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Abstract

The effects of 1.5- and 10-Hz electromagnetic fields (EMFs), 0.2—0.4 gauss, on the intrinsic electrical activity of the human brain at these frequencies was studied. Each of 19 subjects exposed for 2-sec epochs exhibited altered brain electrical activity at the frequency of the EMF during the time of stimulation, as determined by spectral analysis of the electroencephalogram. Since brain activity at specific frequencies could be altered by applied EMFs, the results suggest that it may be possible to use EMFs to determine whether particular intrinsic frequencies subserve specific physiological or behavioral responses.

Key words: EEG; Electromagnetic field; EMF; Magnetic field; Brain; Power spectra; Fourier transformation

1. Introduction

Human brain electrical activity manifested in the electroencephalogram (EEG) consists primarily of frequencies below 20 Hz (Niedermeyer 1987). The question whether these frequencies directly mediate behavioral and physiological effects, or are epiphenomena, has not been resolved (Adey 1970). Externally applied electromagnetic fields (EMFs) may provide a means to alter specific intrinsic frequencies by driving or otherwise interacting with physiological oscillations within the brain, thereby permitting direct assessment of their biological significance in both normal and pathological processes.

EMFs at 2—12 Hz have been reported to affect behavior in man and animals (Hamer 1968; Gavalas et al. 1970; Gavalas-Medici and Day-Magdaleno 1976; Wever 1987), and several animal studies suggest that EMFs can specifically alter brain electrical activity. A relative peak in spectral power was seen in monkeys at the frequency of the EMF (Gavalas et al. 1970), and EMFs amplitude-modulated at 3—14 Hz were reported to reinforce the occurrence of spontaneous rhythms in cats when the EMF frequency was tuned to the frequency of the intrinsic rhythm (Bawin et al. 1973). EMFs at 5—10 Hz altered intrinsic EEG power in rabbits at the frequency of stimulation (Bell et al. 1992a).

EMFs at 35—40 Hz (Bell et al. 1991) and 60 Hz (Bell et al. 1992b) can non-specifically alter human brain activity, but the effects of EMFs on BEG power at the frequency of stimulation have not previously been described. We report here that human subjects exposed to EMFs exhibited changes in the BEG at the fre quency of stimulation.

2. Methods

EMF exposure

Magnetic fields were produced using a pair of coils, each 130 cm in diameter and consisting of 250 turns of copper wire; the coils were maintained parallel and separated by 65 cm (the Helmholtz condition) using a wooden frame. The coil current was obtained from a signal generator (Model 182A, Wavetek, San Diego, CA), and amplifier (Model 7500, Krohn-Hite, Avon, MA), and controlled by a computer-based timing cir cuit. The subjects sat between the coils (saggital plane

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Table 1
Subject Characteristics and Exposure Conditions
The subjects were clinically normal at the time they entered the study except for those with the listed complaints

Subject (age, sex)			Magnetic field (Gauss)	Frequency (Hz)	Complaint
1	22	F	0.2—0.8	10	None
2	30	M	0.2—0.8	10	None
3	31	F	0.2—0.8	10	None
4	30	M	0.2—0.8	10	None
5	25	F	0.2—0.8	10	None
6	62	M	0.2—0.8	10	None
7	27	M	0.2—0.8	10	None
8	26	F	0.2—0.8	10	Seizures
9	28	F	0.2—0.8	10	Seizures
10	21	M	0.2—0.8	10	Numbness
11	31	F	0.2—0.8	1.5	None
12	30	M	0.2—0.8	1.5	None
13	22	F	0.2—0.8	1.5	None
14	31	F	0.2—0.8	1.5	None
15	35	M	0.2—0.8	1.5	None
16	30	M	0.2—0.8	1.5	None
17	34	M	0.2—0.8	1.5	Seizures
18	51	M	0.2—0.8	1.5	Dizziness
19	42	M	0.2—0.8	1.5	Seizures

perpendicular to the coil axis) on a wooden chair in a dark room with their eyes closed. The head and upper chest were within a magnetic-field region that was uniform to within 5% of its predetermined value (within 20% when the thorax and pelvis are included); the field at the feet was about half of that at the head. The average background 60-Hz magnetic field was about 0.1 mG.

