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Increases in microvascular perfusion and tissue oxygenation via pulsed electromagnetic fields in the healthy rat brain.

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Abstract

OBJECT: High-frequency pulsed electromagnetic field stimulation is an emerging noninvasive therapy being used clinically to facilitate bone and cutaneous wound healing. Although the mechanisms of action of pulsed electromagnetic fields (PEMF) are unknown, some studies suggest that its effects are mediated by increased nitric oxide (NO), a well-known vasodilator. The authors hypothesized that in the brain, PEMF increase NO, which induces vasodilation, enhances microvascular perfusion and tissue oxygenation, and may be a useful adjunct therapy in stroke and traumatic brain injury. To test this hypothesis, they studied the effect of PEMF on a healthy rat brain with and without NO synthase (NOS) inhibition.

METHODS: In vivo two-photon laser scanning microscopy (2PLSM) was used on the parietal cortex of rat brains to measure microvascular tone and red blood cell (RBC) flow velocity in microvessels with diameters ranging from 3 to 50 μm, which includes capillaries, arterioles, and venules. Tissue oxygenation (reduced nicotinamide adenine dinucleotide [NADH] fluorescence) was also measured before and for 3 hours after PEMF treatment using the FDA-cleared SofPulse device (Ivivi Health Sciences, LLC). To test NO involvement, the NOS inhibitor N(G)-nitro-l-arginine methyl ester (L-NAME) was intravenously injected (10 mg/kg). In a time control group, PEMF were not used. Doppler flux (0.8-mm probe diameter), brain and rectal temperatures, arterial blood pressure, blood gases, hematocrit, and electrolytes were monitored.

RESULTS: Pulsed electromagnetic field stimulation significantly dilated cerebral arterioles from a baseline average diameter of 26.4 ± 0.84 μm to 29.1 ± 0.91 μm (11 rats, $p < 0.01$). Increased blood volume flow through dilated arterioles enhanced capillary flow with an average increase in RBC flow velocity by 5.5% ± 1.3% ($p < 0.01$). Enhanced microvascular flow increased tissue oxygenation as reflected by a decrease in NADH

autofluorescence to 94.7% ± 1.6% of baseline ($p < 0.05$). Nitric oxide synthase inhibition by L-NAME prevented PEMF-induced changes in arteriolar diameter, microvascular perfusion, and tissue oxygenation (7 rats). No changes in measured parameters were observed throughout the study in the untreated time controls (5 rats).

CONCLUSIONS: This is the first demonstration of the acute effects of PEMF on cerebral cortical microvascular perfusion and metabolism. Thirty minutes of PEMF treatment induced cerebral arteriolar dilation leading to an increase in microvascular blood flow and tissue oxygenation that persisted for at least 3 hours. The effects of PEMF were mediated by NO, as we have shown in NOS inhibition experiments. These results suggest that PEMF may be an effective treatment for patients after traumatic or ischemic brain injury. Studies on the effect of PEMF on the injured brain are in progress.

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