



Original Research

Exposure to cell phones reduces heart rate variability in both normal-weight and obese normotensive medical students

Mohammed Alassiri^{a,c,*}, Asma Alanazi^{b,c}, Hussain Aldera^{b,c}, Sultan A. Alqahtani^{b,c},
Abdulrahman S. Alraddadi^{b,c}, Meshal S. Alberreet^b, Abdullah I. Alhussaini^b, Yousef Alotaibi^b,
Mahmoud A. Alkhateeb^b, Abdullah S. Shatoor^d

^a Department of Basic Sciences, College of Science and Health Professions, King Saud bin Abdulaziz University for Health Sciences (KSAU-HS), Riyadh, Saudi Arabia.

^b Department of Basic Medical Sciences, College of Medicine, King Saud bin Abdulaziz University for Health Sciences (KSAU-HS), Riyadh, Saudi Arabia

^c King Abdullah International Medical Research Center (KAIMRC), Riyadh, Saudi Arabia

^d College Medicine, Department of Internal Medicine, Cardiology Section, King Khalid University, Abha, Saudi Arabia



ARTICLE INFO

Keywords:

Electromagnetic field
Electrocardiogram
SDNN
SDNNi
SDANN
Pnn50

ABSTRACT

Background: We investigated and compared the effect of the radiofrequency electromagnetic field (RF-EM) emitted by a cell phone on the electrocardiogram and heart rate variability (HRV) of normotensive normal-weight and obese medical students.

Method: Twenty medical student volunteers, normal weight (age = 23 ± 2 , BMI = 23.05 ± 1.72) or obese (age = 24 ± 2 , BMI = 32.39 ± 4.78), were exposed to a cell phone (1) close to the heart in silent mode, no ringing or vibrating; (2) close to the heart in ring and vibration mode; (3) next to the ear (brain) while listening; and (4) next to the ear while listening and speaking.

Results: The average basal HR of obese students significantly increased, while the PR interval; time domains, including standard deviation (SD) of all normal R–R intervals (SDNN), mean of the SD of all normal R–R intervals (SDNNi), SD of the average of normal R–R intervals (SDANN), and percentage of R–R intervals at least 50 ms different from the previous interval (pNN50); and high-power frequency (HF) decreased. The LF/HF ratio also significantly increased. The SDNN, SDNNi, SDANN, pNN50, and HF levels significantly decreased and the LF/HF significantly increased in normal-weight and obese individuals only when the phone was near the apex of the heart in ring and vibration mode. All changes were more profound in obese students.

Conclusion: Keeping the phone in a chest pocket reduced the HRV of normal-weight and obese medical students and exaggerated the effect of obesity on sympathetic activation.

© 2020 Elsevier Inc. All rights reserved.

Introduction

Cell phones have become an integral part of everyday life worldwide given their widespread use in communicating, listening to music, playing games, browsing the internet, and shopping. In 2015, the total number of cell phones was estimated to be around 7.4 billion worldwide, though the number may be higher because many users own more than one device.⁹ Despite the benefits of cell phones in our daily life, they represent a serious environmental health

Abbreviations: SDNN, standard deviation of all normal R–R intervals; SDNNi, mean of the standard deviation of normal sinus R–R intervals; SDANN, standard deviation of the average normal R–R intervals; pNN50, percentage of R–R intervals at least 50 ms different from previous intervals

* Corresponding author at: Department of Basic Sciences, College of Science and Health Professions, King Saud bin Abdulaziz University for Health Sciences (KSAU-HS), Riyadh, Saudi Arabia.

E-mail address: assirim@ksau-hs.edu.sa (M. Alassiri).

<https://doi.org/10.1016/j.explore.2020.02.006>

1550-8307/© 2020 Elsevier Inc. All rights reserved.

hazard because they are a major generator of continuous-pulse microwave and radiofrequency electromagnetic radiation (RF-EM) of frequencies between 900 and 1800 MHz.¹⁷

Because of the paucity of scientific evidence, the World Health Organization (WHO) is currently conducting several large multinational epidemiological studies, including case-control studies and prospective cohort studies, to examine the adverse effect of cell phone use, at levels below the known thermal level, on health. Based on the findings of two international bodies, the WHO recommends reducing exposure time to EM radiation. In addition to the thermal effect on tissue, several experimental and cross-sectional studies have investigated the adverse pathological nonthermal effects of the RF-EM field emitted by cell phones on the health of humans and animals. Most of these effects appeared during or after a call and disappeared after 2 h. Indeed, headache, warmth around the ear and auricles, fatigue, decrease in reflexes, blurred vision, eye inflammation, disturbed memory and sleep, behavioral changes, inner ear

damage, reproductive toxicity, flu-like symptoms, electromagnetic hypersensitivity, and some cases of cancer have been reported.⁵

The effect of cell phone radiation on cardiac health and function remains an important issue. Heart rate variability (HRV) refers to the beat-to-beat variation in heart rate (HR) and is a reliable marker of the autonomic control of the heart.³⁶ In longitudinal studies, a reduced HRV predicts an increased risk of cardiovascular diseases (CVDs) and mortality. On the one hand, several studies on healthy humans of various ages and occupations have shown the adverse effect of RF-EM fields on cardiac health caused by fluctuations in HR and HRV, and some contradictory findings.⁶ Exposure to an EM field (EMF) significantly reduced HRV and increased HR and sympathetic activity. Another study reported that healthy age-matched subjects who used their cell phones for more than 10 years showed a decrease in HRV with an increase in sympathetic activity.¹³ In contrast, adolescent students who had short-term exposure to RF-EM showed a decreased HR and increased parasympathetic activity.²⁷ However, numerous studies have shown no relationship between exposure to RF-EM and blood pressure (BP), HR, and HRV.^{7,34,28,2,3,15,37} The differences between the results of the cited studies could be due to the race, age, and sex of the subjects, exposure time and strength, and method of analysis.

