ELSEVIER

Journal of the Neurological Sciences 144 (1996) 99-106

JOURNAL OF THE
NEUROLOGICAL
SCIENCES

Low-level EMFs are transduced like other stimuli

Andrew A. Marino [a,*], Glenn B. Bell [b,1], Andrew Chesson [c]

[a] *Department of Orthopaedic Surgery and Department of Cellular Biology and Anatomy Louisiana State University Medical Center, P.O. Box 33932, Shreveport, LA 71130-3932 USA*

[b] *Department of Orthopaedic Surgery Louisiana State University Medical Center, P.O. Box 33932, Shreveport, LA 71130-3932 USA*

[c] *Department of Neurology Louisiana State University Medical Center, P.O. Box 33932, Shreveport, LA 71130-3932 USA*

Received 6 November 1995; revised 8 May 1996; accepted 10 May 1996

Abstract

The aims of this study were to test the theory that transduction of low-level electromagnetic fields (EMFs) is mediated like other stimuli, and to determine the false negative rate of the method used to assess the occurrence of transduction (intra-subject comparison of stimulus and non-stimulus states (ICOS)). A light stimulus was chosen as a basis of comparison because light could be applied and removed at precise time points, similar to the manner in which EMFs were controlled. Subjects exposed to a weak light stimulus during 2-second epochs exhibited alterations in brain electrical activity that were similar to those previously observed in subjects exposed to EMFs. The false negative rate of the ICOS method was 61%, since it registered an effect in only 39% of the subjects (11/28) whereas all subjects were actually aware of the light. In a second group of subjects that were exposed to 0.8˚gauss (1.5 or 10˚Hz), 58% (11/19) exhibited similar alterations in brain activity, as determined using ICOS. Previous measurements in the same subjects using a different method showed that the EMFs actually affected brain electrical activity in all subjects; consequently, the false-negative rate was 42% when an EMF was used as the stimulus. The results suggested that the post-transduction brain electrical processes in human subjects were similar in the cases of EMF and light stimuli, as hypothesized, and that the high negative rate of the ICOS method (here and in previous studies) was composed partly or entirely of false negative results.

Keywords: EEG, Electromagnetic field, Brain Fourier transformation, Cognitive stimulus, Non-cognitive stimulus, Power spectrum

1. Introduction

Low-level electromagnetic fields (EMFs) can cause biological changes in animals (Becker and Marino, 1982) and, when present in the environment, can increase the risk for some diseases (Marino, 1993). Neither the transduction locus nor the nature of the biological signals engendered by EMFs are known, and various theories have been proposed (Polk and Postow, 1986). One possibility, by analogy with other stimuli, is that detection of EMFs occurs in the nervous system, leading to afferent electrical signals and subsequent processing events in the brain (Becker and Marino, 1982; Marino, 1993).

There are many generators of the brain's electrical activity. Energy emitted by the underlying electrical events, including those triggered by somatic stimuli, propagates passively and can sometimes be detected using scalp electrodes; occipital alpha waves and evoked potentials are two examples. It seemed reasonable, therefore, to determine whether changes in scalp potentials caused by EMFs might also be observable, thereby providing support for the neurogenic theory of EMF bioeffects. When intra-subject comparisons of stimulus and non-stimulus states (ICOS) were made using spectrally decomposed 2-second epochs, altered brain electrical activity was found in 7 of 14 subjects exposed to 0.25-0.50˚gauss, 35-40˚Hz, and 15 of 20 subjects exposed to 0.78˚gauss, 60˚Hz (Bell et al., 1991; Bell et al., 1992a). But spurious signals due to EMF effects on scalp electrodes have been reported (Takashima et al., 1979); thus, despite the precautions taken previously (Bell et al., 1991; Bell et al., 1992a), it could still be argued that the observed effects were artifacts of the recording system, not true physiological effects.

Another issue raised by the earlier studies (Bell et al., 1991; Bell et al., 1992a) involved the interpretation of the negative results that occurred in 35% ([7+5]/[14+20]) of

* Corresponding author. Tel: +1 (318) 675-6180. Fax: +1 (318) 675-6186. E-mail: amarino@lsumc.edu

[1] Present address: Science Applications International, Inc., 4242 Woodcock Drive, San Antonio, TX 78228

the subjects studied. The subjects could have been inherently unresponsive to the EMF; if so, they would have been true negatives, suggesting that human sensitivity to EMFs was not a universal trait. On the other hand, at least some of the negative cases would have been false, implying that the trait of sensitivity was more general than would have otherwise been concluded.

One aim of this study was to test the parallelism theory between EMFs and other stimuli by determining whether subjects exposed to a light stimulus would exhibit changes in brain electrical activity similar to those observed previously using EMFs (Bell et al., 1991; Bell et al., 1992a), an observation which would suggest that post-stimulus brain electrical activity was indeed similar in the two cases when determined using the ICOS method. And such an observation would also indicate that the previous results were not caused by EMF-induced artifacts at the scalp electrodes.

