Influence of Mobile Phone Electromagnetic Field Exposures on Nervous Function in the Human Brain and Heart

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Summary

It is as yet undetermined whether mobile phone exposures can cause any biological changes and effects that may lead to adverse health effects in humans. At a distance of within 2 cm from a user's head mobile phones can radiate radiofrequency (RF) pulsed signals in the range of 450 - 2500 MHz at specific energy absorption rates (SARs) of no more than 2 W/kg (as measured over 10 grams of tissue), which is a recognised general public limit. At present, the frequency range of mobile phones is known to be far too high to cause cell excitation (occurring at frequencies < 100 kHz) and SAR levels respectively are too low to implicate levels of heating which may cause temporary brain dysfunction such as lowered performance in a complex learned task (associated with SARs in the order of 4 W/kg, or an excess of a 1˚C temperature rise). Accordingly, it is presently not understood whether mobile phone exposures can cause biological effects at all. Nevertheless, given that recent statistics have indicated that the number of mobile phone users worldwide currently exceeds 1.7 billion people, a comprehensive health risk assessment of mobile phone usage is crucial.

This thesis aims to determine whether RF pulsed exposures from mobile phones can influence nervous function within the human brain and heart. In order to address this objective, a robust double-blind study to investigate mobile phone exposures on nervous response has been undertaken by means of analysis of electrophysiological test measures of brain wave activity and heart responses. In general, test measurements of the electroencephalogram (EEG), the electrocardiogram (ECG), and the pulse plethysmogram (PPG) (for indications of blood pressure response) have been undertaken whilst participants are exposed and sham exposed to various GSM mobile phone radiofrequency and low-frequency protocols. In initial work, both

standby and full-power mode GSM exposures have been investigated in a pilot study (Chapter 3 - EEG investigated only), while pulsed 900 MHz and 1800 MHz continuous wave exposures have been investigated in the final experimental work involving 100 participants (Chapter 8 – EEG, ECG, and PPG measures are investigated).

In order to prepare for the conduct of the major study, several preliminary investigations were undertaken to address identified gaps or inadequacies in the current body of literature. This work has led to the development of several novel investigations, solutions, and findings, which form the basis of the thesis Chapters. A number of important points of note that summarise the original contribution of work to the field of research are:

1. An original test handset has been developed to test multiple mobile phone exposures on test participants. This was specifically undertaken to overcome experimental confounding factors such as acoustic cues, heating, and low frequency interrupts, that would otherwise be present with use of a real mobile phone
2. Comparative analysis of results at two mobile phone exposures with carriers 900 MHz and 1800 MHz on the same group of participants in simultaneous experiments, has not been performed earlier, and has importantly contributed to observation of new findings
3. New characteristic changes not reported by others, are demonstrated near to the health protection limits for both exposures investigated, 900 MHz and 1800 MHz
4. The major investigation conducted is the largest known to be undertaken in terms of the number of participants (N = 100) where work has been conducted on the resting EEG effects due to mobile phone exposures in the eyes closed condition (at 900 MHz and 1800 MHz)

5. A novel statistical analysis method which accounts for both type 1 and type 2 error correction is presented. The novel analysis method has been critical for observing previously unseen changes in human brain wave activity
6. New interesting findings are presented, which quantify a characteristic change within the EEG alpha band (8 – 12 Hz) and demonstrates a most interesting new finding, which is specifically that there appears to be a consistent decrease at 900 MHz but a consistent increase at 1800 MHz in power spectral density both at 16 Hz. This result is especially interesting as the experimental protocol required both exposures to tested be over a randomized, counterbalanced sequence
7. Complete quantification of the electric field distribution (at both 900 MHz and 1800 MHz) inside and around a realistic computational head model fitted with modeled electrode leads in a standard array is new. The outcomes of this work is critical to the understanding that EEG leads in the presence of mobile phone exposures do not significantly change recorded SAR levels (changes of 4 - 5 %) inside the heads of test participants. New computed findings that EEG lead lengths are also variables in the behaviour of the electric field distribution inside the head is also an important outcome
8. Original experimental protocol permitting the measure of blood pressure response during the actual time of exposure is presented. Previously, studies have shown that blood pressure requires to be measured manually using an arm-cuff *following* exposure so that movements (sitting and standing) do not interfere with brain-wave activity recordings. The experimental approach incorporated into this study overcomes this dilemma by utilizing an analysis of the recorded 'pulse-plethysmograph' or PPG

alongside the electrocardiograph (ECG), in order to determine simultaneous changes in blood pressure *during* the time of exposure.

Several important outcomes have also been determined from the main experimental work conducted (as described in further detail in Chapter 8). These outcomes specifically include:

- An observed 'characteristic' response in the EEG alpha band (8-13 Hz) as due to 900 MHz exposures at almost all recording sites in both hemispheres. Notably, this response only appears at 900 MHz although is not observable at the 1800 MHz exposure tested. The characteristic response seen in the EEG is considered an important finding as it has not been identified previously, yet it seems to go some way in explaining the partial effects observed in alpha rhythms by others
- An EEG effect due to both 900 MHz and 1800 MHz exposures notably appearing at 16 Hz is observed from almost all recording sites. Most importantly, at this beta rhythm only a decreasing tendency in EEG power can be noted amongst the 16 electrode sites tested at the 900 MHz exposure but only an increasing tendency in EEG power may be noted at 1800 MHz, suggesting that the direction of the effect in EEG power may be dependent on the RF component
- Effects only due to the 'RF' components of 900 MHz and 1800 MHz appear to influence the EEG, and not the 217 Hz pulse component
- Effects at 900 MHz on the contralateral side to exposure, namely the central, temporal, and parietal sites appear to indicate a common median difference response between sham and exposure EEG spectral power, which is not evident on the ipsilateral side to exposure

- Exposures of 900 MHz or 1800 MHz (as pulsed at 217 Hz) *do not* appear to alter heart rate, heart rate variability (HRV), or blood pressure variability (BPV). This result appears to be consistent with most previously published research as indicated for heart and blood pressure responses

All findings determined by the experimental work conducted can be classified into the category of 'short-term' effects as particularly given that maximum exposure durations did not exceed five-minute intervals for any given trial.

In regards to future work, several major points of recommendation are suggested:

1. A very limited amount of studies have been conducted on children to investigate mobile phone exposure effects on brain and heart activity. Although it is understood that the brain response is not fully developed until adulthood, there appears to be no real physiological restriction in examining younger participants of the same age, for example, 50 male 13 yr olds. Examining children for such effects is crucial particularly as it is possible that maturing brain at earlier ages could be more susceptible to adverse health consequences
2. Investigation of longer duration times of continuous mobile phone exposures on test participants at rest is in need to be undertaken in both brain and heart investigations. The present body of literature at best generally indicates outcomes of 30-minute continuous mobile phone exposures. While these duration times are absolutely adequate for trials it would be interesting also to see the effects of mobile phone exposures on brain and heart activity at durations up to one hour or more. In such a study, it would be recommended that vigilance activities such as auditory or visual

tasks would need to be investigated and wisely incorporated during exposure times, in order to sustain consistencies in recorded brain and heart data

3. Based on the results contained within this thesis, the EEG rhythm 16 Hz is strongly outlined as a point of new interest for susceptibility to phone exposures. Consequently, it is recommended that the spectral power density within this band be robustly quantified in any future research. In analysis, care should be taken not to just consider grand-averaging across EEG bands, which as explicitly indicated in Chapter 7, can most easily mask the potential RF effect at 16 Hz
4. The alpha band in EEG studies is still raising ongoing concerns that a change is present in brain waves due to various mobile phone exposures. Future work should focus on incorporating a rigorous high standard of statistical analysis, not yet consistently observed. Replication of the statistical analysis proposed in this thesis is highly recommended
5. Finally, a call for better RF dosimetry, larger sample sizes (at least 50 participants), and double blinding, are all critical factors that need to be incorporated into future research to improve on the quality and integrity of outcomes in both brain and heart research. Observation of Tables 2.1 – 2.4 within the thesis highlight these existing inadequacies

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Chapter 1

Background & Introduction

The following Chapter provides a necessary background and introduction to the investigation of the effects of mobile phone exposures on nervous function in the human brain and heart. Specifically, this chapter outlines the problem associated with mobile phone exposures with respect to health implications, provides a brief on theoretical concepts associated with electromagnetic fields, discusses important aspects regarding the specific energy absorption rate, and outlines the concept of *athermal* effects and mobile phone exposures. In addition, a concise background on the nervous system is provided alongside a necessary outline of the origins of the electroencephalogram (EEG), the electrocardiogram (ECG), and blood pressure.

1.1 The Issue Associated with Mobile Phone Exposures and Human Health

During operation, mobile phones can emit energy in the form of electromagnetic fields, which can be absorbed by a user's head. However, it is presently unclear whether this electromagnetic energy can cause biological consequences or adverse health effects. Mobile phone usage around the world is enormous and is still on the rise. Currently, it is estimated that there is over 1.7 billion mobile phone users globally. Consequently, if there were to be adverse health implications due to exposures from mobile phones, the effects could be widespread amongst vast populations. It is thus vital that all present and valid possibilities of biological effects due to mobile phone radiated fields be examined and resolved.

Various studies so far examining biological effects of mobile phone exposures have included the investigation of potential connections to cancer, cell division, blood pressure alteration, induction of epilepsy, depression in melatonin levels, DNA strand breaks, effects on the eyes, and human cognitive alteration, to name a few. However, although many of these branches of studies continue to provide plausible leads for means of further research, as it stands only questionable evidence supporting the existence of mobile phone exposures causing biological changes may be indicated.

1.2 Theoretical Concepts of Electromagnetic Fields

Exposures generated from mobile phones are produced in the form of Radiofrequency (RF) Electromagnetic Fields (EMFs). EMFs can be mathematically defined in free space using four field quantities namely the Electric field intensity (E), the Electric flux density (D), Magnetic flux density (B), and Magnetic field intensity (H). The four fundamental EMF quantities and corresponding Units are summarised in Table 1.1 below [Cheng D., 1989].

Field	Field Quantity	Symbol	Unit
Electric	Electric field intensity	**E**	V/m
	Electric flux density (Electric displacement)	**D**	C/m²
Magnetic	Magnetic flux density	**B**	T
	Magnetic field intensity	**H**	A/m

Table 1.1 Indicated are the four fundamental electromagnetic field definitive quantities. Source: Cheng D., 1989

In static EMFs, the electric (**E** and **D**) and magnetic (**H** and **B**) fields form two individual vector quantities. However, this is unlike *time-varying* electromagnetic fields, in which case time-varying magnetic and time-varying electric fields are uniformly coupled and propagate dynamically by one field component continuously producing the other over distance. The velocity of EMFs in free space is known to be a constant 299,792,458 m/s

(c, or the speed of light). The medium in which the EMFs exist, directly influence the relationship between **E** and **D** and between **B** and **H.** The relationship in free space between electric flux density **D** and electric field intensity **E** as well between the magnetic flux density **B** and the magnetic field intensity **H** is indicated in Table 1.2 below [Cheng, D., 1989][Benson, H., 1991]. As transverse waves, the electric and magnetic field components of EMF plane waves exist at right angles to one another and also at right angles to the direction of the wave as depicted in Figure 1.1 below.

Relationship between the Electric and Magnetic field quantities	**Applicable Constants**
$\mathbf{D}=\varepsilon_0\mathbf{E}$	Permittivity of free space, $\varepsilon_0 \cong 8.854\times10^{-12}\ \mathrm{F/m}$
$\mathbf{B}=\mu_0\mathbf{H}$	Permeability of free space, $\mu_0 = 4\pi\times10^{-7}\ \mathrm{H/m}$
Relationship between the field constants and EMF velocity: $c=\frac{1}{\sqrt{\varepsilon_0\mu_0}}$	Speed of light, $c \cong 3\times10^{8}\ \mathrm{m/s}$

Table 1.2 Indicated is the mathematical relationship between the Electric and Magnetic field quantities of electromagnetic fields as defined in free space

James Clerk Maxwell was able to describe the propagation of time-varying electromagnetic waves through four fundamental equations (as valid in any point in free space). The differential, and corresponding integral forms of equations are indicated below in Table 1.3 [Cheng D., 1989].

Maxwell's Electromagnetic Field Equations	
Differential form	Integral form
$\nabla \times \mathbf{H} = \mathbf{J} + \frac{\partial \mathbf{D}}{\partial t}$	$\oint \mathbf{H} \cdot d\ell = I + \int \frac{\partial \mathbf{D}}{\partial t} \cdot d\mathbf{s}$
$\nabla \times \mathbf{E} = -\frac{\partial \mathbf{B}}{\partial t}$	$\oint \mathbf{E} \cdot d\ell = -\frac{d\Phi}{dt}$
$\nabla \cdot \mathbf{D} = \rho$	$\oint \mathbf{D} \cdot d\mathbf{s} = Q$
$\nabla \cdot \mathbf{B} = 0$	$\oint \mathbf{B} \cdot d\mathbf{s} = 0$

Table 1.3 Indicated are both the differential and integral forms of Maxwell's equations which define time-varying electromagnetic fields as valid for any point in free space

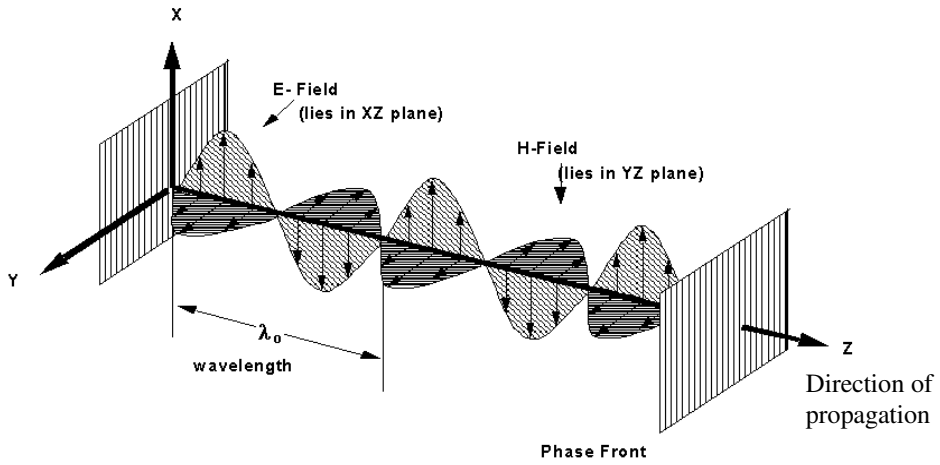


Figure 1.1 Indicated is a theoretical representation of the relationship between the Electric and Magnetic fields as described for EMF propagation as a plane wave (source: www.ehb.itu.edu.tr/~sparker/em_review)

Over a significant propagated distance, EMFs may be categorised into two regions namely the near-field and far-field. The near-field can be further divided into two regions namely the *reactive* near-field and the *radiating* near-field. While specific field characteristics of an EMF may be quantified accurately by calculation in the far-field, in practice near-field calculations can carry large uncertainties given that in this region plane wave characteristics have not been established and the relationship therefore between the field quantities can be highly non-linear. The reactive near-field is definitively the distance within $\lambda/2\pi$ from the radiating source, where λ is the wavelength. Therefore, the reactive near-field distance from a mobile phones radiating antenna at 900 MHz or 1800 MHz can be found to be approximately 5.3 cm or 2.65 cm respectively (notably within the region of a mobile phone user's head). On the other hand the far-field distance may be defined as the region extending beyond $2D^2/\lambda$ (considering an antenna dimension D) or 0.5λ, whichever is the greater [ARPANSA Std., 2002]. In the far-field region the electromagnetic field from the mobile phone has formed a uniform plane wave such that the ratio of electric field intensity 'E' to the magnetic field intensity 'H' is constant. It follows that in free space:

$$\frac{\mathbf{E}}{\mathbf{H}} = \sqrt{\frac{\boldsymbol{\mu}_0}{\boldsymbol{\varepsilon}_0}} = \mathbf{376.7\ \Omega \approx 377\,\Omega}\ \text{(wave impedance)} \qquad \text{(Eq. 1.1)}$$

In the radiating near-field region the finite angular field extent is known to be dependent on the distance from the radiating source and is defined as the region between the reactive near-field and the far-field boundary [ARPANSA Std., 2002].

Far-field estimations of field strength may be given by the power flux density equation:

$$\mathbf{S} = \frac{PG}{4\pi d^2} \qquad \text{(Eq. 1.2)}$$

where S is in units of W/m², P is the power to the antenna in Watts, G is the linear isotropic gain, and d is the distance from the antenna in metres. Corresponding power flux density expressions in terms of E and H are as follows:

$$\mathbf{S} = \mathbf{E} \times \mathbf{H} \qquad \text{(Eq. 1.3)}$$

$$|\mathbf{S}| = \frac{|\mathbf{E}|^2}{377} = |\mathbf{H}|^2 \qquad \text{(Eq. 1.4)}$$

1.3 The Specific Absorption Rate and Athermal Biological Effects due to Mobile Phone Exposures

"Most radiofrequency (RF) field standards are based on the premise that there exists a threshold specific absorption rate (SAR) of RF energy (for frequencies above about 1 MHz) of 1-4 W/kg, above which there is increasing likelihood of adverse health effects" [WHO EH Criteria 137, 1993].

Definitively, SAR is a measure of the rate of energy absorption per unit mass, and is most often expressed in terms of Watts per kilogram (W/Kg) [IEEE 1528 Std., 2003] given that:

$$\mathrm{SAR} = \frac{\sigma |\mathbf{E}|^2}{\rho} \qquad \text{(Eq. 1.5)}$$

where σ (S/m) is the electrical conductivity, E is the RMS value of the electrical field strength vector (V/m), and ρ is mass density of the medium (kg/m³). In standard practice limitations of the amount of RF energy that communication devices may introduce to a user are identified in terms of SAR [ICNIRP, 1998]. SAR is presently determined by robust measurements of the electric field strength using a miniature automated E-field probe inside a specific head model phantom filled with liquid with dielectric equivalent properties for head tissue. Inside the tissue-equivalent liquid, SAR may also be expressed in terms of the rate of temperature rise as is defined by:

$$\mathrm{SAR} = \left.\frac{c\Delta T}{\Delta t}\right|_{t=0} \qquad \text{(Eq. 1.6)}$$

where c is the specific heat capacity, ΔT is the change in temperature, and Δt is the exposure duration. However, currently SAR in terms of temperature is difficult to dynamically assess in practice particularly as there are limitations to the sensitivity and inter-ranges within human head phantoms to which temperature probes can measure low-power devices [IEEE 1528 Std., 2003].

Mobile phones are permitted at most in some countries to produce SAR levels ranging up to 2 W/kg while exposures from mobile phone antennas have been estimated to produce temperature rises inside the head of up to approximately 0.6˚ C [Hirata et al., 2003]. Although mobile phones can produce low-level heating in the head it is currently accepted that the body's normal thermoregulatory system may adequately compensate for this low-level energy deposit. Consequently, given that there is no adequate scientific evidence to substantiate biological effects due to RF fields at subthermal or *athermal* levels, mobile phone exposures are not found to initiate adverse health effects. However, on the other hand established thermal consequences of RF fields as principally arising due to incident *electric* field interaction causing ionic motion and rotation of polar molecules (of mainly water compositions), are well-defined [WHO EH Criteria 137, 1993][Carpenter & Ayrapetyan, 1994]. Coupling of low-frequency time-varying RF *magnetic* fields to the human body can also occur that in turn induce electric fields and currents at magnitudes proportional to the radius of the induced electric field as well as

the electrical conductivity of tissue and the magnitude and gradient of the magnetic flux density [ARPANSA Std RPS3, 2002].

In terms of athermal RF biological effects, related investigations can be found in excess of several thousand, nevertheless there still remains debate as to whether RF biological effects as resulting from mechanisms other than heating actually exist. Interestingly, Adair [Adair, 2003] summarised in existing biophysical terms reasons why some believe that it is theoretically improbable that there are athermal effects of low intensity RF and microwave exposures. Specifically, noted RF interactions and corresponding characteristics addressed on the molecular scale were inclusive of rotational motion of molecules with significant dipole moments, opening of voltage gated channels, electrostrictive forces on cells and induced dipole moments, molecular resonances, as well as magnetic field effects (free radical pair recombination). The examination of these interactions was conducted in the perspective that external influences generating a response would not be masked by biological endogenous noise. Adair concludes, "*I hold that our prior knowledge establishes that it is unlikely that weak fields generate any biological effect. Hence, any experimental results that seem to indicate such effects, effects that are a priori most improbable, must be especially definitive.*"

1.4 Nervous Response in the Human Brain and Heart

The underlying aim of investigations conducted and described in this thesis is to determine whether nervous function in the human brain and heart can be influenced by mobile phone use. In Chapter 3 a pilot study is conducted to investigate the effects of both standby and full-power mode GSM signals on the EEG alone, while Chapter 8 describes the major study incorporating 100 participants where both pulsed 900 MHz and 1800 MHz RF signals are investigated on measures of the EEG and ECG as well as on blood pressure responses via measures of the pulse plethysmograph (PPG). Hence a necessary background to the nervous system is provided with an adequate brief on the EEG, ECG, and blood pressure.

The nervous system may be divided into two formally categorised systems namely the central nervous system (CNS) and the peripheral nervous system (PNS). The peripheral nervous system may also be further divided into two complex networks, namely the sensory somatic system (SNS) and the autonomic system (ANS). For the purpose of this research, it is important to identify that the brain is in particular part of the CNS (together with the spinal cord), while the heart is part of the ANS, by which it may be regulated. As can be observed in Figure 1.2 below, the CNS (as concerned with the brain) and the ANS (as concerned with aspects of heart regulation) are connected to one another at the spinal cord.

The ANS may be divided into two further systems, namely the sympathetic system and the parasympathetic system. The sympathetic nervous system is known to function as an

involuntary preparatory system for an emergency, while the parasympathetic system is known to *involuntarily* rebalance the body - In these circumstances the sympathetic impulses may activate to increase heartbeat and constrict arteries, while parasympathetic impulses may reduce heartbeat and dilate the arteries [Alcamo & Krumhardt, 2004].

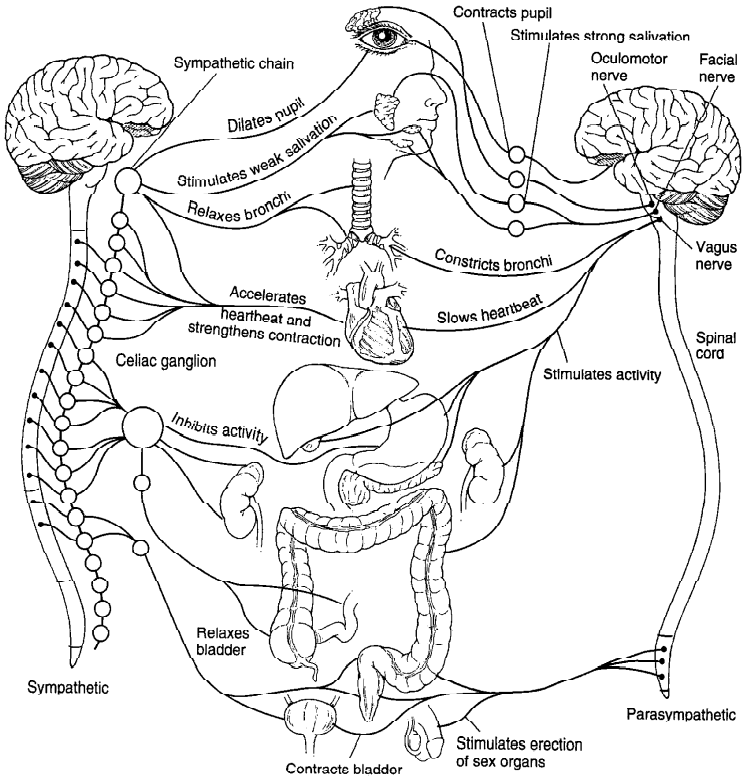


Figure 1.2. Shown is the general network of nerve fibers of the autonomic nervous system within the human body as identified by the sympathetic and parasympathetic nervous systems (Picture source: Alcamo & Krumhardt, 2004)

The medulla oblongata, which forms a major part of the 'brain stem' connecting the brain to the spinal cord, is known to coordinate the sympathetic impulses that regulate heartbeats as well as blood pressure. Blood pressure can be thought of as the pressure that blood forces upon arterial walls within the body. In particular, sympathetic neurons within the medulla oblongata called the 'vasomotor center' can function for example to reduce the diameter of arterial walls, which in turn would increase blood pressure.

1.4.1 Measurement of the EEG, ECG, and Blood Pressure

Unlike blood pressure, the brain and heart can produce measurable electrical responses. Neurons in the brain can produce summated electrical potentials at the cellular level in the order of milli-Volts. In particular, 'pyramidal' cells that are located in the cerebral cortex (the outermost layer of the brain) generate moving currents in the extracellular space amounting from dipoles produced across the active neurons. Potential differences existing between clusters of pyramidal cells themselves and the scalp cause electrical current to flow to the scalp surface where it may be measured by electrodes in the order of micro-Volts [Fisch BJ, 1999]. An electrical current measured over time at the scalp surface will produce a resulting 'EEG' signal. EEG signals may be measured as the electrical difference between two brain site locations such as across the frontal, central, or occipital hemispheres of the brain, called differential recordings, or likewise may be measured as a referential recording of the difference between a ground reference and a single scalp location in order to obtain its full magnitude. An example of a real-time EEG

recorded signal is provided below alongside a Fast Fourier Transform (FFT) of the signal to demonstrate its spectral characteristics (Fig 1.3). Notably, the spectral distribution may be divided into four clinically significant bands of which signal strength has been found to correspond directly to different behavioural states. For example, relatively higher theta rhythms (~4 - 8 Hz) are known to correspond strongly to sleep [Steriade M, 1993], while awake and relaxed states may cause prominent alpha rhythms (~8-13 Hz) and may particularly be found to correspond to long-term memory performance [Klimesch, 1998]. Indeed oscillations of the EEG have been shown to be driven by thalamo-cortical activity [Silva et al., 1991][Sternman, 1996][Mochizuki et al., 2005], although it should be stated that mechanisms of the substantiated rhythmic generation of the EEG is still largely not understood. Power spectral density of the EEG may extend up into the order of 100 μV^2 for referentially recorded responses within the clinically significant range of ~1 – 32 Hz, however responses of much lower spectral densities may be found to extend up to gamma rhythms in the order of 80 Hz where investigations of neuropsychiatric disorders such as schizophrenia may be investigated [Herrman & Demiralp, 2005].

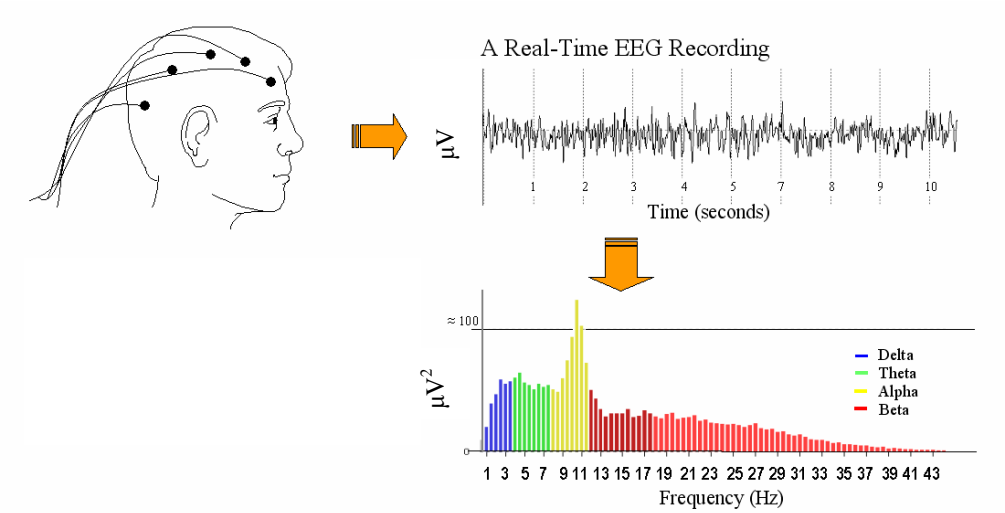


Figure 1.3. Shown is a real-time EEG signal as well as its spectral distribution as generated by performing a FFT. The spectral distribution as illustrated may be divided into four clinically significant bands namely, delta, theta, alpha, and beta.

In comparison to the real-time complex signal of the EEG, the ECG provides a far more basic electrical response. Nonetheless, the ECG provides some very useful information about heart functionality. The ECG may be identified by a characteristic waveform that represents one heart cycle or heartbeat (Figure 1.4). In adults the number of heartbeats per minute typically ranges from approximately 60 – 80, however in newborn children expected heartbeats can range up to and around 150 beats per minute. Each measured heartbeat may be characterised by the P-wave, QRS-wave, and the T-wave as illustrated in Figure 1.4 below. The cardiac cycle begins by 'self-excitation' of the heart's atria muscle through *Purkinje fibers* by impulses sent from the 'sinoatrial' (SA) node located in the right-atrium (Figure 1.4). These impulses firstly depolarise and contract cells of 'autorhythmic' tissue, causing the atria to contract (cycle represented by the P-wave).

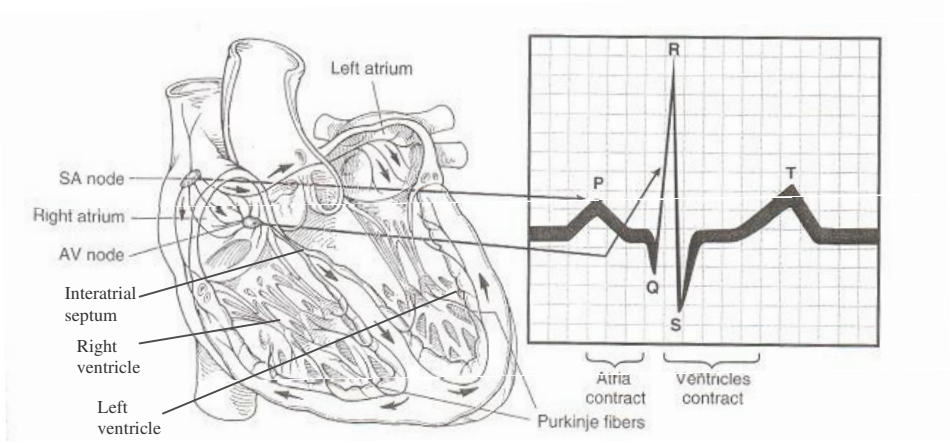


Figure 1.4 Shown is a typical real-time ECG waveform as identified by its characteristics, namely the P-wave, QRS-wave, and T-wave. As illustrated, the atria and ventricle contractions of the heart through one cardiac cycle or heartbeat, correspond directly to the ECG's wave characteristics (Picture source: Alcamo & Krumhardt, 2004)

Next, a second node called the 'atrioventricular' (AV) node, located in the interatrial septum (Figure 1.4), processes signals received from the SA node that are processed through a bundle of Purkinje fibers (the bundle of His) to then contract left and right ventricles of the heart (represented by the QRS-wave). Repolarisation of the heart allows for the recovery from the ventricular contraction (as represented by the T-wave). After a regulated duration by the SA node, the heart is prepared to repeat the process.

Formal measurement of the ECG can be obtained via surface electrodes positioned across the body such as in a standard 12-lead ECG, that is across the chest, wrists, and legs [Rautaharju P. and Rautaharju F., 2007]. Leads measuring voltage potential across the body caused by the depolarisation and repolarisation of heart function may also be

determined by ‘unipolar leads positioning’ requiring leads on the limbs only, such as on both wrists and one on the ankle [Wilson et al., 1934][Goldberger, 1942].

Unlike the EEG and ECG, blood pressure is not an electrophysiological response though may be measured by a ‘sphygmomanometer’ in terms of the ratio of ‘systolic’ over ‘diastolic’ pressure. Typical readings of blood pressure can be expected around 120 mm Hg over 80 mm Hg, respectively. To determine systolic pressure, an inflatable cuff of a sphygmomanometer may be placed around the upper arm to apply pressure to the blood vessel of the ‘ulnar’ artery. At a specific pressure from the cuff (as indicated by a mercury level), blood passing through the vessel is heard using a stethoscope, as is found by lowering the exerted pressure. The sounds detected are called sounds of ‘Korotkoff’ and the pressure at which the sounds are heard signifies the systolic pressure. Similarly, the diastolic pressure is then determined by lowering the pressure further to a point at which the sounds of ‘Korotkoff’ cannot be heard.

Blood pressure variability (BPV) on the other hand may be determined by specialised means requiring observation of differences in time between signal peaks of the ‘pulse plethysmograph’ (PPG) and the ECG R-wave (pulse transit time, PTT). While this method does not provide absolute measures of systolic and diastolic pressures, it has been shown to reliably indicate BPV measures by means of noting variations in the PTT [Bramwell & Hill, 1922][MacKenzie et al., 2002][Ma & Zhang, 2005][Atlasz et al., 2006]. This technique will be specifically elaborated on in the methodology of the main experimental work (Chapter 8).

Chapter 2

Literature Review

Direct comparisons between the reported literature concerning the effects of mobile phone exposures on the electroencephalogram (EEG) may be notably difficult to present mainly due to inconsistencies in methodological practices between studies reported [Barnes & Greenebaum, 2007]. The format of this review has been to firstly present a detailed summary of the literature at hand followed by a critical overview, developed hypotheses, and a series of research questions. In addition, incorporated into this review are studies which have considered mobile phone exposure effects in heart response through measures and derivations of heart rate, heart rate variability (HRV), and blood pressure (BP).

2.1 Effects of Mobile Phone Exposures on the Electroencephalogram

The literature regarding the investigation of mobile phone exposures on measures of the EEG, may be subdivided into 3 major categories, namely:

1. *Mobile Phone Exposure Effects on the Awake response of the EEG*
2. *Mobile Phone Exposure Effects on the Sleep response of the EEG*
3. *Mobile Phone Exposure Effects on Cognitive Performance and Event-Related Potentials*

Although awake studies will later form a key focus of this thesis, to provide a necessary understanding of the broader scope of literature at hand the following

context aims to cover the literature associated with all 3 types of studies outlined above.

2.1.1 Mobile Phone Exposure Effects on the Awake EEG

In an early study, von Klitzing reported on a particular effect on the EEG from the P4 EEG electrode position when a subject was exposed to a 150 MHz electromagnetic field pulsed at 217 Hz. Most notably the spectral EEG power of the 10.7 Hz signal was reported to increase by as much as "5 times". Following the exposure period the power at this EEG frequency was stated to be "slightly reduced or as remaining at this level". Yet in another subsequent exposure period under the same conditions, the indicated increase reportedly remained unchanged. Additionally, the results of this study indicated an increase in EEG power during exposure from electrode positions O1 and O2, however the report was vague in describing this effect.

In a follow-up investigation to the 1992 study, von Klitzing [von Klitzing, 1995] further reported on the results of an experiment where 17 participants were exposed 2-3 times in 15-minute intervals again to a controlled 150 MHz exposure pulsed modulated at 217 Hz. Two 15-minute control EEG recordings were conducted between the exposure periods. Predominantly, results of the investigation demonstrated that during the first control recording following an active exposure period noted increases in EEG power of the ~10 Hz rhythm became evident. Notably, this exposure effect was again sustained as it were in earlier findings, this time throughout the subsequent exposure period as observed from the occipital electrode O2. Notwithstanding this however, no indication of results in terms of statistical significance were reported for von Klitzing's published work.

In 1995 Reiser, Dimpfel, and Schobel, also published work on the effects of mobile phone exposures in the awake EEG [Reiser et al., 1995]. The report addressed exposures of a 150 MHz '*coil* source' pulse modulated at 9.6 Hz (magnetic flux densities ~400 pT) as well as a 902 MHz mobile phone modulated at 217 Hz (8 W). In the experiment 36 participants were required to be awake and at rest during 15-minute exposure periods. Results of the investigation of the 150 MHz exposure source predominantly indicated spectral power increases in the EEG frequency bands of alpha 2 (9.75 – 12.5 Hz), beta 1 (12.75 – 18.5 Hz), and beta 2 (18.75 – 35 Hz) as seen comparably to von Klitzing's reports at the occipital region of the head from the electrode positions O1 and O2. In addition, in the beta 1 band, spectral power increases during exposure were reported in 29 out of the 36 participants as reported with an error probability of less than 0.001. Following cessation of exposure and in support of this finding, the error probability was reported to increase to 0.07 suggesting a parallel influence due to the presence of exposure. The authors interpreted these results as indicating an 'instantaneous' effect due to exposure.

Subsequently, results of the investigation using the mobile phone, which was placed at a distance 40 cm behind the head, indicated small but statistically insignificant power increases in alpha 2 and beta 1 from the electrodes O1 and O2. Additionally, in beta 2 from the same electrode positions, results demonstrated relatively higher power for 25 out of the 36 subjects investigated. Perhaps most notably, resultant increases in the alpha 2 and the beta EEG bands were shown to occur approximately 15 minutes after exposure ceased. Both investigations of Reiser et al. were conducted single blind.

In yet another single blind investigation reported by Röschke and Mann [Röschke et al., 1997], EEG measures of the awake, closed eyed condition of 34 men were investigated during exposure to a 900 MHz field pulsed with a 217 Hz signal from an "aerial" source positioned 40 cm from the vertex of the head (8 W; 0.05 mW/cm^2). However, unlike positive findings as noted in the awake studies by Reiser et al. and von Klizting, results of Röschke and Mann's study did not indicate any notable changes in the EEG during exposure periods of approximately 3.5 minutes as observed in analysis of the EEG frequency bands delta (1-3.5 Hz), theta (3.5 – 7.5 Hz), alpha (7.5 -12.5), and beta (12.5–18 Hz). Nevertheless, all test recordings were measured from the central head locations C3 and C4 and not considered from the occipital sites O1 and O1 as previously reported [von Klitzing, 1992, 1995][Reiser et al. 1995].

In a more extensive mobile phone investigation to those previously mentioned, Hietanen et al. [Hietanen et al., 2000] reported on a study investigating the RF effects of five different cellular phones on the recorded EEG from 19 adults (10 male; 9 female). The exposure sources consisted of 3 analogue NMTs (Nordic Mobile Telephones) with different antenna types, as well as a 900 MHz GSM mobile phone and an 1800 MHz PCN (Personal Communications Network) phone. Each of the participants took part in six tests of which one test was a 30-minute sham. The remaining five tests were 30-minute RF exposure tests which each included 20 minutes of exposure (1- 2 W peak) and 10 minutes of sham exposure. During all trials a full 10/20 EEG system array of electrodes were considered (inclusive of O1, O2, C3, and C4) while participants were instructed to stay awake and be seated with eyes closed. Most interestingly, apart from a significant change in delta band, which

was passed as occurring due to statistical chance, results of statistical comparisons between sham and exposure recordings did not indicate any likely changes in the EEG for any of the analogue or digital mobile phone exposures tested.

In 2002, in another awake study, Croft et al. [Croft et al., 2002] recorded the EEG of 24 participants (16 males, 8 female; ages 19-48 yrs) with eyes open during mobile phone exposures from a standard Nokia 5110 mobile phone operating at 900 MHz (3-4 mW average). The recordings were obtained with 19 electrodes incorporated into a standard EEG electrode-cap. The handset was located via bracket and was positioned radial to the subject's scalp midway between Oz and Pz. A 3-minute auditory discrimination task was conducted followed by a 2-minute resting EEG recording. This procedure was repeated four times constituting one session, which was undertaken three times. The exposure sequence administered was conducted single blind however was counterbalanced with an active mobile present in one session, an EMF attenuator present in a second, and a control condition made in the third. The 3-minute auditory discrimination tasks undertaken during the trials consisted of a series of binaural tones (1.1 kHz and 1 kHz for 60 ms every 1-3 seconds) to which participants were to press a button for target tones allowing for accuracy and reaction time to be measured. Following the experiment, an electro-oculographic correction was conducted on both the discrimination task and resting EEG data. The experimental results for the resting EEG demonstrated spectral power decreases in the 1-4 Hz bands in the right hemisphere and increases in EEG power in the 8-12 Hz in midline posterior sites and near to the exposure source. Other notable results reported were that of attenuation of the 4-8 Hz and a global decrease in the 12-30 Hz band both

as a function of time, and notable increases in the 30-45 Hz responses in the mid frontal and lateral posterior regions.

In an investigation entitled 'Effects of high-frequency electromagnetic fields on human EEG: a brain mapping study' Kramarenko et al. [Kramarenko et al. 2003] later investigated the effects of an active radiophone (100 MHz; 3 mW) and a mobile phone (900 MHz pulsed at 217 Hz) on 10 young male adults and 10 children (12 yrs old). The EEG was acquired using 16 electrodes positioned in a 10/20 standard array while participants sat awake again with eyes open in a comfortable seat whilst exposures were activated or inactivated in the ear-mouth position. The exact period of exposures were not documented. The EEG data was wirelessly transmitted to the EEG recording apparatus. Spectral analysis on the EEG data was carried out including color-coded brain 'mapping' of all recorded EEG amplitude characteristics. Via this mapping any unusual characteristics during the experimental recordings were noted. Most prominent experimental results indicated that following active exposure from the mobile phone for within 15-20 seconds for adults and 10-15 seconds for children, a "short lasting" (1 s for adults and 2-3 s for children) slow-wave activity appeared in the 2.5 – 6 Hz EEG band for adults and in the 1 - 2 Hz band for children. The slow-wave appeared every 10-15 seconds and was reported to have progressively diminished and disappeared following 10 minutes of the phone being turned off. An observed increase in the 'median frequency' was observed prior to the onset of the slow-wave, however following its appearance a reduction in the median frequency was noted. An indication of how many of the adults or children this effect occurred in was not noted.

In 2004 Hinrikus et al. [Hinrikus et al., 2004] reported on a study entitled 'Changes in human EEG caused by low level modulated microwave stimulation'. In this investigation 20 participants (11 male and 9 female, ages 19-23 yrs) lay in a relaxed position with eyes closed and 'ears blocked' whilst exposed to a 450 MHz microwave source modulated at 7 Hz. Experiments were conducted single blind with each participant undergoing exposure and sham exposure once. Exposures were generated by a signal generator which fed the modulated signals to a quarter wave monopole positioned 10 cm 'from the skin of the left side of the head'. From estimations of the field power density at the skin SAR was derived to be 0.35 W/kg. Experimental procedures of the study consisted of an initial 1-minute reference EEG recording and a following 'photic stimulation' applied for 20 seconds. Ten continuous subsequent cycles of 1-minute exposure each followed by a 1-minute pause was carried out while EEG recordings were conducted throughout. For each participant, the same procedure was carried out again with a sham exposure set of cycles. The EEG recordings were acquired via 9 electrodes located in the frontal, temporal, parietal, and occipital regions in accordance to standard 10/20 positioning. Results were generated by visual observation of the EEG and a student two-tailed t-test analysis for differences between exposure and sham exposure recordings as well as an inter-cycle analysis. Results of applied photic stimulation indicated a regular decrease in the alpha band in all channels. Observed changes of the EEG when RF exposure was present included unstable readings beginning at the third recorded cycle of exposure as well as particular suppression or amplification of the alpha or theta rhythms. The authors note particular odd behaviour during exposure between individual participants, noting unusual differences in suppression or amplification trends of the

EEG between the left and right hemispheres. In addition, increases in EEG power and changes in the frontal regions are particularly noted. An observation that repetitive exposure may cause more prominent changes is further noted. Given evidence of observed changes, the authors conclude that although changes from the RF stimulus can be observed, these effects are unique to the individual and furthermore are not detectable using a gross analysis of the group.