The equipment that powered the coils and recorded the EEG was located 15 m from the room occupied by the subjects. The room was partially soundproofed, but occasional sounds that occurred irregularly in an adjacent corridor could be heard in the room. There were no visual, auditory, or tactile cues to the subjects that indicated the presence of the magnetic field.

Subjects

Thirteen normal (non-symptomatic) subjects were chosen from the general population, and 6 symptomatic subjects were selected from among those with neurological complaints who underwent a clinical EEG as a diagnostic procedure (Table 1). The symptomatic subjects were identified for possible inclusion by an EEG technician, who noted the presence of a well-developed occipital alpha rhythm during the clinical EEG. Subjects having this finding were asked to participate in the study, and those consenting and willing to remain in the laboratory area after completion of their clinical EEG, were utilized. No subject who had a seizure during the clinical EEG, or who had persistent focal or generalized slowing was used. All clinical EEGs were normal except for that from one subject (No. 17, Table 1), which exhibited intermittent, dysrhythmic activity, as later interpreted by the neurologist reading the clinical tracings and blinded to whether the subject was a study participant. The results were analyzed without respect to age, sex, or the presence of symp toms.

The study was approved by the Institutional Review Board for Human Research of the Louisiana State University Medical Center.

EEG measurement

Gold-plated surface electrodes 1 cm in diameter (Grass Instrument Co., Quincy, MA) were placed at C_3, C_4, P_3, P_4, O_1, and O_2 (10—20 system); to facilitate quantitative comparisons, the electrodes were referred to a common point (linked ears were chosen for convenience), and the ground was placed on the forehead. Electrode impedances were measured before and after each recording, using an electrode impedance meter (Grass Model EZM SA, Grass Instrument Co., Quincy, MA); typically, the impedances were 2—3 kΩ.

The EEG was filtered to pass 0.3—35 Hz, and then the signal was split and simultaneously recorded on an electroencephalograph (Model 6, Grass Instrument Co., Quincy, MA) and stored on a 40-Mbyte hard drive after sampling at 200 Hz; the stored data was analyzed on a mainframe computer using commercial software (SAS Institute, Inc., Austin, TX). Trials containing obvious movement artifacts were identified on the written record, and the corresponding digitized data was deleted.

The aim was to determine whether brief exposure to an EMF having a frequency of an intrinsic brain rhythm produced an alteration in the FEG at the frequency of stimulation during the application of the field. Characteristic frequencies within the A *(1.5* Hz) and a (10 Hz) bands were therefore chosen for study, and fields ≤ 0.8 gauss were used to avoid the production of magnetophosphenes, which can occur at stronger fields.

A method of analysis was developed to control for Faraday-type induction which occurs in the human head in the presence of a magnetic field. Since the inductive signal is proportional to the rate of change of magnetic flux (Serway, 1990), by applying a relatively strong magnetic field (0.8 gauss rms) and measuring the induced signal under the assumption that the contribution of the EEG was negligible, the strength of the induced signal at lower fields could be determined on the basis of proportionality.

Consider a pair of surface electrodes attached to a volume electrical conductor (Fig. lA). Application of $B_0(f_0)$, a uniform magnetic field at frequency f_0, results in an inductive signal superimposed on the noise (B = 0) signal. The electrode voltage is amplified and Fourier-transformed to produce the power spectrum. Let $P(B_0,f_0)$ be the coefficient of the power spectrum

Fig. 1. Characterization of the signal induced in a volume conductor by a magnetic field. B_0 induces a voltage that is amplified and Fourier-transformed. $P(B_0,f_0)$ is the power at f_0 measured during application of B_0; $P_0(f_0)$ is measured in the absence of the field. Measurements were made during application of B_0, $2B_0$ and $4B_0$ in consecutive time intervals (blocks). For human subjects, the order of field presentation was varied randomly from subject to subject. Within each block the field was on for 2 sec and off for 5 sec (the B_0 block is illustrated). C~ and E~ are the control and exposed epochs in the nth trial (n = 50). For human subjects, the EEG at f_0 during the exposed epochs at B_0 and $2B_0$ was compared with the power in the corresponding control epochs, after the former were corrected for the inductive signal (determined using $4B_0$).