The second aim was to evaluate the reliability of the ICOS method by comparing the results with those obtained using other methods. The reasoning was that if ICOS had a high false-negative rate, it would be more likely that the previous studies underestimated the prevalence of EMF sensitivity in the population. The ICOS false-negative rate was determined using light as a stimulus, and (in a separate group of subjects), using an EMF of a frequency that had no *a priori* theoretical or physiological significance (Polk and Postow, 1986).

2. Materials and methods

2.1. Light

A light stimulus was chosen as a basis of comparison because light could be applied and removed at precise time points, similar to the manner in which EMFs were controlled. Light was obtained from four incandescent light bulbs housed in a plastic box located 1.3˚meters in front of the subject; the bulb current was controlled by a computer-based timing circuit and produced less than 0.1˚watts/m^2 of illumination and less than 0.01˚mG at the subject's forehead. The subjects were instructed to keep their eyes closed throughout the session, including the period during which the light was presented, and compliance was verified by the absence of eye-movement artifacts and by direct questioning. Even though the subjects' eyes were closed, the intensity of the light was sufficient to permit each subject to be aware of whether the light was on or off. Sham exposure was used as a negative control; during that portion of the test session the electroencephalogram (EEG) was recorded but the light was not presented to the subject.

2.2. Magnetic Field

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. A timing circuit controlled the coil current, which was obtained from a signal generator (Model 182A, Wavetek, San Diego, CA), and amplifier (Model 7500, Krohn-Hite, Avon, MA). The subjects sat between the coils (mid-sagittal plane located at the mid-point between the coils and perpendicular to the coil axis) on a wooden chair with their eyes closed. A field of 0.8˚gauss at either 1.5 or 10˚Hz was applied for 2-second epochs to separate groups of subjects; the field was uniform to within 5% in the region of the head (20% with the chest and pelvis included). The average background 60-Hz magnetic field was about 0.1˚mG. There were no visual, auditory, or tactile cues to the subjects that indicated the presence of the field, and hence the subjects were unaware of the field when it was presented.

2.3. Subjects

Normal (non-symptomatic) subjects were recruited from the general population, and additional subjects (patients) were recruited from among those with neurological complaints who underwent a clinical EEG as a diagnostic procedure. The patients were identified for possible inclusion by EEG technicians who, while doing a clinical EEG, noted the presence of well-developed occipital alpha activity. Cooperative patients having this finding were then asked to participate in the study, and informed consent was obtained after the nature and possible consequence of the study were explained. Those consenting to participate and willing to remain in the laboratory area after completion of their clinical EEG, were utilized. Several of the clinical EEGs were subsequently judged abnormal by the neurologist reading the clinical tracings and blinded to whether the patient was a study participant. No patient was used who had a seizure occurring during the tracing, or who had focal or generalized slowing of a persistent nature. However patients that manifested only infrequent, intermittent, dysrhythmic activity were not excluded, as planned, because there was no evidence to suggest that the presence of dysrhythmic activity would differentially affect the responses to the stimuli

The studies involving light were performed on 16 normal subjects and 12 patients. In the EMF studies, 13 normal subjects and 6 patients were used. The results were analyzed without respect to age, sex, the presence of symptoms, or the clinical evaluation of the EEG because preliminary examination revealed no apparent influence of the factors. The study was approved by the Institutional Review Board for Human Research of the Louisiana State University Medical Center.

2.4. Measurement Procedure

Surface electrodes (Grass Instrument Co., Quincy, Massachusetts) were placed at C-3, C-4, P-3, P-4, O-1, and O-2 (international 10-20 system), with ear-linked electrodes as the reference; the ground was placed on the forehead. The EEG was filtered to pass 0.3-35˚Hz, and the signal was divided and simultaneously recorded on an

electroencephalograph (model 6, Grass Instrument Co., Quincy, Massachusetts) and sampled at 200˚Hz; the digital data was stored on a hard drive for later analysis. Electrode impedances were measured before and after each recording (EZM˚5A, Grass Instrument Co., Quincy, MA); typically, the impedances were 2-3˚k‰.

The room in which the measurements were made was dark and partially soundproofed, but occasional sounds that occurred in an adjacent corridor could be heard in the room. The equipment that powered the light and coils and that recorded the EEG was located at a distance of 15 meters.

Each subject underwent a block of trials that included stimulus (either light or magnetic fields) and control epochs; in addition, the group that received the light stimulus also received a sham stimulus as a positive control (a period during which all experimental conditions were maintained except for the absence of a stimulus). A trial consisted in the presentation of a stimulus (or sham) for 2˚sec, followed by a 5-sec stimulus-free interval. The control epoch for each stimulus epoch was the immediately preceding 2-sec interval.

If P(f) represents the power spectrum (the coefficients at frequency f in the fast Fourier transform (FFT) of 2-sec EEG epochs), we hypothesized that P(f) differed reliably between stimulus and control epochs, and therefore that the occurrence of such a difference was evidence that detection of the stimulus had occurred. Approximately 60 trials were obtained for each stimulus, and the first 50 artifact-free trials were used in the subsequent analysis.