Despite these differences, most of the above-mentioned studies were conducted with normal-weight healthy individuals. However, the effect of an RF-EM field on cardiac function in the presence of other cardiovascular risk factors is not well known. Obesity, defined as a body mass index (BMI) of 30 or more, is an independent risk factor for sudden cardiac death and arrhythmias in humans.^{20,10} Obesity can reduce HRV, an effect attributed to a decrease in the responsiveness of adrenoreceptors, withdrawal of parasympathetic tone, and/or increased sympathetic activity.¹⁶ However, the effect of RF-EM from a cell phone on an electrocardiogram (ECG) and HRV in obese individuals remains unknown. This interesting area is the focus of this study.

Medical students are among the busiest individuals in society. Their busy academic schedule leaves them little time for physical exercise, which increases the chance of becoming overweight and obese. In addition, medical students are among the most active in the use of cell phones for communicating, taking photos, recording lectures, and browsing the internet. In this study, we targeted both normal-weight and obese medical students who have used their cell

phones at least 2 h per day in the preceding 2 years to investigate and compare their ECG trace and HRV before and after exposure to the RF-EM generated by their cell phone. The effect of the RF-EM field was studied with the phone in a pocket near the apex of the heart (in ringing and silent modes) and when placed near the ear (brain) while listening and speaking.

Materials and method

Ethical statements

The consent forms and all procedures conducted in this study were approved by the biomedical ethics review board of King Saud Bin Abdulaziz University for Health Sciences under the Institutional Review Board number (IRB #) SP17/064/R.

Study subjects and inclusion and exclusion criteria

Twenty healthy male students in their second or third year of medical school were recruited for this study and assigned to either the normal-weight group ($n = 9$, mean age = 23 ± 2 y, mean BMI = 23.05 ± 1.72 kg/m²) or the obese group ($n = 11$, mean age = 24 ± 2 y, mean BMI = 32.39 ± 4.78 kg/m²). Before the study began, all students were informed about the procedure and the aim of the study and written informed consent was obtained. The average number of years and hours per day using a cell phone are shown in Fig. 1. All participants were asked to not consume any caffeinated beverages or perform any physical exercise 12 h before being examined and to abstain from using a cell phone at least 2 h before the experimental session. The students were not on any pharmacological treatment; had no chronic respiratory, vascular, neurological, or disabling disease or any recent acute illness; and were free of academic or social stress. Members of both groups had normal blood pressure, regular sleep habits, did not smoke 2 days before the test, and were trained and habituated to the laboratory examination room, experimenters, equipment, and protocols. Also, the students underwent a brief medical examination by a physician at the local College of Medicine to ensure they did not have any CVD or respiratory problems. The demographic data of all participants are presented in Table 1.

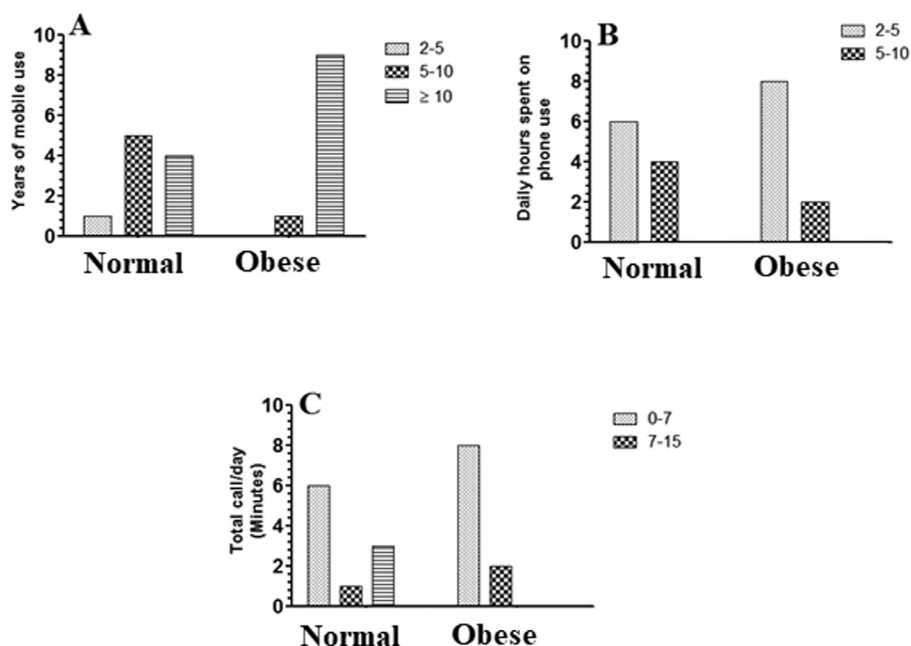


Fig. 1. Descriptive analysis of cell phone use by normal-weight and obese students.

