Abstract

Cardiac diseases are the leading cause of death in population. Diagnostic tests to detect cardiac dysfunction at an early stage of the disease are desirable. The major focus has been centered on tests evaluating the perfusion of the heart with imaging techniques or detecting alterations in electrical or mechanical function of the heart. The heart generates magnetic fields that can be detected by body surface mapping utilizing super conducting quantum interference device sensors giving magnetocardiograms (MCGs). The advantages of MCG over traditional electrocardiograms (ECGs) are increased sensitivity to small signals and lack of conductivity in body tissues, presentation of direct component signals and primary currents. This review will highlight the basic principles and recent advantages of MCGs, and the application of MCG in clinical diagnosis, especially in cases whose ECGs are non-diagnostic or not specific, such as detecting baseline shift in ischemic heart disease, noninvasive His potential recording, detection of arrhythmic mechanism defining reentrant circuits vs non reentrant mechanism, diagnosis of fetal arrhythmias and prolongation of QT interval. Areas of future basic and clinical research are also discussed.

Key words: cardiac mapping, cardiac magnetic fields, electrical currents

Introduction

Cardiac diseases such as ischemic heart disease, heart failure, and hypertension are the major cause of morbidity and mortality in the population (1–3). Various noninvasive tools have been developed to diagnose cardiac dysfunction and for risk stratification, such as perfusion imaging (using thallium, sestamibi), mechanical dysfunction (cardiac echo), and electrical deterioration (Fig. 1). The heart generates electromagnetic fields that are altered with changes in perfusion, structural, or electrical alteration and can be detected by appropriate sensors on the body surface. Electrocardiograms (ECGs) have been traditionally used to detect changes such as ST segment deviation in ischemic heart disease and ventricular hypertrophy (4), the presence of ventricular preexcitation over an accessory pathway (delta wave) in the Wolff-Parkinson-White syndrome (5), an alteration in cardiac electrical activity within various ranges of myocardium as ST elevation in V1-V3 in Brugada syndrome (6), or defects in cardiac repolarization as QT prolongation in long QT syndrome (7). Although, ECGs are widely used as great diagnostic tools, there are certain limitations as some ECGs may not provide the required information for clinical decision mainly, such as in patients with baseline ECG abnormalities, for example, with ventricular hypertrophy, ischemic heart disease, or use of medication. Additional information could be obtained by assessing the magnetic fields generated by electrical alteration of the heart. This is not only of academic importance, but it is of great use in patients who present to the emergency department with acute coronary syndrome and have a normal or non-specific ECG (8, 9). They are more likely not to be hospitalized (odds ratio 7.7) and suffer adverse events, including increased mortality (8). Therefore, it is essential to have additional diagnostic tools to increase the probability of detecting individuals who are likely to suffer from adverse cardiac events.

Magnetocardiograms (MCGs) are body surface mappings that detect the cardiac magnetic fields and especially weak
electrophysiological phenomena that could be missed by ECGs. Advances in magnetic fields have existed for centuries and early navigators have used the earth’s magnetic fields to guide them in navigation of the earth and sea (Table 1). The cardiac magnetic field was detected much later and was first described by Baule and McFee in 1963 (10). Over the years advances have been made in this field of MCGs (10–16) and it has been developed as a useful diagnostic tool.

Table 1. History of Magnetocardiograms

<table>
<thead>
<tr>
<th>Year</th>
<th>MCGs</th>
<th>ECGs and others</th>
</tr>
</thead>
<tbody>
<tr>
<td>11th century</td>
<td>Compasses in China (detection of the earth’s magnetic field)</td>
<td>ECG in human (Waller, 1887)</td>
</tr>
<tr>
<td>1820</td>
<td>Discovery of electromagnetism (Oersted)</td>
<td>ECG system by Einthoven (1903)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fetal ECG (Cremer, 1906)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ECG in sinus rhythm, AF, and VF (Lewis, 1909-11)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Percutaneous transfemoral catheterization (Seldinger, 1953)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intracardiac recording (Maurice, 1945)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Echocardiogram (Edler &amp; Hertz, 1953)</td>
</tr>
<tr>
<td>1963</td>
<td>Detection of the cardiac magnetic fields (Baule)</td>
<td>EP study (Wellens, 1971)</td>
</tr>
<tr>
<td>1970</td>
<td>Single-channel MCG system using rf-SQUID (Cohen)</td>
<td>MR (Lauterbur 1974)</td>
</tr>
<tr>
<td>1980’s</td>
<td>Multi-channel MCG system using dc-SQUID</td>
<td>Catheter ablation in WPW syndrome by radiofrequency current (Jackman, 1991)</td>
</tr>
</tbody>
</table>

with multichannel recordings. Recent reports provide further evidence conforming MCGs as a practical useful tool for supplemental information in addition to other diagnostic modalities including ECGs (17–20), so as to best take care of patients.

