ORIGINAL ARTICLE

An Analysis of the U-Wave and Its Relation to the T-Wave in Body Surface Potential Maps for Healthy Subjects and MI Patients

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Background: The aim of this study was to analyze the U-wave morphology and its relation to the T-wave in one group of healthy subjects and in two groups of myocardial infarction (MI) patients—with and without ventricular tachycardia (VT) episodes. The context of the U-wave origin was also discussed and the U-wave as a potential marker of VT was investigated.

Methods: The study was carried out on three groups of subjects: 20 healthy subjects, 14 MI patients not at risk of VT, and 22 MI patients at risk of VT. The morphology of the repolarization phase was examined in the high-resolution body surface potential maps recorded from 64 surface ECG leads. The temporal and spatial distributions of several ECG parameters were studied.

Results: The U-wave was present in almost all the studied subjects. The spatial heterogeneity and smooth change in both the T- and U-wave shapes on the entire torso were observed in all the studied groups. The statistical significance of discrimination between the MI patients without VT and MI patients with VT was observed for QRS interval, QT interval, U-wave integral, and normalized U-wave integral.

Conclusions: High-resolution measurement of body surface potentials and an advanced data analysis allow for a detailed description of U-wave morphology and its relation to the T-wave. This might be of value in discriminating intracardiac repolarization effects, mechano-electrical feedback, and arrhythmia risk stratification.

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The U-wave origin has not been explained until today. It is the only deflection in the electrocardiogram that is not widely used in clinical practice, although the U-wave presence and duration might influence the assessment of some important ECG parameters such as QT interval and QT dispersion.^{1–3} Many scientists have attempted to unravel the U-wave mystery since the

beginning of the last century. In 1912, Einthoven⁴ identified and labeled the U-wave postulating that the U-wave represented the currents generated by late repolarizing region of ventricular myocardium. In 1939 Nahum⁵ noticed that coupled ventricular extrasystoles usually occurred during the U-wave appearance. In 1954, there was a symposium on the U-wave where different problems of

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measurement, assessment, clinical importance, and significance of the U-wave in 12-lead ECG were discussed.^{6–12} Surawicz¹⁰ studied the polarity of the U-wave in relation to the T-wave. Holzmänn⁹ introduced different types of fusion between T- and U-waves. Lepeschkin and Surawicz⁷ proposed detailed methods for the QT interval measurement in the presence of the U-wave. The abnormality of the complex of T-wave and U-wave complex was studied by Lambert¹¹ and the U-wave in coronary artery disease was investigated by Papp.⁸

In 1979, Spach¹³ studied the surface distribution of the amplitudes of the U-wave and the T-wave in normal subjects suggesting that a similar location of its maximum values might manifest the similarities of the "cardiac electrical generator." In 1991, Antzelevitch and Sicouri¹⁴ reported that the cells in the midmyocardium (M cells) had action potentials lasting well beyond the end of the T-wave. It was, therefore, very attractive to credit this terminal activity with a function in the formation of the U-wave. The experimental confirmation of this hypothesis has been done by Drouin.¹⁵ Recently also Kors¹⁶ has referred to this concept by simulating the U-wave formation in his model of left ventricle including M cells. Duan et al.¹⁷ studied several ECGs and M-mode echocardiography of healthy subjects and they found that the U-wave occurrence was better correlated with the QQ interval than the mechanical valve timing, which might indicate that the U-wave origin was related more to the electrical than the mechanical activity of the heart.

On the other hand, the concept of mechano-electrical feedback observed by Stauch¹⁸ in 1960 and developed i.a. by Lab¹⁹ and Zabel²⁰ was then used by Surawicz²¹ in 1998. He stated that the mechanical stretch of myocardial cells during ventricular diastole might influence the transmembrane potential, which could produce after-potentials. He proposed the after-potentials as the source of the U-wave formation. In 2002 Di Bernardo and Murray²² used that statement in their model of the left ventricle repolarization process and showed the influence of after-potentials on the cardiac action potential and the U-wave formation. In 2008, Riera et al.²³ wrote a review article on the U-wave and its clinical significance. They suggested that the U-wave might be a result of mechano-electrical feedback, except for the long QT syndrome, where the M cells would play a decisive role. Schimpf et al.,²⁴ while studying

the short QT syndrome in electrocardiography and echocardiography simultaneously in several patients, concluded that the U-wave was rather independent of the ventricular repolarization timing and was coincident with the second heart sound which, might be a proof that the U-wave was formed by the mechano-electrical phenomenon.