Table 1
Characteristics of study participants.

	Normal	Obese
N (%)	9 (45)	11 (55)
Age (years)	23 ± 2	24 ± 2
Smokers (%)	0 (0)	0 (0)
Diabetes (%)	0 (0)	0 (0)
Stroke (%)	0 (0)	0 (0)
History of any CVD (%)	0 (0)	0 (0)
BP (mmHg)		
Systolic	115 ± 9	115 ± 13
Diastolic	72 ± 9	70 ± 12
Weight (kg)	68.82 ± 6.28	99.02 ± 17.18 ^a
Height (m)	1.73 ± 0.04	1.75 ± 0.06
BMI (kg/m ²)	23.05 ± 1.72	32.39 ± 4.78 ^a

Values are presented as mean ± SD.

^a Significantly different compared to the weight of lean subjects. Significantly difference is $P < 0.05$.

Experimental procedure

The study was performed in a partly darkened temperature-controlled quiet laboratory at room temperature (22 °C). All phones except the one used in the experimental procedure (iPhone 5 plus) were left outside the laboratory. Before the beginning of the experiments, each volunteer was asked to remove any metal objects such as rings, watch, and belt. Blood pressure, weight, and height of each participant were measured before the ECG was recorded over 2 h using a 3-electrode recording system (Lead II) (PowerLab 8/30 ML870 high-performance data acquisition system, ADInstruments, Dunedin, New Zealand). The ECGs for all volunteers were recorded in four phases. For the first phase, the cell phone in silent mode (no ringing or vibrating) was fixed at the parasternal area between the second and fifth intercostal spaces of the chest using a pocket suspended from the neck. The second phase was like the first phase but the phone was in ring plus vibration mode. In the third phase, the ECG was recorded while the phone was placed by another examiner next to the ear of the volunteer who was asked to listen to a normal call (with no bad news). The fourth phase was like the third phase but the volunteer was asked to speak during the recording. All phases were recorded within 2 h, starting at 8:00 a.m., with each phase recorded over 15 min. The experimental procedure was adopted from previous similar studies.³⁵ However, the ECGs in those studies were recorded over short periods and in some cases without any recovery time, whereas 15 min separated the phases of this study while the phone was turned off and kept outside of the laboratory.

A schematic presentation of the experimental procedure is shown in Fig. 2.

Data analysis

Data were saved on a Dell computer and analyzed using LabChart 7.1 software (ADInstruments). For a spectral analysis of HRV, the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology¹ defined 2 to 5 min as the appropriate amount of time to extract from an ECG. Hence, a 5-min

period was used to analyze both the ECG and the HRV. The first 5 min of all recordings was excluded from the analysis to minimize the inclusion of stressful stimuli, for example, when the phone suddenly rings. ECG intervals were analyzed beat-to-beat using the ECG module of the software, and the periods of the PR, QRS, ST, and QT intervals (in milliseconds) and the HR were derived from the signal using the software. In addition, both the time and frequency domains of the HRV were analyzed using the HRV module of the software. Time domain analysis included (1) the standard deviation of all normal R–R intervals (SDNN, ms), (2) the mean of the 5-min standard deviation of the normal R–R intervals (SDNNi, ms), (3) the standard deviation of the average R–R interval (SDANN, ms), and (4) the percentage of R–R intervals that are at least 50 ms different from the previous interval (pNN50, %). Frequency domain analysis included the low-frequency (LF, 0.04–0.15 Hz) and high-frequency (HF, 0.15–0.40 Hz) components and the ratio LF/HF. SDNN represents the overall HRV (overall autonomic activity).^{21,39} The power LF domain represents mainly the sympathetic modulation activity, while the HF and pNN50 domains represent vagal modulation activity.³⁹ However, the LF/HF ratio is an indicator of sympathovagal balance, where a high value indicates activation of sympathetic tone and/or a decrease in parasympathetic function.^{39,21} All data were analyzed by an assistant who was unaware of the experimental groups and is an expert in the use of the LabChart ECG and HRV modules.

Statistical analysis

All data were analyzed and presented in graph form using SPSS ver. 20.0 statistical software (IBM Corp., Armonk, NY, USA). Descriptive statistics are presented as frequencies and percentages for the categorical variables and as mean ± standard deviation for the numerical variables. A paired *t* test was used to compare the outcome variables within the same group (normal weight and obese). An independent *t* test was used to compare the outcome variable of the same group between 2 different phases. Of the 40 variables, only 5 were not normally distributed, and for those there was no contradiction in the significance of the comparison between a *t* test or a Mann–Whitney test. The years and hours of cell phone use by all subjects were plotted using GraphPad Prism ver. 6.