at f_0; it can be expressed as the sum of $P_{B_0}(f_0)$ and $P_0(f_0)$, the contributions due to the field and the noise, respectively (Oppenheim and Schafer 1975). $P_{B_0}(f_0)$ can therefore be determined from the difference in Fourier coefficients between field and no-field epochs.

$$P_{B_0}(f_0)) = P(B_0,f_0) — P_0(f_0) \quad (1)$$

For a sinusoidal magnetic field, the inductive signal is directly proportional to the product of frequency and field strength; the Fourier coefficient of the induced signal is therefore proportional to the product of the squares of frequency and field strength (Serway 1990). Differences defined as in Eq. 1 but formed at $2B_0$ and $4B_0$ therefore correspond to 4 times and 16 times the difference observed at B_0, respectively;

$$4P_{B_0}(f_0) = P(2B_0,f_0) — P_0(f_0) \quad (2)$$

$$16P_{B_0}(f_0) = P(4B_0,f_0) — P_0(f_0) \quad (3)$$

If the volume conductor is the head of a human subject, and the voltage is the EEG measured from any pair of electrodes during application of the field, the Fourier coefficient corresponding to an exposed epoch can formally be considered to be the sum of three contributions: the inductive signal, the BEG that would be present if no magnetic field were applied, and a putative effect of the field on the EEG. When modified to allow for an effect of the field (μ_1 and μ_2 at B_0 and $2B_0$, respectively), Eqs. 1 and 2 can be rewritten

$$[P(B_0,f_0) — P_{B_0}(f_0)] — P_0(f_0) + \mu_1 = 0 \quad (4)$$

$$[P(2B_0,f_0) — 4P_{B_0}(f_0)] — P_0(f_0) + \mu_2 = 0 \quad (5)$$

where $P_0(f_0)$ now represents the sum of the signals due to the control EEG and noise.
If

$$[P(B_0,f_0) — P_{B_0}(f_0)] — P_0(f_0) \neq 0 \quad (6)$$

it follows that $\mu_1 \neq 0$. Similarly, if

$$[P(2B_0,f_0) — 4P_{B_0}(f_0)] — P_0(f_0) \neq 0 \quad (7)$$

then $\mu_2 \neq 0$. Thus, an effect of the magnetic field (B_0, $2B_0$) on EEG activity (μ_1, $\mu_2 \neq 0$) can be inferred from a comparison between $P_0(f_0)$ and the EEG power measured during application of the field but corrected for the inductive signal ($[P(B_0f_0) — P_{B_0}(f_0)]$, $[P(2B_0,f_0) — 4P_{B_0}(f_0)]$). If, after subtracting the inductive signal, the EEG power were the same as that in the absence of the field, it would follow that the field had no effect on the EEG; otherwise, the null hypothesis must be rejected.

A trial consisted of the presentation of the magnetic field for 2 sec, followed by an interstimulus period of 5 sec (Fig. 1B). $P(B_0,f_0)$ and $P(2B_0,f_0)$ were computed for each exposure epoch in a series of 50 trials; $P(f_0)$ was determined from the 2-sec intervals immediately preceding each of the exposure epochs. Since EEG power was generally negligible in comparison with the inductive signal caused by $4B_0$, $P_{B_0}(f_0)$ could best be estimated from Eq. 3 (highest signal-to-noise ratio). $[P(B_0,f_0) — P_{B_0}(f_0)]$ and $[P(2B_0,f_0) — 4P_{B_0}(f_0)]$ were each compared with $P_0(f_0)$ using the Wilcoxon signed rank test (Pfurtscheller and Aranibar 1977) at a significance level of $P < 0.05$. If statistically significant differences were found in the EEG from a particular electrode, it was inferred that the subject responded to the presence of the field by exhibiting altered brain electrical activity at f_0. The analysis was performed for each of the 6 electrodes (referred to linked ears) for each subject, using 50 trials with $B_0 = 0.2$ gauss and 50 additional trials with $2B_0 = 0.4$ gauss. The overall procedure is depicted in Fig. 1.