2.5. Statistical Design

The statistical justification for the method of analysis is presented elsewhere (Bell et al., 1992a). Briefly, the criterion for accepting the conclusion that an event-related change in scalp potential actually occurred was that the stimulus magnetic field, light, or sham resulted in bilateral differences in at least 2 Fourier frequencies from at least one pair of electrodes, employing the Wilcoxon signed rank test (at $P<0.05$). It was shown that this condition was sufficient to eliminate (at $P˚<˚0.05$) the possibility of a family-wise error regarding rejection of the null hypothesis (Bell et al., 1992a). Thus, for each subject regarded as detecting the stimulus, it was unlikely ($P < 0.05$) that the decision was a Type I statistical error, and each of the frequencies listed as mediating detection in particular subjects exhibited statistically significant differences in spectral energy during presentation of the stimulus, compared with the level observed during the control epochs. After the statistical analysis, the data was subjected to Bartlett smoothing for purposes of illustration.

In the EMF groups, the FFT coefficient at the frequency of stimulation contained a non-physiological component resulting from Faraday induction (Bell et al., 1994). As a precaution against the inclusion of inductive signals, spectral frequencies †2.5˚Hz and 9-11˚Hz were also excluded from analysis in the subjects exposed to 1.5˚Hz and 10˚Hz, respectively.

3. Results

When the electrical activity obtained during the sham-stimulus epochs was compared with that from the corresponding control epochs, no false-positive decisions regarding detection resulted in any of the 28 subjects tested. The results obtained using light are given in Table 1. A total of 11 subjects exhibited statistically significant changes during exposure to the light, thereby indicating that the stimulus had been detected. From subject to subject, responses occurred throughout the spectrum but were more likely in the alpha frequency range (Fig. 1).

Table 1
EEG frequencies affected by exposure to light.

Subject	EEG frequency (Hz) affected
1 (31,F)	P 9.5,10; O 9,9.5,10
2A (36,F)	O 5.5,9.5
2B	O↑ 14,15
3 (24,F)	None
4A (37,M)	O↑ 13.5,16.5; P↑ 1.5,13.5,16.5
4B	O↑ 6.5,18
5 (30,M)	None
6 (23,M)	None
7A (22,F)	C↑ 7.5,11; P↑ 7.5,8.5,10.5,11; O↑ 8.5.10,10.5,15
7B	O↑ 4.5,17.5
8A (47,M)	C↑—2; C 6.5,8; P↑ 3.5,4; P 6.5,7.5, 8.5,9,9.5,10; O 8.5,9,9.5,10
8B	C 6.5,7,7.5,9,10; P 7,7.5,8,9,9.5,10; O 6,7,8,9,9.5,10
9 (23,M)	None
10 (18,M)	None
11 (25,M)	None
12 (28,M)	None
13 (30,M)	O↑—9; O—3.5
14 (36,M)	None
15 (35,F)	None
16 (30,M)	None
17 (40,F) S	None
18 (50,F) S	C↑–14.5; C 9.5,18; O 2.5,10.5
19 (52,M) S	None
20 (33,M) S	None
21 (46,F) S	None
22 (19,F) S,A	None
23 (23,F) S	P↑ 1,6.5,10,10.5,11,13.5; O↑ 9.5,10.5,11
24 (28,F) S,A	O 2.5,8.5
25 (44,F) S,A	P↑ 12.5,14.5,16,16.5,17; O↑ 1.5,7,12,12.5,13.5,14
26 (22,M) S,A	None
27 (61,M) S,A	None
28 (51,F) S,A	C 9.5,17; P↑—2; P 8,9,9.5,17; O↑—2; O 9,9.5,17

The light was 0.25˚watts/m^2 at its source. C, P, O are the central, parietal, and occipital electrodes (international 10-20 system), respectively. F, female; M, male. The power at each frequency measured during presentation of the stimulus was less than that measured during the control epochs except where indicated by the arrow. S, symptomatic; A, abnormal EEG.

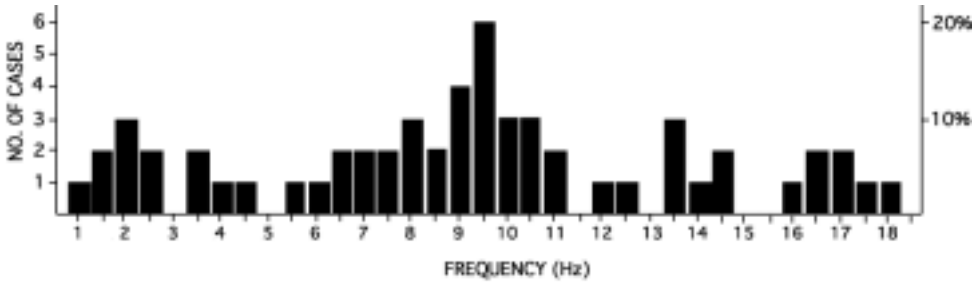

Fig. 1. Number of subjects that responded to light as a function of the spectral frequency at which responses were observed (n = 28). Data from Table 1.

