The Relaxation Response

Helping the Health Care Provider to Restore Balanced Function With Whole Body Magnetic Resonance Therapy





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The Magnesphere[™]

"Not All Magnetic Fields Are Created Equal"

The Magnesphere[™] is the newest innovation in Magnetic Resonance Therapy. These are practical effective devices for clinical use. Studies, some of which are summarized in this publication, have shown our technology to be useful for the promotion of relaxation and health.

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Introduction

Not all magnetic fields are created equal. Certain intensities and frequencies are resonant to living systems, while others produce dissonance (also known as "noise"). All life on Earth evolved within the geomagnetic field, which is steady and relatively unchanging.

As life grew more complex, its natural magnetic profiles became more subtle, to establish bioresonance within the living system at the quantum level of structure and function. Therefore, certain "windows", or narrow ranges of intensity and frequency are natural to living systems for maintenance of communication, equilibrium and balance at atomic, molecular and cellular levels. For Earth's magnetic field, these ranges are about 0.3 gauss to about 0.6 gauss, averaging about 0.5 gauss in North America. Remarkably, these ranges are lower than a millionth of a gauss in humans, the most complex of all species.

It has been demonstrated that pico-Tesla range magnetic fields (pT MF's) are physiologic, and they are associated with the extremely low frequencies as seen in the electroencephalogram (EEG). David Cohen of MIT was the first to use a superconducting magnetometer (SQUID) to measure the normal magnetic fields emanating from the human brain and heart. He found that these naturally-occurring magnetic fields were in the pico-Tesla range, about 10 million times weaker than the geomagnetic field. At the other end of the strength spectrum, Magnetic Resonance Imaging (MRI) magnetic fields are about 10,000 gauss, strong enough to line up nuclei for diagnosis. Human magnetic fields are, naturally, billions of times weaker. It is interesting to note that most of the permanent magnets now used for magnet therapy range in strength from several gauss to several thousand gauss. This is out of the range for biological resonance with humans. By contrast, household appliances, cellular telephones, and computers emit MFs a bit weaker than the geomagnetic, at milligauss levels. These too are outside of the bioresonance windows, which makes them a source of dissonance. Therefore, when considering use of Pulsed Electromagnetic Magnetic Fields (PEMF's) for adjunctive therapy to conventional health care, it is advisable to employ naturally-occurring magnetic field intensities (i.e. pico-Tesla range MF's), or secondarily the geomagnetic, at frequencies within the EEG spectrum.

Dealing With Chronic and Acute Stress

"Stress" refers to a constraining force or influence. For example, forces are exerted when one body part presses on, pulls on, pushes against, or tends to compress or twist another body part. Stress produces a deformation or strain to cause tension, and it is generally a factor in disease causation. The tension can be physical, chemical or emotional. Stress alters an existent equilibrium.

The central nervous system (CNS) and peripheral nervous system (PNS) are significantly affected by stress, as are all other systems of the body, including the immune system. More specifically, stress adversely affects the autonomic nervous system (ANS), which innervates smooth and cardiac muscles, as well as glands. The visceral nervous system is a major component of the autonomic nervous system. Additionally, most peripheral nerves are composed of both motor and sensory neurons.

In the PNS, the nuclei (cell bodies) are clumped together as ganglia. Thus, in the ANS, the ganglia are sites of synapse intermediate between end organs and the spinal cord. The 31 pairs of spinal nerves that emerge through foramina eventually swell into dorsal root ganglia (where cell bodies of sensory neurons are located), and become rami to form the plexi: cervical, brachial, lumbar and sacral.

The ANS functions automatically, below conscious levels, but it is influenced by stress, be it physical or emotional. Effector cells of the ANS can either be stimulated or inhibited, and the ANS regulates a diversity of functions. These functions include blood vessel diameter, blood pressure, GI secretion, pupil size, micturition, sweating, kidney function, bronchi diameter, erectile function, basal metabolism, liver function, body temperature, and pancreatic function. ANS and CNS interact through centers within the hypothalamus, brain stem and spinal cord, integrating the brain's cortex and limbic systems with visceral inputs and the rest of the ANS activity.

