Magnetic Measurement of S-T and T-Q Segment Shifts in Humans

Part I: Early Repolarization and Left Bundle Branch Block

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SUMMARY. The direct-current magnetocardiogram not only shows the features usually seen on the electrocardiogram, but also shows the T-Q (baseline) shift due to cardiac injury current. The first direct-current magnetocardiogram measurements of the human heart are presented here. The hypothesis tested is that there is no injury current associated with the S-T shift seen in two electrocardiogram abnormalities: early repolarization, and left bundle branch block. The data from three typical early repolarization subjects and one typical left bundle branch block patient are presented. It is found, in each case, that although there is appreciable S-T shift, there is essentially no baseline shift on the direct-current magnetocardiogram. The absence of baseline shift proves that the S-T shifts in these cases are not "apparent" shifts, caused by a dc injury current which is interrupted during the S-T interval; instead, these are "true" S-T shifts caused by a current flowing only during systole, presumably due to an altered repolarization of the ventricles. It is also found that the direct-current magnetocardiogram does not have routine clinical application because of a practical problem. This is the presence of false baseline shifts due to noncardiac currents, mostly in the gastrointestinal tract, which could be suppressed in only about one-third of the subjects. However, the direct-current magnetocardiogram may be useful as a research tool, for clarifying the cause of the S-T shift in selected subjects. (Circ Res 53: 264-273, 1983)

THE S-T segment shift of the electrocardiogram (ECG) is an important indicator of cardiac abnormality. It can be produced by two mechanisms. In the first, it is produced by a current that flows only during the S-T interval, caused by a difference in action potentials between regions of the heart. We call this a "true" S-T shift (also called a primary, or a systolic S-T shift). In the second mechanism, the S-T shift is produced by a dc injury current which flows during the entire cardiac cycle except that it is interrupted during the S-T interval; we call this an "apparent" S-T shift (also called a secondary or a diastolic shift). The injury current is caused by an abnormally low membrane resting potential in one region of the heart. It is important to differentiate between the two types of S-T shifts because the apparent S-T shift indicates acute myocardial injury, usually of ischemic origin. This S-T shift would be recognized because it would be accompanied by a shift of the T-Q segment (or baseline), which reflects the injury current; but the T-Q shift cannot be measured on the ECG, hence the ECG cannot distinguish between the two types. However, the T-Q shift can be measured with the direct-current magnetocardiogram (dcMCG), which does therefore distinguish between the two. The dcMCG was previously used to show the type of S-T shift produced by coronary occlusion in the intact dog (Cohen and Kaufman, 1975). It is used here for the first time to show the type of S-T shift present in humans.

In this study, S-T shifts in two types of abnormalities are investigated. The first is the normal variant S-T shift known as early repolarization (ER), and the second is the S-T shift in left bundle branch block (LBBB). Both of these are believed to be true S-T shifts, but up to now there has been no experimental proof of this in either case. Both are believed to be caused by regional differences in action potential waveforms, where the regions in ER are endocardium vs. epicardium (Mirvis, 1982), and in LBBB are right vs. left ventricle (Lamb, 1965). The main objective of our study is to test the hypothesis, using the dcMCG, that the S-T segment shift in these two cases is a true shift.

That the injury current can be measured on the dcMCG, but not on the ECG, is explained in the following way. The dcMCG is a measurement of the magnetic field over the torso produced by the same ionic currents which produce the ECG; the dcMCG therefore has the same features of the ECG, such as P wave, QRS complex, T wave, and U wave (Cohen et al., 1976). However, the dcMCG has an additional feature, a T-Q (baseline) level which shows the dc currents from the heart. If one attempted to record, on the ECG, the dc potentials generated by the heart, they would be greatly overshadowed by dc potentials generated in the skin (Edelberg, 1972). For this reason, dc is deliberately filtered out of the clinical ECG, hence its T-Q level is arbitrary. However, because of the skin's high electrical resistance, skin
electrical sources generate currents which are small enough so that their magnetic fields are negligible, compared with the cardiac fields. Therefore no dc filtering of the magnetic signals is necessary, and the dcMCG is capable of measuring the dc current generated by the heart.

