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Localization of electroreceptive function in rabbits

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Abstract

The detection process by which animals react to the presence of electromagnetic fields (EMFs) may be a form of sensory transduction. However, the anatomic location of signal transduction in most species is unknown. Attempts to solve this problem by applying local EMFs and registering the resulting changes in the electroencephalogram (EEG) have not succeeded because of the nonstationarity of the EEG and the insensitivity of linear methods of analysis. We approached the problem of localizing electroreception in rabbits by using recurrence quantification analysis (RQA), a novel method of nonlinear analysis designed to detect small deterministic changes in a larger signal irrespective of considerations involving stationarity. When 2.5 G, 60 Hz was applied to the entire body, increased determinism in the EEG was found in all 10 animals studied, as evidenced by statistically significant increases in two RQA quantifiers. A similar result occurred when the field was applied only to the front half of each animal, but no effect on the EEG was seen when the field was applied only to the back half. When the field was localized to the head, the effect on the determinism in the EEG was again seen. When the field was further localized to the eye, the effect did not occur. Overall, the results indicated that detection of the field occurred in cells or extracellular structures in the head, probably the brain, although the methods used did not have the resolution to discriminate between specific brain structures. Thus, our results showed that the presence of transient deterministic brain states induced by an EMF signal could be documented using dynamical analysis, thereby allowing us to infer the approximate anatomic location of the signal's transduction.

Keywords: Magnetic field; Nonlinearity; Brain electrical activity; Recurrence quantification analysis; Sensory transduction

1. Introduction

Animals exposed to electromagnetic fields (EMFs) exhibit a broad range of biological effects [1–6]. In some of the studies that reported effects on brain electrical activity, the functional changes occurred immediately upon presentation of the field [7–10], suggesting that the process by which the fields were detected ("electroreception") was a kind of sensory transduction. Under this neurogenic theory, EMF-induced changes in growth and metabolism [1–4] could be explained as indirect consequences of field exposure.

The neurogenic basis of electroreception is firmly established for some aquatic animals; in fish, the process is mediated by cells located along the lateral line, and in platypus, by cells in the bill [5,6]. For most animals, however, the nature and location of the EMF transduction process entailed by the reports of EMF-induced bioeffects remains unknown.

Electroreception could, in principle, be anatomically localized by applying EMFs at specific locations on the body and observing the resulting changes in the electroencephalogram (EEG). A major problem with this approach has been the absence of appropriate mathematical tools to analyze the EEG, which is influenced by nonlinear neuronal-feedback loops, dynamic changes in the states of individual cells, and the presence of other simultaneous inputs to the system. These phenomena combine to produce a nonstationary signal in which it is difficult to detect determinism caused by a particular stimulus, using either Fourier analysis or nonlinear methods such as Lyapunov exponents.

When complex time series like the EEG are transformed mathematically into phase space, it is sometimes possible to

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detect patterns that are inapparent in either the time or frequency domains [11]. Eckmann et al. [12] described the use of a recurrence plot to facilitate visualization of such patterns, and Webber and Zbilut [13–15] developed a method (recurrence quantification analysis [RQA]) for quantifying the recurrence plot so that the amount of determinism in a signal could be ascertained. RQA has been used to detect the onset of muscle fatigue [16], predict the occurrence of cardiac arrhythmia [17], and identify putatively different physiological states [16–20].

We approached the problem of localizing electroreception in rabbits by using RQA to measure the effect on the EEG of fields applied to various regions of the body. Our approach differed from those used previously to study the effects of EMFs on the EEG [8,21–26], principally in that RQA could capture any determinism present in the EEG, not simply linear determinism.

2. Experimental procedures

2.1. Exposure systems

Magnetic fields were obtained using multiple-turn coils of 12-gauge magnet wire. A coaxial configuration of four square coils, each 66 cm on a side, was used to produce full-body exposure to a field that was homogeneous to within 5% throughout the region occupied by the rabbit. The outer coils (85 turns each) were ± 33.4 cm from the unit's centerline; the inner coils (35 turns each) were at ± 8.5 cm. In some experiments, the magnitudes and phases of the coil currents (3–8 A, depending on the experiment) were chosen such that the two halves of the rabbit's body were exposed to fields having predetermined differences (Figs. 1 and 3).

