Lead Selection for Maximal QT Interval Duration Measurement in Patients with Heart Failure and Stroke

I.Mozos

"Victor Babes" University of Medicine and Pharmacy, Timisoara, Romania
Email: ioanamozos@yahoo.de

Abstract. *A prolonged QT interval is a predictor of sudden cardiac death. QT interval is prolonged in heart failure and stroke patients. Interlead variability of the QT interval is considerable, but its measurement is laborious in all 12 standard ECG leads. The objective of the present study was to find the leads with the longest QT interval duration in patients with stroke and heart failure. A total of 122 patients, 43 with heart failure, 61 with stroke and 18 healthy controls, underwent standard 12-lead ECG, and the QT interval was measured in each lead and corrected for heart rate using the Bazett formula (QTc). Maximal QT interval duration (QTmax) was measured most often in the precordial leads: V2, V4, and V3. Considering limb leads, QTmax was measured most often in DII. The highest QTc and QTmax values were recorded in stroke and heart failure patients, respectively. The precordial leads are the most reliable leads for assessment of maximal QT interval duration. DII is the most reliable limb lead for QT interval measurements in patients with stroke and heart failure.*

Keywords: Electrocardiography, QT interval, Heart Failure, Stroke

1. Introduction

The QT interval, including ventricular depolarization and repolarization, begins at the initiation of the Q wave and ends where the T wave returns to the isoelectric line, and reflects the summed ventricular action potential durations [1, 2]. Prolonged QT interval duration continues to be a reliable predictor of syncope, torsades de pointes, ventricular arrhythmia and sudden cardiac death. Significant spontaneous variability in the QT interval duration appears within standard 12-lead ECG leads [1]. Measurement of the QT interval is laborious in all 12 standard ECG leads, the selection of the optimal ECG lead for QT interval measurement may be crucial for further therapy [3], which justifies the objective of the present study to find the leads with maximal QT intervals. With improved survival after major cardiovascular events and aging of the population, stroke, myocardial infarction and arrhythmias will be increasing clinical entities in the coming decades [4].

2. Subject and Methods

Study Design and Population
An observational retrospective study was performed including 122 participants with stroke, heart failure and healthy controls. The investigations conformed to the principles outlined in the Declaration of Helsinki (Cardiovascular Research 1997; 35:2-4) and were approved by the Ethics Committee of the university. Patients with heart failure, diagnosed according to the ACCF/AHA Guidelines [5] and stroke, according to the AHA criteria [6], were included. The most important exclusion criteria were atrial fibrillation and electrolyte imbalances.

Standard 12 Lead ECG
The participants underwent standard 12-leadECG and QT interval was assessed in each lead, according to a previously described methodology [7]. QT interval duration was prolonged if >450 ms in men and >460 ms in women [8]. Borderline QT interval was considered for QT interval

durations of 430-450 ms in men and 450-460 ms in women [9]. The Bazett formula was used for heart rate correction of the QT interval [10].

3. Results

The study population included 122 participants, aged 60±16 years, 54% female, 43 with heart failure stage A, B and C (27 with an old myocardial infarction, 16 hypertensive), 61 with ischemic stroke and 18 healthy controls. QTmax, QTc, HR and QT interval duration in all standard ECG leads are included in Table 1. QTc was prolonged in 73 patients (60%). Borderline QTc values were measured in 15 patients (12%).

Table 1. Maximal QT interval duration (QTmax), heart rate corrected QTmax (QTc), heart rate (HR) and QT interval duration in each of the 12 standard ECG leads.

Variable	Results (Means±SD)
QTmax	421±55 ms
QTc	483±65 ms
HR	81±20 beats/min
QT in DI	380±56 ms
QT in DII	377±59 ms
QT in DIII	363±56 ms
QT in aVL	382±61 ms
QT in aVF	377±55 ms
QT in aVR	376±54 ms
QT in V1	370±54 ms
QT in V2	393±48 ms
QT in V3	393±49 ms
QT in V4	387±52 ms
QT in V5	388±55 ms
QT in V6	387±55 ms

Leads with Maximal QT Interval Duration

Maximal QT was measured more frequent in leads V2 (in 18% of the patients), V4 (15%) and V3 (14%). Considering only patients with a prolonged QTc, QTmax was measured most often in V3 (16%), followed by leads DI, aVL and V2 (14%) and DII, aVF, V4 and V5 (12%), and most seldom in lead aVR (5%). In heart failure patients, maximal QT interval duration was found most frequently in leads V2 (28%), aVF and V4 (21%), and V3 (19%). QT interval duration was frequently found prolonged in DII (20%), V3 and V5 (18%) and V4 (16%), in stroke patients. V3 (33%), V6 (28%) and DII and V2 (22%) were most often the leads with the maximal QT interval duration in the healthy control group.

