Influence of static magnetic fields on pain perception and sympathetic nerve activity in humans

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Kuipers NT, Sauder CL, Ray CA. Influence of static magnetic fields on pain perception and sympathetic nerve activity in humans. J Appl Physiol 102: 1410–1415, 2007. First published December 28, 2006; doi:10.1152/japplphysiol.00734.2006.—Static and pulsed magnetic fields have been reported to have a variety of physiological effects. However, the effect of static magnetic fields on pain perception and sympathetic function is equivocal. To address this question, we measured pain perception during reproducible noxious stimuli during acute exposure to static magnets. Pain perception, muscle sympathetic nerve activity, mean arterial pressure, heart rate, and forearm blood velocity were measured during rest, isometric handgrip, postexercise muscle ischemia, and cold pressor test during magnet and placebo exposure in 15 subjects (25 ± 1 yr; 8 men and 7 women) following 1 h of exposure. During magnet exposure, subjects were placed on a mattress with 95 evenly spaced 0.06-T magnets imbedded in it. During placebo exposure, subjects were placed on an identical mattress without magnets. The order of the two exposure conditions was randomized. At rest, no significant differences were noted in muscle sympathetic nerve activity (8 ± 1 and 7 ± 1 bursts/min for magnet and placebo, respectively), mean arterial pressure (91 ± 3 and 93 ± 3 mmHg), heart rate (63 ± 2 and 62 ± 2 beats/min), and forearm blood velocity (3.0 ± 0.3 and 2.6 ± 0.3 cm/s). Magnets did not alter pain perception during the three stimuli. During all interventions, no significant differences between exposure conditions were found in muscle sympathetic nerve activity and hemodynamic measurements. These results indicate that acute exposure to static magnetic fields does not alter pain perception, sympathetic function, and hemodynamics at rest or during noxious stimuli.

exercise; complementary medicine; alternative medicine; cold pressor test

IN 1997 it was estimated that out-of-pocket expenses for complementary and alternative therapies totaled $27 billion in the United States (7). Kessler et al. (18) reported that demand for complementary and alternative therapies has increased over the last 50 years and will influence all facets of health care in future years. An understanding of the therapeutic efficacy and physiological mechanisms of action for many of these therapies is lacking. Magnets are one form of alternative therapy marketed to treat a variety of health conditions. In fact, sales of therapeutic magnets have been reported to be greater than one billion dollars worldwide (35).

Previous studies have found physiological interactions with magnetic fields. For example, time-varying electromagnetic fields produced by electrical currents are used to treat nonunified bone fractures (cf. Ref. 1). With regard to sympathetic function, time-varying electromagnetic magnetic fields can modify electrical activity in the brain (i.e., transcranial cortical magnetic stimulation). This change in electrical activity of the central nervous system can inhibit muscle sympathetic nerve activity (19) and increase skin sympathetic nerve activity (31). Magnetic devices sold to patients commonly utilize static magnetic fields generated by permanent magnets and not time-varying electromagnetic fields. Like time-varying electromagnetic fields, there is some evidence to suggest that static magnetic fields alter autonomic function in humans. A 2.0-T static magnetic field can increase cardiac cycle length, which may be caused by changes within the sinus node (15). A 0.4-T static magnetic field can alter skin blood flow in humans, possibly caused by alterations in calcium dynamics (21). In rabbits, there is evidence that a 1-mT static magnetic field alters vasomotion by influencing nitric oxide synthase activity and calcium dynamics (26). Likewise, exposure of the neck to a 5.5-mT static magnetic field had a restorative effect on vascular tone and blood pressure during blood pressure modulation by norepinephrine and nicardipine infusions in the rabbit (25). However, there are no data that have examined the effect of static magnetic fields on muscle sympathetic nerve activity at rest and during different physiological stressors in humans. Therefore, the first goal of this study was to determine the influence of static magnetic fields on cardiovascular and sympathetic function at rest and during physiological stress.

