

Fetal and neonatal responses following maternal exposure to mobile phones

Ahmed Y. Rezk, MD, Khaled Abdulqawi, MD, Randa M. Mustafa, MD, Tark M. Abo El-Azm, MD, Hesham Al-Inany, MD.

ABSTRACT

الأهداف: توجد الموجات الكهرومغناطيسية في كل مكان في بيئتنا لكنها غير مرئية لكن يمكن اختبارها، انتشار استخدام التليفون المحمول أثار تساؤل عن إمكانية تأثير الموجات الكهرومغناطيسية على وظائف أعضاء الجسم فهناك العديد من التقارير عن أثار الموجات الكهرومغناطيسية الصادرة من التليفون المحمول في إحداث تغيير بيولوجي للخلايا المتعرضة لها.

الطريقة: صمم البحث كدراسة إكلينيكية مقارنة بمجموعة ضابطة وأجرى في المستشفيات الجامعية بينها وبعض الوحدات العلاجية الأخرى. شملت الدراسة ٩٠ سيدة حامل يحمل غير مصحوب بمشاكل وتتراوح أعمارهن بين ١٨ و٣٣ عاماً وكذلك ٣٠ من مواليدهن المكملي النمو والعمر الجنيني، وتم تعريض الأمهات للموجات الكهرومغناطيسية الصادرة من التليفون المحمول خلال محادثة تستمر ١٠ دقائق أثناء وبعد الولادة وملاحظة الأثار من خلال قياس عدد دقات القلب وكم الدفع الدموي للقلب لكل من الأجنة والمواليد.

النتائج: حدثت زيادة ذات دلالة معنوية في عدد نبض الجنين مع انخفاض لحجم الدم الدافق وكم الدفع الدموي للقلب، لكن تتضاءل هذه الأثار مع تقدم العمر الجنيني.

خاتمة: نستنتج من هذا البحث أن تعرض الأم لمجالات الموجات الكهرومغناطيسية الصادرة من التليفون المحمول لها أثر ذو دلالة معنوية على عدد نبض القلب وكم الدفع الدموي للقلب في الأجنة والمواليد.

Objective: To study fetal and neonatal heart rate (HR) and cardiac output (COP), following acute maternal exposure to electromagnetic fields (EMF) emitted by mobile phones.

Methods: The present study was carried out at Benha University Hospital and El-Shorouq Hospital, Cairo, Egypt, from October 2003 to March 2004. Ninety women with uncomplicated pregnancies aged 18-33 years, and 30 full term healthy newborn infants were included. The pregnant mothers were exposed to EMF

emitted by mobile telephones while on telephone-dialing mode for 10 minutes during pregnancy and after birth. The main outcome were measurements of fetal and neonatal HR and COP.

Results: A statistical significant increase in fetal and neonatal HR, and statistical significant decrease in stroke volume and COP before and after use of mobile phone were noted. All these changes are attenuated with increase in gestational age.

Conclusions: Exposure of pregnant women to mobile phone significantly increase fetal and neonatal HR, and significantly decreased the COP.

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From the Departments of Obstetrics and Gynecology (Rezk), Pediatrics (Abdulqawi), Physiology (Mustafa), and Cardiology (El-Azm), Benha Faculty of Medicine, Zagazig University, and the Department of Obstetrics and Gynecology (Al-Inany), Cairo University, Cairo, Egypt.

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Address correspondence and reprint request to: Dr. Hesham Al-Inany, Department of Obstetrics and Gynecology, Cairo University, 8-Moustapha Hassanin st; Manial, 11451, Cairo, Egypt. Tel. +20 101469022. Fax. +20 013267080. E-mail: kaainih@link.net

