

Short Communication

Rate-Dependent Repolarization Dynamics: Correlation between Electrocardiographic T Wave and U Wave

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Abstract

U wave is the last ventricular repolarization component in electrocardiogram (ECG). Rate-dependent amplitude adaptation of U wave was less known relative to that of T wave. We experienced a patient presenting palpitation and showing ECG associated with clear physiologic upward U wave. Echocardiogram demonstrated no structural heart diseases and normal left ventricular (LV) function. T and U wave amplitudes were simultaneously plotted as a function of the preceding RR intervals using ambulatory ECG. U wave amplitude was augmented monotonously according to an increase of the preceding RR interval, whereas T wave amplitude was increased to the maximum level in the RR interval ranging from 1,000 to 1,500 msec and decreased to a steady-state level as RR interval was further prolonged. These indicate that rate-dependent repolarization gradient in T wave differs from that in physiologic U wave, which is mediated by mechano-electrical coupling under pause-dependent LV pressure and/or volume augmentation.

Keywords: T wave, U wave, Rate-dependency, Wave amplitude.

Introduction

T wave amplitude is dependent greatly on the heart rate. This amplitude variability is useful for risk assessment of the occurrence and recurrence of ventricular tachyarrhythmia in patients with and without structural heart diseases [1-3], whereas U wave amplitude dynamicity is not fully investigated. U wave is a small wave component followed by T wave in the electrocardiogram (ECG). Polarity of the U wave determines its abnormalities, i.e. positive U wave is generally physiologic, whereas negative U wave is pathologic [4]. The amplitude of the physiologic U wave is less than one fourth of the positive T wave amplitude [5]. However, rate-dependent U wave dynamics remains unclear in comparison with T wave dynamics. We experienced an old female patient showing clear physiologic U wave and analyzed rate-dependent dynamics of both T and U waves simultaneously.

Case Description

A 80-year-old woman complained of palpitation and visited our hospital in 2012. On her first visit to our hospital, the results of her serum chemistry were unremarkable, i.e., serum potassium concentration was 4.0 mEq/l, and calcium concentration was 9.0 mg/dl. Automatic measurement of absolute and rate-corrected QT interval indicated 445 msec and 410 msec, respectively (**Figure 1**).

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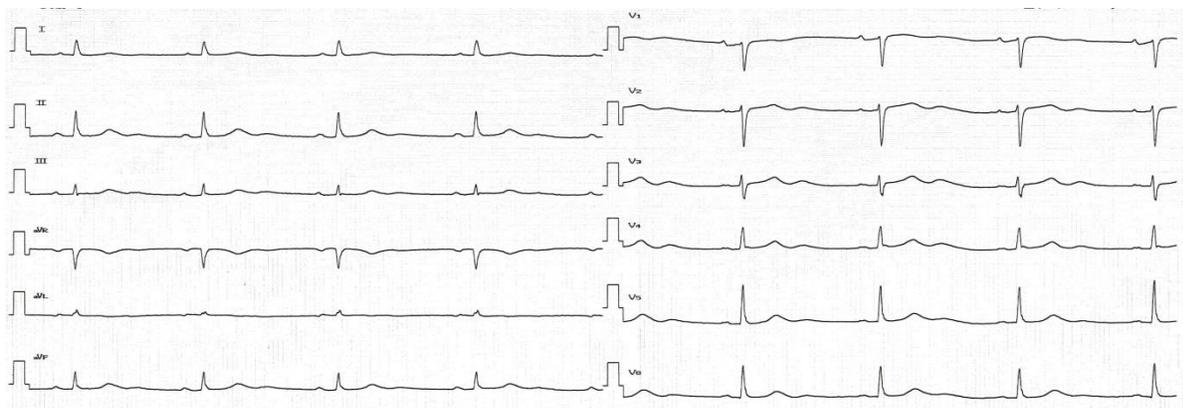


Figure 1: Automatic measurement of absolute and rate-corrected QT interval indicated 445 msec and 410 msec, respectively.

