

differences between this meteorite and the Murchison meteorite. Apart from these differences, a similarity in the amino acid composition of these two carbonaceous chondrites is apparent from the ratios calculated in Table 1. This compositional similarity would seem to further substantiate the conclusion that these compounds are indigenous to type II (C2) carbonaceous chondrites. The Murchison and Murray meteorites are extremely similar in their macroscopic characteristics. Both meteorites have total carbon contents of about 2.2 percent and a total nitrogen content in the range of 0.15 to 0.20 percent (by weight) (15). There is every reason to believe that all the C2 chondrites have a similar origin and similar chemical characteristics. The Allende chondrite, on the other hand, contains 0.25 percent carbon and only 0.006 percent nitrogen (by weight). It remains to be seen whether the amino acid composition reported for the Murchison and Murray meteorites will prove to be representative of type II and possibly type I carbonaceous chondrites.

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References and Notes

1. Work up to December 1966 has been reviewed by J. M. Hayes, *Geochim. Cosmochim. Acta* **31**, 1395 (1967).
2. K. Kvenvolden, J. Lawless, K. Perring, E. Peterson, J. Flores, C. Ponnamperna, I. R. Kaplan, C. B. Moore, *Nature* **228**, 923 (1970); K. Kvenvolden, J. Lawless, C. Ponnamperna, *Proc. Nat. Acad. Sci. U.S.A.* **68**, 486 (1971).
3. See, for example, *Sci. News* **98** (No. 23), 429 (5 December 1970).
4. Water was twice distilled in a glass distilling flask, redistilled from alkaline potassium permanganate, and again redistilled from alkaline sodium hydrosulfite. All glassware was thoroughly washed and held at the Pyrex annealing temperature (560°C) for 15 minutes prior to use.
5. J. V. Benson, Jr., and J. A. Patterson, *Anal. Chem.* **37**, 1108 (1965).
6. R. W. Hubbard and D. M. Kremen, *Anal. Biochem.* **12**, 593 (1965).
7. D. H. Spackman, W. H. Stein, H. Moore, *Anal. Chem.* **30**, 1190 (1958).
8. J. Oro, J. Gilbert, H. Lichtenstein, S. Wikstrom, D. A. Flory, *Nature*, in press.
9. M. Calvin and S. K. Vaughn, in *Space Research*, H. K. Bijl, Ed. (North-Holland, Amsterdam, 1960), p. 1171.
10. I. R. Kaplan, E. T. Degens, J. H. Reuter, *Geochim. Cosmochim. Acta* **27**, 805 (1963).
11. J. Oro and H. B. Skewes, *Nature* **207**, 1042 (1965).
12. J. Raia, thesis, University of Houston (1966).
13. J. Oro, S. Kakaparksin, H. Lichtenstein, E. Gil-Av, *Nature*, in press.
14. The samples used in this study were all from the Center for Meteorite Studies, Arizona State University.
15. E. K. Gibson, C. B. Moore, C. F. Lewis, *Geochim. Cosmochim. Acta*, in press.
16. Supported in part by NSF research grant GA 14389 and NASA grants NGL-03-001-001 and NGR 05-007-215. Contribution No. 64 from the Center for Meteorite Studies.

19 February 1971

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Magnetocardiography of Direct Currents: S-T Segment and Baseline Shifts during Experimental Myocardial Infarction

Abstract. *Magnetocardiograms with a bandwidth of 0 to 40 hertz were recorded from intact dogs undergoing myocardial infarction. This was done with a superconducting magnetometer in a magnetically shielded room. The purpose was to look for the steady currents of injury from the heart which supposedly produce much of the S-T segment shifts during infarction. These heart currents cannot be measured with surface electrodes because of direct-current interference from other sources, such as from the contact potential between electrode and skin. The magnetocardiograms showed both S-T segment shifts and direct currents as a result of infarction. However, they also showed that the S-T segment shifts were not produced by the direct currents. It is unlikely that these direct currents originated from the infarcted area, and their exact origin is not yet known.*

Diagnosis of a myocardial infarction strongly depends on certain telltale signs on the electrocardiogram (ECG); one such sign is a shift of the S-T segment. Although many careful measurements have been made on exposed animal hearts in order to understand the cause of the shift, the results are not consistent and the cause is still not clear. We have started to investigate the S-T shift by using the new technique of direct-current (d-c) magnetocardiography and find that we are obtaining information not previously available. Our work is the first use of this d-c technique and we present here some of our initial results.