Curcio et al. [Curcio et al. 2005], later reported on a double blind study investigating the EEG effects of a 902 MHz signal (pulsed at 217 Hz ; 0.25 W) on 20 participants (10 males and 10 females, ages 22-31 yrs). The exposure parameters were set by a test card in a Motorola 'Timeport 260' mobile phone which was positioned in an ear-mouth position 1.5 cm from the left ear. Standard measurements of SAR were acquired at 0.5 W/kg. For the experimental procedure participants were placed randomly into two groups and were seated awake in a comfortable armchair with eyes closed. Both groups underwent a 45-minute baseline trial where no mobile phone was present, as well as a 45-minute exposure-on period, and 45-minute sham exposure trial. For group 1 recordings of the EEG were undertaken during 7-minute periods following each trial, however for group 2 recordings were conducted during the last 7 minutes of each trial. The EEG recordings were acquired from the frontal (Fz), central (Cz), parietal (Pz), and temporal (T3, T4) regions of the head in accordance with the international 10/20 standard. In addition an EMG recording was conducted (as taken from the submental muscles) as well as an EOG as acquired from the right eye as used for artefact identification and removal. Statistical tests of the EEG across a 1-24 Hz spectrum in 1 Hz bins were conducted between 3 different trial recordings

and between groups. Given a large number of multiple comparisons were conducted in the design the analysis incorporated a Bonferroni correction to appropriately lower the statistically significant probability criteria. Results of the study indicated statistically significant outcomes in the alpha rhythms 9 Hz and 10 Hz from the Cz position when the exposure-on test recording was compared to the baseline and sham. For both results the mean exposure EEG recording was higher than was in the baseline or sham exposure trials suggesting a power increase due to exposure. In addition at Pz, statistically significant difference was shown in the 11 Hz band when drawing results between exposure-on during the recording session (Group 2) and the no exposure condition EEG acquired following a 45-minute exposure session (Group 1). In all statistically significant cases EEG power was increased in the exposure-on condition. The authors furthermore note approaching significance at the 9 Hz bin from the T3 electrode position, although indicate that no significant effects or interactions occurred at the Fz and T4 positions. The authors hypothesise that 'the current resting EEG recorded on Cz and Pz could be the result of a composite activity coming from bilateral areas and that the possible effect of EMF exposure could be induced by a kind of spreading of RF power across the brain'. The paper concludes stating that 'EMFs like the ones emitted by mobile phones influence normal brain physiology' and this is 'probably by means of changes in cortical excitability'.

In addition to effects due to pulsed 902 MHz signals as noted by Curcio et al. in the EEG alpha band, recent studies by Vecchio et al. [Vecchio et al., 2007] and Croft et al. [Croft et al., 2008] have both notably indicated alpha band effects during the awake resting condition due to ~900 MHz exposures pulse modulated at 217 Hz.

Interestingly, Vecchio et al. recently tested (in a sample size of 10 participants) the hypothesis that EMFs from mobile phones affect interhemispheric synchronisation of cerebral rhythms as particularly noted in the alpha band, as their previous work suggests [Curcio et al., 2005]. In a unique approach to other EEG awake investigations, experimental protocol permitted the participants to walk and talk to experimental aids for two 45-minute durations 1 week apart whilst mobile phone exposures were active 1.5 cm from the left ear. In a double blind protocol the 45-minute sessions either consisted of sham or exposure. Exposures were generated from a commercially available 902.4 MHz GSM phone mounted in a worn helmet with reported pulse modulations of 217 Hz (250 mW average; 2 W peak), however it is importantly assumed (as there is no comment made) that the 8 Hz repetition rate in GSM transmission as well as discontinuous transmission (DTX), as associated with 2 Hz in a reduced power mode, was also employed. EEG recordings from a complete 10/20 positioned electrode array were considered in 5-minute rest recordings with eyes closed both before and after the two 45-minute talking sessions. The EEG was investigated in the bands delta (2 – 4 Hz), theta (4 – 6 Hz), alpha 1 (6 – 8 Hz), alpha 2 (8 – 10 Hz), and alpha 3 (10 – 12 Hz). Results indicated that for the alpha 2 and 3 bands interhemispheric coherence was lower for GSM than sham at frontal areas, while for temporal areas (nearest to exposures) alpha 2 band was notably higher for GSM than for sham. Specifically due to these results, the authors suggest in conclusion that “mobile phone emission influences brain physiology, probably by the interhemispheric synchronisation of signals, mainly across the corpus callosum”.

In addition to speculation that alpha rhythm effects may result from mobile phone exposures, Croft et al. presents a relatively large study (N = 120) where the experimental analysis is undertaken upon hypothesis that an alpha band effect exists. The double blind study entitled "The effect of mobile phone electromagnetic fields on the alpha rhythm of human electroencephalogram" describes an investigation of the effects of mobile phone exposures on the awake EEG, which was part of a two-fold study with the other part addressing mobile phone effects on event-related potentials [Hamblin et al., 2006] as will be addressed later in Section 2.1.3. Specifically, Croft et al. examines the 'open eyes' condition of the EEG alpha band (8 – 12 Hz) of 120 seated participants (46 male, 74 female) exposed to 895 MHz exposures pulsed at 217 Hz from a programmable GSM phone (250 mW average; 2 W peak). The phone was set to transmit over the right (N = 60) or left (N = 60) temporal region in an ear-mouth 'touch' position. Experimental protocol required participants to firstly perform a battery of tests (as documented in Hamblin et al., 2006). The awake at rest recordings were subsequently recorded for 10-minutes, followed by another battery of tests, which followed once more with a 10-minute resting awake trial. Maximum SAR levels of the GSM phone tested were 0.674 W/kg at the cheek-bone and 0.11 W/kg at the temporal lobe near the antenna region, as measured over 10 gram averages of tissue.

Alpha power data (Sham and Exposure) as recorded from a comparable 10/10 system array (58 recording sites considered) was grouped into frontal ipsilateral, frontal contralateral, posterior ipsilateral, and posterior contralateral. Results of the study indicated that the low-level exposure tested increased EEG activity in the alpha band, where this effect was reportedly more pronounced at sites ipsilateral to exposure

particularly at posterior sites. Notably, different effects in the posterior region were reported during and after exposure with speculation that there is one effect that lasts only as long as exposure, primarily, and a longer lasting larger alpha increase more frontally potentially due to secondary neural processes (not reportedly as statistically strong). Croft et al. further note that the magnitude of the increase effects were very small and indicate that during the main effect of exposure "only 60% showed an increase in alpha, suggesting that the effect of the phone was not homogeneous and it may be related to individual differences between the participants". Increases in alpha as a function of exposure duration as noted in earlier work and as described previously [Croft et al. 2002] was not replicated by this study.

In yet another recent double-blind study by Hinrikus et al. [Hinrikus et al., 2008], the EEG of four different groups of participants were acquired to investigate the effects of 450 MHz exposures modulated at 7 Hz (1st group; 19 adults), 14 and 21 Hz (2nd group; 13 adults), 40 and 70 Hz (3rd group; 15 adults), and 217 and 1000 Hz (4th group; 19 adults). As indicated in Hinrikus et al.'s 2004 study, participants lay in a relaxed position with eyes closed while modulated signals were fed to a ¼ wave monopole positioned 10 cm 'from the skin of the left side of the head' (field power density 0.16 mW/cm^2; calculated SAR over 1 g = 0.303 W/kg). Exposure for each group (with the exception of the first group) lasted 20 minutes total with every even minute of a continuous 40 minute EEG recording session signifying an exposure period. Specifically, the first 20 minutes of each 40-minute session for group 2 consisted of the 14 Hz modulation while the latter 20 minutes consisted of the 21 Hz modulation. Similarly, groups 3 (40 Hz and 70 Hz) and 4 (217 Hz and 1000 Hz) were

exposed in the same arrangement. Each odd minute was a reference EEG recording. As the first group was only exposed to the 7 Hz modulated signal there was only 20 minute EEG recording session allocated in this instance. The EEG was recorded from the frontal, temporal, parietal, and occipital sites. Excluding investigation of the 1000 Hz modulation which demonstrated null findings, main results of the study indicates that in beta1 band (15 - 20 Hz) that 13 - 30% of all participants in each group indicated significant increased changes, as seen from the parietal recorded data. An indication of increased spectral power in the alpha band (8 – 13 Hz) is also noted. Main results in previous work [Hinrikus et al., 2008a] that demonstrated that microwaves modulated at 14 and 21 Hz were significant during the first 30 s of exposure, was not substantiated in this study. In conclusion to their findings, the authors indicate support for hypothesis that the effect of microwaves differs among individuals.

In another double-blind study, Kleinlogel et al. [Kleinlogel et al., 2008a] reported on the effects of GSM 900 MHz (SAR 1 W/kg) and UMTS 1950 MHz (SAR 0.1 or 1 W/kg) on the resting EEG and well-being of 15 adults during an 'eyes closed' seated protocol. The experimental protocol involved several duties, of which firstly involved the recording of 'vigilance controlled' resting EEG during 4-minute sham-exposure and post 4-minute radomised exposure periods. This session was then followed by a 2.5-minute period of visually evoked potentials induced by participants sighting a checkerboard on a computer screen with alternating (200 times) black and white patterns. Following this period, an 11-minute continuous performance test (CPT) involving 12 randomly presented letters on a computer screen (every 1450 ms for a

period of 200 ms) required participants to press a mouse button each time the letters O and X appeared sequentially – reaction times and button press errors were measured for assessment. Following the CPT, two auditory evoked sessions were also then recorded for 2 minutes then ~6 minutes respectively. The first auditory sessions required participants to simply listen and not respond to 20 ms, 80 dB, 1000 Hz signals every 500 – 1000 ms, via an earpiece to the left ear. The second auditory session required participants to press a mouse button each time a rare 1000 Hz tone was heard amongst many 500 Hz tones, all at 80 dB. Finally, another vigilance controlled resting EEG was recorded for 2 minutes during exposure followed by 6 minutes of vigilance controlled EEG without exposure on. The total experiment was performed by each participant five times over, 1 week apart, with the first time being a training session and the latter 4 times with randomised sham-exposure, GSM 900 MHz, UMTS 'low SAR', or UMTS 'high SAR'. Results of the study for resting EEG did not indicate any statistically significant changes due to exposure when measured over a 10-20 standard array of 19 electrodes. EEG bands considered were 1 - 3.5 Hz, 4 - 7.5 Hz, 8 - 10 Hz, 10.5 - 12.5 Hz, 13 - 18 Hz, 18.5 - 32 Hz. Similarly, no significant changes in any of the ERP measurements including visual and auditory responses could be noted. Results of the ERP work conducted were separately published Kleinlogel et al., 2008b. In conclusion to both studies, the authors remark that outcomes of their work 'do not give any evidence for a deleterious effect of the EMF on normal healthy mobile phone users'.

In a most recent single blind study conducted by Wu et al. [Wu et al., 2009], the resting EEG of 10 adults were examined for exposures to 900 MHz 'cordless phone'

(0.101 μW/cm^2) positioned in an ear to mouth position over the left ear (one trial) and a 2.4 GHz wireless router (0.0676 μW/cm^2) positioned 2 m in front of the head (second trial). While seated and relaxed in eyes closed condition randomised sham and exposure recordings were considered for each participant over three 100-second intervals with 30-second rest periods between recordings. Test EEG recordings were acquired using a 32-electrode cap. Analysis over averaged spectral power across standard delta, theta/alpha, and beta EEG bands did not indicate any statistically significant alteration due to either the cordless phone or the wireless router. Nevertheless, significant increases in EEG power in two of the 10 participants were suggested to indicate the possibility of individual variation to sensitivity to EMFs. However, a limited discussion is provided on this topic particularly as to from what regions of the head the power increases were indicated experimentally. Uniquely, in addition to investigation of resting EEG recordings Wu et al. also recorded the resting EEG with a 'Farabloc hood' over the heads of participants, which was used to shield any RF above 1000 Hz. Interestingly, results of these tests were reported to indicate significantly lowered spectral power in the resting EEG when the hood was on as compared to both control and exposure test recordings. Consequently, the authors state that 'it cannot be excluded that ambient radio waves are chronically affecting human resting EEG'.

In summary, with respect to studies investigating mobile phone exposure effects on EEG recordings in awake participants, it may be said that considerable findings in most related publications have been shown [Reiser et al., 1997][Croft et al., 2002][Curcio et al., 2005][Croft et al., 2007][Vecchio et al. 2007]. Interestingly

however, other investigations such as reported by Röschke and Mann, 1997, Hietanen et al. 2000, have published reports that have indicated null or statistically insignificant findings in EEG responses to similar exposures. In regards to the direction of results indicating positive findings, although outcomes may be shown to be somewhat varied, work conducted by Curcio et al. [Curcio et al. 2005][Vecchio et al. 2007], Croft et al. [Croft et al. 2002, 2007], and Hinrikus et al. [Hinrikus et al., 2008a] appears to demonstrate that an alpha effect due to mobile phone exposures may exist in the awake EEG response. Although, indeed the study as produced by Röschke and Mann [Röschke and Mann, 1997] could not confirm related effects in the alpha response, it may be considered that in this investigation the position of exposure source was located at 40 cm from the head, whereas the test protocol in studies by Curcio et al., Croft et al., and Hinrikus et al. required the exposure source to be positioned in the near-field, close to the ear. However, Kleinlogel et al.'s (2008a) recent double blind study ($N = 15$) is interesting as it is indicated that there was no statistically significant changes evident within the alpha band (8-10 Hz and 10 –12 Hz) when GSM 900 MHz and UMTS 1950 MHz exposures were investigated over the left ear. Notwithstanding this, in summary the direction of results in awake studies most strongly appear to indicate that an alpha band effect may exist in short-term durations as seen in both hemispheres, and potentially following exposure cessation, when mobile phone exposures are tested in close proximity to the head. Nonetheless, more studies are required to confirm these outcomes. Some drawbacks that may be indicated to be addressed in future awake studies include a lack of double blinding, relatively weak sample sizes, and dosimetry which have all been part of the following majority of studies [von Klitzing, 1995][Reiser et al. 1997][Röschke and Mann, 1997][Hietanen

et al., 2000][Croft et al., 2002][Kramarenko et al. 2003][Hinrikus et al., 2004][Wu et al., 2009]. Absence of consistent testing of EEG electrode sites to consider regions over the head has also been a notable drawback in interpreting the direction of noted outcomes [von Klitzing, 1995][Reiser et al. 1997][Röschke and Mann, 1997].

Articles: Mobile phone RF effects on the resting awake EEG	**Main outlined findings**	**Type of exposure**	**No. of participants/ single or double blind**	**Duration of exposure**	**SAR**
von Klitzing, 1995 (Preliminary study: von Klitzing, 1992)	Effect from the P4 EEG electrode position – 10.7 Hz signal increased by "5 times". Signal from position O1 & O2 is indicated to have been affected [1992]. Initial control recording following an active exposure period noted increases in EEG power of the ~10 Hz rhythm from O2 [1995]	150 MHz EMF pulsed at 217 Hz	1992: Undefined; single blind 1995: 17 adults; single blind	1992: undefined 1995: 2-3 exposure periods in 15-minute intervals	undefined
Reiser et al., 1995	Spectral power increases in alpha 2 (9.75 – 12.5 Hz), beta 1 (12.75 – 18.5 Hz), and beta 2 (18.75 – 35 Hz) for the 150 MHz exposure source test at O1 & O2. Statistically insignificant increase power trends seen in alpha 2 and beta 1 at O1 & O2 occurring 15 min after exposure cessation of the mobile phone 902 MHz source.	150 MHz '*coil* source' pulse modulated at 9.6 Hz (magnetic flux densities ~400 pT)/ 902 MHz mobile phone modulated at 217 Hz (8 W) positioned ear-mouth.	36 adults; single blind	15-minute exposure periods recorded	undefined
Röschke et al., 1997	No statistically significant changes in EEG power indicated from the recorded sites, C3 and C4.	900 MHz EMF pulsed at 217 Hz from an "aerial" source positioned 40 cm from the vertex of the head (8 W; 0.05 mW/cm^2)	34 adults; single blind; eyes closed condition indicated	Multiple 3.5-minute exposure periods recorded	undefined
Hietanen et al., 2000	Apart from a statistically significant change in delta band, (indicated as occurring due to statistical chance), no notable changes in the EEG could be noted for any exposure type tested. A full 10/20 EEG electrode array was considered.	3 analogue NMTs with different antenna types, as well as a 900 MHz GSM mobile phone and an 1800 MHz PCN phone (1- 2 W peak)	19 adults; single blind; eyes closed condition indicated	20-minute exposure periods from each of the 5 RF sources recorded	undefined

Table 2.1 (1 of 5) Mobile phone RF effects on the resting awake EEG

Croft et al., 2002	Spectral power decreases in EEG power were noted in delta in the right hemisphere. Spectral power increases were further noted in the alpha band in midline posterior sites and near to the exposure source. Attenuation as a function of time were particularly described for the theta and beta bands. A full 10/20 EEG electrode array was considered.	Nokia 5110 mobile phone operating at 900 MHz (3-4 mW average) was positioned radial to the subject's scalp midway between Oz and Pz.	24 adults; single blind; eyes open condition indicated	Multiple 2-minute exposure periods during resting EEG recordings	undefined
Kramarenko et al. 2003	Following active exposure from the mobile phone for within 15-20 seconds for adults and 10-15 seconds for children, a "short lasting" (1 s for adults and 2-3 s for children) slow-wave activity appeared in the 2.5 – 6 Hz EEG band for adults and in the 1 - 2 Hz band for children. A full 10/20 EEG electrode array was considered.	100 MHz radiophone (3 mW), and a 900 MHz mobile phone (900 MHz pulsed at 217 Hz) positioned in ear-mouth.	10 adults and 10 12yr old children; single blind; eyes open condition indicated	Undefined periods of exposure	undefined
Hinrikus et al., 2004	Particular suppression or amplification of the alpha or theta rhythms is noted. Also reported are unusual differences in suppression or amplification trends of the EEG between the left and right hemispheres amongst participants. A total of 9 EEG electrodes located in the frontal, temporal, parietal, and occipital regions were considered.	450 MHz microwave source modulated at 7 Hz. Exposures generated by modulated signals fed to a ¼ wave monopole positioned 10 cm 'from the skin of the left side of the head'.	20 adults; single blind; eyes closed condition indicated	Multiple 1-minute exposure periods tested	SAR was derived to be 0.35 W/kg from estimations of the field power density
Curcio et al. 2005	Statistically significant increases at 9 Hz and 10 Hz from Cz is noted for periods where exposure is active during EEG recordings. At Pz, at 11 Hz, results were shown to be significantly higher for the exposure-on tests as compared to the exposure-absent recording sessions. EEG recordings from the frontal (Fz), central (Cz), parietal (Pz), and temporal (T3, T4) regions of the head were considered.	Motorola 'Timeport 260' 902 MHz mobile phone (pulsed at 217 Hz ; 0.25 W), positioned in ear-mouth, 1.5 cm from the left ear.	20 adults; double blind; eyes closed condition indicated	Single 45-minute exposure period, with EEG recorded in the last or the following 7 minutes of exposure	0.5 W/kg

Table 2.1 (Continued: 2/5) Mobile phone RF effects on the resting awake EEG

Croft et al., 2008	Increased power in the alpha band is particularly noted, where the effect was more pronounced at sites ipsilateral to exposure mainly at posterior sites. A full 10/10 EEG electrode array was considered over the 8 – 12 Hz alpha band.	895 MHz exposure pulse modulated at 217 Hz from a programmable GSM phone (250 mW average; 2 W peak) positioned ear-mouth over the left or right ear.	120 adults; double blind; eyes open condition indicated	Two 10-minute resting awake exposure trials	0.674 W/kg over 10g of tissue
Vecchio et al., 2007	Results indicated that for the alpha 2 (8 – 10 Hz) and alpha 3 (10 – 12 Hz) bands interhemispheric coherence was lower for GSM than sham at frontal areas, while for temporal areas (nearest to exposures) alpha 2 band was notably higher for GSM than for sham. A complete 10/20 electrode array was considered.	902.4 MHz GSM phone with pulse modulations of 217 Hz (250 mW average; 2 W peak), positioned ear-mouth, 1.5 cm from the left ear	10 adults; double blind; resting, eyes closed EEG recorded before and after exposure	5-minute EEG recordings acquired before and after two 45-minute exposure sessions	undefined
Huber et al., 2003 (preliminary studies: Huber et al., 2000; Borbely et al., 1999)	1st exp: EEG results notably demonstrated increases in power at the central position C3 in the 11 – 11.5 Hz spectral range in the waking period prior to sleep onset 2nd exp: A decrease in spectral power at C3 in the 10.5–11 and 18.75–19.5 Hz EEG rhythms was particularly noted in the waking exposure period. The EEG was measured from sites F3, C3, P3, O1, F4, C4, P4, O2. Conclusive comments suggest that EEG power is manifested rapidly during sleep due to RF exposures. These effects are indicated to outlast exposure by at least 15 minutes when RF is applied during the waking period prior to sleep.	1st exp: 900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm behind the head while lying down 2nd exp: Patch antenna with comparable EMF as in exp 1, positioned ~11 cm from both the left and right side of the head of seated participants	1st exp: 24 adults; double blind; eyes closed during a sleep onset period 2nd exp: 16 adults; double blind;	1st exp: multiple 15 minute exposures 2nd exp: 30 minutes of exposure	Exp 1 and 2: 1 W/kg over 10 g of tissue

Table 2.1 (Continued: 3/5) Mobile phone RF effects on the resting awake EEG

Hinrikus et al., 2008b (Preliminary study: Hinrikus 2008a)	Excluding investigation of the 1000 Hz modulation which demonstrated null findings, main results of the study indicates that in beta1 band (15 - 20 Hz) that 13 - 30% of all participants in each group indicated significant increased changes, as seen from the parietal recorded data. An indication of increased spectral power in the alpha band (8 – 13 Hz) is also noted. Main results in previous work [2008a] that demonstrated that microwaves modulated at low frequency were significant during the first 30 s of exposure was not substantiated in this study. In conclusion to their findings to date, the authors indicate support for hypothesis that the effect of microwaves differs among individuals.	450 MHz modulated signals (i.e. modulated at 7 Hz (1st group), 14 and 21 Hz (2nd group), 40 and 70 Hz (3rd group), and 217 and 1000 Hz (4th group), fed to a ¼ wave monopole positioned 10 cm 'from the skin of the left side of the head' (field power density 0.16 mW/cm^2)	Four groups with 19, 13, 15, and 19 adults respectively; double blind; eyes closed condition indicated	10 1-minute exposures for each low-frequency modulation tested	0.303 W/kg averaged over 1 g of tissue (calculated)
Kleinlogel et al., 2008a	Results of the study for resting EEG did not indicate any statistically significant changes due to exposure over the 1 - 3.5 Hz, 4 - 7.5 Hz, 8 - 10 Hz, 10.5 - 12.5 Hz, 13 - 18 Hz, and 18.5 - 32 Hz.bands. Additionally, no changes due to exposure could be reported on ERPs as reported elsewhere [Kleinlogel et al., 2008b]. In conclusion to both studies, the authors remark that outcomes of their work 'do not give any evidence for a deleterious effect of the EMF on normal healthy mobile phone users'.	GSM 900 MHz and UMTS 1950 MHz exposures, positioned over the left ear	15 adults; double blind; eyes closed condition	6 minutes total resting EEG exposure period	1 W/kg (GSM 900 MHz) and SAR 0.1 or 1 W/kg (UMTS 1950 MHz)

Table 2.1 (Continued: 4/5) Mobile phone RF effects on the resting awake EEG

Wu et al., 2009	Analysis over averaged spectral power across standard delta, theta/alpha, and beta EEG bands did not indicate any statistically significant alteration due to either the cordless phone or the wireless router. EEG recordings were considered over 32-electrodes covering the head. Two of the 10 participants were suggested to indicate the possibility of individual variation to sensitivity to the exposures. Uniquely, lowered spectral power in the resting EEG was indicated when a 1000 Hz shielding hood was placed on subjects' heads as compared to the regular control and exposure test recordings undertaken.	900 MHz 'cordless phone' (0.101 $\mu W/cm^2$) positioned in an ear to mouth position over the left ear (one trial) and a 2.4 GHz wireless router (0.0676 $\mu W/cm^2$) positioned 2 m in front of the head (second trial)	10 adults; single blind; eyes closed condition	Three 100-second intervals of exposure with 30-second rest periods between	undefined

Table 2.1 (Continued: 5/5) Mobile phone RF effects on the resting awake EEG

2.1.2 Mobile Phone Exposure Effects on the EEG during Sleep

Waking EEG effects have also been observed in reported sleep studies investigating the RF effects on participants whilst recording their EEG. Huber et al. [Huber et al., 2003] reported on awake effects of participants prior to sleep over two double blind experiments conducted to extend the analysis of their previous work using different experimental protocols [Borbely et al., 1999][Huber et al., 2000]. During the first experiment the EEG (as observed from sites F3, C3, P3, O1, F4, C4, P4, O2) of 24 men was recorded during 900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm behind the head of participants lying down. The experiment considered two sessions of two consecutive nights sleep (a pre-experimental night and an exposure night) at an interval of one week. During the exposure nights, exposures were continuously switched on and off at 15-minute intervals inclusive of the sleep onset period. In the second experiment 16 participants were required to be seated for 30 minutes prior to a 3 hr morning sleep episode to be exposed to a similar protocol from a patch antenna configuration positioned ~11 cm from both the left and right side of the head. Right hemispheric, left hemispheric, and sham exposures were each considered at intervals 1 week apart with EEG acquisition considered from the same sites as in experiment 1. The SAR of exposures tested in both experiment 1 and 2 were prudently adjusted to a peak spatial-average of 1 W/kg over 10 g of tissue. The resultant outcomes regarding awake effects reported were represented by the waking period measurements recorded prior to sleep onset. The recorded EEG results for this period particularly demonstrated ‘increases’ in power at the central position C3 in the 11 – 11.5 Hz spectral range. In the second

experiment however, a 'decrease' in spectral power at C3 in the 10.5–11 and 18.75–19.5 Hz EEG rhythms were observed in a 30-minute waking exposure period prior to a 3 hr morning sleep procedure. During the sleep periods, main results indicated that under both experimental conditions non-REM sleep EEG power was notably increased in the 9 –14 Hz range *following* RF EMF exposure. These effects were observable in almost all recorded locations. Notably, spectral magnitudes of the effect were found independent of the side of exposure indicating that asymmetric exposure does not necessarily produce asymmetric responses. In conclusion, Huber et al. conclude that changes in EEG power are manifested rapidly when exposure occurs during sleep. It is furthermore indicated that these effects may outlast exposure by 'at least' 15 minutes when RF EMF is applied during waking prior to sleep.

In addition to examination of awake and sleep EEG, Huber et al. also address investigation of the effects of RF exposures on heart rate and heart rate variability. However, the outcomes of these tests, along with related studies, will be addressed in Section 2.2.

Wagner et al. [Wagner et al., 2000] also investigated the effects of mobile phone RF exposures on the sleep EEG to clarify previous results determined in two initial studies [Mann and Röschke, 1996][Wagner et al., 1998]. In the preliminary work conducted [Mann and Röschke, 1996], it was notably shown that suppression of REM sleep occurring due to GSM 900 MHz exposures as well as a shortening of sleep onset latency. Corresponding EEG effects to sleep parameters, demonstrated increased alpha spectral power. In the second study however [Wagner et al., 1998], the initial findings by Mann and Röschke could not be replicated, with the most

common finding to the first study being that of observed effects of REM suppression which could not be shown to be statistically supported. Nevertheless, as estimated power flux densities of 0.5 W/m^2 and 0.2 W/m^2 were respectively incorporated into both the first and second studies, Wagner et al. [Wagner et al. 2000] tested a hypothesis that there may be a dose-dependent response. To do this, 20 participants (mean age 24 yrs) were exposed to circular polarised 900 MHz exposures (pulsed 217 MHz) generated by a horn antenna connected to a digital mobile radio telephone (Motorola 1000). The horn was positioned 40 cm below the pillow of the bed from where participants were exposed at much higher power flux densities than previously tested, as reported in the order of 50 W/m^2. Two consecutive nights of 'exposure' sleep were undertaken for each participant as well as two consecutive nights of non-exposure or 'sham' sleep as undertaken at least one week apart from the exposure nights. The order of sham or exposure consecutive nights was divided such that half of the participants undertook exposure nights first while the remaining half undertook the consecutive sham nights first. The study was conducted single blind. During sleep the EEG was acquired from referential recordings of the 10/20 electrode positions Cz and Pz. Test measurements were considered in the frequency bands of delta (1 – 3.5 Hz), theta (3.5 – 7.5 Hz), alpha (7.5 – 15 Hz), and beta (15 – 23 Hz). Results of the spectral analysis of the EEG indicated that there were no statistically significant differences between sham and exposure nights across the positioned leads or with respect to various sleep stages considered, such as duration from falling asleep to REM latency periods. Notably, in the sleep parameters as well, no significant changes could be noted as due to exposure. Consequently, the hypothesis of a dose-dependent effect could not be confirmed. In conclusive remarks, the authors further

speculate that the null findings in the present study, that were in contrast to their previous work, may have resulted due to experimental differences such as the polarisation of the field source (circular versus linear) and/or averaging over two nights as opposed to one night sleep.

Unlike previous sleep investigations of 900 MHz mobile phone exposures, Hinrichs et al. [Hinrichs et al., 2005] conducted a study to examine the influence of 1800 MHz GSM exposures on 13 healthy participants generated from a flat panel antenna positioned as notably in the far-field 1.5 m behind the subject's head (estimated SAR of 72 mW/kg averaged over 10 g). An eight electrode EEG acquisition was considered in accordance with 10/20 positioning to record brain wave activity of the participants over four nights sleep (~8 hrs per night), of which two nights randomly consisted of the active exposure. The four night procedure which was conducted double blind followed one adaptation night where the participants familiarised themselves with the laboratory environment. Results of the investigation which considered statistical analysis over the delta, theta, alpha, and beta (1 & 2) ranges at each site, global spatially averaged band power, as well as attention to the alpha band for each sleep stage and each position, did not indicate any statistically significant difference between the exposure and sham recordings. A median frequency analysis of the alpha band and total EEG frequency band also did not demonstrate any statistically significant differences due to the RF modulated exposure. Based on the results and as the study was based upon far-field exposures, the authors conclude particularly that base stations of GSM 1800 cellular phone networks are not supported by their investigation.

Amongst sleep investigations addressing the effects of mobile phone exposures, contradictory outcomes as also shown in awake studies may be identified. For example, statistically significant and notable results have been described to have indicated effects in the EEG at different sleep stages, such as observations of suppression during REM [Mann and Röschke, 1996] or varying changes during sleep onset periods or within non-REM sleep periods [Huber et al., 2003]. However in contrast, the replicated studies of Wagner et al. [Wagner et al. 1998, 2000] could not confirm evidence of any significant effects. Fritzer et al. [Fritzer et al., 2007] (which will be relevantly discussed firstly in Section 2.1.3 below) closely replicated the study of Huber et al. 2003, but notably could not substantiate similar increased effects in the 9 – 14 Hz EEG range during non-REM sleep.

From the studies that may be noted with regards to mobile phone exposures on sleep EEG, it may be said that it is notably difficult to identify any direction in findings given variable outcomes noted in only few studies. Current drawbacks however may be noted in the statistical strengths of the studies, given that most studies of concern have only considered relatively small sample sizes [Mann and Röschke, 1996][Wagner et al. 1998, 2000][Huber et al., 2003][Hinrichs et al., 2005] [Fritzer et al., 2007].

Articles: Mobile phone RF effects on sleep EEG	Main outlined findings	Type of exposure	No. of participants/single or double blind	Duration of exposure	SAR
Huber et al., 2003 (Preliminary studies: Huber et al., 2000; Borbely et al., 1999)	During sleep periods investigated, main results indicated that under both experimental conditions non-REM sleep EEG power was notably increased in the 9 - 14 HZ range following EMF exposure. The effects were observable in almost all recorded locations (F3, C3, P3, O1, F4, C4, P4, O2). Conclusive comments suggest that EEG power is manifested rapidly during sleep due to RF exposures. These effects are indicated to outlast exposure by at least 15 minutes when RF is applied during the waking period prior to sleep.	1st exp: 900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm behind the head while lying down 2nd exp: Patch antenna with comparable EMF as in exp 1, positioned ~11 cm from both the left and right side of the head of seated participants	1st exp: 24 adults; double blind; eyes closed during a sleep onset period 2nd exp: 16 adults; double blind;	1st exp: multiple 15 minute exposures 2nd exp: 30 minutes of exposure	Exp 1 and 2: 1 W/kg over 10 g of tissue
Wagner et al., 2000 (Preliminary studies: Wagner et al., 1998; Mann & Röschke, 1996)	No significant changes in EEG spectral power due to exposure with respect to various sleep stages such as in duration from falling asleep or REM latency periods. EEG acquisition was considered from Cz and Pz. Wagner et al. conclude the null findings in the present study, notably in contrast to their previous work showing suppression of REM sleep as well as a shortening of sleep onset latency [1996], may have resulted due to experimental differences – namely, polarisation of the field source (circular versus linear) and/or averaging over two nights as opposed to one night sleep.	Circular polarised 900 MHz exposures (pulsed 217 MHz) generated by a horn antenna, positioned 40 cm below the pillow of participants	20 adults; single blind	Exposure applied for two consecutive nights sleep	Calculated ~2 W/kg over 10 g of tissue

Table 2.2 (1 of 2) Mobile phone RF effects on Sleep EEG

Hinrichs et al., 2005	No significant changes in EEG spectral power due to exposure with respect to sleep stages over an eight electrode EEG acquisition over the head. Particularly as the study was based upon far-field exposures, the authors conclude that base stations of GSM 1800 cellular phone networks are not supported by their investigation.	1800 MHz (GSM) exposures generated from a flat panel antenna positioned 1.5 m behind the subject's head	13 adults; double blind	Two nights of exposure (~8 hrs per night)	Estimated 72 mW/kg averaged over 10 g
Fritzer et al., 2007	Non-significant effects reported on the investigation of sleep EEG, attention, visual searching, mental processing speed, concentration, and short-term memory. Results could not substantiate the effect that non-REM sleep EEG power can be increased in the 9–14 Hz range as indicated by Huber et al., 2003.	900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm above the head while lying down	Two groups of 10 adults; double blind; eyes closed during a sleep onset period	6 nights sleep during exposure	Calculated 1 W/kg indicated

Table 2.2 (Continued: 2/2) Mobile phone RF effects on Sleep EEG

2.1.3 Mobile Phone Exposure Effects on Cognitive Performance and Event-Related Potentials

Recently Fritzer et al. [Fritzer et al., 2007] reported on an investigation of short and long term effects of 900 MHz GSM modulated signals on the EEG from 3 half-wave dipoles at a distance of 30 cm from the top of the head of two independent groups of participants lying down asleep (N = 10 exposure group/10 sham group). The experimental setup itself was in accordance with Huber et al. [2003]. In addition to an investigation on sleep Fritzer et al. investigated the cognitive effects of the same exposure on attention, visual searching, mental processing speed, concentration, and short-term memory. Reported outcomes of this study indicated no significant effects on sleep or any significant impairment on cognitive performance of the participants. In line with reported absences of effects on cognitive performance Russo et al. (N = 168) and Haarala et al. (N = 64) also demonstrated no significant effects on cognitive performance during tests of reaction time and vigilance [Russo et al., 2006] and reaction time and memory [Haarala et al., 2003][Haarala et al., 2004]. Nevertheless, selective alterations in cognitive task reaction times [Preece et al., 1999] and memory [Smythe et al., 2003] have been previously demonstrated. In addition, Fritzer et al. addressed the findings of Huber et al. [Huber et al., 2003] that non-REM sleep EEG power can be increased in the 9–14 Hz range, however results could not substantiate the effect. Fritzer et al. nevertheless comment that a differing 15-minute on/off alternating exposure protocol only used by Huber et al., may have promoted the increased effect previously reported.

Over the last 10 years Finish groups authored by Krause C. have conducted a series of investigations to examine the EEG during cognitive tasks whilst being exposed to mobile phone exposures [Krause et al., 2000a, 2000b, 2004, 2006, 2007]. In a replication study of their previous work [2000b] (N = 24) Krause et al. in 2004 reported on the effects of 902 MHz mobile phone exposures pulsed at 217 (avg. power 0.25 W) to investigate the event-related desynchronisation (ERD) (decrease in oscillatory power) and synchronisation (ERS) (increase in oscillatory power) of the EEG. EEG bands of interest (as examined on a *per subject* basis) were the 4 – 6 Hz, 6 - 8 Hz, 8 -10 Hz, and 8 -12 Hz bands. Results of the replication study indicated contradictory outcomes. In contrast to the previous work conducted where enhancements in both ERD and ERS were observed in the alpha 8 – 10 Hz band during auditory/visual memory load tasks no significant effects could be indicated. Most recently Krause et al. [Krause et al., 2007] once more reported on a study in aim to further assess previous inconsistent outcomes as well as to extend the analysis to examination of continuous wave (CW), pulsed modulated (PM) and hemispherical differences. The results of the study indicated commonality with the earlier results which had observably indicated oscillatory effects in the alpha EEG band [Krause et al 2000a,b], however the results as indicated, and as stated, demonstrated varying and inconsistent outcomes to the previous reports. Conclusive comments by Krause et al in summary of their work were as follows: *"Although we were able to reproduce some of our previous findings, the current results support the hypothesis that the possible effects of EMF on brain oscillatory systems during cognitive processing are very subtle. The EMF effects may be too small to be consistently observed and/or replicated with the EEG. If the differences found in the present studies are*

observations of an actual effect, they are far too subtle to be systematically replicated. Alternatively, the observed effects in the EEG can be a result of chance or confounding factors."

In a recent double-blinded study, Hamblin et al. [Hamblin et al., 2006] investigated a relatively large sample of 120 participants (46 male and 74 female) to examine event-related potentials and reaction time to pulsed 895 MHz exposures at 217 Hz (250 mW; SAR = 0.11 W/kg). The experimental procedure consisted of a randomised counterbalanced series of tasks including an auditory, visual (monitor 1.5 m away), and a Winsconsin card sorting task, which were divided by 90 second resting EEG recording periods. The series of tasks with EEG recording interrupts were conducted during a sham/sham session and an exposure/sham session, in succession. The mobile phone exposure to each participant lasted 30 minutes with one half of the sample population receiving exposures directly to the right hemisphere and the remaining half to the left "using a plastic cradle like apparatus" positioning a GSM phone unit over the temporal region. The total procedure was repeated twice for each participant one week apart, with participants seated in a relaxed 'eyes open' arrangement. Reported findings were only reported for the ERP and reaction time responses with underlying EEG derivations being omitted for the purpose of the study that were later reported elsewhere [Croft et al. 2007]. Given that no statistically significant differences were observed the authors withdrew from findings of their preliminary work [Hamblin et al., 2004] stating in conclusion "that there is currently no clear evidence in support of a mobile phone-related EMF effect on ERPs or reaction time".

In a most recent investigation, Lass et al. [Lass et al., 2008] published a study on "Modulated low-level electromagnetic field effects on EEG visual event-related potentials". Thirty adults were exposed to 450 MHz exposures modulated at 21 Hz (0.16 mW/cm^2; calculated SAR = 0.303 W/kg over 1 g of tissue) from a 13 cm quarter-wave antenna located 10 cm from the skin on the right side of the head. During exposure, EEG was recorded from Fz, Cz, and Pz during a sequence of visual tasks where participants were required to press the mouse button when presented on computer screen with infrequent 2 cm diameter blue circles (25% of the time), but avoid pressing when frequent 4 cm diameter circles were presented (75% of the time). Under single blind conditions during tasks, the test exposures were switched on and off, each minute, for 10 minutes where 400 stimuli in total were presented. This procedure was repeated to obtain a 10-minute sham-exposure recording with the same amount of stimuli, only in this procedure the exposure was not switched on. Two sequences in total of stimuli were presented, one for the 10-minute on/off exposure recording and one for the 10-minute sham trial, which were counterbalanced. 10-minute breaks separated both trials. Results of the study indicated that ERP response latencies and reaction times to visual stimuli were unaffected by the RF modulated exposure at 21 Hz. However, it is demonstrated by observed affects on the N100 amplitude that the earlier stages of visual information processing are more susceptible to RF effects than in later stages.

Amongst publications where RF exposures from mobile phones were investigated during cognitive performance tasks such as to test memory or reaction time, there has been strong evidence to support that there are no associated effects on these

parameters [Krause et al., 2007][Haraala et al., 2004][Hamblin et al., 2006][Russo et al., 2006] [Fritzer et al., 2007][Kleinlogel et al., 2008b][Lass et al., 2008]. However notwithstanding this, notable evidence suggesting that there could be related RF effects associated particularly with reaction time and memory load has been indicated [Freude et al., 1998][Preece et al., 1999][Jech et al., 2001][Smythe et al., 2003]. Indeed where effects have been shown on ERPs outcomes have been very subtle and difficult to accurately replicate. Thus, given that effects have been difficult to substantiate even with large sample sizes it is considered that a trend towards negligible changes due to mobile phone exposures on ERP responses in presently prevailing.

Articles: Mobile phone RF effects on Event-Related Potentials	Main outlined findings	Type of exposure	No. of participants/ single or double blind	Duration of exposure	SAR
Freude et al., 1998	Investigation of slow brain potentials (SPs) notably indicated that exposure affected the temporo-parietal-occipital regions in decreasing the SPs during the VMT. No effect was determined in the induced potentials caused by the finger movement task due to exposure.	916.2 MHz pulsed modulated at 217 Hz (2.8 W peak), positioned over the left ear	16 adults; single blind	~8 minutes exposure total over self paced finger movements and visual monitoring task	0.882 mW/kg over 10 g of tissue
Jech et al., 2001	Over full 10-20 EEG standard recordings, most notable results indicated that 'exposure enhanced the positivity of the ERP endogenous complex in response to target stimuli in the right hemifield of the monitor'. RTs were also shortened in the target stimulus task by 20 ms.	900 MHz pulsed 217 Hz with 2 Hz and 8 Hz components active (2 W peak), positioned over the right ear	22 adult patients with narcolepsy-cataplexy - 17 investigated on visual ERP; double blind	45 minutes exposure during visual monitoring to target stimuli	0.06 W/kg over 10 g of tissue
Hamblin et al., 2006 (Preliminary study: Hamblin et al., 2004)	No statistically significant differences observed [2006], in contrast to preliminary work where latency periods of sensory/cognitive ERP components N100/P300 were affected [2004]. The authors in conclusion to their 2006 study indicated that currently there is no clear evidence in support of a mobile phone-related EMF effect on ERPs or reaction time.	895 MHz mobile phone exposures at 217 Hz (250 mW avg) positioned ear-mouth over the left or right ear	120 adults; double blind; eyes open condition indicated	30 minute exposure (60 subjects with exposure over left ear; remaining 60 subjects with exposure over right ear) during auditory and visual oddball task	0.674 W/kg over 10g of tissue

Table 2.3 (1 of 2) Mobile phone RF effects on Event-Related Potentials

Krause et al., 2007 (Working summary of Krause et al., 2000a, 2000b, 2004, 2006)	Notable conclusive comments to the work of Krause et al., suggest that 'possible effects of EMF on brain oscillatory systems during cognitive processing are very subtle'….'if differences found in the present studies are observations of an actual effect, they are far too subtle to be systematically replicated'.	902 MHz mobile phone pulsed at 217 (avg. 0.25 W); independent tests on left and right ear (CW and pulsed condition tested)	Two groups both with 36 adults; double blind	27-minute exposure for each exposure condition (CW and pulsed modultated)	0.738 W/kg over 10 g of tissue
Fritzer et al., 2007	Non-significant effects reported on the investigation of sleep EEG, attention, visual searching, mental processing speed, concentration, and short-term memory. Results could not substantiate the effect that non-REM sleep EEG power can be increased in the 9–14 Hz range as indicated by Huber et al., 2003.	900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm above the head while lying down	10 adults exposure grp/10 sham; double blind; eyes closed during a sleep onset period	6 nights sleep during exposure	Calculated 1 W/kg indicated
Kleinlogel et al., 2008b	Results of the study for resting EEG [Kleinlogel et al., 2008a] did not indicate any statistically significant changes due to exposure. Similarly, no changes due to exposure could be reported on ERPs or RTs. In conclusion to both studies, the authors remark that outcomes of their work 'do not give any evidence for a deleterious effect of the EMF on normal healthy mobile phone users'.	GSM 900 MHz and UMTS 1950 MHz exposures, positioned over the left ear	15 adults; double blind	2.5 minutes VEP (target stimuli in oddball task); 11 minutes continuous performance test (CPT); two auditory evoked (2 & 6 minutes) sessions (RTs recorded in the second)	1 W/kg (GSM 900 MHz) and SAR 0.1 or 1 W/kg (UMTS 1950 MHz)
Lass et al., 2008	Results of the study indicated that ERP response latencies and reaction times to visual stimuli were unaffected by the RF modulated exposure at 21 Hz. However, it is demonstrated by observed affects on the N100 amplitude that the earlier stages of visual information processing are more susceptible to RF effects than in the later stages.	450 MHz exposures modulated at 21 Hz (0.16 mW/cm^2) from a 13 cm quarter-wave antenna located 10 cm from the skin on the right side of the head	30 adults; single blind	10 minutes exposure every alternate minute during VEP in computer screen target stimulus trial	Calculated 0.303 W/kg over 1 g of tissue

Table 2.3 (Continued: 2/2) Mobile phone RF effects on Event-Related Potentials

2.2 Effects of Mobile Phone Exposures on Heart Function

Apart from studies addressing mobile phone exposure effects on the human EEG, the electrocardiograph (ECG), pulse-plethysmograph (PPG), and blood pressure (BP) parameters have been used previously to assess RF exposures on heart function. The following section summarises the current literature with respect to these investigations.

