Influence of Psychological Stress on Systolic-Diastolic Interval (SDI) Interaction Characteristics Measured from the Electrocardiogram (ECG) Signal

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Abstract

Stress affects the ventricular repolarization and the ventricular mechanical function by increasing the QT interval variability and changing left ventricular ejection fraction respectively. As QT and TQ intervals are considered as surrogates of mechanical systolic and diastolic durations, alteration of QT and TQ interval dynamics can be used to analyse stress related changes in the systolic and diastolic function of the heart. The ratio of QT to TQ interval within a cardiac cycle (i.e. QTTQ ratio) represents the synchronized contraction and relaxation operation of the heart. Besides QTTQ, the ratio of TQ to RR interval (i.e. TQRR ratio) can also be used as a non-invasive measure of the diastolic function to analyse ventricular dysfunction. The stress related alteration of ventricular function is reported in subjects with coronary heart diseases, but the same was not reported in healthy subjects widely. In this study, the effect of stress on systolic-diastolic interval (SDI) ratios is analysed to investigate the stress related alteration in ventricular repolarization in healthy subjects. The study results indicate that stress induction increases the mean QTTQ ratio ($mSDI_{QT\text{-}TQ}$) and decreases the TQRR ratio ($mSDI_{TQ\text{-}RR}$) indicating the effect of stress on systolic and diastolic functions. The increase in $mSDI_{QT\text{-}TQ}$ indicates the increases in the probability of arrhythmogenesis. However, the difference between stressed and unstressed conditions for theses ratio parameters was not significant ($p>0.05$). Alternatively, the variability of SDI parameters (i.e. variance of QTTQ and TQRR ratios) increased significantly, which can detect the induced stress in healthy individuals and significantly ($p<0.05$) differentiated the stressed condition form natural steady state relaxed condition of the same individual. The significant increase in variability in QTTQ and TQRR ratio indicate the increased probability of arrhythmogenesis due to sympathetic activation during stressed conditions.

1. Introduction

Psychological stress is neurologically related with cognitive functions and it increases with the initiation of certain cognitive tasks like problem solving, decision making, playing competitive games, and driving. Stress affects different cardiovascular responses and stress induced autonomic nervous system activation (i.e. mainly the over activation of sympathetic nervous system) might also trigger lethal arrhythmias through alterations of the neural transmissions to the heart both in subjects without and with heart diseases [1-3]. Heart rate and ventricular repolarization characteristics were reported to be affected by stress in several studies [4, 5]. Increase in heart rate (i.e. decreases in RR interval in ECG), heart rate variability, alteration in QT interval (i.e. ventricular repolarization duration), and increases in QT variability are the major changes with stress induction [3, 5-7]. The mechanical function of the ventricles (i.e. Systolic and diastolic function) is also affected by mental stress. Stress can induce myocardial ischemia like exercise induced ischemic responses and contribute to the development of coronary artery diseases (CAD), which affect normal heart function through coronary heart disease[8]. Left ventricular dysfunction in terms of the decreases in left ventricular ejection fraction is also reported in subjects with CAD with the induction of mental stress [9, 10]. Therefore, both the electrical and mechanical characteristics of a normal cardiac cycle are affected by psychological stress. Although stress induced alteration of the left ventricular function is investigated thoroughly in subjects with a history of heart diseases[10, 11], the changes in systolic and diastolic function with stress in healthy human individuals is not reported widely. Hence, the main objective of this study is to investigate the changes in mechanical functionality of the ventricles with stress in healthy subjects from surface ECG.

The contraction and relaxation function of the ventricles is investigated using the cardiac imaging techniques (i.e. Echocardiography, Tissue Doppler Echocardiography) which are not widely available in every clinical setting and relatively costly in comparison to the cheap ECG analysis system. The systolic (i.e. ventricular contraction) and diastolic (i.e. ventricular relaxation and filling) function of the ventricles is controlled by the cardiac action propagation characteristics through the ventricles. Ventricular action potential propagation can be analysed from the different ECG wave interval characteristics from ECG as shown figure 1. The QT interval termed as electrical systole can be considered as a surrogate of mechanical systolic interval within a cardiac cycle of the ECG signal [12, 13]. The TQ interval is considered as a surrogate measure for the electrical diastolic interval and directly affects the QT or systolic interval of the next cardiac cycle [12]. The beat-to-beat QT-TQ interval relationship can be described as the systolic-diastolic interval interaction (SDI) or the balance in the heart's contraction and relaxation operation within one cardiac cycle [14]. Several studies have proposed that the systolic to diastolic interval ratio can be used as an indicator of ventricular dysfunction in cardiovascular disease [12, 15]. A recent study proposed and validated an ECG based measurement technique of systolic-diastolic interval interaction (SDI) characteristics to investigate the synchronized temporal characteristics of the mechanical function of the ventricle[16]. The SDI technique used two measures calculated form the QT, TQ and RR intervals to analyse how alteration in normal VR characteristics affect the normal ventricular function. To the best of our knowledge, the relation between QT, TQ and RR interval in healthy stressed condition is not reported before. In this study, we used this technique to investigate stress induced changes of SDI in healthy subjects, which can provide useful information about mechanical functionality of the ventricles.

