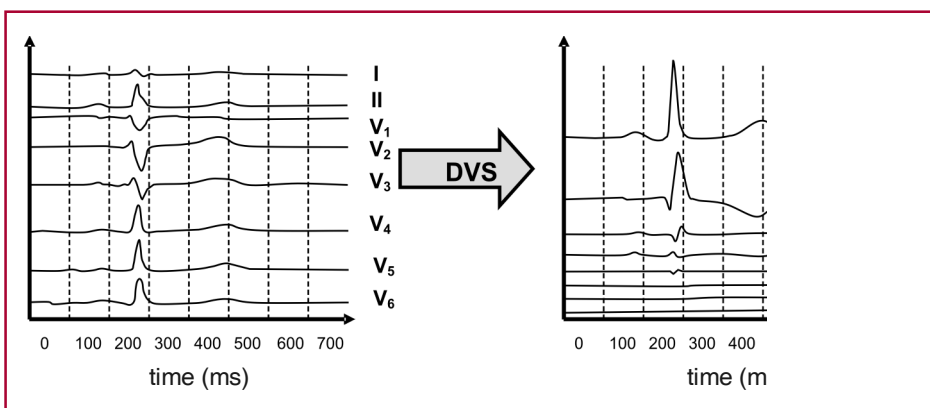


Fig. 3. Standard ECG (left panel) and the 8 pseudo-leads obtained by applying singular value decomposition (right panel). See the maximal amount of energy concentrated in the first three directions S_1 S_2 S_3 after applying SVD.

Wave front direction of repolarization

Ventricular gradient (V_G), investigated by Wilson et al. 75 years ago (44) is the vector resulting from instantaneous ventricular vectors of depolarization and repolarization and can detect primary T wave changes concealed by secondary modifications. (44) This phenomenon can occur under circumstances of ischemia associated with paroxysmal tachycardia, bundle branch block (45, 46), ventricular hypertrophy (47) or Wolff-Parkinson-White syndrome; in these conditions a positive T wave may be abnormal and a negative T wave may be normal. In these cases, estimation of V_G provides a solid basis for the analysis of the surface ECG. (48)

Total R T cosine (T_{CRT}) is a new descriptor of repolarization heterogeneity that quantifies the deviation between the directions of ventricular depolarization and repolarization. It revives the old concept of ventricular gradient. T_{CRT} is estimated as the cosine angle between the direction of the QRS complex and the T wave dominant vectors in an optimal orthogonal space obtained by the SVD from the surface ECG. Negative values of T_{CRT} express that the T-wave loop is opposite to the QRS loop, while positive values represent normal cardiac activation and recovery. T_{CRT} is better than the QT_D to predict risk following a myocardial infarction (49) and SD. (50) It is also useful to quantify the circadian variations of heterogeneity (51) and to discriminate between normality and abnormality in HMC (Table 1). In the same way, estimation of the total angle principal component-to-T (T_{PT}) is useful to study repolarization excluding depolarization as it can discriminate between control or normality in the SVD space (34) (Table 1).

Recently, T_{CRT} was compared to V_G (53) in healthy volunteers and in patients with heart disease (with exercise-induced ST-depression) during Valsalva maneuver. Ventricular gradient and T_{CRT} detect changes in the angles in depolarization and repolarization front waves and contain non-redundant information, probably related to the methodology used for calculation.

Repolarization complexity

As the clinical use of Frank lead system is minor, leads XYZ of the vectocardiogram (VCG) can be obtained by applying Dower inverse transformation (54) or SVD to standard 12-lead ECG.

Under normal conditions, the morphology of T_{WL} is determined by the three eigenvalues $\lambda_1, \lambda_2, \lambda_3$ relative to the principal axes: S_1, S_2, S_3 . In addition, it is equivalent to the vector calculated from integrated T-wave amplitudes of the XYZ leads containing the dipolar energy. (41)

When VRD increases, its eigenvalues change and this variation can be quantified through the repolarization complexity (C_R), mathematically defined as λ_2/λ_1 .

