R WAVE PEAK TIMES DISPLAYED IN BODY SURFACE ISOCHRONE MAPS OF YOUNG ADULT MEN

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Abstract

In standard 12-lead electrocardiography, the peak time or intrinsicoid deflection is defined as the activation time of the ventricular muscle lying under the exploratory electrode. It serves as an easy measurable parameter used in the diagnosis of cardiac diseases related to disturbed or delayed ventricular activation. The aim of this study was to analyse the R wave peak isochrone maps in young adult volunteers using the time normalisation of the QRS complex. Body surface isochrone maps of the peak time were constructed and analysed in 13 healthy young men. It was found to start always on the anterior chest surface and mainly in its upper half. After spreading downward and leftward, it mostly ended mainly in the back chest, mainly in its upper half, and around the right shoulder. This agrees with previously published results of mean maps, also in older subjects without known cardiovascular diseases obtained from different mapping lead systems. The R wave peak times obtained from our maps also agreed with known intrinsicoid deflections for the standard leads V_5 and V_6, published previously. To conclude, the R wave peak isochrone maps allow simple visualisation of activation sequence, which will differ in cardiac diseases related to disturbed or delayed ventricular activation, like ventricular hypertrophy, myocardial infarction or bundle branch blocks.

Keywords

electrocardiography, body surface potential mapping, ventricular activation, intrinsicoid deflection

Introduction

Electrocardiography is a method capable of detecting a wide range of cardiac pathologies, based on the assessment of various parameters of cardiac function. One of them is the ventricular activation time, defined as the activation time of the ventricular muscle lying under the exploratory electrode [1].

In standard 12-lead electrocardiography (ECG), the ventricular activation time is also known as the intrinsicoid deflection or the peak R wave time. It is measured from the beginning of the QRS complex to the peak of the R wave with typical values of 15 ms to 35 ms in the lead V_1 and up to 50 ms in the leads V_5 and V_6 [2]. Prolongation or delay of intrinsicoid deflection is an important criterion for the diagnosis of bundle branch block and ventricular hypertrophy [1, 2]. Delayed intrinsicoid deflection may provide an opportunity to identify patients with undiagnosed heart disease prior to adverse clinical outcomes [3, 4].

Body surface potential mapping or multi-lead electrocardiography is an extension of the concept of the 12-lead ECG, which, by increasing the number of the unipolar leads and distributing them over the whole chest, allows non-invasive mapping of cardiac electrical activity in detail. Instead of evaluating individual curves, two- or three-dimensional images called maps are analysed. They may display the distribution of potentials in a single time moment (isopotential maps), areas under the electrocardiographic curves-time integrals (isointegral maps), or time values that have been predefined in connection with some value of the potential on the surface of the chest (isochrone maps) [5].

Until now, only a very small number of scientific papers have dealt with body surface maps displaying R wave peak times or ventricular activation times. We concentrated here on those which described the activation in healthy controls.

Isochrone maps of 36 clinically normal subjects, all men, 22 to 51 years old, had a consistent pattern, with isochrone lines extending from the right upper anterior chest to the left anterior chest and then to the back [6, 7]. No R wave area was small and was located only on the right upper chest or on the upper back. On the isochrone maps of patients with myocardial infarction (73 men, 12 women, 35 years to 69 years old), abnormal findings were observed, like different locations of no R wave area, delayed ventricular

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activation times near this area, an islandlike zone of delayed activation in the left precordium [6].

Another study evaluated temporal changes in body surface R peak isochrone maps and left ventricular function in 22 patients with myocardial infarction aged 47 years to 69 years [7]. They found decreasing areas of abnormal activation pattern corresponding to the improved ejection fraction of the heart in a few patients. The control group was the same as in the previous study [6].

The next study reported in 50 patients with myocardial infarction aged (57.5 ± 9.9) years (mean ± standard deviation, also in the following text) activation delay on ventricular activation time maps, which was associated with a lower ejection fraction and a higher incidence of ventricular tachycardia compared to patients without activation delay [8]. Furthermore, a typical finding in patients with myocardial infarction was a large area without R waves on the anterior chest. The control group consisted of 40 normal subjects, all men, 21 years to 55 years old.

From these studies, 10 criteria were proposed and applied to a group of 4 healthy girls aged 16 years to 17 years [9]. Although the activation sequence was as expected in healthy subjects, a time shift of about 10 ms to 20 ms was observed, probably due to inappropriate setting of the QRS onset, which was done automatically by the implemented software that used only a single time instant for setting the zero voltage (instead of a baseline).

