

## Assessment of QT and JT Intervals in Patients With Left Bundle Branch Block

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### Abstract

**Background:** Prolongation of the QT interval is considered a risk factor for cardiac adverse events and mortality. Left bundle branch block (LBBB) lengthens the QT interval. The corrected QT interval (QTc) is most likely overestimated because its prolongation is caused by increases in depolarization duration and not in repolarization.

**Objectives:** In this study, we aimed to apply corrected JT interval (JTc) as an appropriate measure of ventricular repolarization for predicting QTc in a formula.

**Patients and Methods:** The study population consisted of 101 patients with sinus rhythm (SR) and narrow QRS complexes (< 120 milliseconds). All patients underwent electrophysiology studies or ablation. A diagnostic catheter was positioned in the right ventricular apex (RVA) to induce LBBB at two different cycle lengths (CLs; 600 and 700 mv). The intrinsic QRS complex, QT time, and JT time were measured during SR and subsequent RVA pacing. The JTc was derived simply by subtracting the QRS duration from the QTc.

**Results:** Stimulation from the RVA increased the QTc from  $456.20 \pm 38.63$  ms to  $530.67 \pm 47.73$  ms at a CL of 600 ( $P < 0.0001$ ) and to  $502.32 \pm 47.26$  ms at 700 CL ( $P < 0.0001$ ). JTc showed no significant changes with stimulation from the RVA ( $102.97 \pm 11.35$  ms vs.  $103.59 \pm 10.67$  ms,  $P = 0.24$ ). There was no significant correlation between JTc and QRS complex duration. A significant correlation was seen between QRS and QTc at both CLs. The ROC curve indicated that sensitivity of 80% and specificity of 67% were obtained with JTc duration of 92.6 ms.

**Conclusions:** Right ventricular pacing increases the QT interval without increasing the JT interval. Our results confirm that JTc, as an index of repolarization, is independent of ventricular depolarization. Therefore, it can be applied for predicting QTc in patients with LBBB.

**Keywords:** Left Bundle Branch Block, QTc, JTc, Electrophysiology Study

## 1. Background

Prolonged QT interval, which is induced by various cardiovascular and non-cardiovascular diseases and drugs, increases the risk of arrhythmia and adverse outcomes. The QT interval encompasses all ventricular depolarization and repolarization periods (1, 2). The corrected QT interval (QTc), which was introduced in Bazett's formula (3), has been shown to depend on the preceding cycle length (CL) (4). However, because the QT interval contains ventricular depolarization, its value is limited when increased QRS duration causes the QT prolongation. In addition, the QT interval requires adjustments for heart rate and gender in narrow QRS complexes (2).

Although the QT interval is widely used by clinicians, inaccurate measurements or interpretations of it in patients with bundle branch block or other arrhythmias with ventricular conduction delays has led it to be replaced by the JT interval for appropriate measurement of ventricular repolarization. Because QT intervals are prolonged in left bundle branch block (LBBB), the JT interval can be used for risk stratification (2).

## 2. Objectives

In this study, we evaluated the correlations between QTc, corrected JT interval (JTc), and QRS interval. Our aim was to derive a simple, clinically useful formula for predicting QTc through JTc during LBBB. For this purpose, we studied patients with narrow native QRS complexes, in which ventricular pacing from the right apex causes widened QRS complexes, by simulating LBBB.

## 3. Patients and Methods

### 3.1. Study Population

The study population consisted of 101 patients (mean age  $46.70 \pm 16.14$  years, 41% male) who underwent electrophysiological studies. All patients were in sinus rhythm (SR) and had a QRS duration of < 120 ms on electrocardiogram (ECG). All parameters were obtained from standard electrophysiological studies. Patients with wide

QRS complexes ( $\geq 120$  ms) and those who were taking anti-arrhythmic drugs with effects on ventricular depolarization and repolarization were excluded. The study was explained in detail to the subjects, all of whom provided signed informed consent. The study protocol and informed consent documents were approved by our local ethics committee in accordance with the Helsinki Declaration of the World Medical Association (2000).

### 3.2. Study Protocol

In all patients, a diagnostic catheter was positioned in the right ventricular apex (RVA) to stimulate 20 ms below the SR CL. The intrinsic QRS complex, QT interval, and JT interval were measured during SR, and subsequent RVA pacing at 600 and 700 mv CLs was performed to induce LBBB. The QRS complex was measured as the interval between the beginning of the QRS complex and the J point, at which the isoelectric ST segment starts. The longest interval in all leads in which the end of the T wave could be clearly defined was considered the QT interval; this was usually V2 or V3. A tangent on the downward limb of the T wave to the baseline was used for determination of the QT interval when the end of the T wave was indistinct. The JT interval and the JTc were derived simply by subtracting the QRS duration from the QT and the QTc, respectively.

