Beat-To-Beat Ventricular Repolarization Duration Variability Assessed By Cardiac Acceleration and Deceleration Phases in Athletes

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Abstract

Dynamic ventricular repolarization duration (VRD) and RR-interval coupling relates to autonomic control and myocardial electrical stability. Phase-rectification of RR-interval series separates acceleration (AC) and deceleration (DC) phases, reflecting sympathetic and parasympathetic influences on heart rate, respectively. To assess the effect of physical conditioning status on dynamic VRD and phase-rectification-driven RR-interval coupling. Controls (n = 10) and Athletes (n = 10) groups underwent 15 min resting ECG. RR-interval histogram was calculated, with 100 ms width classes, ranging from 700 ms to 1300 ms. RR-intervals and respective VRD, defined as the segment between R-wave peak and the apex of the T-wave (RTₐ) were grouped. The averages of RTₐ (MRTₐ) and RR-intervals (MRR) were calculated, using the whole series, AC and DC phases, as well as root-mean-squared difference of consecutive RTₐ (RMS-RTₐ). Values were pooled, and compared inter groups and inter classes. Regression lines were calculated, and slope compared between groups (α<0.05). MRTₐ was larger in Athletes than in Controls. MRTₐ increased proportionally to MRR in both AC and DC, and slope was steeper in Athletes. MRTₐ slopes in AC and DC phases did show differences, in both groups. RMS-RTₐ and RMS-RTₐ DC showed no intergroup differences. An inverse correlation between RMS-RTₐ and MRR was found only in Athletes, similarly in both phases. In athletes, VRD is larger, for all RR-interval durations, and RTₐ slope steeper than in heathy sedentary, indicating faster adaptation. In athletes, VRD variability decreases as RR-interval increases, indicating a beneficial effect of fitness on repolarization stability.

Keywords: Ventricular repolarization duration; Cardiac cycle length; RR-interval histogram; Heart rate adaptation; Phase rectified RR-interval

Abbreviations: ECG: electrocardiogram; VRD: ventricular repolarization duration; AC: acceleration; DC: deceleration; RR: interval segment duration between consecutive R-wave peaks; RTₐ: segment between R-wave peak and the apex of the T-wave; MRTₐ: averages of RTₐ; MRR: averages of RR-intervals; RMS: root-mean-squared; HR: heart rates; ECG: electrocardiogram; VO₂MAX: maximal oxygen consumption; METs: metabolic equivalents; SD: standard deviation; HRV: heart rate variability; PRSA: phase-rectified signal averaging

Introduction

Regular exercise leads to structural and functional adaptations that improve cardiac function as a consequence of increased demand on the cardiovascular system [1,2]. Among them, are i) increases in left ventricular mass and volume [3], ii) reduced resting heart rates (HR) induced by increased vagal tone [4,5] and, iii) electrical changes, characterized by the redistribution of electrical charges on myocardial surface, as indicated by increases in both ventricular activation amplitude and repolarization duration (QT-interval), which are observed in the resting electrocardiogram (ECG) waveforms of athletes [6,7].

The relationship between ventricular repolarization duration (VRD) and cardiac cycle length is a valuable tool to assess cardiac adaptation to autonomic input [8]. It is well known the VRD adapts to changes in HR, which makes it difficult to compare the repolarization interval for subjects with different physiological conditions. The effects of exercise training on VRD indices may have an autonomic nature, partially explained by an increase in cardiac vagal activity [9] and, additionally, have not been extensively studied.

Recently, an approximate isolation of distinct autonomic contribution on HR has been possible by assessing the capability of RR-interval series to accelerate (AC) and decelerate (DC), representing, respectively, sympathetic and parasympathetic contribution phases. To further accomplish this task, it is initially detected whether a particular RR-interval increases or decreases relatively to the previous one, and then such particular RR-intervals are separated in new series [10].

