

The Methodology of Clinical Analysis of Electric Heart Field

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Summary

A brief description of the methodology of analysis of the electric heart field using electrocardiograms, vectorcardiograms, diagrams of potential maxima/minima and body surface potential maps is presented. The text is focused on the description of different kinds of isopotential and isointegral maps and their diagnostic possibilities. A detailed description of the diagram of potential maxima/minima and its place in diagnosis of different disturbances of the heart muscle and conduction defects is given.

Key words

Electric heart field – Electrocardiography – Vectorcardiography – Diagram of potential maxima/minima – Body surface mapping

Introduction

Clinical analysis of the electric heart field is a modern non-invasive electrocardiological diagnostic method which provides information about all physiological and pathological states of the heart that have their electric manifestation. Although other non-invasive and invasive methods can also inform about heart functions, analysis of the electric heart field is distinguished among them by its complexity, reproducibility and is innocuous for the patient (Stojan 1991).

We analyze the cardiac electric field in three stages: electrocardiography (ECG), vectorcardiography (VCG) and body surface mapping using various types of maps manifesting electric heart activity on the body surface. The analyzed signals are recorded during a single heart cycle synchronously in 80 leads on the thorax and in limb and chest standard ECG leads and in leads of the Frank VCG system.

Electrocardiogram

In addition to the standard ECG examination, CARDIAG software facilitates a precise measurement of signal values at any moment by means of a time cursor (not only in any standard ECG lead but also in leads on the back and in the right precordium). We try to diagnose conduction defects, cardiac hypertrophy, fibrosis, local ischaemic changes and their combinations. A comparative study (Stojan *et al.* 1992b) has shown that a standard ECG examination

only detects 55 % of pathological findings in comparison with the complex analysis of the electric heart field. This fact justifies the use of vectorcardiography and body surface mapping.

Vectorcardiogram

During the VCG examination we analyze: a) orthogonal leads X, Y, Z and modules with vector velocity, b) angles of the area vectors, and c) VCG loops of PPQ, QRS and STT intervals in their orthogonal projections.

a) Orthogonal leads of the corrected Frank system determine the character of VCG loops in space. These leads are also used for the determination of PPQ, QRS and STT intervals. The X lead resembles lead I, a pathological Q wave in the Y lead is a sign of necrosis or fibrosis in the inferior heart wall. Absence of the Q wave in the Z lead is a sign of a similar defect in the anterior wall. The space module as well as its projections can manifest preexcitation at the beginning of its rising slope, but notches in their depolarization part are more important because they can be caused by fibrous changes in the myocardium. The instant when they occur must be analyzed on the VCG loops because they can also originate in the spatial loops projection. A split of the depolarization part of the module may indicate branch block, especially the right bundle branch block (RBBB).

b) Angles of area vector projections are evaluated for all the three intervals: PPQ, QRS and

STT. We can diagnose, for example, right atrial hypertrophy from the position of the PPQ area vector in the frontal plane. The interval of ventricular depolarization in the frontal plane determines the heart electric axis the value of which exceeds normal limits in left anterior or posterior hemiblock (LAH, LPH). Characteristic manifestation of these diagnoses can also be found in depolarization isopotential maps. The angle between the spatial area vectors is normally less than 90 degrees during depolarization. When its values are greater than 90 degrees, this is a sign of ventricular hypertrophy (especially in combination with an overload), of a ventricular activation defect or of ventricular ischaemic changes.

c) The VCG loops of heart cycle intervals are displayed in all the three orthogonal projections. A close relation exists between the loops and angles of the area vectors. Changes of vector velocity can be assessed by the distance of points representing the vector position at 2 ms intervals. The position of VCG loops in quadrants, their shape, orientation, rotation, and velocity are considered for the diagnosis using standard VCG criteria. Anterior, inferior and posterior wall fibroses have their characteristic manifestation on the VCG loops, but their exact localization and extent can only be determined using isopotential maps. Changes in loop shape during the first 10 to 20 ms of ventricular depolarization are caused by interventricular septal fibrosis, later deformations are

most often caused by fibrous changes or activation defects of the ventricles. Great deformations of repolarization loops are manifestations of ischaemic changes.