### 3.3. Statistics

Continuous data were expressed as mean value  $\pm$  standard deviation, and categorical data were presented as numbers (percentages). All variables were tested for normal distribution with the Kolmogorov-Smirnov test. Correlations between the parameters were estimated by Pearson's  $r$  (for normally distributed data) and Spearman's  $\rho$  (for non-normally distributed data) correlation coefficients. The ROC curve was used to determine

the area under the curve (AUC), and the optimum JTc cut-off level and the 95% confidence interval (CI) were obtained. Statistical analysis was performed with SPSS 15 for Windows (SPSS Inc., Chicago, IL, USA). P values of  $< 0.05$  were considered significant.

## 4. Results

### 4.1. Electrophysiological Parameters

All patients' demographic data are shown in Table 1. Electrophysiologic study parameters are depicted in Table 2. In sinus rhythm, the mean QRS duration was  $92.07 \pm 16.66$  ms, which increased to  $152.57 \pm 22.42$  ms by RVA stimulation at 600 CL ( $P < 0.0001$ ), and to  $156.92 \pm 18.73$  ms at 700 CL ( $P < 0.0001$ ). Stimulation from the RVA increased the QTc from  $456.20 \pm 38.63$  ms to  $530.67 \pm 47.73$  ms at 600 CL ( $P < 0.0001$ ) and to  $502.32 \pm 47.26$  at 700 CL ( $P < 0.0001$ ). The JTc interval showed no significant changes with stimulation from the RVA ( $102.97 \pm 11.35$  ms vs.  $103.59 \pm 10.67$  ms,  $P = 0.24$ ).

### 4.2. JTc and QTc Correlation

The correlations between QRS and JTc are shown in Table 3. There was no significant correlation between JTc and QRS complex duration, but there were significant correlations between QRS and QTc at both CLs.

The QTc can be predicted by the following formula:  
 $QTc = (0.786 \times QT) + (0.305 \times CL) - 188.733$ .

According to a previous study, 450 ms was considered the cut-off value for prolonged QTc. The AUC in the ROC for JTc was 0.798 (95% CI, 0.682–0.914,  $p < 0.0001$ ) (Figure 1). The curve indicated that a sensitivity of 80% and a specificity of 67% were obtained with a JTc duration of 92.6 ms.

**Table 1.** Demographic and Baseline Data of All Patients

Variable	Mean $\pm$ SD or No. (%)
Age, y	46.70 $\pm$ 16.14
<b>Gender</b>	
Male	41 (40.6)
Female	60 (59.4)
<b>Most common diagnoses</b>	
PSVT	51 (50.5)
PVC	14 (13.9)
WPW	12 (11.9)
VT	6 (5.9)
AFL	5
<b>Risk factors</b>	
DM	13 (12.9)
HTN	23 (22.8)
DLP	17 (16.8)
EF	50.20 $\pm$ 10.51

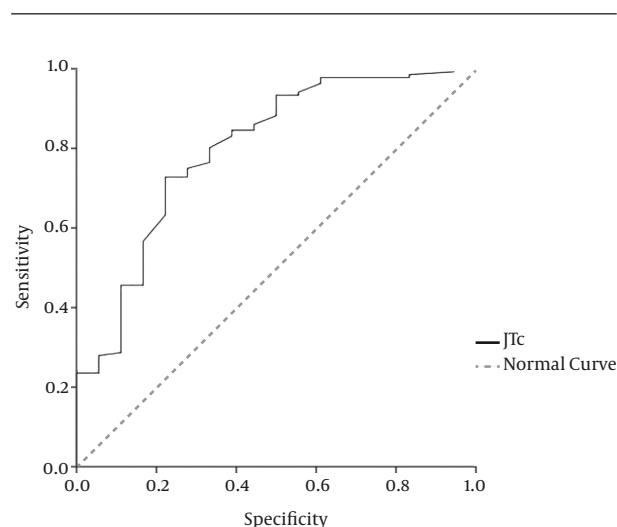
Abbreviations: AFL, atrial flutter; DLP, dyslipidemia, DM, diabetes mellitus; HTN, hypertension; PSVT, paroxysmal supraventricular tachycardia; PVC, premature ventricular contractions; VT, ventricular tachycardia; WPW, Wolf-Parkinson-White.

