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Short-term effects of GSM mobiles phones on spectral components of the human electroencephalogram

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Abstract— **The aim of the study was to investigate whether the GSM (Global System for Mobile) signals affect the electrical activity of the human brain. Nine healthy subjects and six temporal epileptic patients were exposed to radiofrequencies emitted by a GSM mobile phone signals. Electroencephalographic (EEG) signals were recorded using surface electrodes with and without radiofrequency. In order to obtain a reference, a control session was also carried out. The spectral attributes of the EEG signals recorded by surface electrodes were analyzed. The significant decrease of spectral correlation coefficients under radiofrequency influence showed that the GSM signal altered the spectral arrangement of the EEG activity for healthy subjects as well as epileptic patients. For the healthy subjects, the EEG spectral energy decreased on the studied frequency band [0 - 40 Hz] and more precisely on occipital electrodes for the α-band. For the epileptic patients, these modifications were demonstrated by an increase of the power spectral density of the EEG signal. Nevertheless, these biological effects on the EEG are not sufficient to put forward some electrophysiological hypothesis.**

I. INTRODUCTION

Mobile phones emit pulsed high-frequency electromagnetic fields that may have some adverse effects on the brain. Several studies have investigated the interaction between mobile phones and brain functioning in humans. Reiser [1] reported a change in the EEG tracing on exposure to 900 MHz radiation but Roschke and Mann [2] found no changes in healthy male volunteers exposed to GSM mobile phone signals. Borbely [3] found a slight reduction in the duration of waking, after sleep onset had occurred. Krause [4] reported EEG changes in healthy volunteers exposed to an electromagnetic field of 902 MHz during performance of an auditory task, and obtained similar results in subjects performing a visual memory task [5]. In France, the COMOBIO project (a program of the National Network for Research on Telecommunications) aimed at evaluating the medical effects of mobile phones. In this paper, we present works performed in the COMOBIO sub-project in which we were involved and which focused on the effects of GSM electromagnetic fields on human brain activity.

II. PROTOCOL

Two groups were considered: nine healthy subjects and six patients suffering from right temporal lobe epilepsy. These patients with intractable focal epilepsy resistant to anti-epileptic drugs have been studied under the hypothesis that they would show a different pattern of modifications in response to the radiofrequencies. All participants had normal hearing and took part in two recording sessions, one experimental and one control. The experimental session consisted of two sequential recording phases: in Phase 1, there was no radiofrequency (RF) emission; in Phase 2, the emission power was maximal. The control session was also composed of two phases which were the same length as in the experimental session, but, in each phase, the mobile phone did not emit any radiofrequency. The EEG activity was recorded on the surface of the scalp using a 32 electrode helmet lasting 250 seconds per phase. This continuous signal was sampled at 1 kHz and in order to delete the spurious frequencies at 50 Hz, 83.3 Hz, 133.4 Hz and 216.7 Hz revealed in [6], a low-pass filter with a 40 Hz cutoff frequency was applied.

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III. MATERIAL AND METHODS

With any information on EEG signal shape, it is difficult to judge RF influence by analyzing signals characteristics in the time domain. As a consequence, we turned to the frequency domain to study differences between the EEG signals recorded with or without RF. The analyzed band is [0 - 40 Hz].

A. Artifact rejection

During recordings, perturbations, such as eyes' blinking, may entail artifacts in the EEG signal (Fig. 1). So, the continuous EEG signal was first split into 250 epochs of 1000 time samples each. The localized power was computed for each epoch.

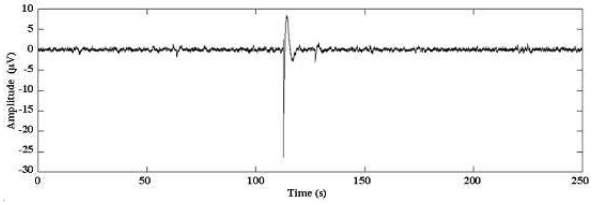

Fig. 1. EEG signal containing an artifact

Fig. 2 shows that an artifact made the localized power increase. To solve this problem, epochs having the highest localized power, with a 5% proportion of the entire number of epochs, were removed.