Some papers dealt with ventricular activation maps in children with renal diseases with a control group consisting of 26 children aged (15.2 ± 1.2) years. On their mean map of normal subjects, ventricular activation started—the shortest median time was 12 ms, located on the anterior thorax around the midsternal line and on the upper back along the left scapular line [10]. The highest values of ventricular activation time (median 62 ms) were found around the right shoulder and on the right upper back. In patients of comparable age with renal diseases, this time was statistically significantly longer (median 110 ms), although activation started at comparable times, only on the lower sternum, not on the upper part of the chest or the back.

Prolonged activation times were found in 33 teenagers aged (15.0 ± 2.1) years with type 1 diabetes, although the activation started at similar times as in the control group made up of 30 age-matched healthy adolescents [11]. It should also be mentioned that all the patients included in this study had normal findings on the standard ECG.

The same control group was used to assess ventricular activation times after kidney transplantation in 5 patients aged (20.8 ± 1.2) years [12]. A relationship was demonstrated between changes in the maps and the duration of dialysis. After kidney transplantation, the abnormal distribution of isochrones and ventricular activation times showed some significant and specific regression.

All these studies used a full grid of electrodes: 87 body surface leads with the mapping system HPM-5100S, Chunishi Denshi Company [6, 7, 8], or the mapping system HPM 7100, Fukuda Denshi [10, 11, 12]) or 80 body surface leads with the mapping system CARDIAG 128.1 [9]. A full grid system means that all electrocardiograms are measured at each node of the grid. Therefore, we were interested in ventricular activation maps measured with a limited lead system where electrocardiograms are measured in selected grid nods and the remaining are reconstructed [13] to determine whether they will provide comparable information about the activation sequence.

The aim of this study was to analyse the R wave peak isochrone maps in young adult volunteers using the time normalisation of the QRS complex.

Materials and Methods

Ventricular activation time maps of 13 men without known cardiovascular diseases aged (19 ± 1) years were examined. Although this is a retrospective study, all subjects provided their informed written consent to participate in body surface potential mapping studies prior to data recording.

In each subject, unipolar electrocardiograms for body surface potential mapping were registered using the limited 24-lead system after Barr [13], based on a 150-point grid of 10 rows and 15 columns implemented in the ProCardio mapping system (Institute of Measurement Science, Slovak Academy of Sciences) as previously described in detail [14]. All data were recorded in the supine position during normal expiration at a sampling rate of 500 Hz and filtered using a Pipberger filter.

Linear baselines (zero isoelectric lines) were established along the TP segments of each electrocardiogram, defined as the interval from the end of the T (or U) wave to the onset of the P wave in the next heartbeat, when no electric activity of the heart is recorded and voltage is effectively zero [15]. The onset and offset of the QRS complex were manually established from the root mean square (RMS) signal as the time instants when the signal approached the baseline (in the RMS signal of 0.01–0.02 mV, approximately) and stopped to decrease for at least three milliseconds when reading the values from the middle of the QRS complex.

Electrocardiographic curves in the 150 grid points were reconstructed according to the procedures described by Barr [13]. These data were exported from the mapping system to MS EXCEL files, from which one of the authors constructed maps of the peak R wave time [9]. These maps display the instant time values

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(assigned to the sequential number of the map obtained from the time normalisation), in which the potential on the chest surface reached the peak of the R wave being at least 0.05 mV high. When the R wave was absent in a lead, this was called the "no R wave area". In the case of two R waves in the ECG trace, the greater one was taken for the measurement.

For time normalisation, the duration of the QRS complex of each subject was divided into 20 equidistant parts [15]. The activation sequence represented by the time steps (21 steps in total) was analysed in each subject, as well as in the group mean R wave peak map. For easier comparison with earlier published maps, we present the original time values starting from the QRS onset.

Results

The mean duration of the QRS complex in the group was (89 ± 9) ms, range from 80 ms to 110 ms. The mean time between two subsequent time steps was (4.4 ± 0.5) ms (concerns time normalisation). The values of the R wave peaks reached up to 2.8 mV.