In this review, we will highlight the basic principles of MCG and its potential utility and application in clinical practice.

### Basic Principles of Magnetocardiography

#### Biomagnetic fields

In 1820, Hans Oersted found that every time an electrical current is switched on, a compass needle near the wire carrying the currents moves (Table 1). The needle is moved by magnetic fields accompanying the electrical currents. This principle also applies to the currents associated with the electrophysiological phenomena in the human body. Action potentials originating in myocardial cells create both electrical currents and magnetic fields. At the body surface, the

![Figure 2. Relationship between signals and sensors in electrocardiograms (left) and that in magnetocardiograms (right).](image)

One heart beat was simultaneously recorded by electrocardiograms (lead II, left top) and magnetocardiograms (approximately V1 position, right top) in the same subject, a 60-year-old healthy male. SQUID: superconducting quantum interference device.

![Figure 3. Biomagnetic fields. SQUID: superconducting quantum interference device (21, 22).](image)

![Magnetic strength (Tesla)](chart)

**Environmental fields**
- Earth field
- Urban noise
- Car at 50 m
- Screwdriver at 5 m
- Transistor IC chip at 2 m
- Transistor die at 1 m

**Biomagnetic fields in human**
- Heart
- Skeletal muscle
- Fetal heart
- Eye
- Brain

SQUID
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Table 2. Advantages in Magnetocardiograms

<table>
<thead>
<tr>
<th>Advantage</th>
<th>MCGs</th>
<th>ECGs</th>
<th>Intracardiac electrode recording</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) effects of body tissues on conductivities</td>
<td>low</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>2) contact to skin or heart required</td>
<td>No</td>
<td>Yes, noninvasive</td>
<td>Yes, invasive</td>
</tr>
<tr>
<td>3) skin-electrode or tissue-electrode interference</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>4) components of volume currents</td>
<td>low</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>5) direct-current filtering required</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>6) use for fetal study</td>
<td>Yes, high</td>
<td>Yes, low</td>
<td>No</td>
</tr>
<tr>
<td>7) spatial resolution</td>
<td>intermediate</td>
<td>low</td>
<td>high</td>
</tr>
</tbody>
</table>

Disadvantage

| 1) environmental noise | high | low | low |
| 2) portability | No | Yes | No |
| 3) costs | high | low | high |
| 4) clinical evidence | low | high | low |

Table 3. Electrical Conductivities of Body Tissues

<table>
<thead>
<tr>
<th>Material</th>
<th>$\sigma$ ($\Omega^{-1}$ $m^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sea water</td>
<td>5</td>
</tr>
<tr>
<td>Ringer solution</td>
<td>1.1</td>
</tr>
<tr>
<td>Blood</td>
<td>0.6</td>
</tr>
<tr>
<td>Lung</td>
<td>0.08</td>
</tr>
<tr>
<td>Fat</td>
<td>0.05</td>
</tr>
<tr>
<td>Skull</td>
<td>0.005</td>
</tr>
<tr>
<td>Cardiac muscle along fiber</td>
<td>0.4</td>
</tr>
<tr>
<td>across fiber</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Reference 21.

cardiac electrical currents are measured by ECGs, while the cardiac magnetic fields are measured by MCGs. In other words, ECGs and MCGs both provide information about the same myocardial activity (Fig. 2).

The cardiac electrical currents are strong enough to be recorded at the body surface despite the presence of urban noise, while the cardiac magnetic field is a million times weaker than the earth’s magnetic field and a thousand times weaker than the magnetic fields associated with urban noise (21, 22) (Fig. 3). Picking up these weak physiological signals in a noisy environment is therefore one of the biggest issues in clinical MCG studies. Current systems use superconducting quantum interference devices (SQUIDs), a gradiometer (first-order or up to a third-order), a magnetically shielded room, filtering, and signal averaging (22).