There is also the third hypothesis that indicates the Purkinje fibers repolarization as the source of the U-wave deflection in the ECG signal.²⁵ This proposal, however, is thought to be the least likely explanation of the U-wave origin due to a small mass of Purkinje fibers.

The debate is still open.^{17,26} Despite the fact that the U-wave is commonly claimed to be present in electrocardiogram in up to 75% of population,^{4,27} recent magnetocardiographic study by Goernig²⁸ has revealed the U-wave activity in every healthy volunteer, in whom the detection of a distinct U-wave activity was not possible in the standard 12-lead ECG. That result suggests that the U-wave activity should be a regular phenomenon, which has a distinct spatiotemporal assembly.

The aim of this study was to analyze the U-wave morphology, duration, and spatial heterogeneity as well as their relation to the T-wave in one group of healthy subjects and in two groups of MI patients—with and without VT episodes. To obtain a precise distribution of the low-amplitude U-wave potentials we have used high-resolution body surface potential map (BSPM) measurements^{29,30} and aim-oriented methods for signal analysis. The U-wave as a potential marker of ventricular tachycardia (VT) was also investigated.^{31–33}

METHODS

Subject Population

The study included three groups of subjects. The first group comprised 20 healthy subjects, the second group comprised 14 myocardial infarction (MI) patients not at risk of ventricular arrhythmia (non-VT), and the third group consisted of 22 MI patients at risk of ventricular arrhythmia (VT) with an implanted cardioverter-defibrillator (ICD). The basic data for all the three studied groups are presented in Table 1.

The control group consisted of volunteers with normal electrocardiograms, no history of cardiovascular disease, and who were not receiving

Table 1. General Data for Studied Groups

Studied Groups	Number of Subjects	Age (years)	Heart Rate (bpm)	BMI (kg/m ²)	LVEF (%)
Healthy	20	49 ± 15	66 ± 8	25 ± 6	>65
Non-VT	14	61 ± 11	64 ± 12	29 ± 3	Data not avail.
VT	22	64 ± 11	62 ± 11	29 ± 4	38.8 ± 13 (n = 16)

VT = ventricular tachycardia; BMI = body mass index; LVEF = left-ventricular ejection fraction.

any medications. The eligibility criteria for the group of MI patients at risk of VT were documented by the spontaneous episodes of VT before or after ICD implantation. The study was approved by an institutional ethics committee and the subjects gave informed consent.

Measurements and Data Processing

The patient examinations were carried out in General Hospital of Medical University of Vienna (Austria). A high-resolution multilead ECG system Active Two³⁴ (Biosemi Inc.) with 64 surface electrodes was used to record high-resolution BSPMs. Active electrodes containing an Ag₂Cl contact sensor with a preamplifier were located over the entire torso according to the modified Amsterdam lead system^{35–37} as shown in Figure 1.

The 64 unipolar ECGs were simultaneously recorded at a bandwidth of DC to 800 Hz in a period of 15 minutes. Next, the signals were amplified and converted into a digital form with a 4096 Hz sampling frequency and a 24-bit amplitude resolution. Then the signals were sent through a fiber-optic link to the computer and stored on a hard disk for off-line processing.

The raw ECG data were filtered using a low-pass Butterworth filter limiting the frequency to 300 Hz and a decimate filter decreasing the sampling frequency to 1024 Hz. A high-pass filtering was not applied. Baseline wandering was removed using linear interpolation between isoelectric levels estimated from the 20 ms intervals preceding P-waves in consecutive ECG beats. The UP interval was chosen as the isoelectric line, because as we noticed, during the PQ interval atrial repolarization is still present, which was confirmed by Ihara et al.³⁸.