Then, a 2048-point FFT (Fast Fourier Transform) was applied to all the remaining epochs which were centered

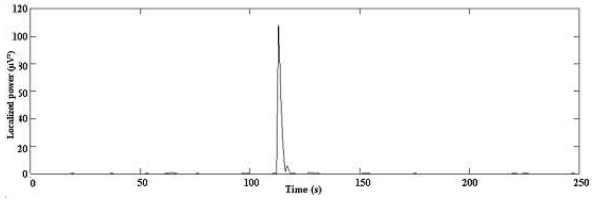

Fig. 2. Localized power computed on an artifacted signal

to remove the continuous component. For each phase and each electrode, we computed the power spectral densities (by using the periodogram method) in Phase 1 and in Phase 2 for each session, respectively named PSD_{Cont_1} and PSD_{Cont_2} for the control session, and PSD_{Exp_1} and PSD_{Exp_2} for the experimental session.

B. Spectral correlation coefficients

To evaluate the influence of RF on the spectrum global shape of the EEG signals, we analyzed the spectral correlation computed between two spectra of signals collected on the same electrode in Phase 1 and Phase 2, according to the formula:

$$\sum_{k=1}^{80} PSD_{S_1}(k).PSD_{S_2}(k) \quad (1)$$

where $PSD_{S_1}(k)$ and $PSD_{S_2}(k)$ are the power spectral densities computed on the frequency band [0 - 40 Hz], S denotes the session ($Cont$ or Exp) and k the frequency bin. Moreover, this correlation was normalized to obtain the correlation coefficient $CSpec$ equal to one if the spectra were exactly similar. For each session (control and experimental), we obtained these correlation coefficients between spectra, named $CSpec_{Cont}$ and $CSpec_{Exp}$, respectively.

C. Frequency bands power spectral densities

The second aspect of this study was based on the power spectral densities for different frequency bands. The EEG rhythms which are correlated with different behaviors such as the emotional level, the sleep and waking states, are conventionally classified [7] into different frequency bands: δ ($<$ 4 Hz), θ [4 - 7 Hz], α [8 - 12 Hz] and β [13 - 30 Hz]. Then, for the studied frequency band [0 - 40 Hz] and the δ-, θ-, α-, β-bands, the power spectral densities were estimated from EEG signals recorded in Phases 1 and 2 of the control and experimental sessions. To take into account the variability that exists between Phase 1 and Phase 2 [8], we computed the following parameters $\Delta PSD_S = PSD_{S_2} - PSD_{S_1}$, where S denotes the session ($Cont$ or Exp).

D. Method

Our objective was to compare the spectral correlations $CSpec$ and the variations ΔPSD between the experimental and control sessions. Therefore, the possible differences obtained between the control and experimental sessions may be attributed to the radiofrequencies. The Jarque-Bera test [9], based on skewness and kurtosis, indicating a normality rejection of $CSpec$ and ΔPSD, we had to use the non-parametric Wilcoxon signed-rank test [10] with a 95% confidence level on these parameters to define their evolution from the control session to the experimental one. At first, we computed the parameters $CSpec$ and ΔPSD for each healthy subject and each epileptic patient, using the 32 electrodes, in each session. In our results, we have indicated the mean for each session of $CSpec$ and ΔPSD for each subject with an upper bar and the mean on the whole group of subjects with a double upper bar. We also represented the topographic maps of the statistical comparison with the following colour code:

- dark grey: the parameter decreases significantly from the control session to the experimental one,
- white: there is no significant variation between parameters obtained in the two sessions,
- light grey: the parameter increases significantly from the control session to the experimental one.

IV. RESULTS

The results obtained on healthy subjects and on epileptic patients will be presented separately because the EEG signals of both groups are not comparable.

A. Spectral correlation coefficients

1) Healthy subjects: From the study carried out for each healthy subject, we noticed that for six subjects the $CSpec$ parameters were significantly lower in the experimental session than in the control one (TABLE I). The other subjects had a significant increase of $CSpec$ from the control session to the experimental one. If we perform the study on the nine healthy subjects, the Wilcoxon test showed that the $CSpec$ parameters obtained were significantly lower in the experimental session ($\overline{\overline{CSpec_{Exp}}} = 0.984$) than in the control one ($\overline{\overline{CSpec_{Cont}}} = 0.991$) with a p-value of less than 0.05.

TABLE I

$CSpec$ ANALYSIS FOR NINE HEALTHY SUBJECTS

Healthy subjects	$Cont \rightarrow Exp$	$\overline{CSpec_{Cont}}$	$\overline{CSpec_{Exp}}$	Significativity
CA	-	0.996	0.927	YES
EM	-	0.993	0.987	YES
LC	+	0.993	0.996	YES
MP	-	0.998	0.985	YES
MR	+	0.988	0.997	YES
NG	-	0.996	0.995	YES
OR	+	0.957	0.990	YES
SB	-	0.997	0.974	YES
SD	-	0.997	0.989	YES

The study on each electrode did not show any special area in which the radiofrequencies revealed some influence.