In a double blind investigation by Tahvanainen et al. [Tahvanainen et al., 2004], 32 participants underwent several measurements of heart rate and blood pressure (systolic/diastolic) by use of an arm cuff during mobile phone exposures. 35-minute measurements were acquired both during and after a randomised sequence of 900 MHz (2 W peak; SAR = 1.58 W/kg) and 1800 MHz (1 W peak; SAR = 0.7 W/kg) exposures generated by a Nokia dual band mobile phone controlled via serial port by a notebook computer. The authors investigated the cardiovascular responses during controlled deep and spontaneous breathing as well as during Valsalva manoeuvre during supine rest and 'head-up' tilt test. Although results of the investigation indicated a slight, progressive increase in systolic and diastolic blood pressure, it was suggested that randomisation of the experimental protocol dismissed this observation to be independent of a resultant effect due to RF exposure. Furthermore, there were no resulting statistically significant changes in blood pressure or heart rate during exposure, as well as no evident changes in heart rate in investigated post-trial periods.

Consequently, Tahvanainen et al. conclude that 900 MHz or 1800 MHz mobile phone exposures do not acutely affect arterial blood pressure and heart rate.

In a somewhat similar and earlier investigation to Tahvanainen et al., Braune et al. [Braune et al., 1998] recorded test measures of blood pressure, heart rate, and capillary perfusion of 9 participants during a continuous 35-minute exposure to a remote controlled GSM 900 MHz pulsed 217 Hz (2 W) mobile phone. Following the exposure period, test measurements were taken over a 1-minute period during supine rest and standing up, deep breathing, and during the Valsalva manoeuvre. The test procedure was repeated 5 times for each participant on different days. Results of the investigation indicated that systolic and diastolic measures of blood pressure at rest were observed to be considerably higher in the exposure recordings as compared to recorded sham. In addition blood pressure during standing was found to be significantly higher during exposure. During rest significant differences of blood pressure were found to be lower during the first phase of the Valsalva manoeuvre but were higher in the subsequent phases II an IIe. The group interpreted these findings as occurring due to the noted increase of blood pressure at rest. In addition to these results capillary perfusion measurements showed a significantly greater vasoconstriction (as indicated by recordings of the plethysmograph), while the heart rate both for rest and Valsalva manoeuvres measures, was indicated to be significantly decreased. Conclusions of this investigation stated that the RF exposures caused an increase in sympathetic efferent activity with increases in resting blood pressure however such findings were conclusively withdrawn upon a subsequent investigation of these results which indicated non-significant outcomes [Braune et al , 2002].

In addition to Braune et al, Atlasz et al. [Atlasz et al., 2006] also used plethysymograph measures to investigate heart function. In an aim to test whether 900 MHz GSM mobile phone exposures (2 W; pulsed 217 Hz) result in an influence in heart rate variability (HRV), Atlasz et al. incorporated 35 adults in one randomised trial in a 5-minute supine rest position followed by a 5-minute standing position during continuous exposure. Post-exposure plethysmograph measurements were taken during an allocated recovery period. SAR measurements were reported at 1.3 W/kg. Results indicated that there was no significant difference between HRV in sham and exposure outcomes, however observed heart rate variations in 3 of the 35 participants were in particular observed when the subjects were in the standing position following exposure. The authors report that it may be possible that there is a proportion of the population that may be more sensitive to RF exposure than the average population. In conclusion the authors report that based on their experimental conditions RF fields emitted by cellular phones do not cause observable effects on the regulation of heart rate.

In a most recent double-blind study on the effects of RF exposures from mobile phones on HRV, Parazzini et al. [Parazzini et al., 2007] reported on an investigation where 26 participants were exposed to a PC controlled 900 MHz GSM cellular phone operating at full power (2 W; SAR < 0.02 W/kg) for 26 minutes in one session and 26 minutes during a sham trial. The test procedure was in accordance to a rest-stand protocol with each participant resting in the supine position for 13 min and then standing for the latter half. Continuous ECG recordings were acquired throughout

both the sham and exposure periods. A novelly implemented approach to analyse HRV by measures of 'HRV indexes' was performed. Reported results of the study indicated that 26 minutes RF field exposure from a 900 MHz mobile phone did not induce any acute change in central regulation of cardiovascular functions. No statistically significant difference could be shown during rest conditions for all HRV measures, however significant but weak increase in spectral ECG power was indicated in the standing condition as well as a decrease in the variability of the ECG cyclic length as measured by the index analysis. In addition gender dependent decreases were observed to be statistically significant for the standing condition for women (N = 12) during exposure. The authors present the possibility of the weak observed changes as potentially occurring due to a sympathetic drive to the heart. Furthermore it is noted that other possible RF effects were observed only during sympathetic response or when the responsiveness to stand was evaluated. The report concludes generally that EMF RF exposure produces no effects on HRV, however suggests that the findings given for the minor indexes of the investigation should be considered in amongst the framework of future comparable studies.

In addition to studies investigating the RF effects of mobile phone exposures on heart function, Mann et al. [Mann et al., 1998], and Huber et al [Huber et al., 2003] in addition to their work on EEG response, both reported on the effects of mobile phone exposures on heart rate during sleep. Whilst Mann et al. did not indicate any significant influence in ECG recordings of 12 men during sleep (900 MHz pulsed 217 Hz), Huber et al. reported on interesting findings using a similar RF source response. In two types of sleep experiments conducted by Huber et al. [Huber et al., 2003]

where participants were investigated during 15-minute on/off intervals of pulsed 900 MHz signals in the first experiment (N = 24) and 30 minutes of the same exposure prior to a morning sleep in the second (N = 16), the resultant outcomes were shown to differ. During the first experiment although the heart rate did not indicate a change, ECG spectral power measures were shown to statistically significantly decrease in the narrow band 0.1 – 0.11 Hz following RF exposure in the sleep onset period (R-R spectra; low frequency range) as well as increase mean spectra over the first three non-REM sleep episodes in the 0.29 – 0.31 Hz band (5 bins around peak; high frequency range). During the second of the two experiments no differences in heart rate were observed during the 30-minute exposure period however the authors report observations of reductions in ECG amplitude in the waking and stage 1 of sleep following exposure to both hemispheres as well as prior to the sleep onset period following exposure to the right hemisphere. During the sleep period following exposure power in the 0.18 – 0.22 Hz range was also noted to increase in 11 spectral bins surrounding the peak (high frequency range) during left and right hemisphere exposure. The authors interpret the difference in location of these high frequency peak effects between experiments as potentially resulting from "circadian factors" during night sleep as versus daytime sleep. In noting a relatively small sample size of the experiments conducted, in conclusion it is suggested by the authors that the subtle changes in cardiac activity presented require further confirmation.

In relation to reported RF effects on heart function, the studies collectively suggest that there is no clear evidence to support RF associated effects with the ECG, PPG, or blood pressure parameters [Mann et al., 1998][Braune et al., 2002][Tahvanainen et

al., 2004][Atlasz et al., 2006]. However given that only few studies have been published in this area of research and that few positive findings have also been noted in both sleep and awake investigations [Huber et al., 2003][Parazzini et al., 2007] further work would need to be conducted to replicate these studies and independent analytical approaches before any reasonable hypothesis can be made.

Articles: Mobile phone RF effects on Heart Function	Main outlined findings	Type of exposure	No. of participants/ single or double blind	Duration of exposure	SAR
Mann et al., 1998	No significant changes reported on the ECG, specifically on heart rate during sleep.	900 MHz pulsed 217 Hz mobile phone, positioned 40 cm below the pillow of participants	12 adults; single blind	8 hrs (1 night)	Max. 0.6 W/kg
Braune et al , 2002 (Preliminary study: Braune et al., 1998)	No statistically significant differences observed [2002] in contrast to previous findings [1998] where Systolic and diastolic measures of blood pressure at rest were observed to be considerably higher due to exposure. Also, capillary perfusion measurements indicated a significantly greater vasoconstriction, while heart rate for both rest and Valsalva manoeuvres was significantly decreased.	900 MHz pulsed 217 Hz (2 W) mobile phone, positioned in an ear-mouth position on the right side	9 adults; single blind	35-minute exposure periods (five periods total, administered each on different days)	undefined
Huber et al., 2003	During the first experiment ECG spectral power measures were shown to statistically significantly decrease in the narrow band 0.1 – 0.11 Hz, following RF exposure in the sleep onset period (R-R spectra; low frequency range). An increase was also shown in mean spectra over the first three non-REM sleep episodes in the 0.29 – 0.31 Hz band (5 bins around peak; high frequency range). During the second experiment no differences in heart rate were observed however observations of reductions in ECG amplitude in the waking and stage 1 of sleep following exposure to both hemispheres as well as prior to the sleep onset period following exposure to the right hemisphere.	1st exp: 900 MHz exposures (pulsed 2 Hz, 8 Hz, and 217 Hz) from a 3 half-wavelength dipole array positioned 30 cm behind the head while lying down 2nd exp: Patch antenna with comparable EMF as in exp 1, positioned ~11 cm from both the left and right side of the head of seated participants	1st exp: 24 adults; double blind; eyes closed during a sleep onset period 2nd exp: 16 adults; double blind;	1st exp: multiple 15 minute exposures 2nd exp: 30 minutes of exposure	Exp 1 and 2: 1 W/kg over 10 g of tissue

Table 2.4 (1 of 2) Mobile phone RF effects on Heart Function

Tahvanainen et al., 2004	No resulting statistically significant changes in blood pressure or heart rate during exposure, as well as no evident changes in heart rate in post-trial periods. It is concluded that 900 MHz or 1800 MHz mobile phone exposures do not acutely affect arterial blood pressure and heart rate.	Nokia dual band mobile phone (900 MHz/2W peak; 1800 MHz/1W peak), positioned ear-mouth over the ear of the dominant hand side	32 adults; double blind	35-minute exposure period at 900 MHz and 1800 MHz	1.58 W/kg (at 900 MHz); 0.7 W/kg (at 1800 MHz)
Atlasz et al., 2006	No significant changes in HRV due to exposure, however observed heart rate changes in 3 of the 35 participants were in particular observed when participants were in a standing position following exposure.	900 MHz pulsed 217 Hz (2 W) mobile phone	35 adults; single blind	10 minutes continuous exposure	1.3 W/kg
Parazzini et al., 2007	No statistically significant difference could be shown during rest conditions for HRV measures, however significant but weak increase in spectral ECG power was indicated in a standing condition as well as a decrease in the variability of the ECG cyclic length.	900 MHz mobile phone operating at full power (2 W; GSM), positioned ear-mouth over the ear of the dominant hand side	26 adults; double blind	26 minutes of exposure	< 0.02 W/kg

Table 2.4 (2 of 2) Mobile phone RF effects on Heart Function

2.3 Summary of Review

To enable valid assumptions or hypotheses to be extracted from the literature concerning the effects of mobile phone exposures on the EEG there are a number of common experimental variables that need to be considered. These variables include the exposure source and it's location with respect to test participants' heads, the exposure source operating frequency and associated modulations, the exposure field strength and SAR with respect to the participant's head, as well as any noted dosimetry undertaken. Moreover, the duration of exposure, the participant conditions (such as awake, seated, with eyes closed), the number of recording electrodes positioned on the head, the statistical analysis method used, and the sample size in support of the statistical strength of the experiment, are all factors importantly required to be considered. However, it is arguable that due to an apparent lack of replication of the described common test variables across the literature, it may be considered that as it stands published experimental outcomes concerning RF effects on the EEG cannot still in most cases be directly compared.

However in stating this, it should also be clearly noted that there has been research groups who have undertaken ongoing replication studies of their own work [Wagner et al 1998] [Haarala et al., 2003][Krause et al., 2004][Huber et al., 2003][Hamblin et al., 2007] as well as few who have replicated the work of others [Röschke et al., 1997][Fritzer et al., 2007]. In overview of this replication work, it may said that until now results of these studies have not demonstrated any consistent findings with previous reports which can be evidently interpreted as positive RF effects on the EEG. Krause et al [Krause et al., 2007] in observation of the drawbacks in identifying

consistent findings amongst a series of their investigations on mobile phone exposures on human cognitive tasks, recently reported in summary (as outlined) that effects on brain oscillatory systems during cognitive processing may be either very subtle or too small to be consistently observed or systematically replicated. The group interestingly also express that there is still possibility that observable effects which have been shown may have occurred due to statistical chance or experimental confounding factors.

Apart from replication investigations, if on a global basis, the current experimental outcomes of mobile phone effects on brain wave activity are to be compared, an EEG response in the alpha band or alpha rhythms may be indicated in many and during all three participant conditions including the awake still position, sleep, and during cognitive tasks [Reiser et al., 1995][Croft et al., 2002, 2007][Huber et al., 2003] [D'Costa et al., 2003][Hinrikus et al., 2004][Curcio et al., 2005]. Amongst these reports, effects in the alpha band may be shown to be interestingly found at ipsilateral and/or contralateral sites with respect to the position of the exposure source and may have be shown to demonstrate both increases and decreases in spectral power. One plausible hypothesis for effects as seen at different locations on the head was presented by Curcio et al. [Curcio et al., 2005] who in reference to their own outcomes deduce that as an effect was presented at the recorded sites Cz and Pz (that represent the summated activity from bilateral areas of the brain) EMF exposure could be induced by a possible spreading effect of RF power across the brain.

2.4 Critical Research Questions

Based on the provided literature, the following critical research questions were determined to aid in forming a basis upon which the main experimental research (Chapter 8) was to be conducted.

1. Only one study conducted, namely by Hietanen et al. 2000, appears to have investigated the awake response of 1800 MHz mobile phone exposures on the EEG. Similarly only one investigation, namely by Tahvanainen et al. 2004, appears to have been reported that has investigated the effects of 1800 MHz exposures on heart function by measures of blood pressure. No studies appear to have been reported on the effects of 1800 MHz exposures on the ECG or heart rate variability.

   Given the above gaps in the current body of knowledge, does 1800 MHz exposures as also known to be emitted by mobile phones influence either the awake EEG response in humans, or measures of ECG or blood pressure?

2. Some studies, such as by D'Costa et al. 2003, Curcio et al. 2005, and Croft et al. 2007, have all noted effects in the EEG from head regions nearest to the exposure source.

   Does the position of the mobile phone exposure source correspond to an influence in brain wave activity from adjacent recording sites?

3. Many investigations as described have indicated a significant RF influence on the EEG typically due to pulsed 900 MHz exposures at near field distances. These changes may be noted to have been observed within all four EEG spectral bands, namely within delta, theta, alpha, and beta. Apart from very few, all reported studies have indicated results with relatively low sample sizes ranging between 10 – 35 participants. Furthermore it is prevalent that no study with a relatively large sample size (> 50) has yet been reported for the most stable EEG recording condition, specifically where the participant is awake with eyes closed.

   Are there statistically significant alterations within any of the four distinct EEG bands namely delta, theta, alpha, or beta, when the heads of test participants at near-field distances from mobile phone exposure source? Do related effects in the EEG significantly occur under the awake, closed eyed conditions for larger sample sizes, such as a sample ranging up to 100 participants?

4. There have been only very few studies reported which have analysed the EEG at a higher spectral resolution than that provided by averaged EEG data across the clinically significant bands of delta (1-4 Hz), theta (4- 8 Hz), alpha (8 – 12 Hz), and beta (12–32 Hz) [Curcio et al. 2005].

   Do mobile phone exposures cause statistically significant power increases or decreases in *discrete* EEG rhythms over the distinct spectral range of 1 – 32 Hz (i.e. at least at 1 Hz, 2 Hz, 3 Hz)? Do any positive findings occur at high statistical strength, such as would be provided by sample sizes ranging up to 100 participants?

5. It appears that in no study to date, has mobile phone exposures on the awake EEG been investigated at close to acceptable maximum general public SAR limits. The ARPANSA standard (RPS3) 2002, specifies a maximum general public limit to the head and torso of 2 W/kg over any 10 gram of tissue.

   Does both 900 MHz and/or 1800 MHz mobile phone exposures as close to the maximum allowable SAR limit influence nervous function such as in the EEG or in heart responses (ECG, PPG, or blood pressure)?

6. The literature predominantly indicates that 'short-term' mobile phone exposure effects may be result in the EEG or in heart responses of ECG or blood pressure.

   Are there 'short-term' influences in the EEG, ECG or blood pressure during RF exposures from mobile phones?

7. As EEG electrode leads are present in the vicinity of RF exposures during test measurements some investigators have examined the possibility that 900 MHz exposures may be picked up or shielded by the electrode leads to an extent that may adversely influence predicted SAR levels inside the heads of test participants [Wood et al. 2001][Croft et al. 2001][Hamblin et al., 2007][D'Costa et al., 2003, 2004]. While some minimal influences on the SAR have been noted, no investigation has studied the effects of 1800 MHz exposures, or clearly mapped whether the distribution of energy or spatial-peak SAR may re-locate, or whether observed changes in SAR are specifically due to RF coupling, shielding, or both. In addition, current outputs of software RF modelling techniques have been limited and have not necessarily considered a realistic full array of electrode leads on a head model. All

outlined gaps in the current body of knowledge concerning the influence of EEG recording leads on SAR is vital aspect, if positive findings from mobile phone/EEG investigations are to be interpreted correctly.

Are 900 MHz or 1800 MHz mobile phone exposures shielded by, or coupled to EEG electrode recording leads to an extent that may influence the peak spatial-average SAR or its location inside the head?

8. There are currently no investigations in the current body of literature that have investigated the correlation of effects due to mobile phone exposures between measurements of EEG and the ECG.

   Do potential effects due to mobile phone exposures in brain wave activity correspond to heart responses such as between trial measures of the EEG and ECG?

Chapter 3

EEG measures during Radiofrequency Exposures from Mobile Phones: A Pilot Study

3.1 Introduction

Before administering a larger study to investigate EEG effects resulting from mobile phone exposures (as described in Chapter 7), a pilot study incorporating 10 participants was first investigated to establish direction of results and to gain a understanding of the fundamentals of this work. The following chapter aims to describe this pilot study where the awake EEG was recorded and participants were required to be seated still with eyes closed whilst being exposed to GSM RF emissions from mobile phones in standard operating conditions of *full-power* and *standby* mode. Perhaps one of the most important outcomes of this investigation was that statistical endpoints were later (Chapter 7) able to

be used to confirm and confidently identify major significant increase findings in the alpha band, particularly in the 9 Hz rhythm from all surrounding regions of the head.

3.2 Materials and Methods

3.2.1 Sources of Exposure

This investigation was conducted in two trials using two different mobile phones in different power output modes. In the first trial, RF exposures were generated by a modified Nokia 6110 mobile phone which had its speaker disabled to eliminate possible auditory cues. The phone was configured to be enabled via control software to transmit at a nominal average power of 250 mW (2 W peak output) with a 900 MHz carrier modulated at 217 Hz. As a consequence of this configuration, standby mode exposures could not be activated and any power variability caused by GSM power control modes such as discontinuous transmission (DTX) [ETS 300 578 std., 1999] was removed. Thus only nominal full-radiated power was transmitted. In the second trial, standby mode exposures were generated by a standard Ericsson GH388 GSM mobile phone. In this mode of operation, GSM mobile phones such as these have been found to emit low frequencies within the EEG spectrum 1-32 Hz [Heath et al., 1998]. The Nokia 6110 programmable mobile phone was supplied by Telstra Research Laboratories (2003)[*].

[*] *Telstra Research Laboratories, Clayton, Victoria*

3.2.2 Participants

Ten healthy subjects in total participated in this investigation. This included 5 men and 5 women with ages ranging from 18 to 30 years. All participants gave verbal consent that they were not on any medication in the recent month prior to the trial period. The age selection of the participants was based on the principles of cerebral maturation. As may be observed below (Figure 3.1) cerebral maturation may be established as a function of age, indicating that cerebral function approximately begins to plateau around the age of 18 while cerebral functioning following age 45 may be shown to be in decline [Niedermeyer & Da Silva, 1993]. Hence in order to select participants from a consistent sample, only participants between the ages of 18 – 45 years were permitted into the experimental work.

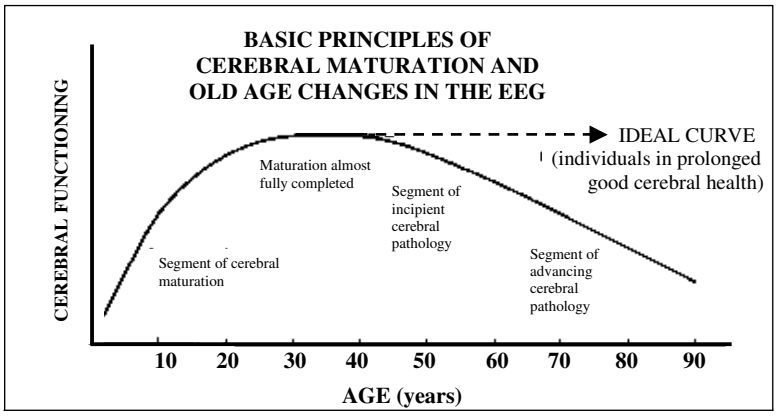


Figure 3.1. Cerebral maturation and old age changes in the EEG [Niedermeyer & da Silva, 1993].

3.2.3 Participant Conditions

For each subject EEG electrodes were positioned at the Fp1 & Fp2, C3 & C4, O1 & O2 symmetrical recording sites according to the 10/20 international standard [Jasper, 1958][Carr & Brown, 1998]. To sustain good conductivity during trials, the electrodes were checked for secure contact between all EEG recordings. The electrode leads were twisted as far as possible, as a measure against the potential for RF pickup, and then fed into a differential amplifier (Mindset EEG acquisition unit).

A mobile phone was placed horizontally behind the subject's head with the antenna positioned 2 cm away from the occipital region (between O1 and O2) as shown in Figure 3.2 below. The position of the antenna was located at the back of the head as to be able to most easily quantify the effects of mobile phone exposures directly on the recoded EEG both near (O1 and O2), midway (C3 and C4), and away (Fp1 and Fp2) from the exposure source. It was envisaged that the outcomes gathered from this arrangement would later provide critical information for cross-examination purposes within the body of literature as well as in the major experimental work where the handset was to be placed in an ear-mouth position (as comparable to real mobile phone use - Chapter 8).

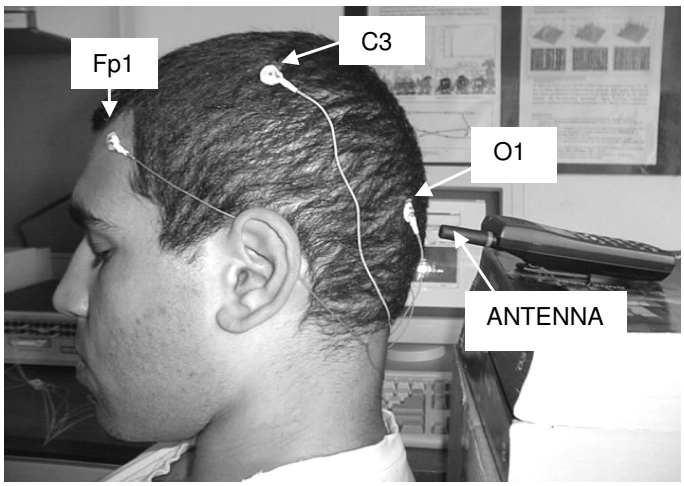


Figure 3.2. Photograph of phone/antenna positioning with respect to the head and the EEG electrode recording sites (symmetrical locations Fp2, C4, and O2, not shown).

During the EEG recordings subjects were required to have their eyes closed, and to be awake whilst sitting still in a comfortable back supported chair. Subjects were able to maintain this position in a relaxed state.

3.3 Trial Procedure

Two trials were conducted. During the first trial, the real-time EEG of each subject was recorded for a total duration of 50 minutes in a silent room. This consisted of ten 5-minute EEG recordings of which five were recorded whilst the Nokia mobile phone was in *full-power mode* and positioned at the rear of the head (Figure 3.2). The remaining five recordings were sham exposure recordings. During the sham exposure recordings the mobile phone was switched off completely though remained in position. For each subject, the five exposure and five sham EEG recordings were randomised such that participants were not aware of the state of exposure of the mobile phone. After every 5-minute EEG

recording subjects took a 10-15 minute break. During the second trial of this investigation, the Ericsson mobile phone in *standby mode* was used as the source of exposure. Apart from this, the procedure of the second trial was identical the first. The same ten subjects participated in both trials.

3.4 Analysis and Results

All EEG data collected during the two trials was realised in the frequency domain between 1 - 32 Hz using the Mindset EEG processing software (Wavelab V.1.0). For each subject, the EEG data obtained under both sham and exposure conditions was then classified into the four clinically significant EEG bands, namely, delta (1 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz), and beta (13 – 32 Hz). The five sham recordings as well as the five exposure recordings (obtained from each subject in both trials) was then averaged within the EEG bands so that there was one set of sham mean data and one set of exposure mean data per recording site. Hence, as EEG data was simultaneously accumulated from three recording sites (Figure 3.2), there were three sets of sham/exposure EEG data to be analysed for each subject in the full-power mode and standby mode trials.

A paired t-test was conducted to statistically analyse whether there were any significant differences between the resultant averaged power in the sham and exposure data sets. The results of the t-test are outlined in Tables 3.1 & 3.2 where p-values are considered significant at less than 0.05.

FULL-POWER MODE TRIAL								
	DELTA		THETA		ALPHA		BETA	
Recording Site	95% CI (μV)	p-value	95% CI (μV)	p-value	95% CI (μV)	p-value	95% CI (μV)	p-value
Frontal	-2.3, 7.0	0.281	-1.3, 3.8	0.289	-0.5, 1.8	0.264	-0.6, 1.1	0.519
Central	-1.0, 9.0	0.106	-1.9, 7.0	0.232	0.1, 3.7	**0.038**	0.03, 1.9	**0.045**
Occipital	-0.4, 11.3	0.065	-1.3, 8.8	0.13	-0.1, 5.3	0.06	0.01, 3.0	**0.049**

Table 3.1 T-test results indicated for the full-power mode trial where averaged sham exposure recordings were compared to averaged exposure test recordings in the delta, theta, alpha, and beta EEG bands. Statistical levels were considered significant at p-values < 0.05 (shown in bold).

STANDBY MODE TRIAL								
	DELTA		THETA		ALPHA		BETA	
Recording Site	95% CI (μV)	p-value	95% CI (μV)	p-value	95% CI (μV)	p-value	95% CI (μV)	p-value
Frontal	-2.1, 0.4	0.166	-0.6, 0.2	0.320	-0.4, 0.1	0.337	-0.3, 0.1	0.332
Central	-1.5, 2.3	0.621	-0.8, 2.3	0.287	-0.3, 0.6	0.442	-0.2, 0.3	0.775
Occipital	-0.5, 1.7	0.215	-1.0, 0.9	0.916	-1.2, 1.2	0.959	-0.2, 0.3	0.583

Table 3.2 T-test results indicated for the standby mode trial where averaged sham exposure recordings were compared to averaged exposure test recordings in the delta, theta, alpha, and beta EEG bands. Statistical levels were considered significant at p-values < 0.05.

In addition to determining the statistical levels of difference between the mean sham and exposure EEG recordings within the delta, theta, alpha, and beta bands, further analysis was undertaken to observe whether there were any trends in the number of subjects indicating a decrease or increase in EEG power as a result of the full-power mode and standby mode exposures. To do this, for each subject, the median sham data minus the median exposure data was calculated for each discrete EEG rhythm between 1 – 32 Hz. As the exposure data was subtracted from the sham data, a negative result was labelled an

'increase' in EEG power, a positive result was labelled a 'decrease' in EEG power, and a null result was labelled a 'no difference' in EEG power. Figures 3.3 & 3.4 indicate the percentage of subjects that indicated an EEG power decrease in each EEG rhythm as well as show the corresponding distributions of the frontal, central, and occipital recording regions of interest.

Importantly, it should be recognised in viewing results that a 'decreased' difference for example in EEG spectral power from differential recordings (i.e. the spectral power 'difference' usually between two symmetrical sites) as applied in this study, does not correspond to decreases that may otherwise be shown in referential recordings, such as EEG electrode sites referenced to the mastoid (or a near approximate zero electrical potential). Rather, a 'decreased' difference between differential recordings at symmetrical locations (such as recorded from Fp1 and Fp2 over comparison of sham and exposure conditions) may be logically determined to represent 'increased' spectral coherence between paired sites. Likewise, in contrast, an 'increased' difference in EEG spectral power between differential recordings (sham vs. exposure) from the same paired sites, may be logically determined to represent 'lowered' spectral coherence between the recordings.

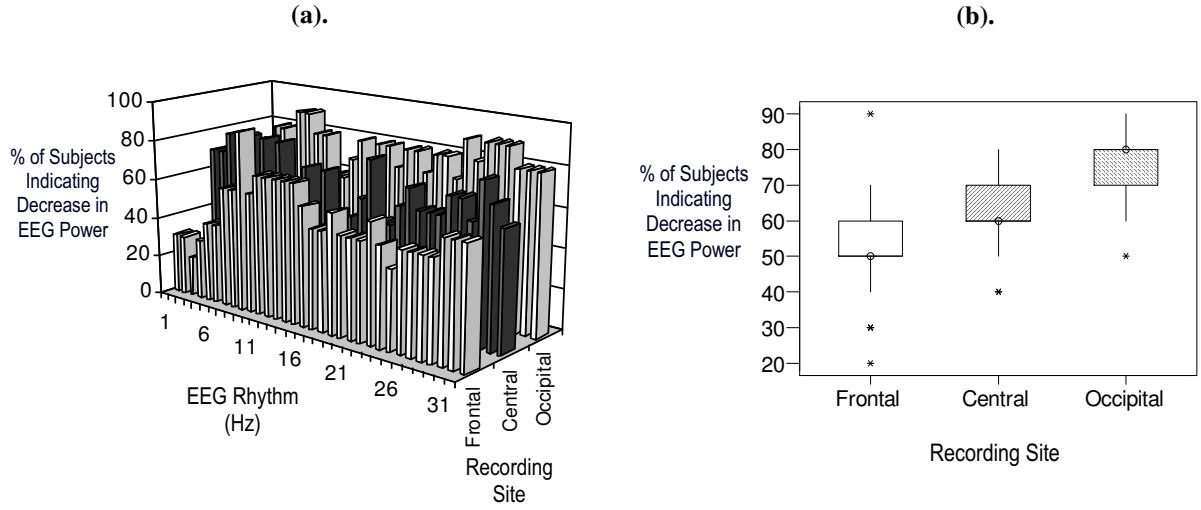


Figure 3.3 Full-power mode trial results. **(a).** For each subject, the median sham data minus the median exposure data was calculated for each discrete EEG rhythm between 1 – 32 Hz. Resultant decreases in EEG power were identified. The percentage of subjects indicating a decrease in EEG power are shown in each rhythm from the standard frontal, central, and occipital regions. **(b).** The corresponding distribution plots of Figure 3.3(a). The 'boxes' indicate where at least 50% of the distribution lies. The 'box-whiskers' and asterisks (data outliers) together indicate where at least 25% of the distribution lies. The circles indicate the median point of the distribution.

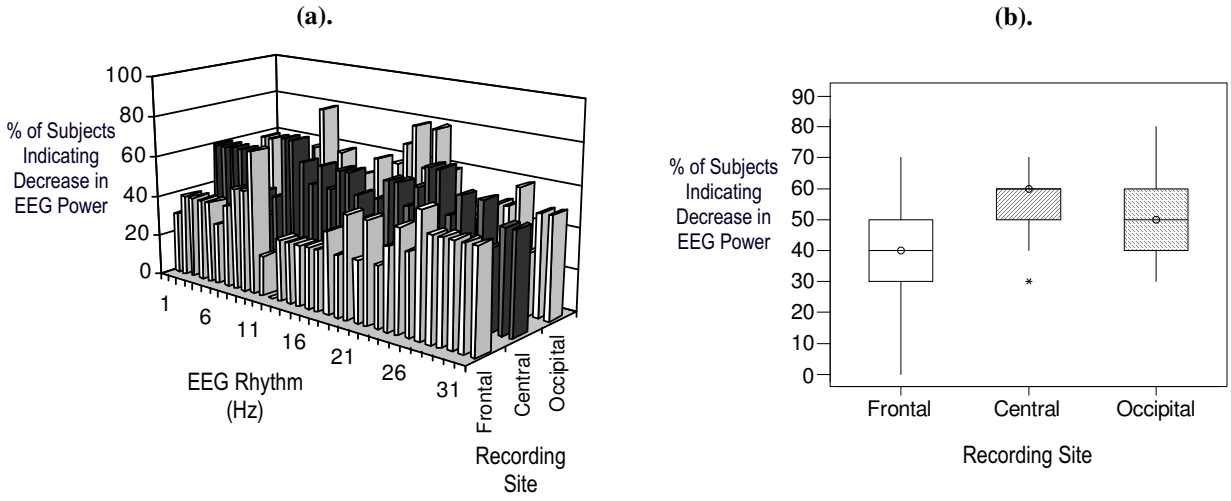


Figure 3.4 Standby mode trial results. **(a).** For each subject, the median sham data minus the median exposure data was calculated for each discrete EEG rhythm between 1 – 32 Hz. Resultant decreases in EEG power were identified. The percentage of subjects indicating a decrease in EEG power are shown in each rhythm from the standard frontal, central, and occipital regions. **(b).** The corresponding distribution plots of Figure 3.4(a). The 'boxes' indicate where at least 50% of the distribution lies. The 'box-whiskers' and asterisks (data outliers) together indicate where at least 25% of the distribution lies. The circles indicate the median point of the distribution.

A subsequent paired t-test was conducted on the overall median EEG paired sample to assess the significance of difference in each band where a very high percentage (80% +) of subjects indicated a resultant decrease in EEG spectral power. Rhythms where a low percentage (20% or less) of subjects indicated a resultant decrease were also of interest as this was equivalent to high percentage of subjects indicating an increase in EEG power. The results of the t-test are indicated in Tables 3.3 & 3.4 for the full-power mode and standby-mode trials respectively. The tables indicate the EEG rhythms and corresponding recording sites at which the high tendency decreases and increases occurred as well as the resultant p-values (considered significant at $p<0.05$).

FULL-POWER MODE TRIAL				
EEG Rhythm (Hz)	Rec. Site	% of Subjects Indicating Decrease	95% CI (μV)	p-value
3	F	20	-3.1, 4.6	0.673
9	F	90	0.1, 0.6	**0.009**
3	C	80	-1.9,10.2	0.155
5	C	80	-1.3, 7.5	0.141
7	C	80	0.6, 5.0	**0.020**
9	C	80	0.5, 4.9	**0.022**
19	C	80	-0.1, 1.7	0.076
30	C	80	-0.1, 1.7	0.070
4	O	80	-0.9,10.3	0.089
5	O	80	-0.8, 9.7	0.087
7	O	90	0.5, 9.0	**0.032**
8	O	90	0.7, 8.7	**0.026**
9	O	80	0.9, 6.9	**0.017**
10	O	80	-0.4, 7.5	0.073
14	O	80	-0.3, 5.2	0.071
16	O	80	-0.2, 5.7	0.065
17	O	80	0.0, 5.3	**0.050**
19	O	80	0.0, 4.9	**0.050**
20	O	80	-0.4, 5.4	0.086
22	O	80	-0.1, 4.3	0.063
23	O	80	-0.1, 3.6	0.056
25	O	90	-0.1, 3.5	0.061
26	O	80	-0.2, 3.0	0.082
27	O	90	0.1, 2.6	**0.034**
28	O	90	-0.0, 2.4	0.055
29	O	90	-0.2, 2.4	0.078
30	O	80	-0.5, 2.7	0.151
31	O	80	-0.7, 2.4	0.248
32	O	80	-0.5, 1.8	0.231

Table 3.3 T-test results in the full-power mode trial following analysis of differences in sham and exposure median spectral power. Shown are the significant levels of cases where a high and low percentage of subjects indicated resultant decrease in EEG power. F = Frontal; C = Central; O = Occipital.

STANDBY MODE TRIAL				
EEG Rhythm (Hz)	Rec. Site	% of Subjects Indicating Decrease	95% CI (μV)	p-value
11	F	20	-0.8,0.03	0.069
12	F	0^	-0.5,-0.1	**0.004**
9	C	80	-0.2, 5.1	0.062
19	C	80	-0.1, 0.9	0.090
21	C	80	-0.1, 0.6	0.093

Table 3.4 T-test results in the standby mode trial following analysis of differences in sham and exposure median spectral power. Shown are the significant levels of cases where a high and low percentage of subjects indicated resultant decrease in EEG power. F = Frontal; C = Central; O = Occipital

^ 0% decrease though 90% of subjects indicating increase in EEG power due to one subject showing a 'no difference' between sham and exposure data.

In addition to the full-power and standby mode trials, electrode lead susceptibility to RF emissions from a Nokia 6110 mobile phone in active *talk mode* was tested. In this trial two EEG electrodes were positioned 10 cm apart on an adults thigh, perpendicular to the length of the leg. The subject was seated still with the thigh horizontally at rest to prevent possible pickup of biopotential activity caused by movement. As in the full-power and standby mode trials, five 5-minute differential sham exposure and exposure recordings were logged by the Mindset EEG acquisition unit. The exposure recordings required the mobile phone to be suspended 2 cm above the thigh, at the mid-point between the two electrodes. A paired t-test was conducted to compare the average spectral sham exposure and exposure data in the EEG delta (1 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz), and beta (13 – 32 Hz) bands. Resultant statistical levels in all four bands indicated p-values greater than 0.2 indicating no significant difference between the mean sham exposure and exposure data (p-values considered significant at $p<0.05$).

3.5 Discussion

Results of the full-power mode trial where the average spectral power in the delta, theta, alpha, and beta EEG bands were considered (Tables 3.1 & 3.2) indicate that there were statistically significant levels of difference between the sham and exposure tests conducted. These results can be seen in Table 3.1 (highlighted) in the alpha and beta bands from the central recording site ($p = 0.038$ and $p = 0.045$ respectively) and in the beta band from the occipital region ($p = 0.049$). The 95% confidence intervals of these significant results

suggest that there was a decrease in mean EEG potential between as low as 0.01 μV to 3.7 μV during the full-power mode exposure. Notably, there was no apparent alteration in EEG activity from the frontal region. This absence of change in frontal region activity coincides with reports made by Reiser, et al. [Reiser et., 1995] where the active mobile phone source was also placed behind the subject's head. No statistically significant results are apparent in the standby mode trial (Table 3.2) over the delta, theta, alpha, and beta bands.

Also indicating no statistically significant levels in the four EEG bands of interest were the results of the electrode lead susceptibility trial where EEG electrode leads (positioned on a subject's thigh) were exposed to mobile phone talk mode emissions. The results of this trial suggest that neither the GSM talk mode emissions nor the data acquisition equipment influenced the electrode leads during EEG recordings. These findings are in accordance with other investigators who also report negligible effects of mobile phone RF emissions on EEG electrode leads [Hietanen et al. 2000].

Apart from analysis of average spectral power in the delta, theta, alpha, and beta EEG bands, analysis of sham and exposure median differences in discrete EEG rhythms were considered (Figures 3.3 & 3.4). Figure 3.3(a) indicates results of this analysis for the full-power mode trial. Observation of the figure shows a particularly high percentage (80% +) of subjects indicating a decrease in EEG power over most of the occipital EEG rhythms. Furthermore, the occipital results can be seen to be evidently higher than the frontal and

central region results. The distribution plots (box graphs) of Figure 3.3(b) outline this particular observation. The occipital distribution in Figure 3.3(b) demonstrates that in at least 75% of the 32 EEG rhythms analysed 70% – 90 % of subjects indicated a decrease in EEG power. Neglecting data outliers, approximately 25% of the corresponding central and 75% of the frontal distributions lie below the occipital plot. Consequently, the full-power mode distributions of Figure 3.3(b) appear to steadily decline from occipital (highest), to central, to frontal (lowest). It is speculated that this may occur due to the mobile phone exposure being applied from the rear of the head (closer to the occipital region and furthest from the frontal region).

Unlike the full-power mode trial results described, the corresponding standby mode graphs of Figure 3.4 do not appear to demonstrate any outstanding characteristics. Nonetheless, at 9 Hz, 19 Hz, and 21 Hz a high percentage of subjects (80% +) indicated a decrease in EEG spectral power.

Tables 3.3 and 3.4 indicate all high and low percentages of subjects indicating a decrease in EEG power for the full-power mode and standby mode trials respectively. As outlined previously, a low percentage (20% or less) in number of subjects indicating a decrease in EEG power is equivalent to a high percentage of subjects indicating an increase in EEG power. Thus, it can be seen that a high percentage of subjects indicated an increase in EEG power at 3 Hz in the full-power mode trial (Table 3.3) and at 11 Hz and 12 Hz in the standby mode trial (Table 3.4). Furthermore, at 12 Hz the difference in sham and exposure

median spectral power was found to be statistically significant (p = 0.004; Frontal region; CI: -0.5,-0.1) as highlighted in Table 3.4.

Significant p-values in the full-power mode trial for a high percentage of subjects indicating a decrease in EEG power were also identified (highlighted in Table 3.3). They occurred at 9 Hz in the frontal region (p = 0.009, CI: 0.1, 0.6), in the central region at 7 Hz (p = 0.020, CI: 0.6, 5.0) and 9 Hz (p = 0.022, CI: 0.5, 4.9), and in the occipital region at 7 Hz (p = 0.032, CI: 0.5, 9.0), 8 Hz (p = 0.026, CI: 0.7, 8.7), 9 Hz (p = 0.017, CI: 0.9, 6.9), 17 Hz (p = 0.05, CI: 0.0, 5.3), 19 Hz (p = 0.05, CI: 0.0, 4.9), and 27 Hz (p = 0.034, CI: 0.1, 2.6). Of particular interest amongst these significant levels are 7 Hz (Theta rhythm), 8 Hz (alpha rhythm), and 9 Hz (alpha rhythm) as these frequencies are in succession and therefore are less likely to have occurred by statistical chance. Furthermore, these frequencies have relatively substantial 95% confidence intervals ranging minimally from 0.5 μV to at least 6.9 μV. It is also very interesting to note that EEG rhythms 7 Hz and 9 Hz indicate statistically significant values (for decrease in EEG power) in both the occipital and central recording sites. Moreover, statistically significant levels are evident at 9 Hz in all three recording sites.

3.6 Conclusions

This preliminary study suggests that a change in human brain wave activity occurred in the alpha (8 – 13 Hz) and beta (13 – 32 Hz) EEG bands during exposure to a GSM mobile phone configured to transmit at full-radiated power. A corresponding statistical analysis in

EEG bands during exposure to a GSM mobile phone in standby mode did not indicate any significant levels of change. Aside from this analysis in EEG bands, median differences between sham and exposure EEG data in each discrete EEG rhythm (from 1 - 32 Hz) were observed. Results of the full-power mode trial showed that in at least 75% of the 32 EEG rhythms analysed 70% – 90% of subjects indicated a decrease in EEG power. This again suggested that the full-radiated power might be responsible for alteration in the normal (sham exposure) EEG recordings. No predominant characteristics were apparent under standby mode conditions. A subsequent statistical analysis was conducted for the cases where a very high percentage of subjects indicated a resultant decrease or increase in EEG power. Results of particular interest were consecutive findings at 7 Hz (Theta rhythm), 8 Hz (Alpha rhythm), and 9 Hz (Alpha rhythm) that indicated statistically significant decrease in EEG power from the occipital region in the full-power mode trial. In addition, a decrease in EEG power at 7 Hz and 9 Hz were found to be statistically significant in both the occipital and central recording sites. Moreover, in the full-power mode trial, statistically significant levels were apparent at 9 Hz from all three recording sites (frontal, central, and occipital).