Subsequently, the ECG signals were averaged in time using the cross-correlation method. To achieve a low level of noise, both the number of the averaged cycles (usually ca. 100 cycles) and the value of the correlation coefficient (≥ 0.98) were

fitted. The level of noise was measured in the 20 ms isoelectric U–P interval. The obtained root mean square (RMS) value of noise in the averaged ECG signals varied from 0.1 μ V to 1.8 μ V, with the mean value equal to 0.7 μ V.

Data Analysis

To detect the ECG characteristic time instances (the onsets and offsets of both depolarization and repolarization waves) the algorithm based on Singular Value Decomposition (SVD) proposed by Acar³⁹ was applied. For each subject a rectangular matrix M (n, m) containing n averaged ECG signals, each of m samples length, was decomposed using the following estimation:

$$M = U \Sigma V^T, \quad (1)$$

where

U (m, m) and V (n, n) are square, orthogonal matrices.

Σ is a diagonal matrix (m, n) of singular values.

Next, the reduced-space data matrix S was obtained by projecting matrix M down into the reduced space defined by only the first three left-singular vectors of unitary matrix U . From these three vectors of matrix S (S_1, S_2, S_3) the RMS signal was calculated. The ECG characteristic time instances were found first in the RMS signal and then in each lead separately (Fig. 2). First, R-peak was marked as the maximum of the RMS signal. Then, T-wave maximum was marked in relation to R-peak. The beginning of the P-wave and the end of the U-wave were calculated by inspecting the stationarity of the time relation between vectors S_1 and S_2 in the, respectively, defined time windows. Next, the onset of the Q-wave and the offset of the S-wave were calculated from the difference RMS signal using the threshold method.

The T-wave end was established as the minimum of RMS signal in the time window related to the T-wave maximum. Then, in each lead individually, the ECG fiducial points were established in

Figure 1. Lead arrangement on the torso for high-resolution ECG measurements. Standard ECG leads are marked with dots.

Figure 2. ECG characteristic time instants detected on RMS signal obtained using Singular Value Decomposition method.

relation to the global time instants of the waves using the threshold method for the Q onset, and P, S, T, U offsets and extrema search procedures for P, R, T, U peaks.

Morphology Examination

In the present study the morphology of the repolarization phase was examined in the

Figure 3. Classification and distribution of the TU complex shapes observed on the entire torso.

(A) Morphology classification of TU complex. The column numbers refer to the T-wave shape and the row letters to the U-wave shape. The A-I shape is the most common shape. The vertical lines indicate the end of the T-wave and the beginning of the U-wave.

(B) The simplified spatial distribution of the TU-complex shapes on the torso observed in the group of healthy subjects. The locations of the ECG leads are marked with black and white (standard) dots. The positive T- and U-waves (the dark gray area) are observed in the lower central chest area, precordial area, and left lower back area. The positive or biphasic T- and U-waves (medium gray) are observed on the upper left side of the chest. The biphasic T- and U-waves (light gray) are observed on the left and on the right side of the torso. The negative T- and U-waves (white) are observed on the upper right side of the chest and on the upper right side of the back.

high-resolution BSPMs recorded from 64 surface leads. The TU complex shapes were divided into 24 classes as shown in Figure 3(A). In the suggested classification 16 classes had already been proposed by Lepeschkin.⁷ The observed

T-waves were classified as monophasic positive (I), biphasic positive/negative (II), biphasic negative/positive (III), monophasic negative (IV). The U-waves were divided into classes: monophasic positive (A), biphasic positive/negative (B), biphasic

negative/positive (C), monophasic negative (D), the U-wave was not in either monophasic or biphasic shape and its interval was covered by a descending slope of the T-wave (E) and there was an isoelectric line after the end of the T-wave (F). Several parameters describing mainly the TU complex and its relation to the RR and QT intervals were assessed for each subject and each lead.⁴⁰ The following parameters were studied: T-wave amplitude, U-wave amplitude, T-wave duration, U-wave duration, QRS interval, QT interval, QT interval dispersion, QU interval, QU interval dispersion, Tpeak-Tend interval, Tend-Upeak interval.

The integrals of the T-wave and the U-wave were obtained from a calculation for each lead, the algebraic sum of all the instantaneous potentials from the wave onset to the end of the wave multiplied by the sampling interval. Additionally, the normalized parameters of the T-wave integral and U-wave integral were obtained by dividing them by the maximum value of the respective integral for each patient.