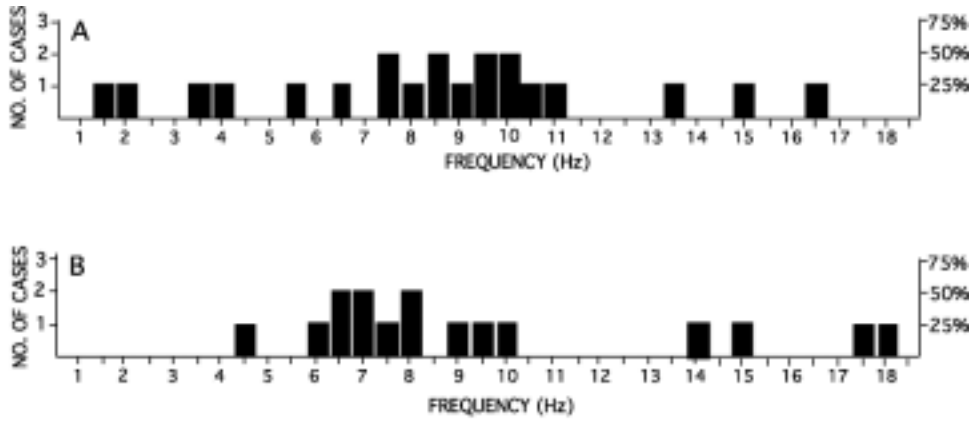

Fig. 2. Test/re-test results in four subjects exposed to light. A, number of subjects (of 4, subject no. 2, 4, 7, 8, Table 1) affected at each spectral frequency. B, results from the same subjects after 1-2 weeks.

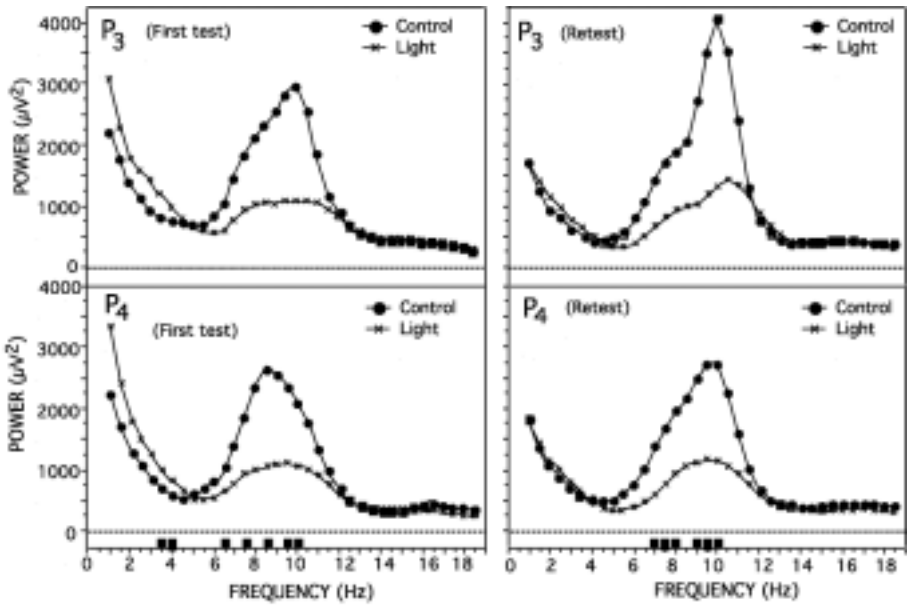

Fig. 3. the effect of light on the EEG at the parietal electrodes in a subject who was tested twice (subject no. 8, Table 1). Each curve is the mean of 50 epochs (subjected to Bartlett smoothing). The spectral energies in the light and control epochs at each frequency were compared using the Wilcoxon signed rank test. Frequencies that differed significantly ($p < 0.05$) from the control at both P_3 and P_4 during the first test are indicated by the solid squares on the frequency axis in the left panel. Comparable results obtained during the re-test are shown in the right panel.

Table 2

EEG frequencies affected by exposure to magnetic field of 0.8˚Ga

Subject	EMF frequency (Hz)	EEG frequency (Hz) affected
1 (22,F)	10	None
2 (30,M)	10	C↑ 14.5,17
3 (31,F)	10	None
4 (30,M)	10	C↑ 11.5-13,14-18.5
5 (25,F)	10	O↑ 12.5,13.5
6 (62,M)	10	C↑ 6,11.5-14.5
7 (27,M)	10	None
8 (26,F) S	10	C↑ 8.5,11.5-15.5,17; P↑ 2.5,11.5,13.5
9 (28,F) S	10	None
10 (21,M) S	10	None
11 (31,F)	1.5	O 10.5; O↑ 14
12 (30,M)	1.5	None
13 (22,F)	1.5	C↑ 16.5,17.5-18.5
14 (31,F)	1.5	None
15 (35,M)	1.5	None
16 (30,M)	1.5	C↑ 15,15.5,17,18.5
17 (34,M) S,A	1.5	O↑ 11,18.5
18 (51,M) S	1.5	C↑ 16.5,17.5,18.5; O↑ 3.5,5,8,13.5,14,15.5,18.5
19 (42,M) S	1.5	C↑ 4.5,5,6-7.5,8.5-10,11.5,12, 13,14.5,15.5,16,17

C, P, O are the central, parietal, and occipital electrodes (international 10-20 system), respectively. F, female; M, male. The power at each frequency measured during presentation of the stimulus was less than that measured during the control epochs except where indicated by the arrow. S, symptomatic; A, abnormal EEG. Frequencies 9-11˚Hz and †2.5˚Hz were excluded from consideration in subjects 1-10 and in subjects 11-19, respectively.

