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Brief Communication

Pain Control Using High-Intensity Pulsed Magnetic Stimulation

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High-intensity pulsed magnetic stimulation (HIPMS) non-invasively depolarizes neurones, which can be deeply embedded in local tissues. Trans- or subcutaneous electrical stimulation can produce analgesia. To test the hypothesis that similar analgesia could be obtained using HIPMS, analgesia was determined in ten blinded subjects following HIPMS. Analgesia was consistently produced in all subjects with long-lasting pain relief occurring in half of the cases. ©1993 Wiley-Liss, Inc.

Key words: analgesia, depolarization, neurogenic inflammation

INTRODUCTION

Clinical experience with trans- and subcutaneous electrical stimulation supports the proposition that electromotive perturbations affecting peripheral nerve trunks or nerve networks can facilitate the recovery of function in nerve-injured individuals [Ellis, 1987]. There are, however, a number of problems with this methodology, including the daily insertion of needles, poor accuracy in the repeatability of needle insertions, and scant data concerning the field/tissue interactions. Thus, a means of repeatedly hypo- or depolarizing a small volume of neural tissue non-invasively was sought.

d'Arsonval's results on magnetically induced visual phosphenes suggested that high-intensity pulsed magnetic stimulation (HIPMS) might provide a way of achieving repeatable non-invasive depolarizations of small neuronal volumes [d'Arsonval, 1896; Ellis, 1989].

Based on concurrent work with neuropeptides, it was further hypothesized that chronic pain was often mediated by aberrantly functioning small neural nets involved in self-perpetuated neurogenic inflammation [Ellis, 1990]. Electrical stimulation served to interrupt this neurogenically perpetuated inflammatory feedback loop. Thus, one could reasonably assume that HIPMS, by generating local tissue eddy currents, would be suitable in rhythmically depolarizing deep neural structures, otherwise accessible only through surgery [Geddes and Bourland, 1989].

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McRobbie, Barker, and others have constructed and used HIPMS diagnostically [McRobbie, 1985; Barker et al., 1985]. However, attempts to procure a similar device locally for therapeutic use proved futile until recently when a suitable stimulator was built in the Ukraine by former Soviet defense engineers. Informal experimentation using this stimulator has indicated the presence of the hypothesized analgesic effect.

Pain change was chosen as the experimental variable for several reasons. In previous work with electrical fields, it was noticed that pain reduction is usually the first symptom or sign to change with treatment. The extent of change, when properly measured, is easily determined, and there is the obvious social and economic utility of demonstrating a new pain-control methodology [Huskisson, 1974]. Thus, this study asks if HIPMS of specific intensity and duration can lessen pain.

MATERIALS AND METHODS

Population

Ten adult human subjects were studied following informed consent. Ages ranged from 38 to 68. There were five females and five males. All subjects had been symptomatic and stationary for at least 1 year with their specific pains. Medication/analgesic usage had remained constant over the last year in the five subjects using analgesics. Four subjects suffered from post-traumatic or post-operative low back pain, one from reflex sympathetic dystrophy, two from peripheral neuropathy, two from thoracic outlet syndrome, and one from endometriosis.

Apparatus

The magnetic stimulator is a handmade opto-electrically triggered capacitative discharge system fed by 220 volts. The maximum charge voltage is 500 volts. This device generates up to 1.45 Tesla, as measured by a coil and oscilloscope at the center of an 8-cm inductor/coil which is freely movable and can be easily applied to the surface of the subject. Repetition rates range from 10 impulses per min to 45 impulses per min. There is a corresponding reduction in magnetic field strength with increased frequency. Forty impulses per min with a field of 1.17 Tesla at the surface of the coil was chosen as the experimental regimen. The magnetic field decreases smoothly to 0.13 T at 50 mm from the surface of the coil. The pulsed magnetic field is critically damped with a 0.4 n-sec rise time and an exponentially decaying pulse lasting 250 microseconds as measured at 1% of its maximal height, as visualized on a coil and oscilloscope. The rate of change of magnetic flux exceeds the 9 kT per s threshold for neuronal depolarization [McRobbie, 1985].

