EVIDENCE OF A NONLINEAR HUMAN MAGNETIC SENSE

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Abstract—Human subjects respond to low-intensity electric and magnetic fields. If the ability to do so were a form of sensory transduction, one would expect that fields could trigger evoked potentials, as do other sensory stimuli. We tested this hypothesis by examining electroencephalograms from 17 subjects for the presence of evoked potentials caused by the onset and by the offset of 2 G, 60 Hz (a field strength comparable to that in the general environment). Both linear (time averaging) and nonlinear (recurrence analysis) methods of data analysis were employed to permit an assessment of the dynamical nature of the stimulus/response relationship. Using the method of recurrence analysis, magnetosensory evoked potentials (MEPs) in the signals from occipital derivations were found in 16 of the subjects (P<0.05 for each subject). The potentials occurred 109–454 ms after stimulus application, depending on the subject, and were triggered by onset of the field, offset of the field, or both. Using the method of time averaging, no MEPs were detected. MEPs in the signals from the central and parietal electrodes were found in most subjects using recurrence analysis, but no MEPs were detected using time averaging. The occurrence of MEPs in response to a weak magnetic field suggested the existence of a human magnetic sense. In distinction to the evoked potentials ordinarily studied, MEPs were nonlinearly related to the stimulus as evidenced by the need to employ a nonlinear method to detect the responses.

Key words: evoked potentials, magnetic detection, nonlinearity, electroencephalogram, recurrence analysis, transduction.

Low-intensity electric and magnetic fields (EMFs) can affect brain activity in human beings and animals (Bawin et al., 1973; Gavallas-Medici and Day-Magdaleno, 1976; Sartucci et al., 1997; Preece et al., 1998; Thoss and Bartsch, 2003; Cook et al., 2004; Ghione et al., 2005), and it is a central problem in biology to understand the nature and physiological significance of the phenomenon. Using power-spectra analysis of electroencephalograms (EEGs), we showed that low-frequency EMFs altered brain activity in at least half of the human subjects studied (Bell et al., 1991, 1992a; Marino et al., 1996). When EEGs were analyzed by means of a method capable of detecting nonlinear stimulus-response relationships, EMF-induced changes were found in almost all the animal and human subjects (Marino et al., 2002, 2004).

We proposed that the changes in brain activity were a result of transduction of the fields (Sonnier and Marino, 2001). In this view, the field interacts with membrane-bound or intracellular structures in specialized neurons or neuroepithelial cells, resulting in an afferent signal. EMF receptor cells have been identified in the nervous system of some species of fish and lower mammals (Wachtel and Szamier, 1969; Manger et al., 1995, 1996; Walker et al., 1997; Nemec et al., 2001), but not in human beings.

Evoked potentials are stimulus-induced changes in brain electrical activity that occur by means of superposition on the EEG (Regan, 1975), or as a result of nonlinear processes (Breakspear, 2002; Tass, 2003; David et al., 2003; Stam, 2005). If EMF detection were a form of sensory transduction, we would expect that they could evoke brain potentials, like other sensory modalities. If the hypothesized field-induced potentials occurred as a result of nonlinear brain processes, we would expect that a method of analysis designed to detect nonlinear stimulus–response relationships would be more sensitive than methods designed to detect linear relationships.

One objective of this study was to determine whether the onset or offset of 2 G, 60 Hz induced transient potentials in the human brain, and to assess their duration and latency. A second objective was to test the hypothesis that the potentials were nonlinearly related to presentation of the stimulus; this was accomplished by comparing the results obtained using nonlinear (recurrence analysis) and linear (time averaging) methods to detect the putative potentials.

EXPERIMENTAL PROCEDURES

Subjects

Seventeen clinically normal subjects were studied: eight males (age range 20–51 years) and nine females (18–50 years). The subjects were informed of the goals, methods, and general design of the investigation, but were not told exactly when or for how long the field would be applied. Written informed consent was obtained from each subject prior to participation in the study. The institutional review board at the Louisiana State University Health Sciences Center approved all experimental procedures.