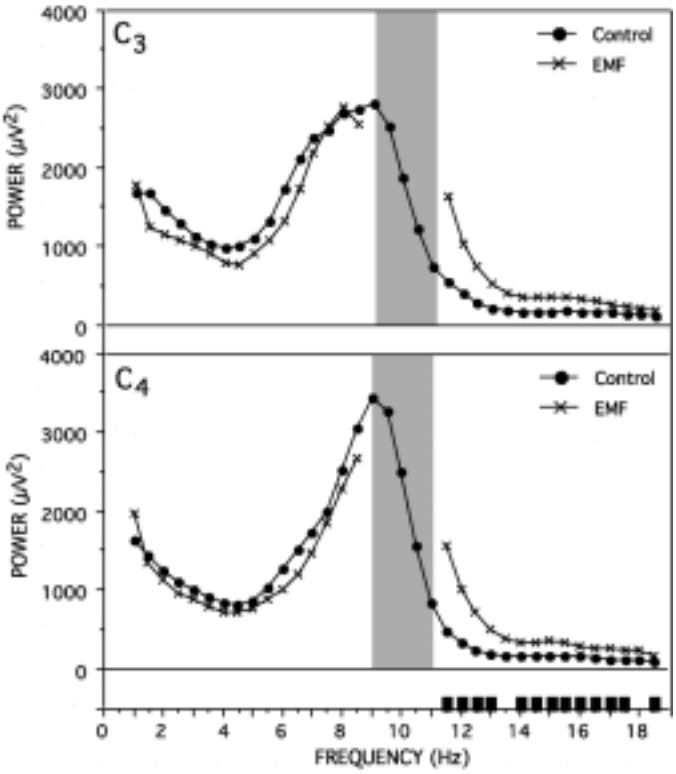

Fig. 5. Effect of 0.8 Ga, 10 Hz on the EEG at the central electrodes (subject No. 4, Table 2). Each curve is the mean of 50 epochs (subjected to Bartlett smoothing). The spectral energies in the EMF and control epochs at each frequency were compared using the Wilcoxon signed rank test (9-11 Hz were not considered). Frequencies that differed significantly ($p < 0.05$) at both electrodes are indicated by the solid squares on the frequency axis.

Four responsive subjects were re-tested, and the occurrence of a response to light was confirmed in each subject; however, the frequencies at which the responses were manifested during the re-test were not identical to those found during the initial test (Fig. 2). Typical results from a subject who displayed an alpha responsiveness during the test and the re-test are shown in Fig. 3. At the parietal electrodes, responsiveness to the field was found at 7.5 and 9-10˚Hz in both tests; other frequencies were not

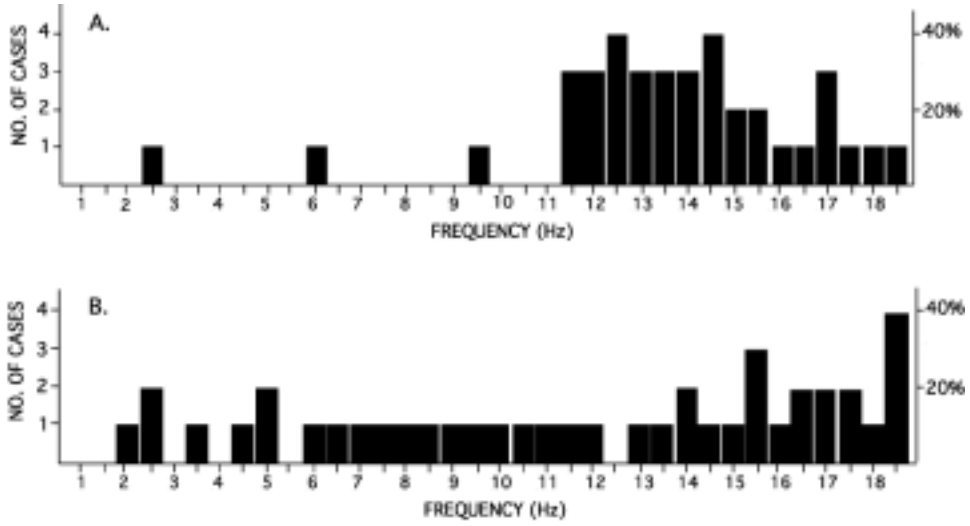

Fig. 4. Number of subjects that responded to magnetic fields as a function of the spectral frequency at which the responses were observed. A, 0.8 Ga, 10 Hz ($n = 10$); B, 0.8 Ga, 1.5 Hz ($n = 9$). Data from Table 2.