Statistical Analysis

For each subject the ECG intervals were assessed on the basis of RMS signal. The T- and U-wave amplitudes were chosen as the maximum values from all the leads for each subject. The T- and U-wave integrals were calculated as the mean value from all the leads for each subject. For each studied group the mean values and standard deviations of the mentioned parameters were assessed. The statistical significance of the difference between the studied patient groups (non-VT vs VT) was estimated using the nonparametric Mann-Whitney test. The statistical significance level of $P < 0.05$ was chosen.

To assess the effectiveness of statistically significant parameters in the VT patient discrimination and to define the risk criteria of VT for MI patients, the specificity and sensitivity values of the proposed parameters were calculated. The diagnostic criteria of the proposed parameters were chosen due to the optimal sensitivity and specificity values.

RESULTS

Morphology Classification

The classification and simplified spatial distribution of the TU complex shapes on the entire torso is presented in Figure 3 and Table 2.

The morphology of the TU complex smoothly changes its form on the entire torso from the negative signals (D-IV shape) in the right upper chest area (leads 1, 2, 4, 10), through the biphasic negative/positive (A-III, F-III) waves in the lower right part of the chest (leads 3, 5, 6, 7), to the positive, separate or partly merged T- and U-waves (A-I) observed in the lower central chest area, precordial area, and left lower back area (up to leads 61, 64). On the upper left side of the back (leads 43, 44) both the T- and the U-wave might be biphasic (F-II) or positive (A-I). On the upper right side of the back and central back (leads 55, 56, 58, 59, 62, 63) both the T- and the U-wave become negative again (D-IV shape).

The maximum positive T- and U-wave are usually present in lead 18 (precordial lead V2) and above or below this location (leads 17 and 19), where the angle of the heart vector projection is equal to 90° .^{41,42} At the same time the maximum negative T- and U-wave are observed on the back, in lead 56 and in the neighborhood of this location, where the angle of the heart vector projection is about -90° . The absence of the U-wave (type E), if observable, is located in the upper left side of the chest as well as the prolonged T-wave (type F).

In both patient groups the observed U-wave shapes are more miscellaneous than in the group of healthy subjects.⁴³ Although the positive signals of the T- and U-wave (A-I shape) are always present in the central part of the chest, around the V2 location, the biphasic or negative T- and U-waves appear more frequently below and above the precordial leads V4, V5, and V6, especially in the group of VT patients. The maximum negative T- and U-wave are observed on the back, in lead 56, and in its neighborhood.

Quantitative Analysis

The quantitative analysis aimed to describe the basic statistics of the obtained parameters in the studied groups. In Table 3 the mean and standard deviation values of the assessed parameters are shown for all the studied groups along with the results of the comparison between the VT and non-VT patients using the Mann-Whitney test.

The longest ECG intervals were observed in the VT group and the shortest ones in the healthy group. The T- and U-wave durations were equal to 351 ± 44 ms and 221 ± 73 ms, respectively, in the VT group and 315 ± 28 ms and 228 ± 35 ms, respectively, in the healthy group. The range of the

Table 2. Main Groups of TU Complex Observed in Healthy Subjects

TU Complex Main Groups	T-Wave and U-Wave Shapes According to Classification
Positive T- and U-waves	A-I
Positive or biphasic T- and U-waves	A-I, A-II, A-III, B-I, B-II, B-III, C-I, C-II, C-III, E-I, E-II, E-III, F-I, F-II, F-III
Biphasic T- and U-waves	A-II, A-III, B-II, B-III, C-II, C-III, F-III
Negative T- and U-waves	D-IV

Table 3. Mean and Standard Deviation Values of Studied Parameters for all Groups, as well as, the Results of Mann-Whitney Test for Discrimination between VT (Ventricular Tachycardia) and non-VT Groups