Recently, people have become concerned with the safety of wireless communication, such as the cellular phones that are rapidly gaining popularity, and yet little is known on the health hazards of the radio frequency fields of these cellular phones.¹ The dominant access technique in Egypt is the so-called time division multiple access, which is used in the global system for mobile communication system (GSM). The carrier frequency bands allocated for this service are set mainly in the spectrum regions of 800-900 megahertz (MHZ).² There are several reports, which indicate that electromagnetic radiation such as from mobile phones at non-thermal levels may elicit a biological effect in target cells or tissues.³ Electromagnetic fields (EMFs) and their potential effects on human health have been

investigated. They may play a role in a number of disorders, such as depression and memory loss. More recently, neurological, degenerative, and heart diseases have also been reported to be related to such EMFs. Furthermore, the increased use of mobile phones worldwide has focused interest on the possible link to increased risk of leukemias, sleep disturbances, and the more serious brain tumors.⁴ The effects of magnetic field on embryonic mortality in chickens were studied. Embryonic mortality in eggs exposed to the magnetic field during their incubation was higher. The negative effect of magnetic field manifested also by a lower weight of the hatched chicken.⁵ The aim of this work is to study fetal and neonatal heart rate (HR) and cardiac output (COP) following acute maternal exposure to EMF emitted by mobile phones during a telephone-dialing mode for 10 minutes (min) by mothers during pregnancy and after birth.

Methods. The present study was carried out at Benha University Hospital and El-Shorouq Hospital in Cairo, Egypt, on 90 healthy women and 30 healthy newborn infants, from October 2003 to March 2004. Healthy pregnant women aged 18-33 years, (mean age, 24±3years) with uncomplicated pregnancies, healthy full term newborns with no perinatal risk factors including both genders of newborn infants, were included in the study. Exclusion criteria were presence of high risk factors (antepartum, intrapartum, postpartum) and unhealthy premature infants with perinatal risk factors. Ethical approval from the University Hospital was obtained before the study period.

They were classified into 4 main groups: group I included 30 pregnant women with gestational age from 25-30 weeks; group II included 30 pregnant women with gestational age from 31-35 weeks; group III included 30 pregnant women with gestational age from 36-40 weeks; group IV included 30 newborn babies of 90 women. In group I, II, and III, the fetal (F) HR and COP following acute maternal exposure to EMF emitted by mobile phones during a telephone dialing mode for 10 min by mothers during pregnancy were studied: FHR (baseline, acceleration, and deceleration), fetal end diastolic volume (FEDV), fetal end systolic volume (FESV), fetal stroke volume (FSV), and FCOP. In group IV, the neonatal HR and COP following acute maternal exposure to EMF emitted by mobile phones during a telephone dialing mode for 10 min by mothers after birth were studied: neonatal HR, neonatal EDV, neonatal ESV, neonatal SV, and neonatal COP.

Written informed consent was obtained from each participant. All cases from group I, II, and III were subjected to the following: a) clinical history, including age, parity, date of last menstrual period for detection of the gestational age, and application of mobile phone;

b) ultrasonographic examination (Medison 3200, South Korea) to check for fetal lie and presentation, and confirmation of the gestational age using the biparietal diameter and femur length; c) FHR recording, a full 15 min of FHR recording using an electronic FHR monitor (Sonic aid LTD, Model Fm7L, Chichester, England) was performed for all cases.⁶ Women were placed in the semi-recumbent position to avoid supine hypotension, and the procedure was explained to the patients and they were instructed to press the button of a hand-held event marker on the feeling of fetal movements, to be recorded on the trace paper. The abdominal belt was placed around the patient's abdomen and the transducer was positioned on the area of maximum heart pulses (usually on the lower abdomen to one side). Ultrasound gel was applied to the site of application of the transducer (as air is a poor conductor for ultrasound). The Doppler mode was used with a paper speed of one centimeter per min. The FHR recording was performed while the patients were holding a mobile phone of GSM 900 type, at 900 MHZ on the right-hand side of the head in a typical telephoning position on dialing mode, for 10 min.⁷ The recording was successfully completed in all patients for a period of 20 min (10 min without the use of mobile phone, and 10 min with it). There were no other devices that could produce high EMF in the vicinity. The FHR analysis was based on the description of HR patterns regarding baseline HR. d) FCOP recording using Doppler echocardiography (Toshiba SSH-140A color Doppler, continued and pulsed power, Japan) was performed for all cases. Women were examined in the left lateral supine position, and echocardiogram was performed using a 2.5-3.5 MHZ transducer. The M-mode was recorded while the cardiac anatomy was being visualized by 2-dimensional echocardiography.⁸ By M-mode, we measure left ventricular end systolic diameter (LVESD) and left ventricular end diastolic diameter (LVEDD). By measuring the LVESD and LVEDD, we can calculate the left ventricular end systolic volume (LVESV, the smallest left ventricular systolic volume) and the left ventricular end diastolic volume (LVEDV, the largest left ventricular diastolic volume) through equation of Teichhols:⁹