**Advantages of MCGs over ECGs**

As mentioned above, ECGs and MCGs provide information about the same phenomena by using different methods.
Signals thus show a similar pattern in the two kinds of mappings (Fig. 2). MCGs have some advantages over ECGs (21–25) (Table 2). First, they are recorded by a completely non-invasive system measuring the spontaneous magnetic fields that accompany the heartbeat. There is no need for electrodes, radiation, or stimulation procedures. A second advantage is that MCGs are less affected by body tissues than ECGs are (21) (Table 3). A third advantage is that skin electrode interference (Fig. 4) (26) does not exist in MCGs. These three advantages make MCGs unique values in fetal diagnosis (see section V: Clinical application of MCGs: Fetal diagnosis). Moreover, direct-current components are not filtered in MCGs and this advantage makes them valuable for analyzing baseline shift in cardiac ischemia (see section III: Clinical application of MCGs: Ischemic heart disease).

**MCG Measurement**

Because magnetocardiography systems have not been internationally standardized their gradiometers, sensor numbers, sensor intervals, measuring areas, and shielding systems differ between laboratories. The number of measuring points ranges from 1 to 64, and this section describes the 64-channel system used in our laboratory.

**MCG system**

Our studies are done at the MCG laboratory (27) of the University of Tsukuba, Tsukuba, Japan. The magnetocardiography system there was developed in collaboration with Hitachi Central Research Laboratory, Hitachi Ltd. (Tokyo, Japan). After official approval from the Japanese Ministry of Health, Labor, and Welfare in 2002, it was commercialized in 2003 as the first system in Japan (Hitachi MCG model MC-6400, Fukuda Denshi, Tokyo, Japan).

SQUID sensors are placed in a dewar (a cooling system using liquid helium for SQUID sensors, arrow in Fig. 5A). They are first-order gradiometers and are arranged in an 8-by-8 matrix with 2.5-cm intervals (Fig. 5B). The cardiac magnetic fields are measured as a vector marker consisting of three special components, customarily called X, Y, and Z components and respectively corresponding to the horizontal, longitudinal, and vertical components in the anterior-posterior view. We measure the normal components (Z components) of the cardiac magnetic fields with a minimal interval of 0.5 ms and analyze both the normal and the tangential components (X and Y components). The tangential components are calculated from the normal components by using the equation:

\[
B_{xy} \propto \sqrt{\{\frac{\partial B_z}{\partial x}\}^2 + \{\frac{\partial B_z}{\partial y}\}^2},
\]

**Figure 5.** 64-channel magnetocardiographic system. Overview of the system (A), a measuring area of 8 by 8 matrix (sensor interval: 2.5 cm, a measuring area: 17.5 by 17.5 cm) superimposed on magnetic resonance image (B), and signals at 9 out of 64 channels (C). pT: pico tesla.
where $B_z$ is the measured-normal component (in pico tesla, pT) and $B_{xy}'$ is the calculated tangential component (pT/m) (28–30).

**MCG measurement**

To reduce noises, MCGs are measured in a magnetically shielded room at subway level (magnetic detection limit: < a few femto tesla/√Hz). Accessories producing magnetic noise, such as watches, pagers, and coins must be kept away from the sensors. And patients with an implanted pacemaker or a history of gun-shot wounds to the chest are excluded because pacemakers and bullets produce a large amount of magnetic noise. Patients with a prior history of cardiac surgery or stenting for coronary revascularization, who thus have magnetic noise originating from wires, clips, prosthetic valve, or stents, may have a large amount of noise, but most of them are good candidate for MCGs. Applicants need not remove their clothes for MCG measurement because clothes do not affect magnetic fields and because SQUID sensors placed in a dewar do not have to be in direct contact with the skin.

The 64-channel MCG system reduces examination time because the 17.5-cm-square measuring area is large enough to cover the four chambers of the heart (Fig. 5B, a system with a smaller measuring area would require multiple measurements to cover the four chambers) and because this system can simultaneously record the signals in all 64 channels plus the ECG signals from limb leads. One examination including two measurements (anterior-posterior and posterior-anterior projections) is usually completed within 15 minutes.

**MCG analysis**

Measured data is sent to network computers and is analyzed by several methods: grid maps investigating the spatial distribution of the cardiac magnetic fields (Fig. 5C), overlapped waveforms, iso-magnetic field maps (see section: Clinical application of MCGs: Arrhythmias), and integral values (see subsection: At-rest phase abnormalities in angina pectoris). Filtering (standard filter: 0.1–100 Hz), baseline correction, signal-averaging, and time frequency analysis (see section: Clinical application of MCGs: Arrhythmias) are used according to the noise level.

