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Effects of ECG sampling rate on QT interval variability measurement

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ABSTRACT

Beat-to-beat variability of the QT interval (QTV) has been used as a marker of repolarization lability and sympathetic activation. The aim of this study was to establish ECG sampling rate requirements for reliable QT interval variability measurement.

We measured QTV in high resolution simulated (1000 Hz) and real ECG (1600 Hz; in the supine position during rest and during sympathetic activation upon standing), using time and frequency domain metrics as well as measures of symbolic dynamics for complexity assessment. We successively halved the sampling rate and investigated its effect on the QTV metrics.

Reduction in sampling rate below 400 Hz and 500 Hz, respectively, resulted in a significant overestimation of QTV variability and also affected complexity measurement of QTV. QTV increased during standing compared to the supine measurement. At 100 Hz, the posture related change in QTV was completely masked by the measurement noise introduced by the low sampling rate.

In conclusion, ECG sampling rates of 500 Hz yields a reliable QTV measurement, while sampling rates of 200 Hz and below should be avoided.

1. Introduction

The QT interval of ECG is a measure of ventricular depolarization and repolarization duration. Corrected for heart rate, it is clinically used to diagnose congenital or acquired QT syndromes [1,2]. The QT interval fluctuates from beat to beat and quantification of this so-called QT interval variability (QTV) has received increasing interest as evidence of the association between increased QTV and elevated risk of cardiac and overall mortality is mounting [3–5]. Aside from its potential use for cardiac risk stratification QTV may be used as a noninvasive marker of sympathetic outflow to the heart, because studies in humans have repeatedly demonstrated increased QTV during periods of acute cardiac sympathetic activation, elicited by pharmacological beta receptor activation or by orthostatic challenge, in particular in healthy subjects [6–8] or subjects with structurally normal hearts [9,10]. The level of QTV measured during rest was shown to be directly correlated with cardiac noradrenaline spillover in hypertensive subjects [11], but not in normal subjects or patients with major depressive disorder or panic disorder [12]. A canine model of tachycardia induced heart failure showed a direct correlation between the QTV and integrated left stellate-ganglion nervous activity [13].

Although QTV has been widely studied in various clinical research settings, some basic technical considerations regarding the QT measurement have been insufficiently addressed. Technical aspects are of particular significance since beat-to-beat fluctuations in the QT interval are typically small, with a standard deviation of less than 5 ms when measured at stable heart rates during rest [14]. Addressing the issue of high precision QT interval delineation of body surface ECG, we have previously shown that template matching algorithms specifically dedicated to the analysis of QTV are better suited to measure subtle beat-to-beat changes in QT interval than conventional delineation techniques [15]. At a more fundamental level, however, ECG data acquisition requirements for measuring meaningful QTV have not been thoroughly investigated yet. Previous studies performing QTV analysis used ECG that was sampled at rates as low as 128 Hz [16]. The aim of this study was to systematically explore the effects of sampling rate on QTV metrics, using simulated ECG with predefined QTV as well as real ECG recordings from healthy subjects to offer guidance for ECG recording requirements. Experimental conditions characterized by a low and high sympathetic drive were considered (i.e. resting in the supine position and standing, respectively).

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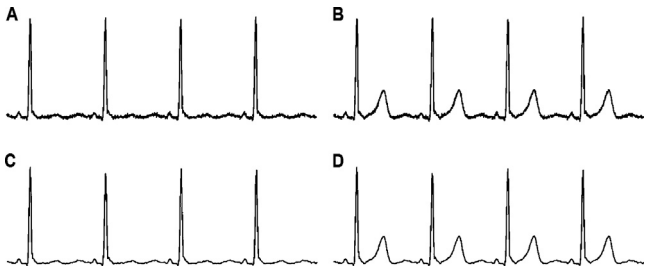

Fig. 1. Examples of simulated noisy ECG with artificially imposed QT variability in the high and low frequency range. (A) T wave acquisition range: 0.6%, sampled at 1000 Hz; (B) T wave acquisition range: 6.4%, sampled at 1000 Hz; (C) T wave acquisition range: 0.6%, sampled at 125 Hz; (D) T wave acquisition range: 6.4%, sampled at 125 Hz.