In conclusion, from the perspective that far greater statistical significance and notable characteristics were evident in the full-power mode trial than in the standby mode trial (almost negligible significance), it is speculated that the field characteristics of the full-power mode exposure may influence human brain activity. However, this is a pilot study

and should be considered as such. Aspects for further research (as addressed in Chapter 8) expand on this study by the implementation of a randomised and fully counterbalanced double-blind investigation to examine both 900 MHz and 1800 MHz pulsed exposures on the EEG as well as on the ECG and blood pressure. Notably, for the first time in an awake EEG study a significant sample size of 100 participants are tested considerably near to SAR levels of exposures close to the maximum allowable limit of 2 W/kg over any 10 g of tissue.

Chapter 4

Development of a Test Handset to Better Assess Bioeffects from Mobile Phone Exposures

4.1 Introduction

In regards to neurophysiological studies investigating mobile phone exposures on test participants, there are a number of critical factors that should be considered in the selection or design of the exposure source so as to avoid misleading results or anomalies arising in experimental test measurements. Amongst reported studies where biosignal test measures have been recorded while active mobile phone exposures are present, the type of RF source has ranged considerably. Exposure sources which have been previously adopted have included real mobile phones positioned at near and far field distances from the head [Krafczyk et al., 2000][Gehlen et al., 1997], different antenna arrangements including externally driven dipoles and monopoles [Huber et al., 2000][Papageorgiou et al., 2001] [Fritzer et al., 2007][Regel et al., 2007], while others

have implemented programmable handsets [Hamblin et al., 2006][Maby., 2006]. Exposure system positioning with respect to the participants head has also varied including methods where the exposure source has been active and near to the participants ear, behind the head, or from distances spanning from beneath a bed whilst participants are lying asleep or at rest [Mann et al., 1998] [Wagner et al., 1998][D'Costa et al. 2003]. However, while most reported studies have generally considered a seemingly stringent exposure source arrangement, confounding factors resulting from the EMF source or exposure position are not often accounted for, and thus has in many cases presented ambiguity as to whether the trialed exposure itself has been responsible for positive outcomes noted. Such experimental confounding factors due to the exposure source can include -

1. Heat generated from internal circuitry from mobile phones radiated next to the head which may cause temperature rises of up to 4.5 $^{\circ}$C to the ear [Gandhi et al., 2001], as well as heating due to the RF from handset antennas may also be accounted, but for a fraction of a degree [Hirata et al., 2003]. Considering that heating may cause significant temperature rise, increased SAR variation, as well as discomfort to the participant during lengthy experimental procedures, possibly undetectable artifacts could arise in physiological measures.
2. Pulsed low frequency fields from a battery or internal circuitry from a mobile phones such as at 2 Hz [Heath et al.., 1998], may cause confounding effects in

biosignal acquisition units measuring low frequency producing biological signals such as the EEG or ECG, consequently making it a perplexing task to distinguish the signals from real test measures.

3. Power and frequency variability in modulation modes of mobile phone transmission, such as mode switching to discontinuous transmission (DTX) may also invariably cause fluctuations in test measures and consequent inconsistencies in experimental outcomes unless appropriately accounted for [Tillmann et al., 2006].
4. Acoustic cues or data processing responses from an active mobile phone may also alter sensitive test measures by inducing acoustic artifacts in the biosignal responses [Niedermeyer & Da Silva, 1993][Fisch, 1999].
5. Distance variability of the RF exposure source from the head (although not associated with the exposure source operation itself) may be shown to considerably alter the SAR into the head even at small distances (~1 mm) [Schonborn et al., 1998]. Consequently uncertainties in interpretation of test measures may result if the exposure source is not monitored in a fixed position with respect to the test participants head.

In the pilot investigation, as described in Chapter 2, all five of these confounding factors were accounted for by - 1. Using controlled exposures excluding low frequency

interrupts (900 MHZ pulsed at 217 Hz; nominal average output: 250 mW), and 2. Placing the handset in a horizontal position behind the head so that internal circuitry and battery heating was considerably away from the head. However, for the major study (described in Chapter 7) it was intended to place the handset in an ear to mouth position, as would be the case in normal mobile phone use. In important consideration that in practical assessment standard protocols to determine SAR levels from behind the head do not exist, one main reason for intending to place the handset near the ear was so specific energy absorption rate (SAR) levels inside the head could be validly measured, assessed, and reported. However, to accomplish keeping the handset near the ear as well as to avoid all noted confounding factors above, a handset other than the programmable mobile phone as used in the pilot study needed to be selected. Due to this necessity, it was decided to design and construct a suitable experimental handset.

The following chapter aims to outline the design and development, construction phase, and SAR testing of a modeled mobile phone handset for use in the major investigation (Chapter 7). The design of the experimental handset was mainly inspired by mobile phone models as described and implemented by Reiser et al. [Reiser et al., 1995] and Mochizuki et al. [Mochizuki et al., 2002]. In addition, subsequent sections of this chapter will describe the computational analysis undertaken to validate practical SAR measures as well as to estimate the electric field distribution surrounding a head. The important design of handset's holder is also described.

4.2 The Design and Construction of Two Test Model Handsets

Two different dummy mobile phone handsets were initially designed for experimental purposes including an aluminium body model (as similar to Mochizuki et al. 2004 and Rowley, 1999) and a perspex model (similar to Reiser et al. 1995). Both handsets adopted the common design configurations of a rectangular block body with an internal coaxial feed through its length to accommodate for an external RF signal feed at its base and interchangeable antennas at its top. Four antennas were designed for the units including two ¼ λ monopole antennas and two helical antennas that would accommodate both 900 MHz and 1800 MHz signals. The antennas of the designed models were uniquely made by stripping a short length semi-rigid coaxial cable with an SMA connection end. The SMA connector end provided an ideal RF feed point and a secure mounting base to connect to the handset's coaxial feed presumably with a good match which would later be measured. Insulation or heat-wrap was used to cover the antennas on both sets of antenna types made. The antennas for both handset models were centered at the half-width point of the top left side of the rectangular bodies as shown in Figure 4.1 below. Accurate centering of the antennas between the top widths of the handsets were ensured so that the handset could be optionally used on both sides of a participants head considering that even minimal variations in the distance from the heads of test participants can cause a considerable variation in SAR [Schönborn et al.,

1998]. Design implications to accomplish this included having to securely and precisely embed the inner coaxial feed cable lengths to the correct depth within each rectangular block. A thin (~3 mm) aluminium and perspex lid was constructed to conceal the embedded coaxial feeds in both the aluminium and perspex handsets.

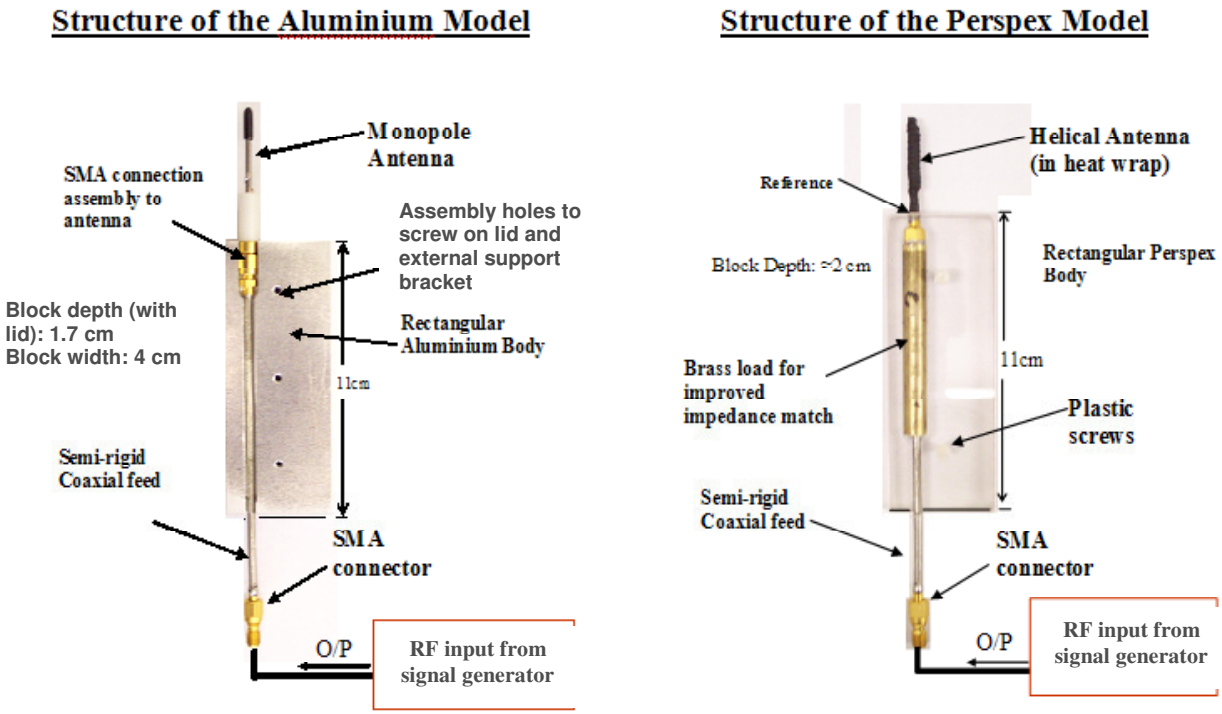


Figure 4.1 Shown above are the structural details and dimensions of the constructed Aluminium and Perspex handset models designed.

Important design considerations when imbedding the coaxial feed included: 1. Provision for allowances in appropriate cutaways in the handsets main body to prevent ease of interchange of the antenna type for use during experimental trials; 2. Allowances for the SMA antenna mounting connection assembly to sit flush at the top surface of the handset, so as to maximize the effective ground plane of the aluminium handset body and minimize non-linearaties; and 3. Provision for a brass load for the perspex body handset, which was uniquely adjoined at the semi-rigid coaxial feed point on the antenna's SMA mounting base, hence providing adequate ground plane conditions for the near lossless body. This cylindrical brass load can be observed within the transparent designed perspex handset model shown in Figure 4.1 above. Following construction of the handsets the antenna input impedance was assessed. Measurements of the impedance at the antenna feed point were determined by measurement of the RF return loss of the handsets at both operating frequencies with the corresponding monopole or helical antenna in place. Using a network analyzer (HP 8753ES), the complex impedance of the handset with the aluminium body was measured at approximately 40 – 60j (Input power: ~170 mW). However, using the same protocol, the impedance of the perspex handset was shown to provide an almost idealistic match at ~50 Ω. Although the perspex model had indicated a more ideal electrical match it was ultimately decided that the aluminium body should be used later for experiments. This decision was mainly made upon the basis that the aluminium

body was more comparable to a real handset given that the body of the phone is metalised and forms part of the radiating source [Kivekäs and Ollikainen, 2004]. Additionally, it would be easier for other investigators to reproduce this model if necessary.

The following sections aim to describe the SAR compliance measurements and RF modeling undertaken to further evaluate the RF exposure response of the selected aluminium test handset.

4.3 Compliance Measurement of the Aluminium Model Handset

In order for the handset to be officially compliant for use in experiments with test participants the handset was required to be tested for its rated Specific Absorption Rate (SAR) at both 900 MHz and 1800 MHz. This would also later aid in obtaining the appropriate Ethics approval for its use in human experiments. As identified in Chapter 1, the ARPANSA RPS3 standard [ARPANSA, 2002] specifies a maximum allowable general public limit for RF exposure to the head and torso at 2 W/kg over any 10 gram of tissue. To ensure the handset's radiated output produced a SAR below this limit, several official SAR evaluations of the experimental handset were undertaken at EMC Technologies Pty Ltd in Australia. In addition to ensuring compliance, it was of

interest to also tune the handset to as close to 2 W/kg as possible to obtain test conditions close to a *worst case scenario*. In order to tune the handset three SAR evaluations for both 900 MHz and 1800 MHz operating frequencies were conducted. SAR measures were acquired by use of a standard automated robotic arm facility used to probe a liquid tissue-equivalent filled phantom head or Specific Anthropomorphic Mannequin (SAM) with the aluminium active test handset in position below it in accordance with applicable IEEE 1528 Standard recommendations for SAR measurements [IEEE 1528, 2003]. Figure 4.2 below indicates the SAR output for one of the "touch left" positions as evaluated beneath the left side of the SAM head. The handset was tuned close to the maximum allowable limit by proportionally increasing the handset's input power levels via an RF signal generator following each evaluation, for each test frequency. The SAR robotic probing system, which incorporated an E-field probe (ET3DV6), was automated using Dosimetric Assessment System (DASY4) software.

As the SAR measurements require a very meticulous setup, a total of approximately 10 hrs was required to tune the handset to produce adequate SAR levels. Time and expense was a restriction on how well the handset may have been tuned to desired maximum levels, however the final measurements at tuned input powers of the handset provided suitable readings of the SAR of up to 70%-75% of the maximum allowable limit. However, considering that SAR measurement uncertainty levels can be quite

significant (± 26.4 %) [IEEE 1528, 2003], the test handset results overall demonstrated very good approximated levels for use to test worst case conditions with test participants. The resultant SAR levels for both 900 MHz and 1800 MHz CW exposures were as follows – 1. At 900 MHz CW exposure, the peak spatial-average SAR was **1.56 W/kg** over any 10 gram cube at an input power of **276 mW;** At 1800 MHz CW exposure, the peak spatial-average SAR was **1.4 W/kg** over any 10 gram cube at a handset input power of **163 mW**.

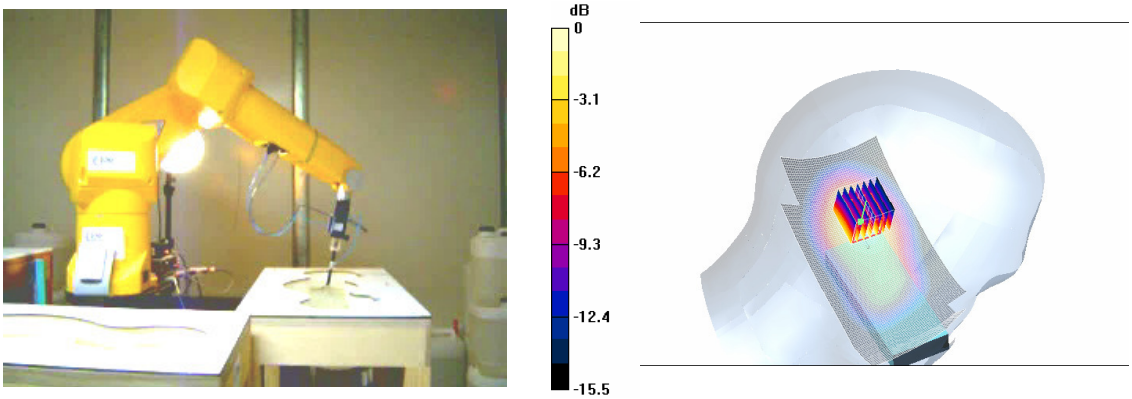


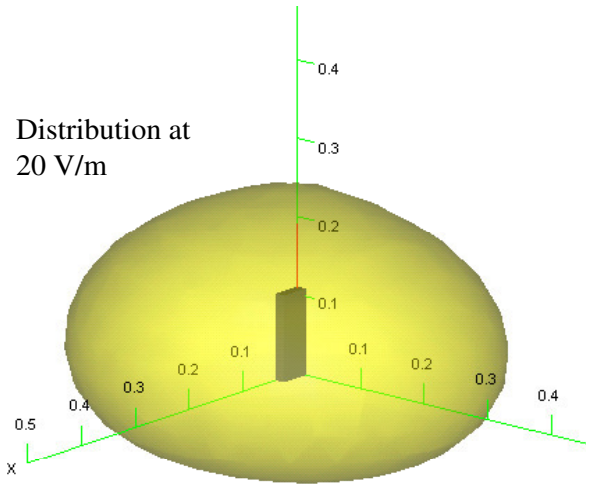
Figure 4.2 Shown above (left) is the SAR robot test facility as used to probe a SAM phantom head model where the radiating test handset was positioned below. The liquid dielectric properties for average head at 900 MHz were $\sigma = 0.97$ S/m $\varepsilon'_r = 41.5$; and at 1800 MHz were $\sigma = 1.38$ S/m, $\varepsilon'_r = 40$. An example of the corresponding 'DASY 4' software output response of the evaluation is also shown (right) indicating the SAR 10 gram cube and spatial-peak location for the 900 MHz touch 'left' position.

4.4 Computational Modeling of the Test Handset and Validation of SAR Measurements

To evaluate and characterise the RF response of the test handset at the predetermined input power levels, computational modeling of the handset's RF field distribution was undertaken. A 3D electromagnetic modeling software package FEKO V.5.2 (Method of Moments (MoM) based – see Meyer et al., 2001), was used to create a dimensionally correct model of the handset in free space to be simulated for both 900 MHz and 1800 MHz continuous wave (CW) test exposures. Results of these simulations are provided below in Figure 4.3 indicating the electric field response with corresponding gain plot at both operating frequencies. In accordance with the practical SAR measurements for compliance of the test handset which indicated 1.56 W/kg at 900 MHz and 1.4 W/kg at 1800 MHz (Section 4.3), the electric field distribution at 900 MHz may also be similarly shown to be approximately 15 - 25% greater than the electric field estimate for the 1800 MHz exposure, at the same distance in the far-field region (as indicated approximately 20 – 30 cm from the reference antenna and handset body).

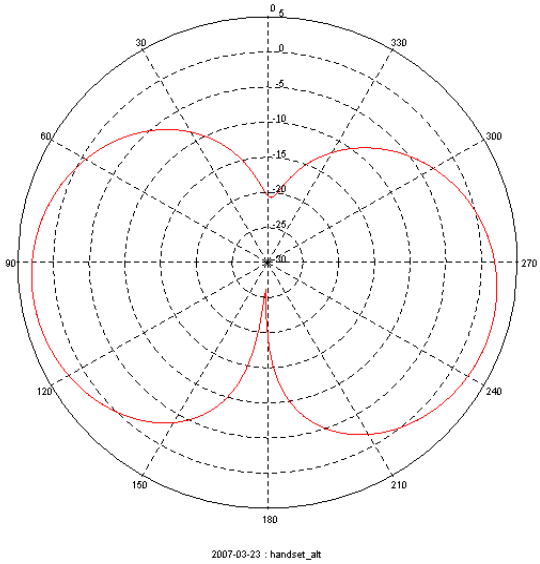
Handset E-Field plot at 900 MHz CW

Input power: 276 mW * Axis labels in metres (m)



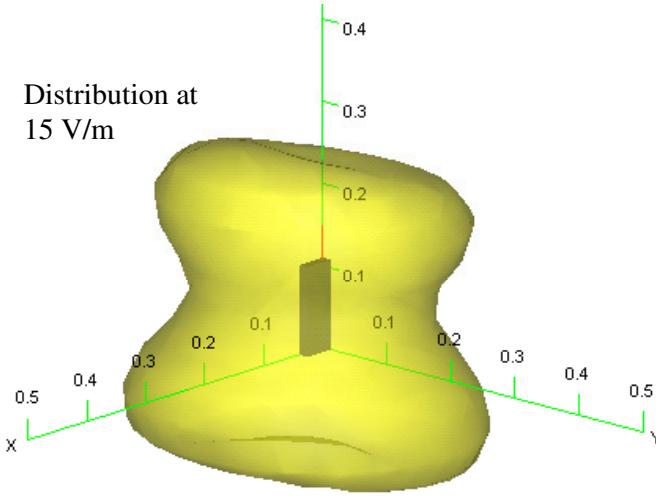
Gain (dB) at 900 MHz

VRP Gain total



Handset E-Field plot at 1800 MHz CW

Input power: 163 mW * Axis labels in metres (m)



Gain (dB) at 1800 MHz

VRP Gain total

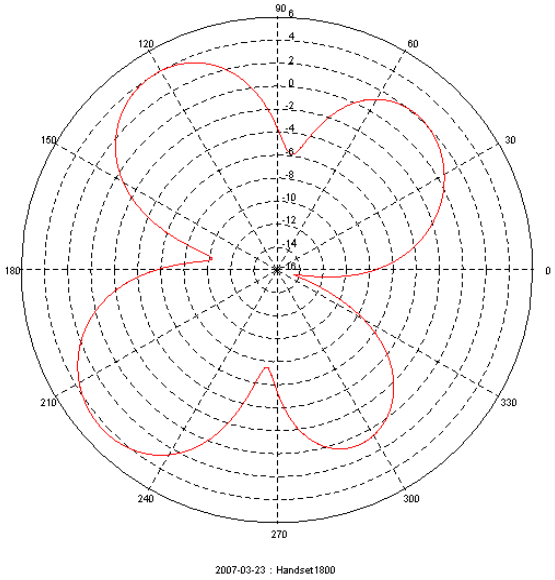


Figure 4.3 Shown are the computational results of the electric field response and corresponding gain plot for the modelled experimental handset operating at both 900 MHz and 1800 MHz CW frequencies.

Given in Section 4.3, that in standard practice SAR uncertainty measurement estimates can be quite large, it was also of interest to determine computational estimates SAR levels of the model handset. Specifically, it was of interest to indeed ensure that SAR levels would be near the maximum allowable limit for experimental purposes. Moreover, given that the handset would later be mounted near to the heads of many test participants and that reports using different antenna models have been shown to indicate significant changes in SAR at small (millimeter) varied distances [Schönborn et al., 1998][Bahr et al., 2006][Beard and Kainz, 2006] it was also necessary to quantify the effects of varying the modeled handset at different angles from the head.

To determine the computational outputs of SAR under the described conditions, a FEKO homogeneous head model[1] as based on dimensions of the IEEE SAM phantom [IEEE 1528, 2003] was incorporated with the geometric model of the experimental handset as shown in Figure 4.4 below. Three positions of the handset with respect to the head were arranged. As indicated in the first diagram (Figure 4.4 - far left), the handset was modeled in the ear-mouth position near the ear (1 mm away) as an estimate of the practical SAR assessment of the handset as described in Section 4.3. In the second arrangement (Figure 4.4 – middle) the handset was oriented in a tilt position with the antenna rotated outward 15 degrees away from the head (about its vertical plane), specifically with the top edge of the handset near to the mid-point of the pinna.

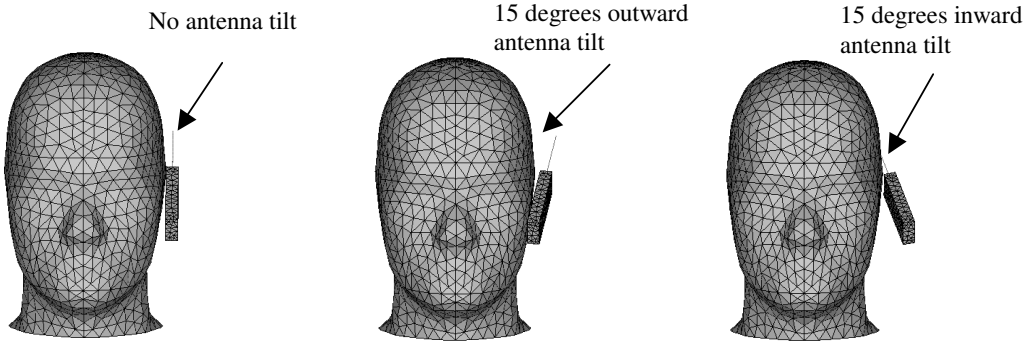
[1] FEKO head model - provided under licenced membership (see www.feko.co.za)

In the third arrangement (Figure 4.4 – right) the antenna was tilted inward 15 degrees toward the head. For this condition the handset was required to be moved ~ 2 mm away from the head so that the lower body of the handset would not touch the head model.

In summary the simulation protocol was to determine the peak spatial-average SAR over any 10 gram of tissue at 900 MHz (276 mW) and 1800 MHz (163 mW) for the following three test conditions:

1. No antenna tilt
2. 15 degrees outward antenna tilt
3. 15 degrees inward antenna tilt

The results of the peak spatial-average SAR level for each condition as well as the corresponding spatial-peak location are indicated below in Figures 4.4 and 4.5, respectively.



No antenna tilt	Antenna at 15 degrees outward tilt	Antenna at 15 degrees inward tilt
SAR over 10 g (W/kg)	**SAR over 10 g (W/kg)**	**SAR over 10 g (W/kg)**
At 900 MHz – 1.525 At 1800 MHz – 1.959	At 900 MHz – 1.33 At 1800 MHz – 0.761	At 900 MHz – 1.415 At 1800 MHz – 2.386

Figure 4.4 Shown are the computational SAR results for varying the handset (15°) with respect to the head. Peak spatial-average SAR levels are as indicated inside the 'FEKO' model head, at generated handset exposures of 900 MHz and 1800 MHz. Dielectric constants for head were specified at 900 MHz: $\sigma = 0.97$ S/m and $\varepsilon'_r = 41.5$; and at 1800 MHz: $\sigma = 1.38$ S/m, $\varepsilon'_r = 40$ [IEEE Std 1528, 2003].

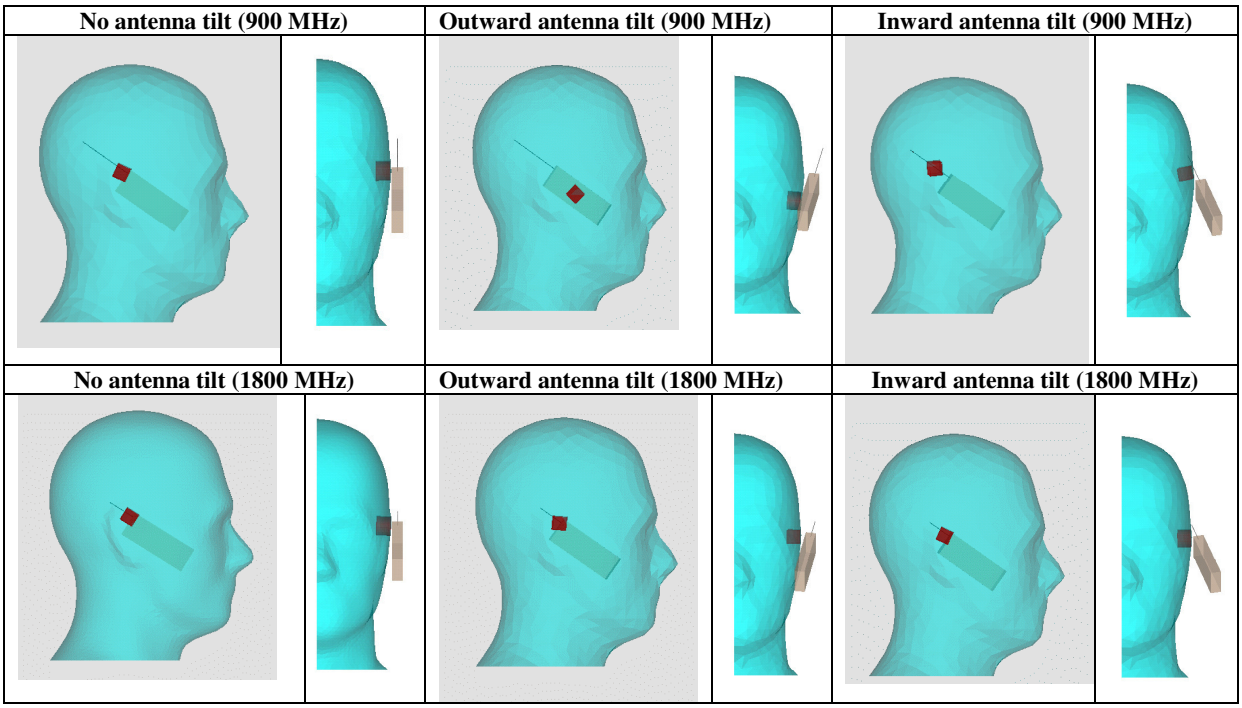


Figure 4.5 Shown are the computational results for the location of the peak spatial-average SAR as estimated over 10 g of tissue (red cube), when tilting the modelled handset (15°) with respect to the head. Exposures from the handset were generated at both 900 MHz and 1800 MHz. Dielectric constants for head were specified at 900 MHz: $\sigma = 0.97$ S/m and $\varepsilon'_r = 41.5$; and at 1800 MHz: $\sigma = 1.38$ S/m, $\varepsilon'_r = 40$ [IEEE Std 1528, 2003].

By observation of Figure 4.4, it may be noted that the outcomes for the 'no antenna tilt' condition indicated SAR outputs of 1.525 W/kg and 1.959 W/kg for the 900 MHz and 1800 MHz test exposures respectively. In comparison, the practical measurements of SAR (Section 4.3) that most closely correspond to the no tilt condition, indicated peak spatial-average SAR levels of 1.56 W/kg at 900 MHz and 1.4 W/kg at 1800 MHz. As may be noted at 900 MHz there is a marginal difference in the SAR level (2.3%) however at 1800 MHz the difference is more considerable (28.5%). Three factors which may account for these discrepancies include, 1. There is a shell thickness

associated with the SAM phantom head (2 mm), which is absent in the computational FEKO model, hence increasing the difference; and 2. The computational head model has a lossy exterior ear (~2 mm thick) with dielectric properties as nominated for head, while the SAM phantom head has an approximate lossless ear which withholds the handset approximately 4 mm from liquid properties inside the shell, and 3. Formal uncertainty as discussed in the standard practical test measurements can be considerable (±26.6 %). Despite these three differences between the practical and computational head models, the corresponding measures of SAR with the test handset may still be said to show fair agreement with one another, particularly as it may be said that all four values of SAR ranged between 1.4 W/kg and 1.96 W/kg. This issue shall nevertheless be readdressed in more detail in Chapter 6 where a novel head model is demonstrated which better clarifies this difference. However, for the purpose of this Chapter, it is perhaps more important to note that SAR outcomes for the test handset placed in a standard ear-mouth position is unlikely to exceed the maximum allowable limit of 2 W/kg however will likely fall near it for both exposures as required.

Apart from a comparison of standard positioning, by observation of the effects between the 15° inward and outward tilt conditions of the test handset as shown in Figure 4.4, it can be seen that the SAR levels may vary more considerably at 1800 MHz than at 900 MHz. At 900 MHz, in comparison to the 'no antenna tilt' condition as described above, the results for the 'outward antenna tilt' and the 'inward antenna tilt' indicated

only marginal differences, specifically a 13 % decrease and 7 % increase in SAR respectively. However as noted, the same results as indicated for the 1800 MHz were considerably much larger indicating a 61.2 % decrease for the outward antenna tilt and an 18 % increase in SAR level for the inward tilt condition. Nonetheless, the smaller changes in SAR levels for the 900 MHz exposures, unlike at 1800 MHz, can be seen to notably influence the location of the 10 gram cube as indicated in Figure 4.5. In particular, at 900 MHz the location of the SAR 10 gram cube inside the head is shown to relocate to the projected mid-section of the handset's body for the antenna outward tilt, and in contrast, towards the projected mid-section of the antenna for the antenna inward tilt condition. The corresponding 1800 MHz exposure results however can only be observed to indicate negligible changes between the handset tilt conditions in terms of the location of the peak spatial-average cube. Notably, the actual 'orientation' of the cube itself can be noted to be the result of the specific placement (by the FEKO simulator) of the cube's faces parallel to the closest dielectric boundary surface inside the head model.

4.5 The Handset Support Apparatus

The computational results as indicated in Figures 4.4 and 4.5 provide a theoretical prediction of the considerable variability in SAR that may occur when handset position variations are shifted near the head. From these simulated findings it was affirmed that a rigid support for the test handset was critical for use in experimental trials with participants. Consequently, a rigid but flexible bracket apparatus as indicated below in Figure 4.6 was designed and improved to meet this criteria. The figure indicates the characteristics of the handset bracket assembly as demonstrated next to the head.

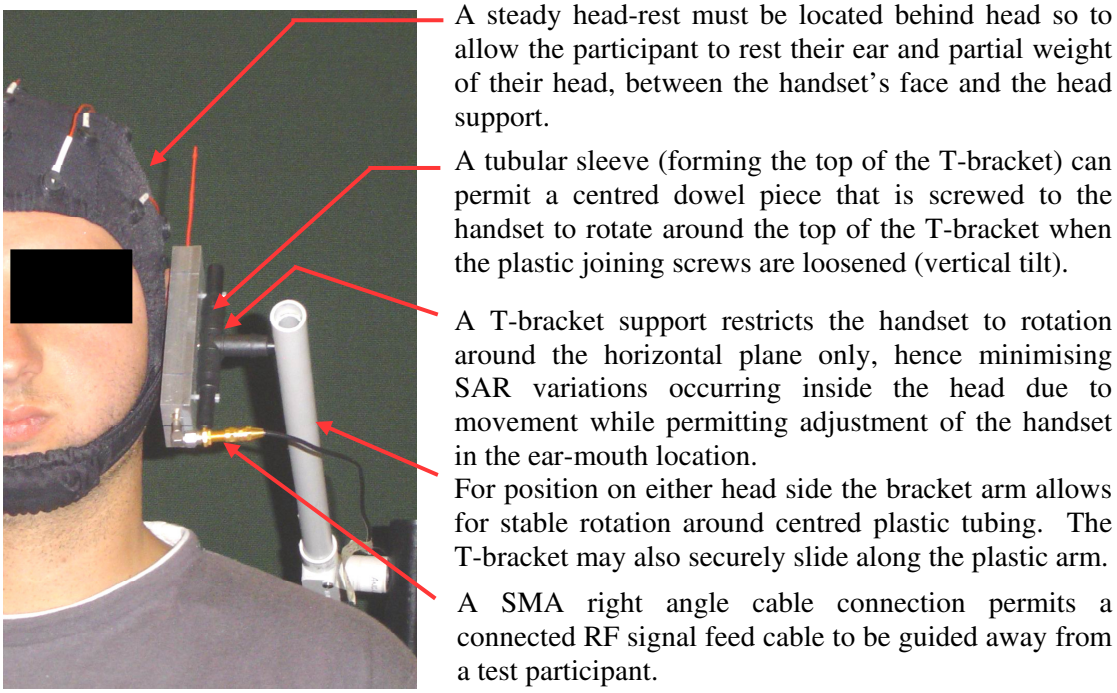
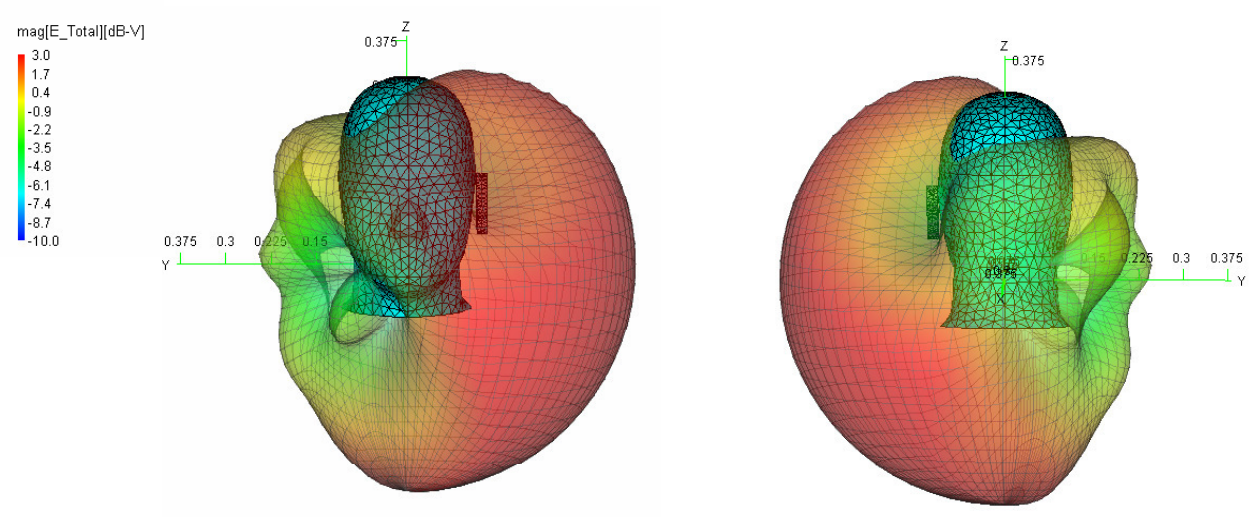


Figure 4.6 Shown is the handset support apparatus as designed to secure the handset in a fixed position during experimental trials in order to minimise SAR variations occurring inside the test participants head.

4.6 Modeling of the Electric Field Distribution Surrounding the Head

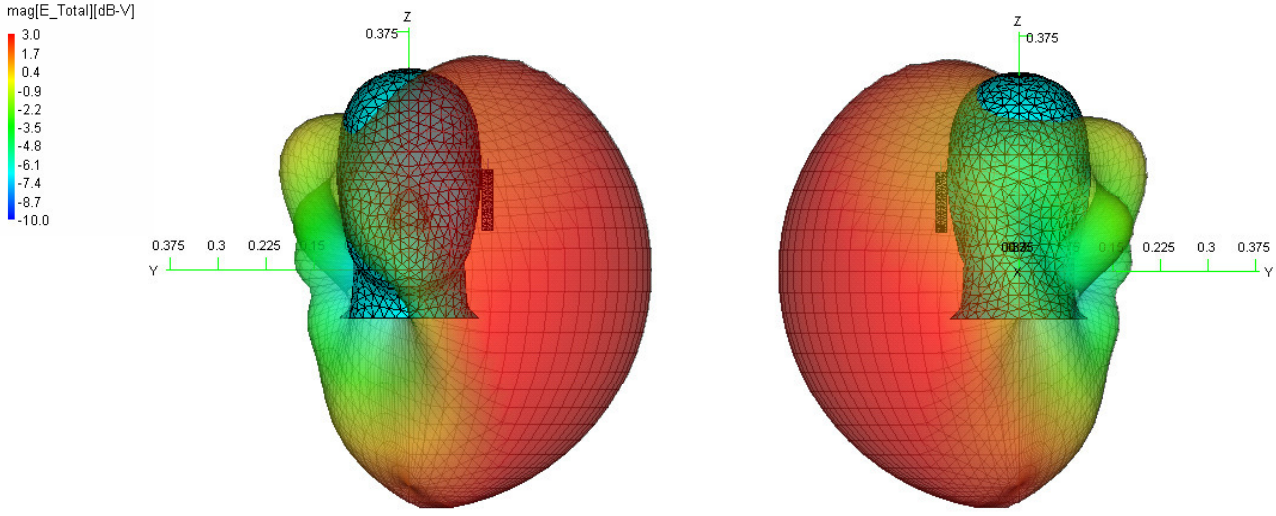
A subsequent computational assessment of the model handset was also undertaken to determine the electric field distribution surrounding the head as results could potentially later be correlated to outcomes of EEG findings at particular recording regions of the brain. The interesting results of these final simulations to assess the test handset are presented below in Figure 4.7 for both handset operating frequencies of 900 MHz and 1800 MHz at the predetermined applicable input power levels of 276 mW and 163 mW. Notably, this analysis shall be extended in Chapter 6 where modeled EEG electrode leads are positioned on a novel computational head to represent a real experimental condition as pictured in Figure 4.6 above.

3D E-field distribution at 900 MHz – Front and back view



Frequency: 900 MHz
Input power: 276 mW
Tissue dielectric constants for head: $\sigma = 0.97$ S/m; $\varepsilon'_{r} = 41.5$

3D E-field distribution at 1800 MHz – Front and back view



Frequency: 1800 MHz
Input power: 163 mW
Tissue dielectric constants for head: $\sigma = 1.38$ S/m; $\varepsilon'_{r} = 40$

Figure 4.7 Shown is the electric field distribution surrounding the head (as indicated in dB-V) which was of interest to determine as results could potentially aid later in correlating outcomes of EEG findings at particular recording regions of the brain to visible field strengths outside.

4.7 Discussion and Conclusion

The results as provided in this Chapter, are represented by the outcomes of the development stages undertaken to novelly design, construct, and develop a test handset to a controlled and usable level for use in future experimental work. As outlined in Introduction, the main advantage of using a test handset rather than a real mobile handset, is that it eliminates important experimental confounding factors where investigation of RF effects on neurophysiological responses is of concern. Furthermore, as the test handset is externally driven by a RF signal generator it is identified as having a foremost advantage of being able to test different RF protocols such as pulsed 900 MHz or 1800 MHz exposures on the one unit. Indeed at both 900 MHz and 1800 MHz CW exposures, the handset was described to be novelly tuned to meet close to the maximum allowable SAR limit so that a worst-case scenario may be investigated in the EEG experimental procedures and results of the major study (outlined in Chapter 8). Additionally, a computational assessment of the handset was undertaken to reasonably ensure that SAR outcomes of the practical compliance measurements, that are known to have a considerable uncertainty level of ±26.6%, were presumably closer to the maximum allowable limit. This assessment indicated fair agreement with the practical measures with neither assessment exceeding the 2 W/kg limit or falling below 1.4 W/kg. In further addition to these tests, the angular tilt of the handset with respect to the head was also assessed in terms of SAR level and peak

spatial-average location. Outcomes of this computational assessment most clearly and importantly indicated that the experimental handset's RF energy distribution inside the head would be considerable if moved only slightly during trials. Specifically, for tilts up to 15 degrees, a 60% decrease change in SAR was found to occur at 1800 MHz, while at 900 MHz the peak spatial-average SAR, although not changing considerably (maximum 12.8 % decrease), could be noted to significantly shift from the upper antenna projected region to the corresponding lower handset region inside the head. These findings suggested that a robust unit would need to be designed to support the test handset if the tilting variable was to be controlled. To facilitate this need, in consideration that double blind conditions would need to later be administered, a rigid and customized apparatus to enable the handset in a fixed position, was successfully designed and constructed.

Chapter 5

Effects of EEG Electrode Leads on the Specific Absorption Rate of Radiofrequency Exposures from Mobile Phones

5.1 Introduction

In assessment of studies adopting the EEG to investigate the effects of mobile phone exposures on human brain function, there has been questions raised as to whether the EEG electrode recording leads in the vicinity of the exposure source may affect the level of SAR induced inside the heads of participants. An assessment of induced RF pickup into the EEG recording leads as described in the pilot study (Chapter 3), provided some evidence that there was no statistically significant differences between sham and exposure trials when leads were attached to an adults thigh at rest (‘*flat-line’* recordings) and connected to the active EEG acquisition unit (Mindset 1000). However, on the other hand these trials

could not assess the potential for RF 'shielding' due to the electrode leads in terms of the amount energy that may be restricted into the head. If there is high RF shielding effects due to electrode leads are current EEG studies measuring the effects of SAR levels which are not comparable to mobile phone usage?

In terms of RF induction from mobile phone exposures into the EEG recordings leads alone, the literature suggests there may be only negligible associated effects [Hietanen et al., 2000][D'Costa et al. 2003][Wood et.al, 2003]. However, although directly comparable, quite a recent report by Angelone et al. [Angelone et al., 2004] suggested that much lower frequency electromagnetic fields as produced by MRI scans (128 MHz, 300 MHz), may indeed induce pickup into EEG recording leads and rather significantly increase SAR levels inside the head.

To address these concerns, this chapter describes a study aimed at investigating whether SAR levels due to 900 MHz and 1800 MHz MP exposures are affected by standard arrays of EEG recording leads as arranged in accordance with the EEG 10/20 and 10/10 International Systems on the left side of a Specific Anthromorphic Mannequin (SAM) phantom.

5.2 Materials and Recording Methods

A DASY-4 robotic SAR measurement facility* was used to measure the SAR distribution of mobile phone exposures inside a SAM phantom head model fitted with a standard 10/20 EEG electrode cap (Quik-Cap). The EEG cap in total consisted of 19 tin electrodes with outer-side accompanying leads of approximately 1 m in length. However, due to the right side of the SAM phantom head requiring clearance for SAR probing only the middle and left-half of the cap's electrodes and leads were positioned. This arrangement was secured to the head phantom with durable tape about the peripheral lining of the cap. Stray lead lengths which fed towards the back of the cap were taped along and away from the spine of the phantom. To ensure a realistic setup, EEG electrode gel was also inserted beneath each of the positioned electrodes.

