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Case Blog

Title: Nerve Regeneration and Magnetic Resonance Energies

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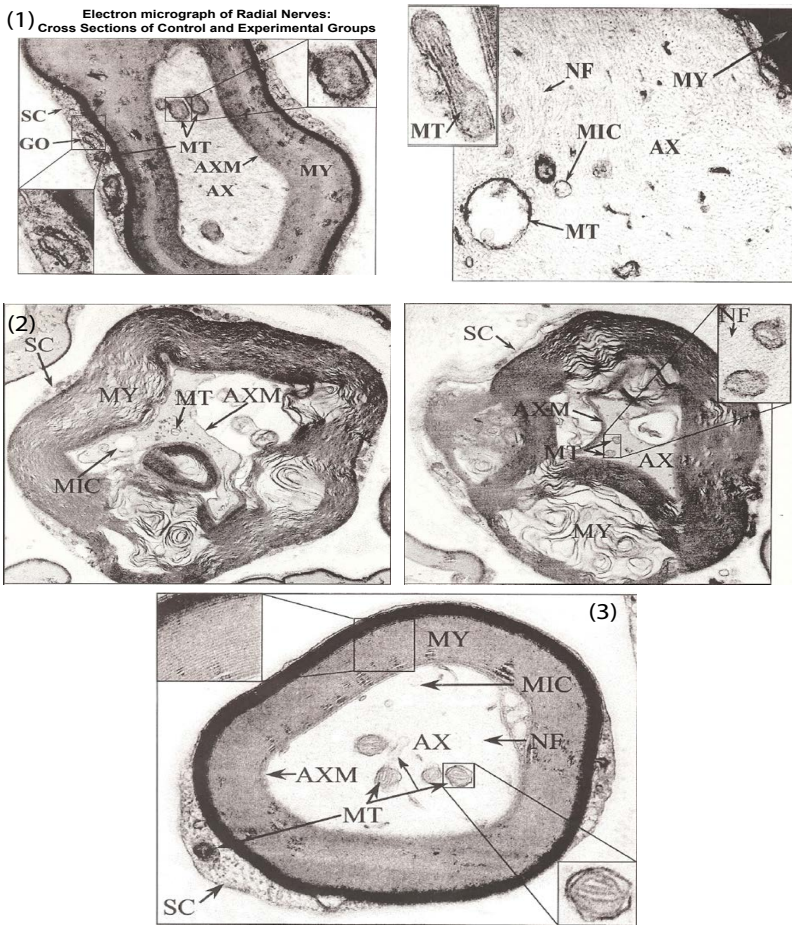

Figure 1: Electron micrograph (EM) of cross sections of radial nerve of mice from control Group 1, indicating Axon (AX), Axonal membrane (AXM), Golgi bodies (GO), Microtubule (MIC), Mitochondria (MT), Myelin sheath (MY), Neurofilament (NF), Schwann cells (SC), A. (Top) GO, MT, B. (Bottom Left) MT binary fission, C. (Bottom) NF. EM Magnification x 19,000. Scale Bar=1"µm".

Figure 2: Electron micrograph (EM) of cross sections of radial nerve of mice from IDPN treated Group 3 unexposed to EMF indicating Axon (AX), Axonal membrane (AXM), Microtubule (MIC), Mitochondria (MT), Myelin sheath (MY), Neurofilament (NF), Schwann cells (SC). A. (Top) MY, MT, SC, AXM, MIC, B. (Bottom) MY, AXM, MT, NF. EM Magnification x 10,000. Scale Bar=1 "µm" .

Figure 3: Electron micrographs (EM) of cross sections of radial nerve of mice from IDPN treated Group 2 exposed to EMF, indicating Axon (AX), Axonal membrane (AXM), Golgi bodies (GO), Microtubule (MIC), Mitochondria (MT), Myelin sheath (MY), Neurofilament (NF), Schwann cells (SC). A. (Top) MT, MY, AXM, NF, MIC, B. (Bottom Left) GO, MIC, C. (Bottom Right) GO, NF, MY, MT. EM Magnification A, B x 19,000, C x 4,800. Scale Bar=1 "µm" .

Nerve Regeneration Studies

It is noted that the energy state and bioelectric potential of nerves may be modulated by pico-Tesla electromagnetic fields (PTEMF's) [1-8]. For the following nerve regeneration studies, the field intensities, gradients and frequencies were calculated with the Jacobson Resonance equations; considering subcellular components vital for nerve function. Target molecules included nerve growth factors, dynein, kinesine, microtubule associated protein (MAP), neurofilaments, tubulin, cholinesterase, acetylcholine, and calmodulin. It was determined that the natural EMF profile for mice is in the micro gauss range (1 µG=100 pT). A sequence of extremely low-level EMF magnitudes with correspondent biological frequencies (<300 Hz) was employed.