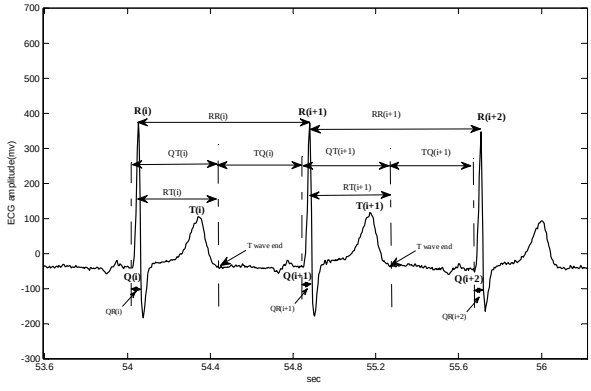

Figure 1. Schematic representation of different ECG wave intervals (RR, QT, TQ, and QR intervals) of two cardiac cycles. QT and TQ intervals are used as electrical systolic and diastolic time intervals.

2. Subjects and ECG processing

ECGs were taken from Physionet Stress Recognition in Automobile Drivers (drivedb) database from this database, a total of 16 healthy subjects' data was taken out of 17 subjects' recordings. One recording (drive01) was dropped from our study due to problems in the T wave analysis. The detail of this study protocol, i.e., driving protocol, driving period, stress measurement and validation of stress level assessment techniques etc. was described by Healy et al. [17].

In this study, we have used 5 minutes ECG and respiration signal during resting and high stress (city driving) conditions. Recordings of resting condition were treated as stable physiological condition and grouped as "Unstressed", whereas recordings of city driving condition were considered as stressed condition data and grouped as "Stressed". The ECG sampled at 496Hz was recorded with a modified lead II configuration for reducing the effect of motion artefact and for better detection of *R* waves.

QT intervals were detected using a semi-automated template-matching algorithm proposed by Berger et al [18]. This algorithm is used in many clinical studies with reliable results describing ventricular repolarization variability. The QT interval was calculated as the difference between Q wave onset and T wave end point (i.e. QTend interval).

2.1. Systolic-Diastolic interval interaction (SDI) parameter measurement

The SDI parameters calculate the beat-to-beat variation of the ratio of electrical systolic (i.e. QT interval) and diastolic time (i.e. TQ interval) interval time series. We used the same methodology to calculate the SDI parameters as described in [16]. The two SDI parameters calculated are termed as QTTQ and TQRR using the following equations, which actually characterizes the beat-to-beat systolic-diastolic interval interaction within each cardiac cycle:

$$\mathrm{QTTQ(i)} = \frac{\mathrm{QT(i)}}{\mathrm{TQ(i)}} \quad (1)$$

$$\mathrm{TQRR(i)} = \frac{\mathrm{TQ(i)}}{\mathrm{RR(i)}} \quad (2)$$

Where, i=1...N and N is the total number detected intervals. The relationship between the proposed TQRR as reported in [16] with the existing QTTQ measure can be shown using the following equations:

$$TQRR(i) = \frac{TQ(i)}{RR(i)} = \frac{TQ(i)}{QT(i) + TQ(i)}$$

$$= \frac{1}{1+\frac{QT(i)}{TQ(i)}} \approx f\left(\frac{QT(i)}{TQ(i)}\right) = f(QTTQ(i)) \qquad (3)$$

Therefore, TQRR actually signify the beat-to-beat variation of the QTTQ (i.e. systolic-diastolic interval interaction) as both can be represented as a function of the beat-to-beat QTTQ parameters (Eqn. (3)) and found more sensitive in characterizing ventricular dysfunction [16] than QTTQ. Detailed analysis technique of the SDI measures was discussed in [16].

The mean and variability of the SDI parameters were calculated by the average and the variances of QTTQ and TQRR time series as described in [16], which were denoted as $mSDI_{QT\text{-}TQ}$, $mSDI_{TQ\text{-}RR}$, $vSDI_{QT\text{-}TQ}$ and $vSDI_{TQ\text{-}RR}$ respectively. The values of cardiac cycle (i.e. RR interval), electrical systolic (i.e. QT interval) and diastolic intervals (i.e. TQ interval) were also reported for comparing with the SDI measures in investigating stress related alteration in ventricular function in healthy subjects. Non-parametric Mann-Whitney U-Test was carried out for statistical comparisons between 'Stresssed' and 'Unstressed' group. A value of $p < 0.05$ was considered significant. All the statistical calculations were carried out in MATLAB R2012b.