The mobile phone exposures were generated from the experimental mobile phone model as described in Chapter 3. The model handset was placed against the phantom head's left ear and was oriented in accordance with the 'touch' position as outlined in the SAR measurement protocols specified by the IEEE [IEEE P1528, 2003]. However, this placement was restricted by the ~2 mm lining on the EEG cap which narrowly prevented the handset from touching the cheek between the required ear to mouth alignment. A

* SAR measurements were conducted using professional facilities at EMC Technologies Pty. Ltd., Australia

photograph of the phone model's setup position with respect to the SAM phantom and EEG electrode cap is shown below in Figure 5.1.

In separate tests, both 900 MHz and 1800 MHz CW exposures were generated inside the head phantom using the model phone. RF signals were fed from an Agilent function generator via coaxial cable to an amplifier and then directly to the handset's coaxial feed point as shown in Figure 5.1. The power supplied to the feed point of the handset was 276 mW at 900 MHz and 163 mW at 1800 MHz as previously determined to produce comparable mobile phone peak spatial SARs near to 2 W/Kg over any 10 g of tissue. An adjustable perspex mount allowed for the positioning of the handset assembly against the phantom head (Figure 5.1).

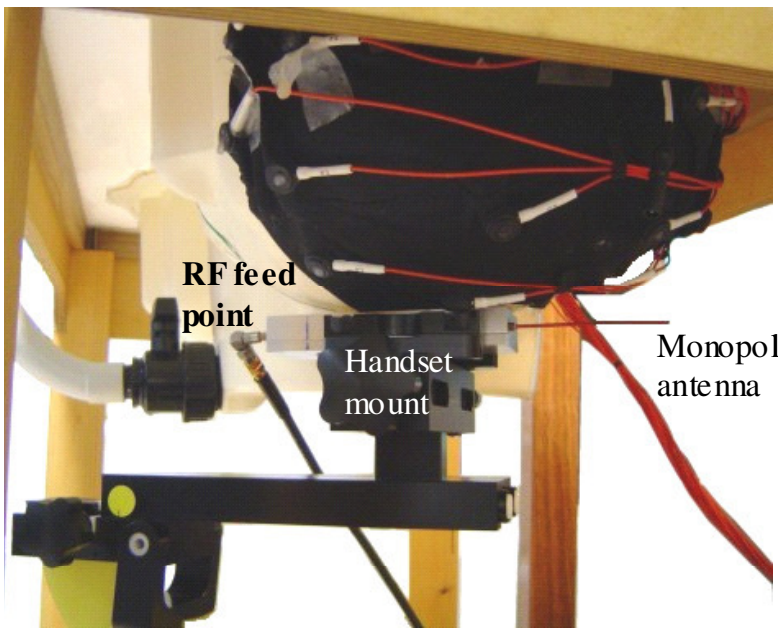


Figure 5.1. Shown is the left underside of the SAM phantom as positioned with the EEG electrode cap and handset arrangement.

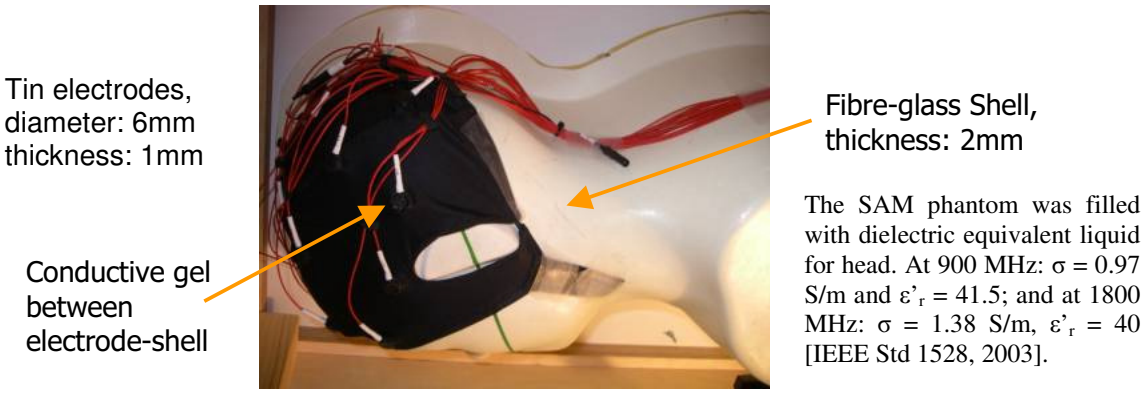
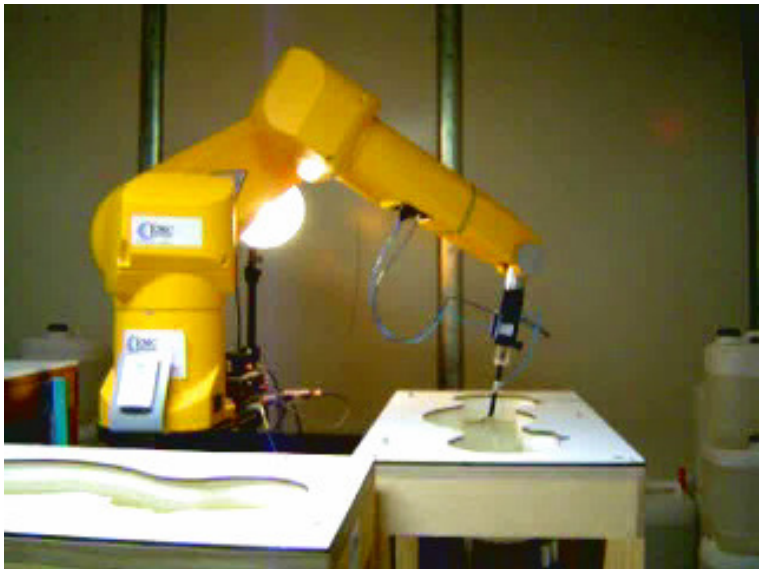


Figure 5.2. Shown above is an additional view of the SAM phantom head as clearly fitted with the left half of a 19-electrode cap.

Before SAR measurements could be recorded the phantom head model was filled with a brain tissue equivalent mixture according to standard mobile SAR testing guidelines as specified by the Australian Communications (and Media) Authority [ACA Std, 2003]. At 900 MHz the relative permittivity and conductivity of the liquid mixture was 41.5 and 0.97 S/m respectively. Similarly, at 1800 MHz the permittivity of the solution was 40 and the conductivity was 1.38 S/m [Gabriel et al., 1996]. The same arrangement was used to determine the input power levels to the model handset.

To acquire SAR measurements an automated robotic arm was used to probe the liquid from the right side of the head phantom. This automation was configured and controlled via DASY-4 software. Each SAR test required the handset's peak SAR to be determined by scanning the handset's projected surface area about the inner left-side of the head. A

scan within a 30 mm cubic volume (7x7x7 points) was then conducted around the handset's peak SAR to produce estimates of the spatial peak SAR averaged over any 10 gram of tissue. Approximately 20 minutes was required to complete each SAR test.



The first SAR test recording was acquired with the EEG cap in place and the handset active at 900 MHz. After this recording the model handset was lowered vertically on its adjustable mount and the EEG cap and remaining traces of EEG gel were carefully removed from the phantom head. The handset was then raised again on its vertical mount to the prior touch position and SAR measurements were repeated with the 900 MHz

exposure present. After this test, the electrode cap was arranged back into position and the 900 MHz antenna that was fixed on the model handset was replaced with a shorter monopole to accommodate the 1800 MHz exposure. SAR measurements with the 1800 MHz exposure were then taken with and without the EEG cap present in accordance with the preceding trial.

In addition to these trials, the SAR levels of the programmable Nokia 6110 mobile phone as used in the pilot study (Chapter 2) (GSM 900 exposures) was also assessed, though this time in the presence of two different EEG cap types with much denser lead populations. These caps both consisted of 62 tin electrodes arranged in similar arrays as comparable to the 10/10 International System however one EEG cap had externally located electrode leads (Quik-Cap) and the other incorporated the leads beneath the cap (Electro-Cap).

The Nokia 6110 exposure source was set to transmit at 895 MHz, with a mean output power of 250 mW (2 W peak). Apart from the exposure source only, the cap arrangement and SAR testing procedures were identical to that of the trials conducted with the model handset.

Interestingly, during the trials using the Nokia handset, it was found that the spatial peak average SAR levels were located near the phone's body, rather than near the phone's

antenna. Consequently, to obtain SAR measurements in closer proximity to the electrode leads, subsequent tests were required to record a programmed volume SAR scan around the projected antenna region of the phone inside the SAM phantom.

The SAR measurement facility used in all trials was fully calibrated within current month of testing.

5.3 Results

The results of the SAR test measurements of the active mobile phone exposures as recorded with and without the EEG caps present, are summarised below in Table 5.1.

Exposure Type	Status of Cap on Phantom	Cap Type	Peak 10 g SAR in Phantom (W/Kg)	Difference between SAR with and without Cap
Model Handset Tx at 900 MHz	Present	19-electrode Quik-Cap	1.56	0 %
Model Handset Tx at 900 MHz	Removed	--------	1.56	--------
Model Handset Tx at 1800 MHz	Present	19-electrode Quik-Cap	1.33	5 % decrease
Model Handset Tx at 1800 MHz	Removed	--------	1.4	--------
GSM Nokia 6110 Tx at 895 MHz	Removed	--------	0.674	--------
GSM Nokia 6110 Tx at 895 MHz	Present	62-electrode Electro-Cap	0.574	14.8 % decrease
GSM Nokia 6110 Tx at 895 MHz	Present	62-electrode Quik-Cap	0.552	18.1 % decrease
GSM Nokia 6110 Tx at 895 MHz	Removed	--------	0.110†	--------
GSM Nokia 6110 Tx at 895 MHz	Present	62-electrode Electro-Cap	0.095†	13.6 % decrease
GSM Nokia 6110 Tx at 895 MHz	Present	62-electrode Electro-Cap	0.090†	18.2 % decrease

Table 5.1 Results of the SAR measurements taken with and without the EEG caps present.
† SAR measurement taken at a volume scan located in the antenna region of the GSM mobile phone.

5.4 Discussion & Conclusions

Several observations can be made from the results of this study shown in Table 5.1. For tests conducted with the model handset at 900 MHz it can be seen that no change in the measured SAR was evident when the 19-electrode cap was present on the SAM phantom. A small a decrease (5 %) in the recorded SAR was observed with the model handset operating at 1800 MHz under the same test conditions.

Also indicating decreases in the SAR when the EEG cap was present were the results for the first set of test recordings taken with the GSM exposure. These results are shown to indicate greater changes of up to 14.8 % for the tests conducted with the 62-electrode 'Electro-Cap', and an 18.1 % decrease in SAR with the presence of the 62-electrode 'Quik-Cap'. Demonstrating similar results to these was the second set of averaged SAR test recordings taken with the GSM exposure at a volume scan positioned in the antenna region of the phone. In these results, respectively a 13.6 % and an 18.2 % decrease in the measured SAR was observed when the same 'Electro-cap' and 'Quik-Cap' was positioned.

In this type of work, measurement uncertainties may be quite significant (± 26.4 %)[8] and hence while this study suggests that there may be effects in the averaged SAR from mobile

phone exposures due to the presence of EEG recording leads, these effects are relatively minimal. With the exception of the model handset operating at 900 MHz where there was no change, these effects are observed as decreases in the spatial peak SAR in all other trials (Table 5.1). In addition, it is particularly interesting to note that these reductions are predominately higher in the presence of the 62-electrode EEG caps with much denser lead populations. It is suggested that this effect may be due to increased RF shielding from the additional positioned leads in the vicinity of exposure. Nevertheless, this presumption is not in agreement with Angelone et. al. [Angelone et. al., 2004] who suggest potentially unsafe increases in SAR may be produced inside the heads of participants undergoing MRI RF exposures with positioned EEG electrode leads.

In conclusion, this investigation indicates that the measured SAR from mobile phone exposures may be influenced in the presence of EEG recording leads, but these changes are not substantial enough to cause significant perturbation of mobile phone SAR levels inside the heads of test participants.

Chapter 6

A Novel Computational Head Model for Dynamic Assessment of RF Exposure Shielding and Coupling Effects due to EEG Leads

6.1 Introduction

In Chapter 4 the effects on mobile phone exposure SAR levels due to EEG leads positioned on a participant's head was assessed using a SAM phantom. This investigation indicated that there may be up to a 5 % decrease in SAR levels for 900 MHz continuous wave (CW) exposures using the experimental handset (leads in a 10/20 EEG system arrangement) and up to an 18 % decrease for denser lead populations using the GSM 900 programmable handset (leads in a 10/10 system arrangement) [D'Costa et al., 2004]. It was thus confirmed to rather use the 10/20 International System cap to overcome larger SAR variations when considering investigation of the EEG during mobile phone exposures in future experiments. However, in recognition that a 5 % decrease in SAR levels may also pose to represent a redistribution of the electric field inside the head, it became necessary to understand whether the RF field distribution is significantly changed due the leads present.

In Chapter 3 (development of the test handset), computational validation techniques of practical SAR measurements were undertaken using a commercial homogeneous head model (FEKO model – MoM solver). However, in consideration to modify this model to incorporate electrodes onto the head surface it was found that geometrical limitations restricted the design and simulation. In consequence to overcome, a novel computational head model based on standard dimensions of the head was designed which could assess the handset's field distribution inside the head as well as model positioned electrodes and leads closely in accordance to a 10/20 system array.

Only two recent related studies can be found which have investigated the RF exposure electric field distribution and SAR level changes inside the head with respect to EEG recording leads [Schmid et al., 2007][Hamblin et al., 2007]. Together, these studies are notably useful in indicating SAR differences with respect to angular changes of lead orientation, though are both limited in the dynamic assessment of the electric field response inside the head and the specific causes due to an experimentally comparable set of positioned leads. In the investigation by Schmid et al., outcomes of FDTD analysis of 900 MHz and 1970 MHz exposures assessed over a 15 EEG electrode 10/20 array (using an anatomical correct head model) indicated a notable decrease at 900 MHz in the 1g-averaged spatial peak in the brain cortex (46%) and a significant increase in the same measure at 1970 MHz (22%). These changes were notably indicated when the electrode leads were oriented parallel to the incident electric field vector, however when leads were oriented horizontally, the uncertainty level was considerably reduced to ±5%. Similarly Hamblin et al., in an experiment defining three parallel leads and electrodes on the flat surface of a modified sphere (FDTD homogeneous model defined with dielectric

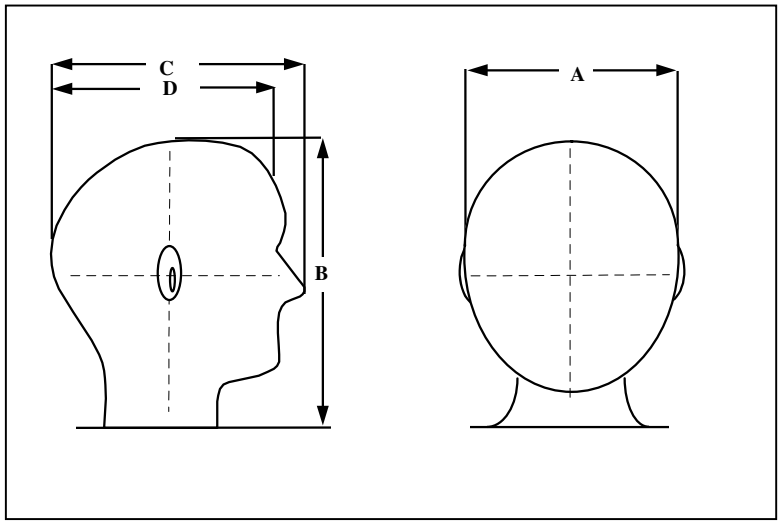
constants for head) found that the peak 10 g averaged SAR was reduced by 38% when the leads were parallel to a 900 MHz dipole orientation and considerably reduced to almost a 0% difference when leads were perpendicular.

The following Chapter enables an extended analysis of previous studies through a flexible design of a novel human head model (using CADFEKO) with an array of 14 electrodes and leads arranged in accordance to the EEG 10/20 system. The head model and lead arrangement was designed to be comparable to the real experimental conditions that would be administered in the major investigation as will be described in Chapter 8. Specific outcomes of this study are demonstrated over measures of SAR output, novel indication of the peak spatial-average location, and the RF distribution inside the head, as due to both 900 MHz and 1800 MHz MP exposures.

6.2 Computational Modeling of a Human Head with a Complete Array of Leads

A computational head model was designed with a total array of fourteen modelled EEG recording electrodes and leads divided on both hemispheres. The modelled head was designed within the 'CADFEKO' module of electromagnetic modelling software 'FEKO' V.5.2. Selected head dimensions of the design were based upon the recommended dimensions of a human head phantom, as indicated in the ACA Radiocommunications (Electromagnetic Radiation – Human Exposure) Standard 2003 [ACA, 2003]. Recommendations published within this standard indicate the physical dimensions of two suggested phantom head models for practical SAR measures, which were developed and provided respectively to the standard by Motorola as well as Neils Kuster and Schmid &

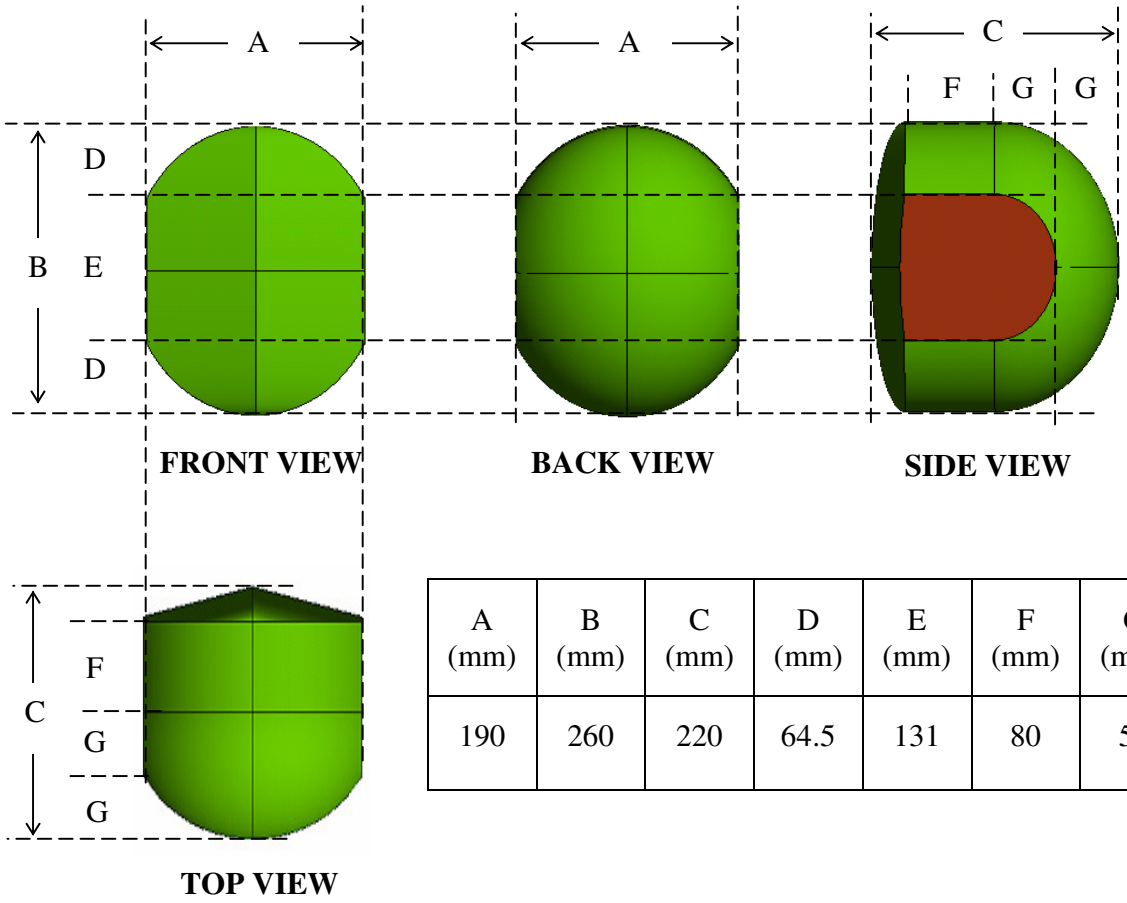
Partner Engineering. An extract from this standard is provided below which indicates the basic dimensions for the two described head specifications (Figure 6.1).



	Model 1 (millimetres)	Model 2 (millimetres)
A	190	190
B	260	240
C	229	230
D	190	210

Figure 6.1 Shown are two specifications for geometric parameters of a human head phantom as published in the ACA Radiocommunications Standard, 2003. The parameters indicated for 'Model 1' were developed by Motorola while the dimensions for 'Model 2' were developed by Kuster, N. and Schmid & Partner Engineering.

As noted, the dimensions of the computational head model as indicated for this study were based upon the geometric parameters as indicated in Figure 6.1. The geometric design of the computational head model (without EEG electrode leads) is indicated below in Figure 6.2.

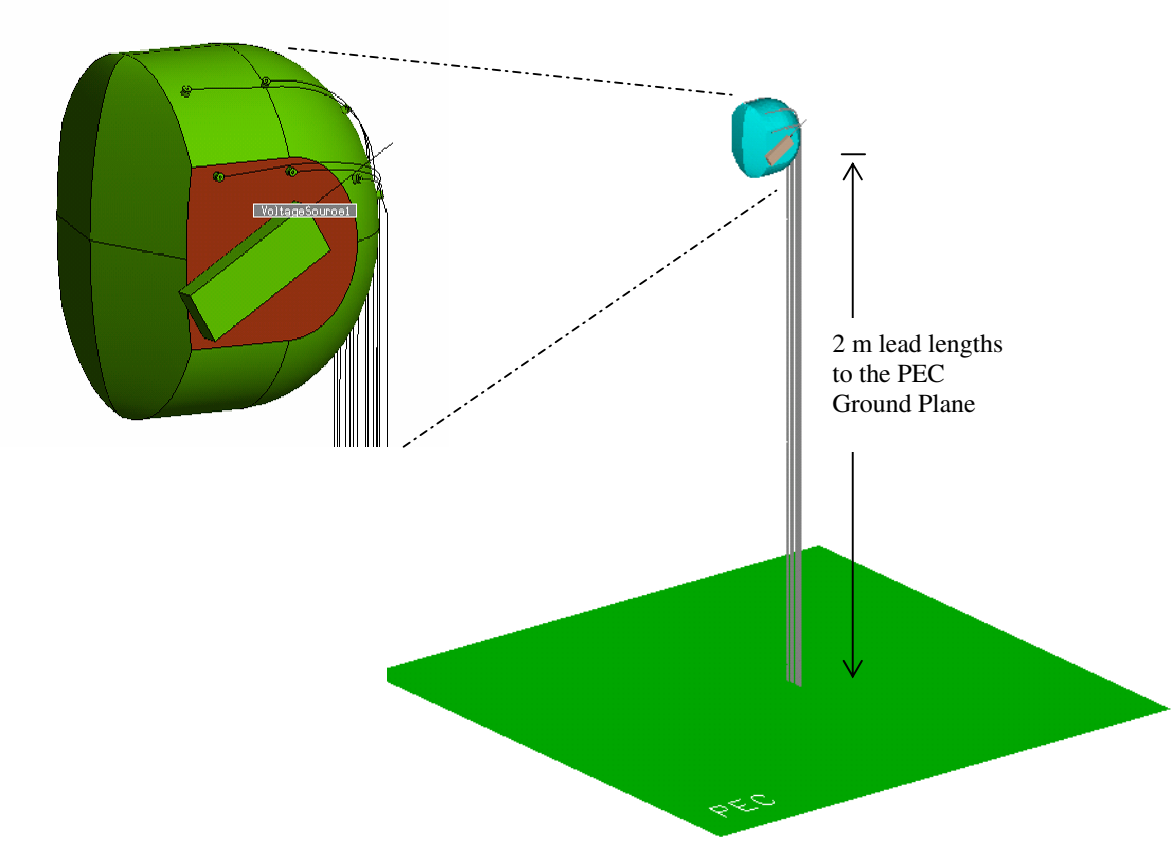


A (mm)	B (mm)	C (mm)	D (mm)	E (mm)	F (mm)	G (mm)
190	260	220	64.5	131	80	55

Figure 6.2 Outlined above are the geometric parameters of the designed head model as based on model designs for a phantom head as recommended by Radiocommunications (Electromagnetic Radiation – Human Exposure) Standard [ACA, 2003].

To define the geometric measures of the EEG electrodes and leads to be incorporated with the modelled head, physical measures of the electrodes and leads of the 10/20 system electrode-cap to be used in the major investigation were acquired. In addition, the mobile phone test handset as described in Chapter 4 was re-modelled near to the head in an 'ear to mouth position' along the vertical plane. The total geometry of the designed computational head model with the electrode leads and positioned handset is illustrated below in Figure 6.3. The geometric model may also be observed to be connected to a Perfect Electrically Conducting infinite plane or 'PEC' ground plane via longer modelled lead lengths. This ground plane model was also incorporated for some of the test conditions in order to simulate a worst case scenario for RF coupling into the electrode leads. This condition was in accordance with real experimental conditions where leads are fed into a differential amplifier that are in turn realised, following signal processing channels, at a ground point within the amplifier.

All modeled electrode lead dimensions are also provided in Figure 6.3 below in addition to the illustration of the geometric model.



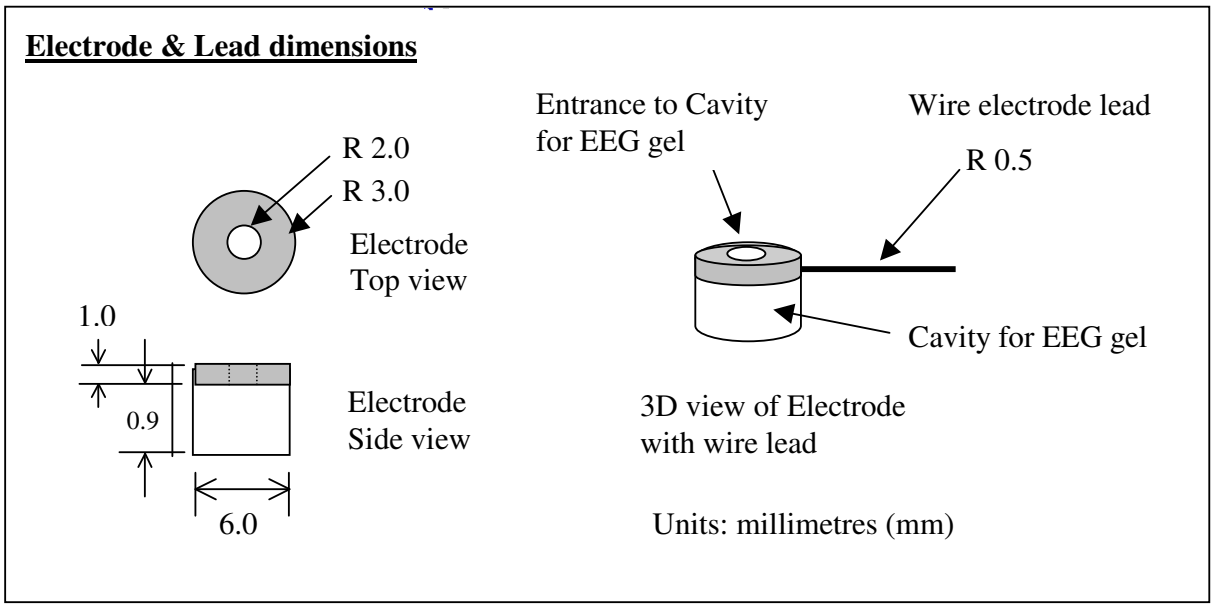


Figure 6.3 Shown is the total geometry of the designed computational head model with electrode leads and positioned handset. The geometric model may also be observed to be connected to a Perfect Electrically Conducting infinite plane or 'PEC' ground plane via modelled electrode leads which would later be used for part of the test simulations to represent a worst case scenario for RF coupling into the lead array.

6.3 Dielectric Properties of the Head Model

Notably, the modelled head was also novelly incorporated with the flexibility to include or omit an outer shell as equivalent to that used on specific anthromorphic mannequins (SAM) [IEEE 1528 Standard, 2003]. To nominate the material quantities of the computational head model, standard dielectric properties for the phantom shell, tissue, electrodes, and electrode gel were defined in FEKO. Figure 6.4 below indicates a mid cross-sectional view of the head model at a position where the most central positioned electrodes are halved to demonstrate the dielectric properties of the model.

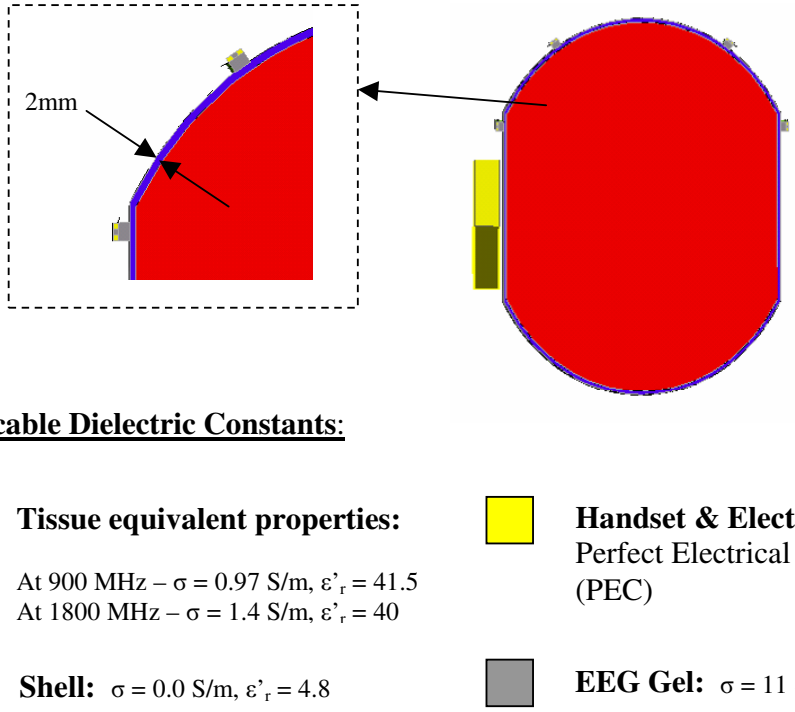


Figure 6.4 Shown is the mid cross-sectional view of the head model indicating the dielectric properties of the designed head model, including the EEG electrode gel, exterior shell, and tissue equivalent. The shell (blue) representing the shell properties of a real SAM phantom, were substituted for the dielectric quantities of tissue for approximately half of the total test simulations. Outlined in red are the dielectric properties for tissue as specified in accordance to the IEEE 1528 Standard. * EEG gel properties were provided by Telstra Research Laboratories.

Importantly, for approximately half of the test simulations conducted with the head model (as will later be described) the exterior shell of the modelled head, was substituted for the properties of tissue equivalent dielectric properties as specified by the IEEE 1528 Standard, 2003. This was undertaken to quantify any differences that could exist between the electrodes positioned on the tissue, and 2 mm away from the tissue as comparable to the practical trials described in Chapter 5 where a real SAM phantom head model with the same shell thickness was utilised. The slight increase in head size due to substituting in tissue for shell, was assumed to have a negligible effect on test outcomes.

6.4 Simulation of the Computational Head Model

6.4.1 Validation of the Head Model

To replicate real experimental conditions to be conducted test simulations of the handset with the computational head model were run in FEKO V.5.2, once again at the actual handset's tuned operating frequencies of 900 MHz (I/P: 276 mW), and at 1800 MHz (I/P: 163 mW). Although final test simulations of the designed head model were ultimately run with the modelled handset 3 mm from the head, initial development simulations undertaken to determine the selected distance, were considered at both 3 and 4 mm. The test distances were allocated to account for the absence of the near lossless outer ear or pinna, which would otherwise appear on an official phantom head to ensure a defined spacing between the handset and head [IEEE 1528 Standard, 2003]. A table of results indicating SAR outcomes of these initial tests (without leads present) alongside findings from previous real SAM phantom tests and the 'FEKO head' model tests (as described in Chapter 4), is summarised in Table 6.1 below.

Handset operating frequency	**Practical SAR measurements using a real SAM phantom head (W/kg)**	**Simulated SAR measurements using the FEKO homogeneous head model (W/kg)**	**Simulated SAR measurements using the designed homogeneous head model (W/kg)**
900 MHz *with shell*	1.56 (~4mm at the truncated pinna)	N/A	1.28 (3 mm) 1.27 (4mm)
1800 MHz *with shell*	1.40 (~4mm at the truncated pinna)	N/A	1.74 (3 mm) 1.54 (4 mm)
900 MHz *shell removed*	N/A	1.53 (~3 mm)	1.6 (3 mm) 1.34 (4 mm)
1800 MHz *shell removed*	N/A	1.96 (~3 mm)	2.24 (3 mm) 1.96 (4 mm)

Table 6.1 Shown are the SAR results of the initial test simulations conducted to validate the designed human head model (no leads attached) against real SAR tests and outcomes of the 'FEKO head' simulations as described in Chapter 4. As neither the commercially produced computational head model nor the originally produced model may allow the handset to touch the head, the simulated handset distances (mm) from the head are provided next to corresponding results

By observation of Table 6.1 it may be noted that the designed human head model in this study indicates excellent agreement with both the commercially designed head model (FEKO head) and the standard practical SAR measurements, with the maximum discrepancy shown to be ~18.5 % (shell present at 900 MHz). Importantly recalling that uncertainty levels in practical test measurements can indeed be quite considerable (± 26.4 %) [IEEE 1528, 2003] the maximum difference was still considered to be reasonable. Notably, direct comparisons of difference in SAR levels between the designed head

model and the commercially available head model did not exceed 12.5% for either exposure tested.

6.4.2 Simulation of the Head Model with EEG Electrodes and Recording Leads

In consideration of all results in Table 6.1, it was decided ultimately to keep the handset at 3 mm from the head during simulation with the EEG electrode lead array on the developed model. This decision was most significantly based on the intent to test the worst case condition. At the 3 mm distance the designed head model was run at both 900 MHz and 1800 MHz to determine the volume average SAR as well as the peak spatial-average SAR over a 10 gram cube. The following six points indicate the test conditions simulated:

1. The head model with the outer shell present and electrode leads grounded
2. The head model with the outer shell present and electrode leads not grounded
3. The head model with the outer shell not present (i.e. tissue only) and electrode leads grounded
4. The head model with the outer shell not present (i.e. tissue only) and electrode leads not grounded
5. The head model with the outer shell present and with electrode leads removed
6. The head model with the outer shell not present (i.e. tissue only) and with electrode leads removed

The following seven tables provided below indicate the results of the above described simulated conditions for both 900 MHz and 1800 MHz simulated exposures.

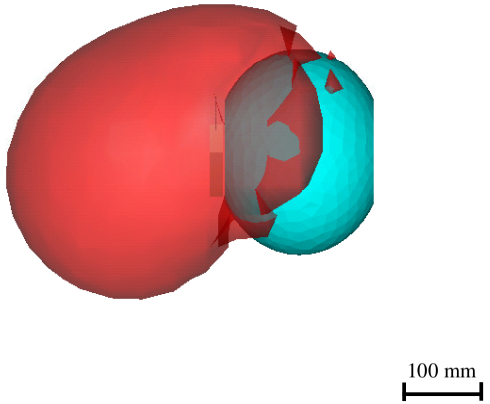
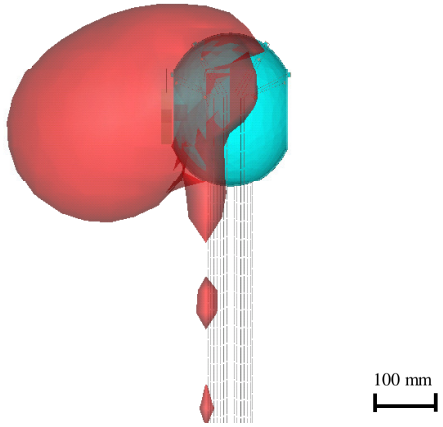
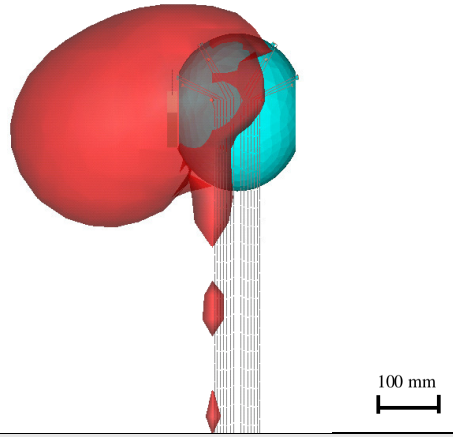
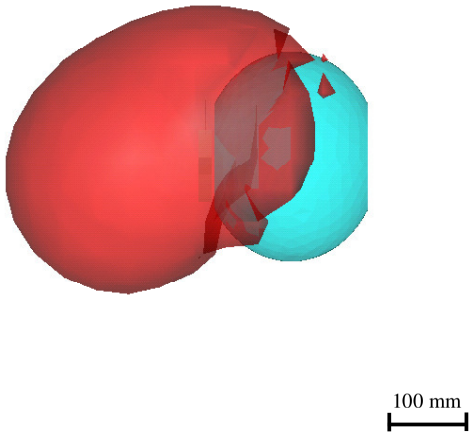
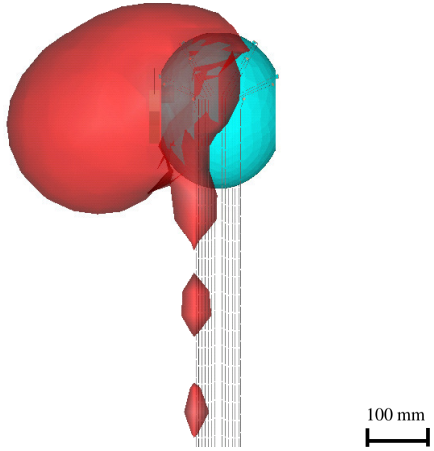
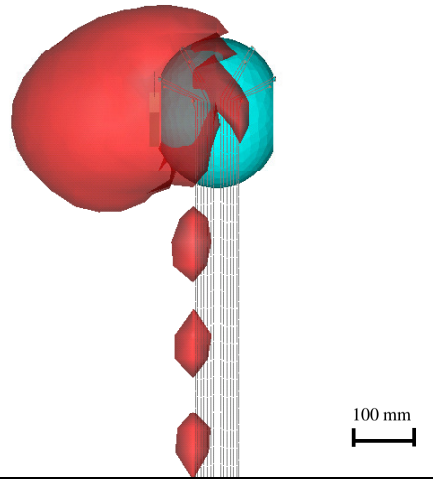
f = 900 MHz; No electrode leads; shell present	f = 900 MHz; leads not grounded; shell present	f = 900 MHz; leads grounded; shell present
ISOVIEW: 11 V/m (rear head view) 	ISOVIEW: 11 V/m (rear head view) 	ISOVIEW: 11 V/m (rear head view) 
f = 900 MHz; No electrode leads; head only	f = 900 MHz; leads not grounded; head only	f = 900 MHz; leads grounded; head only
ISOVIEW: 11 V/m (rear head view) 	ISOVIEW: 11 V/m (rear head view) 	ISOVIEW: 11 V/m (rear head view) 

Table 6.2 Shown are the electric field distributions at 900 MHz as produced by the handset (on left-side) around the modeled head as indicated with and without electrode leads present. Isoviews at 11 V/m were selected as this field strength most clearly indicates where corresponding RF coupling and potential reradiation may be generated on the electrode leads

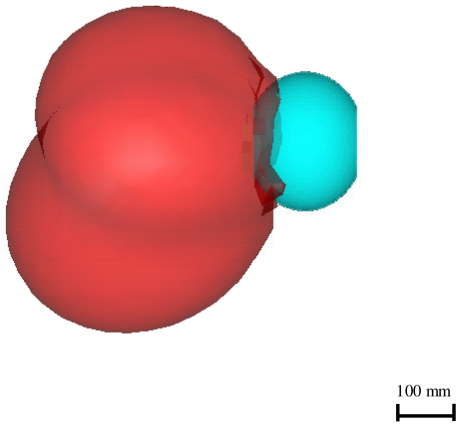
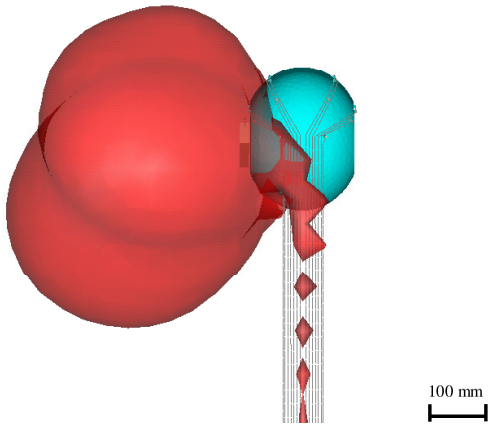
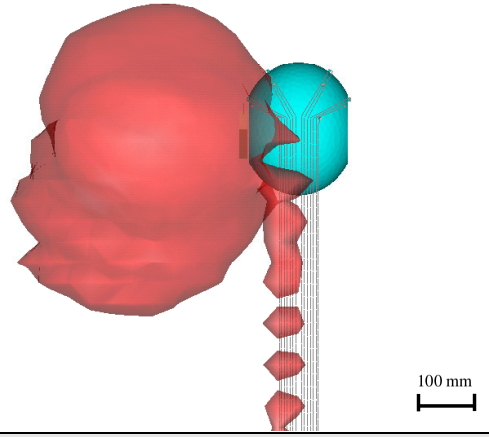
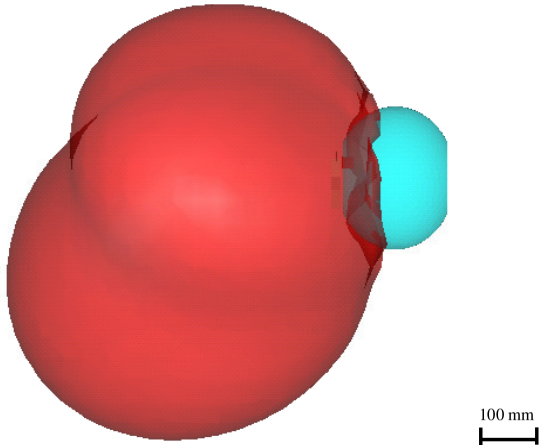
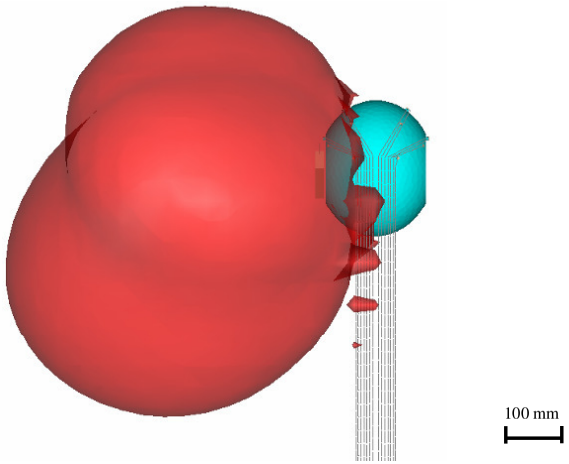
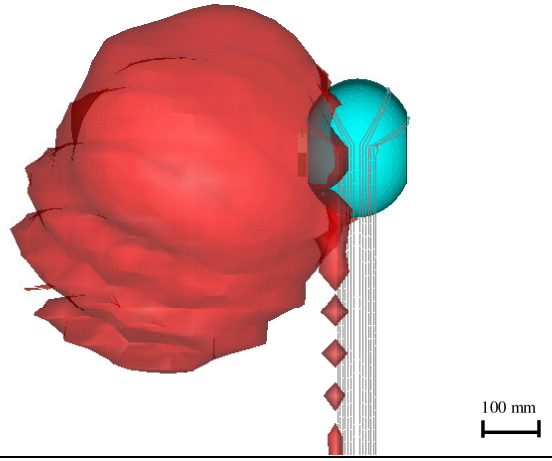
f =1800 MHz; No electrode leads; shell present	f =1800 MHz; leads not grounded; shell present	f =1800 MHz; leads grounded; shell present
ISOVIEW: 6.5 V/m (rear head view) 	ISOVIEW: 6.5 V/m (rear head view) 	ISOVIEW: 6.5 V/m (rear head view) 
f =1800 MHz; No electrode leads; head only	f =1800 MHz; leads not grounded; head only	f =1800 MHz; leads grounded; head only
ISOVIEW: 4.5 V/m (rear head view) 	ISOVIEW: 4.5 V/m (rear head view) 	ISOVIEW: 4.5 V/m (rear head view) 

Table 6.3 Shown are the electric field distributions at 1800 MHz as produced by the handset (on left-side) around the modeled head as indicated with and without electrode leads present. Isoviews at 11 V/m were selected as this field strength most clearly indicates where corresponding RF coupling and potential reradiation may be generated on the electrode leads.