$$V = 7.0 (2.4 + D) \times D^3$$

Where: V = Volume, and D = Diameter

The apparatus is computerized to receive the measured diameters, and transform it into volumes directly. We measured the stroke volume by the equation:

$$SV = LVEDV - LVESV$$

Finally, COP was calculated from the equation:

$$COP = SV \times HR$$

All newborn cases of group IV were subjected to the following: full clinical history including perinatal history; gestational age assessment through clinical examination for all systems, application of mobile phone by their mothers for 10 min, and neonatal COP recording. All neonates were examined in the first 48 hours after delivery, while their mothers are carrying them in the semisitting position. Neonatal COP recording using Doppler echocardiography (Toshiba SSH-140A color Doppler, continued and pulsed power, Japan) was performed for all cases. Patients were examined in the supine position, and sedation was given to the babies at the start of assessment in the form of chloral hydrate syrup in a dose of 25-50 mg/kg/dose peroral, with careful observation for respiratory depression. Echocardiogram was performed using 5 MHZ transducer. The M-mode recordings were carried out while the cardiac anatomy was being visualized by 2-dimensional echocardiography. The fetal and neonatal COP recording was performed while their mothers were holding a mobile phone of GSM 900 type, at 900 MHZ on the right-hand side of the head in a typical telephoning position on dialing mode.

According to the given designed sheet, the collected data and results were fed to a special well-prepared biocomputer program, on IBM computer of the third generation. Statistics were carried out using Arcus Quickstat (Version I). Different outcome measures were compared and statistically analyzed using Student's t-test, and the chi-square test to evaluate significance of results (*p*-value).

Results. In group I, there was a significant statistical change in FHR, EDV, ESV, and SV (*p*<0.001). Also, there is a significant statistical decrease in COP (*p*<0.025) (Table 1). Similar statistical changes were noticed in groups II and III (Tables 2 & 3). Table 4 shows the comparison between the mean±SD in neonatal HR, EDV, ESV, SV, and COP before and after postnatal maternal exposure to EMFs of group IV. There was a significant increase in HR, a significant decrease on EDV and ESV, in SV, and COP was noted. Table 5 showed comparisons of the percentage of change in fetal and neonatal HR, EDV, ESV, SV, and COP before and after exposure to EMFs (use of mobile phone) in group I, II, III, and IV. The increase in FHR resulting from exposure to the mobile phone was attenuated with increase in the gestational age, as it was 13.1% at gestational age (25-30) weeks, and 7.3% at gestational age (31-35) weeks. At gestational age (36-40) weeks, there was further decrease to 5.9%, while in group IV, there was a further decrease in neonatal HR to 4.3%. There was a reduction in the percentage of decrease in EDV as the gestational age increases. The percentage of

Table 1 - Changes in fetal heart rate (FHR), end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), and cardiac output (COP) before and after use of mobile phone, in pregnant women of gestational age ranged 25-30 weeks (Group I).

Variables (n = 30)	Before	After	t	P-value
	Mean ± SD			
FHR (bpm)	124.3±12.9	140.6±12.6	2.539068	<0.011
EDV (ml)	9.1±1.3	6.3±1.0	2.56732	<0.0003
ESV (ml)	3.1±0.5	2.5±0.47	1.293742	<0.02
SV (ml)	5.8±1.2	3.7±1.0	1.929875	<0.001
COP (ml/min)	721.3±175.3	528±172.1	2.197321	<0.025

Table 2 - Changes in fetal heart rate (FHR), end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), and cardiac output (COP) before and after use of mobile phone, in pregnant women of gestational age ranged 31-35 weeks (Group II).