Magnetocardiography is the measurement of the weak magnetic field around the torso produced by natural ion currents from the heart. The currents in the torso which produce the heart's magnetic field are powered by the electrical activity of the heart muscle; it is these same currents that also produce the ECG. A record of the heart's pulsating magnetic field is called a magnetocardiogram (MCG). Because the MCG has its origins in the same currents as the ECG, the MCG has features similar to the ECG, such as the QRS complex and the T-wave. It differs from the ECG by sampling the heart currents differently, and in prin-

ciple can show some heart events not detectable with the ECG (1). At the peak of QRS the heart's magnetic field rises to about 5×10^{-7} gauss or one-millionth of the earth's magnetic field (2). The first MCG was recorded by using two large coils on the chest, connected in opposition to reduce the background magnetic disturbances (3). Another detection scheme made use of a single, compact coil situated in a magnetically shielded room to reduce the background; with this system the heart's magnetic field was verified, the brain's alpha-rhythm field was detected at about 1×10^{-9} gauss, the heart's QRS field was mapped in detail, and a steady magnetic field of about 1×10^{-6} gauss was found at the abdomen (4). Recently, a more heavily shielded room was constructed at Massachusetts Institute of Technology (5) and a newly devel-

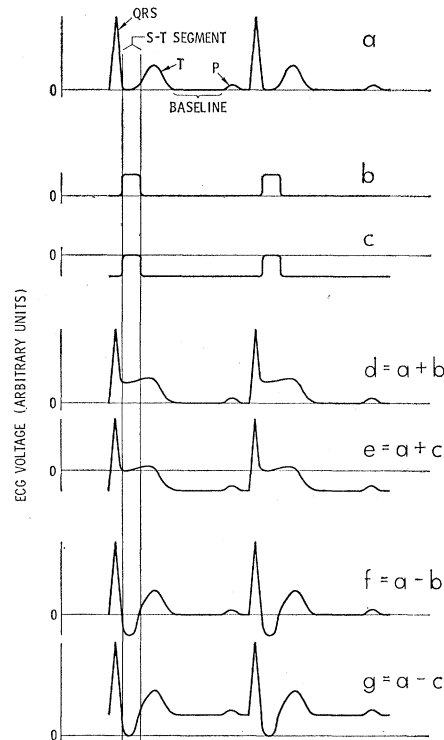


Fig. 1. Often-used explanation of the S-T segment shift on the ECG during infarction. Before infarction, the ECG is normal, as in *a*. After the start of infarction, the infarcted area generates new voltages *b* or *c*, or both; *b* can be produced by two different physiological events and *c* by yet another, third event. The lowered baseline level of *c*, which is d-c, is called the "current of injury." The various combinations of *a* with *b* and *c*, shown in *d*, *e*, *f*, and *g* appear as surface voltages. The standard ECG cannot tell the difference between *d* and *e* or between *f* and *g*.