Although the dcMCG is not subject to large dc interference by the skin, it is nevertheless subject to some degree of dc interference by organs near the heart, such as those in the gastrointestinal (GI) tract; these generate dc currents which appear as false T-Q shifts. This problem is troublesome enough to raise the question of the clinical practicality of the dcMCG. The second objective of this work is therefore to assess methods of coping with this problem.

**Methods**

**The Principle of S-T and T-Q Shifts**

There is a principle, first applied in animals (Cohen and Kaufman, 1975), which is used here in the interpretation of the human dcMCG. We consider Figure 1, in which the relationship between the S-T and T-Q shifts of the dcMCG is shown. Figure 1A shows the relationship in a true S-T shift. During the S-T interval, a shortened and reduced action potential in the hatched region creates a potential gradient, which produces a current flow and magnetic field; the current flows from the normally depolarized region (unhatched) to the incompletely depolarized region (hatched). Similarly, Figure 1B shows the relationship in an apparent S-T shift. During the T-Q interval, a decrease of the level of the membrane resting potential in the hatched region creates a potential gradient which also produces a current flow and a magnetic field; the current flows from the partially depolarized region (hatched) to the normally polarized region (unhatched). However, during the S-T interval, all regions in this idealization are depolarized similarly; hence, there is no potential gradient, no current flow, and no magnetic field. As a result, the S-T shift (with respect to the T-Q level) is equal and opposite to the T-Q shift (with respect to the magnetic zero level).

The principle can be stated as follows: Whatever the location around the torso of the magnetic detector and its "angle-of-view," if an S-T shift is seen, then no T-Q shift will be seen if it is a true shift, and an equal and opposite T-Q shift will be seen if it is an apparent shift. Further, a smaller and opposite T-Q shift will be seen if it is a mixture of the two. The simplicity here is that the S-T/T-Q relationship is independent of detector location and angle-of-view (or "lead" in ECG terminology). It is only necessary to choose a location and angle-of-view so that the S-T shift can be seen, if it exists at all. This principle allows a simple and rapid determination of the fraction of the S-T shift which is true or apparent. However, there is the problem of false T-Q shifts.

**Modulating the S-T Shift; the Use of Maps**

Organs near the heart, especially those in the GI tract, are active generators of dc currents. These currents, via their steady magnetic field, shift the entire dcMCG trace up or down. They are not immediately distinguished from cardiac injury currents, and they appear as false T-Q shifts. This is the main problem in these measurements. However, there is one procedure in which this problem can be circumvented; this is when the S-T shift is modulated. If the S-T shift is turned on and off, then any associated injury current will also be turned on and off, and the T-Q shift will move up and down accordingly; this separates it from the false T-Q level, which is not modulated. In this procedure, it is necessary to record the dcMCG at only one location around the torso, where this modulation can be seen. This was the case with coronary occlusion in dogs (Cohen and Kaufman, 1975) and with exercise-induced angina (part II of this work).

When no modulation procedure is possible, the problem of false T-Q shifts must be handled in a more elaborate way. Whether or not a T-Q shift originates in the heart or in a nearby organ can, to some extent, be determined by the spatial distribution of the field over the torso. That is, a mapping of the T-Q shift over the anterior of the torso can determine the location of the source. A mapping is necessary because, from the dcMCG recorded at only one location ("one lead"), it would not be known whether the T-Q level was due only to the heart, or to a nearby organ, or to a combination of both. For the human S-T shift in ER and LBBB investigated here, no modulation is possible, in that they are stable shifts in which we cannot intervene. We are therefore forced to perform the mapping procedure. (In part II no mapping is necessary.)
In a map, the extent of discrimination between cardiac and extraneous sources depends on the angle of view (spatial sharpness) of the dcMCG, which in turn depends on the configuration of the dcMCG coil which detects the field; this is similar to the ECG, where its angle of view depends on the spacing between electrodes. For maximum discrimination, the MCG coil should have a narrow angle of view. For ease of discrimination, it should also have a direction of greatest sensitivity which is in front of the coil (on its axis), not off to its sides. Both of these characteristics are lacking in the coil configuration conventionally used for MCG measurements (Cohen and Hosaka, 1976), which is a simple loop; it is therefore not suitable for mapping here. However, these characteristics are present in a configuration called the 2-D system (Cohen et al., 1980), which we therefore use here.