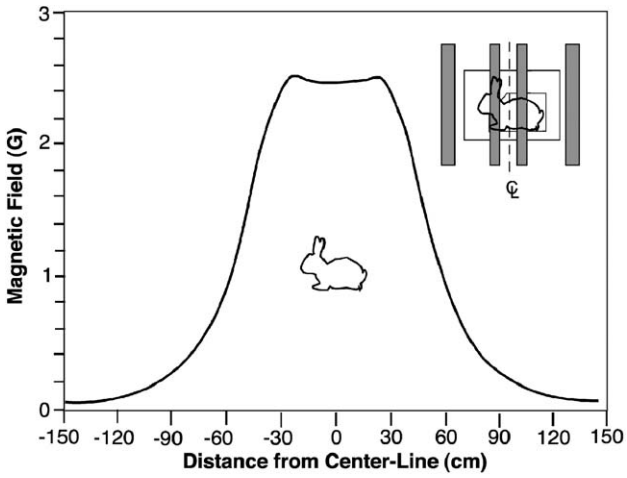

Fig. 1. Magnetic field used for full-body exposure. The coils (shown in a side view as shaded bars) were energized to produce a homogeneous field in the region occupied by the rabbit (drawn approximately to scale).

Localized exposure of the brain was produced using a pair of 14-turn coils, each 5 cm in diameter and located 9 cm apart (2.86 A) (Fig. 6). Localized exposure of the eye was produced using a 24-turn coil, 2 cm in diameter (1.45 A) (Fig. 9). The experiments were performed at or near 2.5 G, 60 Hz, because earlier work had shown that this field consistently altered the EEG in the rabbits [27]. All coil fields were calculated using commercial software (MF3D, ERM, Pittsburgh, PA), and measured with a three-axis magnetic field sensor (Bartington MAG-03, GMW, Redwood City, CA).

The four-coil unit produced no detectable change in temperature at the location of the rabbit (<0.01 °C). The circular coils produced temperature changes of 0.1–0.2 °C at the location of the rabbit. As a control, coils were wound such that the current flowed in opposite directions in adjacent turns; when energized in the same way as the conventionally wound circular coils, the control coils produced the same heat as the conventionally wound coils, but no field. The rise times of all the coils were <1 μs.

During an experiment, the rabbit was restrained in an acrylic box, which was positioned inside a light-tight wooden box to minimize environmental influences and standardize the rabbit's sensory environment. For exposure using the four-coil unit, the wooden box was centered in the unit such that its axis and the rabbit's rostral–caudal axis were parallel. To produce localized exposure, the circular coils were positioned at appropriate locations inside the wooden box. The magnetic field was a subliminal stimulus as judged by the absence of any somatic response when the field was switched on or off; presentation of the field was not accompanied by any sensory cues to the rabbit. The average geomagnetic field at the location of the rabbit was 305 mG, 22.6° below the horizontal. The geomagnetic component along the direction of the 60-Hz field was 239 mG.

2.2. Animal procedures

Five female and five male New Zealand rabbits were used in the study. All animal procedures were approved by the Institutional Animal Care and Use Committee. The EEG was recorded over the occipital region, which was under the easily palpable suture of the parietal and interparietal cranial bones. The indifferent and ground electrodes were respectively 2.5 cm and 5 cm more rostral. The electrodes (0.5 cm in diameter) were attached to the shaved scalp using conducting paste (EC2, Grass, Quincy, MA); the impedance (1–3 kΩ) was measured before and after each experiment (EZM 5, Grass).

The EEG was measured using an amplifier capable of resolving 0.1 μV (Model 4400, Nihon Kohden, Irvine, CA). The signal was filtered to pass 0.3–35 Hz, amplified, digitized at 512 Hz (12-bit), and stored on a hard drive.