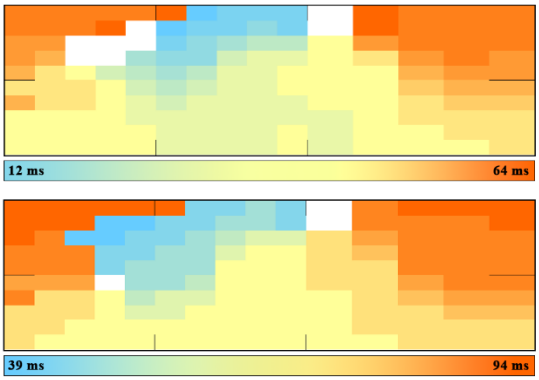

Fig. 1: Examples of R wave peak body surface isochrone maps of 2 different subjects. Each rectangle represents the chest surface enrolled into the plane along the midaxillary line. The left half of the rectangle corresponds to the anterior chest and the right half to the back. The upper edge of the map is at the level of the jugular notch, the lower edge at the level of the navel. The short horizontal bars indicate the position of the fourth intercostal space. Short vertical bars (from left to right) indicate the positions of the sternum and the left midaxillary line, respectively. The darkest blue colour represents the first occurrence of the R wave peak displayed on each map, and the darkest red colour represents its last occurrence. No R wave areas are displayed in white colour. The time values on the scales are in milliseconds.

The very first peak of R waves (the start of the activation sequence) on the maps of individual subjects was found 12 ms after the QRS onset in the left clavicular area (Fig. 1, top).

The very last peak of R waves was found on the maps of individual subjects 94 ms after the QRS onset in a larger area around the right shoulder (Fig. 1, bottom).

No R wave areas were found mainly in the middle left anterior chest and the lower part of the left back, as well as around the right shoulder (Fig. 2, top). In individual maps, they represented 3% ± 2% of the chest surface.

The first occurrences of the R wave peaks in the whole group covered mainly the upper right anterior chest and were found in about 16% of the chest surface (Fig. 2, middle). Regardless of the location on the chest, the mean first occurrence of the peak of the R wave was at (21 ± 9) ms, range (12–39) ms.

The last occurrences of the R wave peaks in the whole group covered mainly the upper chest, partially overlapping with the first occurrence locations and were found in about 19% of the chest surface (Fig. 2, bottom). Regardless of the location in the chest, although usually around the right shoulder, the peak of the last occurrence of the R wave occurred at the mean time (77 ± 9) ms.

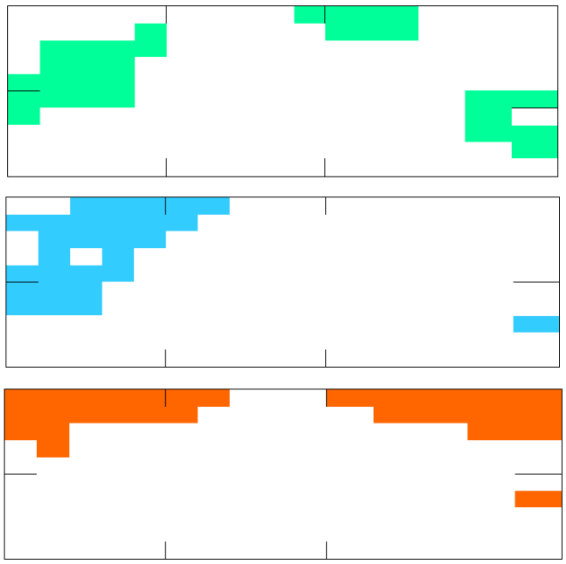

Fig. 2: Areas on the chest surface with no R waves (top), the start of activation (middle) and ending activation (bottom), all in individual maps.

In the locations of V_1 to V_6 typically analysed for the standard ECG leads, the mean R wave peak times were as follows:

- V_1: (28 ± 10) ms, range (20–46) ms;
- V_2: (29 ± 11) ms, range (20–51) ms;
- V_5: (49 ± 9) ms, range (40–66) ms;
- V_6: (50 ± 10) ms, range (40–72) ms.

When considering the whole chest (the whole map), the time interval between the 'onset' of activation (the very first peak of the R wave) and the 'end' of activation (the very latest R wave) regardless of its location was (56 ± 4) ms, range (48–66) ms.

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We found that ventricular activation (depolarisation) started in the upper half of the anterior chest (Fig. 1, blue colour); In the standard 12-lead ECG this corresponds mainly to the areas above the positions of V_1 and V_2. Then the spread of activation continued downward, approximately along the main axis (anatomical) toward the apex, then mainly leftward towards the positions V_4 to V_6 (Fig. 1, light green to yellow). On the back, the activation spread from the lower part of the chest towards the right shoulder (toward the heart base), sometimes even reaching the anterior chest (Fig. 1, red colour), i.e., the latest R wave peaks occurred in areas which are not explored by the standard chest leads. However, the activation never spread from the starting positions directly rightward and directly upward.

This fact may be regarded somewhat misleading in the group mean R wave peak map (Fig. 3, top), where the activation seems to spread also in these directions, although much more slowly than elsewhere. This 'spread' results from averaging the values in the overlap areas of the earliest and latest R wave peaks (Fig. 2, middle and bottom), which is also supported by the standard deviation map (Fig. 3, bottom). The highest variability is seen on the right upper anterior chest, partially overlapping with no R wave area (Fig. 2, top).

