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Reconstructive: Lower Extremity: Original Articles

Pulsed Electromagnetic Fields Accelerate Normal and Diabetic Wound Healing by Increasing Endogenous FGF-2 Release

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Abstract

Background: Chronic wounds, particularly in diabetics, result in significant morbidity and mortality and have a profound economic impact. The authors demonstrate that pulsed electromagnetic fields significantly improve both diabetic and normal wound healing in 66 mice through up-regulation of fibroblast growth factor (FGF)-2 and are able to prevent tissue necrosis in diabetic tissue after an ischemic insult.

Methods: *Db/db* and C57BL6 mice were wounded and exposed to pulsed electromagnetic fields. Gross closure, cell proliferation, and vascularity were assessed. Cultured medium from human umbilical vein endothelial cells exposed to pulsed electromagnetic fields was analyzed for FGF-2 and applied topically to wounds. Skin flaps were created on streptozocin-induced diabetic mice and exposed to pulsed electromagnetic fields. Percentage necrosis, oxygen tension, and vascularity were determined.

Results: Pulsed electromagnetic fields accelerated wound closure in diabetic and normal mice. Cell proliferation and CD31 density were significantly increased in pulsed electromagnetic field-treated groups. Cultured medium from human umbilical vein endothelial cells in pulsed electromagnetic fields exhibited a three-fold increase in FGF-2, which facilitated healing when applied to wounds. Skin on diabetic mice exposed to pulsed electromagnetic fields did not exhibit tissue necrosis and demonstrated oxygen tensions and vascularity comparable to those in normal animals.

Conclusions: This study demonstrates that pulsed electromagnetic fields are able to accelerate wound healing under diabetic and normal conditions by up-regulation of FGF-2-mediated angiogenesis. They also prevented tissue necrosis in response to a standardized ischemic insult, suggesting that noninvasive angiogenic stimulation by pulsed electromagnetic fields may be useful to prevent ulcer formation, necrosis, and amputation in diabetic patients.

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