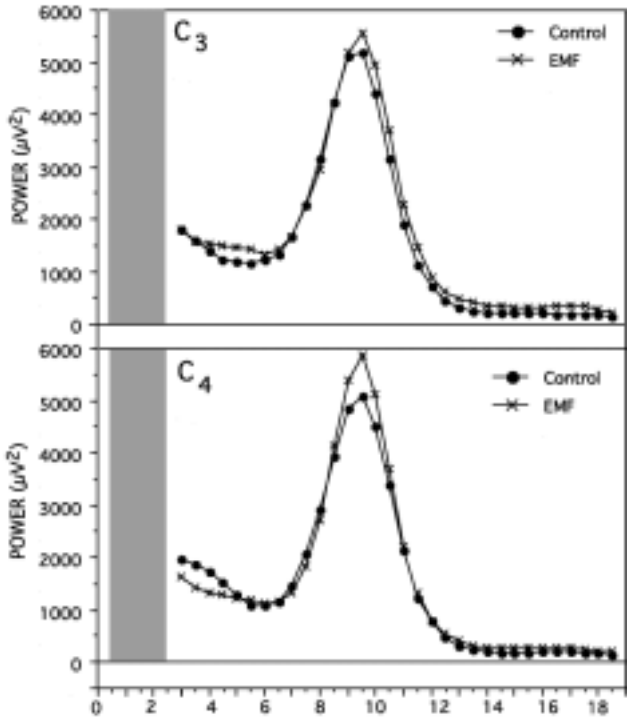

Fig. 6. Effect of 0.8 Ga, 1.5 Hz on the EEG at the central electrodes (subject No. 16, Table 2). Each curve is the mean of 50 epochs (subjected to Bartlett smoothing). The spectral energies in the EMF and control epochs at each frequency were compared using the Wilcoxon signed rank test († 2.5 Hz not considered). Frequencies that differed significantly ($p < 0.05$) at both electrodes are indicated by the solid squares on the frequency axis.

affected identically (6.5˚Hz and 8.5˚Hz in the first test versus 7˚Hz and 8˚Hz during the re-test). Moreover an increase in power at 3.5˚Hz and 4˚Hz was seen initially, but not during re-test.

In the magnetic field studies, 50% (5/10) of the subjects responded to the EMF at 10˚Hz, and 67% (6/9) responded at 1.5˚Hz (Table 2). Again, responses occurred throughout the spectrum but, for both fields, the detection processes were mostly mediated by changes at the higher spectral frequencies (Fig. 4). Examples of the effect of EMFs on the power spectrum are shown in Fig. 5 and 6.

4. Discussion

Using the ICOS method, it was found previously that most subjects exhibited changes in brain electrical activity during exposure to EMFs; depending on the subject, the changes occurred throughout the power spectrum and consisted of induced increases and decreases in spectral energy of 100-4000˚ V^2 (Bell et al., 1991; Bell et al., 1992a). That is, the specific observation that was relatively reproducible from subject to subject was the occurrence of changes in brain electrical activity somewhere within the range of 1-18.5˚Hz; EMF-induced changes in the same frequencies in each subject were not observed.

On the basis of the theory that the early events in EMF transduction are similar to those caused by ordinary somatic stimuli, similar results regarding brain electrical activity should occur following exposure to such stimuli. That prediction was tested using light as the stimulus because light could be applied and removed with roughly the same temporal precision as was possible using an EMF. Approximately 39% (11/28) of subjects exposed to light exhibited altered brain activity which consisted of both increases and decreases in energy at various frequencies, up to a maximum change of about 4000˚ V^2 (Table 1) (Fig. 3). Again, the reproducible observation (39% of subjects) was that a change occurred in the power spectrum, but not at the same frequencies in each subject. Moreover, when a light-induced change occurred, it could be demonstrated again in the same subject during a re-test 1-2˚weeks after the first test. Consequently, the predicted similarity between the brain responses to EMFs and light when assessed using ICOS was confirmed, because for both stimuli (1)˚the phenomenon of an induced change in brain electrical activity in at least some subjects was proved; (2)˚the effect was not manifested at the same frequency in each subject. These results indicate that the post-transduction processes in the two cases were similar in the sense that they produced the same kind of electrical perturbations at the scalp. Further, since it is unlikely that distinctly different stimuli such as a 60-Hz magnetic field and a weak visible light signal would produce exactly the same kind of artifact at scalp electrodes, it is reasonable to conclude from the present results that previous observations involving EMFs (Bell et al., 1991; Bell et al., 1992a) were not artifacts of the system.

All of the subjects that received the light stimulus were aware of it even though the light was dim and their eyes were closed. Since awareness is a centrally mediated phenomenon, it was certain that brain electrical activity was altered during presentation of the light; it can be concluded, therefore, that the negative rate for responsiveness to light of 61% when assessed by ICOS (Table 1) consisted entirely of false-negatives.