Saline, tap water and a melon were used to evaluate the accuracy of the measuring system and the validity of the procedure for determining the effect of field strength on the magnitude of the inductive signal. For measurements in saline and tap water, 6 measuring electrodes were attached to the curved surface of a plastic cylinder (20 cm in diameter), which was immmersed in either normal saline or tap water. The electrodes were placed in one plane, with spatial separations and locations that approximately corresponded

Table 2
Evaluation of the method of measuring spectral power during application of a magnetic field
Fields of 0.2, 0.4 and 0.8 gauss at either 1.5 or 10 Hz were applied to normal saline, tap water, and a melon, as depicted in Fig. 1. The power at the frequency of the applied field (averaged over 50 2-sec epochs) was computed for electrodes located to correspond to the indicated standard (10—20 system) EEG electrodes. The measurements obtained at 0.2 and 0.4 gauss are expressed as a percent difference relative to that obtained at 0.8 gauss, taking into account the expected proportional differences between different field strengths. The results are expressed in terms of the percent difference (PD) between the observed (O) and expected (E) values: PD = 100X |(O—E)/E|. Individual measurements were repro ducible to within 5%. Electrode resistances 0.1—0.2 kG, 1—2 kG, 2—3 kG for saline, tap water, and melon, respectively

Electrode	Percent difference in spectral power (%)									
	1.5 Hz				10Hz					
	Saline		Tap		Saline		Tap		Melon	
	0.2 G	0.4 G	0.2 G	0.4 G	0.2 G	0.4 G	0.2 G	0.4 G	0.2 G	0.4 G
C_3	5.1	6.6	10.9	3.0	6.1	2.3	7.6	4.0	15.0	4.2
C_4	2.3	0.0	16.1	7.8	9.7	3.7	7.8	7.4	5.6	5.6
P_3	7.1	14.3	7.0	0.0	10.8	8.7	6.6	3.8	11.2	21.5
P_4	8.7	6.5	3.3	0.8	6.1	2.3	5.3	2.9	18.5	1.6
O_1	18.8	2.2	4.8	7.4	16.9	7.7	2.2	9.1	13.7	7.7
O_2	1.1	2.5	3.1	7.4	8.3	3.1	8.0	4.0	3.8	4.0
Mean±SE	6.7+1.1%						7.4±1.0%			

to the central, parietal, and occipital electrodes on the human subjects. All measurements were referred to a single reference electrode, located anteriorly, and in the same plane as the measuring electrodes. The locations of the electrodes on the melon were also similar to the electrode locations on the human subjects.

3. Results

The contribution of the inductive signal in each of the passive conductors was found by applying 0.8 gauss and using Eq. 3, and the value thus determined (the expected value) was compared with those found during application of 0.2 and 0.4 gauss, respectively, using Eqs. 1 and 2 (the observed values). The inductive signal at 0.2 and 0.4 gauss was essentially as expected on the basis of the assumption of a proportional response (Table 2); the mean percent differences for the 3 conductors were 7.4 and 6.7% at 10 Hz and 1.5 Hz, respectively. Thus, the response of the measuring system to induced signals in passive conductors was proportional to the square of the field strength and frequency, as expected.

