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Mobile-phone pulse triggers evoked potentials

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ABSTRACT

If mobile-phone electromagnetic fields (EMFs) are hazardous, as suggested in the literature, processes or mechanisms must exist that allow the body to detect the fields. We hypothesized that the low-frequency pulses produced by mobile phones (217 Hz) were detected by sensory transduction, as evidenced by the ability of the pulses to trigger evoked potentials (EPs). Electroencephalograms (EEGs) were recorded from six standard locations in 20 volunteers and analyzed to detect brain potentials triggered by a pulse of the type produced by mobile phones. Evoked potentials having the expected latency were found in 90% of the volunteers, as assessed using a nonlinear method of EEG analysis. Evoked potentials were not detected when the EEG was analyzed using time averaging. The possibility of systematic error was excluded by sham-exposure analyses. The results implied that mobile-phones trigger EP at the rate of 217 Hz during ordinary phone use. Chronic production of the changes in brain activity might be pertinent to the reports of health hazards among mobile-phone users.

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Mobile phones are increasingly more common, and questions have been raised concerning whether the electromagnetic fields (EMFs) they emit are partly responsible for brain cancer or other diseases [11]. Mobile phones transmit and receive high-frequency EMFs (~1 GHz), and also emit low-frequency magnetic pulses (217 Hz) from the phone's circuitry and battery currents [16] (Fig. 1a). If exposure to mobile-phone EMFs is hazardous, processes or mechanisms must exist that allow the body to detect at least one field. One possibility is that the EMF is detected by sensory transduction, like other environmental stimuli [1]. EMFs of the type produced by the electrical power system triggered evoked potentials (EPs) having latencies of about 250 ms [6], but the ability of mobile-phone EMFs to trigger EPs, as assessed using a standard stimulus-response protocol [17], has not been studied.

Reports that brain electrical activity was affected during exposure to simulated mobile-phone EMFs supported the transduction hypothesis. As examples, high-frequency mobile-phone EMFs altered the amplitude of the P50 component of the auditory evoked potential [15], spectral coherence during an auditory memory task [10], and alpha power during sleep [2]. Both high-frequency EMFs and low-frequency magnetic pulses from the phone's battery currents altered the contingent negative variation triggered by acoustic stimuli [8].

Evidence that at least one of the types of EMFs produced by mobile phones was capable of eliciting brain potentials would provide a possible basis for explaining how chronic phone use leads to disease. Consequently, in a study of 20 clinically normal volunteer subjects, 7 males (age range 22–62 years) and 13 females (18–53 years), we addressed the question of whether a low-frequency pulse of the type produced by mobile phones was capable of triggering EPs.

The subjects gave written informed consent prior to participating in the experiment. They were informed of the goals, methods, and general design of the investigation, but were not told exactly when during the experimental session that the EMF stimulus would be applied. The institutional review board at the LSU Health Sciences Center approved all experimental procedures.

Mobile phones emit a complicated temporal array of electromagnetic, acoustic, thermal, and tactile stimuli. To avoid confounding effects and to facilitate use of a standard protocol for detecting EPs [17] we applied a simulated mobile-phone pulse. The strength of the pulse was chosen based on measurements of a typical mobile phone (Model 6085, Nokia, Helsinki, Finland) made during a phone call (3 km between the base tower and phone). The peak strength of the magnetic pulse 10 cm from the phone was 3 μT, and the duration of each pulse was 0.7 ms (MAG-03, GMW, Redwood City, CA, USA) (Fig. 1b).

Several factors entered into our considerations regarding the design of the apparatus necessary to repetitively apply a