2) Epileptic patients: According to TABLE II, four epileptic patients had a significant decrease in the CSpec parameters when passing from the control session to the experimental one. Among the two others, one showed no

TABLE II

$CSpec$ ANALYSIS FOR SIX EPILEPTIC PATIENTS

Epileptic patients	$Cont \rightarrow Exp$	$\overline{CSpec_{Cont}}$	$\overline{CSpec_{Exp}}$	Significativity
BB	-	0.995	0.989	YES
CR	-	0.990	0.983	YES
LG	+	0.980	0.993	YES
MG	0	0.995	0.995	NO
SC	-	0.988	0.936	YES
TL	-	0.994	0.991	YES

significant variation (MG) and one a significant increase (LG). On the six epileptic patients, the radiofrequencies showed also a significant decrease of CSpec when passing from the control session ($\overline{CSpec_{Cont}} = 0.990$) to the experimental one ($\overline{CSpec_{Exp}} = 0.981$). In the same manner as for the healthy subjects, this decrease appeared on all electrodes.

B. Power spectral densities

The study on the $CSpec$ parameters showed that the radiofrequencies induced some modifications on the spectral components. Now, in a second step, we are directly interested by the contents of the power spectral densities.

1) Healthy subjects:

TABLE III

ΔPSD ANALYSIS ON [0 - 40 HZ] FOR NINE HEALTHY SUBJECTS

Healthy subjects	$Cont \rightarrow Exp$	$\overline{\Delta PSD_{Cont}}$	$\overline{\Delta PSD_{Exp}}$	Significativity
CA	-	0.010	-0.029	YES
EM	+	-0.017	0.044	YES
LC	-	0.020	0.002	YES
MP	+	0.012	0.030	YES
MR	-	0.074	-0.031	YES
NG	+	-0.010	0.003	YES
OR	+	0.047	0.076	YES
SB	+	-0.026	-0.009	YES
SD	-	-0.008	-0.122	YES

a) Frequency band [0 - 40 Hz]: The EEG spectral modifications observed by the $CSpec$ analysis were verified by the ΔPSD analysis on the entire frequency band [0 - 40 Hz] under study: four subjects (TABLE III) had a significant decrease of spectral power induced by the radiofrequencies, whereas five others had a significant increase. On the entire database of the healthy subjects, the Wilcoxon test with a p-value of less than 0.05 showed that the ΔPSD_{Cont} parameters ($\overline{\Delta PSD_{Cont}} = 0.012$) were higher than the ΔPSD_{Exp} ones ($\overline{\Delta PSD_{Exp}} = -0.004$). By carrying out the study for each electrode, we observed that this EEG spectral power decrease took place in parietal, centroparietal and central areas (Fig. 3).

b) δ-band: For the nine healthy subjects, the ΔPSD variations decreased when passing from the control session to the experimental one. This modification which took place in nearly all of the electrodes was the opposite to the result of Hietanen's study [11] which showed no radiofrequency influence on the EEG δ-band.

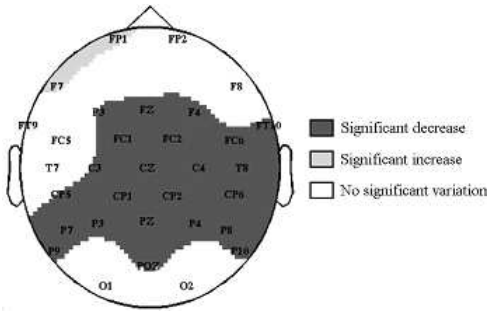

Fig. 3. Scalp topography of p-values from the Wilcoxon test on ΔPSD computed on the frequency band [0 - 40 Hz] for the healthy subjects

c) θ-band: With a p-value of less than 0.01, the Wilcoxon test showed a ΔPSD decrease in the θ-band and more precisely for some electrodes in parietal area without any laterality (Fig. 4a).