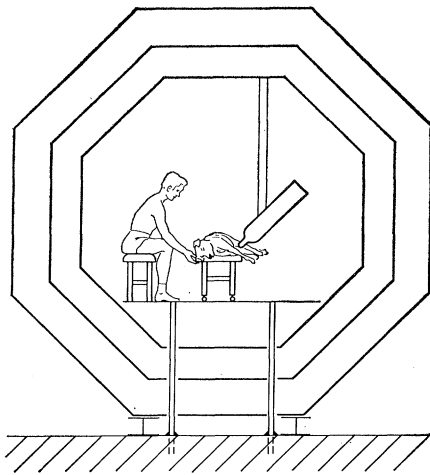


Fig. 2. Magnetocardiograms being taken of an infarcted dog in the shielded room. The SQUID detector is situated in the tail of the Dewar containing liquid helium, which is shown fixed to the ceiling. The detector output, which is proportional to the horizontal component of the magnetic field, goes to an external display station. The dog lies on a rolling table that can easily be moved around in order to place different parts of the dog's body close to the detector. Not shown are the tube and syringe for producing the infarction, or the ECG limb leads. All magnetic material, such as the experimenter's shoes, trousers, and so forth, is removed from the room.

oped SQUID (superconducting quantum interference device) (6) magnetometer was installed in the room, which resulted in new, higher sensitivity (7). One use of the room-SQUID system is the measurement of d-c fields; the previous detectors we mentioned, which were coils, could respond only to alternating current, but the SQUID has a flat response down to d-c, which is its characteristic that is most valuable to this study. The shielded room has a low and stable residual field and is therefore suitable for d-c studies.

Direct current is involved in the S-T shift through the concept of "current of injury"; most authors prefer to explain the shift as largely caused by this current. In this explanation, illustrated in Fig. 1, three different physiological events take place in sequence at the start of infarction. The first event is a shortening of the action potential of the infarcted cells and macroscopically appears as a new voltage *b* from the infarcted area. This first event lasts only a few minutes, during which the second event appears. The second event, which can last for many hours, is a decrease of the resting potential of the infarcted cells and macroscopically appears as *c*, which contains the steady (d-c)

current of injury as a displacement of the baseline. The third event, beginning about 30 minutes after infarction, is a failure of the infarcted area to depolarize due to further decrease of the resting potential; this macroscopically appears as *b* again even though the fundamental mechanism is different from the first event. Curves *b* and *c* are added to or subtracted from *a*, depending on relative infarct-torso geometry, to produce *d* and *e* or *f* and *g*. The major contributor to the S-T segment shift is considered to be *c*; its associated steady (d-c) current of injury shifts the ECG baseline in Fig. 1, *e* and *g*. If d-c levels could be measured with electrodes on the skin, then this theory of the S-T shift could be experimentally verified; if found to be true, measurements of these levels could eventually become standard diagnostic information. However, it is difficult to measure d-c on the skin from a deep generator because of local effects such as electrode polarization and the galvanic skin effect; the standard ECG, for example, has a lower cutoff at about 0.1 hz and does not measure d-c such as the baseline levels in Fig. 1, *e* and *g*. Because of these skin voltage difficulties, investigations of d-c from infarcts have usually (8) been made directly on the surface of surgically exposed hearts of animals in situ, where the d-c levels are much larger than at the skin. Such measurements by several groups (9) support the explanation of Fig. 1, while similar careful measurements by others (10) give different results.