Previous studies reproduced the VRD dependence on the cardiac cycle lengths, indicating that the separation by RR-interval classes can be useful to compare different populations based on pairing the bands [11]. Additionally, depending on vagal stimulus intensity, the ascending rate of the RR-interval series (DC phase) changes accordingly, determining steepest slope variation [12]. Then, discriminating the DC and AC series, a strongest vagal stimulus would determine faster ventricular adaptations (steepest curve) and vice-versa, potentially affecting the relation between them (hysteresis). Thus, the objective of the study was: i) to develop an analysis that relates the ventricular repolarization duration and its variability to RR-intervals, stratified by RR histogram classes, comparing healthy sedentary subjects and athletes, and ii) to assess AC and DC phases of RR-interval series, discriminating sympathetic and parasympathetic effects.

**Materials and Methods**

**Study population**

The analyzed signals were extracted from an existing high resolution ECG database as described previously [5]. The study protocol was approved by Instituto Nacional de Cardiologia Ethics Committee and informed consent was obtained from each volunteer. Ten elite runners ([mean ± SD] 8.9 ± 3.2 years of training; six to eight training sessions/week; 90 to 120 min/session; 90 to 110 km/week) were enrolled (Athlete group). A group of 10 healthy sedentary volunteers were included as control (Control group). Inclusion criteria, physical assessment procedures and maximal oxygen consumption (VO$_{2\text{MAX}}$) estimation protocol have been published elsewhere [5].

VO$_{2\text{MAX}}$ was divided by the constant 3.5 mL kg$^{-1}$ min$^{-1}$ to be converted into metabolic equivalents (METs). Control and Athlete groups were separated according to estimated VO$_{2\text{MAX}}$ by arbitrarily defining as less than 11.5 METs for sedentary controls and more than 16.0 METs for athletes.

Both groups were adjusted by age, gender and matched by anthropometric data (Table 1). The aim of anthropometric data intergroup matching was to minimize inter-group physiological and anthropometric variability, thus reducing potential effect of thoracic geometry on surface ECG signals.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controls</th>
<th>Athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>29.0 ± 5.4</td>
<td>24.4 ± 7.2</td>
</tr>
<tr>
<td>BMI (Kg/m$^2$)</td>
<td>23.8 ± 3.8</td>
<td>20.7 ± 1.9</td>
</tr>
<tr>
<td>BSA (m$^2$)</td>
<td>1.8 ± 0.2</td>
<td>1.8 ± 0.2</td>
</tr>
<tr>
<td>APTD (cm)</td>
<td>21.3 ± 1.9</td>
<td>21.1 ± 1.2</td>
</tr>
<tr>
<td>LLTD (cm)</td>
<td>28.1 ± 3.2</td>
<td>28.0 ± 1.2</td>
</tr>
<tr>
<td>METs</td>
<td>8.7 ± 1.9</td>
<td>19.6 ± 1.3*</td>
</tr>
</tbody>
</table>

*Table 1: Anthropometric and demographic characteristics (mean ± SD) of the subjects who participated in the study. BMI: body mass index; BSA: body surface area; APTD: anteroposterior thoracic diameter; LLTD: laterolateral thoracic diameter; METs: metabolic equivalents *p = 0.001.

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Signal acquisition and processing
The high resolution ECG signals were acquired using modified bipolar Frank XYZ orthogonal leads shortly after application of the questionnaire and physical examination. Before a 15 min continuous signal acquisition, subjects remained in the supine position for 5 min for stabilization of autonomic modulation after the change from the orthostatic position. Detailed description of the equipment was previously described and digital data were processed with custom-made pattern recognition software [13].

Wave detection
The analysis of the RR-interval length was carried out after detection of the QRS complex. Artifacts and ectopic beats were excluded by correlation, precocity and visual inspection, by one expert.

The analysis of the VRD was carried out with the signal low-pass filtered at 15 Hz (Butter-worth, 2nd order). The distance between the top of the QRS complex (R) and the apex of the T wave (T) in normal beats defined RTA interval, which was employed in a sole purpose of analyzing repolarization adaptation over instantaneous cardiac cycle [11]. The R point was defined as the absolute maximum of the R-wave, as well as the apex of the T-wave (T, Figure 1). The RR- and RTA-intervals were analyzed on X lead.