Multiple independent experiments were performed on each rabbit to allow a determination of its ability to detect the fields produced by the various coil arrangements, as assessed on the basis of deterministic changes in the EEG. A trial consisted in the application of the field for 2 s (*E* epoch), followed by a field-free period of 5 s. At least 60 trials were run on each rabbit (7 min), during which the EEG was measured continuously. The EEG signal from the last 2 s of each trial was used as the control (*C* epoch) for the corresponding *E* epoch. The signal from the 2 s proceeding the *C* epoch was defined as the sham (*S* epoch). Both the *C* and *S* epochs were control epochs in the sense that the field was not applied during either interval. We used the *C* epoch for comparison with the *E* epoch to assess the effect of the field. Additionally, we compared the *S* and *C* epochs to evaluate the possibility that any positive results might be attributable solely to our statistical method. A minimum of 60 trials was run. The EEG was measured continuously during an experiment, commencing 5 min prior to initiation of the trials.

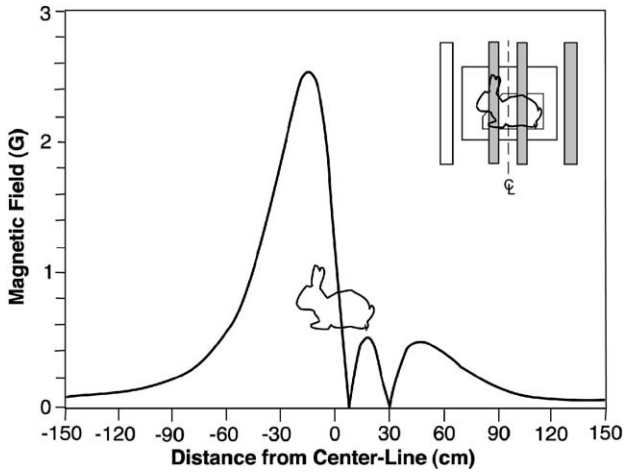

Fig. 3. Magnetic field used for half-body field exposure. The coils were energized (shaded) to maximize the difference in average field between the halves of the body. For exposure of the cranial half-body region, the rabbit was positioned in the coil unit as shown. For exposure of the caudal region, the box containing the rabbit was reversed (drawn approximately to scale).