The 2-D system is illustrated in Figure 2A. Each of the two coils is connected to a sensitive magnetic transducer, routinely used in biomagnetic measurements, called a SQUID (Superconducting Quantum Interference Device), which is followed by an amplifier. The output of the two channels are usually combined to form the x and y components of an arrow, which mimics the x-y projection of the underlying currents, as in Figure 2, B and C. (The analogy in the ECG would be a pair of bipolar electrodes about 1 cm apart feeding a differential amplifier, crossed at 90° with another identical pair, where the two amplifier outputs are combined to form a frontal-plane vector.) To produce a map, the dcMCG signals are first recorded at grid points over the torso; then from the raw traces, the T-Q levels are determined for each point. These are next combined to form an arrow at each point, and plotted to yield a map of T-Q arrows. This map, then, visually indicates the location of the dc sources. To use our S-T/T-Q principle, we also obtain an S-T map from the same tracings; in this way, the amount of T-Q shift which is equal and opposite to the S-T shift over the heart region can be seen.

The characteristics of the dcMCG arrow map, due to a dipole in the heart region, are illustrated in Figure 3, along with the ECG map for comparison. These maps are theoretically calculated, assuming the torso to be a semi-infinite volume conductor (Cohen and Hosaka, 1976). When the dipole is oriented along the x-axis, parallel to the anterior wall, as in Figure 3B (left), the largest arrow is seen to mimic the source dipole in position and direction; also, there is a circular pattern on each side of this arrow. Thus, the location and orientation of a parallel source is readily determined from the arrow map. However, when the dipole is oriented along the z-axis, perpendicular to the anterior wall (Fig. 3C), there is no significant external magnetic field (in contrast to the ECG map). This is a well-known property of the MCG (Baule and McFee, 1965), and is important here in the interpretation of the dcMCG.

For example, a subject may show an S-T shift on ECG and is important here in the interpretation of the dcMCG. If, instead of a semi-infinite volume conductor, a more realistic torso model had been used, these properties would remain valid (Hosaka et al., 1976).

The magnetic field (actually the gradient), measured by the 2-D detector, is expressed in picotesla per centimeter (pT/cm). For example, a current dipole of moment 1 ma-

\[ \text{cm, at a distance of } 10 \text{ cm, will produce a magnetic gradient of } 10.5 \text{ pT/cm. For comparison with the magnetic unit of gauss, which had been used until recently, } 1 \text{ pT} = 10^{-4} \text{ gauss.} \]

The Magnetic Measurements

The dcMCG measurements took place inside the MIT magnetically shielded room (Cohen and Kaufman, 1975). This was done for two reasons: first, to eliminate time-varying magnetic background signals due to trucks, rotating machinery, etc., and second, to greatly reduce the earth's magnetic field, because the human body becomes weakly magnetic in the presence of an externally applied magnetic field (Wikswo et al., 1973). To eliminate magnetic fields due to zippers, nails in shoes, etc., the subjects wore special nonmagnetic clothes. The two magnetic sig-
Measurements were usually made at 20 points of a rectangular grid marked over the chest and abdomen. For each measurement the subject first stood out of range of the detector, then brought the appropriate grid point against the cross-hairs of the detector for a few seconds, then stepped out of range again. The T-Q shift at each location was measured as the difference between the T-Q level when he was in range, and the baseline level when he was out of range. The S-T shift was measured as the difference between the S-T and T-Q level at about 40 msec after the J-point on lead aVF. The S-T and T-Q shifts for each of the 20 points were digitized from the raw traces on the strip charts, and stored in a computer. Arrow maps corresponding to S-T and T-Q shifts were then plotted by the computer. The precision of this digitizing procedure was ±0.2 pT/cm, or <10% of the larger S-T shifts.