Results

General signs and ECG changes

During the study period, all subjects had sinus rhythm. No arrhythmias were noted in the analyzed records of all phases from both normal-weight and obese students. No participant had any symptoms such as a headache, pain, or stress during the study period. The mean HR and QRS, PR, QT, and ST segments of both normal-weight and obese students remained not significantly different from their baseline (recovery) levels during all phases of recording after cell phone exposure (Table 2). There was also no significant difference in the mean HR and the segments of an obese student compared with those a normal-weight student in all phases (Table 2). In

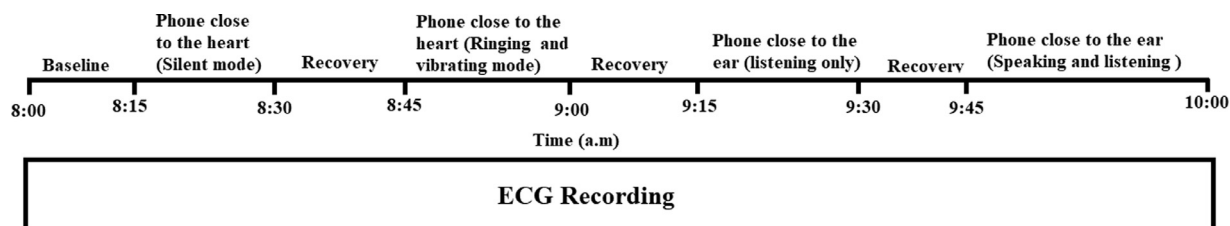


Fig. 2. Schematic diagram of the experimental procedure used to record ECGs.

Table 2

Analysis of ECGs of normal-weight and obese subjects recorded during all phases of the study.

Treatment		Phase 1		Phase 2		Phase 3		Phase 4	
parameter		BL	EX	BL	EX	BL	EX	BL	EX
HR (bpm)	Normal	71 ± 8	69 ± 7	68 ± 5	68 ± 7	69 ± 5	69 ± 8	79 ± 30	69 ± 6
	Obese	77 ± 12 ^a	75 ± 11	76 ± 9 ^a	74 ± 8 ^a	73 ± 10	74 ± 9	73 ± 10	74 ± 10
QRS (ms)	Normal	88 ± 9	88 ± 9	88 ± 10	89 ± 8	89 ± 9	88 ± 9	88 ± 9	88 ± 9
	Obese	83 ± 13	83 ± 13	82 ± 13	83 ± 13	84 ± 12	85 ± 12	84 ± 13	85 ± 12
QT (ms)	Normal	334 ± 13	339 ± 13 ^b	340 ± 13	343 ± 15	340 ± 14	341 ± 15	341 ± 14	340 ± 14
	Obese	318 ± 24	329 ± 23	318 ± 19	325 ± 24	324 ± 25	323 ± 25	324 ± 26	324 ± 25
PR (ms)	Normal	196 ± 40	186 ± 45	178 ± 28	175 ± 28	181 ± 36	177 ± 32	183 ± 36	178 ± 30
	Obese	161 ± 22 ^a	161 ± 22	165 ± 22	163 ± 22	164 ± 21	172 ± 41	165 ± 20	179 ± 60

Values are presented as mean ± SD (*n* = 9 for normal weight and 11 for obese).^a Significantly different when compared to normal subjects in the same phase.^b Significantly different when compared to the baseline within the same group. Values were considered significantly different when *P* < 0.05.

BL: baseline (recovery); EX: exposure. Phase 1: phone in a pocket close to the apex of the heart in silent mode. Phase 2: phone in a pocket close to the apex of the heart in ring plus vibration mode. Phase 3: phone next to the ear while the subject just listens. Phase 4: phone next to the ear while the subject listens and speaks.

contrast, the resting basal PR interval of the obese subjects was significantly lower than that of the normal-weight students during all phases, but remained within physiological levels (Table 2). Similarly, resting HR levels were significantly higher in obese students than in normal-weight students.

Changes in HRV

The calculated basal time domains of SDNN, SDNNi, SDANN, and pNN50 and the calculated basal HF power domains of the obese volunteers were significantly lower than those of the normal-weight individuals (Tables 3 and 4). However, the calculated LF/HF ratio of obese subjects was significantly higher than that of normal-weight subjects (Table 4). Cell phone exposure during phase 2 only (phone in ring plus vibration mode and near the apex of the heart) significantly decreased the HF domain but increased the LF/HF domain in both normal-weight and obese students (Table 4). During this phase, SDNN, SDNNi, SDANN, and pNN50 significantly decreased by 14.8%, 20.5%, 19.2%, and 14.5%, respectively, in normal-weight students and by 23.8%, 31.5%, 27.6%, and 26%, respectively, in obese students (Table 3). The calculated HF power during phase 2 significantly decreased by 21% and 31% in normal-weight and obese students, respectively (Table 4). However, normal-weight students exposed to the cell phone during phase 2 had a 31% increase in the LF/HF ratio whereas obese students had a 39.1% increase (Table 4). All time and frequency domain exposure values for normal-weight and obese students during phases 1, 3, and 4 did not differ much from the measured baseline (recovery) recordings (Table 4).

Discussion

This study investigated and compared the effect of the RF-EM field emitted from a cell phone on the electrical activity and autonomic regulation of the heart in normal-weight and obese normotensive medical students. This study had a unique design in that the effect of cell phone exposure was studied using real situations in which the phone was in a pocket near the heart while silent or ringing and when placed near the ear (brain) while listening or speaking during a call. Although the prolonged use of a cell phone did not affect the ECG trace of both normal-weight and obese individuals in all phases of the study, the findings of this study showed that there was a reduction in HRV, characterized by the dominance of the sympathetic tone and withdrawal of the parasympathetic tone, in both normal-weight and obese students during phase 2.

