Phantom Pain Reduction by Non-ionizing Electromagnetic Treatment

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Abstract

Based on our years of experience testing a technique called non-ionizing *Electromagnetic-Own-Signal-Treatment (EMOST)*, here, we present our preliminary results for phantom pain reduction by this method under clinical circumstances. Our preliminary results showed not only a reduction in phantom pain in patients but also a marked improvement in their sleep and mood quality after the treatments. Here, we also suggest that some of the important effects of our method may be achieved via the redox balance of the body and redox-related neural plasticity. However, low-frequency and intensity electromagnetic fields can potentially provide useful methods for the treatment of various problematic disorders and can play important roles in public health of the 21st century medicine.

Introduction

But the health effects of low-frequency and intensity electromagnetic fields (LFI-EMFs) are controversial, increasing evidence suggests that non-ionizing LFI-EMFs can influence various cell functions and are capable of initiating diverse healing processes, such as the delay of fractures, induction of analgesia, acceleration of wound re-epithelialization, inhibition of inflammatory processes, reduction of fatigue, improvement of multiple sclerosis and chronic pulmonary disease etc.

The EMOST device
(BioLabor-MCC HI 2.5.2)
is controlled by a personal computer

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Methods

Limb amputees were recruited at the National Institute for Medical Rehabilitation in Budapest, Hungary. The limb amputees (experimental amputees (n=10) and control amputees (n=5)) were randomized to receive either an active EMOST treatment or a sham treatment.

The EMOST device (*controlled by a personal computer*) operates with the non-linear, electromagnetic signals of the patient within a defined frequency range, i.e., from 1 Hz to 1 MHz. The parameters and exposure times can be pre-programmed. The electromagnetic intensity of the device is 0.1-10 microteslas.

Pain intensity was quantified upon admission using a 0-10 verbal numerical rating scale.

Photograph of an amputee during an EMOST treatment

Results 1

Patients	Phantom pain intensity before the treatments	Phantom pain intensity after six EMOST treatments	Interval between the amputation date and the EMOST treatments
I.	6	0	1 month
II.	7	3	1/2 year
III.	8	4	2 years
IV.	6	2	1 month
V.	1	0	8 years
VI.	3	2	1 month
VII.	7	2	1 month
VIII.	7	4	1 month
IX.	7	0	3 years
X.	7	6	5 years

This table summarizes the phantom pain intensity observed after six EMOST treatments. (The control group is not shown).

Results 2

The reduction of phantom limb pain by EMOST was statistically significant (*P < 0.05).

While our main goal was to reduce phantom limb pain by EMOST treatments, most of the patients also reported a marked improvement in their sleep and mood quality, or a reduction in the frequency of phantom pain after the treatments.

No improvements in the reduction of phantom limb pain or sleep and mood improvement were reported in the control group.

Discussion 1

Low-frequency and low-intensity electromagnetic fields redox processes and membrane-bound receptors

A growing body of evidence indicates that several effects of extremely low-frequency electromagnetic therapies may be explained (*or connected*) by redox regulation and membrane processes. Many experiments have provided evidence that reactive oxygen species and reactive nitrogen species and their derivatives act as essential signals in intracellular and intercellular communication.

The effect of LFI-EMFs on cell membranes and membrane-bound receptors can stimulate Ca²⁺-related pathways and free radical and redox-regulated processes.

Discussion 2

EMOST, Sleep and Redox-related Plasticity

The reason for phantom pain is not well understood, but most investigators agree that phantom pain and phantom sensations can originate from the central nervous system. According to the neuromatrix theory, the regular neurosignature may be responsible for painless phantom limb sensations, but phantom pain could be due to an anomalous reorganization of the neuromatrix.

There are emerging evidences that sleep can promote brain plasticity. It is possible that one of the major effects of the EMOST is to influence redox processes in cells and tissues. However, redox modulation has been recognized as a fundamental system in the regulation of the NMDA receptor. The NMDA glutamate receptor is the most important molecular structure in controlling synaptic plasticity and memory functions. Thus, some of the essential effects of the EMOST treatment can be achieved via the redox balance of the body and redox-related plasticity during sleep.

Some References

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