To ensure that the separation of extraneous fields from cardiac fields be effective from the mapping, it was important to understand the spatial and temporal characteristics of the extraneous fields. It was also important that the extraneous fields not be too large so that they would not spill over and dominate the heart region of the map. The extraneous field characteristics therefore were studied at some length, and a search was made for interventions to reduce the fields. These are described in the Appendix; however, we here outline the measures finally chosen as our standard procedure in coping with extraneous fields.

(1) The ferromagnetic contaminants in the GI tract, which can produce fields indistinguishable from that of currents, were demagnetized by using a hand-held magnetic eraser. (2) An overnight fast was performed before the measurements took place (in the morning). (3) To reduce time-varying GI fields, a signal-averaging procedure was used in which eight T-Q maps recorded on four different days were averaged. (4) For each map to be acceptable, two criteria were to be fulfilled: the TQ level had to be <8 pT/cm in row N, and <5 pT/cm in rows F to L.

FIGURE 3. Characteristics of the dcMCG arrow map compared with the ECG isopotential map. The maps are theoretical, due to a dipole source in a semi-infinite volume conductor. Part A: the grid used, consisting of 20 points 5 cm apart marked directly on the torso; location L25 is always at the xiphoid. Part B, left: the dcMCG arrow map due to a current dipole (open arrow) located behind point J27 and oriented along the x axis. Part B, right: the ECG map due to the same dipole; the thick line is zero potential. Part C, left: the dcMCG arrow map due to a dipole oriented perpendicular to the surface; the external magnetic field in this case is zero, therefore each arrow is null. Part C, right: the ECG map due to the same dipole showing, in contrast, a potential maximum over the source.

The Study Group

The data from three ER subjects and one LBBB patient are presented here. These are persons selected from two larger groups because only in these persons were the extraneous fields acceptably low. Although three and one would usually be too small a number from which to draw conclusions, these cases all are typical as far as their electrocardiographic and clinical data are concerned; hence their dcMCG data can be presented as typical, with reasonable confidence.

All subjects were paid volunteers, and informed consent procedures were followed. The three ER subjects were selected by screening from a group of 124 male student volunteers, age 19–24. This type of group was chosen because the largest of the S-T shifts in ER can be observed most frequently in young adults (Lamb, 1965; Spach et al., 1979; Kambara and Phillips, 1976). The screening consisted of selecting those who had the largest S-T shifts, as measured at the 20 grid points with a single-channel acMCG (bandwidth: 0.05–100 Hz). This was used for screening instead of the dcMCG because, here, no demagnetizing or fasting was required, hence, the screening was rapid, and the S-T shifts are almost the same. Seven subjects showed a significant S-T shift (>3 pT/CM). Of these, three were acceptable after being tested for extraneous fields, using the criteria described in the Appendix. These subjects all had normal physical examination, medical history, urine test, and chest x-rays. Their 12-lead ECGs all were similar and typical of ER, with S-T elevation.
of about 1.5 mm in lead II and about 2.5 mm in V4.

The LBBB patient was selected by first screening a large number of LBBB patient records for S-T elevations of >2 mm in the ECG limb leads. Three patients met this criteria; their dcMCG then was measured, and only this one patient, a 61-year-old male, met the criteria for extraneous fields. His record showed a history of hypertension and chronic left bundle branch block for several years. He first experienced angina pectoris 7 years previously, and, 6 weeks before our measurements, was admitted to a hospital with chest pain. An acute myocardial infarction was diagnosed on the basis of S-T elevation in the right precordial leads, in addition to the typical S-T change due to LBBB (Madias et al., 1975), and marked rise of myocardial fraction of creatine kinase. He was discharged in 2 weeks and was placed on quinidine for supraventricular and ventricular arrhythmias, hydralazine, coumadin for a ventricular aneurysm, digoxin, and nitroglycerin. During the dcMCG measurements, his ECG was similar to those recorded before infarct. A bicycle ergometry test in conjunction with thallium 201 myocardial perfusion scintigraphy did not reveal redistribution of radiotracer at rest. Thus, there was absence of clinically significant myocardial ischemia during exertion, and ischemia at rest could be excluded.