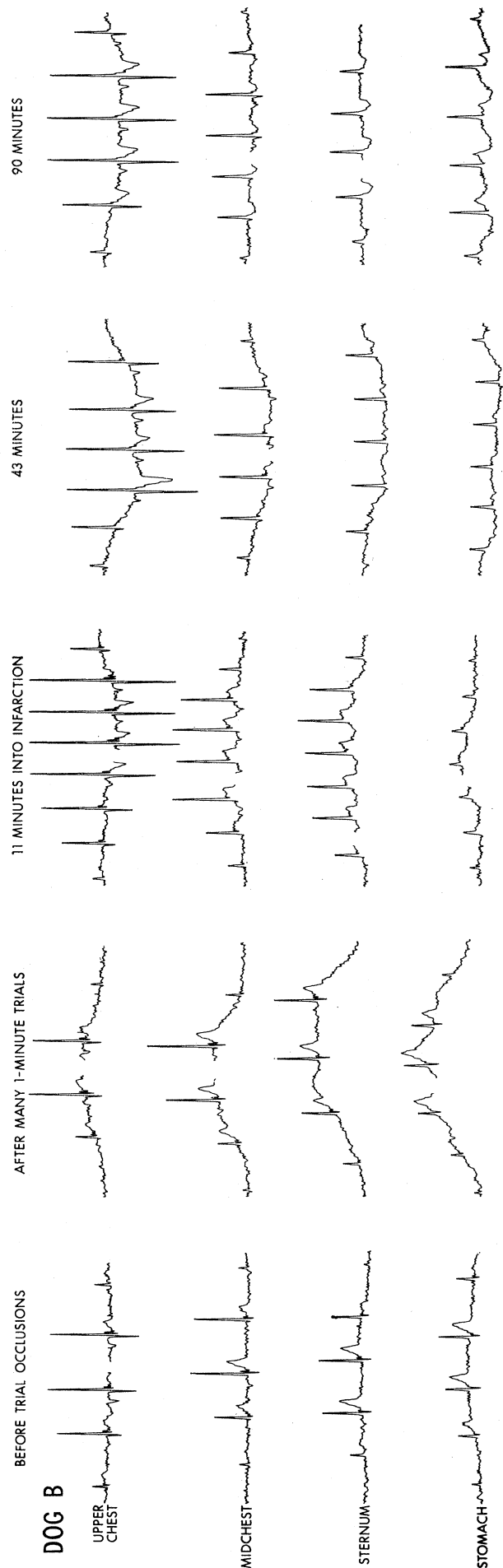
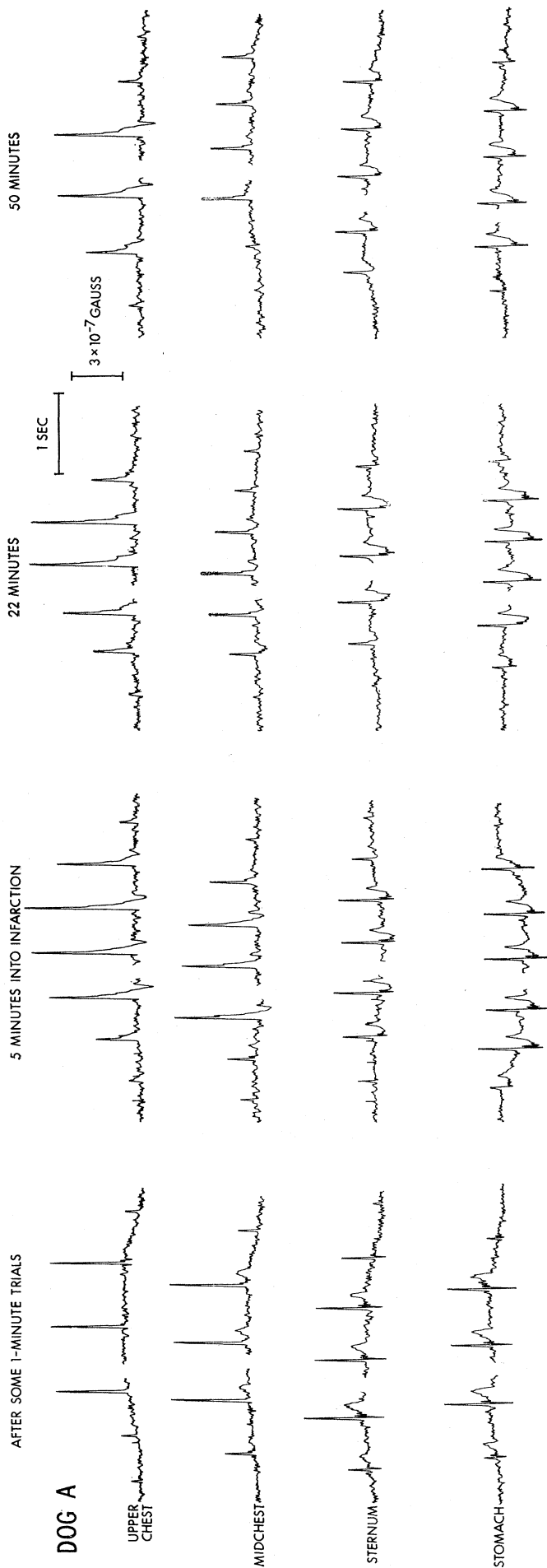
We have combined two techniques in order to bypass some of these difficulties in measuring d-c and to learn more about the connection between d-c and the S-T segment shift. One technique is the new room-SQUID capability of measuring small d-c fields (11). The other technique is to produce infarctions in the intact dog (12); this is done by externally (through Silastic tubing) occluding a cuff around a coronary artery, the cuff having been surgically implanted a week or two earlier. Combining these techniques consisted of recording the d-c MCG's of intact dogs undergoing infarction in the shielded room. The d-c MCG, which is defined by a bandwidth of 0 to, say, 40 hz, has the features usually seen on the ECG as well as a baseline position which is a measure of the d-c magnetic level; it therefore can show the difference between, for example, *f* and *g* in Fig. 1. Because the MCG involved