Typical results obtained from a human subject using 10 Hz are shown in Table 3, which lists the mean 10-Hz power from the control and exposed epochs from subject No. 1, after correction for the inductive signal. The 10-Hz power during the control epochs measured from C_3 and C_4 was 454 and 768 μV^2, respectively; during application of the field the corresponding values were 1514 and 3756 ALV^2, and each of the distributions differed significantly from their corresponding controls as determined by the Wilcoxon test. Significant differences were also found at the other electrodes during application of 0.2 gauss, and at the C and P electrodes during application of 0.4 gauss. The results for all 10 subjects exposed to the 10-Hz field are summarized in Fig. 2, which gives the observed significant changes in spectral power caused by the magnetic field, expressed in terms of the magnitude of the percent difference between the observed and expected values. The 10-Hz field produced significant effects in the 10-Hz EEG power from 1 or more electrodes in 8

Table 3
EEG spectral power at 10 Hz in subject no. 1 exposed to 10-Hz magnetic fields
The absolute value of the mean ± SE are listed for the Control epochs [P_0(10)] and for the exposed epochs after correction for the inductive signal [P(0.2,10)—P_{02}(10)], and IP(0.4,10)—$4P_{02}$(10)]. The distributions were compared using the Wilcoxon test

Electrode	Spectral power at 10 Hz (μV^2)					
	0.2 gauss			0.4 gauss		
	Control	Exposed	*P*	Control	Exposed	*P*
C_3	454± 82	1514±350	(0.002)	407± 55	15321±1004	(0.0001)
C_4	768±162	3756±418	(0.0001)	443± 68	14820±1119	(0.0001)
P_3	704± 164	307±139	(0.0001)	453± 68	522± 529	(0.04)
P_4	1282±327	603±130	(0.0001)	822±174	2004± 376	(0.0001)
O_1	793±101	2326±485	(0.0001)	1100±222	604±1592	(0.47)
O_2	797±127	6907±648	(0.0001)	2038±643	2287±2368	(0.99)

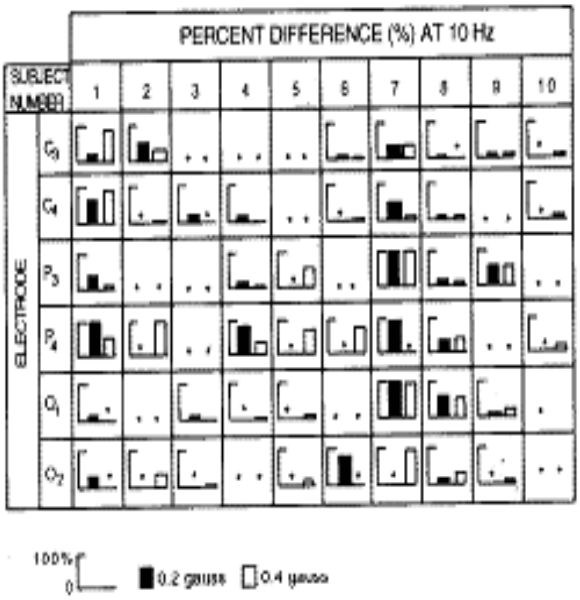

Fig. 2. Change in spectral power of human subjects at 10 Hz during application of a 10-Hz magnetic field. The results are expressed in terms of the percent difference between the observed and expected power. The solid and open bars correspond to 0.2 and.O.4 gauss, respectively. Data from nonsignificant comparisons are omitted (indicated by asterisks). Percent differences greater than 100% are shown as 100%.