Use of static magnetic fields for healing and diminishing pain remains controversial. Studies examining the efficacy of treating pain with magnets have produced conflicting results, with some studies finding positive treatment effects and others not (6, 8, 10). Furthermore, studies examining pain perception and magnetic therapy have been scrutinized for poor study design (8). For example, studies examining pain in disease states are limited by the fact that disease-induced pain can be very variable. To address this limitation, we had healthy subjects perform three noxious physiological stressors that increase heart rate, blood pressure, and sympathetic outflow. These stimuli were exercise, muscle ischemia, and cold stress. These physiological stressors allow reproducible measures of pain perception to be obtained, allowing for better comparison across various treatment conditions (4, 5, 27). Moreover, exercise-induced and ischemic pain is common in patients suffering from peripheral artery disease and claudication. Therefore, the second goal of the present study was to investigate the influence of static magnetic fields on pain perception during noxious stimuli.

The strength of the static magnetic fields used in the present study was chosen based on what is commonly available in...
commercial devices used by the public and marketed as having health benefits. Because these magnets generate small static fields with limited tissue penetration, we hypothesized that the 0.06-T static magnetic fields would not alter cardiovascular responses or pain perception during the noxious stimuli. This study is the first randomized, double-blinded, placebo-controlled crossover experiment to test these physiological effects in humans.

METHODS

Subjects. Fifteen volunteers (8 men and 7 women) (age 25 ± 1 yr, weight 70.9 ± 3.2 kg, height 171.0 ± 2.4 cm) who were normotensive, did not smoke, and were not taking any medications participated in this randomized, double-blinded, placebo-controlled crossover study. Each subject was required to give signed written consent before participating in the study. All female subjects were tested in the same period of the menstrual cycle and not during ovulation or menstruation. The Institutional Review Board of the Pennsylvania State University College of Medicine approved the study.

Experimental design. Subjects participated in two testing sessions. During one testing session, subjects were placed supine on a rippled rubberthane mattress (Nikken, Ultra Kenkopad model 1225, Irvine, CA). The mattress was purchased from the manufacturer without their knowledge of this study. Within the mattress were magnets arranged in a grid composed of 21 rows separated by 7.5 cm. The rows alternated between four and five magnets. Each magnet was separated by 15.5 cm from adjacent magnets in the same row. The rows containing four and five magnets were staggered from the sides of the mattress by 21.5 and 13.5 cm, respectively. Each individual magnet was a small disk with a diameter of 1.5 cm. The north pole of the magnets faced toward the subjects. Using a Tesla meter (Bell, model 4048, Orlando, FL) with ±2.0% accuracy, we measured the magnetic field strength at the surface of a magnet to be 0.06 T. Vertically from the surface of the magnet, magnetic field measured 1 cm, 6.8 mT; 2 cm, 1.52 mT; 3 cm, 0.5 mT; 4 cm, 0.3 mT; 5 cm, 0.2 mT; 6 cm, 0.15 mT; 7 cm, 0.08 mT; 8 cm, 0.06 mT; 9 cm, 0.05 mT. At 9 cm, field strength was undetectable relative to Earth’s magnetic field. At the midway point between four magnets, magnetic field strength at the surface of the mattress was 0.1 mT. Going up vertically from the midway point, field strength measured 0.09 mT at 1 cm, 0.06 mT at 2 cm, and undetectable relative to Earth’s magnetic fields at distances greater than 3 cm. During the second testing session (2–7 days later), each subject was placed on an identical textured rubberthane mattress from which all magnets had been removed. Both the control and magnetic mattresses were enclosed in identical covers, thereby prohibiting the investigators and research participants from identifying the type of mattress. This sleeve remained on the mattress during all testing sessions. All other testing procedures and interventions were the same for the two sessions. The order of the two sessions was randomized. For blinding purposes, another researcher not involved in data analysis selected the mattress by flipping a coin. This investigator held the randomization key. All other investigators were blinded to the experimental conditions until completion of the study and data analysis.

Experimental protocol. Each subject was required to rest on the mattress for 1 h. During the rest period, subjects were instrumented for the study. Instrumentation and electrode insertion for sympathetic nerve recordings were done in the first 15 min of the rest period. After 1 h, variables were measured and the subjects then performed three interventions known to increase sympathetic outflow and act as noxious stimuli. The interventions used were 1) isometric handgrip, 2) postexercise muscle ischemia, and 3) cold pressor test. Each intervention consisted of an initial 5-min baseline followed by the intervention and a 3-min recovery period. During isometric handgrip, subjects squeezed a dynamometer with their nondominant hand for 2 min at 30% of their previously determined maximal voluntary contraction. Five seconds before the end of isometric handgrip, blood flow was occluded to the exercising arm by inflating an arm cuff to suprasystolic pressure for 2 min to induce postexercise muscle ischemia. The cold pressor test required that the subject’s nondominant hand be placed in a container of ice water for 2 min. During the cold pressor test the same tissue area was exposed to cold stress during each visit (30).