Magnetic field

Uniaxial magnetic fields, 2 G (200 μT), 60 Hz, uniform to within 5% in the region of the head, were applied by means of two sets of...
three coaxial coils (Fig. 1a). A field of 2 G was chosen for study because it is present in the general and work environments (Bernhardt, 1988). Each set consisted of a circular coil (21 turns, radius of 21.6 cm), and two square coils (85, 120 turns, respective side length of 48.3 cm and 66 cm). The coil current was obtained using a function generator (Model 182A, Wavetek, San Diego, CA, USA) and amplifier (Model 7500, Krohn-Hite, Avon, MA, USA), and was applied by means of a zero-crossing switch controlled by a computer-generated timing signal (Fig. 1b).

Field exposure took place in a darkened isolation chamber that reduced the subject’s exposure to random ambient stimuli. The subjects sat in a comfortable wooden chair with their eyes closed and their sagittal plane perpendicular to the field. The equipment that controlled the coils and recorded the EEG was located outside the chamber, thereby eliminating the possibility of auditory or visual cues from the experimental apparatus. No subject consciously perceived the magnetic field because it was below the threshold for awareness. The absence of sensory cues was verified by interviewing each subject at the end of the experimental session. The background 60-Hz magnetic field (field present during the sham-exposure and control epochs) was 0.1 mG; the geomagnetic field was 599.8 mG, 68.4° below the horizontal (component along the direction of the applied field, 360.5 mG). All magnetic-field measurements were performed using a triaxial magnetometer (Bartington, MAG-03, GMW, Redwood City, CA, USA).

Our goal was to detect potentials caused by onset and by offset of the field. To avoid confounding the two putative effects, the EMF was applied for 2 s and the EEG voltage signal, V(t), was analyzed to detect the effects of field onset and field offset; a portion of the signal recorded during the interstimulus period (see below) served as the control.

The coil voltage was applied instantaneously, and the coil current reached its predetermined steady-state value in approximately 10 ms (Fig. 1c, left). The change in current produced a voltage spike in V(t), in accordance with Faraday’s law; the spike
was broadened to 30 ms because of the time-constant of the measuring circuit (Fig. 1c, right). Similar spike phenomena occurred when the field was terminated. In preliminary studies, the faradic origin of the spike was established by means of measurements on electrical phantoms of the human head (a melon, and a spherical volume of 0.9% NaCl); when electrodes were attached to the phantom and the magnetic field was applied, the spike was duplicated exactly. The spikes in V(t) due to onset and offset of the field were deleted by removing the first 30 ms (10 points, see below) after presentation of the stimulus.

As a positive control, a binaural 424-Hz tone (10 ms rise and fall times) was presented for 2 s, with an interstimulus period of 5 s; the sound pressure at the location of the subject was 65 dB.

### EEG recording

EEGs were recorded from O1, O2, C3, C4, P3, and P4 (International 10–20 system) referenced to linked ears, using gold-plated electrodes attached to the scalp with conductive paste. Electrode impedances (measured before and after each experiment) were 3–5 kΩ in 11 subjects and 5–10 kΩ in six subjects. The signals were amplified (Nihon Kohden, Irvine, CA, USA), filtered to pass 0.5–35 Hz, sampled at 10 kHz using a 12-bit analog-to-digital converter (National Instruments, Austin, TX, USA), and stored on a computer hard drive. For analysis, the signal was divided into consecutive 7-second intervals (trials); in each, field onset was at t=0, field offset at t=2 s, and t=2–7 s was the interstimulus period. Trials containing artifacts as assessed by visual inspection were discarded. Various algorithms for automatic or semi-automatic removal of artifacts from the EEG are being developed, but thus far none has been shown to be superior to visual inspection (Krishnaveni et al., 2006). The artifact-free trials were sub-sampled at 300 Hz and digitally filtered between 0.5–35 Hz after removal of the spikes. All results were based on data from at least 50 trials.

### Nonlinear and linear analysis

A description of the general mathematical properties of recurrence analysis when used to evaluate brain potentials is given elsewhere (Carrubba et al., 2006).