Parameter	Healthy Subjects	Non-VT Patients	VT Patients	P Value VT vs non-VT
T amplitude (μV)	751 $\pm$ 219	535 $\pm$ 277	483 $\pm$ 235	ns
U amplitude (μV)	73 $\pm$ 29	69 $\pm$ 48	54 $\pm$ 25	ns
T duration (ms)	315 $\pm$ 28	342 $\pm$ 48	351 $\pm$ 44	ns
U duration (ms)	228 $\pm$ 35	255 $\pm$ 71	221 $\pm$ 73	ns
QRS interval (ms)	99 $\pm$ 9	107 $\pm$ 14	133 $\pm$ 35	0.01
QT interval (ms)	414 $\pm$ 27	449 $\pm$ 43	484 $\pm$ 47	0.03
QT dispersion (ms)	57 $\pm$ 21	77 $\pm$ 25	77 $\pm$ 32	ns
QU interval (ms)	641 $\pm$ 51	704 $\pm$ 100	705 $\pm$ 98	ns
QU dispersion (ms)	152 $\pm$ 74	206 $\pm$ 74	215 $\pm$ 84	ns
Tpeak-Tend interval (ms)	114 $\pm$ 13	116 $\pm$ 26	127 $\pm$ 23	ns
Tend-Upeak interval (ms)	72 $\pm$ 12	70 $\pm$ 24	67 $\pm$ 28	ns
U integral (ms $\cdot\mu V$)	2688 $\pm$ 1625	1862 $\pm$ 1242	1037 $\pm$ 902	0.03
U integral norm	30 $\pm$ 11	22 $\pm$ 6	14 $\pm$ 9	<0.005

U-wave duration in the healthy group varied from 160 ms (HR = 69 bpm) to 300 ms (HR = 54 bpm and HR = 60 bpm). The Tpeak-Tend interval was the longest in the VT group and equal to 127 $\pm$ 23 ms, while in the healthy group it was the shortest and equal to 114 $\pm$ 13 ms. However, the Tend-Upeak interval was the shortest in the VT group and equal to 67 $\pm$ 28 ms, while in the healthy group it was equal to 72 $\pm$ 12 ms. Both QT dispersion and QU dispersion were the largest in the VT group.

While the temporal parameters were the longest in the VT group, the potential-correlated parameters were the smallest in this group. The mean values of the amplitudes of the T- and U-wave were equal to 483 $\pm$ 235 μV and 53 $\pm$ 26 μV , respectively. In the healthy group those parameters were equal to 751 $\pm$ 219 μV and 73 $\pm$ 29 μV , respectively. The integral parameters were also the smallest in the VT group. The U-wave integral and the normalized U-wave integral parameters values in the VT group were equal to 1037 $\pm$ 902 (ms $\cdot\mu V$) and 14 $\pm$ 9, respectively, whereas in the non-VT group 1862 $\pm$ 1242 ms $\cdot\mu V$ and 22 $\pm$ 6, respectively. In the healthy subject group they were equal to 2688 $\pm$ 1625 (ms $\cdot\mu V$) and 30 $\pm$ 11, respectively.

The following parameters: T-wave amplitude, U-wave amplitude, T-wave duration, Tpeak-Tend interval, Tend-Upeak interval showed a potential ability to differentiate between the non-VT and VT groups, however, they did not separate the VT and non-VT patient groups at the chosen, statistically significant level ($P < 0.05$). While using the nonparametric Mann-Whitney test for discrimination between the non-VT and VT groups, the statistically significant difference was observed for QRS interval, QT interval, U-wave integral, and normalized U-wave integral. The lowest P-value was found for the normalized U-wave integral parameter ($P < 0.005$). The mean value of the U-wave integral for each subject comprises information on both the amplitude and duration of the U-wave. It expresses the mean spatial and temporal electrical activity of the heart during the U-wave segment.

In the VT patient group, the values of the U-wave integrals were lower than in both the non-VT group and healthy group due to the more negative or low-amplitude signals present, especially in the left lower chest and on the back areas. The representative maps of the U-wave integral for each studied group are shown in Figure 4(B).

Figure 4. Body surface maps of T- and U-wave. (A) Body surface potential maps of T- and U-wave amplitude averaged in the group of healthy subjects. (B) Body surface integral maps of U-wave for the healthy subjects, non-VT patients, and VT patients.

Figure 5. Sensitivity and specificity of normalized U-wave integral in differentiation between non-VT and VT groups.

