Nocturnal Exposure to Intermittent 60 Hz Magnetic Fields Alters Human Cardiac Rhythm

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Heart rate variability (HRV) results from the action of neuronal and cardiovascular reflexes, including those involved in the control of temperature, blood pressure and respiration. Quantitative spectral analyses of alterations in HRV using the digital Fourier transform technique provide useful in vivo indicators of beat-to-beat variations in sympathetic and parasympathetic nerve activity. Recently, decreases in HRV have been shown to have clinical value in the prediction of cardiovascular morbidity and mortality. While previous studies have shown that exposure to power-frequency electric and magnetic fields alters mean heart rate, the studies reported here are the first to examine effects of exposure on HRV. This report describes three double-blind studies involving a total of 77 human volunteers. In the first two studies, nocturnal exposure to an intermittent, circularly polarized magnetic field at 200 mG significantly reduced HRV in the spectral band associated with temperature and blood pressure control mechanisms ($P = 0.035$ and $P = 0.02$), and increased variability in the spectral band associated with respiration ($P = 0.06$ and $P = 0.008$). In the third study the field was presented continuously rather than intermittently, and no significant effects on HRV were found. The changes seen as a function of intermittent magnetic field exposure are similar, but not identical, to those reported as predictive of cardiovascular morbidity and mortality. Furthermore, the changes resemble those reported during stage II sleep. Further research will be required to determine whether exposure to magnetic fields alters stage II sleep and to define further the anatomical structures where field-related interactions between magnetic fields and human physiology should be sought. *Bioelectromagnetics 19:98–106, 1998.* © 1998 Wiley-Liss, Inc.

Key words: power-frequency; heart rate variability; EKG; HRV; EMF; Fourier transform

INTRODUCTION

Since the late 1960s many research studies have examined the biological consequences of exposure to power frequency electric and magnetic fields. In the first human exposure study performed in our laboratory, a large number of physiological and performance variables were screened with generally negative results [Graham et al., 1987]. The most relevant exception for purposes of this report was a biologically small but statistically significant slowing of mean heart rate (lengthening of the cardiac interbeat interval) resulting from exposure to combined 60 Hz electric and magnetic fields. Field-related slowing of heart rate has continued to be observed in subsequent studies performed with healthy young men in our laboratory [Maresh et al., 1988; Cook et al., 1992; Graham et al., 1994], and this response appears attributable to the magnetic aspect of exposure [Graham et al., 1993].

The mechanisms by which power frequency fields alter cardiac rhythm are not understood, and little attention has been paid to whether field exposure alters the natural variability in the human cardiac rhythm. A metronome beats at a constant rate; a healthy heart does not. Even in quiescent conditions, heart rate typically exhibits significant beat-to-beat variability. This type of variability is not consciously perceived by a person; and it should not be confused with heart rate reactivity, the slowing or speeding of heart rate in direct response to perceived situational or personal stimuli (eg., exercise, anxiety, relaxation, etc.). Heart rate variability

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(HRV) is a well-established physiological phenomenon, a consequence of the action of a number of neuronal and cardiovascular reflexes, including those involved in blood pressure control, thermoregulation, and respiration. Alterations in HRV are widely recognized in physiological research as reliable indicators of autonomic nervous system activity [Kitney and Rompelman, 1980, Akselrod et al., 1981; Pomeranz et al., 1985; Porges and Bohrer, 1990, Berntson et al., 1993; Kamath and Fallen, 1993]. HRV is mediated by the activity of the sympathetic and parasympathetic branches of the autonomic nervous system. In general, the sympathetic branch acts to speed up heart rate and the parasympathetic branch acts to slow heart rate. Extensive basic biomedical research has indicated that quantitative analyses of HRV provide useful in vivo indicators of beat-to-beat variations in sympathetic and parasympathetic nerve activity [Kamath and Fallen, 1993; Malik and Kamm, 1995].