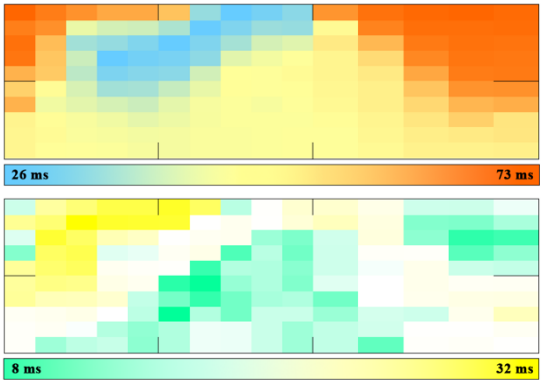

Fig. 3: Group R wave peak body surface isochrone map (top) and map of standard deviations (bottom). The map of means is coloured the same way as the individual maps in Fig. 1 and shows the time interval from 26 ms to 73 ms. In the map of standard deviations, the lowest variability of values is displayed in green (from 8 ms) and the largest in yellow (till 32 ms).

Discussion

The typical duration of the QRS complex in men aged 18 to 29 years is (96 ± 9) ms, range from 80 ms to 114 ms [1], which corresponds to our data. Because we are more interested in the sequence of electrical activation in the heart than specific activation times, time normalisation allows for a more suitable comparison of individual events in different QRS complex durations than evaluation using maps constructed with a constant time step [16]. Time normalisation avoids comparing, for example, the activation of the heart apex in one patient with the activation of the ventricular base occurring in another patient at the same time, mainly when analysing mean maps.

The use of a limited lead system of electrodes [13] allowed us to cover the whole chest regardless of the shape and dimensions of any subject as described in Methods. Full grid lead systems usually do not have this possibility as they use strips of electrodes with strictly set distances between them [10–12].

The amplitudes of the R wave, when measured using standard chest ECG leads, range from 0.1 mV in lead V_1 (the lowest values) to 3.6 mV in lead V_4 (the highest values) [1]. Our maximum R waves were in this range.

As the map leads cover not only the area of precordial leads V_1 to V_6, for the evaluation of R wave peaks, the value of 0.05 mV was selected as the recognition threshold. In a 12-lead ECG, this value represents the amplitude of a half-millimetre wave when using the standard voltage calibration of 10 mm / 1 mV. Therefore, it is low enough to display smaller or more distant activation fronts in the heart, but it should be high enough to exceed the unavoidable noise of the recorded signals.

In any 12-lead ECG, intrinsicoid deflection is measured from the onset of the QRS complex to the peak of the R wave in each individual lead separately. When using body surface maps, the QRS onset is set as a single although the same time instant for all evaluated leads. This may lead to somewhat prolonged times when comparing the data. Despite this fact, our values were in the published ranges [1] in V_5 (41 ± 13) ms, range (26–85) ms, as well as in V_6 (41 ± 12) ms, range (22–87) ms. The influence of such delay caused by improper software settings of the QRS complex was published earlier [9] where it led to abnormal findings in healthy volunteers. On the other hand, it should be mentioned that the intrinsicoid deflection slightly decreases with increasing age.

Regarding the activation sequence on the mean map, our findings are in agreement with previously published maps, although they used different mapping lead systems, examined younger and older subjects, and a higher number of volunteers than we did [6–12]. The activation sequence started on the anterior chest around 20 ms after the QRS complex and ended on the upper back or around the right shoulder after 60 ms or later.

In addition to the retrospective observational study design, our study had the following limitations.

The number of volunteers in this study was small, and therefore, the findings should be interpreted as exploratory in nature.

Using the time normalisation of the QRS complex, the R peaks could be measured with an uncertainty of approximately ± 2 ms in each lead. However, this did

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not affect the sequence of activation, as shown by the isochrone maps on the chest surface.

Conclusions

The R wave peak time is an easily measurable parameter in the ECG traces, as the R wave is a positive deflection of the QRS complex representing ventricular activation. It can facilitate or differentiate the diagnosis in a variety of cardiac diseases.

Despite the different methods used, our results are consistent with those of previously published studies. We showed that the R wave peak isochrone maps allow for simple visualisation of activation sequence also when using a limited lead system. This may be a suitable basis for further evaluation of patients with cardiac diseases related to disturbed or delayed ventricular activation, such as ventricular hypertrophy, myocardial infarction, or bundle branch blocks.