For detection of evoked potentials by means of recurrence analysis, the epochs of interest in V(t) (t=0.03–1 s, 2.03–3 s, and 5.03–6 s, corresponding to onset, offset, and control intervals, respectively) were embedded in separate phase spaces, and recurrence plots for each of the epochs were generated and quantitated by means of percent recurrence, %R(t), which was the nonlinear quantifier that we used to characterize the effect of the field. %R(t) was uniquely determined from V(t) by means of mathematical methods described elsewhere (Eckmann et al., 1987; Webber and Zbilut, 1994; Jeong et al., 2001). Briefly, the first 100 ms of each V(t) epoch was embedded in a five-dimensional phase space using a time delay of 5 ms (Jeong et al., 2001), and the corresponding recurrence plot was generated (Eckmann et al., 1987) (scale, 15%) and quantified using %R, defined as the number of recurrent points in the plot divided by the total number of points in the recurrence matrix (Webber and Zbilut, 1994). The process was repeated using a sliding window of 1 point in V(t), yielding the time series %R(t), which was smoothed using a 100-ms, step-1 averaging window; the resulting time series, %R(t), was analyzed for the presence of evoked potentials.

From a formal viewpoint, %R is a measure of the extent to which the EEG is correlated with itself in phase space. The advantage of %R is that it can quantify the recurrence plot, which is a useful device for revealing patterns of dynamical activity not detectable by eye or by conventional analysis. The disadvantage of %R is that it does not provide direct insight into the physiological basis of the dynamical activity.

To synchronize the graphical representation of V(t) and %R(t), we adopted the convention that each point in %R(t) was plotted at the time corresponding to the middle of the interval in V(t) from which it was computed. For example, the value of %R(t) determined by the 100-ms interval in V(t) beginning at t=30 ms appeared in a plot of %R(t) at t=80 ms; when that point was the first in the 100-ms averaging window for %R(t), it was plotted at t=130 ms. Thus, %R(t=130) reflected the determinism that occurred in V(t) within 30–230 ms.

The adjustable parameters in the analysis were chosen on two bases. First, we performed mathematical modeling in which 300-ms segments of linear and nonlinear signals having spectral properties typically found in evoked potentials were added to baseline EEGs, and optimal parametric values for detecting the added determinism were ascertained by trial and error (Carrubba et al., 2006); the parameters thus fixed were embedding dimension, time delay, scale for calculating %R from the recurrence plot, V(t) window for calculating %R(t), and %R(t) averaging window for calculating %R(t). Second, EEGs from the first three subjects enrolled in the study were used to delineate conditions for digital filtering and to identify the expected latency range of the evoked potentials (see below).

As with recurrence analysis, linear analysis was performed for each subject to permit a statistically-based decision for each subject regarding the detection of brain potentials. V(t) was averaged over the exposure trials, and the averaged signal was examined for the presence of evoked potentials based on statistical comparisons of the epochs of interest in V(t) (t=0.03–1 s, t=2.03–3 s, t=5.03–6 s).

### Experimental design and statistics

We chose an intra-subject design so that the effect of the field could be assessed in individual subjects. Each subject underwent three blocks of 80 trials (Fig. 1d); the magnetic field was applied in either the first or third block, as determined randomly from subject to subject. In the block where the field was not applied, the data were analyzed as a negative control (sham exposure). Sound was applied in the middle block (positive control).

On the basis of a discriminant analysis of the EEG from the first three subjects enrolled in the study, we found that onset potentials occurred within 209–354 ms in %R(t), and that they were detected more robustly if, when the relative alpha power (8–13 Hz) was ≥30%, V(t) was digitally filtered to remove the 9–12 Hz power prior to computing %R(t). We chose a level of 30% because it was maximally effective in revealing potentials in the three subjects. In several instances (noted in the figures) 8–10 Hz power was removed because it revealed a stronger effect. The aim of filtering was to improve the discrimination between the exposed and control epochs. The usefulness of alpha filtering was shown in a previous study (Marino et al., 2003b). After the conditions regarding latency range and alpha filtering were delineated, they were applied prospectively to a determination of the offset potentials in the three subjects, and to both onset and offset potentials in the remaining 14 subjects.