![Figure 1: Identification of the apex (point) on R- and T-waves, which allowed precise identification of the ventricular repolarization duration by RTA interval.](image)

Instantaneous RR and RTA interval analysis
The histogram was constructed for each individual RR-interval series, and divided into classes of 100 ms width, ranging from 700 ms to 1300 ms, which represents a variation between 46 and 100 bpm in HR. For each histogram class, and respective to each RR-interval series, it was calculated mean (MRR), standard deviation (SDRR) of consecutive normal RR-intervals, and mean (MRTA), standard deviation (SDRTA) and root-mean-squared difference (RMSRTA) of consecutive normal RTA intervals. Only pairs of consecutive normal RR and RTA intervals for individual series that lied inside a particular class of the RR histogram were analyzed together.

For a particular histogram class (class) of the i-th series, containing N class,i RR-intervals, the calculus of the mean (Mx i, class), standard deviation (SDx i, class), root-mean-squared difference (RMSx i, class) of the normal RR and RTA intervals was performed as follows:

\[
M_{x,i,\text{class}} = \frac{\sum_{k=1}^{N_{\text{class},i}} x_k}{N_{\text{class},i}} \quad (1)
\]

\[
SD_{x,i,\text{class}} = \sqrt{\frac{\sum_{k=1}^{N_{\text{class},i}} (x_k - M_{x,i,\text{class}})^2}{N_{\text{class},i}}} \quad (2)
\]

\[
RMS_{x,i,\text{class}} = \sqrt{\frac{\sum_{k=1}^{N_{\text{class},i}} (x_k - x_{k-1})^2}{N_{\text{class},i}}} \quad (3)
\]

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where x represents either RR or RT\textsuperscript{a} interval.

For each histogram, classes with 20 or less intervals were excluded of analysis to avoid bias due to lack of statistical precision.

The values of the variables \( Mx_{\text{class}} \), \( SDx_{\text{class}} \), and \( RMSx_{\text{class}} \) were aggregated to the respective histogram class. The pooled mean \( (Mx_{\text{class}}) \), standard deviation \( (SDx_{\text{class}}) \) and root-mean-squared difference \( (RMSx_{\text{class}}) \) of RR and RTA intervals for each histogram class, weighted by respective degree-of-freedom \( (\eta_{\text{class}}) \), were calculated according to:

\[
Mx_{\text{class}} = \frac{\sum_{i=1}^{20} Mx_{i,\text{class}} \cdot (\eta_{i,\text{class}} + 1)}{\sum_{i=1}^{20} (\eta_{i,\text{class}} + 1)}
\]

\[
SDx_{\text{class}} = \sqrt{\frac{\sum_{i=1}^{20} (SDx_{i,\text{class}})^2 \cdot \eta_{i,\text{class}}}{\sum_{i=1}^{20} \eta_{i,\text{class}}}}
\]

\[
RMSx_{\text{class}} = \sqrt{\frac{\sum_{i=1}^{20} (RMSx_{i,\text{class}})^2 \cdot (\eta_{i,\text{class}} + 1)}{\sum_{i=1}^{20} (\eta_{i,\text{class}} + 1)}}
\]

where x represents either RR or RT\textsuperscript{a} interval.

The variables \( MRT^a_{\text{class}} \) and \( RMS-RT^a_{\text{class}} \) were plotted and correlated with \( MRR_{\text{class}} \).