**Table 2.** Electrophysiologic Study Parameters Before and After RVA Stimulation

Electrophysiology Parameters	Normal QRS Interval	Wide QRS Interval (CL: 600)	Wide QRS Interval (CL: 700)
Heart rate, bpm	80.48 ± 18.04	99.15 ± 4.07	85.77 ± 5.96
QRS interval, ms	92.07 ± 16.66	152.57 ± 22.42	156.92 ± 18.72
QTc, ms	456.20 ± 38.63	530.67 ± 47.73	502.32 ± 47.26
JTc, ms	102.97 ± 11.35	103.59 ± 10.67	97.47 ± 10.38
JT interval, ms	294.79 ± 52.19	270.16 ± 28.41	273.57 ± 29.65

**Table 3.** Correlation Between QRS and JTc/QTc

JTc/QTc	Correlation coefficient	P Value
JTc (600 CL)	.17	0.11
JTc (700 CL)	.08	0.45
QTc (600 CL)	.544	< 0.0001
QTc (700 CL)	.547	< 0.0001

**Figure 1.** ROC of JTc (AUC: 0.798; 95% CI, 0.682 – 0.914)

## 5. Discussion

It is well known that the risk of malignant ventricular arrhythmia is associated with a prolonged QT interval (5). Patients with LBBB have increased QTc, which is calculated with Bazett's formula. Because the increased QT interval in LBBB, caused by prolongation in the depolarization period and not repolarization, the measurement of the QT interval is almost overestimated (2). Theoretically, the QT interval might be judged by instead using the JT interval as a more appropriate measure in arrhythmias with ventricular conduction delays, in which repolarization has been altered (1).

In this study, the intervals for each patient during SR were directly compared with stimulated RVA at two different CLs. The JT interval during stimulation from RVA

was not significantly changed. There was no significant correlation between JT interval and QRS complex duration. It seems that JT intervals with normal QRS at shorter RR intervals may be attributed to the smaller action potential duration at faster heart rates (6). Previous studies demonstrated that the repolarization period is better described by the JT interval than the QT interval because the QT interval encompasses both the depolarization and the repolarization period, whereas the JT interval characterizes the repolarization period in the presence of conduction abnormalities, such as LBBB. The JT interval is independent of QRS duration, which illustrates its crucial role in the determination of prolonged ventricular repolarization and its independent prognostic value for cardiac events (2, 6).

A study by Salim et al. (7), which was conducted on patients with Wolf-Parkinson-White syndrome, demonstrated that QTc and QRS intervals were shortened after ablation compared to before ablation, whereas the JTc intervals remained unchanged. It was therefore concluded that there is a relative independence of JTc in wide QRS complexes. Since the JTc interval is used as an appropriate measure of ventricular repolarization in LBBB, we decided to obtain JTc intervals and to predict the QTc with a formula that considers the CL of the stimulation.

Bazett's formula, which is frequently used by physicians, significantly exaggerates QTc/JTc rate dependency during ventricular pacing (2). The Rautaharaju formula improves upon this because of its valuable correction of QT changes in the presence of ventricular pacing, but it may lead to different results if the intrinsic conduction rate and ventricular pacing change (2, 8). Previous studies showed an association between a QTc interval of > 450 ms and adverse events and mortality (9, 10), so we used a cut-off point of 450 ms for QTc prolongation, based on the literature (9, 11). At this cut-off point, JTc duration was 92.6 ms with a sensitivity of 80% and a specificity of 67%.

### 5.1. Limitations

Our study had some limitations, mainly the assumption that the pacing-induced LBBB pattern is equal to the 'natural' LBBB pattern, which may or may not be true. The other shortcoming is that the study subjects presumably had no structural heart disease. Many patients with LBBB on ECG have structural heart disease, and the behavior of the JT interval in those patients may be different from a pacing-induced LBBB in patients with normal hearts.

### 5.2. Conclusion

Our results confirm that JTc, as an index of repolarization, is independent of ventricular depolarization. Therefore, it can be applied for predicting QTc in patients with LBBB or other arrhythmias with ventricular conduction delays. We obtained a rate of 92.5 ms for the JTc at a cut-off point of 450 ms, with a sensitivity of 80% and a specificity of 67%.

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### Footnotes

**Authors' Contribution:** Study concept and design: Abolfath Alizadeh; acquisition of data: Peyman Tabatabaei, and Masome Zeighami; analysis and interpretation of data: Hooman Bakhshandeh; drafting the manuscript: Behshid Ghadrdoost, Ala Keikhavani; critical revision of the manuscript for important intellectual content: Abolfath Alizadeh; statistical analysis: Behshid Ghadrdoost, and Hooman Bakhshandeh; technical and material support: Majid Haghjoo, and Amirfarjam Fazelifar.

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