QT interval rate correction was based on Bazett's formula ($QTc = QT/RR^{1/2}$). Transthoracic echocardiogram demonstrated normal left ventricular (LV) function without any structural heart diseases or false tendons. Papillary muscles size was intermediate. Two-channel ambulatory ECG (Digital Walk, FM-160, Fukuda Denshi, Tokyo, Japan) demonstrated 69,980 total heart beats per day, and average heart rate was 49 beats per minute associated with nocturnal sino-atrial conduction block and repetitive premature supraventricular contractions (PSVCs). Based on symptom diary, her palpitation was considered to be due to the short runs of PSVCs, and antiarrhythmic agents (diltiazem 60 mg per day or bisoprolol 1.25 mg per day) were prescribed. U wave polarity was persistently positive. She complained no symptoms except for palpitation and demonstrated no signs susceptible for heart failure and coronary heart disease. Ambulatory ECG showed no diurnal ST-T changes. These indicate that her U wave is not pathological.

Ambulatory Data Analyses

Based on the ambulatory monitoring applied to this case, U wave amplitude as well as T wave amplitude was plotted as a function of preceding RR interval. Ambulatory lead showing greater T and U wave complex (CM5) was adopted. On this analysis, ECG data satisfying the following three criteria were

acceptable; 1) ambulatory data showing clear positive T wave and consecutive physiologic U wave without baseline drift, and 2) data demonstrating that incisura between the T wave and U wave was above the baseline, 3) whether sinus beats or PSVCs did not matter if the beats satisfied the above two conditions.

The amplitudes of repolarization waves and the preceding RR intervals were measured manually by two independent cardiologists, who were unaware of this patient's baseline characteristics. The amplitudes of the T and U wave complex in CM5 were persistently greater than those in CC5 and measured from isoelectric line including the consecutive P wave onsets to the highest levels of the T and U wave, respectively. The intra-observer and inter-observer coefficient of variance for manual measurements of preliminary ECG was 1.4% and 2.5%, respectively. Representative ambulatory monitors containing long RR intervals were demonstrated (**Figure 2**).

Repetitive PSVCs yielded compensatory pause, and pause-dependent behavior of T wave amplitude differed from that of U wave. The amplitude of T wave preceded by long pause was smaller than that of the preceding T wave, whereas the amplitude of the corresponding U wave was greater than that of the preceding U wave (**Figure 2A**).