Spectral analysis techniques have long been applied in the field of cardiovascular physiology [e.g., see monographs by McDonald, 1974 and by Milnor, 1982], and their use in performing quantitative analyses of HRV has become an area of emerging importance in clinical medicine. Certain alterations in HRV have newly-recognized prognostic value in a number of important conditions [coronary artery disease, Hayano et al., 1990; post-infarction risk, Kleiger et al., 1987; Bigger et al., 1993; diabetic autonomic neuropathy, Bernardi et al., 1992; systemic hypertension, Huikuri et al., 1996]. In addition, investigators of the Framingham Heart Study—the best-known community-based, continuously followed cohort study for cardiovascular disease in the world—have reported that quantitative spectral assessment of HRV offers prognostic information for mortality risk beyond that provided by the evaluation of traditional risk factors [Tsuji et al., 1994].

The quantitative methods used by basic physiologists and clinicians are essentially identical to those we report here in our evaluation of alterations in HRV resulting from magnetic field exposure. This report describes three recent human studies in which volunteers slept through the night in the laboratory while being exposed to intermittent or continuous power-frequency magnetic fields. Sufficient cardiac data were collected to make a detailed examination of HRV. The main purpose of these studies was not to evaluate HRV, but rather to determine if nocturnal exposure to magnetic fields suppressed plasma levels of the pineal hormone melatonin [Graham et al., 1996, 1997]. These studies provided the additional opportunity to obtain continuous recordings of cardiac interbeat interval (IBI) throughout the night for evaluation of HRV during magnetic field exposure. To our knowledge, our spectral studies of HRV in human volunteers exposed to power-frequency magnetic fields [Sastre et al., 1993; 1994; 1995] provide the first application of these well-validated physiological techniques and concepts to bioelectromagnetics research.

METHODS

Subjects

The participation of volunteers in these studies was reviewed and approved by the MRI Institutional Review Board for Human Studies in accordance with Federal guidelines and regulations [*Federal Register*, 1991]. Volunteers were recruited by posting informational notices at local colleges and universities. Inquiring individuals who telephoned about the study were given a complete and accurate description of the purpose, procedures, risks and benefits of participation and were screened to assure they met the specific criteria set for participation (male, age 18 to 35 years, no chronic disease or disability, no recent serious acute illness, no medications, regular sleep habits, no night work). All volunteers provided written informed consent.

Procedures

Procedures common to all studies are described first, followed by those specific to each of the three studies.

Common procedures The identical protocol was followed in each study. On arrival at the laboratory at 2200 h, the volunteer changed to a surgical scrub suit. His vital signs were recorded, and the indwelling catheter for the collection of multiple blood samples was inserted in an arm vein. IBI was recorded using silver-silver chloride biopotential electrodes attached with double-stick adhesive tape to prepared skin sites on the right clavicle and the seventh intercostal space under the left ancillary midline, corresponding to the standard electrocardiographic Lead II configuration. Beckman electrode paste served as the contact medium.

Each man slept overnight in the Human Exposure Test Facility at MRI from 2300 h to 0700 h under double blind test conditions. After the volunteer got into bed in the exposure room, a blood sample was obtained. The double-blind/field control system was activated at 2300 h. On sham control nights, the control system did not energize the field generating equipment. On exposure test nights, the field generating equipment was energized to present the magnetic field either intermittently or continuously until 0700 h. The nurse entered the exposure facility each hour to collect blood samples from the catheter. These collections occurred between 5 min before the hour to on the hour through the night. In the morning, the volunteer provided a final blood sample, his vital signs were recorded, and the catheter and sensors were removed.

It should be noted that collection of cardiac data was not the major purpose of the studies reported here. If for any reason problems were encountered with the cardiac recording system (e.g., an electrode became loose), the experimenters were instructed to turn the system off and to do nothing that might interfere with the major thrust of the study, the collection of blood samples for melatonin assays. Therefore complete cardiac data was available for only a subset of the subjects in each of the studies. Except for a requirement for completeness of the data, there were no other exclusions in the subset of subjects included in the cardiac analyses reported here.

Study 1 Study 1 used an independent groups design. Thirty-three healthy young men were randomly assigned to three groups matched on age, body size and sensitivity of melatonin to light exposure: a sham control group; a group exposed to a 10 mG magnetic field, and a group exposed to a 200 mG magnetic field. Exposure to the circularly-polarized field during the night was intermittent: 1 h off/1 h on. For example, midnight to 0100 h was ''on,'' 0100 to 0200 h was ''off'' and so on. During the hours designated as ''on,'' the field switched on and off every 15 s throughout the hour. For example, from 12:00:00 to 12:00:15 the field was on, from 12:00:15 to 12:00:30 the field was off, and so on. IBI was recorded continuously through the night. Complete cardiac data were collected on 29 of the 33 volunteers.