Maps

Information about the electric heart field may preferentially be obtained by analyzing various types of maps of cardiac electric activity and of diagrams of potential maxima/minima that concentrate information about body surface potentials during ventricular depolarization and repolarization. The development of amplitudes in the diagram (Fig. 1) differs typically in various pathological states, e.g. in ventricular hypertrophy and bundle branch block and in their combinations with necrotic or fibrous regions, when the diagnosis from standard ECG is not possible. The diagram is also useful in the diagnosis of the Wolff-Parkinson-White syndrome.

The ischaemic heart disease manifests itself in the diagram of potential maxima/minima by further symptoms: elevation or depression of the PPQ segment greater than $150 \mu\text{V}$ is a sign of atrial infarction when the values gradually decrease. On the contrary, when these values rise this is characteristic for Ta (T-auriculaire).

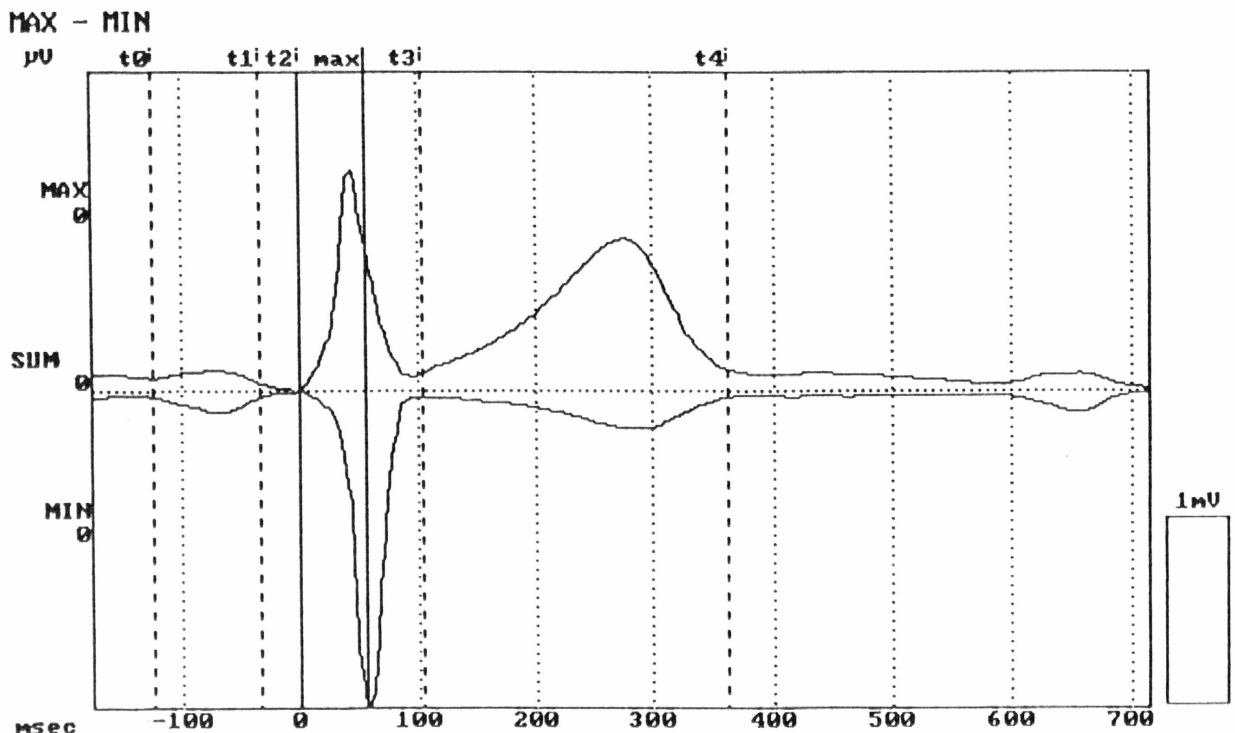


Fig. 1
Diagram of potential maxima and minima in a healthy man

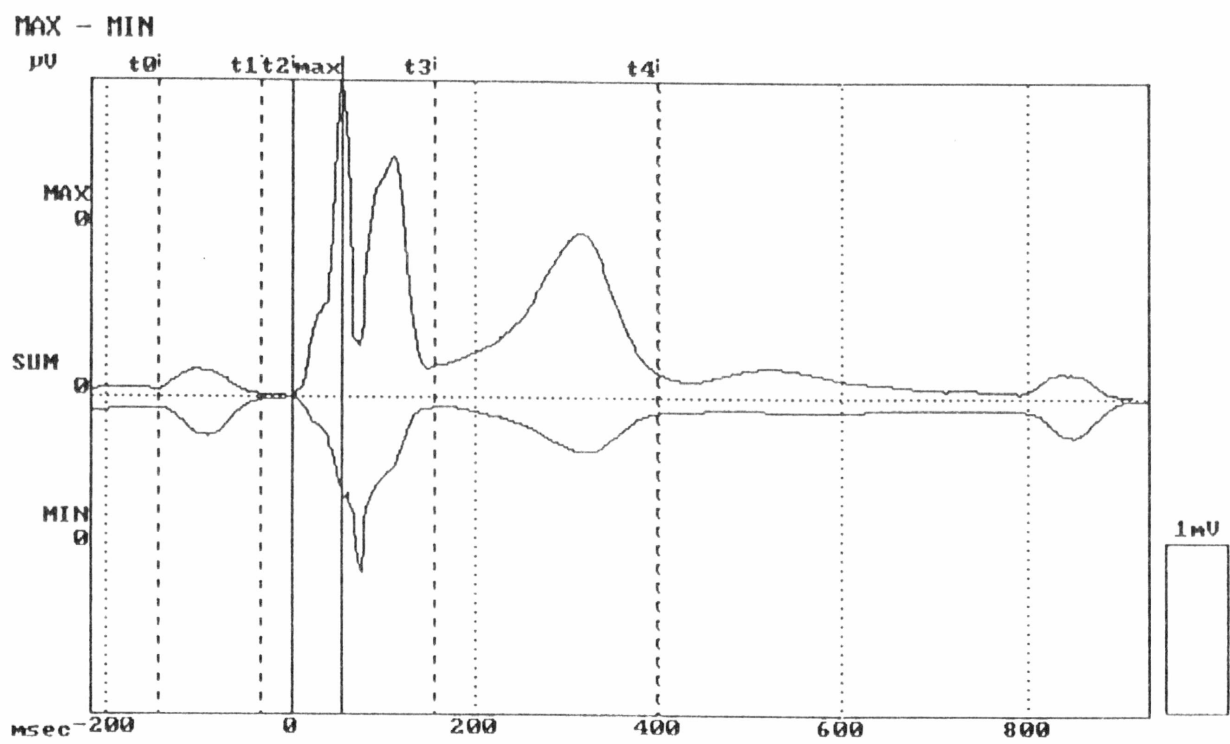


Fig. 2
Diagram of potential maxima and minima in right bundle branch block

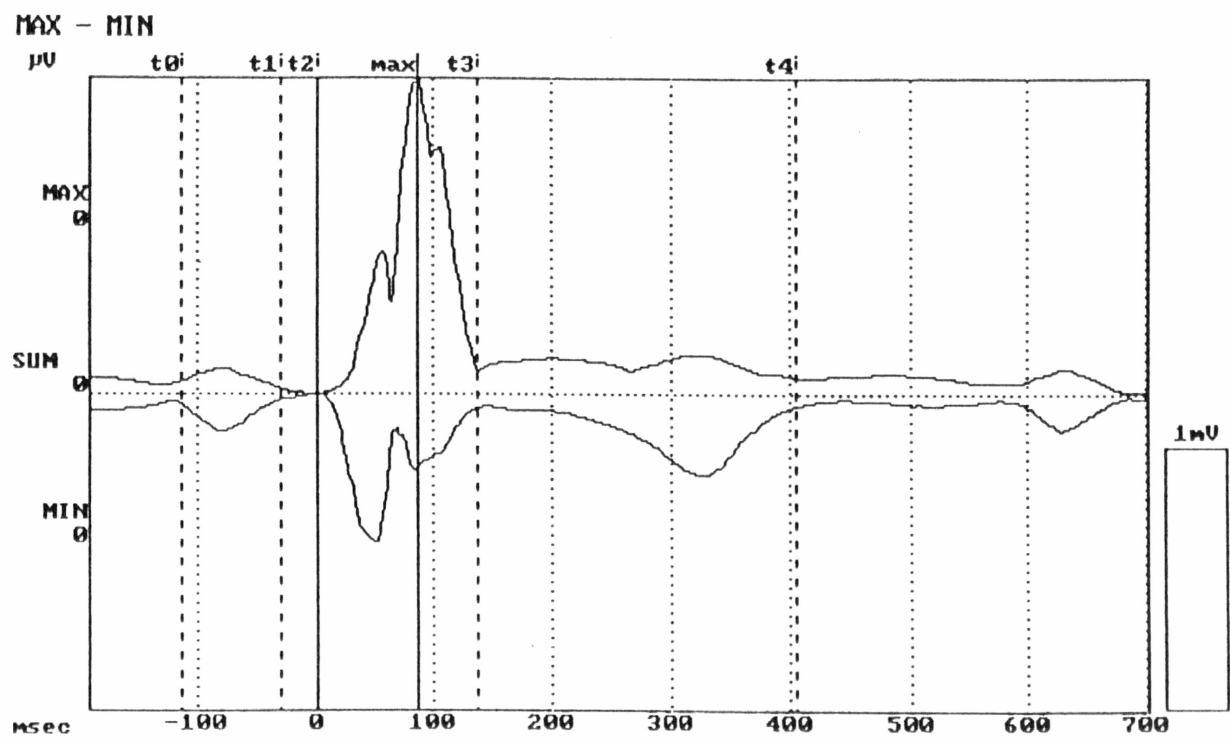


Fig. 3
Diagram of potential maxima and minima in right bundle branch block with old myocardial infarction

Necrosis or fibrosis of the ventricular myocardium are characterized in the depolarizing part of the diagram by a decrease of electropositive values at the instant when the damaged heart region is activated. In the case of interventricular septum, anterior wall and periapical region this occurs in the first 30 or 40 ms of depolarization, in the case of inferior and free left ventricular wall in the middle of depolarization whereas for the posterior wall it occurs at the end of the QRS interval.

When fibrous changes are combined with left ventricular hypertrophy, the diagram discloses the increase of the sum of potential maxima caused by the hypertrophy and a decrease at the moment when the heart region with fibrosis is activated.