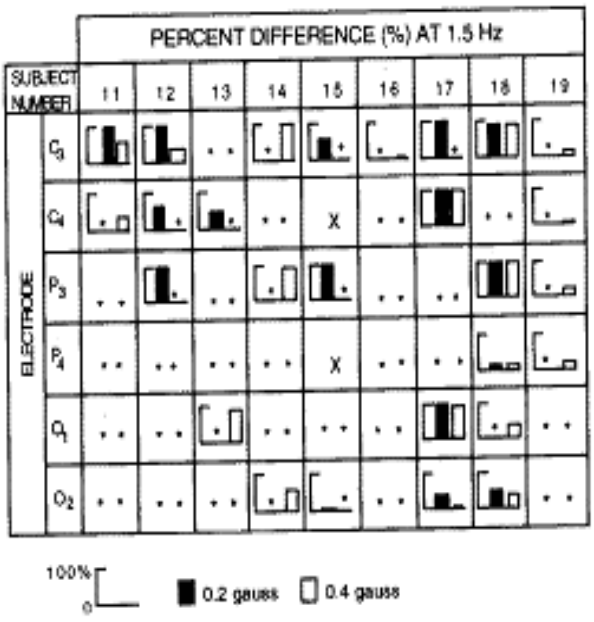

Fig. 3. Change in spectral power of human subjects at 1.5 Hz during application of a 1.5-Hz magnetic field. The results are expressed in terms of the percent difference between the observed and expected power. The solid and open bars correspond to 0.2 and 0.4 gauss, respectively. Data from nonsignificant comparisons are omitted (indicated by asterisks). Percent differences greater than 100% are shown as 100%. X, data not recorded.

of 10 subjects exposed to 0.2 gauss, and in all 10 subjects when exposed to 0.4 gauss.

Table 4 shows typical results obtained during application of 1.5 Hz, and the results for all 1.5-Hz subjects are summarized in Fig. 3. Significant effects on EEG power from I or more electrodes was found in 6 of 9 subjects exposed at 0.2 gauss, and in all 9 subjects at 0.4 gauss (Fig. 3).

In all 4 of the field conditions studied, statistically significant responses were usually observed at 2 or more electrodes (Fig. 4).

4. Discussion

In each subject, the magnetic field altered ongoing brain activity at the frequency of stimulation from one or more electrodes (Figs. 2 and 3). The effect was more likely at 10 Hz compared with 1.5 Hz, and more likely at 0.4 gauss compared with 0.2 gauss (Fig. 4), but the strength of the effect was not proportional to either frequency or field strength (Figs. 2 and 3). A possible source of error that could vitiate these inferences would be the existence of passive contributions to the signal during presentation of the magnetic field other than Faraday induction. If so, every comparison between field-on and field-off groups of data would be biased because no corrections were applied for non-inductive signals. But non-inductive contributions were not seen during measurements of the passive volume conductors, where the observed signal was as expected on the basis of induction alone to within an average of about 7%. Consequently, any putative non-inductive contribution must be such that it exists only for some volume

Table 4
EEG spectral power at 1.5 Hz in subject no. 11 exposed to 1.5-Hz magnetic fields
The absolute value of the mean±SE are listed for the control epochs [P_0(1.5)] and for the exposed epochs after correction for the inductive signal [P(O.2,1.5)—P_{02}(1.5)], and [P(O.4,1.5)—$4P_{02}$(1.5)]. The distributions were compared using the Wilcoxon test

Electrode	Spectral power at 1.5 Hz (gV^2)					
	0.2 gauss			0.4 gauss		
	Control	Exposed	*P*	Control	Exposed	*P*
C_3	3836±551	2013±618	(0.003)	3656±893	674+756	(0.007)
C_4	2111±318	1898±369	(0.50)	2391±272	1548~5O3	(0.05)
P_3	2895±419	3223±-634	(0.91)	3662±679	2942+611	(0.15)
P_4	2133±352	2664±601	(0.94)	3OO2±464	1775±458	(0.13)
O_1	2845±448	4343±798	(0.23)	4251±655	4500±876	(0.70)
O_2	3304±702	3483±634	(0.92)	4412±705	4857±932	(0.55)