3. Results and discussion

Table 1 shows the variation of the calculated values of different ECG wave durations and SDI parameters between the two groups. Heart rate increases with stress induction, which is evident from the decrease in the mean RR interval in the stressed group of the unstressed group subjects. However, the decrease in heart rate is not significantly different between the groups. Heart rate variability parameter, SDRR also increases insignificantly with stress induction.

Electrical systolic and diastolic time intervals calculated as the QT interval and TQ interval respectively, showed a decreasing trend with stress induction. The decrease in QT and TQ interval were also not significantly different between the groups. The variability of QT interval (i.e. SDQT) increases significantly with stress induction in the healthy subjects. SDTQ (i.e. variability of the electrical diastole) increased with stress induction, but the increase was not significant.

The mean of the SDI parameters, $mSDI_{QT\text{-}TQ}$ increased and $mSDI_{TQ\text{-}RR}$ decreased insignificantly with stress induction in the healthy subjects of this study. The variations of these two measures indicate the prolongation of systolic interval and shortening of diastolic time intervals with stress induction, which could be arrhythmogenic [14, 16]. The variability of these two measures calculated as the variance of $SDI_{QT\text{-}TQ}$ and the $SDI_{TQ\text{-}RR}$ (i.e. $vSDI_{QT\text{-}TQ}$ and $vSDI_{TQ\text{-}RR}$ respectively) were found significantly different in the stressed group with $p<0.001$. Both $vSDI_{QT\text{-}TQ}$ and $vSDI_{TQ\text{-}RR}$ increased significantly with stress induction indicating that the temporal synchronization between systolic and diastolic interval is affected by the induction of stress from normal relaxed condition.

Table 1: Variation of heart rate, electrical systolic and diastolic intervals and different SDI parameters due to stress induction in healthy subjects

SDI parameters	Unstressed	Stressed	p value
Mean RR interval (ms)	834.38 ± 110.14	770.50 ± 103.45	0.206
SDRR (ms)	58.38 ± 21.30	68.15 ± 18.37	0.102
Electrical Systolic interval(QT interval in ms)	339.93 ± 29.22	329.68 ± 35.53	0.346
SDQT (ms)	6.63 ± 4.01	13.39 ± 6.31*	0.005
Electrical Diastolic interval(TQ interval in ms)	496.45 ± 88.78	440.82 ± 81.18	0.172
SDTQ (ms)	58.31 ± 20.70	68.65 ± 17.63	0.075
Mean QTTQ ($mSDI_{QT\text{-}TQ}$)	0.69 ± 0.09	0.75 ± 0.10	0.182
Var [QTTQ] ($vSDI_{QT\text{-}TQ}$)	50± 20	160 ± 80*	1.39e-4
Mean TQRR ($mSDI_{TQ\text{-}RR}$)	0.59 ± 0.03	0.57 ± 0.03	0.2345
Var [TQRR] ($vSDI_{TQ\text{-}RR}$)	5 ± 1.1	14 ± 5*	5.079e-5

All values are shown as Mean ± STD.
* indicates significant difference of the feature from unstressed condition

In this study, we investigated the stress-induced alteration in systolic and diastolic interval interaction changes derived from surface ECG, which can provide useful information about the alteration in normal synchronized mechanical function of the ventricles in healthy subjects. The study findings indicate that the variability of both QTTQ and TQRR interval interactions (i.e. $vSDI_{QT\text{-}TQ}$ and $vSDI_{TQ\text{-}RR}$) changes significantly with stress induction in healthy individuals indicating increased complexity in the interaction of systolic and diastolic time intervals.

The heart rate and heart rate variability showed an increasing trend with stress induction, which supports previous study findings [1, 4, 6] though the increases in the duration and the variability were not found significantlyconditionst between the two psychological condition. QT interval (i.e. electrical systolic interval) decreased with stress induction, which supports the previous findings of the shortening of the QT interval in healthy subjects due to sympathetic activation [1, 19, 20]. TQ interval was found to decrease insignificantly with stress indicating that ventricular relaxation duration is affected by stress induction that might alter the proper systolic function[13, 14]. The increase in $mSDI_{QT\text{-}TQ}$ in stressed condition can be arrhythmogenic [14]. The increase in $mSDI_{QT\text{-}TQ}$ indicates that the heart is taking more time for the systolic phase and limits the proper

recovery time of the ventricles by affecting the proper filling of blood for the next cardiac cycle function. $vSDI_{QT-TQ}$ and $vSDI_{TQ-RR}$ both increase significantly with stress induction indication the increase in complexity of the temporal synchronization of the systolic and diastolic function. Only the variability measures of the SDI parameters and the electrical systolic time interval (i.e. SDQT) can significantly differentiate the stressed condition form normal unstressed state. These results represent that stress affects the normal ventricular repolarization process and normal mechanical function of the ventricles, which is reflected through the changes of variability of different ECG wave intervals. The variability and not the duration of the electrical systolic and diastolic interval interaction characteristics can be used for analysing stress-induced changes in synchronized mechanical function of the ventricles, which might provide useful information about arrhythmogenesis.

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