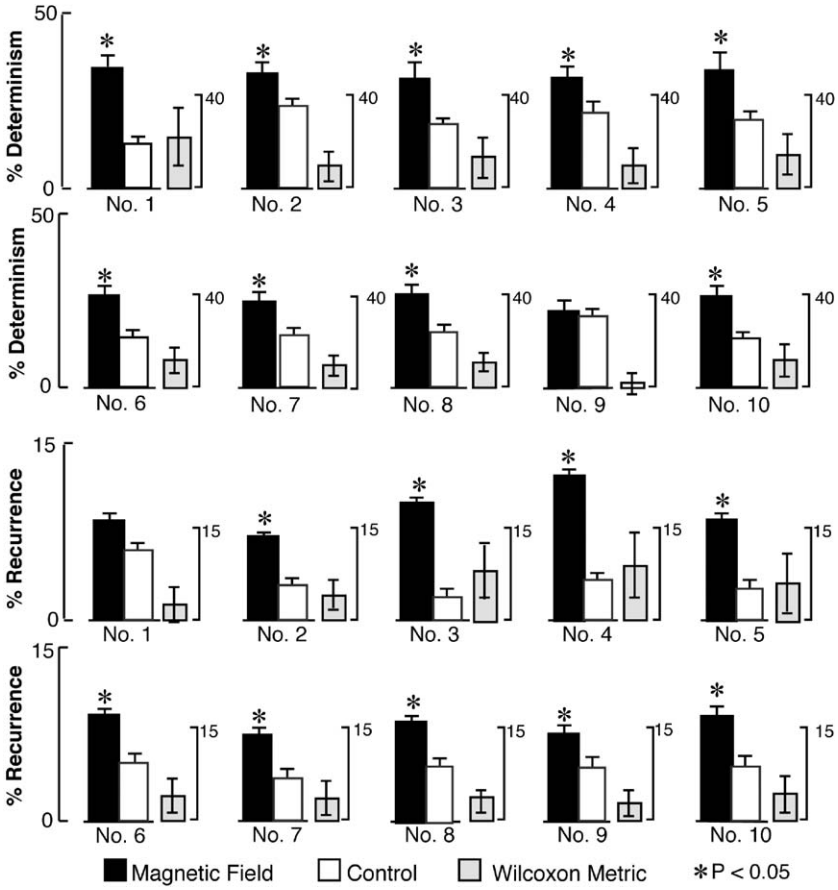

Fig. 2. Effect of full-body exposure to 2.5 G, 60 Hz, on the EEG of female (Nos. 1–5) and male (Nos. 6–10) rabbits (see Fig. 1). For each rabbit and each quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values (± S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

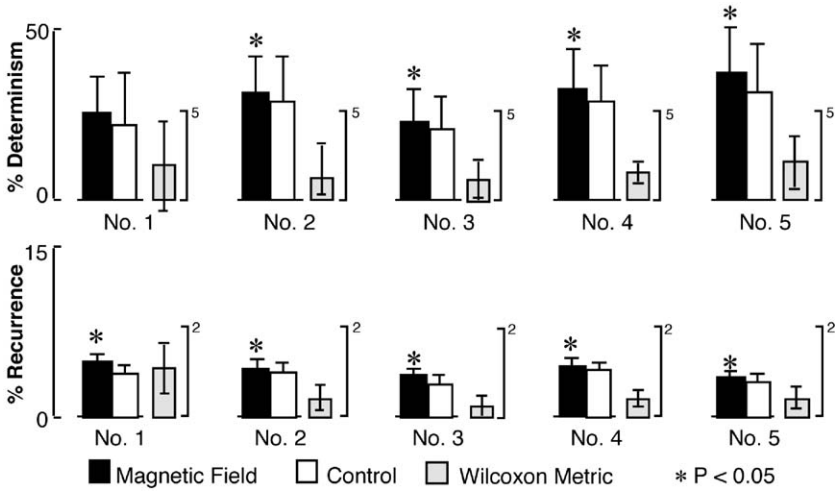

Fig. 4. Effect of exposure to 60-Hz magnetic field such that the cranial and the caudal half-body regions were exposed to 2.2 ± 0.6 and 0.5 ± 0.3 G, respectively (see Fig. 3). For each rabbit and quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values ($\pm$ S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

At the conclusion of the experiments, the rabbits were euthanized by intravenous injection of pentabarbitol (100 mg/kg).

2.3. Analysis

Movement artifacts were identified by inspection of the EEG while blinded to the type of epoch in which they occurred, after which the trials that contained the artifacts were removed from the recorded voltage; movement artifacts occurred approximately equally in exposed, control, and sham epochs. The resulting scalar time series, S, was embedded in a five-dimensional phase space using a time delay of 1 [28]; the values were chosen because they resulted in the most sensitive characterization of the EEG, as determined during preliminary studies. The local recurrence plot was obtained from the state vector $\boldsymbol{X}=(S_t, S_{t+1}, S_{t+2}, S_{t+3}, S_{t+4})$ by plotting a point in two-dimensional space at the location addressed by (i,j) whenever $\boldsymbol{X}_j$ was near $\boldsymbol{X}_i$ [12]. Two states were defined as near when they were within a five-dimensional hypersphere having a radius less than 15% of the minimum radius such that all points were near. The plots were quantified using percent recurrence (%R) and percent determinism (%D),