A characteristic sign of complete bundle branch block is a prolongation of the ventricular depolarization interval and two positive peaks in the diagram. In RBBB the first positive peak corresponding to the activation of the left ventricle is higher than the second one which corresponds to the activation of the right ventricle (Fig. 2.). When the RBBB occurs in a combination with necrosis or fibrosis of the anterior heart wall, the amplitude of the first positive peak decreases proportionally to the extent of fibrosis so that it has the same amplitude or is even smaller than the second (Fig. 3). This corresponds to a decrease of the positive potential in the fibrous region activated during the first 40 ms of ventricular depolarization. Sometimes a larger first wave in the electronegative part is also present.

Left bundle branch block is characterized by a prolonged depolarization with at least a small second peak and amplitudes of electropositivity smaller than those of electronegativity. Necrosis or fibrosis decrease the amplitude of the positive component of the diagram at the moment when the damaged region of the heart is activated.

The absolute amplitudes of the ST segment are smaller than 200 μV in both polarities under normal conditions and in cases with fibrous changes.

Amplitudes greater than 200 μV in the ST interval immediately following the J point in the positive part of the diagram are characteristic for subepicardial ischaemic changes caused by injury currents. The same signs in the negative part are characteristic for subendocardial ischaemic changes. From the relation between positive values in the ST interval and negative values during the T wave, and *vice versa*, we can judge the corresponding phase of myocardial infarction or aneurysm, when these findings can be localized in the same area of the isopotential maps.

According to the phase of myocardial infarction, various changes appear in the diagram but we can hardly make the correct diagnosis without their verification by isopotential maps, especially since we

cannot localize the damaged region using the diagram of potential maxima/minima only.

High positive potentials during depolarization and high negative potentials during repolarization are characteristic for the ventricular hypertrophy, especially left ventricular hypertrophy with strain. Marked metabolic changes can also be observed in the diagram, e.g. maximum positive values increase in hyperkalaemia while negative values increase in hypokalaemia.

Analysis of the diagram is just a part of the complex analysis of the heart electric field. The next phase consists of analysis of isopotential and isointegral maps with regard to the findings on the diagram of potential maxima/minima. We search for auricular infarctions and Ta, auricular hypertrophies and conduction defects.

Ventricular activation is first assessed by analyzing the isopotential depolarization block of maps and of selected "large" maps. We can then evaluate local changes of the potential on the profile section. The maps of potential divergence show regions with local changes of potential more distinctly because they emphasize deformations of isopotentials. We observe not only the shape of isopotentials but also the values and shift of electropositivity to the left in correspondence with the rotation of the VCG depolarization loop. We can thus evaluate not only local changes of the electric field but also diffuse changes. Evaluation of the development of the field is important for the differentiation of fibrosis and local activation block. Manifestation of the inactive region on the maps in fibrosis is hidden in the potential of surrounding normal regions while delayed activation of the region can be observed by additional electropositivity in the case of activation block. The isointegral maps are also important for the analysis of depolarization because they emphasize those heart field abnormalities that take place during longer periods of time. The isoarea maps are in fact isointegral maps of a chosen interval of depolarization and they emphasize the manifestation of electric structures activated during this interval.