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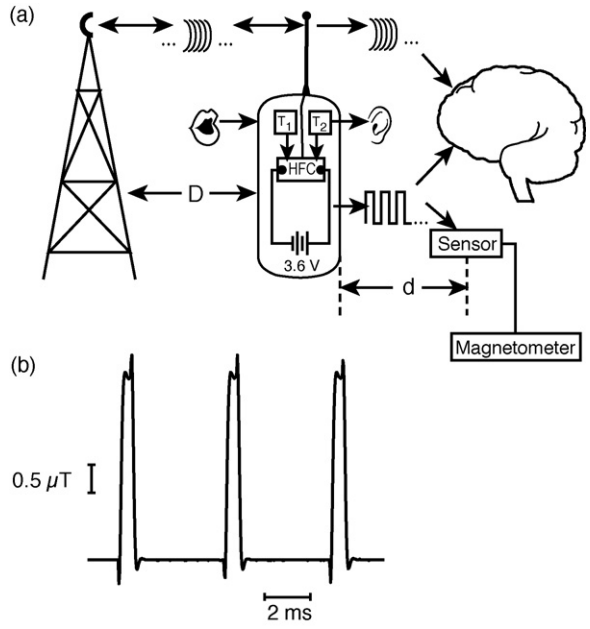

Fig. 1. Mobile-phone EMFs in the brain. (a) Mobile phones produce high- and low-frequency EMFs. A battery supplies current to a high-frequency circuit (HFC) that encodes speech (transducer T_1) on the transmitted signal and decodes speech (transducer T_2) in the received signal. The battery current produces magnetic pulses whose strength depends on D and d, the distances between the base tower and the phone and between the phone and the field sensor, respectively. (b) Portion of the magnetic pulse train from a Nokia 6085 mobile phone (D = 3 km, d = 10 cm).

3-μT, 0.7-ms pulse and an appropriate inter-stimulus period. To minimize variability in the responses of the subjects, it was desirable that the pulse strength be relatively uniform throughout the brain, irrespective of the motion of the subject's head relative to the source of the field (actual mobile-phone magnetic fields do not exhibit this property). The standard method for applying uniform magnetic fields involves the use of multiple current-carrying coils of magnet wire [14], but the impedance of typical coil systems [3] prevents generation of the narrow pulse (Fig. 1b) required for the present study [12]. Fortunately, however, we recently discovered that subjects exposed to an EMF stimulus responded not to its magnetic component but rather to the electric component induced in the brain as a consequence of the rate of change of the magnetic flux [7]. External electric fields that produce the same induced electric field as that from a magnetic pulse can conveniently be produced by applying a voltage to a pair of parallel metal plates.

An external electric-field pulse was generated by applying a 65-V, 0.7-ms pulse (Hewlett Packard 8015A, Palo Alto, CA, USA; Krohn-Hite 7500, Avon, MA, USA) to two metal plates (65 cm apart) located on each side of the head (Fig. 2a). It can be shown that this pulse produced an unperturbed electric field (field in the absence of a subject) of about 100 V/m that, in turn, induced a brain electric field comparable to that induced by the magnetic pulse (Fig. 1b).

The stimulus duration and inter-stimulus period were 0.7 ms and 2.9993 s, respectively, resulting in 3-s trials (Fig. 2a). The subjects were exposed in an isolation chamber to reduce the effect of random ambient stimuli; all electrical equipment was located outside the chamber. The absence of both uncontrolled sensory cues and direct perception of the field was verified by interviewing each subject at the end of the experimental session.

Electroencephalograms (EEGs) were recorded from O_1, O_2, C_3, C_4, P_3, and P_4 (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 below 10 kΩ in all subjects (below 5 kΩ