Results

Typical dcMCG data of one of the ER subjects are presented in Figure 4. We first examine the raw traces in part A. It is seen that the S-T shifts are largest over the heart (J25, J27, L25, L27). Generally, they show the same polarity as the T waves; further, their slope is concave-upward and extends toward the peak of the T wave, which is typical of ER in the ECG (Wasserburger et al., 1961). These features are common to the three ER subjects. It is then seen that there are almost no T-Q shifts associated with the largest S-T shifts, at L27 for example. However, some locations showing no S-T shifts do show a T-Q shift, the largest of which can be observed over the abdomen, at N29 of the lower panel for example; this indicates an extraneous field of GI origin.

However, the spatial characteristics of the S-T and T-Q shifts are best seen in the corresponding arrow maps in Figure 4B. In the S-T map, the largest arrows are over the heart as they should be because, in analogy with Figure 3, the source is in the heart. However, the arrow pattern for this subject is some-
what more complicated than that of Figure 3, indicating that the source configuration is more complex than that of a dipole. Whatever its exact figuration, the general source direction is seen to downward and leftward. The T-Q map is quite different. The largest arrows are not over the heart, but are instead located over the subject's lower left abdomen, confirming that the sources are in that region; the arrows are small enough to allow this map to meet the acceptance criteria. Over the heart, the T-Q arrows are small, compared with S-T arrows; therefore, in this case, there is no significant T-Q shift of cardiac origin. In contrast to the small extraneous field of Figure 4B, recorded after an overnight fast, the large field in Figure 4C, due to eating, serves to illustrate the problem of extraneous fields. Whereas the S-T map is similar to that of Figure 4B, the T-Q map shows a much larger amplitude. The counter-clockwise swirling pattern, with the largest arrows mostly over the stomach, is certainly due to the electrical activity in the GI tract. When T-Q maps show this large an amplitude, they do not meet the extraneous-field criteria and are rejected.

To illustrate the total data of all three ER subjects, the S-T and T-Q maps recorded on four different days were averaged for each subject and are shown in Figure 5. Their standard ECGs show S-T shifts of 1.0–1.5 mm in lead II, and 2.0–3.0 mm in V2 and V6.

**Figure 5. Averaged dCMCG maps and standard ECCs of the three ER subjects A, B, and C.** The arrow maps of each subject are due to the standard procedure, where each map is the average of eight maps recorded on four different days after an overnight fast. Subject A is the same as in Figure 4. The heart rate was about 55 beats/min for all three subjects. The ECG calibration bars are 1 mV and 1 second, while the dCMCG bar is 10 pT/cm.
V3. The shape of the S-T segment is upwardly concave, followed by an upright T wave in the precordial leads. The S-T arrow patterns of subjects B and C are alike, and differ somewhat from that of subject A. They resemble the pattern in Figure 3; hence, their source can be approximated by a dipole oriented leftward, but also somewhat downward. The S-T pattern in each individual map used in the averaging was essentially the same as in the averaged maps shown here; thus, comparison of the unaveraged S-T map of the subject in Figure 4 with his averaged map (Fig. 5A) reveals little difference. In contrast, the averaged T-Q map of this subject in Figure 5A compared with his unaveraged map in Figure 4B shows a decrease in overall T-Q amplitude due to averaging. This is because, as previously noted, the T-Q signals varied in time, so that the averaging process was effective. Again, as in Figure 4B, the T-Q shifts in each case are much smaller than the S-T shifts, and show no relation to them. Thus, the peak T-Q shifts are about 1 pT/cm and are located over the abdomen, whereas the S-T shifts are 3-5 pT/cm over the heart. The ratio of S-T to T-Q is about 10:1 over the heart.

