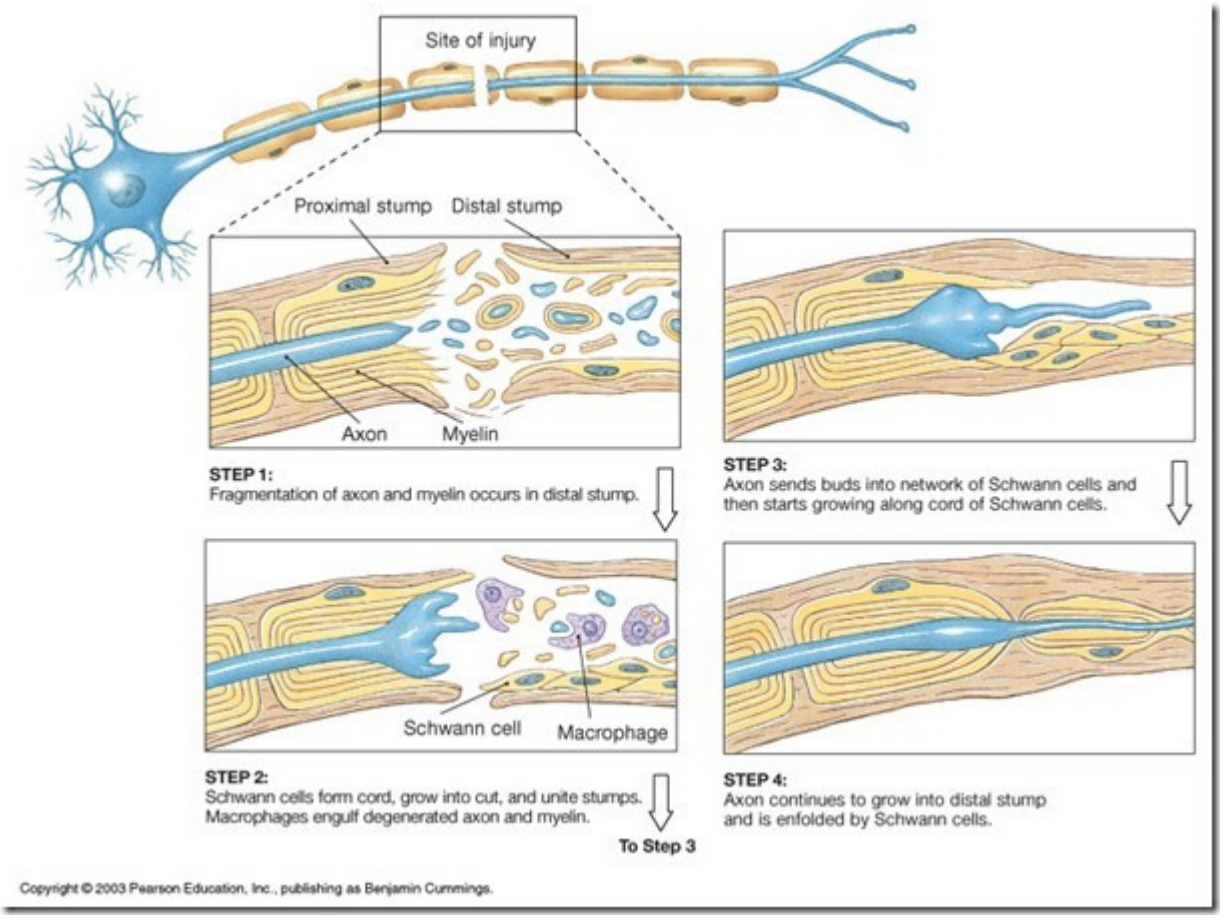
Medical PEMF Studies

NERVE REPAIR

Electromagnetic fields influence NGF activity and levels following sciatic nerve transection.



1. J Neurosci Res. 1999 Jan 15;55(2):230-7.

Longo FM(1), Yang T, Hamilton S, Hyde JF, Walker J, Jennes L, Stach R, Sisken BF.

Author information:
(1)Department of Neurology, UCSF/VAMC, San Francisco, California, USA.
LFM@itsa.UCSF.edu

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Pulsed electromagnetic fields (PEMF) have been shown to increase the rate of nerve regeneration. Transient post-transection loss of target-derived nerve growth factor (NGF) is one mechanism proposed to signal induction of early nerve regenerative events. We tested the hypothesis that PEMF alter levels of NGF activity and protein in injured nerve and/or dorsal root ganglia (DRG) during the first stages of regeneration (6-72 hr). Rats with a transection injury to the midthigh portion of the sciatic nerve on one side were exposed to PEMF or sham control PEMF for 4 hr/day for different time periods. NGF-like activity was determined in DRG, in 5-mm nerve segments proximal and distal to the transection site and in a corresponding 5-mm segment of the contralateral nonoperated nerve. NGF-like activity of coded tissue samples was measured in a blinded fashion using the chick DRG sensory neuron bioassay. Overall, PEMF caused a significant decrease in NGF-like activity in nerve tissue ($P < 0.02$, repeated measures analysis of variance, ANOVA) with decreases evident in proximal, distal, and contralateral nonoperated nerve. Unexpectedly, transection was also found to cause a significant ($P=0.001$) 2-fold increase in DRG NGF-like activity between 6 and 24 hr postinjury in contralateral but not ipsilateral DRG. PEMF also reduced NGF-like activity in DRG, although this decrease did not reach statistical significance. Assessment of the same nerve and DRG samples using ELISA and NGF-specific antibodies confirmed an overall significant ($P < 0.001$) decrease in NGF levels in PEMF-treated nerve tissue, while no decrease was detected in DRG or in nerve samples harvested from PEMF-treated uninjured rats. These findings demonstrate that PEMF can affect growth factor activity and levels, and raise the possibility that PEMF might promote nerve regeneration by amplifying the early postinjury decline in NGF activity.

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