no voltage contact with the skin and responds mostly to deep, strong currents, the difficulty with surface electrode measurements of d-c is bypassed. Also, using an intact dog eliminates the biochemical and electrical complications of measurements on a surgically exposed heart.

In these experiments the baseline of the d-c MCG is only the relative d-c level at the magnetometer; the zero d-c level was arbitrary. This relative level, however, gives the dog's absolute d-c level in the following way. With the dog out of range of the magnetometer, the magnetometer output level was noted on a chart recorder. The dog was then wheeled up to the magnetometer, which was fixed in position, and placed in a predetermined orientation with respect to the magnetometer. As a result, an MCG appeared on the recording with a baseline that had shifted from the original level. After a few seconds in this position, the dog was wheeled out of range again and the baseline returned to its original position. The difference between the MCG baseline and the out-of-range level is then the absolute d-c level of the dog's MCG.

Figure 2 shows the experimental arrangement. An infarction was produced by gradually forcing about 1 ml of liquid from the outside, with a

Fig. 3. Direct-current MCG's before and during myocardial infarctions of two dogs. Only MCG's of the magnetic field component which was normal to the torso are shown here. In dog A, the anterior descending branch of the left coronary artery was occluded; in dog B, the circumflex branch was occluded. Each MCG was made by continuously recording the detector output during the sequence of (i) dog away, (ii) dog rolled to detector, and (iii) dog rolled away. For the purpose of this display, the ends of each MCG have been trimmed off so that steps (i) and (iii) are short, but just visible; also several heartbeats were usually removed from step (ii), shown by a short gap. The ends of each MCG define the zero level, and the shift of the baseline from this zero level is the d-c level of the MCG. Note the d-c baseline shifts as a result of trial occlusions and infarction; the largest are from dog B after the trials. Note also the S-T segment shifts in both dogs, as a result of infarction. The upper two rows of dog A and the 11-minute column of dog B show S-T elevation; the lower two rows of dog A and two MCG's in the 90-minute column of dog B show S-T depression. One general conclusion from these MCG's is that there is no clear relation between the S-T segment shifts and the baseline shifts.



syringe, through the tubing into the balloon cuff. Before infarction, a determination was made of the exact amount of liquid necessary to just occlude the artery. This was done in the shielded room by the experimenter (W.H.) who made a series of 1-minute trial occlusions with increasing amounts of liquid; he watched the dog's ECG on a wall-mounted cathode-ray tube during these occlusions until there was an appreciable T-wave or S-T segment change. The permanent infarction was then begun with less than this critical amount of liquid which was increased over a period of 20 minutes or so. Direct-current MCG's were usually recorded just before and just after the trial occlusions, then periodically during the infarction. The dogs were conscious but lightly sedated in some instances with about 15 mg of morphine sulfate given intramuscularly. Infarctions, verified by eventual autopsies, were produced in five dogs weighing between 25 and 30 kg. Preliminary d-c studies were made during the infarctions of the first two dogs, and much MCG and ECG data were taken during the infarctions of the last three. Some of the data from two of these three are shown in Fig. 3. Both dogs were scanned for unwanted (ferromagnetic) d-c fields (13) at various times before the infarction, and it was determined that they were magnetically "clean." A typical scan is shown in the first column ("Before trial occlusions") for dog B where only a small amount of d-c exists at the stomach and sternum. The infarction of dog A was straightforward, as shown by the ECG, MCG, and autopsy. With dog B, many trial occlusions of the anterior descending branch produced no sign of infarction on the ECG, so we then occluded the circumflex branch, which resulted in infarction; the dogs were usually prepared with a cuff around each branch.