The data of the LBBB subject are shown in Figure 6. The ECG is characteristic of LBBB, with a QRS duration of 140 msec, a QRS axis of 0°, slurred R waves, depressed S-T segments, and inverted T waves in leads I and II (Lamb, 1965). The raw dcMCG traces also show a 140-msec QRS, slurred R waves, and inverted T waves. The averaged S-T arrow map (part D) is seen to be similar to the single-scan arrow map (part C), indicative of the stability of this S-T shift, as in ER. However, the S-T arrow pattern is different from that in ER. Here, the largest arrows are located in the left precordial.

![Diagram of ECG and dcMCG maps](http://circres.ahajournals.org/lookup/suppl/doi:10.1161/01.RES.53.2.270/-/DC1)

**Figure 6.** The dcMCG maps and standard ECG of the LBBB patient. Part A: the standard ECG; the calibration bar indicates 1 mV for leads I to aVF, and 2 mV for the leads V1 to V6 Part B: the raw traces of one scan after an overnight fast. The largest peaks are cut off. The calibration bars indicate 20 pT/cm and 1 second. Part C: the dcMCG levels of part B displayed as arrow maps. Part D: maps due to the standardized procedure. The calibration bar, for both Parts C and D, indicates 10 pT/cm.
region, oriented toward the subject’s right; the amplitude (10 pT/cm) is much larger. Using Figure 3 as a guide, the equivalent dipole source is located in the left ventricle and oriented parallel to the chest and to the subject’s right. The T-Q map (part D), shows the expected reduction in amplitude due to averaging, in comparison to that in part C. The T-Q shifts are not related to the S-T shifts, and are much smaller, and once again of abdominal origin. In this case, the ratio of S-T to Q is about 10:2 over the heart.

Discussion

Our main objective was to test the hypothesis that the S-T shifts in ER and LBBB are true S-T shifts, caused by current flowing only during the S-T interval. In the subjects we examined, all of whom were typical, our results prove that the S-T shifts are indeed true S-T shifts, and are exclusively of this type. The T-Q shifts were much smaller than the S-T shifts, and were of noncardiac origin.

The absence of significant T-Q shifts on the dcMCG thus confirms the view that the S-T shift in ER does not result from regional differences of membrane resting potential, but from an altered repolarization process (Chelton and Burchell, 1955). In this view, the terminal period of the action potential (phase 3) begins earlier in some cells, for reasons not clearly understood; the S-T shift then forms the initial part of the T wave, and therefore is of the same polarity. To explain its polarity, it is then only necessary to explain the T wave polarity, presently considered to be due to a more rapid terminal repolarization in the epicardium of the left ventricle than in the endocardium, resulting in a more negative intracellular potential in the epicardium; this yields a larger positive charge density in the membrane surfaces of the epicardium and, hence, positive potential in the lower left torso, seen for example in the T waves and S-T shifts of Figure 5. These potential differences across the ventricular wall have been seen in animals, during the S-T and T period, by Spach et al. (1975, 1979).

This view is supported not only by the absence of the T-Q shift, but also by the orientation of the equivalent source deduced from the dcMCG. For all three subjects, it is generally leftward and downward (more downward for subject A), forcing current around oppositely in the volume conductor; this agrees with the larger positive charge density on the epicardium of the left ventricle, which would generate the same volume currents. Because the dcMCG does not see the z-component of the source, we deal only with x-y sources and currents, and do not here consider the anterior wall potentials (ECG precordial leads) and the projections which produce these. Finally, we note that—although this phenomenon has been called “early repolarization” (Chelton and Burchell, 1955)—the use of this term to designate the normal S-T variant was recently challenged by Mirvis (1982); he also found that the overlap between repolarization and depolarization was not the major determinant of S-T shift.