### Instantaneous AC and DC analysis

RR-interval histograms in AC and in DC phases were also built, following the rules described above. RR-interval in AC (RR\textsuperscript{AC}) and in DC (RR\textsuperscript{DC}) phases was classified accordingly. To further accomplish this task, it was initially isolated the data points as either acceleration (AC) or deceleration (DC) phases. If a particular RR-interval increased relatively to the previous one, a DC interval occurred. As the instantaneous RR-interval increased, it characterized parasympathetic input (DC; lozenge symbols in Figure 2). Conversely, a sympathetic effect on the cardiac cycle length was represented whenever the RR-interval decreased relatively to the previous one, and AC interval was defined (AC; represented by circle symbols in Figure 2). After RR-intervals classification, RT\textsuperscript{a} intervals histograms were built, respectively, following the correspondent RR-intervals phases: RT\textsuperscript{a, AC} derived from RR\textsuperscript{AC} and RT\textsuperscript{a, DC} from RR\textsuperscript{DC} intervals.

### Statistical analysis

RT\textsuperscript{a} and RR of each subject were pooled and averaged in a class-by-class basis in Controls and Athletes groups, and compared intergroup by non-paired Student t-test. Kruskal-Wallis ANOVA was employed to compare VRD inter classes. The RT\textsuperscript{a} length and its variability (MRT\textsuperscript{a} and RMS-RT\textsuperscript{a}) was analyzed employing all beats as well as beats selected from AC and DC phases, MRT\textsuperscript{a, AC} MRT\textsuperscript{a, DC} RMS-RT\textsuperscript{a, AC} and RMS-RT\textsuperscript{a, DC} respectively. Regression lines of MRR vs. MRT\textsuperscript{a}, and RMS-RT\textsuperscript{a}, were analyzed and angular coefficients (slope) compared between Controls and Athletes groups using non-paired Student t-test. Correlation coefficients (r) were assessed by Pearson correlation tests. All tests considered the significance level \( \alpha < 0.05 \).

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Results

Linear correlation coefficient (r) and respective angular coefficient (slope) of regression lines between MRR and other each pooled MRT\textsuperscript{a} and RMS-RT\textsuperscript{a} variables in both AC and DC phases are presented in Table 2. The r values were significant for all regression lines except in Control group for RMS-RT\textsuperscript{a}. No intergroup slopes differences were observed for all comparisons (p > 0.05).

<table>
<thead>
<tr>
<th></th>
<th>MRR vs. MRT\textsuperscript{a}</th>
<th>MRR vs. RMS-RT\textsuperscript{a}</th>
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<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Athletes</td>
</tr>
<tr>
<td>AC</td>
<td>r</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.98*</td>
<td>0.99*</td>
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<tr>
<td></td>
<td>slope</td>
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<tr>
<td></td>
<td>0.0933</td>
<td>0.1112</td>
</tr>
<tr>
<td>DC</td>
<td>r</td>
<td></td>
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<tr>
<td></td>
<td>0.98*</td>
<td>0.99*</td>
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<tr>
<td></td>
<td>slope</td>
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<tr>
<td></td>
<td>0.0981</td>
<td>0.1169</td>
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</table>

Table 2: Correlation and slopes of pooled mean RR-interval (MRR) vs. pooled mean ventricular repolarization parameters (MRT\textsuperscript{a} and RMS-RT\textsuperscript{a}) in AC and DC phases:
*p < 0.05, RMS: pooled root-mean-squared.

The pooled MRT\textsuperscript{a}\textsubscript{AC} and MRT\textsuperscript{a}\textsubscript{DC} were presented for each group, respectively, in figure 3 (a) and (b), as a function of pooled MRR. MRT\textsuperscript{a} showed significant difference between groups for all MRR classes (p < 0.05).

The MRT\textsuperscript{a} showed no significant intra-group differences between AC and DC phase, in all MRR classes (p > 0.05). The interclasses comparison of MRT\textsuperscript{a} variables showed significant difference in both groups (p < 0.05, Figure 3 (a) and (b)).

The pooled RMS-RT\textsuperscript{a}\textsubscript{AC} and RMS-RT\textsuperscript{a}\textsubscript{DC} were presented for Athletes group in figure 4, as a function of MRR. The slopes showed no significant difference between AC and DC phases (p > 0.05). In Control group, no significant linear correlation (r) between MRR and RMS-MRT\textsuperscript{a} was found.