We conclude from the five infarctions, and especially from the infarctions of dogs A and B, that myocardial infarctions produce d-c. This d-c can appear strongly within a few minutes after the start of infarction or even during short trial occlusions, then it seems to die away during the first hour or so. As to the connection between this current and the S-T segment shifts, we make the following arguments. (i) It is certain that the d-c and the S-T segment shifts do not completely come from the same cellular events, otherwise

there would be an exact correlation between these, both in various MCG positions and at various times; that is, the amplitude of the baseline shifts would always be tied to the amplitude of the S-T segment shifts. It is seen from Fig. 3 that there is no clear correlation. (ii) It is unlikely that any of the d-c seen in Fig. 3 is directly connected with the S-T segment shifts. For example, consider the right-hand column for both dogs; although there are large S-T segment shifts in three of the MCG's of dog A and one or two of dog B, there are no significant baseline shifts in any of these five MCG's. The baselines have almost returned to their preinfarction positions, yet the large S-T segment shifts have remained. These S-T shifts, therefore, do not have any appreciable associated d-c.

It follows from argument (ii) that the data of Fig. 3 do not support that part of the theory of Fig. 1 which involves the current of injury. Not only do the latter five S-T segment shifts exist without any d-c, but also the S-T segment shifts in other MCG columns generally do not have associated baseline shifts with proper polarity or enough amplitude. This is seen, for example, in the two middle MCG's of dog B (11 minutes) or the four MCG's of dog A (22 minutes). Stated in other terms, none of the MCG's of Fig. 3 have the character of either *e* or *g* in Fig. 1, which have opposing S-T segment and baseline shifts.

Even though the d-c does not seem to be connected with the S-T segment shifts, do they nevertheless come from the same location in the heart? That is, conceivably the d-c could originate in the same region (presumably the infarct) as the S-T segment shifts but has a different time sequence and directionality. In answer, there is some evidence in Fig. 3 which suggests that almost none of the d-c came from the infarct. For example, consider the 22-minute column of dog A; the upper two MCG's have S-T elevation and the lower two have S-T depression. This distribution results from the relative geometry of the infarct and the torso. If the d-c originated at the infarct, one would expect some kind of polarity split of the baseline shifts between the two top and two bottom MCG's of any column of dog A; however, the new d-c in the 5-minute column of dog A, for example, shows no such split. This suggestion that almost no d-c comes from the infarct is seemingly at variance with

the d-c voltage measurements on the exposed hearts (9, 10). Direct-current voltages at the infarct were certainly seen and measured. One would expect these voltages to give rise to currents in the torso, hence also to d-c magnetic fields which we would identify as coming from the infarct by comparison with the S-T segment shifts. One explanation may be that exposure of the heart changes the bioelectrical situation as compared with the intact animal. Perhaps the d-c voltage on the exposed heart originates from a high-impedance source that produces almost no current when in contact with the low-impedance chest wall.

If the currents seen in Fig. 3 do not seem to come from the infarct, what is their source? As yet we do not know; they may not even come from the heart. Some MCG's taken during the infarctions of dogs A and B at angles other than normal-to-torso only roughly indicate a source somewhere within the central torso region, including the heart. More work is needed to further localize the d-c source and to gather more data on the relation between d-c and the S-T segment shift.