The absence of significant T-Q shift in LBBB confirms the view here, as well, that the S-T shift does not result from differences in resting potential, but, again, is caused by an altered repolarization, in particular, by a delayed repolarization of the left ventricle (Lamb, 1965). In this view, ventricular depolarization begins in the right endocardial septal area, then progresses across the free wall of the right ventricle, then moves through the septum and into the left ventricle; this is in contrast to the normal sequence, where excitation begins in the left septal area and first moves into the left ventricle. This altered sequence is then repeated in the repolarization process, which generally follows the same pathways as depolarization. Thus, during the S-T period, repolarization is more advanced in the right than in the left ventricle, so that the right intracellular potentials are more negative than those on the left. There are therefore larger positive charge densities on the right membrane surfaces than on the left, which produce larger positive potentials in the right torso, hence S-T depression in ECG lead I (as in Fig. 6). This view is again supported not only by the absence of T-Q shift on the dcMCG, but also by the orientation of the equivalent S-T dipole source, determined from Figure 6 to be oriented to the right. This orientation indicates that the internal source currents flow from left to right, forcing a flow in the volume conductor from right to left, as would be produced by the larger positive charge densities on the right membrane surface in the above view. Again, as in ER, we do not here deal with the anterior potential and precordial ECG leads.

As for our second objective, to assess the problem of false T-Q shifts, we found no reliable way of suppressing extraneous dc sources in most subjects. Only in about one-third of the subjects were the false T-Q shifts small enough to allow ready measurement. Because of this, the dcMCG cannot at this time be used as a clinical diagnostic tool. Perhaps, in the future, a method can be found to suppress the sources; if so, the dcMCG could then be clinically promising. However, our results here indicate that the dcMCG can now be useful as a research tool to clarify the cause of S-T shift in special subjects. These are where the extraneous fields are small, where the S-T sources are oriented in a direction which has a significant x-y component, and where the S-T shift is stable enough to allow dcMCG measurement.

The following are some of the special cases which we believe can be investigated for research purposes with the dcMCG. (1) The S-T depression seen in patients with coronary artery disease undergoing a stress test; here, both true and apparent S-T shifts should be found because of the effects of ischemia both on action potential shape and on membrane resting potential (Samson and Scher, 1960; Kléber

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et al., 1978). (2) The S-T elevation seen in patients with ventricular aneurysm developed after extensive myocardial infarction (Laham, 1954); the dcMCG can determine whether this elevation is caused by an injury current due to mechanical stress at the border of the aneurysm, or by an altered repolarization due to activation delay around the aneurysm. (3) The persistent S-T shift seen in patients with left ventricular hypertrophy; the S-T shifts here are believed to be true shifts, due to a delayed repolarization resulting from the increased period of time required for left ventricular excitation (Lamb, 1965). (4) The S-T depression seen in lead II in patients receiving digitalis; this is also believed to be a true shift resulting from a shortening of the action potential (Rosen et al., 1973). In cases (3) and (4) the dcMCG would reveal any injury current caused by concomitant medication. In Part II of this work, we investigate the first of these special cases. (Part II is published in this issue of Circulation Research as a Brief Communication.)

Appendix

Dealing with Extraneous Fields

Nature of the Fields; Reduction by Interventions

Extraneous dc magnetic fields are produced by both ferromagnetic contaminants in various organs (lungs, the GI tract, skin) and by ionic currents generated by muscle and nerve (Cohen et al., 1980). The fields produced by the contaminants are readily eliminated in nearly all cases by moving a hand-held 60-Hz magnetic eraser (demagnetizer) over the torso, leaving only the fields due to currents. The nature of these fields was studied by mapping the dcMCG over the torsos of six male subjects at the grid points previously described: the physical examinations, ECGs, and MCGs of these subjects were normal.

The major extraneous field was usually located over the abdomen. Its amplitude rose to levels of >75 pT/cm after eating, or after drinking cold water; for comparison, we recall that the maximum S-T shifts are about 5 pT/cm in the abdomen. Its amplitude rose to levels of >75 pT/cm after eating, or after drinking cold water; for comparison, we recall that the maximum S-T shifts are about 5 pT/cm in the abdomen. When not this low, the temporal behavior of the signals recorded over the fasting stomach differed with subjects and circumstances. The signals could at times be quite steady, or could also exhibit oscillations with a period of 20 seconds, apparently due to the rhythmic contractions of the stomach (Linkens, 1979). In almost all interventions and manipulations in which we attempted to reduce this abdominal field, we succeeded only in stimulating it further. These interventions included pumping the stomach to reduce its electrolyte content, ingesting aspirin or ethanol to reduce the gastric mucosal potential difference and acid secretions (Tarnawsky et al., 1978; Biggerstaff and Leitch, 1977), and drinking warm water to reduce peristalsis. No attempt was made to use drugs which could be clinically unacceptable. The only intervention we found which reduced the dc magnetic field from the abdomen was an overnight fast; if the dcMCG measurements were made in the early morning, say before 9 a.m., then the abdominal field was usually reduced.