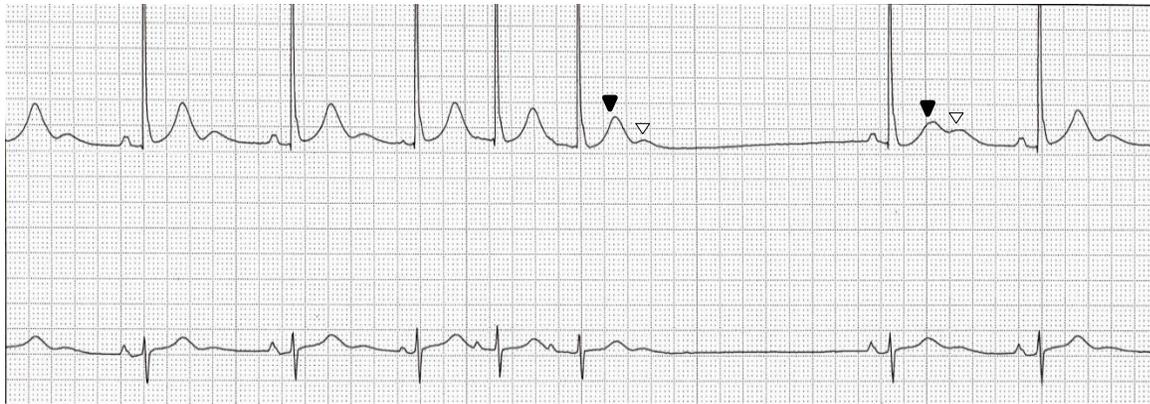


Figure 2A Premature supraventricular contractions

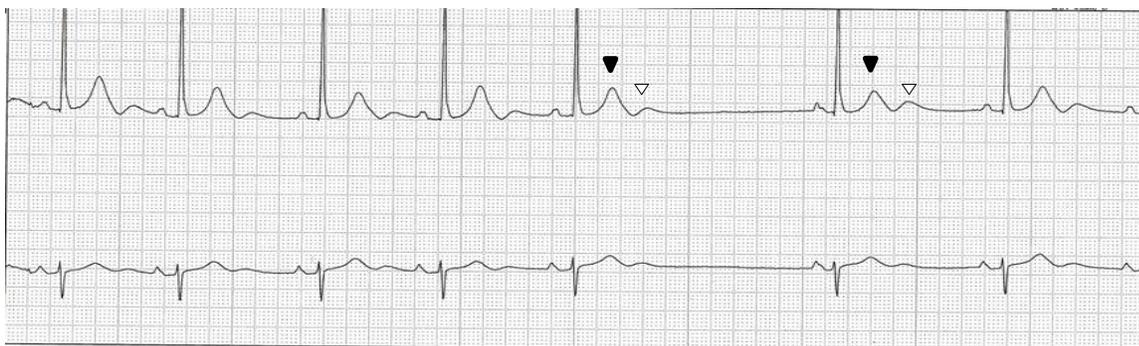


Figure 2B Sino-Atrial Block

These phenomena were observed also immediately after the sino-atrial block (**Figure 2B**). The amplitudes of T and U waves were plotted simultaneously as a function of the preceding RR intervals. To avoid data plots deviation, equal numbers of data points ($n = 10$) were plotted within a same RR segment of 500 msec (i.e., 500 to 1,000 msec, 1,500 to 2,000 msec, 2,000 to 2,500 msec and 2,500 to 3,000 msec segments) except for RR segment ranging from 1,000 to 1,500 msec, which corresponds to sinus beats. Sinus beats data were selected randomly as one datum sampling of every hour segment ($n = 20$), if aforementioned inclusion criteria were met. This trial of random sampling was repeated, and the number of this trial was 10. Inter-trial variance of sampled data ($n = 60$ in one trial) showed 8.2%, which was considered to satisfy the multiple randomsampling validation. Least square method was applied to fit the scattered plots to polynomial regression

curves by using Microsoft Excel 2010 (Redmond, WA, USA), and regression curve showing the greatest correlation of coefficient (R) was accepted. The amplitudes of both T and U waves were abbreviated according to the shortening of the preceding RR interval less than 1,000 msec as observed in the occurrence of PSVCs. However, the behaviors of repolarization dynamics under the preceding RR interval ranging from 1,000 to 1,500 msec in T wave were quite different from those of physiologic U wave. The amplitude of T wave in this RR interval range was scattered but was the maximum, whereas the amplitude of U wave was augmented as the RR interval was lengthened. According to the further prolongation of the preceding RR interval, T wave amplitude was diminished and saturated to the steady-state amplitude around 0.6 mV. On the other hand, physiologic U wave amplitude was further augmented and reached to the maximum level of approximately 0.25 mV (**Figure 3**).