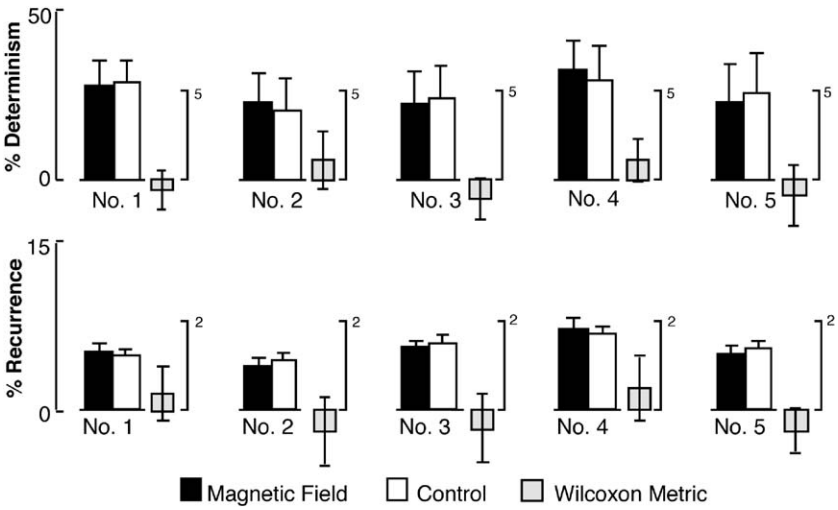

Fig. 5. Effect of exposure to 60-Hz magnetic field such that the cranial and the caudal half-body regions were exposed to 0.5 ± 0.3 and 2.2 ± 0.6 G, respectively (see Fig. 3). For each rabbit quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values ($\pm$ S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

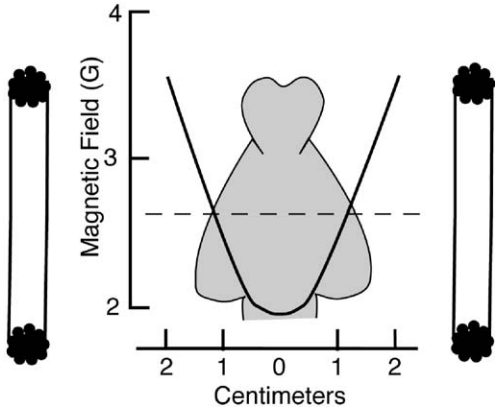

Fig. 6. Magnetic field used for exposure of rabbit brain (shaded outline). The field (averaged over a circular area in the sagittal plane 4 cm in diameter centered on the coil axis) is shown as a function of distance from the mind-point between the generating coils. The average field in the brain (assumed to be at −1.5 to 1.5 cm) was 2.5 ± 0.3 G, 60 Hz. Common axis of coils is shown as a dashed line. Drawn approximately to scale.

defined respectively as the number of recurrent points divided by the possible number of recurrent points, and the number of recurrent points located on lines parallel to the main diagonal of the plot divided by the number of recurrent points [13–16]. Calculations of %*R* and %*D* were carried out using software provided by Webber [13] and independently verified using a custom code (Matlab, Mathworks, Natick, MA); the parameters radius and line were set to 15 and 2, respectively.

2.4. Statistics

In each experiment, the first five trials were discarded and the next 50 artifact-free trials were used to compare the values of the nonlinear quantifiers, using the Wilcoxon signed-rank test. The data are presented in terms of the mean ± S.D. of %*R* and %*D*, and the mean ± 95% confidence limits of the Wilcoxon statistic (MINITAB, Minitab, State College, PA). The statistic was presented for each comparison to more clearly illustrate the ability of the test to detect small differences between exposed and control conditions. The quantifiers were regarded as independent planned comparisons, and therefore no corrections were made even though two tests were performed on each rabbit in each experiment. The level of significance was set at $P < .05$.

3. Results

Initially, Rabbit No. 1 was exposed to a full-body field (Fig. 1), and the EEG was analyzed to ascertain the conditions that maximized discrimination between the *E* and *C* epochs. Corresponding segments of the EEG in the *E* and *C* epochs were systematically compared by unfolding them in phase space, calculating %*D* from the recurrence plot of each epoch segment, and comparing the results using the Wilcoxon signed-rank test to identify the signal segment that was most responsive to the field. We found that a 250-ms segment ("window") centered at 250 ms after commencement of application of the field yielded the lowest *P*'s for *E* vs. *C* when $P > .05$ for *S* vs. *C*. The result for %*D* was 34.6 ± 3.4% for the *E* segments centered at 250 ms with a width of 250 ms, compared with 12.8 ± 1.9% for the controls (5.25 s, 250 ms, $P < .05$); the %*D* in the sham segments, 12.1 ± 1.9%, did not differ from the controls. The %*R* values were 8.6 ± 0.4%, 6.1 ± 0.5%, and 5.8 ± 0.3% for the exposed, control, and sham segments, respectively.