The false-negative rate of ICOS for EMFs was determined by comparing the present results (Table 2) with those obtained previously with the same subjects (Bell et al., 1994). Each subject (Table 2) was previously presented with 0.2-0.4˚gauss and shown to exhibit altered electrical activity at the frequency of the EMF (either 1.5 or 10˚Hz) during its presentation. Since each subject actually detected 0.2-0.4˚gauss, it is reasonable to infer that each also detected 0.8˚gauss. Moreover, considering both the physical basis of brain electrical activity and the mathematical properties of the FFT, it can be concluded that the EMF-induced spectral changes could not have been limited to changes at the frequency of stimulation. Although the FFT can be used to represent and analyze brain activity, the results of the mathematical decomposition of the scalp potentials do not imply the existence of actual, physical oscillators (Gevins, 1984). More likely, neuronal events observed in the form of a change in power at the frequency of the applied field when the scalp potentials were spectrally decomposed (Bell et al.,

1994) actually originated from field-induced membrane events that were not monochromatic oscillators throughout the 2-sec exposure epoch (Gevins, 1984; Nunez, 1989). If so, there must also have occurred a concurrent (over 2 seconds) and corresponding change in other frequencies in the power spectrum. Consequently, ICOS should have been 100% positive for the subjects in Table 2; any other result would indicate the occurrence of one or more false negatives. But a detection rate of 58% was found, indicating that the false-negative rate for observing EMF-related changes in scalp electrical potential was 42% (Table 2). This suggests that the negative results in some subjects in previous studies (Bell et al., 1991; Bell et al., 1992a) were false negatives and did not indicate true non-responders.

What could have accounted for the high false-negative rates (42-61%) using ICOS to detect electroneurophysiological responses to stimuli? The possibilities include: (1)˚a smaller response (less energy emitted at the frequencies subserving detection compared with the responders); (2)˚increased noise levels (non-event-related electrical energy); (3)˚non-ideal placement of the electrodes relative to the locus of the underlying membrane events. The results obtained from 4 subjects who were re-tested using light (Fig. 2) showed that responses large enough to be observed initially were also sufficiently large to be measured at another time, suggesting that the strength of a response is an individual trait. Increased levels of endogenous noise seemed an unlikely explanation because, if true, it would have been expected in at least one of the re-tested subjects, resulting in a negative finding; in each case, however, the original results were confirmed. The third possibility cannot be ruled out because there is no *a priori* reason to expect that an event-related change should be manifested only at the scalp locations sampled. That is, although a particular electrode placement may be the optimal location for detecting brain electrical changes due to particular stimuli (for example, occipital electrodes and light), the failure to observe a change at those locations does not rule out the possibility that changes occurred at other scalp locations. Studies that employed a larger electrode montage are needed to evaluate the issue.

The overall results obtained in this and past studies when subjects were exposed to EMFs and evaluated using ICOS are shown in Fig. 7. In contrast to the distribution obtained using light (Fig. 1), subjects were less likely to detect EMFs via processes manifested as changes in the theta and alpha ranges. The neural circuits that mediate detection of EMFs might not contain cortical projections (because EMFs don't result in awareness). If so, since any event-related processes must propagate electrotonically to the scalp to be detected using scalp electrodes, electrical phenomena originating in the cortex would contribute noise but no signal during measurements of responses to EMFs. Thus, one possible explanation is that ICOS is inherently insensitive to changes

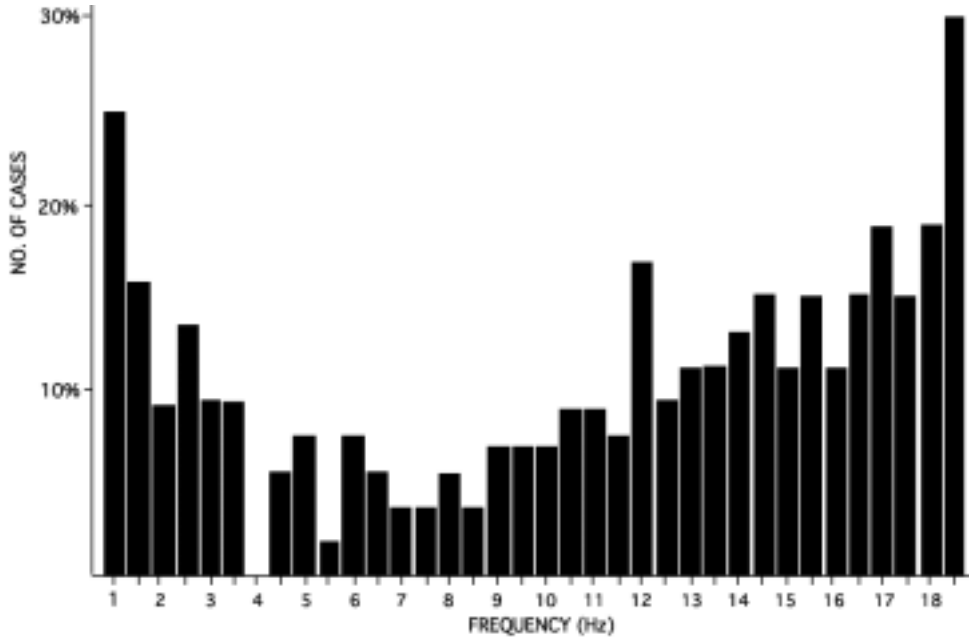

Fig. 7. Overall total percent of subjects affected by EMFs as a function of frequency (Fig. 3 and Refs. 3 and 4). The data includes the responses observed from the subjects exposed to 0.8 Ga at 10 Hz and 1.5 Hz (n = 10, 9, respectively (Fig. 3)), 0.25-0.50 Ga, 35-40 Hz (n = 14) [3], and 0.78 Ga, 60 Hz (n = 20) [4]. The data has been adjusted to reflect the fact that only 44 subjects were eligible to contribute to observation † 2.5 Hz, and only 43 subjects were eligible to contribute to observations at 9-11 Hz.

in those ranges because of the randomizing influence of cortical processes near the electrodes that generate energy in the alpha band (Pfurtscheller, 1989).