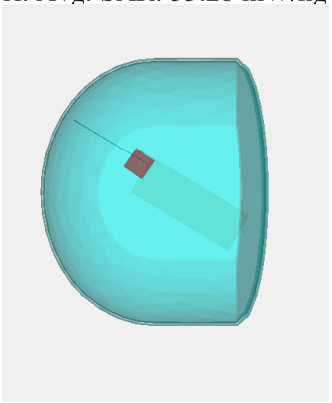
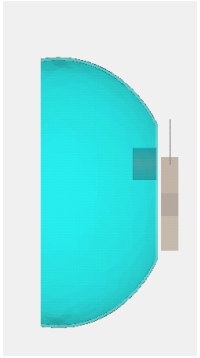
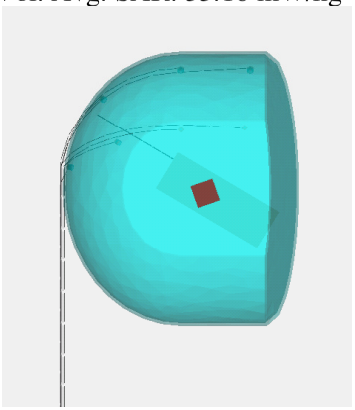
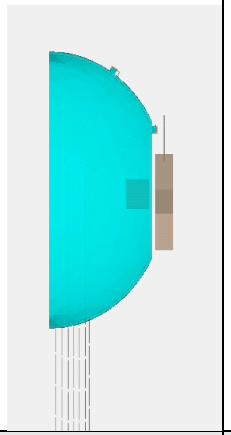
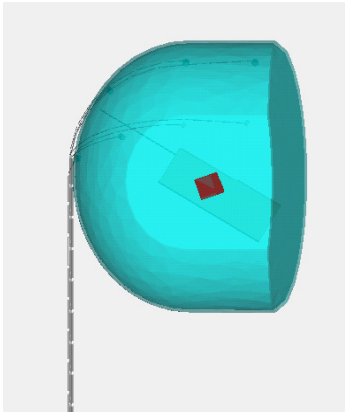
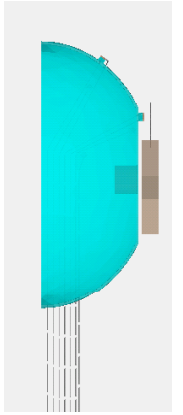
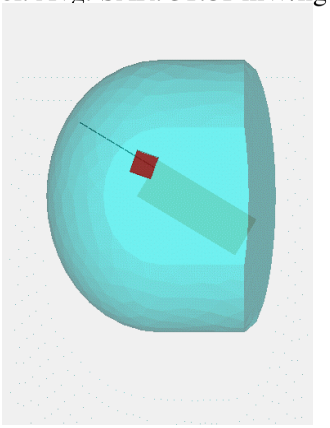
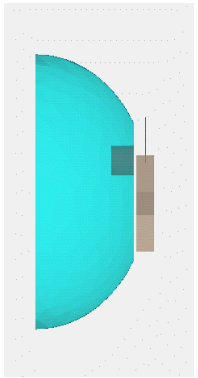
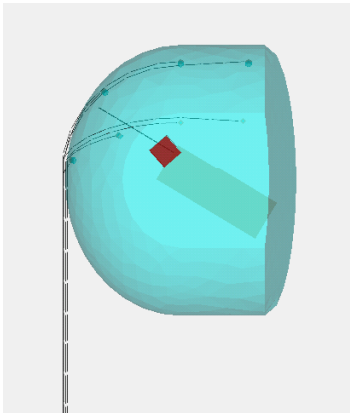
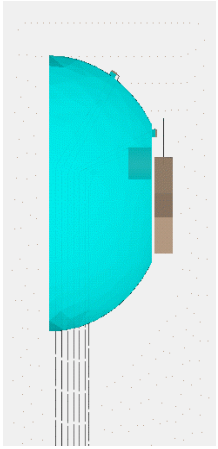
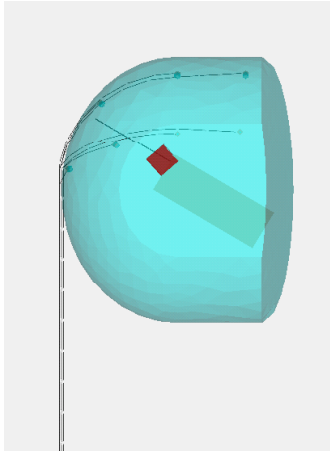
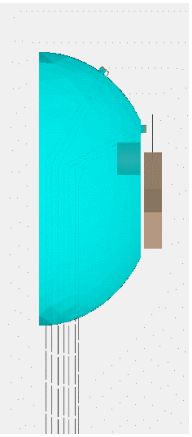
f = 900 MHz; No electrode leads; shell present		f = 900 MHz; leads not grounded; shell present		f = 900 MHz; leads grounded; shell present	
SAR over 10 g: 1.28 W/kg Vol. Avg. SAR: 33.28 mW/kg	**Handset: left-side**	SAR over 10 g: 1.231 W/kg Vol. Avg. SAR: 33.16 mW/kg	**Handset: left-side**	SAR over 10 g: 1.231 W/kg Vol. Avg SAR: 33.16 mW/kg	**Handset: left-side**
					
f = 900 MHz; No electrode leads; head only		**f = 900 MHz; leads not grounded; head only**		**f = 900 MHz; leads grounded; head only**	
SAR over 10 g: 1.6 W/kg Vol. Avg. SAR: 31.81 mW/kg	**Handset: left-side**	SAR over 10 g: 1.621 W/kg Vol. Avg. SAR: 31.72 mW/kg	**Handset: left-side**	SAR over 10 g: 1.6 W/kg Vol. Avg SAR: 31.77 mW/kg	**Handset: left-side**
					

Table 6.4 Shown are the 900 MHz results of the six described simulations conducted to determine the peak spatial-average SAR levels inside the head for conditions with and with and without electrode leads present. Shown in red is the position of the peak spatial-average 10 gram cube inside the head with respect to the handset position on the far left-side.

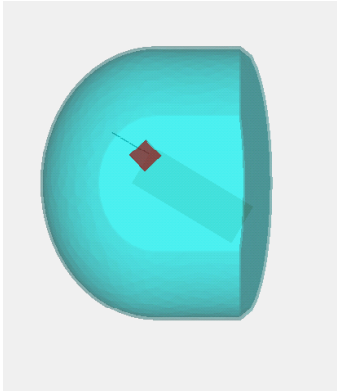
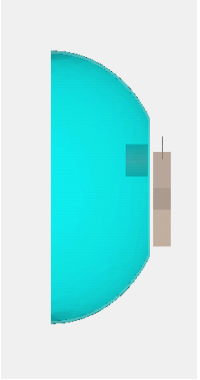
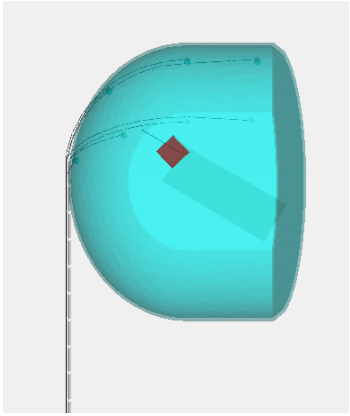
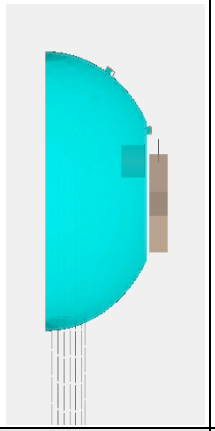
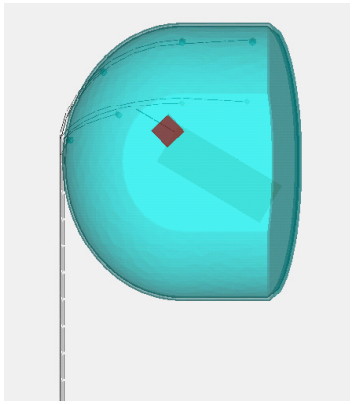
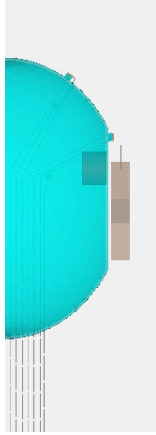
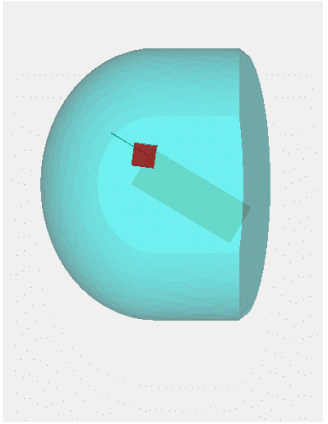
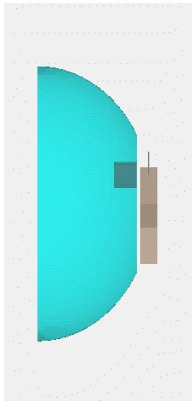
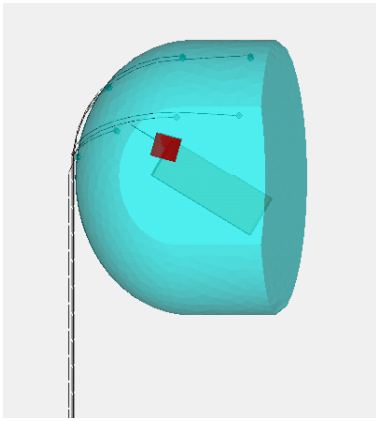
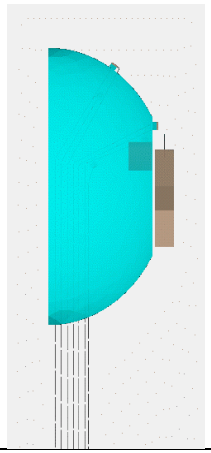
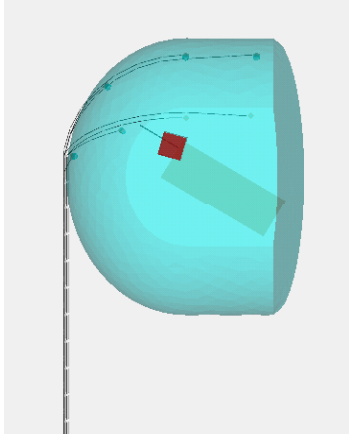
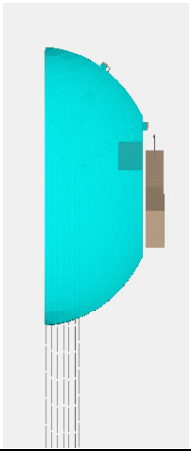
f =1800 MHz; No electrode leads; shell present		f =1800 MHz; leads not grounded; shell present		f =1800 MHz; leads grounded; shell present	
SAR over 10 g: 1.736 W/kg Vol. Avg. SAR: 18.02 mW/kg	**Handset: left-side**	SAR over 10 g: 1.746 W/kg Vol. Avg. SAR: 18.19 mW/kg	**Handset: left-side**	SAR over 10 g: 1.756 W/kg Vol. Avg SAR: 18.30 mW/kg	**Handset: left-side**
					
f =1800 MHz; No electrode leads; head only		f =1800 MHz; leads not grounded; head only		f =1800 MHz; leads grounded; head only	
SAR over 10 g: 2.24 W/kg Vol. Avg. SAR: 17.59 mW/kg	**Handset: left-side**	SAR over 10 g: 2.287 W/kg Vol. Avg. SAR: 17.76 mW/kg	**Handset: left-side**	SAR over 10 g: 2.288 W/kg Vol. Avg SAR: 17.79 mW/kg	**Handset: left-side**
					

Table 6.5 Shown are the 1800 MHz results of the six described simulations conducted to determine the peak spatial-average SAR levels inside the head for conditions with and with and without electrode leads present. Shown in red is the position of the peak spatial-average 10 gram cube inside the head with respect to the handset position on the far left-side.

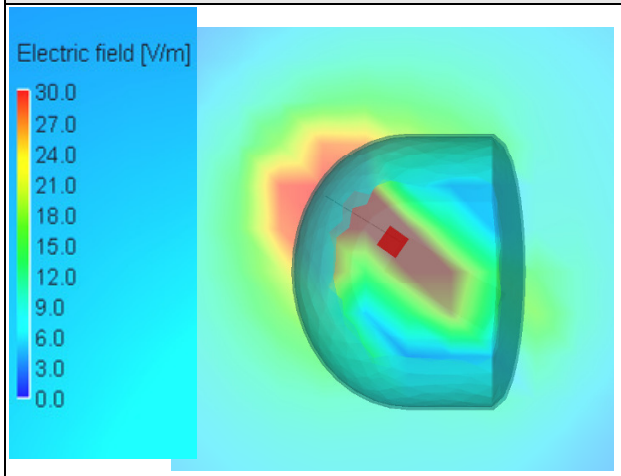
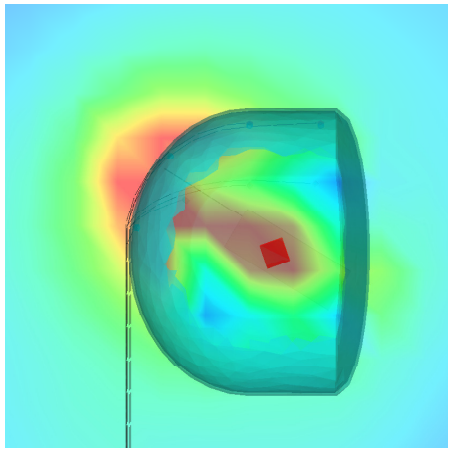
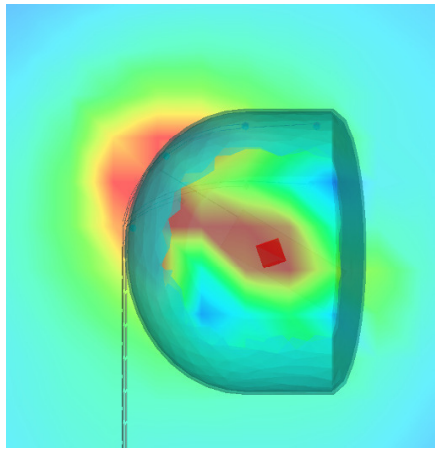
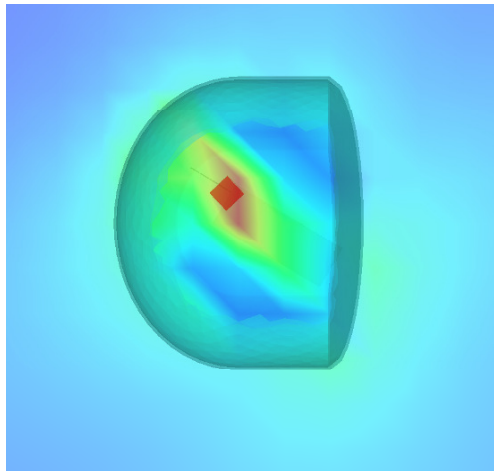
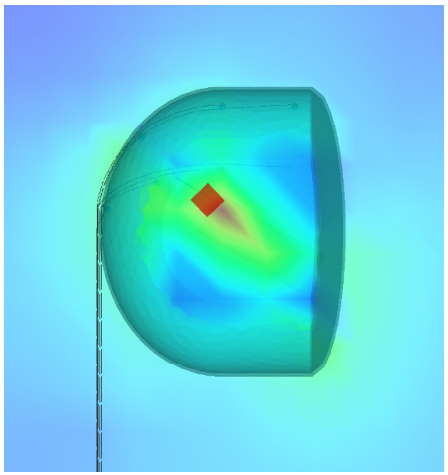
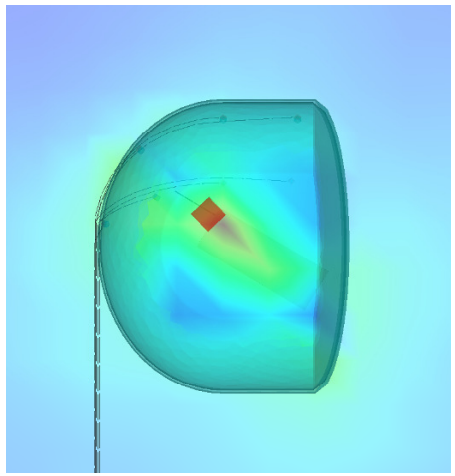
f = 900 MHz; No electrode leads; shell present	f = 900 MHz; leads not grounded; shell present	f = 900 MHz; leads grounded; shell present
		
f =1800 MHz; No electrode leads; shell present	f =1800 MHz; leads not grounded; shell present	f =1800 MHz; leads grounded; shell present
		

Table 6.6 Shown is a cut-plane across the peak spatial-average 10 gram cube (far left-side) which indicates the redistribution of E-field at the inner surface of the head (nearest to the handset). The upper-row 900 MHz illustrations demonstrate the peak spatial-average SAR shift (red cube) with respect to the E-field distribution when electrode leads are present, while for the same conditions at 1800 MHz as indicated in the lower-row there is no notably evident shift.

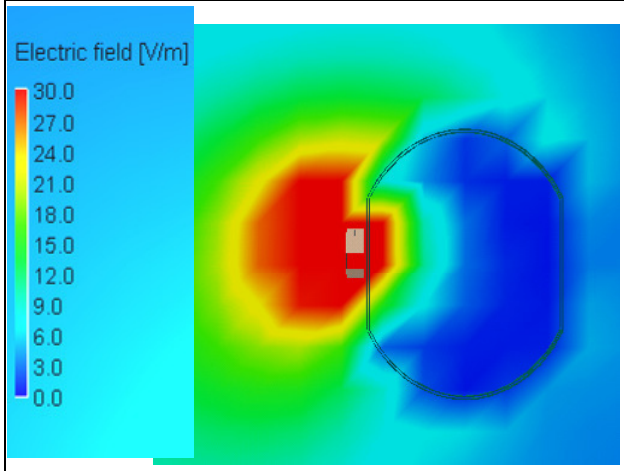
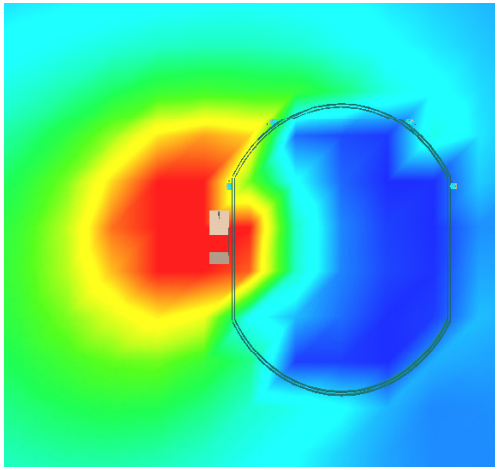
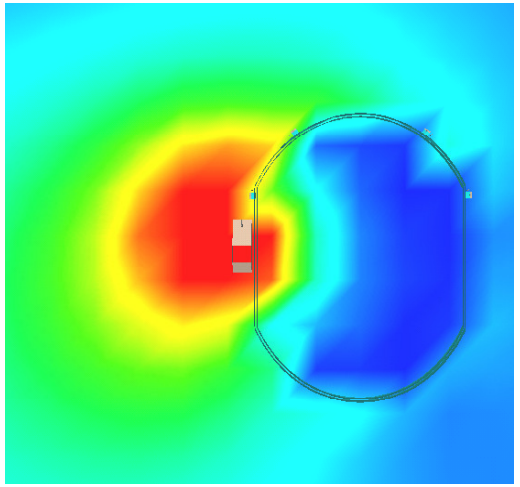
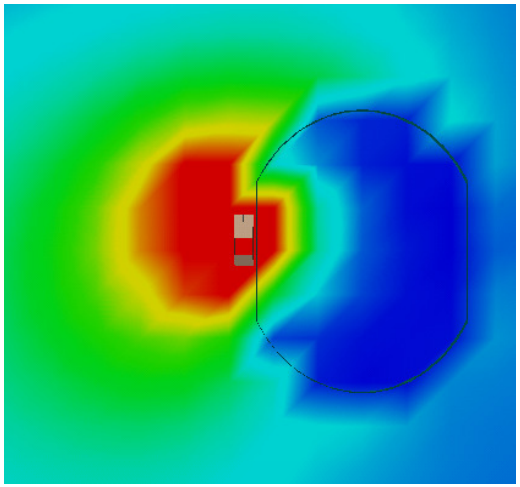
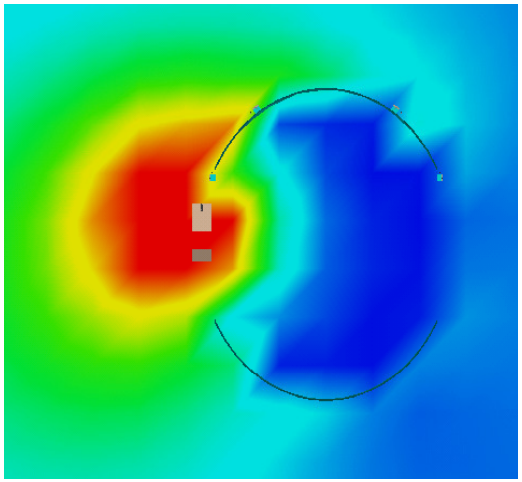
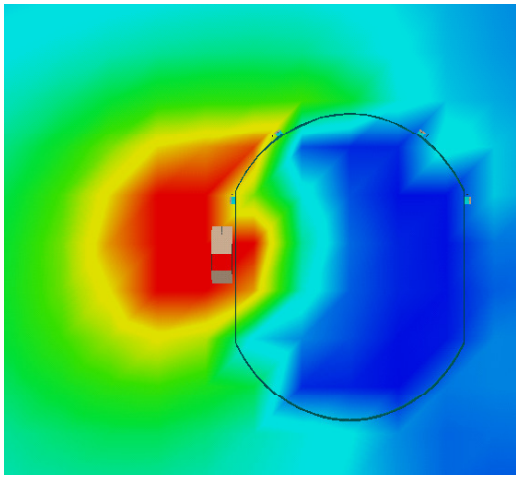
f = 900 MHz; No electrode leads; shell present	f = 900 MHz; leads not grounded; shell present	f = 900 MHz; leads grounded; shell present
		
f = 900 MHz; No electrode leads; head only	f = 900 MHz; leads not grounded; head only	f = 900 MHz; leads grounded; head only
		

Table 6.7 Shown is a cut-plane through the four mid section electrodes (~T7, C3, C4, and T8) demonstrating the E-field distribution at 900 MHz as indicated around the head, around the electrodes and leads, and inside the head with respect to the radiating handset (rear view of head).

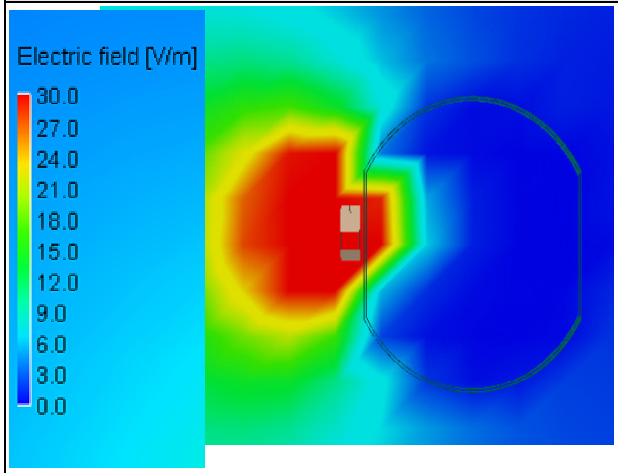
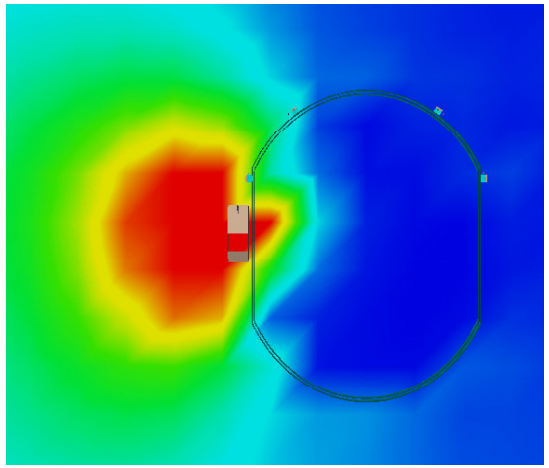
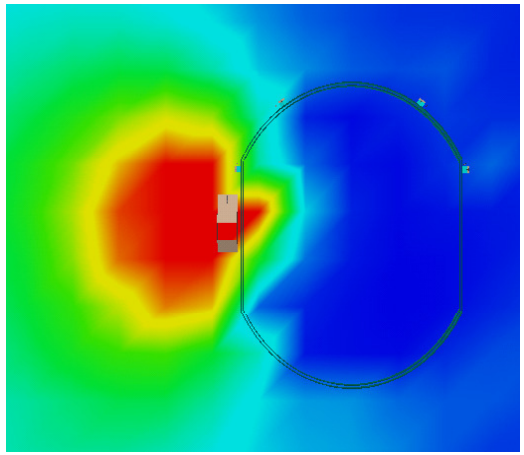
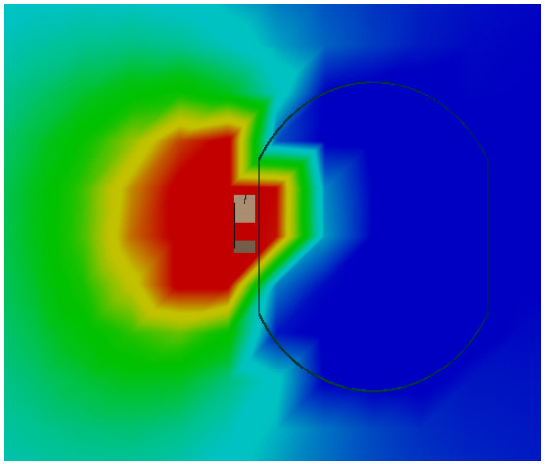
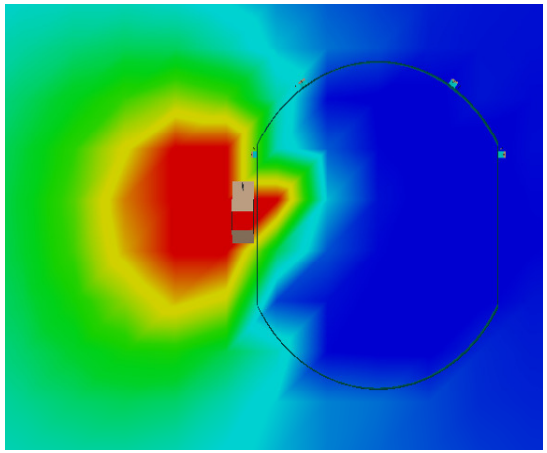
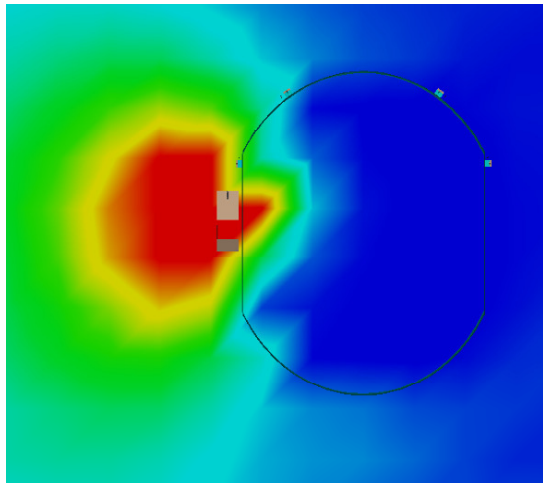
f =1800 MHz; No electrode leads; shell present	f =1800 MHz; leads not grounded; shell present	f =1800 MHz; leads grounded; shell present
		
f =1800 MHz; No electrode leads; head only	f =1800 MHz; leads not grounded; head only	f =1800 MHz; leads grounded; head only
		

Table 6.8 Shown is a cut-plane through the four mid section electrodes (~T7, C3, C4, and T8) demonstrating the E-field distribution at 900 MHz as indicated around the head, around the electrodes and leads, and inside the head with respect to the radiating handset (rear view of head).

6.5 Discussion

By observation of the 900 MHz and 1800 MHz exposure results (Tables 6.2 – 6.7) it may be observed that there are evident differences in the E-field distribution and SAR levels between simulated models with and without electrode leads present and evident differences also between grounded lead and non-grounded lead scenarios. In Tables 6.2 and 6.3, three-dimensional E-field diagrams of the exposure distribution, or *isoviews*, surrounding the handset near the head may in particular be observed to show exposures reradiating at regions near to the electrode leads extending downward from the back of the head for both simulated handset operating frequencies. At 900 MHz (Table 6.2) this reradiation is clearly visible at 11 V/m, while at 1800 MHz (Table 6.3) results evidently show reradiating exposure near to the electrode leads most clearly at 6.5 V/m or 4.5 V/m depending respectively on whether the shell forms part of the head model or not. Notably, corresponding SAR level results of these scenarios (Tables 6.4 and 6.5) are also shown to clearly vary, demonstrating that the spatial-peak SAR can shift in location due to the EEG electrode leads as indicated by the repositioning of the 10 gram average cube (shown in red) for both 900 MHz and 1800 MHz handset exposures. Specifically, observation of Table 6.4 demonstrates that at 900 MHz the spatial-peak SAR may reduce by up to 49 mW/kg (3.8 % decrease when leads are both not grounded and grounded) when EEG leads are introduced onto the head with shell, however can increase by up to 21 mW/kg (1.3 % increase: leads not grounded; 0% change: leads grounded) when electrode leads are present with no shell (i.e. electrodes attached directly to tissue or head). Likewise for the 1800 MHz results, Table 6.5 indicates that when electrode leads

and the shell are present an increase of up to 20 mW/kg (0.6 % increase: leads not grounded; 1.2% increase: leads grounded) is noted, while for the respective case where only the head is present (no shell) there is a slightly greater proportional increase of 48 mW/kg (~2.1 % increase when leads are both not grounded and grounded). Corresponding shifts in location of the peak spatial-average SAR 10 gram cube at 900 MHz may be observed to relocate from the handset's projected lower antenna region inside the head to its central body region, which arises irrespective of whether the electrode leads are grounded and only when the electrodes are positioned on the shell (Table 6.4). At yet a closer observation of this case, Table 6.6 indicates an evident redistribution of the peak electric field regions inside the head model specifically moving from the projected antenna region towards the centre of the handset body when the electrode leads are presented. This is notably unlike the same condition at 1800 MHz where only marginal changes in the 10 g cube location may be observed (Tables 6.5 and 6.6).

Noting that the EEG leads were indeed altering SAR levels and the RF field distribution inside the head, a further cross-sectional analysis across the width of the head (specifically a cut-plane through the four centrally located electrodes) was conducted for each simulated condition. Results of this analysis are indicated in Tables 6.7 & 6.8. As may be noted, a comparison of these results for both the 900 MHz and 1800 MHz exposures from the handset indicated significant differences between the 'no leads' and the 'leads present' condition. Specifically, for the 900 MHz results (Table 6.7), the outcomes for both shell and no shell conditions may be observed to indicate higher E-field levels in the order of 20 - 30 V/m (for both non-grounded and grounded conditions)

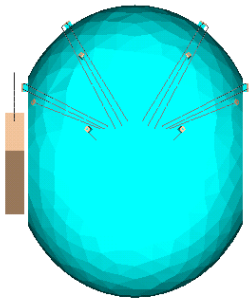
as appearing to be visible from the upper left (near the handset) towards the upper mid section of the head, illustrating indeed that the 900 MHz exposures *couple* and *redistribute* higher field strengths inside the head due to the lower (~F7, T7, P7) and upper row (~F3, C3, P3) of the EEG 10/20 system positioned electrodes near the handset. Similarly, but at lower field strengths, this outcome also holds true for the 1800 MHz exposures (Table 6.8) where coupled RF fields to the same leads redistribute field strengths in the order 10 - 20 V/m. Another important point of observation for both 900 MHz and 1800 MHz results as also indicated in Tables 6.7 and 6.8, is that the lower positioned central electrode (~T7, nearest to the handset) in all cases seems to clearly shield higher RF field strengths from appearing inside the head, in its vicinity. Notably, this shielding effect may be most predominately shown for the 'leads grounded' no shell condition at 900 MHz (Table 6.7), where there is an evident vacancy of the higher field strengths immediately behind the lower central electrode described.

Furthermore, given in summary that exposures at both operating frequencies are shown to couple to the leads falling behind the head as well as to the leads nearest to the handset, it was hypothesised that if the leads were shortened to a point where leads only extended to the back of the head (i.e. without falling – see Table 6.9 below), the incident RF energy should again redistribute and localise higher field strengths at the leads nearest to the handset, thereby causing increased SAR levels. To test this interesting hypothesis, subsequent simulations were carried out using the 900 MHz model with shell present (overall worst-case scenario identified), though the lead lengths at the back of the head were considerably shortened to the rear region of the head, as described for Table 6.9 below. Results of these tests indicated that SAR levels indeed increase when shorter

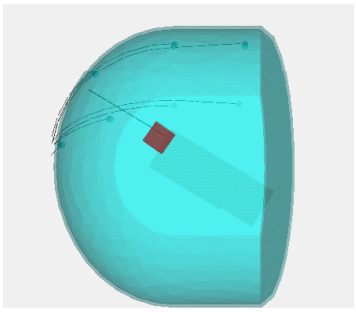
leads are present, but only marginally - specifically from 1.231 W/kg to 1.294 W/kg (i.e. causing a 1.1% increase over the no lead condition) as may be shown by drawing comparison between Figure 6.13 and 6.8. Notwithstanding this marginal difference however, this novel result demonstrates that the length of the falling EEG electrode leads may have a direct bearing on the SAR inside the head under real experimental conditions. For denser lead populations such as used in a 10/10 EEG system (up to 62 leads) the ratio between the lead lengths versus induced SAR is speculated to notably increase.

In an important addition to the 'shortened leads test', it was also most interesting to note in results that, unlike the corresponding results noted for leads at full length (Table 6.9), the peak spatial-average SAR *did not* shift from the projected antenna region to the handset's body, most clearly indicating that length falling leads may furthermore play a role in relocating the peak spatial-average SAR.

Shortened leads test (Handset on left-side of head)



Rear view of head



SAR over 10 g: 1.294 W/kg

Table 6.9 The shortened leads test indicated that by reducing the extent of falling leads prevents a shift in the peak spatial-average SAR over 10 g of tissue. Shown are the rear view and side view of the head indicating the lead extents and the SAR results

6.6 Conclusions

This investigation provides an original assessment and findings of the effects of RF exposures on biosignal leads positioned on the head in an arrangement similar to that of the International 10/20 EEG System array. In particular, the study indicates original findings that when mobile phone exposures are active near to the head in an ear-mouth position with EEG electrode leads present as described, the peak spatial-average SAR inside the head may only be marginally decreased or increased (i.e. within ±4 %) depending mainly on the mobile phone operating frequency and the EEG lead extension lengths. Although while RF coupling onto the leads has indeed been identified to direct energy into the head at both ipsilateral lower and upper leads to exposure (increasing SAR), there is also described evidence of a clear shielding effect due to the electrodes themselves (decreasing SAR)(Tables 6.7 – 6.8). Additionally it was found that the length of the falling leads themselves might couple and reradiate energy that would otherwise be more concentrated into the head (decreasing, and possibly shifting peak SAR)(Table 6.9). Consequently, the total net SAR inside the head when electrode leads are present is identified here to be due to a combination of coupling effects into leads which directs RF energy both into the head as well as away, and a shielding effect that prevents normally higher RF energy levels entering the head. As demonstrated by previous investigation the number of leads may support a dominant decrease effect in the net SAR level by increasing the number of electrodes and leads in a similar configuration to the present study (incident E-field vector ~45° to the leads)[D'Costa et al. 2004](Chapter 5), or otherwise likely produce a dominant increase net effect if one were to choose to orient

the incident E-field vector more parallel to the leads [Schmid et al., 2007][Hamblin et al., 2007].

Regarding comparison of this study with that indicated in Chapter 5, where a SAM phantom head was described to acquire practical measures of SAR with a 10/20 cap fixed to the left side, results may be said to be consistent for both 900 MHz and 1800 MHz exposures. At 900 MHz in this study the novel head model was shown to indicate an approximate 4 % decrease in the 10 g average SAR level (equivalently for both non-grounded and grounded conditions; shell present), as compared to a 5 % decrease as shown in the same measure for the practical condition (leads not grounded). Similarly, at 1800 MHz the results of this study indicated a marginal 1.3% increase (leads not grounded; shell present) and a 0% change (leads grounded; shell present) in the 10 g average SAR computed measure, while for the corresponding measures obtained at 1800 MHz for the practical condition there was a 0 % change indicated. Notably, in summary the proportional changes for leads present versus not present as compared within the 'shell present' and 'shell not present' conditions (i.e. electrodes positioned on tissue equivalent), are both within a ±4 % bracket. Thus, in conclusion it is suggested that for active exposures from mobile phones placed in an ear-mouth position near a 10/20 EEG system array, although complex changes within and around the head may occur in terms of the electric field distribution, these changes are not considerable enough to significantly alter the standard SAR result as produced when leads are not present.

Note: Computational detail of how FEKO determines the location of the 10 gram cube is based on "method of the tangential face" proposed by CENELEC [CENELEC, 2001].

Chapter 7

Statistical Methods to Distinguish Mobile Phone Exposure Effects in EEG Data

7.1 Introduction

The following Chapter aims to outline the development of a novel approach to statistically examining, and graphically communicating, the effects of mobile phone exposures on EEG data. Generally, the Chapter begins in Section 7.2 by describing the justification behind the novel statistical methods that were applied in the pilot study (Chapter 3), and in later sections subsequently describes why and how this method was further developed for a robust examination of the main experimental work involving 100 participants (Chapter 8). The statistical approach applied in the main study has been an invaluable tool for the detection of novel findings including a 'characteristic' response for the first time within the alpha band of the EEG, as due to the 900 MHz exposure signal from mobile phones. This is an important finding that will be described in Chapter 8, however it is significant to outline here that this result would have otherwise been impossible to detect without applying the developed statistical methodologies described in the following work.

7.2 Statistical Analysis of the Pilot Study and the Bonferroni Correction

In Chapter 3, a pilot study was described that incorporated a statistical analysis of EEG data acquired from ten participants during short-term mobile phone exposures. In that study, a novel statistical approach was undertaken whereby only EEG rhythms that were likely to be indicating a spectral power change due to the applied exposures were investigated. Specifically, this was undertaken by only considering the control and exposure data samples in the EEG rhythms where a high percentage (i.e. ±80%) of participants indicated either a decrease or relative increase in EEG spectral power. By doing this, the number of overall paired t-test comparisons that were required to be conducted were significantly reduced and hence so too was the probability of producing a type I error, i.e. the chance of indicating a false statistically significant result. In particular, by applying this approach the number of statistical tests conducted in the pilot study was reduced from as high as '192' to as low as '35' as in total over both talk-mode and standby-mode investigations. Interestingly, if this procedure were not undertaken, the common approach would have been to apply a 'Bonferroni correction' for multiple comparisons to reduce type 1 error on the '192' tests to theoretically maintain a 95% confidence level (for techniques see Bland et al., 1995). In practice it is not uncommon for the applied Bonferroni correction to consider correlation factor, k to lower otherwise overly conservative outcomes generated by the Bonferroni

correction alone [Sankoh et al., 1997][Curtin et al., 1998]. The reason for protecting against this conservativeness is to avoid over-correction of the significant p-value threshold, α. If an over-correction does occur it can lead to generating type II error, that is, the error of losing true positive outcomes in results. This is especially true where a greater number of comparisons are required to be accounted. One suggested method to include a correlation factor in the Bonferroni adjustment of α, is to apply the Dubey and Armitage-Parmar method [Sankoh et al., 1997], which is defined as follows:

$$P_{ax} = 1-(1-P_x)^{m_x} \quad \text{and,} \qquad \text{eq. 7.1}$$

$$\alpha_x = 1-(1-\alpha)^{1/m_x} \qquad \text{eq. 7.2}$$

where: $m_x = X^{1-k_{jx}}$ and,

$$k_{jx} = (X-1)^{-1}\sum_{j\neq x}^{X} k_{jx}$$

In equations 7.1 and 7.2, P_x and P_{ax}, may be respectively defined as the observed and adjusted x^{th} p-values, and α is the adjusted level for p-value significance for the x^{th} hypothesis for x = 1,….X. $k_{jx,}$ is the correlation coefficient between the j^{th} and x^{th} endpoints.

If a direct Bonferroni correction were to be applied to the 192 tests as described above for the pilot study, it would require the statistically significant p-value criteria of α = 0.05 to be lowered to α = 0.0002. Notably, because the lowest p-value outcome in the

pilot study was shown to be 0.004 (in the standby-mode trial), *no* statistically significant outcomes or effects due to the mobile phone exposures on the EEG data would have been noted. Similarly, if a Dubey and Armitage-Parmar method is applied to consider a correlation factor, k (correlating factor making the Bonferroni correction not so conservative), where it may be found to be k = ~0.2, the significant p-value criteria would 'rise' from $\alpha = 0.0002$ to $\alpha = 0.0007$. Nevertheless, it can be noted that even with a correlation factor considered, no statistically significant results would be evident in the pilot study. Considering the reduced number of tests of 35 which was actually administered in the preliminary study, the Dubey and Armitage-Parmar method would indicate to lower α from 0.05 to 0.0034 (k = ~0.2). Interestingly, in this case, it appears that the 0.004 p-value as described above for the pilot study approaches being statistically significant, as opposed to be being completely omitted when all 192 potential tests are considered. Thus, a question is raised here as to how applicable is the Bonferroni correction to EEG data considering differences between control and mobile phone exposure recordings? Figure 7.1 below, indicates the relationship between the Bonferroni correction and the correlation factor k described, for both the 192 tests and 35 tests that are considered relevant to the preliminary study (Chapter 3).

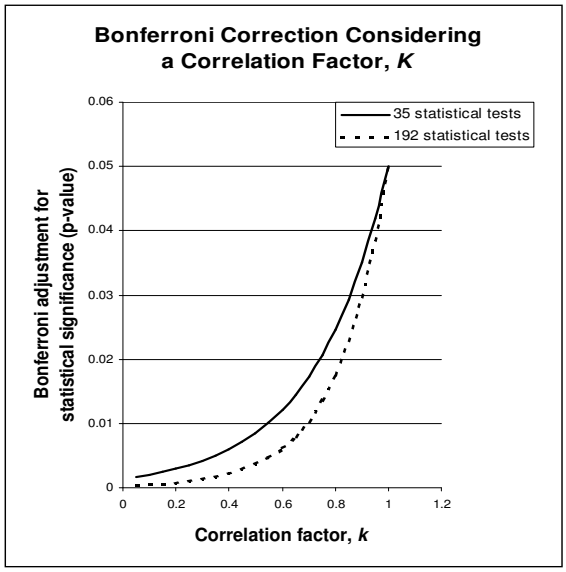


Figure 7.1 Shown is the graphical relationship between the Bonferroni correction and the correlation factor *k,* for the possible multiple t-test comparisons possible in the pilot study.