Measurements. Multifiber recordings of muscle sympathetic nerve activity were made by inserting a tungsten microelectrode into the peroneal nerve at the head of the fibula. A reference electrode was inserted subcutaneously 2–3 cm from the recording electrode. Both electrodes were connected to a differential preamplifier and then to an amplifier (total gain between 40,000 and 80,000), where the nerve signal was band-pass filtered (700–2,000 Hz) and integrated (time constant, 0.1 s) to obtain a mean voltage display of the nerve activity. A satisfactory recording of muscle sympathetic nerve activity was defined as spontaneous, pulse-synchronous bursts that increased during end-expiratory apnea and did not change during stroking of the skin or auditory stimulation (yell).

Mean forearm blood velocity was recorded on a beat-by-beat basis using a 4-MHz pulsed Doppler ultrasound probe (model 500M Multigun; Yonkers, NY) with Zero Crossing (Hokanson; Bellevue, WA) during rest and isometric handgrip. The probe was taped over the brachial artery proximal to the antecubital fossa on the nondominant arm. The distance between the medial epicondyle of the humerus and the probe was the same during both visits. Likewise, focus, depth, power, and gate of the probe were the same during both visits.

Because we cannot measure forearm blood velocity and brachial artery diameter simultaneously, we measured brachial artery diameters in four subjects at rest and during static handgrip exercise after 1 h of exposure to both conditions with high-resolution Doppler ultrasound (HDI 5000, ATL Ultrasound, Bothell, WA). A 5- to 12-MHz transducer was positioned longitudinally over the brachial artery. Arterial diameter measurements were made at end diastole (determined by ECG) by measuring the distance between near and far wall intima media.

Continuous measurements of mean arterial blood pressure and heart rate were made using a Finapres (Ohmeda, Louisville, CO). A three-lead system was used for recording an electrocardiogram during each study. All data were collected online (Maclab 16sp, ADI Instruments, Newcastle, Australia) for later offline analyses.

During isometric handgrip, postexercise muscle ischemia, and cold pressor test, subjects were asked to rate their perception of pain by using a visual analog scale with anchors of 0 and 10 every 15 s (5).

Analysis. All data were analyzed offline using Chart 5.4.2 software (ADI Instruments). Sympathetic bursts were identified from inspection of mean voltage neurograms. Muscle sympathetic nerve activity data are presented as changes in absolute burst frequency. A paired t-test was used to compare values at rest between the two exposure conditions. A one-within, one-between, repeated-measures ANOVA (time × condition) was used to examine the effects of exposure (magnet, placebo) on the dependent variables during isometric handgrip, postexercise muscle ischemia, and cold pressor test. Statistical significance was accepted at the $P < 0.05$ level for all analyses. All values are presented as means ± SE.

RESULTS

Rest. Hemodynamic and sympathetic results during exposure to the magnets and the placebo condition at rest are presented in Table 1. No significant differences were observed between muscle sympathetic nerve activity, mean arterial pressure, heart rate, and forearm blood velocity at rest after 1 h of exposure to the two conditions. Brachial artery diameters did
not differ at baseline between either exposure condition (0.39 ± 0.05 and 0.39 ± 0.05 cm for magnet and placebo, respectively).

**Isometric handgrip.** During isometric handgrip, pain perception (Fig. 1) and ratings of perceived exertion increased similarly with magnet and placebo exposure. Changes in muscle sympathetic nerve activity, mean arterial pressure, and heart rate during isometric handgrip are presented in Fig. 2. During isometric handgrip there was no time × condition interaction or main condition effect, but as anticipated there was a main time effect for increases in muscle sympathetic nerve activity, mean arterial pressure, and heart rate. No significant differences in forearm blood velocity existed between magnet exposure and placebo exposure during the first minute (Δ 1.1 ± 0.6 and 0.6 ± 0.4 cm/s, respectively) and the second minute (Δ 4.3 ± 0.8 and 2.6 ± 0.8 cm/s, respectively) of isometric handgrip. During handgrip there was no significant change in brachial artery diameter from baseline during either exposure condition. Because diameters did not differ between the two exposure conditions, blood flow velocity was used as a surrogate for blood flow.