Interlead variability

The leads proving the closest approximation to QTmax were: V3 (26%), V2 (21%), V6 and DII (20%). In patients with a prolonged QTc, the leads providing the closest approximation to QTmax were: V3 (23%), V2 and V6 (21%) and DI and V4 (15%); V5 (35%), DII, V3 and V6 (28%) and aVL (23%) in patients with heart failure; V3 (26%), V6 (20%) and DI (18%) in stroke patients; V2 (28%), DII, aVF, V3 and V4 (22%) and DI, aVR and V6 (17%) in the control group. DIII, DI, aVR and aVL were the leads with the highest deviation from the QTmax value. Considering only the patients with a prolonged QTc, the leads with the highest deviation from the maximal QT interval value were as follows: V1 (23%), DIII (22%) and aVR (18%). In patients with heart failure, DIII (33%), aVR (21%) and DI, DII were the leads with the highest deviation from the maximal QT interval. V1 (25%), DIII (23%) and DI (18%) were the leads with the highest deviation from the maximal QT interval in stroke patients, and aVL (44%), V1 (22%) and DIII (17%) in the control group.

ANOVA

The highest QTc and QTmax values were recorded in stroke and heart failure patients, respectively (Table 2). Heart rate showed no significant differences between the patients of the 3 groups (p=0.462).

Table 2. Maximal QT interval duration (QTmax), heart rate corrected QT interval (QTc) and heart rate (HR) in patients with heart failure, stroke and the control group

Variable	Heart failure group (n=43)	Stroke group (n=61)	Control group (n=18)	p values by ANOVA
QTc (ms)	490±61	494±69	429±25	0.00054
QTmax (ms)	434±57	424±57	382±19	0.003
HR (beats/minute)	79±21	83±21	76±9	0.462

QT interval duration and QTc in different leads was further compared in 30 participants with measurable QT intervals in all leads. The differences were not statistically significant (p by ANOVA = 0.232 and 0.355 for QT and QTc, respectively) (Table 3). The highest QT and QTc values were also

Table 3. QT interval duration in different leads for 30 study participants with measurable QT interval in all leads

Lead	**QT** (ms)	**QTc** (ms)
DI	368±50	429±61
DII	385±51	448±66
DIII	364±51	423±71
aVR	370±48	431±48
aVF	361±54	420±55
aVL	381±50	440±51
V1	367±49	427±42
V2	385±38	445±51
V3	388±42	448±46
V4	385±40	445±50
V5	384±53	443±57
V6	387±58	447±52

recorded in the precordial leads, especially V3, V6 and V4. QTc in lead DII was also very high. Table 1 also shows the highest QT interval durations in leads V2 and V3 for all study participants.

4. Discussion

The present study found the precordial leads as the most reliable leads for maximal QT interval measurements, especially: V3, V2, and V4 in patients with heart failure, stroke, prolonged QT intervals and healthy controls. Considering limb leads, QTmax was measured most often in DII.

It is known that the measured QT interval is influenced by the leads available for analysis [2]. The projections of the electrical activity on the various leads [11] are, probably, responsible for the QT interval differences in different leads. Electrical activity needs a longer period to travel from the heart to the peripheral electrodes [11], which explains the differences noticed in the limb leads. Lead DII is used for QT measurements in pharmacological studies [1]. The precordial leads are known to contribute significantly to the diagnostic utility of standard 12-lead ECG. Cowan et al demonstrated previously, in patients with anterior and inferior myocardial infarction, that V2 and V3 provide the closest approximation to QTmax [12], and interlead variability is, mainly, due to variation in QRS onset and T wave offset [11, 12]. There is a need for standardization of lead selection for QT interval duration measurement [12]. Sylven at al. suggested the possibility of uniform repolarization abnormalities throughout the ventricular myocardium, in patients with QT prolongation [13], which would explain why QTmax was often measured in several precordial and limb leads in the present study. Yeragani et al. found a highly significant difference between QT variability measures in leads V5 and V1 compared to V3 [14]. Monnig et al. recommended lead DII as a first choice and V5 as a second choice for clinical management of patients with congenital long QT syndrome, considering the risk of syncope and sudden cardiac death [3]. Yamaki et al. demonstrated that the sites of prolonged QT intervals corresponded to the sites of infracted areas in patients with anterior and postero-inferior myocardial infarction [15]. Alvarado-Serrano et al. observed that the QTpeak and JTpeak intervals prolong in old myocardial infarction patients when these intervals are measured in the infarct related leads [16].