In both the onset and offset epochs, the values of %R(t) between 209 and 354 ms (45 points, which described the determinism in V(t) at 109–454 ms) were compared separately with the corresponding points in the control epochs using the paired t-test at a pair-wise significance level of P<0.05 (identical results were found using the Wilcoxon signed rank test). It can be shown that the probability of observing ≥6 significant differences by chance at P<0.05 in 45 tests is 0.024. In a previous study involving dynamical analysis of the EEG we found that O1 and O2 were the most sensitive derivations for detecting an effect of a field (Marino et al., 2004); we therefore based the statistical design of this study on the likelihood of detecting an effect from the occipital electrodes. We planned to conclude that a subject had exhibited an...
evoked potential if ≥6 consecutive tests were pair-wise significant in O1 or O2, or both. The family-wise error rate for our statistical hypothesis was \( P = 1 - (\frac{1}{1} - 0.024)^2\) = 0.047, which was sufficient to reasonably exclude the role of chance. We also evaluated the reliability of this statistical design a posteriori, by analyzing the sham-exposure data to empirically determine the likelihood of a false-positive decision regarding detection.

We regarded a potential as nonlinear if it was detected in \( \%R(t) \) but not in \( V(t) \).

**RESULTS**

Following application of the magnetic field, changes in the signals from the occipital electrodes were detected by recurrence analysis but not by time averaging; typical results are shown in Fig. 2. \( V(t) \) after field onset (\( t = 0.03–1 \)) did not differ from the control (\( t = 5.03–6 \)), as determined by comparing the onset and control epochs, point by point, using the paired \( t \)-test (Fig. 2, first column). In contrast, when the determinism in the onset epochs was captured using \( \%R(t) \), differences in the point-by-point comparisons between onset and control epochs were detected at 268–354 ms (27 points) and 232–344 ms (35 points) in O1 and O2, respectively (Fig. 2, second column) (\( P < 0.05 \) for each pair-wise comparison in each interval). No difference in \( \%R(t) \) for the sham-field-onset epochs was found (Fig. 2, third column).

Changes in the occipital signals due to field onset were detected in \( \%R(t) \) from 16 of the 17 subjects studied (Fig. 3). The conditions for detecting field-induced potentials (optimized using the onset potentials from the first three subjects) were used prospectively in all analyses. In subject 1, between 246 and 318 ms in \( \%R(t) \) computed from O2, each of the 23 points was pair-wise significant (\( P < 0.05 \) in each \( t \)-test) when compared with the corresponding points in the control epochs. The proportion of significant tests (23 of the 45 points in the interval 209–354 ms) was far too high to attribute to chance. In the sham-onset epochs, no false-positive evoked potentials occurred in O1 or O2 (< six significant tests in each time series, data not shown). In subject 2, between 288 and 318 ms (10 points) in \( \%R(t) \) computed from \( V(t) \) measured at O1, each point was significantly greater (\( P < 0.05 \)) than the corresponding point in the control. In subject 4, two intervals in \( \%R(t) \) from O1 differed from the controls; the first at 242–308 ms (21 points), where \( \%R(t) \) was greater than in the controls, and

![Fig. 2. Effect of onset of a magnetic field on brain electrical activity of a 20-year-old male. (a, b) O1, O2, respectively. First column, average value of V(t) for the onset and control epochs and point-by-point comparison-wise probability of a difference between the two curves, assessed using the paired t-test. Second column, comparable curves for \( \%R(t) \) (computed from V(t)). Third column, results for \( \%R(t) \) from the sham-field experiment. Solid line, \( P = 0.05 \). Onset (or sham-onset) and control epochs, black and gray curves, respectively. All curves are shown after use of a 30-point smoothing window.](image-url)
Fig. 3. Onset MEPs measured from occipital electrodes. Latency and duration in each subject are indicated by a bar under the time axis, which shows the location of the points in the onset epochs that differed from the corresponding control; the number of significant points is shown above the axis. Bar graphs indicate the mean ± SD. of the MEP observed in %R t (average of the significant points); black and white bars correspond to onset and control epochs, respectively (SD; not resolved at scale presented). Alpha filtering was performed in nine subjects: S1, 3 (8–10 Hz), 5, 6, 9 (O1), 11, 12, 16, 17. ND, not detected. * Three subjects used to help establish the conditions for detecting onset potentials.
the second at 325–351 ms (nine points) where it was smaller. In subjects 9–12, 16, and 17, evoked potentials were found in both occipital derivations; in subjects 9 and 17 the direction of the effect differed between the two electrodes. An onset evoked potential was not detected in subject 7.