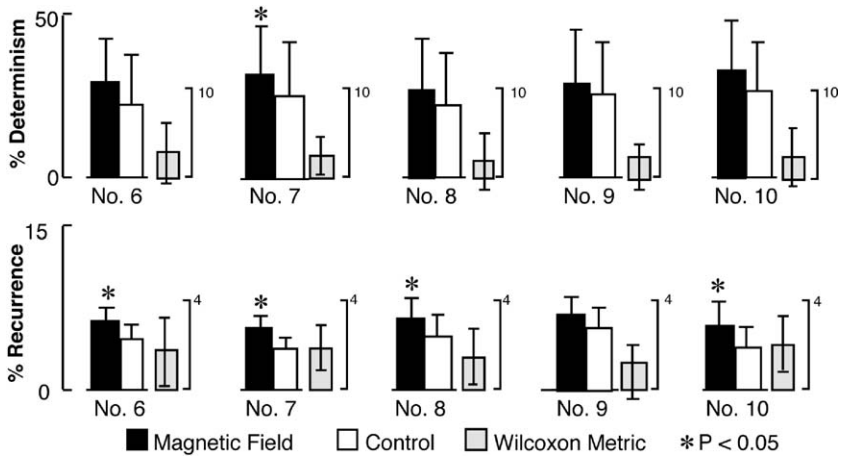

Fig. 7. Effect of exposure of the brain to 2.5 ± 0.3 G, 60 Hz ($n = 5$) (see Fig. 6). For each rabbit and quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values (± S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

The window width and location developed for Rabbit No. 1 were applied prospectively to four additional female and five male rabbits that were similarly exposed to a full-body field, and statistically significant differences in %*D* and %*R* were found in all cases, with two exceptions (Fig. 2). There were no cases of a false-positive result, as assessed by comparing the sham and control segments (data not shown).

One explanation for the results in Fig. 2 was that electroreception occurred throughout the body as, for example, in somatosensory transduction. Alternatively, electroreception might have been localized, such as for the special senses. To help choose between the two possibilities, we first determined the currents and phases under which the coils would produce the maximum average difference in half-body exposure, subject to the constraint that the maximum field would be 2.5 G. Then, each of five rabbits was positioned in the field so that the cranial half was exposed to the higher field (2.2 ± 0.6 G), and the caudal half was exposed to the lower field (0.5 ± 0.3 G) (Fig. 3). Exposure under these conditions resulted in effects in each case when the EEG was analyzed as previously (Fig. 4), with one exception; there were no false-positive results (data not shown). When the experiment was repeated with the cranial half in the low-field region and the caudal half in the high-field region, no effect on the EEG was observed (Fig. 5).

The possibility of a direct effect on the brain was evaluated in five rabbits, using a pair of coils positioned beside the head so that the field in the brain was 2.5 ± 0.3 G (Fig. 6). Significant effects on the EEG were found (Fig. 7). The effects were not seen when the experiment was repeated using coils that generated no field but the same amount of heat as the coils used previously (Fig. 8). In both experiments, the previous window parameters were used, and there were no false-positive results (data not shown).