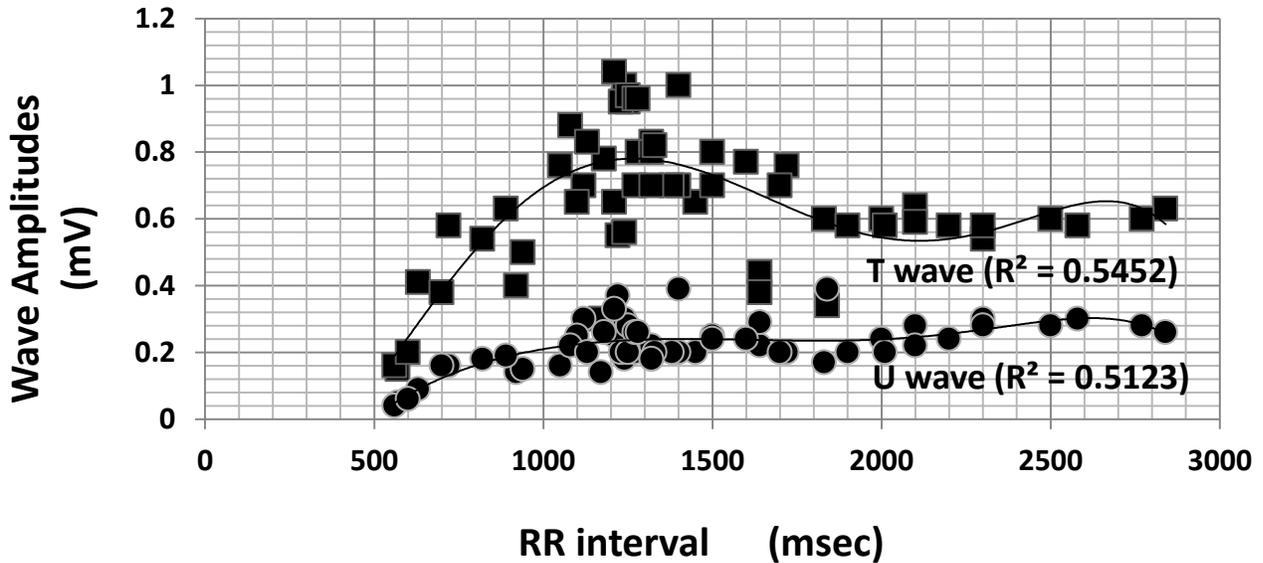


Figure 3

Discussion

Physiologic U wave is sometimes clear in healthy individuals [4]. In this study using ambulatory data in a female patient showing physiologic U wave, polynomial curve fitting was attempted (Figure 3), which yielded curvilinear augmentation of U wave amplitude according to an increase of the preceding RR intervals ($R^2 = 0.51$) and the maximum hump of the T wave amplitude around the RR interval of 1,000 to 1,500 msec ($R^2 = 0.55$).

Ventricular repolarization is greatly influenced by the preceding RR interval and additionally modified by autonomic nervous system, electrolytes, gender and many other factors. Rate-dependent adaptation of repolarization amplitude is useful for risk stratification of ventricular tachyarrhythmia in patients with and without structural heart diseases [1-3]. Among repolarization waves, genesis of T wave completely differs from that of physiologic U wave [5], i.e., T wave represents overall ventricular repolarization, whereas physiologic U wave reflects local repolarization of Purkinje fibers, M cell layer or papillary muscles [4]. Electrophysiologic features of rate-dependency between the whole ventricle and the local components underlie the differences between the T wave and U wave dynamics.

Amplitude of the T wave is reduced according to an increase of the constant atrial pacing rate, whereas that of the U wave remains unknown [6]. However, such rate-adaptation of repolarization wave amplitudes in steady state differs from dynamic relationship between the wave amplitudes and the preceding RR intervals. As a matter of fact, pause-dependent augmentation of repolarization wave amplitude was clear in physiologic U wave but not in positive T wave (Figure 3).

T wave amplitude in CM5 is influenced by LV pressure and/or volume [7], i.e., T wave amplitude is augmented by increased LV filling pressure and/or volume after the long RR intervals in healthy individuals [8]. Temporary changes of LV loading condition modulates transmural repolarization gradient and hence repolarization wave morphologies, which is mediated by mechano-electrical coupling [9]. However, mechano-electrical coupling depends heterogeneously on the period when and the extent to which LV preloading affects i.e., preload is reduced during PSVCs and increased after the following compensatory pause.

These complexities lead to complicated T wave amplitude change as a function of RR interval, whereas U wave reflecting local component repolarization may have been less influenced by such complexities.

Our results should be interpreted carefully due to some limitations. First, this analysis is based on a single patient showing clear physiologic U wave. Second, alteration of T and U wave amplitude as a function of the preceding RR interval was mainly obtained at nighttime due to analytical inclusion criteria. Reportedly, nocturnal rate-dependent repolarization dynamics are different from daytime ones [10]. Third limitation is manual measurement of repolarization waves using analog ambulatory data. In spite of such limitations, repolarization dynamics of physiologic U wave is concluded to be different from that of T wave in structurally normal heart. Rate-dependent U wave amplitude behavior in heart diseases is a matter of future study with perspective of arrhythmogenicity.

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