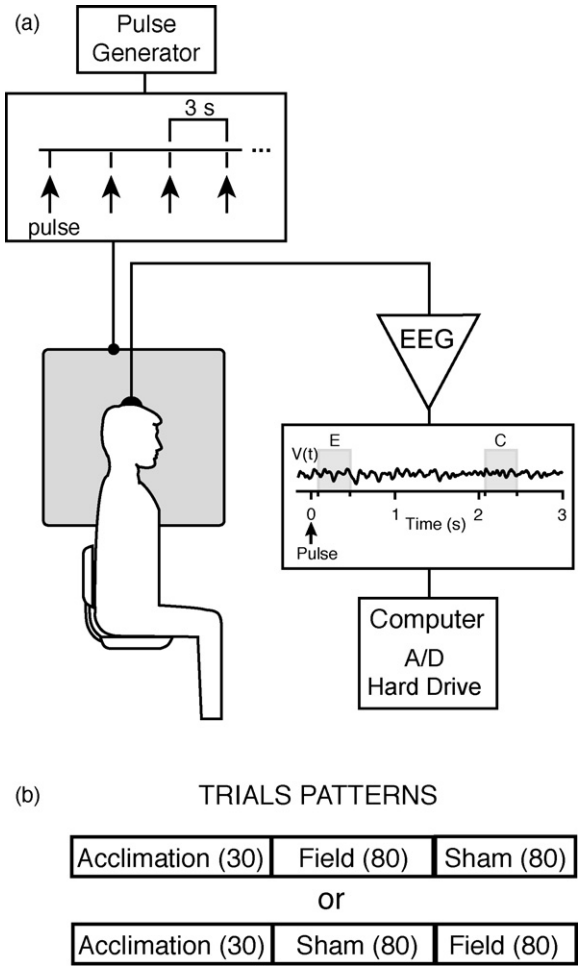

Fig. 2. Procedures for determination of effect of mobile-phone pulse on brain activity. (a) Schematic diagram of the exposure and EEG-detection systems; E (C), latency (control) epoch for assessment of the presence of an evoked potential. A/D, analog-to-digital conversion. (b) Sequence of experimental conditions; number of 3-s trials shown in parentheses.

in 90% of the derivations). The signals, $V(t)$, were amplified (Nihon Kohden, Irvine, CA, USA), analog filtered to pass 0.3–35 Hz, sampled at 300 Hz, and analyzed offline.

Application of the pulse produced a spike in the EEG of less than 1 ms which, in studies on electrical phantoms of the head, we established had been generated by Faradaic induction. The spike was removed from the signal by deleting the first 9 points (30 ms) from each epoch (see below) prior to analyses of the signal. Trials containing movement or other artifacts (as assessed by visual inspection) were discarded (<5% of the trials). The remaining trials were digitally filtered between 0.5 and 35 Hz, and the latency interval 76–471 ms (E epoch) was analyzed for the presence of an evoked potential by comparing each point in the interval with the corresponding point in 2.076–2.471 s (the control (C) epoch). All results were based on data from at least 50 trials.

Following an acclimation period, there were two experimental periods during which either a field or a sham field (no-field condition) was presented (Fig. 2b); the order of presentation of the field and sham varied randomly from subject to subject.

The method used to analyze the data was the same as previously described [3,5,6] and will be mentioned here only in summary. Epochs of interest in the EEG were embedded in five-dimensional phase space, and the resulting trajectory was mapped to a two-dimensional recurrence plot. The plots were quantified using two recurrence variables [21]: (1) percent recurrence (%R), defined as

the ratio of the number of points in the plot to the total number of points in the recurrence matrix; (2) percent determinism (%D), defined as the fraction of points in the plot that formed diagonal lines. The process was iterated, yielding the time series, $\overline{\%R(t)}$ and $\overline{\%D(t)}$ [20], which contained the determinism in $V(t)$ but in a more compact time interval. For example, the analyzed interval in $\overline{\%R(t)}$ (176–371 ms) corresponded to 76–471 ms in $V(t)$.

Each of the 60 points at 176–371 ms in $\overline{\%R(t)}$ and $\overline{\%D(t)}$ was compared with the corresponding point in the control epochs (Fig. 2a) using the paired t-test at a pair-wise significance level of $p < 0.05$. As previously [4], when ≥10 tests were pair-wise significant at $p < 0.05$, we regarded the result as demonstrating the presence of an EP.

Filtering the EEG to remove alpha frequencies facilitates detection of EMF-induced evoked potentials [4–6]; sometimes removal of 9–12 Hz but not 8–10 Hz was effective, and sometimes conversely. Use of $\overline{\%R}$ and $\overline{\%D}$ often gave the same result, but sometimes only one of them revealed a field-induced change in the EEG [6]. Based on these prior observations, we systematically considered all conditions of analysis previously shown capable of revealing an EMF-induced EP [6]. First, we analyzed $\overline{\%R(t)}$ in all 6 electrodes. If we found an EP (≥10 pair-wise significant tests within the expected latency interval) in at least 3 electrodes, no further analyses were conducted. If fewer than 3 EPs were found, we analyzed $\overline{\%D(t)}$. If a total of 3 EPs were still not detected, we filtered $V(t)$ prior to calculating $\overline{\%R(t)}$ and $\overline{\%D(t)}$ and continued the analysis until either 3 EPs were detected or all the 6 predetermined conditions (combinations of recurrence variable and filtering conditions) were considered. The overall results did not depend on the order; for presentation, we chose the sequence $\overline{\%R(t)}$, $\overline{\%D(t)}$, $\overline{\%R(t)}$ after filtering out 8–10 Hz, $\overline{\%D(t)}$ after filtering out 8–10 Hz, $\overline{\%R(t)}$ after filtering out 9–12 Hz, $\overline{\%D(t)}$ after filtering out 9–12 Hz.