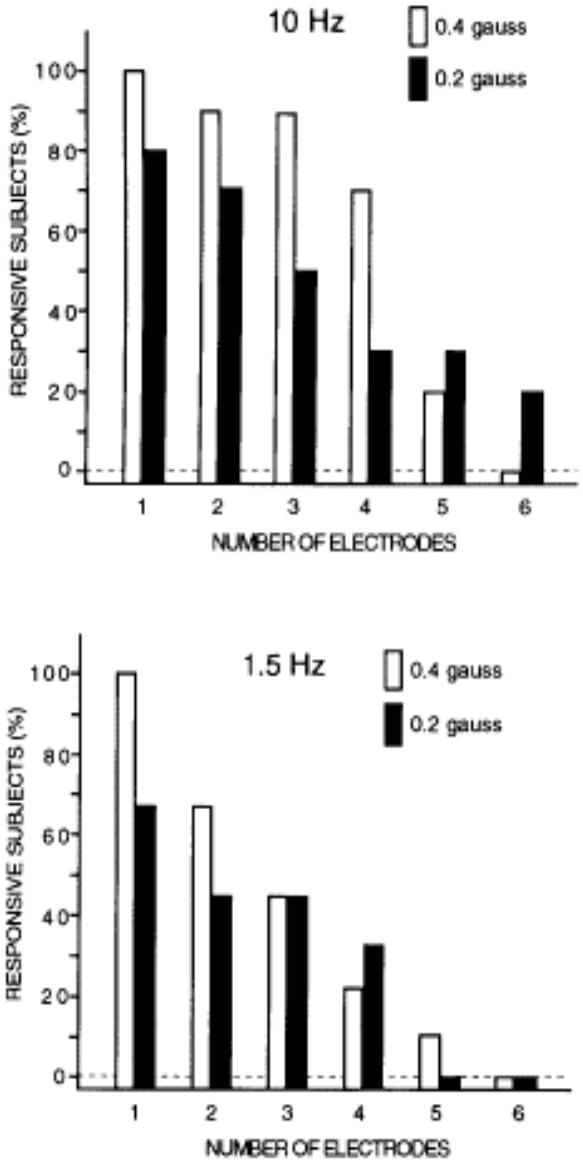

Fig. 4. Percent of subjects that manifested a significant response to a field at t or more electrodes, as a function of the number of electrodes.

conductors (the human head) and not others (saline, tap water, and a melon). Further, if non-inductive signals were present during measurement of the EEG, they must have (1) occurred in some subjects and not others, (2) occurred at some electrodes and not others, and (3) been independent of field strength; these characteristics seem incompatible with passive physical systems. Other potential sources of error include artifacts due to movement of the head or electrode wires, and interference due to respiration. There is, however, no reason to expect that such events occurred differently or more often in exposed than control epochs; uncontrolled motion would therefore have biased the results toward acceptance of the null hypothesis.

Neither age, sex nor the presence of symptoms had any apparent differential role in the elaboration of the observed effects, but the number of subjects within each such class was too small to permit reliable evaluation of class-specific effects. The question whether the effects were due to a direct interaction or were a consequence of an alerting response were not addressed in this study.

Examples of specific frequencies within the central nervous system in relation to particular biological endpoints include 8—9 Hz in the relaxed state and 1—2 Hz in deep sleep. In general, however, it is not clear how most of the various frequencies that can be extracted from the EEG using techniques such as the fast Fourier transform should be regarded. They may originate from relatively monochromatic physiological oscillators, or perhaps they are merely mathematical constructs useful for representing the sum of myriad post-synaptic potentials and action potentials that propagate to the scalp by volume conduction. The results reported here indicate that the external EMF was transduced, resulting in an alteration in the EEG at the frequency of stimulation. Thus, if electrophysiological activity at a particular frequency were a part of the mechanistic chain leading to a particular response, then observations of the response could be used to assess CNS function. Time-estimation tasks, for example, appear to be particularly sensitive to specific EMF frequencies (Medici 1988).

The locus of EMF transduction is still undetermined, but it occurred during a 2-sec exposure epoch, thereby suggesting that it is in a neural or perineural cell. The transduction mechanism may involve sub-threshold changes in cell membrane potential (unpublished data).

In summary, the EMF affected the EEG at the frequency of stimulation in all subjects tested; the spectral power depended on both the subject and the conditions of measurement, and there was a nonlinear relationship between field strength and the magnitude of the response. Similar results were found previously in rabbits exposed to 1—1.5 gauss, 5—10 Hz (Bell et al., 1992a). The similarity in the responses observed in the animal and human studies may indicate that the mechanism of interaction of the EMF with neural tissue is similar in the two species. Thus, the rabbit may be an appropriate species for studying mechanistic and dosimetric aspects of tissue-EMF interactions.

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