Investigations of the effect of a cell phone on cardiac electrical activity have yielded conflicting data. Hussain et al.¹⁹ showed a time-dependent decrease in QT with an increase in PR intervals in healthy female subjects (34–38 years old) exposed to cell phone radiation.¹⁹ In contrast, Tamer et al.³⁵ found that placing the cell phone on the apex of the heart of healthy male subjects (average age = 32 years old) had no effect.³⁵ As in the latter study, we observed no changes in the measured times of the RRS and QT intervals and the ST segment in both normal-weight and obese male students during all phases of cell phone exposure. Hence, the difference between our findings and those of Hussain et al. could be due to the sex and age of the participants.

However, two interesting findings of our study were the significant decrease in the resting basal PR interval (but within the

Table 3

Time domain heart rate variability parameters in all subjects.

		Phase 1		Phase 2		Phase 3		Phase 4	
Parameter		BL	EX	BL	EX	BL	EX	BL	EX
SDNN (ms)	Normal	151 ± 13	159 ± 15	155 ± 12	132 ± 12 ^a (14.8%)	158 ± 1	149 ± 14	157 ± 14	152 ± 17
	Obese	127 ± 13 [*]	135 ± 12 [*]	134 ± 10 [*]	102 ± 8 ^{*a} (23.8%)	129 ± 15 [*]	134 ± 16 [*]	126 ± 11 [*]	122 ± 14 [*]
SDNNi (ms)	Normal	54 ± 9	56 ± 6	49 ± 12	38.3 ± 8 ^a (20.5%)	52 ± 7	54 ± 13	51 ± 16	49 ± 14
	Obese	36.7 ± 7 [*]	34 ± 8 [*]	38 ± 8 [*]	26 ± 4 ^{*a} (31.5%)	35 ± 6.8 [*]	35 ± 8 [*]	35 ± 8 [*]	35 ± 7 [*]
SDANN (ms)	Normal	89 ± 11	79 ± 12	86 ± 7.8	69 ± 9 ^a (19.2%)	88 ± 11	84 ± 7	85 ± 13	87 ± 11
	Obese	61 ± 12 [*]	68 ± 9 [*]	65 ± 9.4 [*]	47 ± 6 ^{*a} (27.6%)	68 ± 10 [*]	67 ± 5 [*]	66 ± 5 [*]	65 ± 4 [*]
pNN50 (%)	Normal	5.6 ± 0.8	5.6 ± 0.4	5.5 ± 0.7	4.7 ± 0.18 ^a (14.5%)	5.2 ± 1.9	5.4 ± 1.2	5.8 ± 1.1	5.3 ± 1.1
	Obese	4.3 ± 0.3 [*]	4.2 ± 0.4 [*]	4.2 ± 0.3 [*]	3.1 ± 0.28 ^{*a} (26%)	3.4 ± 0.9 [*]	3.3 ± 1.2 [*]	3.4 ± 0.8 [*]	3.8 ± 3.2 [*]

Values are presented as mean ± SD (*n* = 9 for normal weight and 11 for obese).^{*} Significantly different when compared to normal subjects in the same phase. Values were considered significantly different when *P* < 0.05.

BL: baseline (Recovery). EX: exposure. Phase 1: phone in a pocket close to the apex of the heart in silent mode. Phase 2: phone in a pocket close to the apex of the heart in ring and vibration mode. Phase 3: phone is next to the ear while the subject just listens. Phase 4: phone is next to the ear while the subject listens and speaks.

Table 4

Frequency domain heart rate variability parameters in all subjects.

Parameter		Phase 1		Phase 2		Phase 3		Phase 4	
		BL	EX	BL	EX	BL	EX	BL	EX
LF (ms ²)	Normal	748 ± 145	767 ± 134	745 ± 114	716 ± 12	698 ± 144	712 ± 143	716 ± 103	736 ± 132
	Obese	754 ± 116	724 ± 132	734 ± 111	689 ± 78 (6%)	683 ± 123	689 ± 83	697 ± 43	704 ± 117
HF (ms ²)	Normal	442 ± 73	454 ± 69	435 ± 65	343 ± 49 ^b (21%)	414 ± 69	423 ± 89	418 ± 79	432 ± 65
	Obese	336 ± 49 ^a	356 ± 59 ^a	345 ± 44 ^a	238 ± 3 ^{a,b} (31%)	356 ± 89 ^a	312 ± 98 ^a	311 ± 103 ^a	362 ± 82 ^a
LF/HF	Normal	1.69 ± 0.04	1.68 ± 0.3	1.7 ± 0.1	2.0 ± 0.2 ^b (31%)	1.68 ± 0.3	1.68 ± 0.3	1.7 ± 0.2	1.7 ± 0.31
	Obese	2.2 ± 0.03 ^a	2.0 ± 0.3 ^a	2.1 ± 0.3 ^a	2.9 ± 0.3 ^{a,b}	1.92 ± 0.3 ^a	2.2 ± 0.2 ^a	2.2 ± 0.3 ^a	1.9 ± 0.19 ^a

Values are presented as mean ± SD (*n* = 9 for normal weight and 11 for obese).^a Significantly different when compared to normal subjects in the same phase.^b Significantly different when compared to the baseline within the same group. Values were considered significantly different when *P* < 0.05.