Fig. 4. Offset MEPs measured from occipital electrodes. Latency and duration in each subject are indicated by a bar under the time axis, which shows the location of the points in %R(t) in the onset epochs that differed from the corresponding control; the number of significant points is shown above the axis. Bar graphs indicate the mean±SD of the MEP observed in %R(t) (average of the significant points); black and white bars correspond to offset and control epochs, respectively (SD; not resolved at scale presented). Alpha filtering was performed in nine subjects: S1, 2, 3, 5, 6, 8 (O1, O2 at 8–10 Hz), 11, 16, 17. ND, not detected.
Changes in the occipital signals due to field offset were detected in subjects (Fig. 4). Offset potentials were found in two of the three subjects used to help establish the conditions for detecting onset potentials.

Onset and offset potentials were detected in subjects from central and parietal derivations (Figs. 5 and 6). Responses evoked by field onset occurred in at least one derivation in 13 subjects, including subject 7, who did not

<table>
<thead>
<tr>
<th>Subject</th>
<th>Gender</th>
<th>Derivation</th>
<th>Latency</th>
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<td>C3, C4</td>
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<td>P4</td>
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<td>P3</td>
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<td>C4, P3</td>
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<td>C3, C4</td>
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<td>P4</td>
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**Fig. 5.** Onset magnetosensory potentials measured from central and parietal electrodes. Latency and duration in each subject are indicated by a bar under the time axis, which shows the location of the points in subjects in the onset epochs that differed from the corresponding control; the number of significant points is shown above the axis. Bar graphs indicate the mean ± SD of the MEP observed in subjects (average of the significant points); black and white bars correspond to onset and control epochs, respectively (SD; not resolved at scale presented). Alpha filtering was performed in seven subjects: S1, 3, 5, 7 (C, P at 8–10 Hz), 11, 15 (6–10 Hz), 17 (8–10 Hz). ND, not detected.
Fig. 6. Offset magnetosensory potentials measured from central and parietal electrodes. Latency and duration in each subject are indicated by a bar under the time axis, which shows the location of the points in TRF in the onset epochs that differed from the corresponding control; the number of significant points is shown above the axis. Bar graphs indicate the mean ± SD of the MEP observed in TRF (average of the significant points); black and white bars correspond to offset and control epochs, respectively (SD; not resolved at scale presented). Alpha filtering was performed in eight subjects: S1, 5 (8–10 Hz), 6, 8 (P3), 9 (P3, P4, and C3 at 8–10 Hz), 11 (C4, and P3 at 8–10 Hz), 16 (8–10 Hz), 17 (8–10 Hz). ND, not detected.
exhibit a response in the signals derived from the occipital electrodes (Fig. 5); responses evoked by field offset occurred in eight subjects (Fig. 6). The average effects in $\%R(t)$ (differences between stimulus and control epochs in bar graphs in Figs. 3–6, expressed as a percent of the mean of the stimulus and control epochs) were 25.1 $\pm$ 8.4% and 23.6 $\pm$ 5.0% for the onset and offset potentials.

Every test for a difference between the control and exposed epochs was also performed on the corresponding sham-exposed and sham-control epochs, employing the same conditions used to compare the exposed and control epochs. There was one case of a false positive detection (O1 in initial measurement of S2) in the 34 sham analyses (17 subjects with respect to both onset and offset of the field).

Using the method of time averaging, magneto-sensory evoked potentials (MEPs) due to either onset or offset of the field were not detected in any electrode derivation in any subject (data not shown).

Following sound onset, auditory evoked potentials were detected by time averaging, as expected, and by recurrence analysis (Fig. 7). Similar results were found in all subjects (data not shown).