Study 2 Study 2 used a repeated measures design in which each subject served as his own control. Forty healthy, young men participated in two all-night test sessions. In one test session, all of the men were sham exposed from 2300 h to 0700 h. In the other test session, all of the men were exposed to the identical 200 mG intermittent exposure condition used in Study 1. Since the 10 mG exposure condition used in Study 1 gave negative results on all measures examined, this condition was not used in Study 2 or Study 3. The volunteers were assigned randomly to two counterbalanced orders of testing (sham/exposed, exposed/sham), and all sessions were conducted double-blind. Complete cardiac data were collected for 22 volunteers (44 all-night test sessions). Data were missing from proportionately more subjects in Study 2 than in Study 1, because complete data for both sessions were required for a subject to be included.

Study 3 Study 3 followed the design of Study 2. An additional 40 healthy, young men served as their own controls by participating in two all-night test sessions. As before, test sessions were counterbalanced across volunteers, and each man was sham exposed in one session and exposed to the magnetic field in another test session. This study differed from the previous ones in that it evaluated the effects of continuous exposure to the magnetic field. The 60 Hz, circularly-polarized magnetic field was activated at 2300 h at a field strength of 200 mG, and it remained on continuously until 0700 h. Complete cardiac data were collected for 26 volunteers (52 all-night test sessions). Data were missing from proportionately more subjects in Study 3 than in Study 1, because complete data for both sessions was required for a subject to be included.

Exposure Facility

Characteristics and control systems of the MRI Human Exposure Test Facility have been documented and are described in Cohen et al. [1992] and in Dietrich et al. [1995]. A systematic protocol using test instruments traceable to the National Institute of Standards and Technology was followed to verify the exposure characteristics of the facility and to calibrate the recording equipment.

Subjects were exposed to a uniform (4%–7%) circularly-polarized 60 Hz magnetic field generated in the facility by six Helmholtz coils surrounding each of the exposure rooms in both the vertical and horizontal axes. The horizontal field axis is oriented from the doorway to the rear of the exposure room, and the vertical axis, from floor to ceiling. Each field axis is independently energized from an adjustable autotransformer. The horizontal field current is shifted from the vertical field current by a phase angle of 90 degrees. Subjects slept on a cot in the facility with their bodies oriented in line with the horizontal field component (North to South). The facility was composed of two test rooms, A and B. In room A, the geomagnetic field was 44.1 μT; the horizontal component was 10.1 μT and the vertical component was 42.9 μT. In room B, the geomagnetic field was 53.0 μT; the horizontal component was 22.3 μT and the vertical component was 48.1 μT. Illumination in the facility is provided by incandescent lamps located above translucent ceiling panels and maintained at less than 10 lux during test sessions. Illumination levels were measured using a Digital Photometer (Model J16, Tektronix, Beaverton, Oregon).

Measures

Cardiac Interbeat Interval, or IBI, is a measure of the duration in milliseconds between successive R waves in the cardiac cycle. As such, it provides a direct and sensitive measure of heart rate. The physiological recording system consisted of six special-purpose isolation amplifiers with high common mode rejection and high input impedance, operating as the input to a Beckman Type R612 multi-channel recorder. The amplifiers were operated from an isolated power supply, and optical couplers were used to further isolate the amplifiers from the field generator. Physiological signals were selectively filtered for residual 60 Hz interference. The cardiac signals were conditioned through the amplifiers in the measurement system, passed to a hardware detector (Beckman Instruments, coupler Type 9857), that generated a pulse each time it determined the presence of the R wave event. The time between each two pulses, corresponding to one IBI, was determined using a digital counter with better than 0.1 ms accuracy. Data were stored on magnetic media for off-line analysis.

Signal Processing

Each volunteer generated 25,000 to 30,000 heartbeats over the course of a night. The series of continuously recorded interbeat time intervals was converted to instantaneous heart rate, which provided a regularly-spaced time series with a 1 s resolution. The time period selected for analysis was midnight to 0600 h. Since blood was drawn every hour, a 10-min segment (5 min before and after the hour) was deleted to remove possible artifacts associated with obtaining blood samples. The six 50-min ''hours'' were each further divided into three ''Periods'' corresponding to the first, middle and last thirds of the hour.