**Postexercise muscle ischemia.** Pain perception (Fig. 1) increased significantly with time during magnet and placebo exposure, but no significant interaction or condition effect was observed. Muscle sympathetic nerve activity, mean arterial

<table>
<thead>
<tr>
<th>Variable</th>
<th>Magnets</th>
<th>95% Confidence Interval</th>
<th>Placebo</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>91 ± 3</td>
<td>86–97</td>
<td>93 ± 3</td>
<td>88–99</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>63 ± 2</td>
<td>58–68</td>
<td>62 ± 2</td>
<td>58–66</td>
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<tr>
<td>Forearm blood velocity, cm/s</td>
<td>3.0 ± 0.3</td>
<td>2.4–3.7</td>
<td>2.6 ± 0.3</td>
<td>2.0–3.2</td>
</tr>
<tr>
<td>Muscle sympathetic nerve activity, bursts/min</td>
<td>8 ± 1</td>
<td>6–10</td>
<td>7 ± 1</td>
<td>5–9</td>
</tr>
</tbody>
</table>

Values are means ± SE. There were no significant differences between experimental trials.

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**Fig. 1.** Changes in pain perception during isometric handgrip, postexercise muscle ischemia, and cold pressor test. Pain perception did not differ between magnets and placebo exposure conditions during all interventions. Values are means ± SE.

**Fig. 2.** Changes (Δ) from baseline in muscle sympathetic nerve activity (MSNA), mean arterial pressure (MAP), and heart rate during isometric handgrip (IHG) and postexercise muscle ischemia (PEMI). No significant differences between exposure conditions were found in any of the variables. Values are means ± SE.
pressure, and heart rate responses during postexercise muscle ischemia are presented in Fig. 2. All three measures were elevated from baseline levels, and no significant condition or interaction was found between treatment conditions.

Cold pressor test. Pain perception (Fig. 1) increased significantly with time during magnet and placebo exposure, and no significant interaction or condition effect was observed. Muscle sympathetic nerve activity, mean arterial pressure, and heart rate responses during the cold pressor test are presented in Fig. 3. During both exposure conditions, all three measures significantly increased with time, but no interaction or main condition effect was observed.

Placebo effect. At the completion of the second study, all subjects were asked if they could differentiate between the two exposure conditions. Twelve of fifteen subjects said they were unable to distinguish between the two exposure conditions. Of the three subjects who claimed a difference, only one properly determined what type of mattress they were on. Subjects were also asked verbally if the texture of the mattresses was different. Subjects reported no difference in the texture of the two mattresses.

Sex differences. There were no sex differences between the two exposure conditions for the measured variables.

### DISCUSSION

The goal of this study was to design a randomized, double-blinded, placebo-controlled crossover study to determine if static magnetic fields altered pain perception, cardiovascular responses, and sympathetic outflow during three reproducible physiological stressors. The major novel findings of this study are that acute exposure to 0.06-T magnets did not alter pain perception duringnoxious stimuli elicited by physiological stress or influence sympathetic and cardiovascular parameters at rest or during physiological stress.

We found that acute exposure to static magnetic fields, at strengths commonly used by the public, did not alter pain perception during exercise, muscle ischemia, and cold stress. Previous studies reported conflicting results regarding the impact static magnetic fields exert on pain perception (6, 8). Part of these inconsistencies may be attributable to the fact that prior studies examining pain perception as a result of injury or disease have not accounted for changes in perceived pain within the same subject over time. In the present study, three noxious stimuli were applied to each subject repeatedly, producing similar painful stimulation during the two exposure conditions. These findings suggest that differences reported in perceived pain in previous studies may be related to intra-individual differences to pain perception and not exposure to static magnetic fields. However, as the physiological cause of pain may differ from disease to disease and to physiological stress, the efficacy of static magnetic fields to reduce pain perception may depend on the origin and type of pain.

Previously, it has been observed that analgesics, such as morphine, can alter resting cardiovascular function, including decreasing resting muscle sympathetic nerve activity and arterial pressure and increasing heart rate (3). Exposure to static magnetic fields in the present study was not associated with increased muscle sympathetic nerve activity, heart rate, or blood pressure at rest. This finding suggests that if acute exposure to static magnetic fields alters pain perception, they do not have an influence on the endogenous opioid system.