Whenever tests were done to compare evoked potential and control epochs, the conditions being evaluated were also applied to the sham data (sham evoked potential versus sham control). We calculated the *a posteriori* false-positive rate (number of false-positive effects in the sham data divided by the total number of tests performed), and used that error rate to estimate the family-wise error (P_{FW}) for the decision that a subject had exhibited field-induced evoked potentials.

Evoked potentials were more likely to be observed at some derivations compared with others, depending on the stimulus [4,7]. Prior to the study we were unaware of how the probability for detection of pulse-induced EPs depended on derivation. We therefore computed the contributions to the family-wise error rate separately for the central, occipital, and parietal electrodes, using the binomial formula; P_{FW}, the error rate for the occurrence of EPs in each subject, was determined by the law of compound probability.

To examine for the presence of linear evoked potentials, the EEG was also evaluated directly (no unfolding in phase space) by time averaging [18]. The estimation of the *a posteriori* false-positive rate and the family-wise error was identical to the analysis used to evaluate the recurrence time series.

We regarded a potential as nonlinear if it was detected by recurrence analysis but not by time averaging.

Using the nonlinear variable $\overline{\%R(t)}$, brain potentials evoked by the simulated mobile-phone pulse were detected in 14 of 20 subjects (Table 1 , first data column). In subject S3, for example, when the E and C epochs in $\overline{\%R(t)}$ were compared point by point, an EP (>10 pair-wise significant tests) having the expected latency was observed at C_3 and P_3 (Fig. 3, left panels); sham-field exposure (the negative control procedure) yielded no false-positive results (<10 significant tests in each derivation (Fig. 3, right panels). A total of 120 statistical tests involving the $\overline{\%R(t)}$ time series were performed to evaluate the effect of the mobile-phone pulse (6 derivations × 20 subjects), resulting in 19 EPs (Table 1, first data column).

For subjects who exhibited EPs from fewer than 3 derivations, $\overline{\%D(t)}$ was computed and analyzed; EPs were found in S1, S7, S12, S13, and S15 that had not been detected with $\overline{\%R(t)}$ (Table 1, second data column). Filtering the EEG to remove 8–10 Hz or 9–12 Hz prior to computing $\overline{\%R(t)}$ or $\overline{\%D(t)}$ revealed additional potentials. For example, when the 8–10-Hz energy was removed from the EEG signals prior to computing $\overline{\%R(t)}$, previously undetected potentials were found in 5 subjects (S2, S7, S10, S14, S16). Overall, 90% of the subjects (18/20) satisfied the criterion for the presence of an effect (at least 3 pair-wise significant tests from any combination of derivations).

The *a posteriori* comparison-wise error rate was 21 false-positive tests in the sham data/470 total tests = 0.0413. We used this error rate to compute P_{FW}, the family-wise error for a decision that a subject detected the field; $P_{FW} < 0.05$ in 78% of the subjects (14/18), and $P_{FW} < 0.085$ in the remaining 22% of the subjects. There were no cases of false-positive results (no instances where >3 pair-wise significant tests were found in the sham data).

Neither the latency nor duration of the potentials depended on gender or electrode derivation (Table 2). When the value of the

Table 1
Evoked potentials in subjects exposed to mobile-phone pulse. Column heads indicate conditions of analysis. Effects in $\overline{\%D(t)}$ are shown in bold. X, evoked potentials not detected. Dashes indicate conditions not analyzed. P_{FW}, family-wise error for the decision that the subject exhibited evoked potentials. NE, no effect.