For the U-wave integral parameter the value of 1277 ($\text{ms} \cdot \mu\text{V}$) was optimal for separating the non-VT and VT groups while using sensitivity and specificity functions. The value of sensitivity was 73% and the value of specificity was 64%. For the normalized U-wave integral parameter the classification value was 18.9. The sensitivity and the specificity values of the normalized U-wave integral were, respectively, 77% and 64% (Fig. 5).

DISCUSSION

The aim of this study was to analyze the U-wave morphology and its relation to the T-wave in the group of healthy subjects and in the two groups of MI patients—with and without VT episodes. To obtain a precise distribution of the low-amplitude U-wave potentials we have used the high-resolution BSPM measurements and the aim-oriented methods of signal analysis.

The maps of the U-wave amplitudes and integrals, obtained in our study, showed a higher spatial resolution than the first body surface maps of the U-wave obtained by Spach.¹³ In his work the U-waves with the amplitudes lower than 15 μV had not been analyzed. In our study all U-wave potential values were taken into account. We have noted that in all the studied subjects with the heart rate <97 bpm the end of the T-wave was definitely not the end of the electrical activity of the heart in a single cardiac cycle, in a few BSPM leads at

least. The U-wave in the shape of a single hill or partly merged with a descending T-wave slope was present and detected in all the healthy subjects, in all the non-VT patients and in 20 out of the 22 VT patients. The U-wave as a separate monophasic wave was not observed in two VT patients. In one patient the high heart rate (97 bpm) caused a shortening of the TP interval to a few milliseconds and the U-wave in this case could have been totally overlapped by the T-wave, prolonging its duration to the beginning of the P-wave. In the second VT patient with the heart rate equal to 58 bpm, the dispersion of the T-wave end was large and equal to 202 ms, which was almost twice as long as the largest observed QT dispersion in all the studied groups. The ECG signals from all the leads of the second VT patient are shown in Figure 6(A). The large dispersion of the T-wave interval is clearly visible.

The presence of the U-wave in all the studied healthy subjects is in agreement with magnetocardiographic studies²⁸ where the U-wave was detected in all the healthy subjects without the U-wave visible in the standard 12 lead ECG. Moreover, it is also coincident with the hypothesis propounded by Kors and Ritsema¹⁶ who suggested the presence of the U-wave in each person due to its origin in the ventricular repolarization process. Also Postema⁴⁴ in his work detected the U-wave in all studied subjects using the fiducial point averaging method in the 12 lead ECG.

The negative potentials of U-waves were present in the healthy subjects not only on the right upper chest, but also on the right upper and central area of the back as shown in Figure 3(B). We have also observed the similarity of the spatial distribution between the T- and U-wave potentials, especially the positive maximum values localized in the same or very close area of the V2 precordial lead (Fig. 4 A). The same results were obtained by Spach⁵ who concluded that such proximity of the T- and U-wave amplitude location might be the evidence for "similarities of the cardiac electrical generator (i.e., the spatial distribution of intracellular potentials) during the T-wave and the U-wave."

The observed spatial heterogeneity of the TU complex morphology present in the healthy subject group and a smooth change in both the T- and U-wave shapes on the entire torso surface support the concept of the ventricle repolarization process as the origin of both the T-wave and the U-wave,

Figure 6. The time courses of TU complexes in 64 leads recorded for two studied subjects. The longest and the shortest TU complexes are marked with black lines. (A) TU complexes observed in patient without visible U-wave. The large dispersion of the T-wave end is noticeable. (B) An example of heterogeneity of U-wave durations in the healthy subject. A long duration of U-wave is clearly visible.

especially that in some leads there is no clear border between T- and U-waves, and the nadir between T- and U-wave practically never reaches the isoelectric line. It is in agreement with the theoretical works of Kors and Ritsema,^{16,41} who wrote "T and U form a continuum. Together they are the resultant of one and the same process of repolarization of the ventricular myocardium." The same conclusion was drawn by Hopenfield et al.⁴⁵ who demonstrated that delayed M-cell repolarization in their model could produce the spatial U-wave distribution observed earlier by Spach.¹³

However, the U-wave duration is too long to be the only result of repolarization process, that is, in precordial leads it can last to the beginning of the P-wave as shown in Figure 6(B). The concept of mechano-electrical feedback might have an influence on the U-wave forming as well. The U-wave beginning is coincident with the second heart sound.²¹ The U-wave occurrence is also coincident with the third heart sound, which is audible in many patients, and according to Manson⁴⁶ it should occur in all subjects. The third heart sound in phonocardiogram is caused by the vibrations of the entire cardiohemic structure, that is, the ventricular wall, intraventricular blood pool, and surrounding structures during the decelerating of blood flow through the mitral valve. The diastole of the heart muscle might manifest itself in the electrical potential between the T-wave end and P-wave beginning, and together with the ventricular repolarization constitutes the U-wave on the body surface.