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References and Notes

1. Various references below [in (3) and (4)] describe how the MCG is produced in various cases by a selection and summation of heart currents in the torso. For simplicity one may think of the MCG as being similar to the ECG except it is produced by a new special "system of leads"; each MCG recorded at a particular location around the torso can be considered as having its own set of leads, where the leads vary with MCG location.
2. The earth's steady field is about 0.5 gauss; the fluctuations in an urban environment might be about 5×10^{-4} gauss (root-mean-square) in a bandwidth of 0 to 40 hz, hence these background fluctuations must be reduced by a factor of about 10^4 in order to see features of the MCG.
3. G. M. Baule and R. McFee, *Amer. Heart J.* **66**, 95 (1963); *J. Appl. Phys.* **36**, 2066 (1965); Y. D. Safonov and V. M. Provotorov, *Bull. Exp. Biol. Med.* **64**, 1022 (1967).
4. D. Cohen, *Science* **156**, 652 (1967); *ibid.* **161**, 784 (1968); *Circulation* **39**, 395 (1969); *J. Appl. Phys.* **40**, 1046 (1969).
5. —, *Rev. Phys. Appl.* **5**, 53 (1970).
6. This belongs to a class of devices that use the Josephson effect, and the particular version we used is described by J. E. Zimmerman, P. Thiene, J. T. Harding [*J. Appl. Phys.* **41**, 1572 (1970)].

7. D. Cohen, E. A. Edelsack, J. E. Zimmerman, *Appl. Phys. Lett.* **16**, 278 (1970).
8. We have found one report of a measurement of d-c on the skin of intact dogs during infarction but with no discussion of the problems of drift and reliability [L. Soloff, G. A. De Los Santos, M. Oppenheimer, *Circ. Res.* **8**, 479 (1960)].
9. M. Kardesch, C. E. Hogancamp, R. J. Bing, *ibid.* **6**, 715 (1958); W. E. Samson and A. M. Scher, *ibid.* **8**, 780 (1960); M. Prinzmetal, K. Ishikawa, M. Nakashima, H. Oishi, E. Oszan, J. Wakayama, J. M. Baines, *J. Electrocardiol.* **1**, 161 (1968).
10. A. H. Katcher, G. Pierce, J. J. Sayen, *Circ. Res.* **8**, 29 (1960).
11. The idea of magnetically detecting the direct current from the heart was put forth by Prof. Otto Schmitt during the 1967 meeting of the Biophysical Society and restated by D. Geselowitz and O. Schmidt [in *Biological Engineering*, H. P. Schwan, Ed. (McGraw-Hill, New York, 1969), p. 365].
12. W. B. Hood, Jr., J. Joison, R. Kumar, I. Katayama, R. S. Neiman, J. C. Norman, *Cardiovasc. Res.* **4**, 73 (1970).
13. Some studies have been made in the shielded room of d-c fields in general from both humans and dogs. In both cases, the abdomen is the largest source of d-c, occasionally higher than 10^{-6} gauss after drinking cold water, or still higher if minute amounts of magnetic material exist in the abdomen.
14. Supported by grants B014040 from NSF; 69-555 and 70-734 from the American Heart Association; PH 43-68-684, HE 5244, HE 10539, SF 57-111, and AM 10517 from NIH; 1020 from the Massachusetts Heart Association; and from the John A. Harford Foundation. We thank Elmer Davis for his administrative advice, Edward Givler for engineering of the MCG equipment, and Peter Howland for his assistance in surgery and occlusions. D.C. is an Established Investigator of the American Heart Association.

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Schöpfung, Maclure, Werner, and the Earliest Work on American Geology

Abstract. Schöpfung produced the first substantial report on the geology of North America, a work that has long suffered unjustified oblivion. In at least one feature this treatise is superior to that of Maclure (the "Father of American Geology")—Schöpfung attempted interpretation whereas Maclure did not. The suggestion is here offered that Maclure, who adopted Werner's classification of the stratigraphic succession, was simply shying away from Wernerian interpretation because he did not like the looks of it.