Variables (n = 30)	Before	After	t	P-value
	Mean ± SD			
FHR (bpm)	129.5±17.1	139±14.2	2.37052	<0.047
EDV (ml)	11.6±4.1	8.5±3.4	3.37278	<0.0003
ESV (ml)	2.8±0.33	2.4±0.25	2.34067	<0.05
SV (ml)	8.9±4	6.8±3.57	2.247863	<0.05
COP (ml/min)	1137.5± 403.8	894.7±372.8	1.3810641	<0.015

Table 3 - Changes in fetal heart rate (FHR), end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), and cardiac output (COP) before and after use of mobile phone in pregnant women of gestational age ranged 36-40 weeks (Group III).

Variables (n = 30)	Before	After	t	P-value
	Mean ± SD			
FHR (bpm)	130.9±12.4	138.6±15.3	2.83464	<0.004
EDV (ml)	17.7±1.9	14.7±1.9	2.895271	<0.002
ESV (ml)	2.9±0.33	2.5±0.5	3.293062	<0.003
SV (ml)	14.6±0.9	12.4±2.2	2.92236	<0.004
COP (ml/min)	1932.2±210.12	1662.75±282.26	1.3811641	<0.016

Table 4 - Changes in fetal heart rate (FHR), end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), and cardiac output (COP) before and after use of mobile phone by their mothers for 10 minutes (Group IV).

Variables (n = 30)	Before	After	t	P-value
	Mean ± SD			
Neonatal HR (bpm)	129.2±10.3	134.8±11.1	2.91235	<0.004
EDV (ml)	16.9±1.3	14.3±1.8	2.87434	<0.002
ESV (ml)	2.7±0.41	2.4±0.3	3.29218	<0.003
SV (ml)	13.9±0.7	12.1±2.4	2.89321	<0.005
COP (ml/min)	1877.1±219.14	1676.3±277.19	1.38624	<0.018

Table 5 - Degree of change in fetal and neonatal heart rate (HR), end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), and cardiac output (COP), before and after use of mobile phone in pregnant women of all groups.

Variables	25-30 weeks		31-35 weeks		36-40 weeks		Newborn	
	Before	After	Before	After	Before	After	Before	After
FHR	124.3 (12.9)	140.6 (12.6)	129.5 (17.1)	139 (14.2)	130.6 (12.4)	139 (15.3)	129.2 (10.3)	134.8 (11.1)
FHR increase (%)	13.1		7.3		5.9		4.3	
EDV	9.1 (1.3)	6.3 (1.0)	11.6 (4.1)	8.5 (3.4)	17.7 (3.4)	14.7 (1.9)	16.9 (1.3)	14.3 (1.8)
EDV reduction (%)	30.7		26.7		16.9		15.4	
ESV	3.1 (0.5)	2.5 (0.47)	2.8 (0.33)	2.9 (0.25)	2.5 (0.3)	2.4 (0.5)	2.7 (0.41)	2.4 (0.3)
ESV reduction (%)	19.3		14.3		13.8		11.1	
SV	5.8 (1.2)	3.7 (1.0)	8.9 (4.0)	6.8 (3.57)	14.6 (0.9)	12.4 (2.2)	13.9 (0.7)	12.1 (2.4)
SV reduction (%)	36.2		23.6		15.1		13.0	
COP	721.3 (175.3)	528 (172.1)	1137.5 (403.8)	894.7 (210)	1932.2 (210)	1662.75 (282.26)	1877.1 (219.14)	1676.3 (277.19)
COP reduction (%)	26.8		21.3		13.4		10.7	

decrease in EDV was 30.7% in group I, decreased to 26.7% in group II, decreased to 16.9% in group III, and further decrease to 15.4% in group IV. There was also a reduction in the percentage of decrease in ESV as the gestational age increase. The percent of decrease in ESV was 19.3% in group I, decreased to 14.3% in group II, decreased to 13.8% in group III, and further decrease to 11.1% in group IV. The percent of decrease in SV was changed from 36.2% in group I, to 23.6% in group II, to 15.1% in group III, and 13.0% in group IV (**Figure 1**). The percent of reduction in EDV is more than the percent of reduction in ESV in all groups, denoting that the reduction in the SV is mainly due to the reduction in the EDV more than ESV. The percent of decrease in COP showed progressive reduction from 26.8% in group I, to 21.3% in group II, to 13.4% in group III, and decreased to 10.7% in group IV, denoting that the effect of exposure to EMF on the COP become less as the gestational age increased (**Figure 2**).