**Clinical Application of MCGs: Ischemic Heart Disease**

MCGs and ECGs differ in the concept of the baseline or zero level. One of the reasons is that direct currents are filtered in ECGs but not in MCGs. The ECG baseline is determined as the PR segment (9) [or the TP segment (31)], while the MCG baseline is determined on the absolute scale measured by the SQUID sensors. In other words, amplitudes in ECGs are based on external voltage standard rather than an absolute scale (31). Thinking that MCGs would be better than ECGs for determining the baseline value, Cohen and colleagues investigated the mechanism of ST changes during cardiac ischemia (13, 16, 32). Subendocardial ischemia causes depression of the ST segment in ECGs, while
transmural ischemia causes ST elevation. But it is not clear when ST elevation occurs, whether an injury current flows only during ST segment or during both baseline and ST segments.

Detection of baseline shift in cardiac ischemia

Cohen and co-workers first investigated the ST elevation after coronary occlusion in dog models in 1975 (Fig. 6) (13) and then investigated ST depression during exercise in patients with stable angina pectoris in 1983 (Fig. 7) (32). They found that cardiac ischemia changed both the baseline and ST segments, shifting them in opposite directions. During acute coronary occlusion the size of the baseline shift was approximately equal to that of the ST segment change (Fig. 6C), but in angina pectoris it was about 70% the size of the ST segment shift (Fig. 7D). MCG baseline shift was not observed in left bundle branch block or early depolarization, a normal variant of ST change (16). He concluded from these results that the ST elevation during acute coronary occlusion is a secondary result of a primary injury current that is interrupted during the S-T interval (13).

At-rest phase abnormalities in angina pectoris

The first indications of ischemic heart diseases are subjective symptoms and ST changes in ECGs. Both of these parameters, however, differ between individuals. About half of the patients with new onset myocardial infarction have no symptom before the onset. ECG abnormalities are observed in 70–90 percent of angina pectoris patients during exercise tests, but in only about half of angina pectoris patients at rest.
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Clinical tools identifying ischemic heart disease in at-risk individuals and in the asymptomatic phases are thus limited.

We studied 30 patients (10 myocardial infarction patients, 10 angina pectoris patients without a prior history of unstable angina or myocardial infarction, and 10 controls) at rest (34) and compared integral values in MCGs (35–38) between the three groups. Comparing the infarction and the control groups, we found that both their ECGs and their MCGs showed differences: the infarction patients had longer QT intervals in their ECGs and had smaller integral values in their MCGs. A new finding is that the integral values in MCGs of the angina pectoris patients were smaller than those in the MCGs of the controls, while ECG parameters (QT interval, QT dispersion, and ST changes) did not differ between the control and the angina pectoris groups (Fig. 8A). This study revealed that the MCGs of unstressed and asymptomatic individuals with angina pectoris have potential abnormalities. Moreover, the MCG integral values decreased more in the myocardial infarction group than in the angina pectoris group and they increased after the intervention (89±8 vs 106±12%, p=0.05, Fig. 8B), thus indicating that integral values in MCGs can reflect myocardial viability and treatment effects.

Other ventricular abnormalities

Clinical MCG studies on ventricular hypertrophy due to pressure overload (39–42) or congenital heart disease (43), myocardial infarction (44–47), pharmacological stress (48), cardiomyopathy (49), Kawasaki disease (50), diabetes mellitus (51), and heart transplantation (25) have been reported.

Table 4. Special Accuracy in Magnetocardiograms: Location of Accessory Pathways in Wolff-Parkinson-White Syndrome