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The effect of these low-level, non-ionizing EMF's on the restoration of forelimb grip strength and radial nerve ultrastructure was studied in mice with induced motor neuropathy.

The Control Group 1 (n=10), was neither poisoned nor treated with EMF. Groups 2 and 3 (n=20) were poisoned to induce motor neuropathy. Group 2 (n=10) after poisoning, was treated with EMF, while Group 3 was poisoned but not treated. Correlation of forelimb grip strength of all mice (n=30) at baseline was closely analogous (with no significant difference). Motor neuropathy was induced by administration of a neurotoxin (IDPN), in drinking water ad lib, for 9½ weeks. Forelimb grip strength of mice, as measured by a force gauge meter, declined to 47% compared to the Control Group 1, a significant difference (Group 2, $p<0.004$; Group 3, $p<0.00$). The normal age related increase in grip strength in the Control Group 1 was considered for the statistical analysis. The IDPN treated Group 3 (without EMF treatment) persisted to have a 56% decrease in grip strength; and radial nerve electron micrographs showed axonal demyelination, mitochondria in an orthodox state of conformation (inactive), and uneven dispersion of neurofilaments and microtubules. In contrast, IDPN treated Group 2 (with EMF exposure) exhibited axonal remyelination, condensed state of mitochondria (indicative of anabolic activity) and evenly dispersed neuro-filaments and microtubules, consistent with grip strength recovery.

EMF exposure was accomplished with the prototypical 18" Jacobson Resonator (Helmholtz configuration), built at the John C. Stennis Space Center by NASA engineers. Two mice at a time were held in two chambered (8 inch by 6 inch) Lucite perforated boxes. EMF's were applied twice weekly for 8½ weeks to Group 2 that resulted in 87% recovery. ($p<0.05$) of grip strength that was sustained after termination of exposure at an 82% level until the 27th week of observation. In the absence of EMF exposure, IDPN treated Group 3 had significantly low grip strength as compared to both EMF exposed Group 2 ($P<0.01$) and the control Group 1 ($p<0.000$) The neurotoxin effect persisted in Group 3 with 56% lower grip strength, as compared to Control Group 1. A consistent increase in grip strength, after termination of EMF exposure, was observed in Group 2 as it approached the level of the Control Group 1.

The gradual loss in forelimb grip test values in IDPN treated mice was indicative of a change in the nerve conduction in the forelimb. This was substantiated by an uneven distribution of axonal neuro-filaments, which determine growth of axonal diameters, and slow axonal transport for impulse conduction. The uneven dispersion of microtubules affected function in normal longitudinal growth and in fast axonal transport; and was a vital sign of nerve degeneration. The orthodox state of mitochondrial conformation indicates ADP deficiency. Reversal to a condensed state (EMF treated, Group 2) corresponds to an oxidative phosphorylation reaction and ATP synthesis, dependent on ADP, and proton permeability of mitochondria. Thus, Group 3, poisoned but not treated, showed reduced metabolic activity. Whereas, the condensed state of mitochondria in Group 2 (IDPN +EMF) indicated a metabolically active condition in axons and Schwann cells.

In a previous study at Cornell, excised pieces of sciatic nerves of mice in-vitro culture medium maintained a normal myelin sheath structure and grew longer and wider during EMF exposure. This could be attributed to Schwann cell activity, a source of neurotropin for nerve growth. Schwann cells produce polypeptide nerve growth factors (NGF). Nerve injury induces an increased output of NGF from Schwann cells.

In the *in vivo* study, two sources of NGF were available, one from CNS neurons and the other from Schwann cells. EMF exposure may have enhanced the action of Schwann cells in IDPN treated Group 2 mice. These Schwann cells indicated distinct Golgi bodies that are the source of NGF secretion, resulting in the remyelination of axons.

Indeed, a link between non-ionizing EMF and renormalized Schwann cell function indicated that a non-neuronal control in the regeneration and growth of peripheral nerve fibers are definitely possible, i.e. the microscopic or quantum field regulation through inter-atomic and inter-molecular communications networks which were regulated by EMF. The renormalized, physiological state of mitochondria, as observed, indicated its normal membrane permeability and a recovery of ATP synthesis essential for nerve growth and repair. Other ATP dependent processes such as the organization of neurofilaments and microtubules for axonal slow and fast transport systems were also restored. The molecular signaling across an axonal membrane may be extensively modified by a low energy level of an applied EMF, and is attainable by cooperative amplification that can restore cellular function. A role of PTEMF (for humans) in recovery from nerve injury, spinal cord traumas, and peripheral neuropathies may be postulated on the basis of selectively modulating neurotropins and their receptors with PTEMF resonant energies (Figures 1-3 excerpted from Medical Hypotheses, 60(6): 821-839).

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