2. Methods

2.1. Data

2.1.1. Simulated ECG

Simulated ECG signals were generated as described previously [15,17]. Briefly, we obtained a noise-free cardiac cycle (starting from a QRS peak and ending at the subsequent QRS peak) of an ECG (lead II) that was recorded in a healthy 26 years old volunteer. The ECG was digitized with an A/D board of 12-bit resolution at a sampling rate of 1000 Hz. Considering the overall input range (4096 quanta), the two R-peaks spanned a range from 1983 to 2940 quanta (i.e. the R-peak amplitude covered 957 quanta), while the T-wave spanned the range from 1984 to 2246 quanta (i.e. the T wave amplitude covered 262 quanta). Hence, the R-peak and T-wave occupied 23.4% and 6.4%, respectively of the A/D board's range. The T-wave amplitude was scaled by factor k, where $k \in \{0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 1.0\}$, resulting in 10 cardiac beats with decreasing T-wave amplitudes. The 10 cardiac cycles were then repeated 500 times, resulting in ten synthetic signals with 500 cardiac cycles each, characterized by null variability in heart rate and ventricular repolarization duration, but different T wave amplitudes (Fig. 1). White Gaussian noise was superimposed to each of the ten synthetic ECG to provide a realistic signal. The mean value of the noise was zero and the standard deviation was 3% of the T-wave amplitude of the original cardiac cycle.

Artificial QT variability was introduced by modulating the ST interval of the simulated ECG with oscillatory components at frequencies of 0.1 and 0.25 Hz, respectively, mimicking the effects of the low frequency oscillations (LF) that have been associated with the Traube–Hering–Mayer waves that can be observed in the cardiovascular system and the high frequency oscillations (HF) associated with respiration. The power of LF oscillations was 2.2 ms^2 and the HF power was 12.8 ms^2.

2.1.2. Real ECG

The study conformed to the principles outlined in the Declaration of Helsinki. We enrolled 10 healthy athletes (5 males, age: 26.6 years [26.5–28.8]; 5 females, age: 24.8 years [24.7–26.4]. Participants took part in a two-week training camp and measurements were repeated after the first week of training as well as 3–4 days after completing the camp. All participants provided written informed consent. Details have been published previously [18,19]. As part of a study on the effects of overtraining on autonomic cardiovascular control, 3-lead ECG (modified Frank lead system) was recorded at 1600 Hz during rest in the supine position for 30 min and subsequently during standing for another 20 minutes. QTV analysis was performed on the lead with the tallest T-wave, which spanned 4683 quanta on average and represents 27% of the R-amplitude, on average. For the purpose of this study we collated the ECG from all athletes obtained during the three recording sessions, distinguishing only between resting and standing, respectively, thus enabling the assessment of QTV with respect to sampling rate during conditions of low and high sympathetic activity.

2.1.3. Down-sampling

To investigate the effect of sampling rate on QTV measurement, we generated versions of the data down sampled at 500 Hz, 250 Hz and 125 Hz for simulated ECG, and 800 Hz, 400 Hz, 200 Hz and 100 Hz for real ECG, respectively, using a poly phase finite impulse response filter, where the number of taps was $N = 2 \times 20 \times$ (original sampling rate/down-sampling rate) + 1, employing Kaiser windowing. Examples of original and down sampled simulated ECG are shown in Fig. 1.

2.1.4. Beat-to-beat QT interval measurement

For this study, we employed a template matching technique that relies on the recently developed two-dimensional warping (2DSW) algorithm [20]. The method is able to account for complex morphological changes in the ECG waveform and was shown to track QT changes with high precision while being robust toward signal artifacts at the same time. Briefly, the algorithm first identifies QRS complexes automatically and uses these fiducial points to generate an averaged template beat, by employing an improved version of Woody's time delay estimation method [21]. Subsequently, points of interest, such as Q-onset and T-wave end, are marked on the template beat by the operator in a semi-automated manner. Using a two-dimensional grid of so-called warping points, the Euclidean distance between template and each beat in the signal is minimized by warping the ECG waveform piece-wise along the warping points. Relative variations in the QT interval, as annotated by the operator on the warped template, are utilized to measure beat-to-beat changes in QT intervals. Time series of RR and QT intervals were visually checked for missing beats and affected beat-to-beat intervals were removed. We then calculated a set of QTV metrics (see below) for each recording, using the last 256 valid consecutive beats of the ECG.

2.1.5. QTV metrics

To quantify QTV we adopted a set of metrics originally proposed for HRV analysis that have been subsequently used for QTV assessment. Time domain assessment comprised:

- SDQT—standard deviation of QT intervals; in ms,
- RMSSD—root-mean-square of successive differences in QT interval; in ms.

For spectral analysis the series were modeled as an autoregressive process describing the dynamics as a linear combination of past samples weighted by constant coefficients plus a zero mean white noise [22]. The Levinson–Durbin recursive algorithm was utilized to estimate the coefficients of the AR model and the variance of the white noise. The number of coefficients was chosen according to the Akaike's figure of merit in the range from 14 to 18 [23]. Power spectral density was computed from the coefficients of the model and from the variance of the white noise according to the maximum entropy spectral estimation approach [22]. The power spectral density was factorized into spectral components each associate to a real pole or pair of complex and conjugated poles, the sum of which provides the entire power spectral density [24]. Power spectral factorization provided the central frequency of the components expressed in cycles $\times$ $beat^{-1}$, converted into Hz by dividing it by the heart period mean, and decomposition of the overall variance. In the frequency domain we quantified: