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Diagnosis of Myocardial Viability Based on Magnetocardiographic Recordings

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Introduction

From the 1960s through the 1970s and the 1980s magnetocardiography data were used predominantly for time domain and signal shape analysis of single leads or non-simultaneously obtained mapping data respectively [1]. This has provided insights into patient risk stratification and prognosis. In the 1990s, however, magnetocardiography began to play an increasingly important role in diagnosing and localising the sources of cardiac magnetic fields. This new application of inverse-solution-based analysis of magnetic fields has become known as Magnetic Source Imaging (MSI). In this respect, the development of source imaging techniques and multisensor devices [2] is advantageously interdependent.

Therefore, magnetocardiography developed as a new experimental discipline of functional imaging. The discipline has moved from no-model signal analysis to a broad emphasis on the functional localisation of cardiac electrical activity and characterisation of patients with known disease [3, 4]. Since that time, the application of new techniques of data analysis has evolved progressively, while the clinical relevance of currently available procedures is being assessed on an ongoing basis [5].

In this paper, following a brief introductory review from the clinical point of view, the major techniques of MSI of distributed cardiac electrical activity used in routine application at the Biomagnetic Centre Jena will be presented and discussed.

Coronary artery disease

Ischemic heart disease is usually due to obstruction of the coronary arteries, which in turn most commonly results from artherosclerosis. The importance of coronary artery disease in contemporary society is attested by the almost epidemic number of persons afflicted. It causes more deaths, disability, and economic loss in industrialised nations than any other group of diseases.

Myocardial infarctions may be divided into two major types: transmural infarcts, in which myocardial necrosis involves the full thickness of the ventricular wall, and subendocardial (nontransmural) infarcts, in which the necrosis involves the subendocardium, the intramural myocardium, or both without extending all the way through the ventricular wall. Eight to 10 days following infarction, the thickness of the cardiac wall in the area of the infarct is reduced as necrotic muscle is removed by mononuclear cells. A loss of functioning myocardium results. As a consequence of infarction, changes in left ventricular size, shape, and thickness involving both the infarcted and the noninfarcted segments of the ventricle often occur. These changes are referred to as "ventricular remodeling", a process that in turn can influence function and prognosis.

One of the most important developments in the treatment of patients with infarction consists of establishing reperfusion of ischemic heart muscle. It improves hemodynamics and decreases infarction size (the latter in early reperfusion).

Significant stenosis of one or more major epicardial arteries, which supply at least a moderate-sized area of viable myocardium, in a patient who has ischemia episodes, angina or major ventricular arrhythmia has to be treated by coronary revascularisation. Efforts to prevent and/or treat the disease have the potential for success. Diagnostic procedures are essential for the correct selection of patients and coronary vessels prior to revascularisation. In this respect it would be of great importance for the future of magnetocardiography to enter the field of assessment of myocardial viability.

Diagnostic procedure

The crucial problem in coronary artery disease refers to the identification of viable myocardium. Stunning and hibernation of myocardial segments may lead to reduced kinesis of viable tissue. Therefore, wall motion analysis fails to correctly diagnose viability. Additional procedures are required.

Myocardial infarction can become visualised by nuclear cardiology either as a "cold spot" or as a "hot spot". Cold spot imaging is based on perfusion defects, whereas the infarct can become visualised as a "hot spot" using a monoclonal antibody specific for intracellular myosin.

Since the gold standard in the diagnosis of myocardial viability (PET: combined perfusional/metabolic imaging) is not available everywere, stress-echocardiography or ^{201}Tl- or ^{99m}Tc-SestaMIBI stress imaging respectively are the most widely used standard procedures.

A precise localisation of myocardial infarction based on the standard ECG is not always possible. The accuracy of such a localisation is influenced, for example, by the distance of the electrode from the heart, which varies considerably among individuals. Therefore, additional anatomical constraints and the application of algorithms to solve the inverse problem of electrocardiography or magnetocardiography respectively are desirable.

Method

23 patients with prior myocardial infarction and 25 healthy subjects/patients without infarction were investigated. The presence of infarction (ECG, history, elevated blood level of cardiac enzymes) was proven by angiography. Fig. 1 displays an example of ECG and angiography of a patient suffering from an inferobasal-inferomedial aneurysm of the left ventricle.

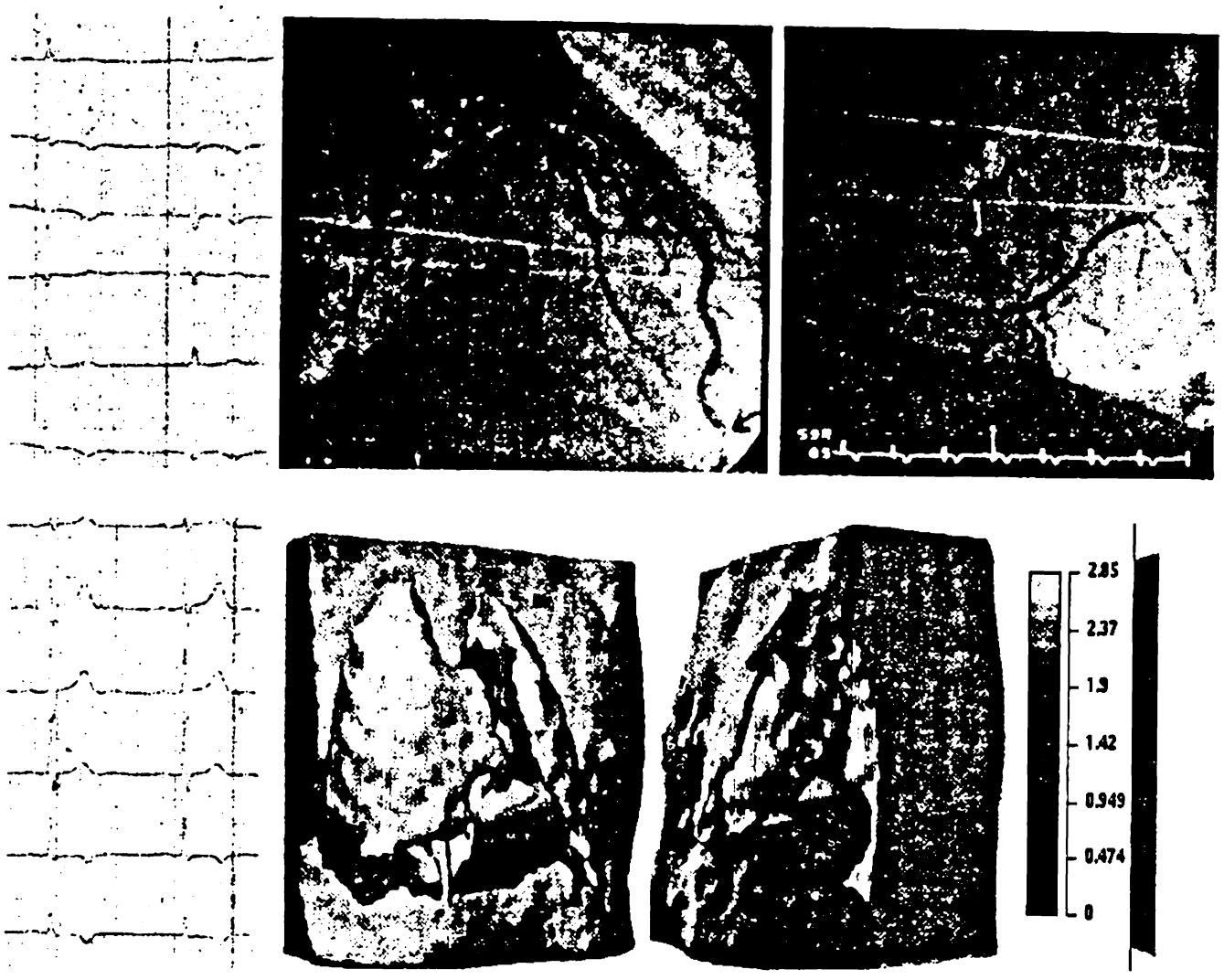

Figure 1: Top: ECG (Einthoven leads), angiogram (30° RAO, 60° LAO); Bottom: ECG (Wilson leads), MSI (30° RAO, 60° LAO); Inferobasal-inferomedial myocardial infarction

Magnetic field maps

Magnetocardiograms were taken using a multisensor device (Philips). The device consists of two sensor heads, each holding 31 sensors [6]. The recording time was set to 100 to 600 s (1000 Hz sampling rate). The sensor heads were tilted by about 30°. The coordinates of the torso relative to the multichannel system were determined by localising the center coordinates of 10 coilsets.

Magnetic resonance images

MRIs were taken with a 1.5 T machine (Philips Gyroscan® ACS II). Prior to investigation, coilsets were replaced by oil pills. A series of 40 to 60 transverse slices (5 mm thick, T1 weighted turbo field echo scan) were taken. The scan time window was placed in the late enddiastolic heart phase at R upstroke.

The data processing, i.e. segmentation of three-dimensional surfaces, calculation of current densities, and presentation of results was done with the software package Curry® [7]. The resulting stack of MRI slices was transformed into a three-dimensional dataset [8].

The surfaces required for volume conductor modeling were segmented by a threshold steered region growing algorithm.The following thinning procedure resulted in a reduced list of surface points. This list was used for the computation of a boundary element volume conductor model. The segmentation of the source structure (left ventricle) was done by hand using a freehand cursor brush in three zoomed orthogonal image planes. The left ventricular endocardial surface was divided into a grid of several hundred points of equal distance using a thinning algorithm. The list of myocardial point coordinates served as dipole positions for reconstruction. The overall time for MRI preparation took about one hour (30 minutes scan preparation and scan time, 30 minutes for 3D surface processing).

Current density images

Current density was calculated for a few hundred support points at the peak of the QRS complex. The reconstruction points were regularly spaced on the left ventricular endocardial surface with a mean distance of 5 mm. The current density had to satisfy the constraints of minimum norm least squares, lead field regularisation,

and noise regularisation. Noise regularisation was performed using the bend values of a L-curve. The resultant current density distribution was presented as a colour-coded surface plot of the left ventricle for the assessment of regional electrical activity. The localisation and extent of the area of low current density in the infarction patients were determined for seven segments of the left ventricle. Low current density was assumed for the currents below an arbitrarily defined value (1 mA/mm²). Localisation results were compared to ECG, echocardiography, scintigraphy, and left heart angiography.

Results

There were marked differences in mean current strength, localisation, and extent of areas of low current density between the infarction patients and the healthy subjects. Mean current densities of the patients turned out to be lower compared to the current densities of the healthy. A mean point distance of 5 mm for the generation of support point lists resulted in a standardised patch area of about 30 mm². According to the ventricular remodeling and enlargement after myocardial infarction, the number of points or currents respectively was considerably higher in patients. Nevertheless, the sum of all currents was lower in patients compared to the healthy subjects.
In table 1 MSI is compared to contrast angiography in the infarction group.

Classification	Myocardial Segment - Infarction Patients						
	antero-basal	antero-medial	apical	infero-medial	infero-basal	septal	postero-lateral
pp	3	13	13	9	1	11	7
nn	15	5	7	9	14	7	12
pn	2	2	2	5	8	0	1
np	3	3	1	0	0	5	3
Σpp+nn	18	18	20	18	15	18	19
Σpn+np	5	5	3	5	8	5	4
pp	abnormal laevocardiography and abnormal MSI						
nn	normal findings for both procedures						
pn	abnormal angiography and normal MSI						
np	normal angiography and abnormal MSI						

Table 1 Comparison of angiography and MSI in 23 infarction patients (i.e. 7 segments in each patient, 161 segments overall)

In 23 infarction patients, the classifications of 126 out of 161 segments (78%) were in agreement (abnormal-abnormal or normal-normal respectively). 35 out of 161 segments (22%) had a different classification (abnormal-normal or normal-abnormal respectively). The interpretation of these findings will be discussed later on. The highest number of mismatch segments was found inferobasally.
Table 2 summarizes the results from 25 healthy subjects/patients without a history of myocardial infarction.

Error! Bookmark not defined. **Error! Bookmark not defined.**Classification	Myocardial Segment - Non-Infarction Patients/Healthy						
	antero-basal	antero-medial	apical	infero-medial	infero-basal	septal	postero-lateral
n	2	2	0	4	5	6	6
p	23	23	25	21	20	19	19
Error! Bookmark not defined.n	normal MSI						
p	abnormal MSI						

Table 2: MSI in 25 normals/patients without infarction (7 segments in each normal/patient, 175 segments overall)

25 out of 175 Segments (14%) turned out to have an abnormal low current density. Note that segments located in an interior or nearly position again had the highest rate of abnormal MSI results.

Discussion

Despite the obstacle posed by the non-uniqueness of inverse solutions, we have shown that an estimation of the myocardial source activity in coronary artery disease can be achieved by a solution which minimizes the square of power integrated over a left ventricular surface. Low regional current density turned out to be the hallmark of infarcted myocardial tissue in coronary artery disease. In this study there are several reasons for the mismatch of angiography-MSI. The interpretation of the coherence between regional wall motion and regional current density is limited by a lack of knowledge about the regional amount of viable myocardium. Because hypokinesis may occur in stunning and hibernating myocardium, as well as in myocardial infarction with varying degrees of myocardial fibrosis, reference procedures (PET, scintigraphy, endocardial catheter mapping) are required in future studies. Reversibility of wall motion abnormalities would be the way to differentiate underperfused but viable myocardium from myocardial scaring in a prospective study.
Assuming that MSI gives a true copy of regional electrical activity, the identification of hypokinetic but viable myocardium (pn-segments, Tab. 1) would become possible. Nevertheless, the results of this preparatory study should considered to be preliminary. Much work is to be done in this field.

Summary

Multisensor magnetocardiography archieved by sophisticated solutions of the inverse problem of electrocardiography may be of additional diagnostic value in clinical cardiology. The enormous hardware costs hinder the in-hospital application of magnetocardiography only in so far as the diagnostic outcome does not justify this expenditure. The non-invasive pre-interventional examination of patients suffering from coronary artery disease may provide an application of magnetic source imaging which is as fascinating as it is difficult.
Images of distributed cardiac source activity were obtained in 23 patients after myocardial infarction and in 25 patients/normals without a history of infarction. Constraints (minimum norm, lead field normalisation, noise regularisation, anatomical restriction) were used to obtain a unique solution. Current density was assigned to patches of the left ventricular endocardial surface. The resultant images were compared to regional wall motion in left ventriculography.
The vast majority of myocardial segments with wall motion abnormalities (hypokinesia, akinesia, dyskinesia) were identified by magnetic source imaging (low current density). However, in some segments (predominantly inferior) disagreement was found. The subjects without a history of infarction had only few segments with abnormal low current density.
The study demonstrates that inverse solutions aimed at current density calculations on realistic myocardial surfaces have the potential to identify and image electrical properties of the myocardium in coronary artery disease. However, further development of reconstruction algorithms and clinical studies including other reference procedures (PET, scintigraphy) are of paramount importance to the method.

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