Subject	$\overline{\%R}$	$\overline{\%D}$	$\overline{\%R}$ (8–10 Hz)	$\overline{\%D}$ (8–10 Hz)	$\overline{\%R}$ (9–12 Hz)	$\overline{\%D}$ (9–12 Hz)	All Effects	No. Tests	P_{FW}
S1 (24/M)	P4	O1	X	**X**	O1 O2	–	O1 **O1** O2 P4	27	0.010
S2 (53/F)	C4	**C4**	C3 P3	–	–	–	C3 C4 **C4** P3	17	0.002
S3 (22/F)	C3 P3	**C3 P3**	–	–	–	–	C3 **C3** P3 **P3**	12	0.001
S4 (22/M)	C3 C4 P3	–	–	–	–	–	C3 C4 P3	6	0.001
S5 (22/F)	O1	X	X	O1 O2	–	–	O1 **O1 O2**	23	0.081
S6 (43/F)	O1 C4	**C4**	–	–	–	–	O1 C4 **C4**	12	0.006
S7 (22/F)	X	**P4**	O2	O2	–	–	O2 **O2 P4**	23	0.031
S8 (50/F)	X	X	X	**O2**	P3 P4	–	**O2** P3 P4	30	0.062
S9 62/M)	X	X	X	X	X	C4	C4	36	NE
S10 (18/F)	X	X	C3	C3	X	**O2**	**O2** C3 **C3**	34	0.078
S11 (36/F)	O2	**O2**	X	X	C3	–	O2 **O2** C3	27	0.043
S12 (47/F)	X	**C3**	X	X	P3	P4	**C3** P3 **P4**	34	0.085
S13 (32/F)	O1	**O1 P3**	–	–	–	–	O1 **O1 P3**	12	0.006
S14 (24/F)	O2	O2	O1	–	–	–	O1 O2 **O2**	17	0.038
S15 (52/M)	C3	**P3**	P3	–	–	–	C3 P3 **P3**	17	0.015
S16 (22/F)	X	X	C3 C4	C3 C4	–	–	C3 **C3** C4 **C4**	24	0.021
S17 (23/F)	C3	C3	X	X	X	X	C3 **C3**	32	NE
S18 (29/M)	C4	**C4**	X	X	P3	–	C4 **C4** P3	27	0.043
S19 (23/M)	P3	X	X	X	C3	C3 P3	C3 **C3** P3 **P3**	34	0.023
S20 (26/M)	P3 P4	P4	–	–	–	–	P3 P4 **P4**	12	0.015

Table 2
Latency and duration of evoked potentials stratified by gender and electrode derivation. Mean ± SD. *N*, number of evoked potentials (from Table 1).

	Gender		Electrode		
	Male	Female	Occipital	Central	Parietal
Latency (ms)	281 ± 51	267 ± 54	293 ± 55	275 ± 44	251 ± 55
Duration (ms)	264 ± 29	262 ± 27	254 ± 24	270 ± 28	263 ± 27
N	23	36	18	22	19

recurrence variable for each potential (Table 1) was compared with its control (expressed as a percent of the average of the sum), the change was sometimes greater than the control, and sometimes less, which is characteristic behavior for a nonlinear system. The average absolute value of the field-induced changes in the recurrence variables was 32% of the control, indicating a more robust effect than that typically observed in auditory or visual EPs.

Using time averaging, EPs were not detected in the EEG from any subject.

A stimulus equivalent to a pulse produced by mobile phones resulted in statistically significant effects on brain electrical activity in 18 of 20 subjects (Table 1). Several considerations indicated that the effects were true EPs. First, the analysis incorporated protection against family-wise error, which obviated an explanation based on chance. Second, comparable changes were not observed in the sham data. Third, the changes occurred several hundred milliseconds after the pulse, which was consistent with the inference that the changes arose from brain processing of afferent signals that resulted from transduction of the pulse. The observed latency was inconsistent with the possibility that the changes could have been generated by a field-electrode interaction because that process has no latency. Fourth, studies using phantoms of the human head verified the absence of electrode signals within the expected latency range.