**DISCUSSION**

Strong magnetic fields (~10,000 G), such as those used for transcutaneous magnetic stimulation, instantaneously activate voltage-sensitive ion channels in axonal membranes. Fields on the order of 1 G cannot do so, and their biophysical mechanism of action is still unknown. Nevertheless, they can produce electrophysiological changes in animals throughout the phylogenetic spectrum (Szabo, 1974; Semm and Beason, 1990; Dobson et al., 2002; Fuller et al., 2003). In some species, specialized receptor organs have been described. The anatomical basis for the detection of weak fields by human beings, however, has not been located. Consequently, the best evidence pres-
ently possible that human beings possess a magnetic sense consists of measurements of potentials evoked by a field stimulus. The primary objective of this study was to provide direct evidence indicating that detection of weak fields was a form of sensory transduction.

Using recurrence analysis, potentials evoked by field onset or offset or both were detected in the occipital electrodes in 16 of 17 subjects; the family-wise error rate for the decision that the subject detected the field was less than 0.05 in each of the 16 cases (Figs. 3 and 4). The potentials occurred with a latency of 209–354 ms in %R(t) (corresponding to 109–454 ms in V(t)), and consisted of statistically significant increases or decreases in %R(t), the quantifier used to capture the nonlinear determinism in V(t). Several considerations led to the conclusion that the observed effects were true post-transduction changes in brain electrical activity triggered by the magnetic stimulus, that is, MEPs.

First, an alternative explanation that the effects resulted from an interaction between the field and the scalp electrodes can be ruled out because, in accordance with Faraday’s law, such interactions begin instantaneously; in our studies they occurred within the first 30 ms after stimulus onset or offset (Fig. 1c). In contrast, the observed potentials occurred several hundred milliseconds after the stimulus, which is a typical latency for evoked potentials, for example, N200 and P300 of the auditory system (McPherson, 1996). In previous studies on rabbits, we showed that field-induced changes in the EEG were extinguished by anesthesia and by death of the animals (Marino et al., 2002), thereby directly establishing the electrophysiological origin of the changes.

Second, sensory evoked potentials are typically produced by both onset and offset of a stimulus (Campen et al., 1997; He, 2002; Takahashi et al., 2004), and both kinds of elicited responses were observed in this study (Figs. 3 and 4). Moreover, potentials are commonly seen more frequently in onset epochs compared with offset epochs (Campen et al., 1997; He, 2002; Takahashi et al., 2004), and such a differential response was also observed here (Figs. 3 and 4).

Third, inter-subject variation in latency within a reasonably well-defined range was seen, as is the case with all other known types of evoked potentials.

Fourth, although our experimental design was based on detecting potentials from the occipital electrodes, signals from other derivations were also examined. The total number of electrodes used was too small to permit localization of the origin of the potentials (Nunez and Srihivasan, 2006). However, our observation of potentials in the central and parietal electrodes strengthened our inference that the occipital potentials were MEPs, even though they may have resulted from spatial aliasing.

Finally, the family-wise error rate for a decision that a subject detected a stimulus was initially estimated at \( P = 0.047 \), and the observed error rate based on the sham analysis was 1/34 = 0.029. Thus on the basis of both a priori and a posteriori considerations, our inferences were adequately protected against chance in each of 16 subjects. The onset data from three subjects was used to help establish the conditions for the analysis, but an offset effect was found in two of those subjects. Thus potentials were detected in 15 of the 17 subjects after all conditions used in the analysis had been fixed.

It follows from all these reasons that the observed changes in brain electrical activity were true MEPs. The results therefore can be interpreted to show that human subjects possess a magnetic sense. The mechanism of this sense as well as the anatomic location at which it is mediated (see below) remains unelucidated. Further, transduction of the field (conversion into an electrical signal by a receptor) did not result in perception, as in the case of the special senses. Thus the “magnetic sense” must be understood more narrowly, similar to the chemical senses for detection of pH, O₂, and blood pressure.