In general, the energy of T_{WL} is concentrated in its preferential plane (the eigenvalues λ_1, λ_2) and the λ_2/λ_1 index allows quantification of the roundness of the T-wave loop. A completely planar T-wave loop will have a $\lambda_3 = 0$.

The R_C differentiates LQTS patients from control subjects (55) and patients with cardiovascular risk. (56) Relative changes of λ_2 and λ_3 regarding total energy are also considered R_C ; Table 1 shows increased R_C in patients with LQTS and during PVS in a experimental model in animals. (43)

Several studies demonstrated that inhomogeneities of repolarization change the morphology of the T_{WL} . A loss of planarity and increased roundness in the T_{WL} was seen in patients with LQTS and in those following a myocardial infarction compared to controls, while QT_D in XYZ could not discriminate between controls and patients with these conditions (57) (Table 1). During coronary angioplasty, the area and the complexity of the T-wave loop are more reproducible compared to the dominant angle of the T_{WL} .

Recently, several VCG parameters obtained from SVD and VCG obtained by Dower (58) were compared; the conclusion was that they are different not only in how they are calculated but also in their results.

CONCLUSIONS

Several indexes can quantify spatial VRD based on different intervals or on the morphology of the T wave.

Standard 12-lead ECG contains the regional information of VR, but the real dispersion of ventricular repolarization is not likely to be obtained through the assessment of Q_{TD} . It is evident that the technical limitations and the projection of the cardiac vector to the linear leads is a restriction for the interpretation of the QT_D and hampers the comparison among studies.

TW_D , evaluated on an integrated signal, might be measuring the dispersion of the apex-to-base gradient, transmural gradient or a combination of both; it should be studied in humans and in diverse conditions. In addition, T_{PE} was measured in few clinical studies and resulted controversial for LQTS and ischemia, (36) while its symmetry and area have been proposed as arrhythmogenic markers although it is not used widely.

Both T_{WR} and T_{CRT} detected patients with intermediate and high risk who could benefit from implantation of an ICD. However, the association between the physiopathological mechanisms and these indexes needs further evaluation.

Multilead systems were used to assess R_C for certain diseases. It is currently analyzed in standard leads and although some commercial devices include measurement of R_C , its role in clinical practice is not well defined yet.

Analysis of T_{WL} is better than 12-lead ECG to detect abnormalities of VR or to determinate certain

diagnosis. Additional studies are needed for the development and validation of new parameters in order to reveal local singularities of the T_{WL} in diseases and incorporate it as a diagnostic tool in routine electrocardiography.

The literature proposes different markers for risk stratification of cardiovascular mortality by ECG analysis; some of them are limited to predict risk, others are promising but have not been validated clinically yet, while some others have been validated as indexes of mortality. However, it would be important to perform a consensus with the participation of experts in order to unify the criteria for estimating the different parameters of spatial VRD, and to establish the most significant indexes and their prognostic value for the different heart diseases. Expert committees have established recommendations for the use of other computerized ECG measurements, such as high-resolution or signal-averaged electrocardiography for the analysis of late potentials (59) or heart rate variability (60).

RESUMEN

Cuantificación de la dispersión espacial de la repolarización ventricular cardíaca a través del electrocardiograma de superficie

La dispersión de la repolarización ventricular (DRV) está determinada esencialmente por la heterogeneidad de los potenciales de acción en diferentes regiones del miocardio. Con frecuencia el corazón responde a ciertos estados fisiopatológicos con la producción de un incremento de la DRV, fenómeno éste que puede devenir en una arritmia ventricular maligna y/o en la muerte súbita. Hace 25 años, con el objetivo de identificar a pacientes de riesgo cardíaco, se comenzó a cuantificar la DRV con diversos índices obtenidos por procesamiento computacional del electrocardiograma. Estos índices se basan en la detección de cambios en la duración o en la forma en la onda T en presencia de cardiopatías. En este trabajo se presenta una revisión de los índices de dispersión espacial y su potencialidad como herramienta de apoyo al diagnóstico de riesgo cardíaco.

Palabras clave > Morfología de la onda T - Dispersión del intervalo QT - Descomposición del ECG - Factores de riesgo - Dispersión de la repolarización ventricular - Muerte súbita

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