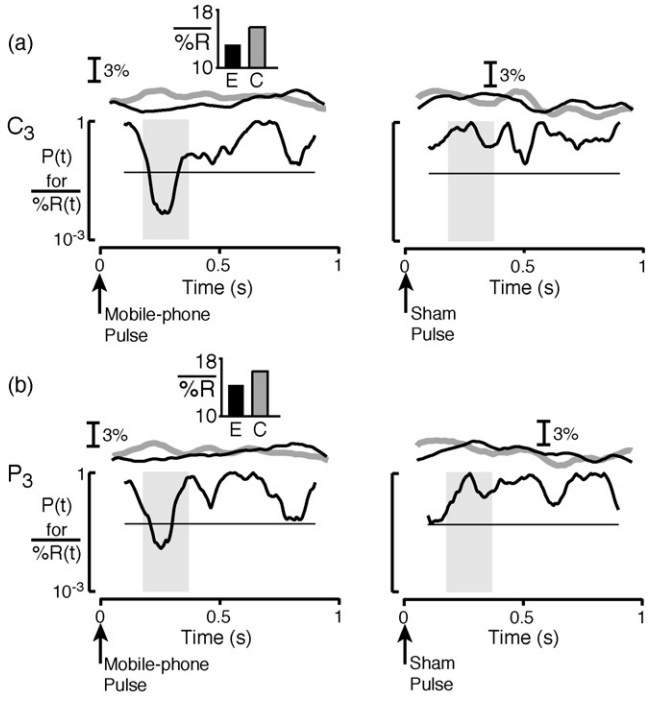

Fig. 3. Evoked potentials in subject S3, observed using the recurrence analysis variable $\overline{\%R(t)}$. Potentials from C_3 and P_3 are respectively shown in (a) and (b). Left panels, mobile-phone pulse stimulus; right panels, sham stimulus. The curves at the tops of the panels show the average values of $\overline{\%R}$ in the E and C epochs (Fig. 2a) ($N \geq 50$ trials). The $P(t)$ curves are the probability that the difference between the means of $\overline{\%R}$ in E and C at time t was due to chance. Bar graphs indicate the average value of $\overline{\%R}$ over the latency interval for which $P(t) < 0.05$ (horizontal line); the standard deviations are not resolved at scale shown. The stippled regions show the expected latency intervals (0.176–0.371 s).

Filtering within the alpha band was sometimes necessary for detection of the EPs (Table 1), as observed previously [4–6]. The rationale for removing alpha energy was that it did not contribute to the response, and therefore that removal of alpha increased sensitivity for detection of the EPs by increasing the signal-to-noise ratio in the system. The increased sensitivity afforded by alpha filtering might mean that the brain region where the alpha activities originate, usually assumed to be the cerebral cortex [19], was not crucial in the brain processing that gave rise to the EPs. This suggestion is consistent with the finding that the subject did not know the mobile-phone field was present even though the subject's brain did. Alternatively, the increased sensitivity afforded by alpha filtering might be related to differences among the subjects in their level of alertness during the experimental session.

The EPs were not detected when the EEGs were analyzed by time averaging, indicating that they were nonlinear in origin, as observed previously [4–6]. The finding that the changes in recurrence variables could be either an increase or a decrease further confirmed the nonlinearity of the response, because only nonlinear systems can exhibit such behavior.

We did not address the question of the anatomical location of the electroreceptor cell. The observed latencies (Table 2) were consistent with a location anywhere in the body. Animal studies, however, suggested the electroreceptor cell was located in the head, possibly the cerebellum [9,13]. The actual transduction process may involve ion channels having field-sensitive gating characteristics [12].

We used a simulated rather than actual mobile-phone pulse as the stimulus. We accepted this limitation to avoid confounding effects, and to increase the reproducibility of the effective stimulus. Nevertheless the pulse was represented verisimilarly enough that the brain potential it triggered could reasonably be imputed to that produced by an actual mobile-phone pulse.

In summary, a pulse of the type produced by mobile phones was transduced by 90% of the subjects studied, as indicated by the occurrence of EPs. The implication of our results is that mobile phones trigger EP at the rate of 217 Hz during ordinary phone use. One possibility is that chronic administration of the periodic changes in brain electrical activity causes or promotes disease.

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