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Figure 3: Pooled MRT\textsuperscript{A} phase analyses (AC and DC) for (a) Control and (b) Athletes groups as a function of RR-intervals. Note that slopes in Athletes group are steeper than in Control group in AC and DC phases.

Figure 4: Pooled RMS-RT\textsuperscript{AC} and RMS-RT\textsuperscript{DC} as a function of RR-intervals and respective slopes in Athletes groups. Linear regression lines in AC and DC phases indicate an inverse dependence of RT\textsuperscript{A} variability vs. RR-interval duration.

Based on the total number of RR-intervals suitting a particular histogram class, the percent value (mean ± SD) of RR-intervals pairs rejected as either one not pertaining to the same histogram class was 30.9% ± 10.5% for the Control group and 50.5% ± 16.5% for Athletes. Figure 5 shows RR-interval pairs histograms for each group.

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Discussion

This study applied a computerized method to automatically analyze the relation between VRD and cardiac cycle length in high performance athletes and healthy sedentary controls. Utilization of \( R^p \) interval as a measure of VRD instead of the conventional \( QT \) interval has been proved to be feasible and has several advantages [14,15]. Most important, \( R^p \) is easily identified in the majority of the cases because it is limited by two sharp edges of the ECG (the R- and the T-wave apexes). The conventional \( QT \) interval requires accurate identification of the \( Q \)-wave and the \( T \)-wave offset, and the latter is subject to controversies related to optimal definition of reference offset point in visual identification processes and to adequacy of automated computer algorithms [14]. The increased accuracy in the VRD measurement based on \( RT^A \) definition, on the other hand, allows the application of non-interactive software [14]. Furthermore, it has been demonstrated that most of both VRD variability and coherence to RR-interval series is concentrated in the first portion of the \( QT \) interval (ending at \( T \)-wave apex) [16]. In fact, recent studies indicate that VRD offset assessed either at the peak of the \( T \)-wave or the inflexion point after the peak show high correlation with the VRD measured at end of the \( T \)-wave, validating this measurement for more accurate determination of ventricular variability [15,17]. The \( RT^p \) calculation by adjusting the parabola to determine the peak of the \( T \)-wave has been previously presented and shown to preserve VRD variability accuracy [11].

The \( QT \) interval reflects the time window extending from the onset of the ventricular activation to the offset of repolarization. Particularly, abnormal repolarization syndromes are often expressed as extreme \( T \)-wave form variation and/or prolongation and all of them are associated with life-threatening heart rhythm disturbances [17,18]. However, the determination of abnormal \( QT \) interval is challenging due to the frequent lack of differentiation of the end of the \( T \)-wave. Furthermore, it undergoes dynamic variations depending on age, instantaneous HR, autonomic status, medications, etc [17].

In both athletes and healthy sedentary subjects, \( QT \) interval duration shows significant dependence on the corresponding cardiac cycle length [8]. Additionally, the autonomic nervous system, by vagal tone predominance (parasympathetic), may also cause \( QT \) interval to prolong, and increase its temporal dispersion in well-conditioned subjects [9]. In elite athletes, besides the vagal tone predominance and, consequently, resting bradycardia, which increase the absolute values of \( QT \) interval duration [19], an increase in left ventricular mass is considered to be an otherwise benign physiological phenomenon, also known as “athlete’s heart”. Thus, an isolated slightly prolonged \( QT \) interval in athletes may reflect delayed repolarization as a result of increased ventricular wall thickness [20] and/or slow heart rates [21]. Thus, the analysis of autonomic contributions to VRD dependence may be a valuable tool to assess VRD adaptation to cardiac cycle length in this population.

Because of the substantial inter-subject variability of the \( QT/RR \)-interval ratio, no mathematical formula holds true for a definite and accurate correction regarding HR status. Consequently, a RR-interval correction formula that performs well in one subject may overcorrect or under correct the \( QT \) interval in other [21]. Hence, the present study relates VRDs to respective RR-intervals, preserving the first from correction formulas and allowing more unbiased comparison between healthy subjects and athletes.