Spectral analyses using the digital Fourier transform were performed on the cardiac time series to determine if exposure altered specific, periodic components of heart rate variability known to be mediated by the activity of the sympathetic and parasympathetic branches of the autonomic nervous system on the heart. For each Period, a time series of 1024 points was analyzed as follows: (1) any linear trend (including the mean value) was first removed; (2) a Hamming window was applied; (3) a digital Fourier transform (DFT) was performed; and (4) the resulting spectrum was smoothed using a 7-point moving average [Blackman & Tukey, 1958; Bloomfield, 1976; Press et al., 1986; Marple, 1987]. The results of the DFT are expressed as the power spectrum, i.e., power at a given frequency in the range of 0.0 to 0.5 Hz. Since the time series had a time resolution of 1 s, the Nyquist limit for this spectral analysis is 0.5 Hz.

Spectral analyses provided measures of total power, absolute and relative power in specific frequency bands (e.g. 0 to 0.1 Hz and 0.15 to 0.40 Hz), as well as the ratios of power between specific bands. Selection of these particular frequency bands as the focus of spectral analysis was based on the results of numerous psychophysical and medical research studies [see Porges and Bohrer, 1990; Kamath and Fallen, 1993 and Malik and Camm, 1995 for extensive reviews of this literature].

Data from Study 1 were also used to determine if exposure influenced the chaotic, aperiodic components of the human cardiac rhythm. For chaos-theory analyses, we used an optomized FORTRAN implementation of Pincus' Approximate Entropy (ApEn) statistic, which is an approximation of the Kolmogorov-Sinai Entropy statistic [Pincus, 1991]. This statistic has the advantage of being relatively insensitive to the unavoidable ''noise'' present in biological systems and has been successfully applied in previous medical research on HRV in patient populations (Pincus et al, 1991; Pincus and Viscarello, 1992).

Statistics

The traditional approach consisted of performing analysis of variance for mixed designs (ANOVA; Systat for Windows, V. 5.0 for Study 1 and BMDP4V for Studies 2 and 3) on both heart rate mean and heart rate standard deviation, as well as spectral data. In Study 1, Group (sham, 10 mG, 200 mG) was the ''between subjects'' variable. Hour (1 through 6) and Period (1 through 3) were the ''within subjects'' variables. In Studies 2 and 3, Order of exposure (sham, real vs. real, sham) was the ''between subjects'' variable. Field (sham, 200 mG), Hour and Period were the ''within subject'' variables. Significant effects ($P < 0.05$) and trends ($P < 0.10$) were followed by simple effects analyses.

RESULTS

Study 1

The data from Study 1 were used to compare and contrast the usefulness of traditional, spectral and chaos-theory based approaches to analysis. Traditional measures of variability, such as the standard deviation (SD), provided a way to compare the present results with the results of previous HRV studies [Kamath and Fallen, 1993; Malik & Camm, 1995]. HRV due to the actions of periodic components in the cardiovascular system was assessed using Fourier spectral analyses. Chaos-theory based analyses allowed the quantification of aperiodic phenomena previously noted in medical studies of cardiac variability in patient populations.

Performance of traditional analyses using ANOVA revealed no significant differences between the test groups in mean heart rate or heart rate standard deviation data. This was not unexpected, given the

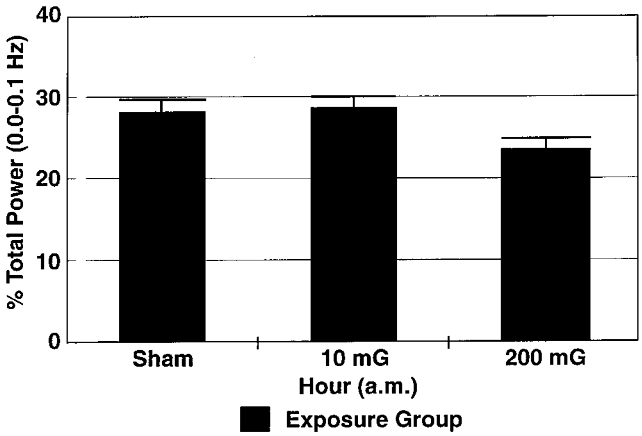

Fig. 1. Study 1. SEM of percent power in the low (0.0– 0.1 Hz) band for three exposure conditions (N = 29). Power in this portion of the spectrum was significantly reduced by exposure at 200 mG, compared to either exposure at 10 mG or sham control conditions (F = 3.89; df = 2,23; P = 0.035).

differences between this experimental design and our previous day-time studies of exposure effects on heart rate. Previously we found changes in mean heart rate primarily around the times when the fields were turned on and off each hour. Since there was a strong possibility of artifactual alterations in HRV resulting from the blood draw maneuvers, the five minutes before and the five minutes after the hour were not analyzed.