We honor William Maclure (1763–1840) as the "Father of American Geology," and in a strictly practical sense this is a just recognition, for it was indeed Maclure's work that started things moving in the study of American geology; and yet, Maclure was not the actual pioneer. He was preceded some 22 years earlier by another man who produced a much larger work (1) which, however, in utterly mysterious circumstance went virtually unknown in America for more than a century (2). This, admittedly, is hard to believe, but extensive investigation leaves it just such—one of those freakish, incomprehensible oddities that do turn up now and then in the long course of human history.

This other man was a young German army surgeon, Dr. Johann David Schöpfung (1752–1800), who came over with the Hessian troops hired by King George III of England to help combat the rebellion in the American colonies, and at the end of the war, instead of going straight home with the soldiers, he stayed here for some 8 months and traveled as far west as Pittsburgh and as far south as Charleston and St. Augustine, gathering data on just about every aspect of American life and environment that anyone could well be expected to observe. The geological part of all this he published in the work

just mentioned (1) and the other parts of general interest in a two-volume work (3) that was translated into English by Professor Alfred Morrison of Hampden-Sydney College and published in 1911 at Philadelphia in an edition (4) that itself soon fell into the category of rare books, but fortunately a facsimile reproduction (5) has recently been issued. I say "fortunately" because the "Reise" is well worth having by anyone who is at all interested in a picture of the newborn United States that evolved through the eye and mind of a highly intelligent and cultured European naturalist who was not only trained in all of his education and experience to observe sharply and critically, but more than that, was definitely a member of the avant-garde in his time (the time, we might recall, of Goethe, Voltaire, and Rousseau). Further, the "Reise" should be of special interest to geologists, geographers, and economists for its many mentions and descriptions of commercially important mineral deposits over and above those dealt with in the "Beyträge," and other natural resources as well.

The main purpose of this note is to bring the whole of Schöpfung's American presence to attention, to compare one aspect of his treatise on geology with that of Maclure, and to offer a suggestion that has apparently never been

ventured in discussions of the history of geology.

Schöpfung's work was done mainly between late July 1783 and January 1784. This was the time when the science of geology was just beginning to take form. In other words, there was no geology; Schöpfung had to create his own. (Note that the title of his book is "Contributions to the *mineralogical* knowledge. . .") The most important feature in which his work is superior to that of Maclure is his constant attempt at interpretation, whereas Maclure modestly abstained from any such enterprise. As Schöpfung made his way across our Coastal Plain, Piedmont, the great limestone valleys, and the Allegheny-Appalachian province, for nearly everything he saw he tried to understand its origin, to explain it—to interpret. Some of his ideas are remarkably sound and prescient; some are wrong or otherwise deficient, but in all such cases he was cautious and in many he realized that he must be in error even though he could not think of anything better. (For example, he thought the whole area, in essentially its present kind of configuration, must have been flooded by universal ocean, but he most positively was uncomfortable with the idea, recognized specific objections to it, and said frankly that he must be missing something, which was indeed the case.)

Now Maclure would have none of such business. He said (6), "In adopting the nomenclature of Werner, I do not mean to enter into the origin or first creation of the different substances, or into the nature and properties of the agents which may have subsequently modified or changed the appearance and form of those substances; I am equally ignorant of the relative periods of time in which those modifications or changes may have taken place; such speculations are beyond my range, and pass the limits of my inquiries."

I fail to see how anybody can read that statement and still call Maclure a Wernerian. If this is not a disclaimer I do not think I have ever seen one. Further, it has occurred to me that Maclure, the canny Scot, was not merely avoiding elaborate speculation as to possible origins, but was just not going to associate himself with ideas (Wernerian) that did not look any too good to him. We remember Maclure's work, and rightly so, first of all for his map, the first of its kind in America. It is hard to believe, however, that he studied and recognized the rock units