The risk of sudden death is high in patients with heart failure, even with aggressive therapy, especially in decompensated heart disease with left ventricular dysfunction, postischemic heart failure, and use of diuretics [17]. Cardiac arrhythmias and ECG cerebrogenic abnormalities are frequent after acute cerebrovascular events [18], mainly due to an autonomic nervous system dysregulation, catecholamine storm, concomitant myocardial ischemia or necrosis, heart failure, cardiovascular risk factors, elevated serum uric acid, inflammation, reactive oxygen species, electrolyte imbalances, especially hypokalemia, and the structural and electrophysiological changes of the senescent heart [18, 19].

The present study is, as far as we know, the first attempt to find the leads with the longest QT interval duration in patients with stroke and heart failure, demonstrating the precordial leads as most reliable. Considering that patients with stroke and congestive heart failure are very often difficult to mobilize, it is very useful to know which ECG leads to select. The leads proving the closest approximation to QTmax were: V3, V6, V2 and DII. Considering that no statistically significant differences were found between the QT intervals in different leads in

patients with a measurable QT interval in all leads, any lead is useful for QT interval measurement, especially if the precordial leads are not available. The last choice for QT interval measurement should be: DIII and aVR, considering that in those leads the highest deviations from QTmax were obtained.

Concluding, the precordial leads are the most reliable leads for assessment of QT interval duration, especially V3, V2 and V4, so QTc should be obtained in one of these leads, in patients with heart failure, stroke, or healthy controls. DII, the inferolateral lead, is the most reliable limb lead for QT interval measurements.

References

[1] Lanjewar P, et al. Issues in QT interval measurement. *Indian Pacing Electrophysiol J*, 4(4): 156-161, 2004.

[2] Goldberger JJ, et al. AHA/ACCF/HRS Scientific Statement on Noninvasive Risk Stratification Techniques for Identifying Patients at Risk for Sudden Cardiac Death: A Scientific Statement from the AHA Council on Clinical Cardiology Committee on Electrocardiography and Arrhythmias and Council on Epidemiology and Prevention. *Circulation*, 118: 1497-1518, 2008.

[3] Monnig G, et al. Electrocardiographic risk stratification in families with congenital long QT syndrome. *Eur Heart J,* 27(17): 2074-80, 2006.

[4] Basile AM, et al. Selective risk factors profiles and outcomes among patients with stroke and history of prior myocardial infarction. The European Community Stroke Project. *J Neurol Sci*, 264: 87-92, 2008.

[5] Yancy CW, et al. 2013 ACC/AHA Guideline for the management of heart failure. *Circulation*, 128(16): e240-e327, 2013.

[6] Sacco RL, et al. An updated definition of stroke for the 21st century: A statement for healthcare professionals from the AHA/ASA. *Stroke*, 44: 2064-2089, 2013.

[7] Mozos I, et al. Factors associated with a prolonged QT interval in liver cirrhosis patients. *J Electrocardiol*, 44: 105-108, 2011.

[8] Rautaharju PM, et al. AHA/ACC/HRS recommendations for the standardization and interpretation of the electrocardiogram. Part IV: The ST segment, T and U waves, and the QT interval: A scientific statement from the AHA Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the ACCF; and the HRS; Endoresed by the ISCE. *Circulation*, 119: e241-e250, 2009.

[9] Goldenberg I, et al. QT interval: How to measure it and what is "normal". *J Cardiovasc Electrophysiol,* 17: 333-336, 2006.

[10] Bazett HC. An analysis of the time-relations of electrocardiograms. *Heart*, 7: 353-70, 1920.

[11] Macfarlane PW, et al. Influence of lead selection and population on automated measurement of QT dispersion. *Circulation*, 98:2160-2167, 1998.

[12] Cowan JC, et al. Importance of lead selection in QT interval measurement. *Am J Cardiol*, 61(1): 83-7, 1988.

[13] Sylven JC, et al. QT interval variability on the body surface. *J Electrocardiol*, 17(2): 179-88, 1984.

[14] Yeragani VK, et al. Significant difference in beat-to-beat QT interval variability among different leads. *Heart Dis*, 4(6): 344-8, 2002.

[15] Yamaki M, et al. The body surface distribution of the QT interval in patients with previous myocardial infarction and normal subjects. *Jpn Circ J*, 51(11): 1289-95, 1987.

[16] Alvarado-Serrano C, et al. Novel indices of ventricular repolarization to screen post myocardial infarction patients. *Comput Biol Med,* 36: 507-515, 2006.

[17] Ferrari R, et al. 150 questions and answers. 2nd edition. IME, Baume-les-Dames, France, 2011.

[18] Katsanos AH, et al. Electrocardiographic abnormalities and cardiac arrhythmias in structural brain lesions. *Int J Cardiol*, 167:328-334, 2013.

[19] Aronow WS. Cardiac arrhythmias. Mechanisms, pathophysiology, and treatment. InTech, Rijeka, Croatia, 2014.