This device has numerous safety features designed to prevent electric shock. The beginning and end of each treatment (10 min maximum because of inductor heating) is signaled by a buzzer. Each stimulation is accompanied by a large striking sound generated by the discharge circuitry as well as by the inductor.

Methods

All ten patients (there was one exception—see Results) received ten treatments. Treatments were given on successive weekdays. Thus, experimental and control

sessions were experienced identically by the subjects. The inductor housing was applied to the areas of maximal pain; the plane of the coil was parallel to the body surface. Four of these treatments, in a randomly determined order, were control (sham) treatment, during which the stimulator was turned on, making its appropriate noises, but the inductor applied to the subject was not connected to the stimulator. After two to three experimental sessions, the mild internal sensation could be recognized by most subjects. The analgesia response was remarkably similar over time despite the subject's familiarity with this sensation.

Prior to experimental and control sessions, each subject was instructed in the use of a visual analog scale on which he/she determined the extent of his/her pain, which was then recorded on a scale of 0–10 [Huskisson, 1974].

A blindfold was then applied so that the subject would not be able to tell if the inductor was connected to the stimulator or not. There were no audible differences to the subjects. Ten minutes of stimulation followed. The inductor was returned to its original position and the blindfold was removed. The visual analog pain scale was repeated and the results were again recorded.

RESULTS

One patient became pain-free after four experimental treatments. The other nine finished the full course of six experimental and four control treatments. Table 1 shows the average improvement in pain scores compared to the control scores for each patient. The maximal pain relief from any one experimental treatment was 5.2 and the minimum was 0.4. The maximum change in any one control (sham) treatment was 0.5 and the minimum was 0.

The average amount of pain relief following any one 10-min experimental treatment in all ten subjects was 1.86 with a standard deviation of 1.14. Controls showed an average pain relief of 0.19 with a standard deviation of 0.13. The difference in pain relief between experimental and control trials was statistically significant ($P < .0001$) using a 2-sample t-test for independent samples with unequal variances, 2-tailed [Snedcor and Cochran, 1967]. Pain relief was gradual over the 10-min treatment time and maximum pain relief occurred approximately 3 h post-treatment in most of the subjects as based on their verbal reports.

Two subjects were pain-free at the end of this protocol as judged by pain scale and subjective verbal reports. They both had been affected by long-lasting moderate to severe low back pain secondary to trauma and multiple operations. Their complete pain relief has lasted at least four months (last follow-up). Three other subjects continued to have partial pain relief at last follow-up. The remaining five subjects experienced pain relief lasting 8–72 h. Their original pain would then recur.

TABLE 1. Average Improvement in Pain Score on a Scale of 0–10

	Subject									
	1	2	3	4	5	6	7	8	9	10
Experimental	0.9	2.9	3.4	1.9	1.1	1.5	2.1	1.8	1.2	2.7
Control	0.1	0.23	0.27	0.3	0.18	0.13	0.28	0.2	0.1	0.18

DISCUSSION

HIPMS applied to painful areas on the human body can achieve significant pain control. Fifty percent of the subjects experienced lasting pain relief while the rest had consistent pain relief lasting 8–72 h. These results were significantly different from the blinded control (sham) results.

These results are most likely mediated by eddy currents induced in the exposed tissues [Marg, 1991]. This would have the effect of hypo- or depolarizing many of the neural structures affected. How can this account for pain control?

One possibility is that the magnetically induced perturbation elicits a sufficient antidromic stimulation to "quiet" the abnormal neural focus via C-fiber activation. Another is that local voltage gated receptors are activated, changing the abnormal neural excitation long enough to insure that function returns to a normal state. Yet a third is that the magnetically induced depolarization is sufficient to interrupt the neurogenic inflammatory cycle by entraining a very low frequency firing rate. Lastly, and more speculatively, the magnetic perturbation of intraneuronal magnetite might contribute to changing aberrant neural output [Kirschvink et al., 1992]. Others have speculated that direct magnetic effects can mechanically produce neuronal activation [Budinger et al., 1984].

These putative mechanisms are all based on the supposition that chronic pain is produced by local neurons firing abnormally and establishing a self-perpetuated neurogenic inflammation of their neuronal fields. This vicious cycle could be interrupted on a number of levels. Exactly how HIPMS elicits this response is unknown, but the mechanism is under active investigation.

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