Another mechanism by which magnets may alter pain perception was proposed by Weintraub et al. (36). Weintraub et al. suggested that magnetic fields decrease C-fiber afferent activity. However, because both Aδ-fiber and C-fiber activation modulate cardiovascular and sympathetic activity during exercise (17) and because no differences in any of these measures were observed during exposure to static magnetic fields, static magnetic fields of the strength used in the present study do not appear to alter Aδ- and C-fiber activity in humans.

Our finding that blood pressure at rest is not altered by static magnetic fields is in agreement with previous studies (11, 16, 33). However, the present study expands on these results by demonstrating that static magnetic fields have no effect on blood pressure responses to physiological stress. Furthermore, our results do not support the concept that static magnetic fields mediate changes in cardiovascular hemodynamics. We observed no changes in forearm blood flow either at rest or during physiological stress. This finding contrasts work by Okano and Ohkubo (26), who reported that a 0.001-T static magnetic field applied for 30 min altered pharmacologically induced vasodilation and vasoconstriction in the microcirculation of rabbits, and Ichioka et al. (14), who reported that an 8-T static magnetic field could alter skin blood flow in rats. Likewise, Mayrovitz

![Fig. 3. Changes from baseline in MSNA, MAP, and heart rate during cold pressor test. No significant differences were observed in any of the measured variables between exposure conditions. Values are means ± SE.](http://jap.physiology.org/DownloadedFrom/vol102/1006SE.html)
and Groseclose (21) reported reduced skin blood flow in humans during 0.4-T static magnetic field exposure. However, our results are supported by studies in humans that report no changes during exposure to magnets with strengths less than 1 T in both muscle and skin blood flow at rest (20, 23) and during deep inspiration (22). Likewise, wrapping the metacarpus region of the horse with 0.027-T magnets did not alter blood flow in that region (32). The finding of the present study supports the conclusion that exposure to static magnetic fields, at the strength commonly used in nominally therapeutic devices, do not elicit changes in local blood flow, blood pressure, or heart rate in humans at rest or during physiological stress.

Conflicting results have been reported on the effect of magnetic fields on nerve function. For example, several studies have reported that exposing nerves to 1.2- to 2.0-T static magnetic fields did not alter nerve conduction velocity, membrane potentials, transmembrane currents, action potential amplitudes, and refractory periods (9, 28, 29). Moreover, in humans exposure to either 1.0-T or 0.045-T static magnetic fields did not alter nerve conduction velocities (34, 36). In contrast, it has been reported that there is an increase in the excitability of motor nerves during exposure to a 1.0-T static magnetic field (12, 13) and that a 0.011-T static magnetic field could block action potentials in cultured mouse sensory cells (24). Our results clearly demonstrate that acute exposure to 0.06-T magnets does not alter muscle sympathetic nerve activity in humans at rest and during physiological stress.

The present study had several limitations. First, the acute exposure to the static magnetic fields used in the present study limits the ability to draw further conclusions about the effects of prolonged exposure. Weintraub et al. (36) did not report significant changes in diabetic neuropathy until the 3rd and 4th mo of daily exposure to 0.045-T static magnets, indicating that chronic exposure to static magnetic fields may be required before therapeutic effects can be observed. Second, the present study used only 0.06-T magnets arranged in a grid. It is possible that other magnet strengths may elicit different effects. Third, the mattress used in the present study placed subjects over magnets spaced throughout the whole body; therefore it cannot be ascertained if magnets were placed in the proper places to alter pain perception. However, the device utilized in the present investigation is available commercially and is marketed as having health benefits. Therefore, because of these reported effects, this mattress was chosen for our investigation. Future studies examining the influence of static magnetic fields on pain perception need to examine influences of exposure duration, dose responses, and placement of static magnetic fields. Different-strength magnetic fields may influence tissues differently because of the varying depths of field penetration. Finally, the noxious stimuli used in the present study produced only moderate pain levels. It is uncertain if more painful stimuli were applied if magnets might have an influence on perceived pain.

In summary, static magnetic fields do not alter pain perception, cardiovascular, or sympathetic responses to three distinct pain inducing physiological stressors in humans. Therefore, the purported therapeutic benefits of static magnets in reducing pain and altering physiological responses must be seriously questioned.

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GRANTS

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