Performance of the chaos-theory based analyses provided negative results. The healthy, young men who participated in Study 1 exhibited chaotic heart rate variations within the normal range reported by others using the ApEn statistic [Pincus et al., 1991]. Under the conditions used in this study, analysis of the ApEn statistic revealed no differences between the sham control and exposed groups.

In contrast to traditional and chaos-theory based techniques, spectral analyses performed on the cardiac time series revealed significant differences between the test groups. As shown in Figure 1, power in the low spectral band was reduced by exposure at 200 mG, compared to either exposure at 10 mG or sham control conditions (F = 3.89; df = 2,23; P = 0.035). In addition, for those volunteers exposed to the intermittent 200 mG magnetic field condition, power at the peak frequency of the high spectral band was greater in the hours when the field was activated than in the hours when the field was turned off (t = 2.13; df = 8; P = 0.06). In contrast, there was no difference in high band spectral power for the sham control group when the same time periods were compared (t = 0.42; df = 8; P = 0.68). These results are shown in Figure 2. Power in the entire high band (0.15–0.40 Hz), however, was not affected by exposure group or by field-on versus field-off hours. Similar negative results were obtained when the ratio between power in the entire high band and total power was examined.

As an example, the three panels in Figure 3 show the power spectra for one volunteer exposed to the intermittent 200 mG magnetic field. Spectra for each analysis period in each of three sequential hours are shown. Increases in peak power in the high, respiratory arrhythmia band, and decreases in power in the low, blood pressure/thermoregulatory control band during magnetic field activation periods, are easily detectable across the three panels.

Study 2

Study 2 was undertaken to replicate and extend the spectral analysis of Study 1 results in a different and larger sample of volunteers, using a more powerful experimental design in which each individual served as his own control. Twenty-three volunteers provided complete cardiac interbeat interval data in both their sham control and magnetic field exposure test sessions. The signal processing methods and time periods selected for analysis were the same as described above, except that the chaos-based ApEn computations were not performed because of the negative results in Study 1.

As shown in Figure 4, compared to sham control conditions, exposure to the intermittent 200 mG magnetic field significantly reduced percent total power in the low spectral band (F = 5.99; df = 1,20; P = 0.02). This result replicates our previous observation in Study

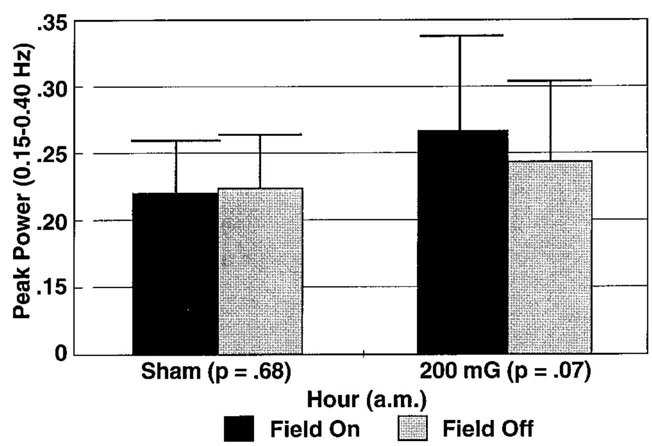

Fig. 2. Study 1. SEM of peak power in the high (0.15–0.40 Hz) band for field-on and field-off conditions. Power was greater in the hours when the field was activated than in the hours when the field was turned off (t = 2.13; df = 8; P = 0.06). No difference in high band spectral power was observed for the sham control group when the same time periods were compared (t = 0.42; df = 8; P = 0.68).