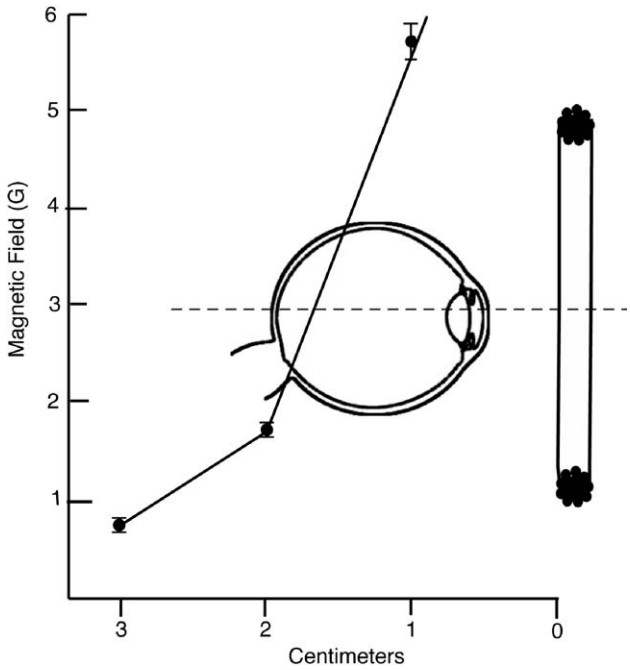

Fig. 9. Magnetic field used for exposure of rabbit eye. The field was produced using one coil (shown on the right). The field (averaged over a circular area in the transverse plane 1 cm in diameter centered on the coil axis) is shown as a function of distance from the coil. The average field over the retina (assumed to be at 1.5–2 cm) was 2.8 ± 0.5 G, 60 Hz. Drawn approximately to scale.

Additional experiments were performed to test the hypothesis that field transduction occurred in the eye, using the 2-cm coil positioned 0.5 cm from the right eye (Fig. 9). No effect on the EEG was seen (Fig. 10).

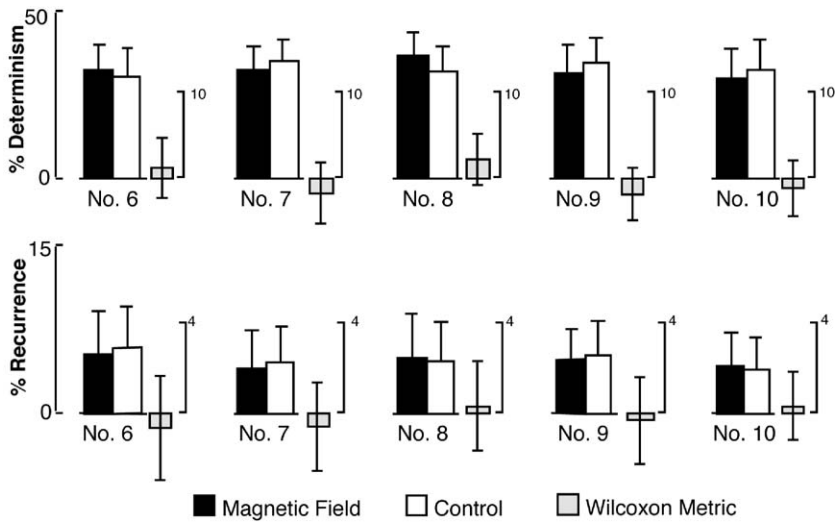

Fig. 8. Sham exposure of the brain of rabbits (temperature control). The current through the coils was identical to that used in Fig. 7, but it resulted in no detectable magnetic field (< 0.01 G). For each rabbit and each quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values (± S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