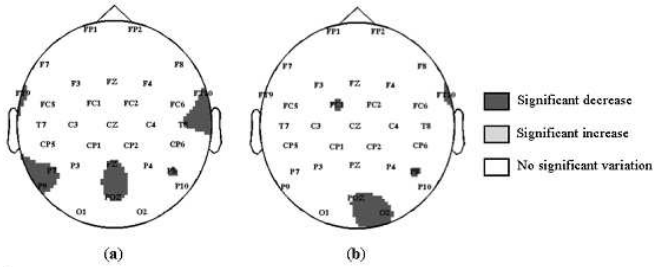

Fig. 4. Scalp topography of p-values from the Wilcoxon test on ΔPSD computed on the θ-band (**a**) and on the α-band (**b**) frequency for the healthy subjects

d) α-band: In this band, the radiofrequencies also permit a decrease of ΔPSD in a significant manner. Few electrodes showed this modification, and like Parts' study [12], an attenuation of EEG energy in the α-band was observed on occipital electrodes (Fig. 4b).

e) β-band: Finally, we observed a significant decrease of ΔPSD in this EEG frequency band due to the radiofrequencies. Furthermore, there was no specific area for which this modification was observed.

2) Epileptic patients:

a) Frequency band [0 - 40 Hz]: TABLE IV shows that all the epileptic patients presented ΔPSD parameters computed in control session that were lower than the ones obtained in the experimental session. For the entire database of epileptic patients, the ΔPSD_{Exp} values ($\overline{\Delta PSD_{Exp}} = 0.052$) were significantly higher than the ΔPSD_{Cont} values ($\overline{\Delta PSD_{Cont}} = -0.026$) with a p-value of less than 0.05.

The study per electrode showed that the EEG spectral power increase took place on the whole scalp.

b) δ-band: For the six epileptic patients, the radiofrequencies emitted by the mobile phone produced an increase of EEG energy in this band. This modification was found on the electrodes close to the vertex (Fig. 5).

c) θ-, α- and β-bands: Finally, for the θ-, α- and β-bands, with a p-value of less than 0.05, the Wilcoxon test

TABLE IV

ΔPSD ON [0 - 40 HZ] ANALYSIS FOR SIX EPILEPTIC PATIENTS

Epileptic patients	$Cont \rightarrow Exp$	$\overline{\Delta PSD_{Cont}}$	$\overline{\Delta PSD_{Exp}}$	Significativity
BB	+	-0.022	0.021	YES
CR	+	-0.014	0.036	YES
LG	+	-0.047	-0.028	YES
MG	+	-0.011	0.019	YES
SC	+	-0.046	0.168	YES
TL	+	-0.015	0.095	YES

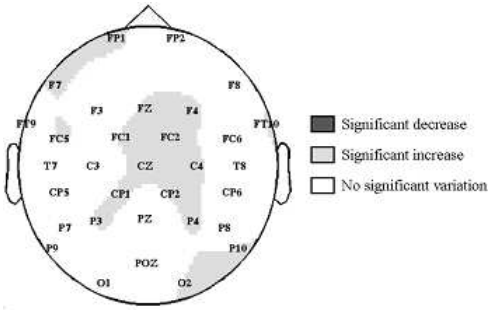

Fig. 5. Scalp topography of p-values from the Wilcoxon test on ΔPSD computed on the δ-band frequency for the epileptic patients

showed a significant increase of ΔPSD variations allowed by the radiofrequencies. For the three bands, almost all the electrodes showed this increase.

V. CONCLUSION

The present study deals with the influence of GSM radiofrequencies on the EEG signals recorded by surface electrodes. For the healthy subjects and epileptic patients, the correlation coefficients in the frequency domain showed some modifications caused by mobile phones. A significant decrease of this coefficient meant that the GSM signal emitted by the mobile phone modified the spectral attributes of the EEG signals. By analyzing the power spectral density of the EEG signals, we studied more precisely the modifications caused by the radiofrequencies in different frequency bands. For the healthy subjects, we observed for all frequency bands a significant decrease of the EEG signal energy in the presence of radiofrequencies. The meaningful result was the EEG signal energy decreases for the α-band in the occipital region. For the epileptic patients, it was the opposite result since the mobile phone signals produce an increase of EEG signal energy in all frequency bands and on all electrodes without actual localization. Based on the current analysis, it seems that the GSM mobile phone does have a biological effect on EEG signals. Nonetheless, it is difficult to compare them to those of previous studies. Reiser [1] found an increase in the EEG waves starting approximately 15 minutes after the exposure, using two separate sources of electromagnetic fields, *i.e.* a therapy instrument (150 MHz) and a mobile digital phone at 900 MHz. In Roschke's study [2] using the same source of electromagnetic fields as in our study, no differences were observed in the spectral power densities of the EEG, between exposure and sham-exposure conditions. On the other hand, there was a difference of the experimental protocol because the mobile phone was placed at 40 cm from the top of the head leading a different specific absorption rate. Finally, although some significant differences are found in analyzing EEG signals, this study is not sufficient to put forward some electrophysiological hypothesis.

VI. ACKNOWLEDGMENTS

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