7.3 Importance of Investigating a Higher Resolution of the EEG Spectrum

One common statistical approach in studies investigating the effects of mobile phone exposures on the EEG has been to investigate and report the EEG in the standard clinically applicable bands of delta (~1-4 Hz), theta (~4-8 Hz), alpha (~8-13 Hz), and beta (~13 –32 Hz). Although these bands are undoubtedly an effective means by which brain dysfunction can be measured, it is not necessarily a viable approach to use these bands when attempting to detect an external frequency source such as mobile phone

exposures. For example, auditory and visually evoked related potentials (ERPs) are clearly a detectable measure in the EEG within these clinical bands and has been investigated considerably, however mobile phone exposures are not necessarily known to evoke brain patterns through sensory stimulus. So, what if EMFs from mobile phones cause a frequency or amplitude modulation only in a part spectral range of these bands? Unfortunately, if this were true, an analysis only in the clinically significant bands would likely mask this effect. This is because in order to investigate the statistical differences between Sham and Exposure recordings, requires the sample comparison of the total average spectral power across a band, such as the alpha band (i.e. the total average power across the alpha rhythms eg. 8 Hz, 9 Hz, 10 Hz, 11 Hz, and 12 Hz), to be calculated for each participant. Thus, if for example a marginal increase in spectral power is evident at 11 Hz and a marginal decrease is evident at 9 Hz, this effect would likely be cancelled due to averaging the total band. Similarly, it may also be imagined that if an effect were to span across the spectral boundary between two clinical bands, such as theta and alpha, the effect in both bands may be masked as analysis in each defined range would be conducted independently. To overcome this issue, it is proposed that the analysis of the EEG to detect mobile phone exposure effects should be interpreted at a high resolution for all non-ERP studies, that is, in at least 1 Hz increments over the 1 – 32 Hz spectrum.

7.4 Tendency, Confidence Intervals, and Paired T-test Outcomes

There have been a considerable number of reports that have indicated that only a notable proportion of the total amount of participants may indicate a change in EEG spectral power due to mobile phone exposures [Krause et al., 2007][Vecchio et al., 2007]. Considering this, it has become of greater importance to not only report on statistically significant outcomes but to indicate notable increased or decreased tendencies in EEG power over the participant load. In the pilot study this was accomplished by indication of the percentage of subjects indicating either an increase or decrease in EEG power within each rhythm over each recording site. However, when hypothetically applying this approach to a greater number of electrode sites such as a 10/20 system array (as would be incorporated into the major investigation – Chapter 8) it was determined that it was not viable to effectively present this dimension with the other two considering so many sites (up to 19 locations). To overcome this, it was decided to utilise 'graphs of the confidence limits' to indicate the probable spread of data over each EEG rhythm, independently for each electrode site. Furthermore, as it may be recalled that if the confidence interval tends completely to one side of the hypothesised mean difference (i.e. applicably the hypothesised mean difference between Sham and Exposure = 0), the sample data pairs are deemed notably statistically significantly different at the

prescribed significance level, α. The 95% confidence interval (α = 0.05) is defined as:

$$\bar{x} \pm 1.96\left(\frac{\sigma}{\sqrt{n}}\right) \qquad \text{(Eq. 7.3)}$$

where: 1.96 is the approximate number of standard deviations from the mean (representing coverage of 95% of the upper tail of the normal curve)

σ = standard deviation;

n = sample size

Consequently, rather than showing the paired t-test outcomes in a separate table to the distribution graphs (as it were for the pilot study) it was found that this information may be presented on one graph, to effectively describe the behaviour of the EEG during exposure conditions for any recording site. By incorporating a plot of the median difference curve between the confidence interval graphs the method on how to represent the EEG data was complete. An example of this graph (as adopted from a real outcome as produced from the major study - Chapter 8) is presented below in Figure 7.2. Notably, the rhythms at which the Upper or Lower confidence limits cross the frequency axis, indicate the rhythms where statistically significant spectral power differences were evident between Sham and Exposure. For example, an *upper* limit crossing the frequency axis at 10 Hz would indicate a significant *increase* in spectral power during exposure at 10 Hz, and vice versa.

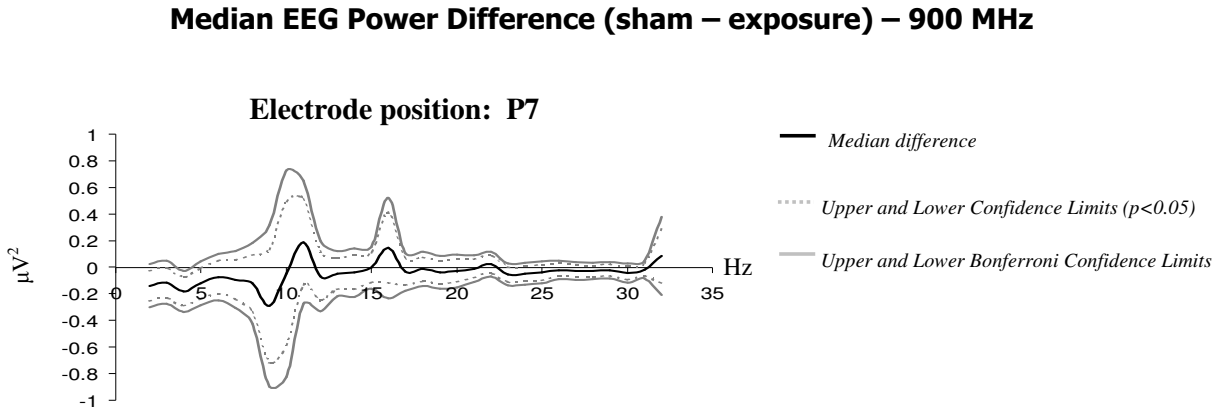


Figure 7.2 Shown is the representation of the method developed to effectively represent the recorded EEG during active exposure periods. Notably, the graphical response indicates the extents of type I and II error probabilities in terms of microvolts as well communicates the EEG rhythms in which statistically significant differences between sham and exposure occurred (i.e. by easily viewing where the Bonferroni or uncorrected (0.05) Upper and Lower limits cross the frequency axis). For example the graph communicates that statistically significant *increases* in EEG power occurred at '4 Hz' by indication of the upper confidence limits falling *below* the frequency axis.

7.5 Including the Bonferroni Corrected Confidence Intervals

Noting in Introduction (Section 7.1), that there is chance of producing a type I error due to the multiple comparisons associated with investigating mobile phone exposure effects in the EEG, and yet further a possibility of producing a type II error when correcting for these comparisons, it was decided to also add to the graphical approach the upper and lower confidence limits that correspond to the Bonferroni corrected significance level considering a correlation factor k (as defined in Section 7.2). This confidence interval may be defined as:

$$\bar{x} \pm z(\alpha_x / 2)\left(\frac{\sigma}{\sqrt{n}}\right) \qquad \text{(Eq. 7.4)}$$

where α_x is the adjusted significance level (as defined in Section 7.2)

σ = standard deviation;

n = sample size

Resultantly, Figure 7.2 may also be noted to include the Upper and Lower 'Bonferroni' confidence limits. Specifically, by indicating these confidence limits it was possible to demonstrate the type I and type II error extents with respect to the Sham and Exposure EEG spectral power differences for each rhythm. This is in accordance with expertise on the matter of statistical corrections who particularly note the importance of communicating in publication the impact of type I and II error, where multiple comparisons are concerned [Feise, 2002].

7.6 Discussion & Conclusion

In this Chapter, the development of a novel statistical approach to investigation mobile phone exposure effects in the EEG is described, which is to be implemented into the major study described in Chapter 8. In particular, this Chapter summarises the different *dimensions* associated with EEG spectral analysis and introduces a novel method to

effectively dealing with the issue of multiple statistical comparisons and a known large spread of collected EEG data, without having to forfeit indicating in results detailed graphical outcomes. In regards to multiple comparisons, the original management of type I and type II errors are specifically addressed, highlighting an approach to interpret the extent of these errors by means of comparatively representing graphs of both the Bonferroni corrected and uncorrected statistically significant levels by means of 'confidence limit' interpretation. Most relevantly, the confidence intervals with a merged plot of the median difference response is described to be an invaluable technique for displaying the behaviour of the resultant EEG of the active EMF exposure period. The approach is particularly effective as the EEG spectral power tendency over the sample size as well as all statistically significant results can be communicated in one generated display.

In conclusion, it may be outlined that the statistical approach as developed and described in this Chapter, has significantly contributed to confidence devoted in the important findings produced by the major investigation (Chapter 8).

Chapter 8

Effects of 900 MHz and 1800 MHz Mobile Phone Exposures on Human Brain and Heart Activity at SAR levels near the Maximum Allowable Limit

The present study aims to investigate whether the EEG, ECG, or blood pressure in resting adults is influenced by pulse modulated 900 MHz and 1800 MHz exposures. Of interest, unlike most previously published research corresponding specific energy absorption rates (SAR) have been measured considerably near to the maximum allowable limit of 2 W/kg [ARPANSA RPS3, 2002].

8.1 Introduction

Over approximately the last 15 years there has been indeed a considerable amount of published papers on the effects of mobile phone exposures on human brain wave activity and fewer regarding effects on heart response. Studies on brain wave activity monitored by the electroencephalogram (EEG) have included investigations of the sleep or awake condition [Fritzer et al., 2007][Röschke et al., 1997], or studies on cognitive performance

examining preparatory thought function or working memory by means of induced event-related potentials (ERPs)[Freude et al., 2000][Krause et al., 2007]. Alongside these reports have also been independent behavioural investigations of mobile phone effects on vigilance and reaction time [Preece et al., 1999][Russo et al., 2006]. In one somewhat similar investigation to the present study, Hietanen et al. [Hietanen et al., 2000] as mentioned in Literature Review (Chapter 2) reported on the resting awake EEG effects of three analogue phones (NMTs) with different antenna types and two digital phones (GSM and PCN) operating at 900 MHz and 1800 MHz (1-2 W peak output). Considering participants (N = 19) exposed over the right ear in 20-minute trials (eyes closed condition), results of an analysis in standard frequency bands only indicated a notable EEG change in the delta band however this effect was omitted as having occurred due to statistical chance. Notwithstanding this outcome however, many other related investigations [D'Costa et al., 2003a][Curcio et al., 2005][Croft et al., 2007] have shown notable positive findings predominately indicating ongoing unsubstantiated support that mobile phone exposures may modulate the alpha band. However, despite these results, based on contradictory and subtle results presented in the literature, inconsistent procedures between studies, and a lack of implemented control of scientific uncertainties, there has been scrutiny that studies using the EEG as a test measure to detect microwave effects are unreliable if not produced under a standardised and improved methodological foundation [Barnes & Greenebaum, 2007].

Apart from an examination of the EEG, investigation of mobile phone exposures on the electrocardiogram (ECG) and blood pressure have been considered. Previous studies [Mann et al., 2002][Braune et al., 2002][Atlasz et al., 2006][Hietanen et al., 2002][Tahvanainen et al., 2004] investigating mobile phone exposures on heart related test measures have generally indicated no prominent positive findings, principally supporting improbable mobile phone exposure affects on vasomotor function and heart regulation. Notwithstanding this however, few studies have indicated positive outcomes [Huber et al., 2003][Parazzinni et al., 2007]. Recently Parazzini et al. [Parazzinni et al., 2007], reported significant but weak increases in spectral ECG power due to GSM 900 MHz exposures (controlled 2 W output) during standing protocol as well as a decrease in variability of the duration of the cardiac cycle over 26-minute trials ($N = 26$). However, no significant effects could be detected during rest periods. Yet in another study incorporating 9 participants Braune et al. [Braune et al., 1998] showed that GSM 900 MHz exposure (2 W peak output) for 35 minutes indicated significant decreased changes in the heart rate and prominent increased changes in blood pressure in both standing and rest. However, in a subsequent investigation Braune et al.'s findings could not be verified to be due to the exposure previously tested [Braune et al., 2002].

8.2 Participants

A total of 100 healthy volunteers participated in this investigation including 56 men and 44 women aged between 18 and 40 years (mean = 25.6 yrs; SD = ±5.1 yrs). Before

inclusion each subject was required to read a two-page summary of the experimental procedures and gave signed consent that they were not on any medication in the recent month prior to the trial period and did not knowingly have any physical or mental health concerns. Each participant received a payment for his or her volunteered time in the experimental procedures that were approved by RMIT University's Human Research Ethics Committee.

8.3 Materials and Recording Methods

During the experiment each participant was required to be seated in a comfortable chair with eyes closed for multiple trials while alternate mobile phone exposures were generated in close proximity to the left side of the head. During the trials three simultaneously recorded test measures were acquired namely, the EEG, ECG, and pulse plethysmograph (PPG). To record the EEG, brain wave signals were acquired with the aid of an electrode cap (Electro-cap) that incorporated positioned electrodes in accordance with the 10-20 EEG International Standard [Jasper, 1958]. Measurements were recorded from all left and right hemispherical sites as referenced to the right mastoid. To enable the EEG recordings, electrode leads extending from the electrode cap were fed to the inputs of an EEG differential amplifier unit, Mindset MS-1000. The Mindset MS-1000 unit was in turn connected to a laptop computer where the EEG signals could be viewed and logged using the software package 'MindMeld live data capture'

V.1.0. EEG gel ('Quik-gel') was used to help ensure low impedance contact between the surface electrodes and scalp over the length of the experiment.

In order to record the ECG, standard electrolyte adhesive electrodes were placed on both inner wrists and on the left ankle in accordance with the augmented unipolar lead system [Goldberger, 1942]. Recording leads extending from snap-points on all three electrodes were then fed into a module-oriented biosignal acquisition unit 'Biopac' that was connected to a second laptop computer to record and view the heart signal. To record the PPG signal, a pulse transducer (TSD-200) was secured comfortably at the tip of the right index finger using a small cuff. A recording lead from the tranducer was then fed to the Biopac system to be viewed simultaneously with the ECG (Figure 8.1). In a unique approach to similar studies, to detect resting blood pressure fluctuations without disturbance to the participant during exposure, the time interval between the recorded ECG and PPG signal peaks or 'pulse transit time' (PTT) was determined (Figure 8.1). In particular the PTT was used to indicate *blood pressure variability* (BPV) in knowledge that as the wave velocity (v) of the PPG measured pressure pulse from the heart (lagging the ECG) *increases*, blood pressure *increases* (as vessel volume decreases), and vice versa (Eq. 1)[Bramwell, 1922] (*see also Biopac application online manual*). Thus, given that the PPG pulse travels a fixed distance from the heart to the measured fingertip, an increase in wave velocity will shorten the transit time, and hence a *decrease* in the PTT may represent an *increase* in blood pressure, and vice versa. Other more recent research based fundamentally on this relationship has been reported [Gribbin et al., 1976][Ma et

al., 2005][Atlasz et al., 2006]. Biopac system acquisition software AcqKnowledge' V.3.7.3, was used to simultaneously record and view the ECG and PPG measurements. In accordance with notes taken during visual monitoring, artefact removal was considered manually for each of the EEG, ECG, and PPG simultaneous measured trials.

$$v = K \bullet \sqrt{V(\Delta P / \Delta V)}$$

(Eq. 1) Bramwell & Hill, 1922

Where: v = Pulse wave velocity
K = constant
V = Initial vessel volume
P= Pressure delta
V= Vessel volume delta

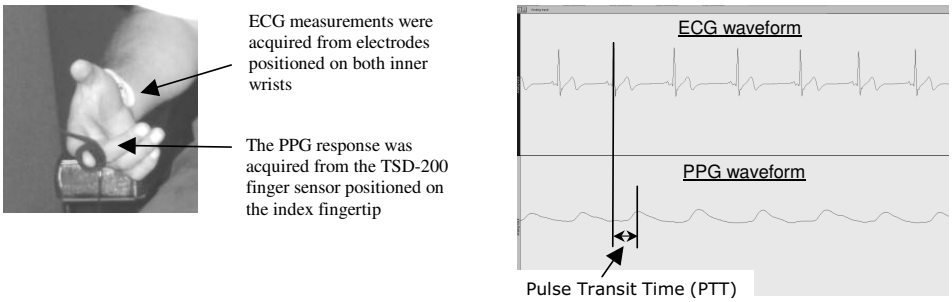


Figure 8.1 Shown are illustrations of the ECG and PPG acquisition technique implemented. Simultaneous measurement of the ECG and PPG allowed for increases and decreases in blood pressure to be noted without disturbing the participant at rest. This was achieved by calculating the time interval between the measured peaks of the ECG and PPG, or pulse transit time (PTT). A noted increase in the PPT is known to represent a decrease in blood pressure, and vice versa.

8.4 Sources of Exposure

During trials, both 900 MHz and 1800 MHz exposures pulsed at 217 Hz were generated by a modeled mobile phone oriented in an ear-mouth position as comparable to that of the touch position as used in standard SAR testing procedures [IEEE 1528 2003]. The experimental handset itself consisted of an 11x4x2 cm rectangular aluminium block with a short interchangeable ¼ λ monopole antenna at its top. The antenna was connected at the top right corner of the handset to a semi-rigid RF feed line that was concealed through

the length of the block. To generate controlled exposures, the pulsed RF signals from a programmable digital pulse generator were fed via coaxial cable to an input point of the RF feed line as exposed at the handset's base. The final design of the handset was mainly influenced by concepts used in other experiments conducted by Mochizuki et al. [Mochizuki et al., 2004] and Reiser et al. [Reiser et al., 1995]. The handset was held in position during trials with the aid of a rigid plastic arm and bracket assembly to secure the handset in one fixed position (Figure 8.3).

Prior to use, several standard SAR tests using a DASY-4 robotic facility[1] were conducted on the test handset in order to adjust its input power levels to meet near to the maximum allowable limit of 2 W/Kg over any 10 gram of tissue [ARPANSA RPS3, 2002]. SAR levels ultimately produced were 1.56 W/kg at 900 MHz (276 mW) and 1.4 W/kg at 1800 MHz (163 mW) as measured with a standard uncertainty level of ± 26.4 % [IEEE 1528, 2003]. Actual design of the test handset as well as indications of unlikely changes in the SAR levels due to potential EEG lead RF shielding has been previously reported [D'Costa et al., 2003a][D'Costa et al., 2003b][D'Costa et al., 2004]. In relation, earlier work testing 'flat-line' recordings using the EEG recording equipment (Mindset-1000) (as acquired from a resting adult's thigh) (Chapter 3) did not indicate any RF pickup on the recording leads when GSM 900 MHz exposures were placed nearby [D'Costa et al., 2003a].

[1] SAR testing conducted at EMC Technologies Pty Ltd, Australia

8.5 Trial Procedure

During the experiment, the test handset was set to one of three possible exposure modes namely, 900 MHz pulsed at 217 Hz, 1800 MHz pulsed at 217 Hz, and sham exposure. Under double blind conditions, each participant undertook five 4-minute seated trials (one session) for each exposure mode, during which time simultaneous measurements of the resting EEG, ECG, and PPG were recorded (eyes closed, awake). Participants were seated inside a large RF shielded room while the RF signal generator was situated outside, connecting to the handset via coaxial cable through a fitted wall adaptor. Between each 4-minute recording, a 2-minute break was provided where the exposure did not cease, however the test handset was moved at least 0.5 m away from the head of the participant who was then free at this time to open their eyes, talk, move their head, or drink water if desired. In addition to these breaks, further 10-minute breaks were allocated between each completed five 4-minute trial session, which was set to allow time for a laboratory assistant to: 1. Discretely replace the interchangeable ¼ wavelength antenna on the handset to correspond to the next frequency mode to be set (regardless if sham mode) in accordance to a randomly counterbalanced sequence; 2. Disguise the differing antenna lengths from the experimenter and participant using a thin rectangular foam block to surround the antenna; 3. Discretely alter the handset's exposure mode on the signal generator (choice of one of 3 'mode-programmed' keys) to match the next relevant mode to be set; and 4. Ensure the input power levels to the handset were correct for the previous and upcoming exposure using a digital power meter, accounting for cable

loss. The timeline of the conducted experimental procedure is illustrated below in Figure 8.2, while Figure 8.3 indicates the participant conditions during trials.

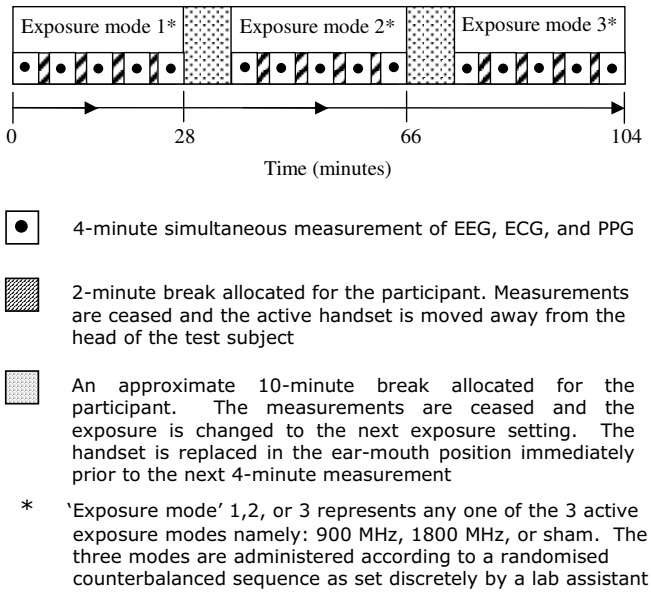


Figure 8.2 Shown is the experimental timeline inclusive of the recording times and break conditions for each participant during the three modes of exposure

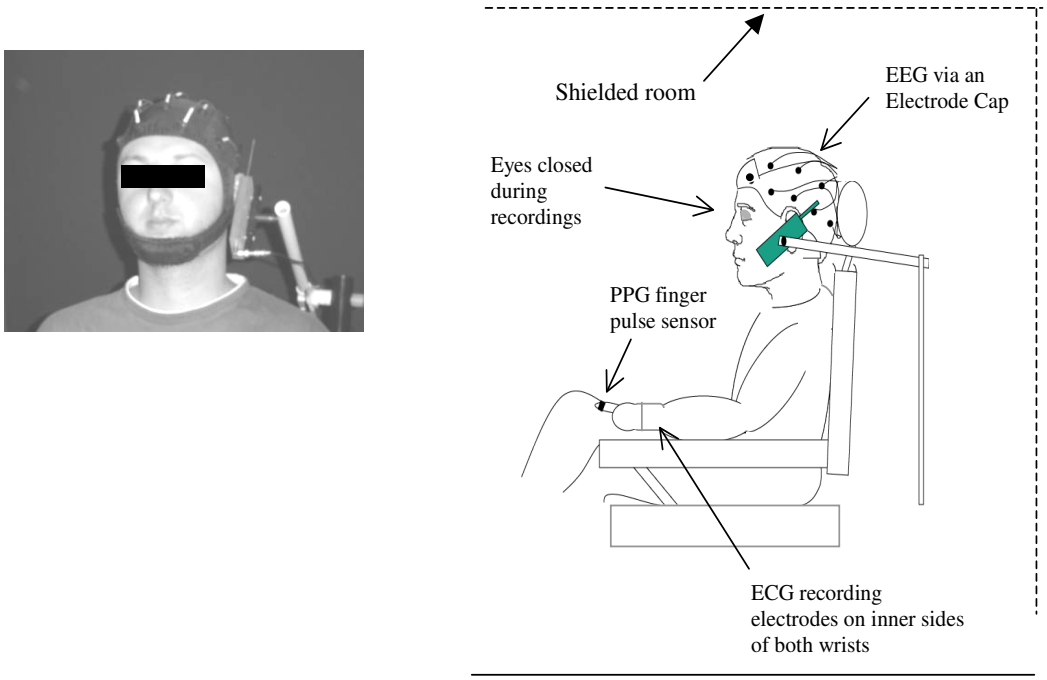


Figure 8.3 Shown are the participant conditions as implemented during the experimental procedures

8.6 Results

The following results indicate the outcomes of paired t-test comparisons of EEG spectral power (Sham vs. Exposure) over two independent approaches. Firstly, Figures 8.4 and 8.5 illustrate the outcomes of the EEG analysis conducted in the four significant spectral bands namely delta (2 - 4 Hz), theta (4 - 8 Hz), alpha (8 – 13 Hz), and beta (13 – 32 Hz)(comparison of grand mean spectral power over each session). And secondly, Figures 8.6 and 8.7 indicate the merged graphs of the median spectral power difference and the 95% confidence limits (Bonferroni corrected (using a correlation factor) and uncorrected) as determined in 1 Hz increments over a 2 – 32 Hz EEG spectrum (comparison of the median spectral power over each session). The 'median' specifically refers to the average of all participants' median spectral power of the five (4-minute) EEG recordings in 1 Hz increments as calculated for each exposure tested. The results shown in Figure 8.6 provide the 900 MHz and 1800 MHz results alongside one another as indicated for the left hemisphere (RF exposed side), while Figure 8.7 demonstrates the EEG results as indicated for the right hemisphere (contralateral side to exposure). Importantly, in order to identify statistically significant outcomes in Figures 8.6 and 8.7, the EEG rhythms at which the 95% upper or lower confidence limit curves respectively cross below or above the frequency axis, must be observed. For example, an *upper* confidence limit curve crossing *below* the frequency axis at 10 Hz indicates a statistically significant *increase* in EEG power at 10 Hz, and vice versa. Additionally, by noting the different extents between the Bonferroni corrected and uncorrected confidence limit graphs, the actual

marginal difference which Type I and Type II error may impose on similar data, of a similar sample size, can be observed. In turn, this may importantly alleviate concern for some previous studies where multiple comparisons have not been appropriately accounted for. Notably, as described in Chapter 7, the applied Bonferroni adjustment here considers a correlation factor (as calculated to be $k = 0.65$) by application of the Dubey and Armitage-Parmar method [Sankoh et al., 1997].

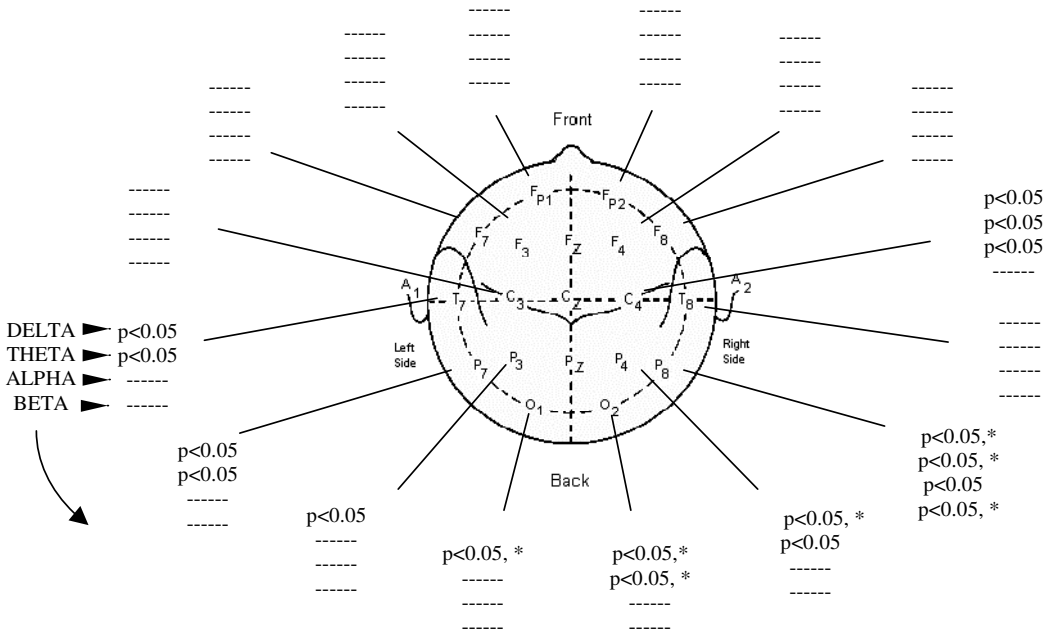


Figure 8.4 Shown are the results of the 900 MHz paired t-test analysis of grand mean spectral power (Sham vs. Exposure) as conducted over the EEG spectral band delta (2 – 4 Hz), theta (4- 8 Hz), alpha (8 – 13 Hz), and beta (13 – 32 Hz) for both left and right sides of the head. All p-values as shown less than 0.05 or the Bonferroni corrected level of 0.0093 (denoted by *) indicate a statistically significant <u>increase</u> in EEG power, not exceeding 5 μV^2. No statistically significant decreases in power could be noted.

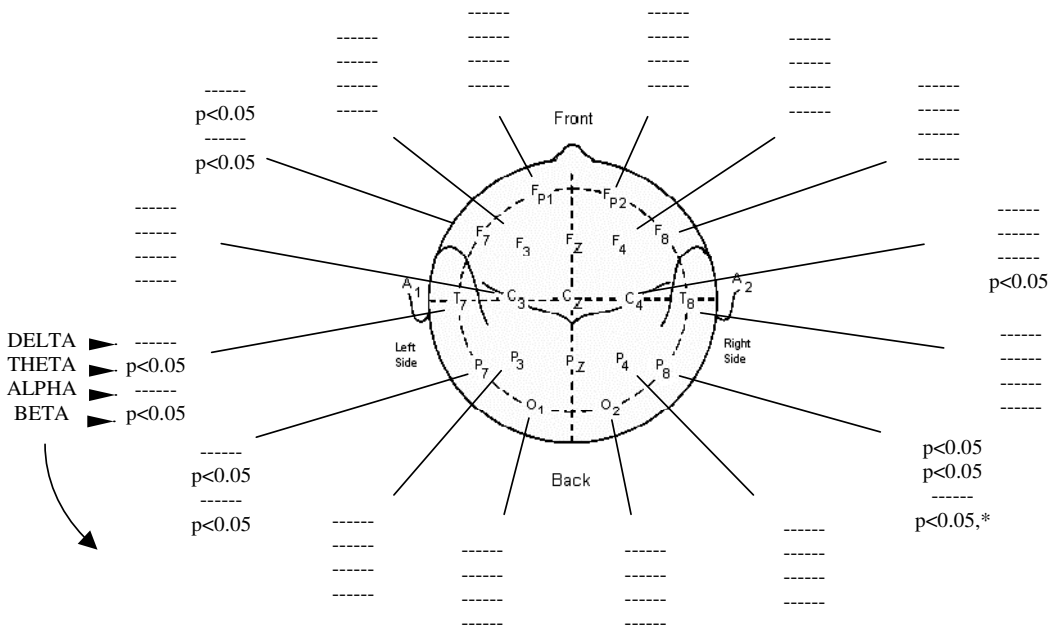


Figure 8.5 Shown are the results of the 1800 MHz paired t-test analysis of grand mean spectral power (Sham vs. Exposure) as conducted over the EEG spectral band delta (2 – 4 Hz), theta (4- 8 Hz), alpha (8 – 13 Hz), and beta (13 – 32 Hz) for both left and right sides of the head. All p-values as shown less than 0.05 or the Bonferroni corrected level of 0.0093 (denoted by *) indicate a statistically significant <u>increase</u> in EEG power, not exceeding 5 μV^2. No statistically significant decreases in power could be noted.

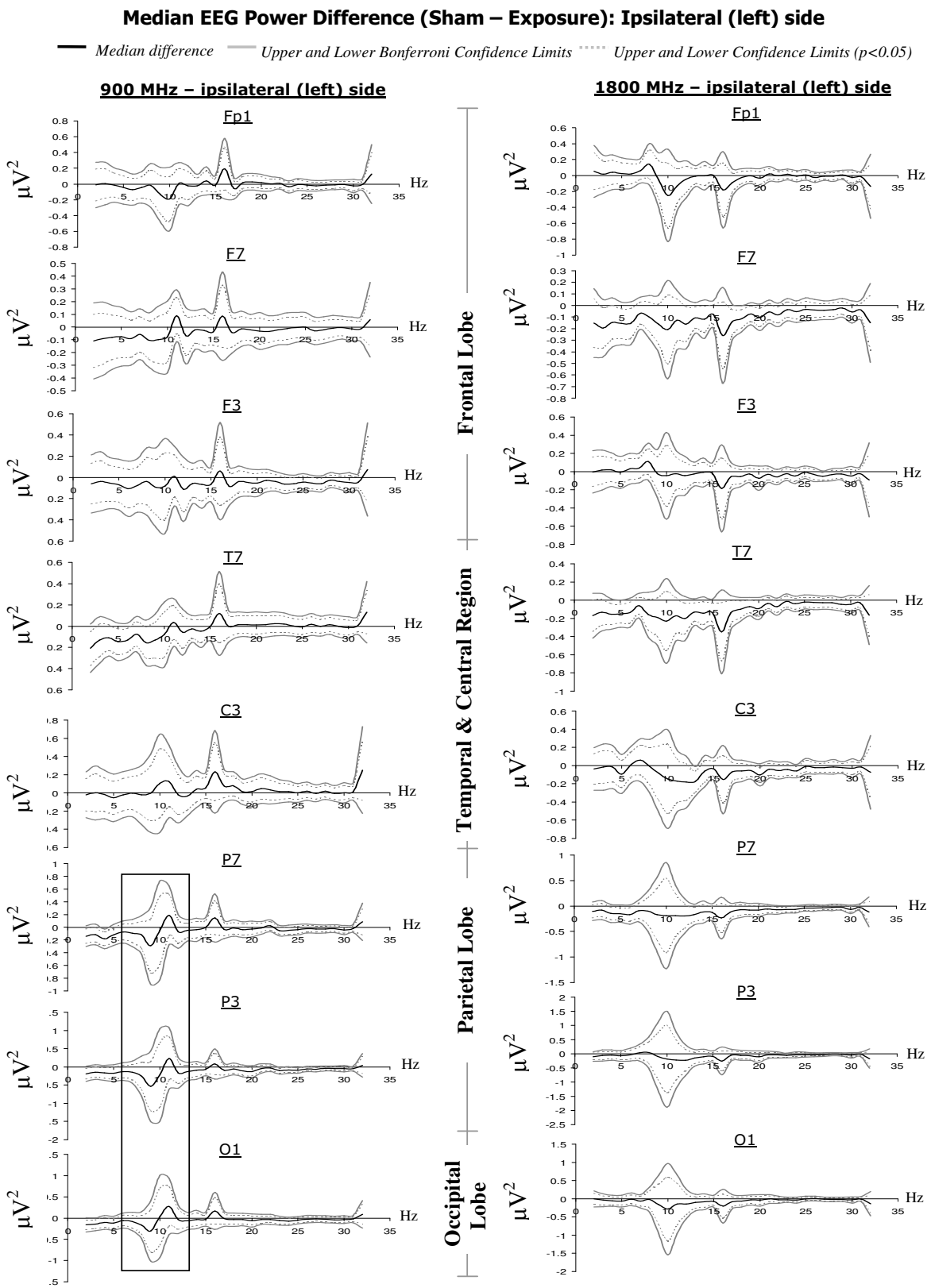


Figure 8.6 Shown are the 900 and 1800 MHz results as indicated for the left side of the head (RF exposed side). The rhythms at which the upper or lower confidence limits (Bonferroni corrected ($p<0.0047$) or uncorrected ($p<0.05$)) cross the frequency axis, indicate the rhythms where statistically significant spectral power differences were evident between sham and exposure eg. An *upper* limit crossing the frequency axis at 10 Hz would indicate a significant *increase* in spectral power during exposure at 10 Hz, and vice versa. 'Median' specifically refers to the average of all participants' Median spectral power of the five (4-minute) EEG recordings in 1 Hz increments (calculated for each exposure mode tested). The 'Median difference' curve is thus a plot of the *average* Median Sham minus the *average* Median exposure spectral power over the EEG spectrum.

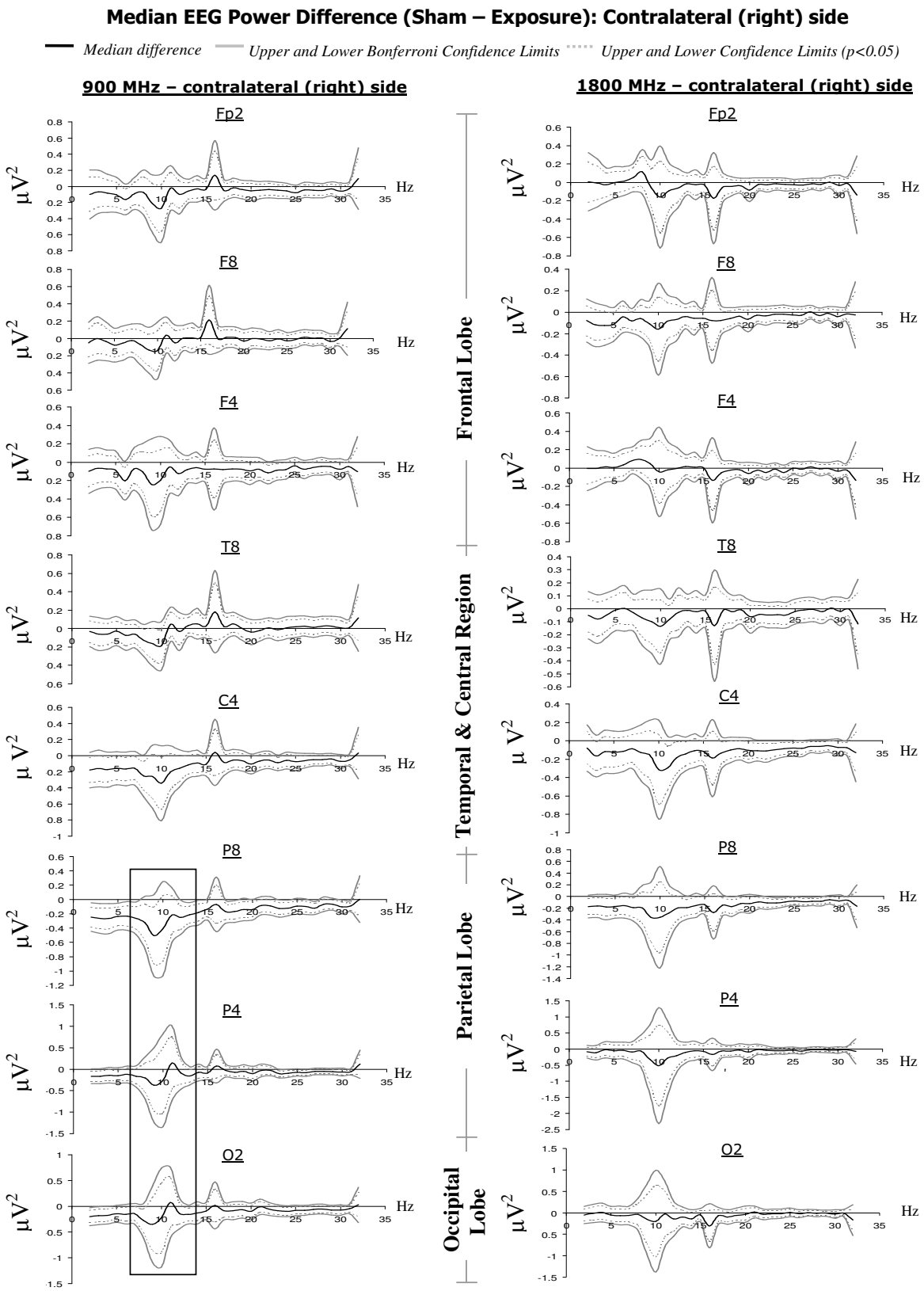


Figure 8.7 Shown are the 900 and 1800 MHz results as indicated for the right side of the head (contralateral to exposure). The rhythms at which the upper or lower confidence limits (Bonferroni corrected ($p<0.0047$) or uncorrected ($p<0.05$)) cross the frequency axis, indicate the rhythms where statistically significant spectral power differences were evident between sham and exposure eg. An *upper* limit crossing the frequency axis at 10 Hz indicates a significant *increase* in spectral power during exposure at 10 Hz, and vice versa. 'Median' specifically refers to the average of all participants' Median spectral power of the five (4-minute) EEG recordings in 1 Hz increments (calculated for each exposure mode tested). The 'Median difference' curve is thus a plot of the *average* Median Sham minus the *average* Median exposure spectral power over the EEG spectrum.

One most apparent note in all results is that only statistically significant *increases* in EEG power can be noted as may be observed up to power spectral densities not exceeding 5 μV^2 (Figures 8.6 and 8.7). For the 900 MHz exposure results shown in Figure 8.4, statistically significant increases in EEG spectral power observed at the $p<0.05$ level were noted at T7 (delta and theta), C4 (delta), P3 (delta), P4 (delta and theta), P7 (delta and theta), P8 (delta, theta, alpha, beta), O1 (delta), and O2 (delta and theta). Statistical significant indications at the Bonferroni corrected level however ($p<0.0093$) were only demonstrated at the occipital and parietal regions, namely O1 (delta), O2 (delta and theta), P4 (delta), and P8 (delta, theta, and beta). Interestingly, as comparable with results found in the pilot study (Chapter 3) no statistically significant outcomes could be noted for any of the frontal electrode positions at 900 MHz for the EEG band analysis (Figure 8.4). For the corresponding 1800 MHz results statistically significant results in EEG power at both p-value levels were notably more limited (Figure 8.5). At $p<0.05$ significant increases in EEG power were indicated at F7 (theta and beta), T7 (theta and beta), C4 (beta), P7 (theta and beta), and P8 (delta, theta, alpha, beta). While at the Bonferroni corrected level of $p<0.0093$, statistically significant differences between sham and exposure were only noted at beta (12-32 Hz) from the P8 electrode position contralateral to exposure.

The higher resolution analysis conducted in 1 Hz increments from 2 - 32 Hz (presented in Figures 8.6 and 8.7) demonstrated greater detail of the experimental results. Results indicated at 900 MHz and 1800 MHz for the RF exposed left-side (Figure 8.6) showed significant increases in EEG spectral power at both statistical levels observed (i.e. at rhythms at which the *upper* confidence limit curves fall *below* the frequency axis). At

$p<0.05$, evidence of a potential increases due to 900 MHz exposure were noted at T7 (2, 5, 6, 8 Hz), P7 (2-5 Hz), P3 (2-5, 20, 23-27 Hz), and O1 (2, 3, 5, 24 Hz). At the corresponding Bonferroni corrected level at 900 MHz ($p<0.0047$), statistically significant increases in EEG spectral power were only commonly indicated from P7 at the theta rhythm 4 Hz, and from P3 at the beta rhythms 23 and 25 Hz. Quite dissimilarly however, at 1800 MHz at the $p<0.05$ level (Figure 8.6) statistically significant increases were evident at F7 (3, 5, 6, 12-14, 17-20, 22 Hz), F3 (22, 27 Hz), T7 (4-6, 12-20, 22 Hz), C3 (13, 20, 24 Hz), and P7 (4-6, 13, 15, 22-24, 30 Hz). While at the Bonferroni corrected level, only one statistically significant outcome was observed, namely from C3 in the 13 Hz alpha rhythm.

Apart from Figure 8.6, Figure 8.7 indicates the 900 MHz and 1800 MHz results for the right-side of the head (contralateral to exposure). Statistically significant increases as for the 900 MHz exposure at the $p<0.05$ level were apparent at F4 (6, 22-24, 26 Hz), C4 (2-8, 10, 13, 15, 20, 23, 30 Hz), P8 (2-9, 12-15, 17-20, 22-27, 29-31 Hz), P4 (2-5, 13, 23-27, 29-31 Hz), and O2 (2-8, 13, 23-26, 29, 30 Hz). Likewise, for the Bonferroni corrected results ($p<0.0047$) statistically significant increases were observed from P8 (2-7, 12-14, 17, 18, 23, 24, 27, 30, 31 Hz), P4 (23, 24 Hz), and O2 (2-4 Hz). For the corresponding 1800 MHz results, statistically significant increases in spectral power ($p<0.05$) were evident from two locations namely C4 (3, 11-13, 17, 18, 20-31 Hz) and P8 (2-6, 13-15, 17-31 Hz). For the Bonferroni corrected results however significant outcomes were only evident from P8 at each discrete beta rhythm from 22-24 Hz and 26-28 Hz.

In contrast to the EEG results, analysis of heart response and blood pressure did not indicate any significant findings during either RF exposure tested. Table 8.1 indicates the mean and standard deviation of the median heart rate (R-R wave peak) and PTT of each of the five 4-minute trials (i.e. median of the five mean heart rates and PTTs resulting from the five 4-minute recordings) attained for each exposure mode (N = 100). Additionally a paired t-test analysis of the median samples of both trial measures (Sham vs. Exposure) was applied. The resulting p-value outcomes and corresponding confidence intervals ($\alpha = 0.05$) of these tests are further indicated in Table 8.1 below.