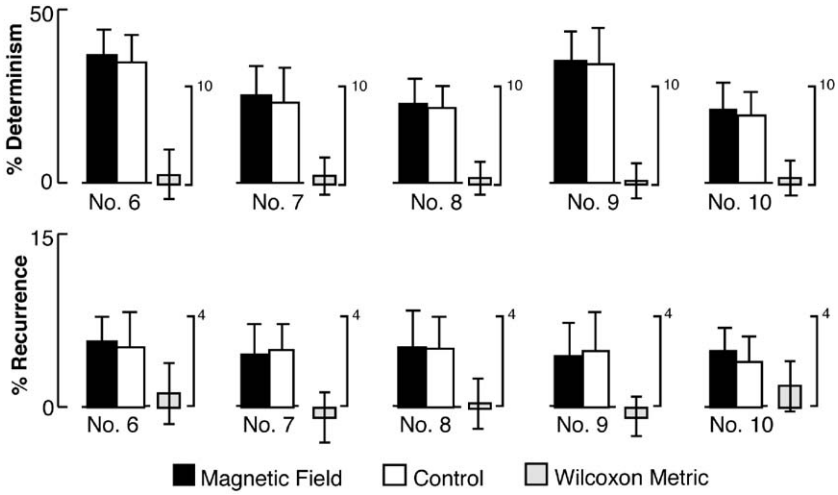

Fig. 10. Effect of exposure to a 60-Hz field of 2.8 ± 0.5 G, averaged over a transverse plane through the retina. For each rabbit and quantifier, the exposed and control EEG epochs were compared using the Wilcoxon signed-rank test ($n = 50$ trials). A 250-ms segment of the data from each epoch (centered at 250 ms from the beginning of the epoch) was evaluated. The average values (± S.D.) of the quantifiers are shown. The average and 95% confidence limits of the test metrics are shown for each rabbit in the third bar.

After the animals were euthanized and cessation of heart activity was verified, each rabbit was exposed to a full-body field of 2.5 G, and the input signal to the amplifier was analyzed as previously to evaluate the possibility of passive field interactions with the electrodes. We found that the RQA parameters were essentially zero, and independent of the presence of the field (data not shown).

4. Discussion

We studied the question whether electroreception in the rabbit, assumed to be a neurogenic process, was generalized or localized. In each of 10 rabbits, the EEG measured while the animal was exposed to a 2.5-G full-body field differed significantly from the EEG measured in the absence of the field, as assessed statistically on the basis of changes in the nonlinear quantifiers of determinism in the signal (Fig. 2). The results were not due to some unrecognized aspect of our analytical method because there were no false-positive results when the same method was used to compare two control epochs (*S* vs. *C*). The effect of the field was undoubtedly physiological in origin because no changes were seen in the input signal to the EEG amplifier when the field was applied to the rabbits after they had been euthanized. In addition, the observed delay of 125 ms between application of the field and the onset of the change in the EEG also indicated that the change was physiological in origin. We infer, therefore, that the field was transduced somewhere in the body, leading to the observed changes in the EEG, as expected under the neurogenic theory.

The possibility that transduction occurred throughout the body was evaluated by applying a field of comparable strength to only the front or back half of the animal in separate experiments, while minimizing the average field applied to the other half of the animal (about 0.5 G). In the former experiment, we found an effect due to the field (Fig. 4); in the latter experiment, no effects on the EEG were found (Fig. 5). Taken together, the two experiments showed that field detection occurred somewhere in the front half of the animals.

When the brain was exposed to an average field of 2.5 G (Fig. 6), the EEG was altered in four of five rabbits studied (Fig. 7); the effect could not be explained on the basis of heat produced by the coils (Fig. 8). The possibility that the transduction was mediated at least partly by retinal cells was evaluated by exposing that region, using a coil that produced an average field of 2.8 G at the retina, and a much lower field at more proximal locations (Fig. 9). Application of the field to the eye did not affect brain activity (Fig. 10), suggesting that the photodetectors in the eye were not the locus of transduction of the field.

Taken together, the results (Figs. 2, 4, 5, 7, 10) can be interpreted to indicate that EMF transduction occurred somewhere in the head, probably the brain, although the methods used did not permit discrimination between specific brain structures that could have been the site of transduction. Central neurons interact strongly via synapses, and neuronal processes are often arranged in parallel, thereby enhancing ephaptic interactions. It is possible that the dense interconnectivity in the rabbit brain amplified transmembrane potential changes induced by the EMF, thereby altering the EEG. Other explanations are also possible. For example, the conditions of exposure and the anatomy of the rabbit's head were consistent with the possibility that transduction occurred in the hair cells of the ear (where the average field was greater than 2.5 G). We did not address the problem of identifying the particular cell or process by which the field was actually detected.

In studies on hippocampal slices [29–33], low-frequency fields produced immediate changes in electrical activity. The field used in the present study was 1–4 orders smaller than that induced in the brain slices. One possibility, among many, is that the brain electroreceptors inferred in the present study were located in the hippocampus. Another possibility is that more sensitive electroreceptors elsewhere in the brain could have been responsible for the effects reported here.

In summary, our results showed that the presence of transient deterministic brain states induced by an EMF signal could be documented using dynamical analysis, thereby allowing us to infer the approximate anatomic location of the signal's transduction.

Acknowledgements

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