BL: baseline (Recovery); EX: exposure. Phase 1: phone in a pocket close to the apex of the heart in silent mode. Phase 2: phone in a pocket close to the apex of the heart ring and vibration mode. Phase 3: phone next to the ear while the subject just listens. Phase 4: phone next to the ear while the subject listens and speaks.

physiological range) and the significant increase in the HR of obese students compared to those parameters of the normal-weight students. No significant changes in the other ECG intervals of the obese students were observed. Some authors have found prolonged QT intervals in obese subjects that seemed to be dependent on the duration of obesity.¹⁶ However, our data agreed with numerous other studies that demonstrated a positive correlation between BMI and an increase in resting HR in obese individuals.^{18,23,29,30,31,32} In general, the HR of mammals is determined predominantly by the sympathetic outflow from the medulla to the heart and is modulated by vagal inputs. A 10% increase in body weight is associated with a decline in parasympathetic tone, which is accompanied by an increase in mean HR; conversely, the HR declines with a decrease in body weight.¹⁸ Hence, it would be reasonable to infer that an increase in sympathetic tone and/or a decrease in parasympathetic function in the obese students are the mechanics behind their high BMI and the observed elevated HR. In support of this inference, obesity has been associated with autonomic imbalance in all age groups and characterized by a decrease in parasympathetic tone and/or an increase in sympathetic modulation, independent of any other associated risk factor.^{11,14,22,25,38}

In contrast, some studies attributed this disparity in HR in the obese to other risk factors associated with obesity such as obstructive sleep apnea (OSA) and diabetes, which exist in some obese individuals and are associated with long-term stimulatory effects on the sympathetic tone.³³ This association does not apply to our study because none of the obese students who participated suffered from a respiratory disorder, sleep disturbance, or OSA. Hence, impairment of the autonomic control of the heart by obesity via a central nervous system-related mechanism is the most likely scenario. Indeed, high levels of adipokines such as leptin in obesity cause enhanced sympathetic flow, as confirmed in experimental animal studies.^{8,12}

An imbalance in the autonomic nervous system can lead to several CVDs and sudden death.³⁹ Monitoring HRV is an acceptable way to study the cardiac autonomic flow of the sympathetic and parasympathetic nervous systems.³⁹ In this study, we examined the effect of cell phone RF-EM field radiation on HRV in normal-weight and obese individuals when the phone was close to the apex of the heart in silent mode and in ring plus vibration mode and when the phone was close to the brain while listening or listening and speaking. However, the listening and listening-and-speaking calls included general talk such as greeting each other and excluded negative news to minimize the effect of stress on the ECG recording.

In the analysis of HRV, both time and frequency domains were calculated. Currently, pNN50 and RMSSD (the square root of the mean squared differences of successive R–R intervals) reflect parasympathetic activity, whereas SDNN and SDANN are related to both sympathetic and parasympathetic modulation of the HR. A reduction in pNN50 is a direct indicator of impaired vagal activity, while

SDANN < 50 ms and SDNN < 100 ms indicate an abnormal sympathetic outflow.³⁹ While there is consensus that HF reflects vagal activity,^{31,26} there is less agreement about the LF component; some authors consider it a measurable marker of sympathetic activity while others consider it a reflection of both sympathetic and vagal influences.^{24,39} Hence, rather than representing a single component, the calculated LF/HF ratio is considered to better reflect the activity of the sympathovagal balance.³⁹ A reduction in the LF/HF ratio is a clear indication of a shift toward sympathetic withdrawal and consequent vagal predominance, and vice versa. Overall, lower HRV values indicate relative sympathetic dominance, which may be due to high sympathetic activity and/or withdrawal of parasympathetic activity. In contrast, higher HRV values reflect a shift in the sympathetic/parasympathetic balance toward increased vagal activity.

In addition to the lower HR in obese students, the basal values of all time domains, including pNN50, SDANN, and SDNN, were significantly lower than those of normal-weight students, although the SDANN and SDNN values remained above 100 and 50 ms, respectively, indicating impaired vagal activity in the obese students. Concomitantly, HF of obese students was significantly lower than that of normal-weight students while the LF values of both obese and normal-weight students were similar which led to a significant increase in LF/HF, indicating withdrawal of parasympathetic activity and a shift toward sympathetic dominance. These data clearly indicate that patients with a reduced HRV had a higher HR. Such findings are not novel and have been reported for obese subjects of all ages by many authors.^{11,14,22,25,26,38}

The most interesting finding of this study is the significant decreases in all measured time and frequency domains in both normal-weight and obese individuals after exposure to the cell phone during phase 2, when the phone was kept in a pocket near the apex of the heart and in the ring plus vibration mode. The decreases in the domains indicate reduced HRV and withdrawal of the parasympathetic modulation. This finding is a clear warning of the effect of keeping a cell phone near the heart, with the effect greater on obese students. This could be due to the dominance of the resting sympathetic tone in the obese, as discussed above, which exaggerates the effect of the RF-EM field. To the best of our knowledge, our study is the first to show the impact of phone use on HRV in obese individuals. Barutcu et al.⁴ reported no significant changes in the time and frequency domains in male volunteers exposed to a cell phone RF-EM field when the phone was in silent mode, which supports our findings in normal-weight subjects. In addition, prolonged use of a cell phone was associated with significant decreases in SDNN, SDANN, RMSSD, and pNN50 and a significant increase in the LF/HF ratio in normotensive volunteers with an average age of 28 years.¹³