Discussion. During recent years, mobile communication systems have experienced wide and rapidly growing use all over the world.⁹ The use of cellular (mobile) phone has widely used worldwide during the last decade. Their rapid adoption by general public has resulted in an increased research interest in possible harmful health effects.¹⁰ This common use of cellular phones has given rise to concerns on the potential influences of EMFs on human physiology.¹¹ The studies of mobile phones and their possible health impact has been focused on the change in electric activity of the brain,^{12,13} sleep pattern,¹⁴ subjectively perceived

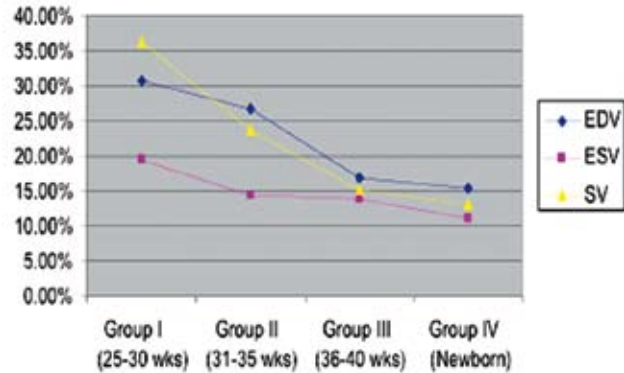


Figure 1 - Percent of reduction in fetal and neonatal end diastolic volume, end systolic volume and stroke volume, before and after exposure to mobile phone in all studied cases.

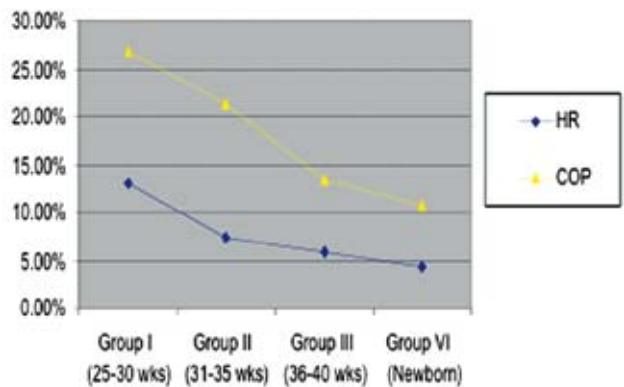


Figure 2 - Percent of change in fetal and neonatal heart rate and cardiac output, before and after exposure to mobile phone in all studied cases.

symptoms,¹⁵ and malignant tumors.¹⁶ Therefore, few human studies concerning the cardiovascular effects of radio frequency fields during mobile phone use, and the results have been controversial.¹⁷ There has been continuing concern on the possible harmful effects of EMF due to increased use of cellular phones in pregnancy, which has been reported to produce subjective disorders such as headache, sleep irregularity, hematological, and cardiovascular abnormalities.¹⁸

Anatomically, the fetus may be in close proximity to the cellular phones during transport and use. Therefore, EMFs emitted by cellular phones may lead to fetal cardiovascular effects.¹⁹ Our results showed that after antenatal and postnatal maternal use of mobile phones in all studied groups, there was a statistical significant increase in fetal and neonatal HR, and significant reduction of EDV, ESV, and SV and COP. In the present study, the percentage of reduction in EDV is more than the ESV reduction in all groups. This prominent decrease in EDV more than the decrease in ESV denotes that the decrease in SV is mainly due to the decrease in contractility. The percentage of decrease in COP showed progressive reduction from 26.8% in group I to 21.3% in group II, to 13.4% in group III, and a 6.9% decreased in group IV, denoting that the effect of exposure to mobile phone on the COP become lesser as the gestational age increased. Thus, the decrease in fetal and neonatal COP as a result of exposure to EMF emitted from mobile phones in our results is explained mainly by the decrease in SV, and this decrease can be explained by a decrease in cardiac contractility as the observed decreased in EDV was more than the decrease in ESV in all the studied groups. Another explanation for this decreased in SV is the decreased cardiac filling manifested by the observed decreased in EDV. This decreased cardiac filling may be explained by the observed tachycardia, which shortens the period of ventricular filling leading to decrease in EDV. However, this is not likely to be an accepted explanation, as the observed tachycardia is within physiological range (not so marked to the extent that it shortens the duration of ventricular filling). The decreased cardiac contractility due to exposure to EMFs can be explained by different mechanisms. The first mechanism is direct toxic effect on cardiac muscle, which was proposed by Fadel et al,²⁰ who found considerable alteration in ECG recordings from rats exposed to EMFs, all of which indicate cardiac toxicity. They found a change in the ECG as an irregular pattern in QRS complexes, which may indicate destruction of cardiac muscle, and/or local blocks in conduction of impulse by Purkinji fibers. Another change was a decreased *p* wave amplitude due to cardiac myopathies that results from myocardial infarction associated with diminished muscle mass, preventing major portions of the heart from becoming massively