Next, we analyze the map of potential maxima of the R1 wave. The isopotential lines have a circular or elliptic shape under normal circumstances. Under abnormal conditions, e.g. transmural fibrosis, we can observe zero values in the region of fibrosis. In non-transmural fibrosis we can find a local potential decrease.

The regions with the R2 wave on the map of its potential maxima can be caused by following mechanisms: most often by a delayed activation in the RBBB, by a delayed activation of the outflow part of the right ventricle, by separated activation of the papillary muscle in the right ventricle or by fibrosis in the posterior wall.

Mapping of the S wave is of lesser practical importance, but maps of the QS wave and Q+QS wave are very important. The QS wave map shows regions where no electrical activity is present during the whole cycle. These regions are, under normal conditions, situated near the base of the heart and in case of transmural fibrosis especially in the anterior, inferior and posterior heart wall. The importance of this map for further analysis of ventricular repolarization is due to the fact that potential values in these regions during repolarization are caused only by a projection of the summed repolarization vector. The local electrical generation is missing because there is no ventricular myocardium near the base of the heart and the region with transmural fibrosis produces no depolarization or repolarization potentials.

Repolarization isopotential maps are formed in a similar way as depolarization maps but the electropositivity does not move across the thorax under normal conditions. We can measure additional electropositivities and electronegativities in profile sections and maps of isodivergence are also available. Using them we can diagnose, for example, ischaemia in subendocardial or subepicardial layers and from their development during repolarization we can estimate their duration. The examination is very sensitive and reveals changes that cannot be observed on standard ECG and VCG.

Similarly as in depolarization, we can display isointegral isoarea maps, and maps of asynchronous maxima/minima of the T wave and diagnose overload, current of injury and phase of ischaemia.

It is important to realize that fibrous changes manifest themselves mainly during depolarization but they also partially influence repolarization maps. Ischaemic changes manifest themselves only during the repolarization phase but they partly affect depolarization maps.

Total isointegral map of ventricular depolarization and repolarization is, in fact, a map of the local ventricular gradient. Local changes on this map can be interpreted as a disposition to extrasystolic arrhythmias.

The last step of this complex examination is the analysis of the ventricular activation map. This map shows, e.g. prolonged activation time above the left ventricle in hypertrophy, above ischaemic regions, in cases of bundle branch or fascicular blocks. In regions

with transmural fibrosis the activation time has zero values.

Conclusions

Findings during the phases of complex analysis of the heart electric field gradually contribute to the diagnosis. Partial findings should not be contradictory because their origin is the same.

The method can be used in clinical practice (Stojan *et al.* 1992a,b). The examination is non-invasive and easily reproducible so that it facilitates a comparison of findings after physical or mental stress, checking of possible restenosis after percutaneous transluminal coronary angioplasty or a bypass, and testing of vasoactive drugs (e. g. isosorbide dinitrate) in primary as well as secondary prevention of ischaemic heart diseases.

The complex analysis of the heart electric field has its place among other non-invasive and invasive examination methods in cardiology. It is more reliable than the exercise test in diagnosing ischaemic heart disease. Though the echocardiographic examination is more reliable for measurements of heart anatomy, it cannot reveal heart activation defects, small myocardial fibrosis and ischaemic regions at all. Coronary angiography provides information about the anatomical state of the coronary bed but the actual state of the myocardium can be inferred only indirectly. This is why the results of coronary angiography sometimes differ from those obtained by the complex analysis. On the contrary, on the basis of the localization of fibrous changes it is possible to estimate the occlusion of the coronary artery that supplies the affected heart region. But the coronary angiography is necessary for cardiac surgery. Scintigraphic examination of the myocardium is not diagnostically reliable in small fibrosis or ischaemia.

We can conclude that the diagnostic possibilities of the complex analysis of the cardiac electric field can be assessed more accurately by comparing the findings obtained by this method with those obtained by other methods and by autopsy.

Acknowledgements

This work was carried out under a project sponsored by IGA, Czech Ministry of Health, Grant No. 1111-1.

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Reprint Requests

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