Several studied parameters have shown a possibility to discriminate between non-VT and VT MI groups. The T- and U-wave amplitudes were smaller in the MI patients group at risk of VT than in the MI patients not at risk of VT. On the other hand, the T-wave interval, as well as, the Tpeak-Tend interval were longer in the VT group than in the non-VT one. There was no difference in the QT dispersion values between the non-VT and VT patients.

The only statistically significant discriminators between the non-VT and VT groups, while using the nonparametric Mann-Whitney test, were the U-wave integral and normalized U-wave integral as well as the known VT-risk factors—QRS duration and QT interval. However, in our study the QT interval in the VT group was significantly longer than in the non-VT group mainly due to the width of the QRS complex. These statistical results should be confirmed by further studies in a larger number of patients.

The mean area under the U-wave (U-wave integral) obtained in this study in the healthy group ($2688 \pm 1625 \text{ ms} \cdot \mu\text{V}$) was similar to the mean area under the U-wave in the 12 lead ECG ($2690 \pm 160 \text{ ms} \cdot \mu\text{V}$), obtained by Postema for the control group.⁴⁴ In both MI patient groups, we observed much lower values of the mean area under the U-wave. Such a result might be explained by the increased disparity of the U-wave morphology, that is, the presence of more

biphasic or negative monophasic U-waves on the body surface, especially in the left precordial area (leads V4, V5, V6). The decreased value of the mean area under the U-wave was obtained by Postema⁴⁴ in patients with a short QT interval and gain-of-function mutation in the gene responsible for I_{K1} . The I_{K1} current helps to establish the resting membrane potential of the cell and indirectly is responsible for the potassium ion current during the third and fourth cardiac action potential phases, which coincide with the U-wave formation.

The obtained results show that the disparity of the U-wave and the TU complex morphology increases in MI patients, especially in those at risk of VT in comparison to the healthy group. It might be the manifestation of both the repolarization dispersion and the impaired left ventricle mechanical function. A quantitative analysis of such a small deflection as the U-wave might contribute to enhancing the VT risk assessment and improve the effectiveness of patient selection to ICD therapy. However, a further evaluation of the effectiveness of the proposed parameters for the malignant VT risk assessment after MI requires additional prospective studies.

The obtained results of the qualitative and quantitative analysis of the TU complex in high-resolution BSPM allowed to form the following conclusions:

- (1) The high-resolution measurement of body surface potentials and the advanced data analysis allow for a detailed description of the U-wave morphology and its relation to the T wave. This might have a value in the discrimination of intracardiac repolarization effects and mechano-electrical feedback.
- (2) The U-wave in the shape of a single hill or partly merged with the descending T-wave slope was observed in all the studied subjects except for two. In those two patients, although the monophasic U-wave was not observed, the ECG potential of that deflection was present and influenced the timing of the T-wave end.
- (3) The observed spatial heterogeneity of the TU complex morphology present in the healthy subject group and the smooth change in both the T- and U-wave shapes on the entire torso surface as well as the similarity of the spatial distribution between the T- and U-wave potentials might support the concept of the ventricle repolarization process as the origin

of both the T- and the U-wave. However, the phenomenon of the mechano-electrical feedback seems to have an influence on the U-wave forming as well as prolonging its duration beyond repolarization timing. Nevertheless, the final answer might be obtained only from further electrophysiological studies.

- (4) The U-wave integral parameter and normalized U-wave integral parameter as well as the QRS interval and QT interval were the only statistically significant discriminators between non-VT and VT groups while using the nonparametric Mann-Whitney test. This might have a diagnostic value in arrhythmia risk stratification.

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