depolarized all at once. A third change in ECG that results from exposure to electromagnetic fields is the elevation of S-T segment, which indicates an acute myocardial injury due to infarction or pericarditis. A second mechanism for decreased cardiac contractility due to exposure to EMFs is increased toxicity in cardiac muscle due to failure in the metabolic activity of the red blood cells (RBC) that occurred by magnetic fields, this was also postulated by Fadel et al,²⁰ who found that exposure of rats to EMFs resulted in the decrease of RBC membrane elasticity and permeability, and changes in the molecular structure of hemoglobin.

A third postulated mechanism for decrease cardiac contractility due to exposure to EMFs is the alteration in calcium channels (Ca^{2+}) homeostasis. Attempts have been carried out to identify possible effects of electromagnetic radiation in Ca^{2+} influx through the cell membrane in non-excitabile cells.²¹ The EMFs may have an influence on calcium homeostasis in different cells including rat thymic lymphocyte, human T-lymphocytes, rat pituitary cells, neurons, and astrocytes via voltage-gated ion channels of the cell membrane.²²⁻²⁴ This last mechanism is the most likely accepted mechanism in our case since the first 2 mechanisms are chronic effects that need long term and massive exposure to occur.

On the effect of EMF on FHR patterns, our result showed that exposure to EMFs emitted from mobile phones leads to fetal tachycardia. In contrast to our results, other investigators found that EMFs produced by cellular phones do not cause any demonstrable effect in FHR, acceleration, and deceleration after holding cellular phone on stand by mode, and then on dialing mode each for 5 min.⁷ This controversy can be explained by the difference in the duration of exposure, in our study it was 10 min, and not 5 min. Considering our results, it is very likely that both differential sensitivity and differential reactivity of individuals and animals to exposure to EMFs is due the ability of magnetic fields to influence large numbers of specific biological target cells (neurons, astrocytes, endocrine cells), biochemical reactions, and different mechanisms of inter cellular communication.^{23,25,26}

There are some postulated mechanisms for the effect of EMFs on HR. One of these mechanisms is the central effect in brain structure that controls autonomic function, specially HR and heart rhythmicity. Both autonomic nervous system branches, the parasympathetic, and sympathetic systems, modulate cardiovascular function, and are tonically active in regulating heart function. The tonic activity of the autonomic nervous system in the cardiovascular system is due to the combined effect of activity in 3 interrelated areas of the medulla and hypothalamus. The medulla contains a vasomotor area comprised of a pressor area that maintains tonic sympathetic outflow to the circulation, and a depressor

area that inhibits the pressor area. The medulla also contains a cardio inhibitory area which, when stimulated, simultaneously increases parasympathetic activity and decreases sympathetic activity to the heart, thereby decreasing heart rate and myocardial contractility. Stimulation of the cardioaccelerating area in the hypothalamus has the opposite effect on the nerve outflow, resulting in increases in HR and myocardial contractility.²⁷

In conclusion, we suggest that this result may be a nonspecific physiological response to the pulsed magnetic fields. We also recommend avoidance of cellular phone use especially in the early weeks of gestation and if necessary, the phone call should be less than 10 min. Further studies are required for testing various exposure times. Further investigations are needed to clarify the exact mechanism by which EMFs affect cardiac output, SV, EDV, and HR.

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