Despite these findings, our study has some limitations, one of which is the small sample size. Studies using a larger number of

participants should be conducted. In addition, the results of this study do not definitively support the proposal that obesity is the leading factor in the observed withdrawal of the resting parasympathetic function and dominance of the sympathetic tone observed in the obese students. Unfortunately, there was a difference in the number of years and hours per day of cell phone use between the normal-weight and obese students. Although obese students spent fewer hours per day using their phones, 90% of them have used their phones for more than 10 years, whereas only 40% of normal-weight students have done so. Hence, independent of obesity, we could suggest that the observed reduction in basal parasympathetic tone observed in obese students could be related to the prolonged use of a cell phone. Indeed, Barutcu et al. found that prolonged use of a cell phone was associated with a significant decrease in HRV.⁴ Hence, further controlled studies may be required to exclude this hypothesis.

Another limitation of this study is that most of the medical students who participated use their cell phone mainly for studying. The average time per day spent speaking on the phone was less than 7 min. Hence, targeting other subjects who use their cell phones for communication for longer periods could provide a bigger picture of the effect of the cell phone on HRV when it is very close to the brain. In addition, HRV follows a circadian pattern where it decreases during the morning and reaches its lowest value in the afternoon (3:00–06:00 p.m.) and then increases in the evening to reach the acrophase in the early morning (3:00–5:00 a.m.). Hence, the use of the LF/HF ratio and HR as markers of more sympathetic modulation, in which they reach their peak value in the afternoon (02:00–04:00 p.m.), makes sense. We tried to minimize the effect of this rhythm by recording all ECGs between 8:00 and 9:00 p.m. However, this needs further verification.

Most importantly, the reduction in HRV was seen only in phase 2, when the cell phone was near the heart and in ring plus vibration mode. Because the peak sensitivity of the human ear is at 2000–5000 Hz, alarm sounds and ring tones are designed to be in this frequency range. Because the ear has a very low threshold for these frequencies (i.e., these frequencies are very penetrating), listening to a ringing and vibrating cell phone for 15 min could be very stressful. This stress may have affected our findings. Unfortunately, it was difficult to study this effect. However, the participants notified us when they were stressed during all phases of the experiment. Hence, experiments designed to exclude the possible effect of stress on these findings should be conducted.

Conclusion

The unique findings of our study show that keeping a cell phone close to the heart and in ring mode could reduce HRV in both normal-weight and obese individuals, with the effect more profound in the obese. Whether obesity alone lowers HRV cannot be deduced from our results. However, these results may have significant practical and clinical applications. Knowing that obesity and the RF-EM field of a cell phone are inversely related to parasympathetic modulation in the heart and the HRV of normal-weight and obese normotensive adults, more care should be taken with respect to the use of a cell phone.

Funding

This work was funded and supported by the King Abdullah International Medical Research Center (KAIMRC) (grant number SP17/064/R).

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:[10.1016/j.explore.2020.02.006](https://doi.org/10.1016/j.explore.2020.02.006).