Circulation Research/Vol. 53, No. 2, August 1983

There were also minor extraneous fields seen over the upper anterior thorax (rows F, H, and N) of amplitude 1 or 2 pT/cm. When observed with a grid finer than 2.5 cm, these fields extended only a few centimeters over the skin; hence, their source was within or near the upper thoracic wall. Because these fields were not altered by sweeping a strong bar magnet over the skin, they were due to ionic currents instead of ferromagnetic contaminants which escaped demagnetization. These thoracic fields did not change significantly when a heatpad was applied to the skin for 15 minutes over the pectoral area, or when shortwave diathermy or therapeutic ultrasound was applied. No significant changes resulted from mechanical massage or from cutaneous application of an analgesic spray (Americaine). However, cooling the skin with an ice-pack for 30 minutes increased the field to 10-15 pT/cm. Also, strong isometric contractions of the pectoral muscle produced a transient dc field, vanishing after about 1 minute: similar magnetic phenomena were reported for the muscles of the upper arm (Cohen and Givler, 1972). The skeletal muscles can therefore be a source of these thoracic fields, but there appeared to be other sources within or near the thoracic wall. All in all, no intervention to reduce these fields was found.

Reduction by Averaging; Rejection Criteria

Because any unwanted quantity can be reduced by time-averaging if it varies with time (the averaging would not affect the stable heart signal), an investigation was made of the temporal characteristics of the extraneous fields. The root-mean-square (RMS) T-Q value in $\Delta B_{z}/\Delta x$ and $\Delta B_{z}/\Delta y$ were determined at each grid point, due to the averaging of six normal subjects ($n = 24$), after four overnight fasts. These were both found to be 2 pT/cm for points on the top three rows (F, H, and J), 3 pT/cm for row L, 8 pT/cm for row N, and 14 pT/cm for an additional row P which passes over the stomach (P25-P27). Further, in any particular subject, averaging different arrow maps found to reduce the overall amplitude of the T-Q magnetic field. In applying this procedure, we noted that arrow maps of the T-Q shift, recorded on different mornings of the same subject, allowed very differences between them than arrow maps recorded at 30-minute intervals. The following averaging procedure thus was developed as part of the standard procedure in this study.

Eight T-Q shift arrow maps of each subject were averaged, as well as the corresponding S-T shift arrow maps. These eight recordings were made on four different mornings after an overnight fasting period, with two recordings made at 30-minute intervals on each morning. It was necessary that each separate T-Q map satisfy two criteria to be selected for averaging: First, that the T-Q shift on both channels be <5 pT/cm for any points on the row N; second, that the T-Q shift on both channels be <3 pT/cm for any points on the upper four rows. If a recording did not meet these criteria, it was rejected, and the recording was repeated 30 minutes later, or on another morning. If the maps of a subject did not meet these criteria on two of three mornings, the subject was rejected from the study.

The first criterion dealt with steady fields originating in the GI tract; the level of 8 pT/cm was chosen by considering T-Q maps where the GI interference was large (usually at N), and seeing the arrow spillover from the abdomen to the heart region (as in Figure 4C); the choice of this level assured that the GI field level over the heart was negligible. The second criterion dealt with the very
localized steady fields originating in the upper thorax. The value of 5 pT/cm was chosen by considering spill-over from various local portions of the thorax to the heart region. Because these regions could be nearer to the heart than the abdomen, the rejection value is correspondingly decreased.

Part II of this work appears in this issue as a Brief Communication.

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