	Heart Rate (R-R wave)	Pulse Transit Time (PTT)
Mean ± SD (Sham)	1.07 ± 0.12 Hz	355.0 ± 43.1 ms
Mean ± SD (900 MHz)	1.07 ± 0.11 Hz	357.7 ± 37.0 ms
Mean ± SD (1800 MHz)	1.07 ± 0.13 Hz	357.3 ± 39.3 ms
95% CI; p-value (900 MHz)	-0.021 to 0.020 Hz; p-value: 0.96	-11.1 to 5.8 ms; p-value: 0.54
95% CI; p-value (1800 MHz)	-0.022 to 0.017 Hz; p-value: 0.96	-10.2 to 5.7 ms; p-value: 0.54

Table 8.1 Shown are the resulting descriptive statistics of the heart rate and PTT analysis, where the 'Mean' represents the average median responses of the five mean heart rates and PTTs resulting from the five 4-minute recordings, as attained for each mode of exposure (N=100). The respective 95% confidence limits and p-values are the result of the paired t-test analysis for mean difference between the Sham and Exposure median samples. P-values were considered statistically significant at $p<0.05$

8.7 Discussion

Apart from the electrode locations P8 and C4 that will be discussed later, one interesting observation as seen in the EEG results is that ipsilateral (left-side) and contralateral (right-side) electrode position results appear to be strikingly similar (Figures 8.6 and 8.7). That is, if the 900 MHz and 1800 MHz results for the recording sites on the left-side of the head (Figure 8.6) are to be superimposed respectively onto the results of the same counter electrode positions on the right-side (in respective exposures)(Figure 8.7), a striking resemblance between the median difference curves as

well as confidence limit plots can be observed. In support of valid trends, this is not the case if the 900 MHz site results are compared with the 1800 MHz site results in the same manner. One notable example of this observation, which may be shown to demonstrate an effect in the alpha band, can be seen if the ipsilateral 900 MHz results provided for P7, P3, and O1 (Figure 8.6) are respectively compared with the contralateral 900 MHz results shown for P8, P4, and O2 (Figure 8.7). For all of these sites, a common EEG spectral power *increasing tendency* in alpha between ~ 8 - 10 Hz (indicated by the 'median difference' curve distinctly falling below the frequency axis), can be seen to be followed by a *decreasing tendency* in EEG power in alpha from ~ 10 – 12 Hz (median difference curve rising towards or above the frequency axis). A resultant *alternating* type characteristic is consequently clearly seen across the alpha band as indicated by the rectangular outlines in both Figures 8.6 and 8.7. Notably, although as a less prominent result, this response can also be observed at 900 MHz at all frontal and temporal regions for both left and right hemispheres (Figures 8.6 and 8.7). This result is notably consistent with results shown at 9 Hz in the pilot investigation (Chapter 3), which demonstrated notable spectral coherence between hemispheres in the frontal, central, and occipital regions as indicated by decreased spectral power in differential recordings. However, as these outcomes in the present study are clearly noticeable from almost each recording site but not necessarily observable to be statistically significant, it is proposed that only a notable proportion of the total population ($N = 100$) may be producing this response.

One speculated explanation for the alternating response observed in alpha, is that a shift of the peak oscillatory power within the band is occurring during the 900 MHz

exposures. More specifically, it appears that a peak shift from approximately 11 Hz to the 9 Hz rhythm is occurring. Although to our knowledge such an effect has not been remarked upon, indications of mobile phone effects potentially elicited from both ipsilateral and contralateral sites surrounding the head in the alpha band have been far from uncommon [D'Costa et al., 2003a][Curcio et al., 2005][Croft et al., 2002][Hinrikus et al., 2004][Croft et al., 2007][[Vecchio et al., 2007]. For example in 2005 Curcio et al. [Curcio et al., 2005], reported on the effects of 902 MHz exposures pulsed at 217 Hz (0.25 W output) as observed from the EEG positions Fz (mid-frontal), Cz (mid-central), Pz (mid-parietal), and the temporal sites T7 and T8. The exposure source was a mobile phone placed similarly in the left ear-mouth position and subjects (N = 20; divided in 2 groups) were seated with eyes closed. Results of the investigation particularly indicated statistically significant increases in alpha rhythms 9 Hz and 10 Hz (Cz), and in 11 Hz (Pz). Consequently Curcio et al. hypothesised that 'the current resting EEG recorded on Cz and Pz, are the result of composite activity coming from bilateral areas, and that the possible effect of EMF exposure could be induced by a kind of spreading of RF power across the brain'. Likewise, in a specific investigation of the alpha band Croft et al. [Croft et al., 2007][Hamblin et al., 2006] (N = 120) indicated that regional spectral power changes across alpha (8 – 12 Hz) occurred in both ipsilateral and contralateral hemispheres, and in further similarity to this study, demonstrated that increases were only present in a proportion of the participants (60%) exposed at rest (895 Hz pulsed at 217 Hz; 2 W peak). Resultantly, Croft et al. suggest that 'the effect of the phone is not homogeneous, and that it may be related to individual differences between the participants'.

Apart from described 900 MHz outcomes, results as provided for the 1800 MHz exposure trials did not indicate any common abnormalities despite consistently similar graphical responses as earlier noted between hemispheres. For example at 1800 MHz the ipsilateral site O1 (Figure 8.6) and the counter contralateral site position O2 (Figure 8.7) both indicated median difference plots randomly near zero microvolts, demonstrating no irregular differences between sham and exposure. Corresponding upper and lower confidence limit plots also appeared to correctly peak (within the alpha band), demonstrating the expectedly high spectral power difference variability for the eyes closed condition in occipital sites.

Aside from indicated consistencies between hemispheres, predominant outcomes in the EEG were observed over specific brain regions such as for the P8 contralateral position, where an unusual amount of statistically significant increases in EEG spectral power were observed for both exposures. To outline, at 900 MHz, increase findings were noted in all four EEG bands in up to 25 ($p<0.05$) and 16 ($p<0.0047$) of the 31 rhythms investigated, while at 1800 MHz significant increases were indicated at P8 in up to 23 ($p<0.05$) and 6 ($p<0.0047$) of the total 31 spectral responses. The statistically significant results for P8 are best represented by the highlighted rhythms as shown in the reproductions of Figure 8.7 below (Figure 8.8) (i.e. where corresponding upper confidence limits are observed to fall below the frequency axis).

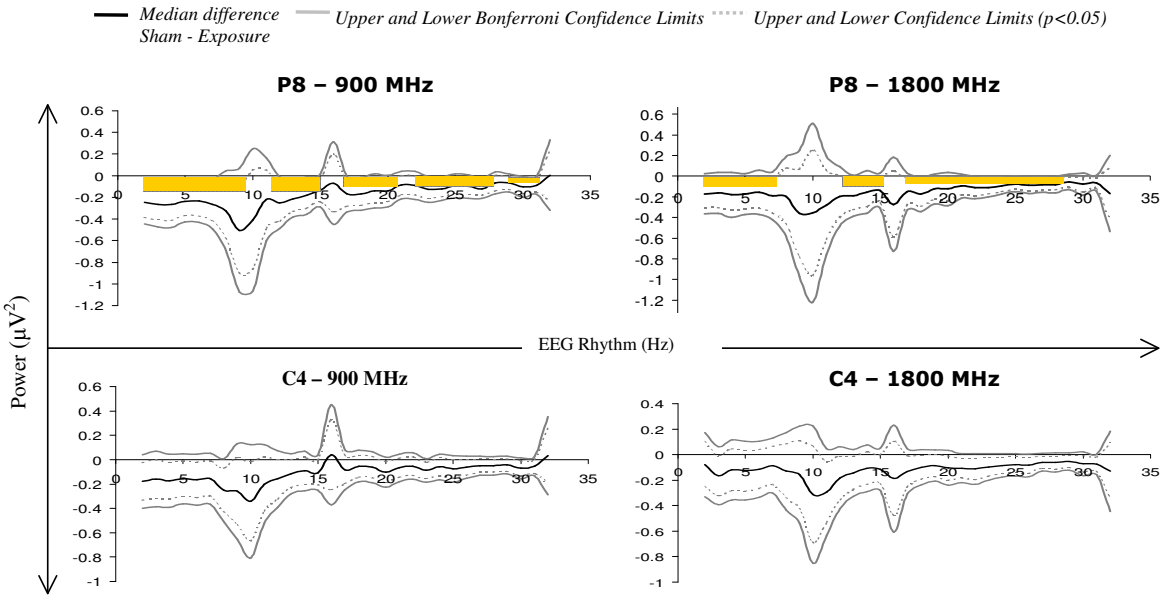


Figure 8.8 Shown are reproductions of the results of Figures 6 and 7 for the contralateral positions P8 and C4 at 900 MHz and 1800 MHz. At P8 in particular, an unusual amount of statistically significant increases in EEG spectral power could be noted for both exposures as highlighted (i.e. where corresponding upper confidence limits are observed to fall below the frequency axis). In addition, the neighbouring central position C4 indicates a strikingly similar median difference response to P8 (in respective exposures only), with corresponding confidence limits indicating similar high tendencies towards EEG power increases across the 2 –32 Hz spectrum.

Although not indicating as many statistically significant trends, the P8 neighbouring central position C4 also indicated unusually high tendencies toward EEG spectral power increases across the 2 – 32 Hz spectrum. Strikingly, the results for C4 can be observed to indicate very similar median difference trends to P8 for both 900 MHz and 1800 MHz exposures, as well as similar upper and lower confidence limit graphs (Figure 8.8), suggesting a related effect at both sites. Moreover, unlike all other recorded sites, both C4 and P8 results are notably dissimilar to their ipsilateral counter side positions C3 and P7, though appear to represent a diminished and distorted form of them for both exposure trials (Figures 8.6 and 8.7). Accordingly, it appears that there is one effect that statistically significantly increases spectral power over many rhythms mainly at C4 and P8 (at 900 MHz and 1800 MHz), and another independent effect that

as earlier described appears to modulate all sites at 900 MHz i.e. the effect noted across the alpha band.

In line with similar changes between neighbouring recorded sites, commonly high statistically significant increase effects in the contralateral parieto-occipital region (O1, O2, P4, P8) may be particularly noted across the delta band (2 – 4 Hz) at 900 MHz (Figure 8.4). In contrast to other studies however, which have indicated comparable experimental conditions to the present investigation, prominent contralateral side effects have not been noted [Röschke et al., 1997][Hietanen et al., 2000]. However, as it is safely assumed that these two particular studies by Hietenan et al. and Röschke and Mann implemented significantly lower SAR levels (as based upon reported exposure source details and position), it is suggested that relatively higher SAR levels as incorporated into this investigation may possibly account for described increase trends in spectral power noted on the contralateral side (1.56 W/kg at 900 MHz; 1.4 W/kg at 1800 MHz). Notwithstanding this however, it is speculated that the alternating median difference response across alpha band as described is not due to the SAR level but rather the 900 MHz carrier signal. This view is deduced on basis that the same effect is not observable at 1800 MHz (as tested at a comparable SAR and pulse modulation) and that other studies have indicated possibly related alpha effects at 900 MHz at much lower SAR levels [D'Costa et al., 2003a][Curcio et al., 2005][Croft et al., 2007][Vecchio et al., 2007]. Notably however, this conjecture opposes hypothesis by Regel et al. [Regel et al., 2007] who over 'post-exposure' waking EEG trials demonstrated that the GSM 'pulse-modulating components' as opposed to continuous wave (CW) signals tested may be responsible for resulting increase and decrease

spectral power changes from 10.5 - 12 Hz as indicated over time (900 MHz; peak SAR: 1 W/g over 10 g).

One final important note in results of the EEG were tendencies clearly seen peaking at both 900 MHz and 1800 MHz for *all* sites at '16 Hz', though this effect is oddly apart from F4 (900 MHz) and F8 (1800 MHz) (see confidence interval diagrams – Figures 8.6 and 8.7). Most importantly however, at 16 Hz only a 'decreasing' tendency can be noted at 900 MHz and only an 'increasing' tendency can be noted at 1800 MHz. While not understood, because these tendencies appear to oppose one another as depending upon exposure at similar SAR levels and pulse, the RF carrier component of these exposures is strongly speculated to be responsible for the amplitude modulation of this beta rhythm.

Apart from investigation of RF effects on the EEG, heart function results were also noted (Table 8.1). For these results, in both consideration of statistical strength (N = 100) and statistically improbable differences indicated between sham and exposure for both heart rate ($p = 0.96$) and PTT ($p = 0.54$), it is speculated that there are no effects of either the pulsed 900 MHz or 1800 MHz exposures on heart rate, BPV, or heart rate variability (HRV) [Atlasz et al., 2006]. This result is in accordance with Tahvanainen et al. [Tahvanainen et al., 2004] who reported no statistically significant changes due to similar exposures of 900 MHz (1.58 W/kg) and 1800 MHz (0.7 W/kg) in measures of heart rate and blood pressure (using a sphygmomanometer) in 35-minute trials (N = 32). Aside from noted non-significant changes, in the present investigation a slight increase tendency (~2.5 ms) in the mean PTT was also observed for both exposures tested (Table 8.1), suggesting in consideration of the outlined confidence limits that a

possible *decrease* in blood pressure may occur in a low proportion of participants. In amongst similar studies, this outcome may be most closely interpreted in line with Atlasz et al. [Atlasz et al., 2006] who in a pilot study also indicated that a minor proportion of the population may express distinct sensitivity in HRV to RF exposures (local max. SAR: 1.3 W/kg).

8.8 Conclusion

There are two important points that should be raised if effects as seen in the alpha band of this investigation are to be replicated:

1. It is proposed to avoid averaging spectral power over the entire alpha band (8 – 13 Hz) during analysis, particularly because *increase* tendencies (8-10 Hz) and smaller *decrease* tendencies (10 –13 Hz) as observed will presumably cancel or possibly appear falsely as a smaller remaining increased tendency.
2. It is proposed to keep eyes closed during trials, particularly as the eyes 'open' condition is known to naturally diminish alpha spectral power [Fisch, 1999], which could likely scale down an already subtle effect.

SAR level, is not speculated to be the cause of the observed alpha effect however, as discussed, it may be critical in causing amplitude modulated effects over to the contralateral region to exposure. However, in consideration that the alternating median difference effect as observed across alpha can be most clearly identified at sites closest to the radiating antenna for 900 MHz exposures (i.e. at P7, P3, O1), the SAR level is speculated to be a factor which may enhance the effect. In support of this, our past

findings and other related accounts have provided evidence of more pronounced effects closer to the radiating source [D'Costa et al., 2003a][Curcio 2005][Croft et al., 2007].

In summary, according to the present results both RF components of 900 MHz and 1800 MHz appear to be critical in inducing common modulated responses in the resting awake EEG in all surrounding regions of the head. However, these effects are notably more substantial at 900 MHz where a clear characteristic effect can be seen across the alpha band. This effect is speculated to be due to the 900 MHz carrier signal, which as outlined may cause a spectral shift in the alpha peak. Effects due to the 217 Hz pulse modulating component cannot be supported by this investigation. Apart from alpha band effects, relatively higher SAR levels of exposure were shown to potentially induce significant increases in the resting EEG spectral power at mid-posterior contralateral sites to exposure by up to 5 μV^2. However in contrast, investigation of resting ECG and blood pressure indicators did not appear to be significantly influenced by the pulsed 900 MHz or 1800 MHz exposures, suggesting that autonomic nervous responses regulating heart and arterial vasoconstriction/vasodilation are not affected during mobile phone use.

In conclusion, the present investigation indicates that there are short-term effects in brain wave activity corresponding to RF carrier signals as used in mobile phone operation. As mechanisms that may produce these RF effects are not presently understood, further research to replicate this study and possibly develop experimental means to clarify the biophysical causes of similarly reported effects is considered vital.

Chapter 9

Conclusions & Future Work

In Chapter 8, several important original findings and observations were determined regarding the effects of both 900 MHz and 1800 MHz mobile phone exposures, particularly on the EEG. These outcomes include:

- An observed characteristic response in the EEG alpha band (8-13 Hz) as due to 900 MHz exposures at almost all recording sites in both hemispheres. Notably, the response did not appear at the 1800 MHz exposure tested. This is the first 'characteristic' response noted in amongst EEG studies reporting on effects of mobile phone exposures
- An EEG effect due to both 900 MHz and 1800 MHz exposures notably appearing at 16 Hz from almost all recording sites. Most importantly, at this beta rhythm only *decrease* tendencies in EEG power can be noted as due to the 900 MHz exposure, and only *increase* tendencies in EEG power can be noted as due to the 1800 MHz exposure
- Effects only due to the 'RF' components of 900 MHz and 1800 MHz appear to influence the EEG, and not the 217 Hz pulse component

- Effects at 900 MHz on the contralateral side to exposure, namely the central, temporal, parietal sites appear to indicate a common median difference response between sham and exposure EEG spectral power, which is not evident on the ipsilateral side to exposure. This case demonstrates that a likely 'neighbouring' effect may be occurring across the contralateral region. In addition, particularly in the temporo-parietal contralateral region, both 900 MHz and 1800 MHz results only appear to indicate statistically significant '*increases'* in EEG power which span in up to 25 of the 31 EEG rhythms investigated (including delta, theta, alpha, and beta rhythms)
- 900 MHz or 1800 MHz exposures *do not* appear to alter heart rate, heart rate variability (HRV), or blood pressure variability (BPV). This result appears to be consistent with most previously published research as indicated for heart and blood pressure responses

In addition to this list of main outcomes the original research questions as outlined in Chapter 2 (Literature Review) are addressed:

Research question 1 required to determine whether 1800 MHz mobile phone exposures could influence the EEG, ECG, or blood pressure measurements - Accordingly, it was determined that 1800 MHz exposures may importantly effect the EEG in particular increasing spectral power in the 16 Hz beta rhythm and furthermore increasing contralateral site EEG spectral power in central to posterior regions. However, heart and blood pressure responses as investigated in terms of heart rate, HRV, and BPV did not appear likely to be affected at this exposure.

Research question 2 required to determine whether the position of a mobile phone corresponds to an influence in brain wave activity at adjacent recording sites – Accordingly, it was found in both the pilot study (Chapter 3) and the main experimental work (Chapter 8) that effects as noted in the alpha band did indeed appear strongest in EEG recording sites nearest to 900 MHz exposure sources, however the same effect did not occur for the 1800 MHz exposure tested. Consequently, as both 900 MHz and 1800 MHz exposures were tested at the same pulsed frequency of 217 Hz as well as similar SAR levels it is speculated that effects appearing strongest nearest to the exposure source may be implicated by the 900 MHz RF component alone.

Research question 3 required to determine whether there were any significant changes in the EEG occurring over large sample sizes for the awake 'closed eyes' condition when the heads of test participants are placed within the near-field of a mobile phone exposure source – Accordingly, for the experiments conducted in the main study (Chapter 8), the experimental handset was positioned in an ear-to-mouth position, placing the heads of 100 awake participants with eyes closed within the reactive and radiating near-field distance from the mobile phone exposure source. As outlined in the summary of important findings above, significant changes did occur in the EEG as seen in all clinically significant bands particularly at the contralateral posterior regions to exposure.

Research question 4 required to specifically determine whether mobile phone exposures cause statistically significant power increases or decreases in discrete EEG rhythms (rather than the clinically significant bands alone) over the distinct spectral range of 1 – 32 Hz – Accordingly, in both the pilot study and the main investigation statistical analysis was conducted in 1 Hz increments across a ~1 – 32 Hz spectrum. This analysis

was undertaken within a developed statistical approach as described in greater detail in Chapter 7. One of the main results as recognised as being seen due to this approach, is the finding of a 'characteristic' median difference response between sham and exposure in the EEG alpha band (8-13 Hz) as observed from almost all recording locations at 900 MHz. This effect is an original finding and has not been identified by others yet it seems to be strongly in accordance with speculative claims regarding effects in the alpha band spectral range. Another important finding was also that of spectral power increases or decreases at the 16 Hz beta rhythm. However, what is interesting here is that only spectral power 'decreases' at 16 Hz occurred at 900 MHz while only 'increases' occurred at 1800 MHz. This effect is seen for both exposures in 15 of the 16 electrode sites investigated. This finding is particularly attributed to the graphical representations of the confidence intervals which appear to allow for this potential effect at 16 Hz to be uncovered.

Research question 5 required to determine whether 900 MHz and/or 1800 MHz RF exposures close to the maximum allowable SAR limit for mobile phone usage influences brain wave activity (EEG) or heart responses – Accordingly, in the major study (Chapter 8) the SAR level of the experimental handset was tuned to be close to the maximum allowable limit of 2 W/kg over any 10 gram of tissue (considering an uncertainty of ±26.6 % in test measurements). Notably, considering that no other comparable study has investigated SAR levels at this magnitude, it is speculated that the higher SAR levels may be responsible for the unique findings of common statistically significant increases and trends observed in contralateral neighbouring regions to the exposure source.

Research question 6 required to determine whether there is a *short-term* influence in the EEG, ECG or blood pressure *during* RF exposures from mobile phones – Accordingly, experimental protocol was particularly arranged in the main study to test short-term exposure periods during EEG, ECG, and PPG measurements. Specifically, short-term periods did not exceed durations of 5-minutes, in any given trial. Consequently, it is important to clearly note that all results provided by the experimental outcomes of this research are concerned only with short-term effects of either 900 MHz or 1800 MHz mobile phone exposures.

Research question 7 required to determine whether 900 MHz or 1800 MHz mobile phone exposures are shielded by, or coupled to, EEG electrode recording leads to an extent where the peak spatial-average SAR or its location inside the head is considerably influenced – Accordingly, to address this point both practical and computational estimates of SAR (from exposures of the experimental handset) were determined in human head models with positioned electrode leads. Main and novel outcomes of these investigations indicated that the peak spatial-average SAR may reduce or increase due the presence of the recording leads depending upon lead numbers and positioning as well as the length of the leads falling behind the head of test participants. Nevertheless, noted changes in the SAR and electric-field distribution inside the head did not appear to be significant enough to reduce or increase levels to extents beyond expected magnitudes of mobile phone exposures.

Research question 8 required to determine whether there are any corresponding effects in brain wave activity and heart responses such as between measures of the EEG and ECG due to mobile phone exposures – Accordingly, it may be noted that due to unlikely

effects occurring due to mobile phone exposures in measures of the ECG and blood pressure response, there does not appear to be potential for correlating effects in heart response to the measured effects observed in brain wave activity.

In conclusion, in regards to future work, several major points of recommendation are suggested:

1. A very limited amount of studies have been conducted on children to investigate mobile phone exposure effects on brain and heart activity. Although it is understood that the brain response is not fully developed until adulthood, there appears to be no real physiological restriction in examining younger participants of the same age, for example, 50 male 13 yr olds. Examining children for such effects is crucial particularly as it is possible that maturing brain at earlier ages could be more susceptible to adverse health consequences
2. Investigation of longer duration times of continuous mobile phone exposures on test participants at rest is in need to be undertaken in both brain and heart investigations. The present body of literature at best generally indicates outcomes of 30-minute continuous mobile phone exposures. While these duration times are absolutely adequate for trials it would be interesting also to see the effects of mobile phone exposures on brain and heart activity at durations up to one hour or more. In such a study, it would be recommended that vigilance activities such as auditory or visual tasks would need to be investigated and wisely incorporated during exposure times, in order to sustain consistencies in recorded brain and heart data

3. Based on the results contained within this thesis, the EEG rhythm 16 Hz is strongly outlined as a point of new interest for susceptibility to phone exposures. Consequently, it is recommended that the spectral power density within this band be robustly quantified in any future research. In analysis, care should be taken not to just consider grand-averaging across EEG bands, which as explicitly indicated in Chapter 7, can most easily mask the potential RF effect at 16 Hz
4. The alpha band in EEG studies is still raising ongoing concerns that a change is present in brain waves due to various mobile phone exposures. Future work should focus on incorporating a rigorous high standard of statistical analysis, not yet consistently observed. Replication of the statistical analysis proposed in this thesis is highly recommended
5. Finally, a call for better RF dosimetry, larger sample sizes (at least 50 participants), and double blinding, are all critical factors that need to be incorporated into future research to improve on the quality and integrity of outcomes in both brain and heart research. Observation of Tables 2.1 – 2.4 within the thesis highlight these existing inadequacies

References

Adair. 2003. Biophysical limits on athermal effects of RF and microwave radiation. Bioelectromagnetics 24:39-48.

Alcamo E & Krumhardt B. 2004. Anatomy and physiology. Barron's educational series, 2nd ed.

Angelone LM, Potthast A, Segonne F, Iwaki S, Belliveau JW, Bonmassar G. 2004. Metallic electrodes and leads in simultaneous EEG-MRI: Specific Absorption Rate (SAR) simulation studies. Bioelectromagnetics 25(4):285-295.

ARPANSA Radiation Protection Standard: Maximum exposure levels to radiofrequency fields – 3 kHz to 300 GHz (RPS-3). 2002.

Atlasz T, Kellenyi L, Kovacs P, Babai N, Thuroczy G, Hejjel L, Hernadi I. 2006. The application of surface plethysmography for heart rate variability analysis after GSM radiofrequency exposure. J Biochem Biophys Methods 69:233-236.

Australian Communications (and Media) Authority, ACA: Radiocommunications (Electromagnetic Radiation – Human Exposure) Standard, 2003.

Bahr A, Dorn H, Bolz T. 2006. Dosimetric assessment of an exposure system for simulating GSM and WCDMA mobile phone usage. Bioelectromagnetics 27:320-327.

Barnes FS, Greenebaum B. 2007. Handbook of biological effects of electromagnetic fields: Biological and medical aspects of electromagnetic fields. 3rd edition, Taylor & Francis Group: 168-179.

Beard BB, Kainz W. 2006. Comparisons of computed mobile phone induced SAR in the SAM phantom to that in anatomically correct models of the human head. IEEE Trans on EMC 48(2):397-407.

Bland JM, Altman DG. 1995. Multiple significance tests: The Bonferroni method. BMJ 310:170.

Borbely AA, Huber R, Graf T, Fuchs B, Gallmann E, Achermann P. 1999. Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. Neurosci Lett 275:207–210.

Bramwell JC, Hill AV. 1922. Velocity of transmission of the pulse wave and elasticity of arteries. Lancet 199:891-892.

Braune S, Riedel A, Schulte-Monting J, Raczek J. 2002. Influence of a radiofrequency electromagnetic field on cardiovascular and hormonal parameters of the autonomic nervous system in healthy individuals. Radiat Res 158:352–356.

Braune S, Wrocklage C, Raczek J, Gailus T, Lucking CH. 1998. Resting blood pressure increase during exposure to a radio-frequency electromagnetic field. Lancet 351:1857–1858.

Carpenter DO, Ayrapetyan, S. 1994. Biological effects of electric and magnetic fields – sources and mechanisms. Academic Press, Inc. San Diego, California. V.1 pg. 57

Cheng, DK. Field and Wave Electromagnetics. 2nd ed. Addison Wesley. 1989

Carr JJ, Brown JM. 1998. Introduction to biomedical equipment technology, 3rd ed., Prentice Hall, New Jersey: 376.

CENELEC. 2001. "Basic Standard for the Measurement of Specific Absorption Rate Related to Human Exposure to Electromagnetic Fields from Mobile Phones (300 MHz – 3 GHz)," Tech. Rep. EN 50 361.

Croft RJ, Chandler JS, Burgess AP, Barry RJ, Williams JD, Clarke AR. 2002. Acute mobile phone operation affects neural function in humans. Clin Neurophysiol 113:1623–1632.

Croft RJ, Hamblin DL, Spong J, Wood AW, McKenzie RJ, Stough C. 2007. The effect of mobile phone electromagnetic fields on the alpha rhythm of human electroencephalogram. Bioelectromagnetics 29(1):1-10.

Curcio G, Ferrara M, Moroni F, D'Inzeo G, Bertini M, De Gennaro L. 2005. Is the brain influenced by a phone call? An EEG study of resting wakefulness. Neurosci Res 53:265–270.

Curtin F, Schulz P. Multiple comparisons and Bonferroni's correction. 1998. Society of Bio Psych. 44:775-777.

D'Costa H, Anderson V, Cosic I. 2003. Development of a test handset to better assess radiofrequency bioeffects from mobile phone exposures. Proceeds of the 28th Annual ARPS conference, Australia. Abstract only.

D'Costa H, Anderson V, Hamblin DL, McKenzie R, Cosic I. 2004. Effects of EEG electrode leads on the specific absorption rate of radiofrequency exposures from mobile phones. Proceeds of the 3rd Int conference on biological effects of EMFs, Greece 2:659-663.

D'Costa H, Trueman G, Tang L, Abdel-Rahman U, Abdel-Rahman W, Ong K, Cosic I. 2003. Human brain wave activity during exposure to radiofrequency field emissions from mobile phones. Aust Phys Eng Sci Med 26:162–167.

ETSI. 1999. ETS 300 578 standard: GSM 05.08 version 4.22.1, 13th ed., 22.
Feise RJ. 2002. Do multiple outcome measure require p-value adjustment? BMC Med research methodology 2(8).

Fisch BJ. 1999. Fisch and Spehlmann's EEG Primer: Basic Principles of Digital and Analogue EEG, 3rd edn. Amster-dam, North Holland: Elsevier BV.

Freude G, Ullsperger P, Eggert S, Ruppe I. 1998. Effects of microwaves emitted by cellular phones on human slow brain potentials. Bioelectromagnetics 19:384–387.

Freude G, Ullsperger P, Eggert S, Ruppe I. 2000. Microwaves emitted by cellular telephones affect human slow brain potentials. Eur J Appl Physiol 81(1):18-27.

Fritzer G, Göder R, Friege L, Wachter J, Hansen V, Hinze-Selch D, Aldenhoff B. 2007. Effects of short- and long- term pulsed radiofrequency electromagnetic fields on night sleep and cognitive functions in healthy subjects. Bioelectromagnetics 28:316-325.

Gabriel S,Lau RW,Gabriel C. 1996. The dielectric properties of biological tissues: III. Parametric models for the dielectric spectrum of tissues. Phys Med Biol 41:2271-2293.

Gandhi OP, Li Q-X, Kang G. 2001. Temperature rise for the human head for cellular telephones and for peak SARs prescribed. IEEE Trans on MW theory & techniques 49(9):1607-1613.

Goldberger E. A simple, indifferent, electrocardiographic electrode of zero potential and a technique of obtaining augmented, unipolar, extremity leads. Am Heart J 1942;23:483–92.

Gribbin, Steptoe, Sleight. 1976. Pulse wave velocity as a measure of blood pressure change. Psychophys 13(1):86-90.

Haarala C, Aalto S, Hautzel H, Julkunen L, Rinne JO, Laine M, Krause B, Hamalainen H. 2003. Effects of a 902 MHz mobile phone on cerebral blood flow in humans: A PET study. NeuroReport 14:2019–2023.

Haarala C, Ek M, Björnberg L, Laine M, Revonsuo A, Koivisto M, Hämäläinen H. 2004. 902 MHz mobile phone does not affect short-term memory in humans. Bioelectromagnetics 25:452–456.

Hamblin DL, Croft RJ, Wood AW, Stough C, Spong J. 2006. The sensitivity of human event-related potentials and reaction time to mobile phone emitted electromagnetic fields. Bioelectromagnetics 27:265–273.

Hamblin DL, Wood AW, Croft RJ, Stough C. 2004. Examining the effects of electromagnetic fields emitted by GSM mobile phones on human event-related potentials and performance during an auditory task. Clin Neurophys 115:171-178.

Hamblin DL, Anderson V, McIntosh RL, McKenzie RJ, Wood AW, Iskra S, Croft RJ. 2007. EEG electrode caps can reduce SAR induced in the head by GSM900 mobile phones. IEEE Trans 54(5):914-920.

Heath, B., Jenvey, S., and I. Cosic. 1998. Investigation of analogue and digital mobile phone low frequency radiation spectrum characteristics. 2nd Int Conference on Bioelectromagnetism. 83-84.

Herrmann C, Demiralp T. 2005. Human EEG gamma oscillations in neuropsychiatric disorders. Clin Neurophys 116(12): 2719–2733.

Hietanen M, Hamalainen A-M, Husman T. 2002. Hypersensitivity symptoms associated with exposure to cellular telephones: No casual link. Bioelectromagnetics 23:264-270.

Hietanen M, Kovala T, Hamalainen AM. 2000. Human brain activity during exposure to radiofrequency fields emitted by cellular phones. Scand J Work Environ Health 26(2):87–92.

Hinrichs H, Heinze HJ, Rotte M. 2005. Human sleep under the influence of a GSM 1800 electromagnetic far field. Somnology. 9(4):185-191.

Hinrikus H, Parts M, Lass J, Tuulik V. 2004. Changes in human EEG caused by low level modulated microwave stimulation. Bioelectromagnetics 25:431–440.

Hinrikus H, Bachmann M, Lass J, Tomson R, Tuulik V. 2008. Effect of 7, 14 and 21 Hz modulated 450 MHz microwave radiation on human electroencephalographic rhythms. Int J Radiat Biol. 84(1):69-79.

Hinrikus H, Bachmann M, Lass J, Deniss K, Tuulik V. 2008. Effect of low frequency modulated microwave exposure on human EEG: Individual sensitivity. Bioelectromagnetics 29:527-538.

Hirata A, Shiozawa T. 2003. Correlation of maximum temperature increase and peak SAR in the human head due to handset antennas. IEEE Trans on MW theory & techniques 51(7):1834-1841.

Huber R, Graf T, Cote KA, Wittmann L, Gallmann E, Matter D, Schuderer J, Kuster N, Borbely AA, Achermann P. 2000. Exposure to pulsed high-frequency electromagnetic field during waking affects human sleep EEG. NeuroReport 11:3321–3325.

Huber R, Schuderer J, Graf T, Jutz K, Borbely A, Kuster N, Achermann P. 2003. Radio frequency electromagnetic field exposure in humans: Estimation of SAR distribution in the brain, effects on sleep and heart rate. Bioelectromagnetics 24:262-276.

ICNIRP. 1998. Guidelines for limiting exposure to time varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Physics 74:494-522.

IEEE P1528: Recommended Practice for Determining the Peak Spatial-Average Specific Absorption Rate (SAR) in the Human Head from Wireless Communications Devices. Measurement Techniques, 2003.

Jasper, HH. 1958. The ten-twenty electrode system of the International Federation. Electroencephal Clin Neurophysiology 10:1426–1437.

Jech R, Sonka K, Ruzicka E, Nebuzelsky A, Böhm J, Juklickova M, Nevsimalova S. 2001. Electromagnetic field of mobile phones affects visual event related potential in patients with narcolepsy. Bioelectromagnetics 22:519–28.

Kleinlogel H, Dierks T, Koenig T, Lehmann H, Minder A, Berz R. 2008. Effects of weak mobile phone-electromagnetic fields (GSM, UMTS) on well-being and resting EEG. Bioelectromagnetics 29:479–487.

Kleinlogel H, Dierks T, Koenig T, Lehmann H, Minder A, Berz R. 2008. Effects of weak mobile phone-electromagnetic fields (GSM, UMTS) on event-related potentials and cognitive functions. Bioelectromagnetics 29:488–497.

Kivekäs O, Ollikainen J. 2004. Bandwidth, SAR, and efficiency of internal mobile phone antennas. IEEE Trans on EMC 46(1):71-86.

Kramarenko AV, Tan U. 2003. Effects of high-frequency electro-magnetic fields on human EEG: A brain mapping study. Int J Neurosci 113:1007–1019.

Krause C, Pesonen M, Björnberg CH, Hämäläinen H. 2007. Effects of pulsed and continuous wave 902 MHz mobile phone exposure on brain oscillatory activity during congnitive processing. Bioelectromagnetics 28:296-308.

Krause CM, Bjornberg CH, Pesonen M, Hulten A, Liesivuori T, Koivisto M, Revonsuo A, Laine M, Hämäläinen H. 2006. Mobile phone effects on children's event-related oscillatory EEG during an auditory memory task. Int J Radiat Biol 82:443–450.

Krause CM, Haarala C, Sillanmäki L, Koivisto M, Alanko K, Revonsuo A, Laine M, Hämäläinen H. 2004. Effects of electromagnetic field emitted by cellular phones on the EEG during an auditory memory task: A double blind replication study. Bioelectromagnetics 25:33–40.

Krause CM, Sillanmäki L, Koivisto M, Häggqvist A, Saarela C, Revonsuo A, Laine M, Hämäläinen H. 2000a. Effects of Electromagnetic field emitted by cellular phones on the electroencephalogram during a visual working memory task. Int J Radiat Biol 76:1659–1667.

Krause CM, Sillanmäki L, Koivisto M, Häggqvist A, Saarela C, Revonsuo A, Laine M, Hämäläinen H. 2000b. Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task. Neuro Report 11:761–764.

Lass J, Kruusing K, Hinrikus H. 2008. Modulated low-level electromagnetic field effects on EEG visual event-related potentials. Est J of Eng 14(2):124-137.

Ma T, Zhang YT. 2005. A correlation study on the variabilities in pulse transit time, blood pressure, and heart rate recorded simultaneously from healthy subjects. IEEE-EMBS 27th Annual Int Conference, Shanghai 996-999.

MacKenzie IS, Wilkinson IB, Cockcroft JR. 2002. Assessment of arterial stiffness in clinical practice. Q J Med 95:67-74.

Mann K, Röschke J. 1996. Effects of pulsed high-frequency electromagnetic fields on human sleep. Neuropsychobiology 33:41–47.

Mann K, Wagner P, Brunn G, Hassan F, Hiemke C, Röschke J. 1998. Effects of pulsed high-frequency electromagnetic fields on the neuroendocrine system. Neuroendocrinology 67:139–144.

Meyer FJC, Palmer KD, Jakobus U. 2001. Investigation into the Accuracy, Efficiency and Applicability of the Method of Moments as Numerical Dosimetry Tool for the Head and Hand of a Mobile Phone User. Applied Comput Electromagnetics Soc 16(2):114-125.

Mochizuki S, Watanabe S, Taki M, Yamanaka Y, Shirai H. 2004. Size of head phantoms for standard measurements of SAR due to wireless communication devices. Elec Comms Japan 87(4):82-91.

Mochizuki H, Ugawa Y. 2005. High-frequency oscillations in somatosensory system. Clin EEG Neurosc 36(4):278–284.

Niedermeyer E, Lopes Da Silva FH. Electroencephalography: basic principles, clinical applications and related fields. 1993. 3rd edition: 168.

Parazzini M, Ravazzani P, Tognola G, Thuroczy G, Molnar FB, Sacchettini A, Ardesi G, Mainardi LT. 2007. Electromagnetic fields produced by GSM cellular phones and heart rate variability. Bioelectromagnetics 28:122-129.

Preece AW, Iwi G, Davies-Smith A, Wesnes K, Butler S, Lim E, Varey A. 1999. Effect of a 915 MHz simulated mobile phone signal on cognitive function in man. Int J Radiat Biol 75(4):447-456.

Rautaharju P, Rautaharju F. 2007. Investigative Electrocardiography in Epidemiological Studies and Clinical Trials. Springer London Pg: 18-28.

Regel S, Gottselig JM, Schuderer J, Tinguely G, Retey J, Kuster N, Landolt H-P, Achermann P. 2007. Pulsed radio frequency radiation affects cognitive performance and the waking electroencephalogram. Neuroreport 18(8):803-807.

Reiser HP, Dimpfel W, Schober F. 1995. The influence of electromagnetic fields on human brain activity. Eur J Med Res 1:27–32.

Röschke J, Mann K. 1997. No short term effects of digital mobile radio telephone on the awake human electroencephalogram. Bioelectromagnetics 18:172–176.

Rowley, JT. 1999. Investigation of mobile phone handset antennas in the presence of the head and development of novel integrated mobile terminal antennas. Dep of Comms & Elect Eng, RMIT University. Thesis publication.

Russo R, Fox E, Cinel C, Boldini A, Defeyter M, Mirshekar-Syahkal D, Mehta A. 2006. Does acute exposure to mobile phones affect human attention? Bioelectromagnetics 27:215-220.

Sankoh A, Huque M, Dubey S. 1997. Some comments on frequently used multiple endpoint adjustment methods in clinical trials. Stats in Med. 16:2529-2542.

Schmid G, Cecil S, Goger C, Trimmel M, Kuster N, Molla-Djafari H. 2007. New head exposure system for use in human provocation studies with EEG recording during GSM900- and UMTS-like exposure. Bioelectromagnetics 28(8):636-647.

Schönborn F, Burkhardt M, Kuster N. 1998. Differences in energy absorption between heads of adults and children in the near field of sources. Health Phys 74:160-168.

Silva LR, Amitai Y, Connors BW. 1991. Intrinsic oscillations of neocortex generated by layer 5 pyramidal neurons. Science 251(4992):432-434).

Smythe J, Costall B. 2003. Mobile phone use facilitates memory in male, but not female, subjects. Neuro Rep 14:243 –246.

Steriade M, McCormick DA, Sejnowski TJ. 1993. Thalamocortical oscillations in the sleeping and aroused brain. Science 262(5134):679 – 685.

Sterman BM. 1996. Physiological origins and functional correlates of EEG rhythmic activities: Implications for self-regulation. Applied Psychophys and Biofeedback 21(1):3-33.

Tahvanainen K, Nino J, Halonen P, Kuusela T, Laitinen T, Lansimies E, Hartikainen J, Hietenen M, Lindholm H. 2004. Cellular phone use does not acutely affect blood pressure or heart rate of humans. Bioelectromagnetics 25:73-83.

Tillmann T, Ernst H, Ebert S, Kuster N, Behnke W, Rittinghausen S, Dasenbrock C. 2006. Bioelectromagnetics Online:1-15.

Vecchio F, Babiloni C, Ferreri F, Curcio G, Fini R, Del Percio C, Rossini PM. 2007. Mobile phone emission modulates interhemispheric functional coupling of EEG alpha rhythms. Eur J of Neuroscience 25:1908-1913.

Von Klitzing, L. 1992. Electromagnetic fields pulsed with 217 Hz alter EEG of man at very low energy levels. 14th Annual Int Conference of the IEEE Eng in Medicine and Bio Soc, Lyon, France. 221-223.

Von Klitzing, L. 1995. Low-frequency pulsed electromagnetic fields influence EEG of man. Physica Medica 11: 77-80.

Wagner P, Röschke J, Mann K, Fell J, Hiller W, Frank C, Grözinger M. 2000. Human sleep EEG under the influence of pulsed radio frequency electromagnetic fields. Neuropsychobiology 42:207–212.

Wagner P, Röschke J, Mann K, Hiller W, Frank C. 1998. Human sleep under the influence of pulsed radiofrequency electromagnetic fields: A polysomnographic study using standardized conditions. Bioelectromagnetics 19:199–202.

Wilson FN, Johnston FD, MacLeod AG, Barker PS. 1934. Electrocardiograms that represent the potential variations of a single electrode. Am Heart J 1934;9:447–58.

Wood AW, Hamblin DL, Croft RJ. 2003. The use of a 'phantom scalp' to assess the possible direct pickup of mobile phone handset emissions by elecroencephalogram electrode leads. Med and Biol Eng & Comput 41:470-472.

Wu K, Sajad A, Omar S, Mackay W. 2009. The effect of high frequency radio waves on human brain activity: an EEG study. J of Undergrad Life Sci 3(1):50-54

Appendix A

List of Publications

A.1 Journal Publications

D'Costa H, Trueman G, Tang L, Abdel-rahman U, Abdel-rahman W, Ong K, Cosic I. 2003. Human brain wave activity during exposure to radiofrequency field emissions from mobile phones. Australas Phys Eng Sci Med 26(4): 162-7.

Journal publication in preparation:

D'Costa H, McKenzie R, Cosic I. 2009. Influence of electromagnetic field exposures from mobile phones on nervous function in the human brain and heart. (will be forwarded to 'Bioelectromagnetics').

Journal publication in preparation:

D'Costa et al. 2009. Modelling the effects of EEG electrode leads on the specific absorption rate of radiofrequency exposures from mobile phones. (will also be forwarded to 'Bioelectromagnetics').

A.2 Full Conference Papers

D'Costa, H, Cosic I. 2005. Statistical Analysis of the Human EEG during RF Exposure from Mobile Phones: An Alternative Method to Analysis of the EEG in Frequency Bands. Biosignal Processing and Classification Workshop Proceedings, 14-17 September, Barcelona, Spain.

D'Costa H, Anderson V, Hamblin DL, McKenzie R, Cosic I. 2004. Effects of EEG electrode leads on the specific absorption rate of radiofrequency exposure from mobile phones, Biological Effects of EMFs, 3rd International Workshop, Kos, Greece.

A.3 Conference Abstracts

D'Costa H, Anderson V, Cosic I. 2003. Development of a test handset to better assess radiofrequency bioeffects from mobile phone exposures. Abstract proceedings of the 28th Annual ARPS conference, Hobart, Australia. pp. 42.