References

- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation*. 1996;93:1043–1065.
- Atlasz T, Kellenyi L, Kovacs P, et al. The application of surface plethysmography for heart rate variability analysis after GSM radiofrequency exposure. *J Biochem Biophys Methods*. 2006;69:233–236.
- Barker AT, Jackson PR, Parry H, Coulton LA, Cook GG, Wood SM. The effect of GSM and TETRA mobile handset signals on blood pressure, catechol levels and heart rate variability. *Bioelectromagnetics*. 2007;28:433–438.
- Barutcu I, Esen AM, Kaya D, et al. Do mobile phones pose a potential risk to autonomic modulation of the heart. *Pacing Clin Electrophysiol*. 2011;34:1511–1514.
- Blettner M, Berg G. Are mobile phones harmful. *Acta Oncol*. 2000;39:927–930.
- Bortkiewicz A, Gadzikwa E, Zmyslony M. Heart rate variability in workers exposed to medium-frequency electromagnetic fields. *J Auton Nerv Syst*. 1996;59:91–97.
- Braune S, Riedel A, Schulte-Monting J, Raczek J. Influence of a radiofrequency electromagnetic field on cardiovascular and hormonal parameters of the autonomic nervous system in healthy individuals. *Radiat Res*. 2002;158:352–356.
- Brydon L, O'Donnell K, Wright CE, Wawrzyniak AJ, Wardle J, Steptoe A. Circulating leptin and stress-induced cardiovascular activity in humans. *Obesity*. 2008;16:2642–2647.
- Carson S & Jonsson P 2015. Ericsson mobility report: On the pulse of the networked society. *Mobility report Stockholm, Sweden*; Ericsson AB.
- Chiuve SE, Sun Q, Sandhu RK, et al. Adiposity throughout adulthood and risk of sudden cardiac death in women. *JACC Clin Electrophysiol*. 2015;1:520–528.
- Dangardt F, Volkmann R, Chen Y, Osika W, Marild S, Friberg P. Reduced cardiac vagal activity in obese children and adolescents. *Clin Physiol Funct Imaging*. 2011;31:108–113.
- Eikelis N, Schlaich M, Aggarwal A, Kaye D, Esler M. Interactions between leptin and the human sympathetic nervous system. *Hypertension*. 2003;41:1072–1079.
- Ekici B, Tanındı A, Ekici G, Diker E. The effects of the duration of mobile phone use on heart rate variability parameters in healthy subjects. *Anatol J Cardiol*. 2016;16:833–838.
- Farah BQ, Prado WL, Tenorio TR, Ritti-Dias RM. Heart rate variability and its relationship with central and general obesity in obese normotensive adolescents. *Ein-stein*. 2013;11:285–290.
- Faust O, Acharya UR, Nergui M, et al. Effects of mobile phone radiation on cardiac health. *J Mech Med Biol*. 2011;11:1241–1253.
- Fraley MA, Birchem JA, Senkottaiyan N, Alpert MA. Obesity and the electrocardiogram. *Obes Rev*. 2005;6:275–281.
- Hardell L, Carlberg M, Hansson Mild K. Use of cellular telephones and brain tumour risk in urban and rural areas. *Occup Environ Med*. 2005;62:390–394.
- Hirsch J, Leibel RL, Mackintosh R, Aguirre A. Heart rate variability as a measure of autonomic function during weight change in humans. *Am J Physiol*. 1991;261:R1418–R1423.
- Hussain D, Ali A, Mazhir S, Juma A. Study the effect of mobile (cell phone) on the heart electricity. *Int J Appl Inform Syst*. 2014;7:1–4.
- Kannel WB, Plehn JF, Cupples LA. Cardiac failure and sudden death in the Framingham study. *Am Heart J*. 1988;115:869–875.
- Kleiger RE, Stein PK, Bigger Jr JT. Heart rate variability: measurement and clinical utility. *Ann Noninvasive Electrocardiol*. 2005;10:88–101.
- Krishna P, Rao D, Navekar VV. Cardiac autonomic activity in overweight and underweight young adults. *Indian J Physiol Pharmacol*. 2013;57:146–152.
- Lee JF, Harrison ML, Christmas KM, Kim K, Hurr C, Brothers RM. Elevated resting heart rate and reduced orthostatic tolerance in obese humans. *Clin Auton Res*. 2014;24:39–46.
- Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. *Circulation*. 1991;84:482–492.
- Souza N, Rossi R, Vanderlei F, et al. Heart rate variability in obese children. *Journal of Human Growth and Development*. 2012;22.
- Martini G, Riva P, Rabbia F, et al. Heart rate variability in childhood obesity. *Clin Auton Res*. 2001;11:87–91.
- Misek J, Belyaev I, Jakusova V, Tonhajzerova I, Barabas J, Jakus J. Heart rate variability affected by radiofrequency electromagnetic field in adolescent students. *Bioelectromagnetics*. 2018;39:277–288.
- Nam KC, Kim SW, Kim SC, Kim DW. Effects of RF exposure of teenagers and adults by CDMA cellular phones. *Bioelectromagnetics*. 2006;27:509–514.
- Peterson HR, Rothschild M, Weinberg CR, Fell RD, Mcleish KR, Pfeifer MA. Body fat and the activity of the autonomic nervous system. *N Engl J Med*. 1988;318:1077–1083.
- Piwonska A, Piotrowski W, Broda G, et al. The relationship between resting heart rate and atherosclerosis risk factors. *Kardiol Pol*. 2008;66:1069–1075. discussion 1076–8.
- Pomeranz B, Macaulay RJ, Caudill MA, et al. Assessment of autonomic function in humans by heart rate spectral analysis. *Am J Physiol*. 1985;248:H151–H153.
- Rossi M, Marti G, Ricordi L, et al. Cardiac autonomic dysfunction in obese subjects. *Clin Sci*. 1989;76:567–572.
- Rumantir MS, Vaz M, Jennings GL, et al. Neural mechanisms in human obesity-related hypertension. *J Hypertens*. 1999;17:1125–1133.
- Tahvanainen K, Nino J, Halonen P, Kuusela T, Laitinen T, Lansimies E, et al. Cellular phone use does not acutely affect blood pressure or heart rate of humans. *Bioelectromagnetics*. 2004;25:73–83.
- Tamer A, Gunduz H, Ozyildirim S. The cardiac effects of a mobile phone positioned closest to the heart. *Anadolu Kardiyol Derg*. 2009;9:380–384.

36. Vanderlei LC, Silva RA, Pastre CM, Azevedo FM, Godoy MF. Comparison of the Polar S810i monitor and the ECG for the analysis of heart rate variability in the time and frequency domains. *Braz J Med Biol Res.* 2008;41:854–859.
37. Wilen J, Johansson A, Kalezic N, Lyskov E, Sandstrom M. Psychophysiological tests and provocation of subjects with mobile phone related symptoms. *Bioelectromagnetics.* 2006;27:204–214.
38. Wu JS, Lu FH, Yang YC, et al. Epidemiological evidence of altered cardiac autonomic function in overweight but not underweight subjects. *Int J Obes.* 2008;32:788–794.
39. Xhyheri B, Manfrini O, Mazzolini M, Pizzi C, Bugiardini R. Heart rate variability today. *Prog Cardiovasc Dis.* 2012;55:321–331.