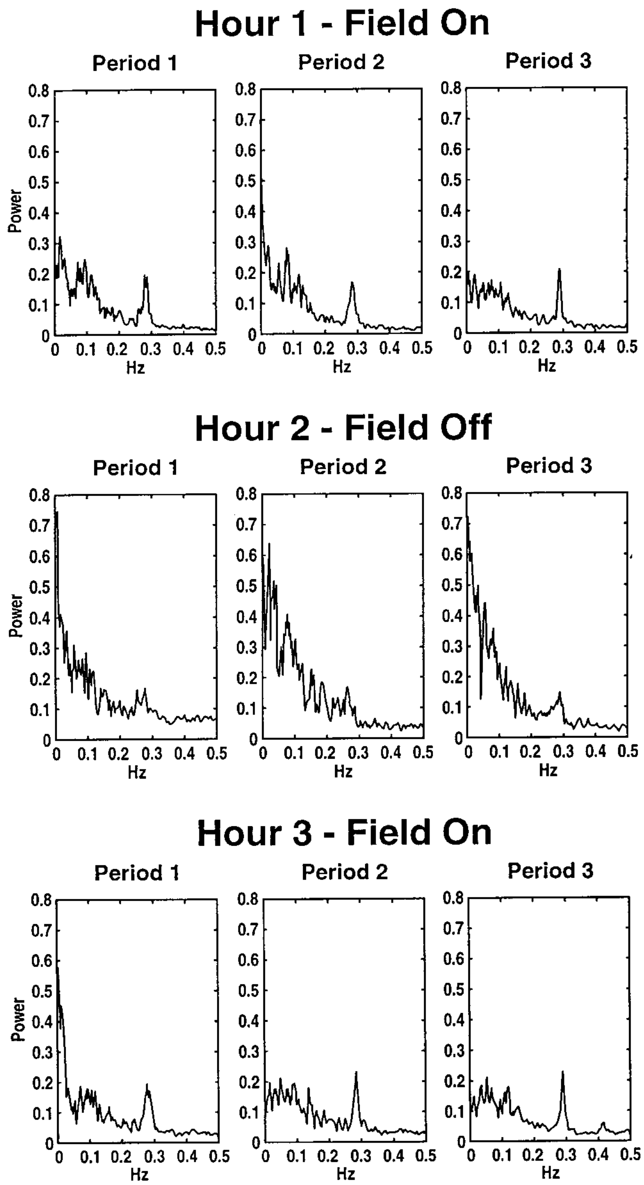

Fig. 3. Study 1. An example of power spectra of the cardiac time series recorded from one volunteer during three sequential hours of nocturnal exposure to an intermittent 60 Hz, circularly-polarized magnetic field at 200 mG. Spectra in each analysis period correspond to the first, middle and last thirds of the hour. Increases in peak power in the respiratory arrhythmia band (0.15–0.40 Hz) of the spectrum, and decreases in power in the blood pressure/thermoregulatory control band (0.0–0.1 Hz) of the spectrum during magnetic field activation periods, are easily detectable across the three sets of panels. Under these conditions, field exposure appears to alter the normal nocturnal variability that is inherent in human cardiac rhythm.

1. The differences in low band power between sham control and magnetic field exposure conditions increased as exposure time increased ($F = 2.92$; $df = 5{,}100$; $P = 0.017$). In contrast, when the same volun-

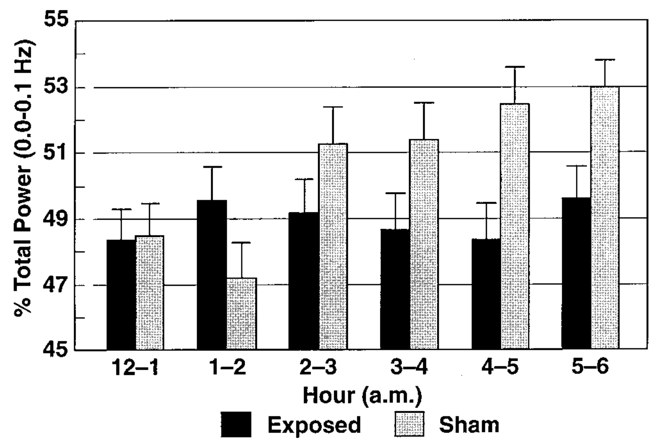

Fig. 4. Study 2. Quantitative spectral analysis of nocturnal heart rate variability measured in 23 healthy, young men under sham control conditions, and again during exposure to an intermittent 60 Hz, circularly-polarized magnetic field at 200 mG. Compared to sham control conditions, field exposure significantly reduced percent total power in the 0.0–0.1 Hz spectral band that reflects blood pressure and temperature control mechanisms ($F = 5.99$; $df = 1{,}20$; $P = 0.02$). The differences in power in this band between control and exposure conditions increased as exposure duration increased ($F = 2.92$; $df = 5{,}100$; $P = 0.017$).

teers were sham exposed, low band activity did not decrease; it showed the typical pattern of increasing power over the night.