<table>
<thead>
<tr>
<th>Author</th>
<th>Group</th>
<th>No. of cases</th>
<th>No. of sensors</th>
<th>Assignment</th>
<th>Mean accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feniti 1989</td>
<td>Rome, Italy</td>
<td>18</td>
<td>1</td>
<td>X-ray</td>
<td>2.0 cm</td>
</tr>
<tr>
<td>Mäkijärvi 1990</td>
<td>Helsinki, Finland</td>
<td>15</td>
<td>1</td>
<td>X-ray</td>
<td>3.1 cm</td>
</tr>
<tr>
<td>Schirdewan 1991</td>
<td>Berlin I, Germany</td>
<td>13</td>
<td>1</td>
<td>MR</td>
<td>2.9 cm</td>
</tr>
<tr>
<td>Weismüller 1991</td>
<td>Ulm, Germany</td>
<td>9</td>
<td>37</td>
<td>MR</td>
<td>1.8 cm</td>
</tr>
<tr>
<td>Oeff 1993</td>
<td>Berlin II, Germany</td>
<td>25</td>
<td>37</td>
<td>MR</td>
<td>0.5–2.0 cm</td>
</tr>
<tr>
<td>Nenonen 1994</td>
<td>Helsinki, Finland</td>
<td>12</td>
<td>1</td>
<td>MR</td>
<td>2.1 cm</td>
</tr>
<tr>
<td>Nomura 1994</td>
<td>Tokushima, Japan</td>
<td>14</td>
<td>7</td>
<td>MR</td>
<td>“good correlation”</td>
</tr>
<tr>
<td>Moshage 1996</td>
<td>Erlangen, Germany</td>
<td>23</td>
<td>37</td>
<td>MR</td>
<td>&lt;2.0 cm</td>
</tr>
<tr>
<td>Yamada 2000</td>
<td>Tsukuba, Japan</td>
<td>1</td>
<td>64</td>
<td>MR</td>
<td>1 cm</td>
</tr>
</tbody>
</table>

MR: magnetic resonance image (25, 58).
Figure 9. An accessory pathway in Wolff-Parkinson-White syndrome. Electrophysiologic study (A), magnetocardiograms before and after radiofrequency catheter ablation (B). A pre-excitation waveform was observed before catheter ablation (CA, arrow in B1 left), and was not after CA (B1 right). A: atrial potential, ABL: ablation catheter, CS: coronary sinus, K: Kent potential, HBE: His bundle electrogram, HRA: high right atrium, pT: pico tesla, RVA: right ventricular apex, V: ventricular potential (58).
Clinical Application of MCGs: Arrhythmias

Magnetocardiography and electrophysiologic (EP) studies are compared in this section, which first discuss spatial accuracy. This result is applied to an electrophysiological phenomena which can be approximated by a single-dipole model, such as a premature complex, an accessory pathway (52, 53), and a His potential. MCGs are more sensitive to His potentials than other body-surface mappings of cardiac activities (18). To reduce the noise, signal averaging was done in these analyses but signal averaging can obscure clinical indications and may distort physiological signals. The topic in the latter part of this section is beat-to-beat analysis using tangential-components in MCGs (54, 55). This analytical method covers the whole sequence of a heart beat and all mechanisms of arrhythmias, from automaticity to random re-entry.

Special accuracy of MCGs

The accuracy of MCGs is affected by many factors: the clinical (Table 4, Fig. 9) or simulated studies (Fig. 10), the numbers and intervals of sensors (Fig. 10), the distance from the magnetic source to the sensor, body motion, models (56), and the comparing tools. In general, the specific error of MCGs is one-third to half that of ECGs (57). The accuracy of the 64-channel system is 1.4±0.7 mm in simulation (Fig. 10), whereas the size of a successful ablation site in the treatment of a premature ventricular complex or Wolff-Parkinson-White syndrome is about 1 cm (58).

His potential recording in MCGs

The origin of an atrioventricular conduction block cannot be determined without an EP study because the amplitude of a His potential is too small to record in standard ECGs. We investigated the feasibility of measuring His potentials in MCGs (Fig. 11) by using two-minute signal averaging (101±36 beats) (18).

In fourteen out of twenty-two patients (64%), the spike potential was recorded between the atrial and the ventricular components (Fig. 11A right). We have reasons to think that the spike potentials in the MCGs correspond to the His potentials in EP studies. One reason is that the potential was recorded in a few channels at the left anterior chest (Fig. 11A middle). Another is that the His-ventricular intervals in the MCGs were significantly correlated with those in the EP studies (Fig. 11B). A statistically significant relation between His-ventricular intervals measured on the body surface and those determined in an EP study has not been demonstrated in previous reports (59–63).
Adult arrhythmias

Atrial tachyarrhythmias are divided into three clinical diagnoses (atrial tachycardia, atrial flutter, and atrial fibrillation) and have three mechanisms: reentry, automaticity, and triggered activity. Clinical diagnosis is currently based on EP study because ECG-based diagnosis does not show a 1:1 correspondence with the mechanisms (64). To identify mechanisms noninvasively, we made a two-step algorithm using MCGs (65). The first step is to visualize electrical currents through an MCG animation. Reentrant circuit numbers (single or multiple) and size (macro- or micro-reentry) are screened through the animation. The second step is a time frequency analysis that can reveal regular activity under a random pattern.