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The behavior of VRD was analyzed by grouping $RT^a$ interval lengths according to the histogram classes of normal $RR$-intervals. This procedure made it possible to cluster beats expected to bear similar influences due to instantaneous time factors. Additionally, $RR$ series were appropriately isolated of distinguish autonomic contribution on HR variability (HRV), by assessing the capability of the heart to accelerate or to decelerate, respectively, sympathetic (AC phase) and parasympathetic (DC phase) contributions.

The $RR$-interval phase-based processing has been successfully employed in previous studies to assess cardiac vagal modulation by phase-rectified signal averaging (PRS) procedure [10,12]. The technique has been shown to be effective in distinguishing athletes from sedentary healthy volunteers. It hypothesized that depending on vagal stimulus intensity, the rate of ascends of the $RR$-interval series would change accordingly, determining slope variation. Thus, a strongest vagal stimulus would determine a steepest slope and vice-versa, affecting the DC measure.

In both groups, the mean VRD measures were strongly dependent on the instantaneous $RR$-interval, confirming previous findings [11,22]. In a physiological range of variability (700 to 1300 ms), pooled $MRT^a$ are greater at larger $MRR$ intervals (Figure 3). This relation held a strong linear dependence ($r > 0.98$; Table 2). $MRT^a$ intra group comparison between AC and DC phases showed no significant differences either in Controls or in Athletes. Thus, it was not possible to isolate any potential hysteresis in VRD adaptation using histogram approach, in the $RR$-interval range analyzed, indicating that beats under a particular $RR$-interval class showed similar repolarization duration regardless cardiac phase. Because AC phase corresponded to sympathetic and DC phase to parasympathetic inputs of the $RR$-interval, based on current data, autonomic phase did not significantly impact repolarization duration in addition to $RR$-interval class itself, in the analyzed range.

On the other hand, beat-to-beat VRD variability has been considered a measure of ventricular autonomic modulation and has prognostic implications [22-25]. In the present study, VRD variability was assessed by root-mean-squared of consecutive $RT^a$ intervals ($RMS-RT^a$) in AC and DC phases using the $RR$-interval histogram approach. In athletes group, $RMS-RT^a$ showed an inverse linear relation with $RR$-interval class, in both phases, evidencing that VRD variability increased as $RR$-interval decreased and vice-versa (Figure 4). Although this inverse relation has been previously reported by Barbosa, et al [11], to the best of our knowledge, this is the first report of the phenomenon in high performance athletes. Although the interpretation of those observations may seem elusive, reduction of VRD variability as $RR$-interval increases clearly points to a beneficial effect of physical conditioning on ventricular repolarisation stabilisation. Those observations were not clearly evident among Controls (Data not shown).

$RR$-interval pairs in both groups (Figure 5) were frequently placed in classes ranging from 800 to 1100 ms, allowing appropriate class-by-class comparison. Additionally, a larger total number of $RR$-interval pairs were rejected in Athletes than in Controls, as consecutive intervals not pertaining to the same class were expected to occur due to larger inter-cycle length variation in the formers.

Study limitations include:
1. Small sample size,
2. Two physiologically well-defined groups,
3. $VO_{2\text{MAX}}$ was estimated indirectly,
4. Groups were loosely matched by age.

It should be emphasized that the present method has not been tested as a risk stratification tool for clinical conditions related to arrhythmias, particularly in athletes. The ability of the method to assess the $RR$-interval to VRD relationship needs further assessments in different clinical settings.

Conclusion

Using RR-interval histogram-based approach, VRD shows linear dependence on $RR$-interval in both healthy sedentary and high performance athletes. This dependence shows no remarkable differences between acceleration and deceleration phases.

Additionally, VRD in athletes is slightly larger than in healthy sedentary volunteers for equivalent RR-interval classes. Among athletes, beat-to-beat VRD variability shows an inverse relation to RR-interval duration, indicating a potential beneficial of physical fitness on ventricular repolarisation stabilization.

Acknowledgment

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Conflict of Interest

No financial interest or any conflict of interest exists.

Bibliography