Analysis of high spectral band activity also revealed field-related differences between sham control and field exposed conditions. Figure 5 illustrates these

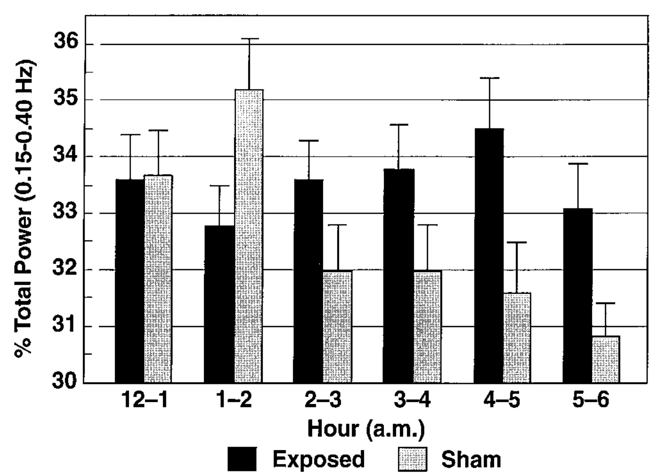

Fig. 5. Study 2. Spectral analysis of heart rate time series obtained in 23 healthy, young men under sham control conditions, and again during exposure to an intermittent 60 Hz, circularly-polarized magnetic field at 200 mG. Magnetic field exposure resulted in increased percent power in the high spectral band (0.15–0.40); sham exposure did not ($F = 3.32$; $df = 5{,}100$; $P = 0.008$).

differences. Magnetic field exposure resulted in increased percent power in the high band; sham exposure did not ($F = 3.32$; $df = 5,100$; $P = 0.008$).

Study 3

This study was undertaken to determine if continuous magnetic field exposure produces differential effects on HRV in human volunteers. Twenty-six additional volunteers provided complete cardiac interbeat interval data in both their sham control and magnetic field exposure test sessions. These data were submitted to spectral analysis using the procedures described above. Unlike our previous results with intermittent magnetic field exposure, we observed no significant decrease in the low band spectral power upon continuous field exposure (Field by Hour interaction $F = 0.71$; $df = 5,120$; $P = 0.61$). Again in contrast to our previous results with intermittent exposure, we also failed to see significant increases in high band spectral power (Field by Hour interaction $F = 1.33$; $df = 5,120$; $P = 0.26$). HRV during exposure to the continuous magnetic field was not significantly different from HRV during sham control conditions.

DISCUSSION

The magnetic field-induced alterations in heart rate variability on healthy human volunteers that we have observed have proven reproducible in two independent studies of strong design and high statistical power conducted under strict double-blind conditions. Exposure of human volunteers to intermittent 60 Hz magnetic fields resulted in alterations in HRV. Spectral analyses of heart rate time series revealed statistically significant reductions in the power of the low-frequency band (0–0.1 Hz) in volunteers exposed to intermittent 200 mG magnetic fields, and a similar reduction in low band power when comparing sham vs. field exposure nights in the same individual. Power in this frequency band is associated with neural control of thermoregulation and blood pressure control.

A reduction in power in the low-frequency band could be due to one of two distinct processes: (1) a reduction in low-frequency *periodic* components in the heart rate spectrum (e.g., Traub–Hering–Meyer wave), or (2) a reduction in *aperiodic* components in the heart rate time series (e.g., chaotic variability). Chaos-theory based analyses of the heart rate time series provided a direct examination of chaotic variability. When chaotic variability is present, it appears in spectral analyses as very low frequency power. Analyses of heart rate time series using the Approximate Entropy (ApEn) statistic did not reveal effects of field exposure on this parameter. The combination of spectral and chaos analyses of these data indicated that the observed reduction in the power of the low-frequency band in subjects exposed to 200 mG magnetic fields most likely results from a reduction in low-frequency *periodic* components in the heart rate spectrum. Thus, intermittent exposure to 200 mG magnetic fields appears to result in an alteration of the normal nocturnal variability that is inherent in human cardiac rhythm. In addition, we also observed an increase in power at a spectral index of the normal respiratory arrhythmia in volunteers exposed to intermittent 200 mG magnetic fields in the first study.