Mechanisms of atrial tachyarrhythmias: MCG animation

We animated MCGs by editing iso-magnetic field maps (the tangential components of the cardiac magnetic fields) with a minimal interval of 1 ms (17). The cardiac magnetic fields (red areas in Fig. 12A) showed a single peak during atrial tachycardia due to a single automaticity, a large circuit during atrial flutter due to macro-reentry along the tricuspid annulus, and a disorganized pattern during atrial fibrillation.

When atrial fibrillation shifted to atrial flutter (Fig. 12B), the disorganized pattern fused to a single pattern, and then evolved into a circle. During common atrial flutter, atrial activation showed counterclockwise rotation in the animation. As left atrial mapping was not performed in this case, it is not clear if the formation of the macro-reentry in the right atrium was due to focal atrial fibrillation originating from the left atrium. This study showed, however, how the pattern of the cardiac magnetic fields reflects the kind of atrial activation in the right atrium: a circular pattern for atrial flutter, and a disorganized pattern for atrial fibrillation. Further studies are required to use MCGs to differentiate atrial activation in the right atrium from those in the left atrium and to classify patterns of atrial flutter, such as those due to lower loop reentry (66) and those due to a circuit in the posteromedial right atrium (sinus venosa) (67).

Classification of atrial fibrillation: time-frequency analysis

As the next step, we classified disorganized patterns hypothesizing that regular signals are amplified through time-frequency, while irregular signals are reduced. Time-frequency analysis showed multiple peaks at a high field strength (0.2–0.6 pT), which were clearly isolated from the
Figure 12. Atrial tachyarrhythmias in magnetocardiograms. Three types of atrial tachyarrhythmias (A), initiation of paroxysmal atrial flutter (B), atrial tachyarrhythmias with (C1) and without firing foci (C2), and chronic phase of atrial fibrillation (D). Magnatocardiogram (MCG) detected a high-frequency component (6 Hz) in partial atrial standstill (arrow in D1 middle), but did not in total atrial standstill (D2). In partial atrial standstill, the 6-Hz component was not evident in electrocardiogram (ECG, arrow in D1).
Figure 13. Fetus magnetocardiograms: normal development. The duration of the QRS complex increased linearly with gestation age (A), while the PQ interval was independent of fetal age (B). (Reproduced from Stinstra J, Golbach E, van Leeuwen P, et al. Multicentre study of fetal cardiac time intervals using magnetocardiography. Br J Obstet Gynaecol 109: 1235-1243, 2002 (20), Copyright 2004, with permission from the Royal College of Obstetricians and Gynecologists).

Table 5. Initiation and Termination Patterns of Fetal Supraventricular Tachycardias

<table>
<thead>
<tr>
<th>Subject</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initiation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initiations observed</td>
<td>3</td>
<td>31</td>
<td>1</td>
<td>5</td>
<td>30</td>
<td>4</td>
<td>17</td>
<td>5</td>
</tr>
<tr>
<td>Spontaneous PAC</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Block of AC in WPW</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus acceleration</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reentrant PAC</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reentrant PAC with transition from fast to slow AC</td>
<td>X</td>
<td></td>
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<td></td>
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<tr>
<td>Termination</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terminations observed</td>
<td>3</td>
<td>31</td>
<td>2</td>
<td>5</td>
<td>30</td>
<td>4</td>
<td>17</td>
<td>5</td>
</tr>
<tr>
<td>Block in AV node</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<td>Block after transition from slow to fast AC</td>
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<td>Block after dual AV pathway oscillation</td>
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<td>Transient bradycardia</td>
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Figure 15. Noninvasive diagnosis of arrhythmic foci: Wolff-Parkinson-White syndrome. A location of accessory pathway calculated by magnetocardiograms is superimposed on two-dimensional (A) and three-dimensional magnetic resonance imaging (red dot, B). This patient is the same patient presented as in Fig. 9. LV: left ventricle, RV: right ventricle, TV: tricuspid annulus.
other peak, in the MCG of a patient with focal automatic atrial tachycardia (arrow in Fig. 12C left), and showed a broad distribution at a range of 6–10 Hz at a low field strength (0.1 pico tesla) in the MCG of a patient with multiple reentrant wavelets (arrow in Fig. 12C right). These two patterns could not be differentiated as a quantitative parameter by ECG or MCG animation. Time-frequency analysis also shows patterns that differ between partial atrial standstill (19) and total atrial standstill (Fig. 12D). This study indicates that time-frequency analysis in MCGs might be used to differentiate atrial fibrillation with multiple wavelets and focal atrial fibrillation with triggered activity and may