The MEPs were detected when V(t) was analyzed by recurrence analysis but not when V(t) was analyzed by time averaging. Recurrence analysis is capable of detecting linear determinism as well as nonlinear determinism (which was the application for which the technique was initially devised), whereas time averaging is capable of detecting only linear determinism (stimulus–response relationships governed by linear differential equations). Thus, taking into consideration the conditions under which we observed the MEPs as well as the mathematical properties of the techniques that we used, it can be concluded that the potentials were nonlinear in relation to the applied field. Nonlinear event-related potentials have been described previously (Breakspear, 2002; Tass, 2003; David et al., 2005).

In designing the exposure apparatus, we assumed that transduction in human beings occurred in the head, as previously reported for rabbits (Marino et al., 2003a); we did not address this issue experimentally. However, it remains possible that transduction occurred elsewhere because the body of the subject was also exposed to a fringing field, although much weaker than the field applied to the head.

We expect that there was post-processing of the afferent signal that resulted from transduction of the field, and that the measured signal was the result of this processing. The situation was probably much the same as that following transduction of light, sound, or touch, as evidenced by the similarity between our latencies and those observed with the common stimuli. The observed inter-subject variation in latency (within the expected 100–450 ms interval) could have been partly due to differences in the cognitive status of the subjects (Lutz et al., 2002).

The likelihood of detecting an MEP at 100–450 ms using recurrence analysis was greater in the occipital electrodes (Figs. 3 and 4 compared with Figs. 5 and 6), confirming our earlier observation (Marino et al., 2004). We also observed earlier that when the effect of the field was averaged over 2 s (spectral analysis), stronger changes in brain activity occurred at the central and parietal electrodes (Bell et al., 1992b). Recurrence analysis and spectral analysis detected different kinds of determinism, possibly indicating that both forms were triggered by the field
and that they occurred at different locations in the brain. On the other hand, using spectral analysis, field-induced changes were found in only 20–65% of the subjects studied (depending on the details of the field), compared with 16/17 = 94% here and 100% previously (Marino et al., 2003b).

Why were the MEPs nonlinear? One possibility is that the sensory system which produced them has no evolutionary purpose. It is reasonable to view the processes responsible for the linear correspondence between the common stimuli and the responses they induce as resulting from evolution by natural selection, leading progressively to physiological systems that conferred a selective advantage because they were reliable. Conversely, in the absence of natural selection there is no process by which the phenomenon of consistency in response to a stimulus can come about. Power-freqency fields were negligible throughout the period of evolution of life on earth and became a prominent part of the environment only within the last century. They could not have served as an agent of evolutionary change, and consequently, a physical mechanism capable of producing a predictable response (a dose-related response that reliably occurs in the same direction) to fields did not develop. In this view, one possible explanation for the existence of a nonlinear human magnetic sense could be that it arose as a vulnerability in the molecular machinery chosen by evolution to mediate other sensory modalities (Nesse and Williams, 1998). Any physical realization of a sensory system for one kind of stimulus is unlikely to be completely immune to all other kinds of inputs. Magnetic phosphenes (Antal et al., 2003) and microwave hearing (Frey, 1962) are two examples of nonfunctional (from an evolutionary standpoint) sensory responsiveness.

Evolutionary considerations also point to another possible basis for a human magnetic sense. Electric and magnetic receptors that facilitate finding food, avoiding predators, and navigating in the environment occur in lower life forms (Wachtel and Szamier, 1969; Manger et al., 1995, 1996; Walker et al., 1997; Nemec et al., 2001). Vestiges of these detection systems might still exist in human beings.

CONCLUSION

In conclusion, human subjects responded to onset and to offset of 2 G, 60 Hz, by exhibiting MEPs with a latency of 109–454 ms (P < 0.05 in 16 of 17 subjects). The potentials were nonlinearly related to the stimulus and were observed by sampling the EEG signals at 300 Hz, unfolding in a five-dimensional phase space using a delay time of 5, computing the %R(t) of the signal as a function of time using a window of 30 points, smoothing the computed time series using a window of 30 points, and then statistically comparing each point in the time series with the corresponding point in the control time series, using the t-test. For subjects having a relative alpha power greater than 30%, the EEG was filtered prior to analysis.

REFERENCES


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