The results obtained in Study 2 confirmed and extended our previous results. Spectral analysis of heart rate time series again revealed a statistically significant reduction in the power of the low-frequency band (0.0–0.1 Hz) in subjects exposed to 200 mG intermittent magnetic fields. The difference observed between sham control and magnetic field conditions was significant, as was the interaction between field conditions and time of night. Since systemic thermoregulation and blood pressure control are active at all times and are known to vary during the night, these results again suggest a field-induced alteration in an underlying normal physiological process.

Spectral analysis of heart rate time series also revealed a statistically significant increase in high band (0.15 Hz to 0.40 Hz) power in volunteers exposed to 200 mG intermittent magnetic fields. The power in this frequency band is associated with the natural respiration-induced alteration of heart rate. The interaction between field conditions and time of night was significant. Since respiration-induced sinus arrhythmia is active at all times, and is known to vary during the night, our results again suggest a field-induced alteration in an underlying normal physiological process.

Unlike previous results with intermittent magnetic field exposure, exposure to the continuous magnetic field did not significantly decrease low band power or increase high band power. This is important because it indicates that intermittency of magnetic field exposure may be an important parameter in the human cardiac responses that result from those exposures. Intermittent exposure resulted in statistically significant alterations in HRV, and these effects were reproducible in two separate studies with non-overlapping human volunteers conducted in two different years. In contrast, continuous field exposure had no observable effect on HRV.

Our analyses of the effects of exposure to intermittent fields (each study individually and combined) have revealed two significant effects in opposite directions, a *decrease* in power in the low spectral band and an *increase* in power in the high spectral band. It is important to note that virtually all clinical studies of HRV as a predictor of cardiovascular risk report that higher risk is associated with a reduction in low band power, as we have observed in this study. The data are less consistent for changes in high frequency power. Some investigators report reductions and others, minor or no changes in high frequency power [Kleiger et al., 1987; Hayano et al., 1990; Bernardi et al., 1992; van Ravenswaaij-Arts et al, 1993; Bigger et al., 1993; Winchell and Hoyt, 1996; Barron and Lesh, 1996; Huikuri et al., 1996]. In the Framingham study [Tsuji et al., 1994] reductions in power in several bands were statistically significant, but only the reduction in low frequency power was an independent predictor of cardiovascular risk. Thus, we do not observe exactly the same pattern of spectral changes seen in the clinical studies, since we observed increases in power in the high frequency band.

As a systemic response in an intact organism, HRV could be altered by the nature of the internal afferent sensory information relayed from the periphery to the central nervous system, by the central processing of that information, or by the nature of the efferent sympathetic and parasympathetic outflow from the central nervous system back to the heart. The pattern of alterations we observed has, to our knowledge, been reported only for HRV patterns associated with ‘‘Stage 2’’ sleep [Vaughn et al., 1995]. Our measures of HRV were obtained only during night sleep. Because we did not record electroencephalographic data, we do not know whether the HRV changes we saw are the result of increased Stage 2 sleep.

Finally, it should be noted that no volunteer has reported any cardiovascular difficulties associated with the night time exposures. The results, however, do indicate a possible interaction between certain types of magnetic field exposure and human physiological activity. In order to determine whether the observed physiological changes have any possible long-term health implications, it will be necessary to perform studies that address the limitations of the present research. These limitations include: (1) only one night of magnetic field exposure was examined in each study; (2) no data were collected to determine whether the observed physiological changes lasted beyond the night-time exposure period; and (3) all studies were carried out on healthy young men. In addition, research involving the simultaneous measurement of relevant brain, cardiac and respiratory indices is needed to narrow down the anatomical structures where field-related biophysical interactions could be sought. Experimental work is continuing in our laboratory to address these limitations and to explore the more complex interaction questions raised by these studies of magnetic field exposure on human heart rate variability.

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