Figure 16. Noninvasive diagnosis of arrhythmic foci: premature ventricular complexes in a patient with a prior history of myocardial infarction and cardiopulmonary resuscitation due to ventricular fibrillation. Premature ventricular complexes (green dots in A and red dot in B) originate from the low septum, close to the area of the myocardial infarction. Ao: aorta, LA: left atrium, LV: left ventricle, PA: pulmonary artery, RV: right ventricle.
also be useful for evaluating degrees of electrical remodeling in atrial fibrillation. MCGs may thus provide key information for determining which strategies to use in the treatment of atrial fibrillation (e.g., pharmacological or nonpharmacological therapy, rhythm control or rate control).

**Clinical Application of MCGs: Fetal Diagnosis**

One of the promising areas for MCGs is fetal diagnosis; it is where the advantages become more prominent (Table 2). Fetal MCGs are unaffected by vernix caseosa and by amniotic fluid and are reliable throughout the second and third trimesters of pregnancy, whereas fetal ECGs measured on the maternal abdomen are reliable only before the 27th week of gestation (68).

Atrial activation, ventricular depolarization, and ventricular repolarization, which respectively correspond to the P wave, QRS complex, and T wave in ECGs, can be observed in the MCG of a fetus after about 15–20 weeks of gestation. Normal development of the heart (20, 69) (Fig. 13), congenital long QT syndrome (70–72) (Fig. 14A), fetal supraventricular tachycardia (Fig. 14B, Table 5) (73), congenital complete atrioventricular block (74, 75), and fetal cardiac hypertrophy (76) have all been diagnosed using MCGs. Although it is controversial whether prophylactic therapy based on MCGs (70) improves prognosis, fetal MCG are useful for detecting high-risk pregnancies and congenital arrhythmias.

**Discussions**

For the forty years that has passed since the MCG was first reported by Baule and McFee (10) Japan has been one of the leading countries in both basic and clinical MCG studies (77, 78). The recent development of a multichannel system there has shifted the main MCG topics from the technical aspects to clinical uses.

The most important advantage of MCGs over ECGs is that they are more sensitive to small signals. Although they may not be necessary in the diagnosis and treatment of individuals who show obviously abnormal ECGs, MCGs can provide unique and additional information when ECGs are not practical or not sensitive enough. Examples of phenomena profitably examined by using MCGs are the baseline shift in ischemic heart disease, at-rest abnormalities in angiography, His potentials, atrial tachyarrhythmias, and fetal myocardial activity. The studies reviewed here suggest that MCGs could be used to detect ischemic heart disease in people who have no symptoms and show no ECG abnormalities and to noninvasively obtain information useful for determining treatment strategies (existence of cardiac ischemia or viability in ischemic heart disease, mechanisms of atrial tachyarrhythmias, degree of electrical remodeling in atrial fibrillation, etc).

One factor limiting the clinical use of MCGs is that their utility has not yet been established. There are very few animal studies and few clinical studies in a large population or from the prospective approach. In addition to ECGs, there is little clinical data with which MCG can be compared, especially data in basic medicine. The potential benefits of MCGs and the most effective way to use them in clinical medicine have therefore not been understood. A second limiting factor is that MCG systems and parameters have not been standardized (79). The cost-effectiveness of MCGs must also be improved.

The search for future uses of MCGs is ongoing. One is in three-dimensional diagnosis combining with electrophysiological, anatomical (80) (Figs. 15, 16), and metabolic information (81). These kinds of information are currently obtained in different images, but a combined image helps provide a comprehensive understanding of a given pathology. Another approach is a therapeutic one. EP study using nonmagnetic catheters under MCG monitoring (82, 83) might reduce radiation time and let us use less invasive ablation procedures to treat arrhythmias.

In conclusion, MCGs, body surface mapping of the cardiac magnetic fields measured using SQUID sensors, are used in clinical diagnosis when ECGs are not practical or not diagnostic, such as in fetal diagnosis and when it is necessary to evaluate baseline shifts or His potentials. Establishing their utility will require further studies in both basic and clinical approaches.

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