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References

[1] Macfarlane PW, van Oosterom A, Pahlm O, Kligfield P, Janse M, Camm J (eds). Comprehensive Electrocardiology. Springer Science & Business Media; 2010.

[2] Pérez-Riera AR, de Abreu LC, Barbosa-Barros R, Nikus KC, Baranchuk A. R-Peak Time: An Electrocardiographic Parameter with Multiple Clinical Applications. Annals of Noninvasive Electrocardiology. 2016;21(1):10–9. DOI: 10.1111/anec.12323

[3] Aiken AV, Goldhaber JI, Chugh SS. Delayed intrinsicoid deflection: Electrocardiographic harbinger of heart disease. Annals of Noninvasive Electrocardiology. 2022;27(3):e12940. DOI: 10.1111/anec.12940

[4] Francia P, Silvetti G, Cosentino P, Cristiano E, Adduci C, Tini G, et al. Relation of delayed intrinsicoid deflection of the QRS complex to sudden cardiac death in patients with hypertrophic cardiomyopathy. International Journal of Cardiology. 2022;366:42–7. DOI: 10.1016/j.ijcard.2022.06.066

[5] Kozlíková K. Povrchové integralové mapy, ich charakteristiky a metódy kvantitatívnej analýzy (Surface integral maps, their characteristics and methods of quantitative analysis). Bratislavské lekárske Listy (Bratislava Medical Journal). 1990;91(11):815–23.

[6] Ikeda K, Kubota I, Igarashi A, Yamaki M, Tsuiki K, Yasui S. Detection of local abnormalities in ventricular activation sequence by body surface isochrone mapping in patients with previous myocardial infarction. Circulation. 1985;72(4):801–9. DOI: 10.1161/01.cir.72.4.801

[7] Ikeda K, Kubota I, Yamaki M, Hanashima K, Nakamura K, Tonooka I, et al. Temporal changes in body surface peak R isochrone maps and left ventricular function in patients with myocardial infarction. Journal of Electrocardiology. 1987;20(3):212–8. DOI: 10.1016/s0022-0736(87)80018-3

[8] Hanashima K, Ikeda K, Yamaki M, Tsuiki K, Yasui S. Clinical-significance of body-surface isochrone maps for predicting ventricular arrhythmias in patients with previous myocardial infarction. Japanese Circulation Journal. 1988;52(3):203–10. DOI: 10.1253/jcj.52.203

[9] Kozlíková K, Hulín I, Kneppo P, Sapáková E, Bakosová M, Molnárová Z. Body surface isochrone maps of peak R in normal adolescent girls. Physiological Research. 1993;42(2):99–102.

[10] Laszki-Szczachor K, Polak-Jonkisz D, Zwolinska D, Makulska I, Rehan L, Sobieszczańska M. Effects of hemodialysis on ventricular activation time in children with end-stage renal disease. Hemodialysis International. 2015;19(1),115–23. DOI: 10.1111/hdi.12189

[11] Zubkiewicz-Kucharska A, Noczyńska A, Sobieszczańska M, Poreba M, Chrzanowska J, Poreba R, et al. Disturbances in the intraventricular conduction system in teenagers with type 1 diabetes. A pilot study. Journal of Diabetes and its Complications. 2021;35(11):108043. DOI: 10.1016/j.jdiacomp.2021.108043

[12] Laszki-Szcząchor K, Zwolińska D, Sobieszczańska M, Makulska I, Polak-Jonkisz D. Dynamics of changes in heart conduction system in dialyzed young adults after kidney transplantation--pilot study. Transplantation Proceedings. 2014;46(8):2708–13. DOI: 10.1016/j.transproceed.2014.09.038

[13] Barr RC, Spach MS, Herman-Giddens GS. Selection of the number and positions of measuring locations for electrocardiography. IEEE Transactions on Biomedical Engineering. 1971;18(2):125–38. DOI: 10.1109/tbme.1971.4502813

[14] Rosík V, Tyšler M, Turzová M. Portable device for ECG mapping. In: Measurement. SAS, Bratislava, 1997:367–70.

[15] Kozlíková K, Trnka M. Varied onset of heart ventricular depolarization in different age groups of healthy volunteers. Physiological Research. 2019;68(Suppl 4):S389–97. DOI: 10.33549/physiolres.934379

[16] Stilli D, Musso E, Barone P, Ciarlini P, Macchi E, Regoliosi G, et al. Newer data on the configuration and variability ranges of body surface maps in a sample of normal subjects. Journal of Electrocardiology. 1988;21(1):1–14. DOI: 10.1016/s0022-0736(88)80018-9

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