It is worthwhile to consider the extent to which the theory that transduction of ordinary (those having no *a priori* theoretical or physiological significance), low-level EMFs occurs within the nervous system has been supported by the data. First, the observed effects (Table 2) occurred rapidly (<2 seconds), which is consistent with electrical transduction processes. Low-level EMFs apparently lack the energy to directly initiate spike potentials, but information may be added to a neuron in the form of subthreshold changes in membrane potential that can initiate or modify neuronal activity (Schmitt et al., 1976; Bialek et al., 1991; da Silva, 1991). Such effects could have constituted an afferent signal occurring within the 2-sec interval. Second, there is no good reason to suppose that the hypothetical post-transduction electrical processes would always consist of only increased or decreased spectral energy at particular frequencies. Both kinds of changes would therefore be expected, depending on the subject, the details of the applied EMF, and host factors. Since both kinds of changes were observed (Table 2) (Bell et al., 1991; Bell et al., 1992a), the relationship anticipated in the model is reflected in the data. Third, since sensory information decussates (Martin, 1989), it is reasonable to expect that event-related signals produced in symmetrically paired electrodes will be similar when, for example, a light stimulus is applied to both eyes or the subject's entire body is exposed simultaneously to an EMF. This consideration does not suggest that the fact of transduction can be inferred only from analysis of responses from paired electrodes, but the successful use of this association in the statistical analysis (Tables 1 and 2) validates the bilateral-symmetry aspect of the proposed model. Fourth, one of the membrane-level hallmarks of sensory transduction the graded response is not observable in event-related scalp potentials because the subsequent processing events occur in a non-linear fashion (Gevins, 1984; da Silva, 1991). It was shown previously that the relationship between applied EMFs and the resulting changes in scalp potentials were non-linearly related in rabbits (Bell et al., 1992b) and human subjects (Bell et al., 1992a). Only when the animals were killed (Bell et al., 1992b) or electrical signals were measured in non-living ionic conductors (Bell et al., 1994) was a linear response between the applied field and the measured voltages observed. Thus, the observed data reflects the anticipated nonlinearity in response, as well as the absence of a specific frequency response to the field.

References

Becker, R.O. and Marino, A.A. (1982) Electromagnetism & Life, State University of New York Press, Albany, NY.

Bell, G.B., Marino, A.A., Chesson, A.L., Struve, F.A. (1991) Human sensitivity to weak magnetic fields. Lancet, 338: 1521-1522.

Bell, G.B., Marino, A.A., Chesson, A.L. (1992a) Alterations in brain electrical activity caused by magnetic fields: detecting the detection process. Electroencephalog. Clin. Neurophysiol., 83: 389-397.

Bell, G., Marino, A.A., Chesson, A., Struve, F. (1992b) Electrical states in the rabbit brain can be altered by light and electromagnetic fields. Brain Res., 570: 307-315.

Bell, G.B., Marino, A.A., Chesson, A.L. (1994) Frequency-specific responses in the human brain caused by electromagnetic fields. J. Neurol. Sci., 123: 26-32.

Bialek, W., Reike, F., deRuyter van Steveninck, R.R. and Warland, D.L. (1991) Reading a neural code. Science, 252: 1854-1857.

da Silva, F.L. (1991) Neural mechanisms underlying brain waves: from neural membranes to networks. Electroencephalog. Clin. Neurophysiol., 79: 81-83.

Gevins, A.S. (1984) Analysis of the electromagnetic signals of the human brain: milestones, obstacles, and goals. IEEE Trans. Biomed. Eng., BME-31: 833-850.

Marino, A.A. (1993) Electromagnetic fields, cancer, and the theory of neuroendocrine-related promotion. Bioelectrochem. Bioenerg., 29: 255-276.

Martin, J.H. (1989) Neuroanatomy, Elsevier, New York, pg. 143.

Nunez, P.L. (1989) Generation of human EEG by a combination of long and short range neocortical interactions. Brain Topography, 1: 199-215.

Pfurtscheller, G. (1989) Functional topography during sensorimotor activation studied with event-related desynchronization mapping. J. Clin. Neurophysiol., 6: 75-84.

Polk, C. and Postow, E. (Eds.) (1986) Handbook of Biological Effects of Electromagnetic Fields, CRC, Boca Raton, FL.

Schmitt, F.O., Dev, P., Smith, D.H. (1976) Electrotonic processing of information by brain cells. Science, 193: 114-120.

Takashima, S., Onaral, B., Schwan, H.P. (1979) Rad. Environ. Biophys., 16: 15-27.