Sympathetic stimulation activates the body in states of stress, fear and rage (the "fight or flight" reaction), and during physical strain. It accelerates norepinephrine and epinephrine. This increases heart rate, blood pressure, blood sugar, and stress. Of course, the result is more strain and tension, in a self-sustaining loop. Parasympathetic stimulation maintains body functions under quiet, day-to-day living conditions. It decreases heart rate; promotes digestion and absorption of food; and promotes regular heart rhythm, normal sleep patterns, kidney function and relaxation, thus promoting reduction of pain, tensions, anxiety and strain.

PEMF stimulation with pico-Tesla magnetic fields has been shown to enhance parasympathetic stimulation to enhance feelings of relaxation. By extension, it provides the basis for lowering blood pressure, restoring cardiac rhythmicity, reducing intraocular pressure, stimulating the intestines and urinary bladder, dilating of peripheral blood vessels, and promoting normal sleeping rhythms. Parasympathetic stimulation is a natural by-product when feelings of relaxation are enhanced, because cortical and limbic brain functions are integrated with ANS functions. It serves as a positive, safe and adjunctive modality, in much the same way that the Chiropractor adjusts the structures of the body to restore alignment, balanced function and homeostasis.. As a direct consequence of this combined approach to wellness, pain is oftentimes reduced from a great variety of etiologies (arthritis, fibromyalgia, headaches sprains, strains, contusions, etc.), with concomitant increase in energy levels.

Basic Science and Clinical Research

Numerous positive basic science research studies, (both in-vitro and in-vivo) have been reported utilizing pT MF's. The results include such benefits as the reduction of heart rate, the increase of the A-H interval, and the reduction of atrial fibrillation; the regeneration of cellular and subcellular structures (myelin sheath, mitochondria, Schwann cells, neurofilaments, and microtubules of peripheral nerves) secondary to induced neuropathy; modulation of afferent inputs that innervate spinal neurons; and wound healing. Double blind, randomized, placebo-controlled studies in Parkinson's disease, fibromyalgia, and osteoarthritis have also shown promising results.

Electromagnetic Theory: The Fundamental Mechanism

Einstein referred to the electromagnetic field as matter and to the gravitational field as space, predicting that these two fundamental realities of nature could be unified through an algebraic theory (Einstein A, 1956).

The theory underpinning the use of pico-Tesla magnetic field therapy is today based on the unified field equation of Jacobson Resonance:

 $Mc^2 = BvLq$

Where Mc² is the intrinsic energy of a target molecule in the body, and BvLg is the energy of the electromagnetic field interaction; the equal energies are connected by a wave of equal gravitational energy. When the energy of the wave is equal to the intrinsic rest mass energy of the molecule, then bioresonance is achieved, and all comparable molecules will coherently vibrate throughout the body. Coherence of vital quantum function may be restored, and amplified to the whole body system, because homeostasis is based upon equilibrium, balance and communications between molecules. The theory depicts biological structures to be piezoelectric, such as bone, keratin, collagen, nucleoproteins, genes, and cytoskeletal structures such as microtubules (an ordered structure of oriented dipoles). Piezoelectricity is known to be a property of semiconductive crystals, by which the application of mechanical force the generates a voltage (defined as energy per unit charge) or, conversely, the application of voltage results in the production of a mechanical force. That is to say, piezoelectricity results in the conversion of mechanical vibrations to electrical oscillations or the conversion of electrical oscillations to mechanical vibrations. An everyday example of this phenomenon can be found in the frequency-stabilized oscillators used in radio and television broadcasting. Another routine example is the operation of a quartz wristwatch.

The effect may be made continuous by altering the pressure, the temperature or the electromagnetic force. (Jacobson, J.I., 1987, 1991). Such interactions may be interpreted as photo-mechanic ponderomotive forces generated by coherent electric forces. Otherwise stated, photon-phonon transductions or non-linear vibrational waves (without losing energy) result from gravity waves (quantum gravity), secondary to electromagnetic forces.

Note that a photon refers to a quantum of light, whereas a phonon is the vibration of a crystal lattice structure.

Jacobson Resonance maintains that piezoelectricity is a property of all matter, as light is transduced to sound and vice versa. When comparing structures, the piezoelectric property is determined as a matter of degree, just as the insulative property of structures is a matter of degree, because all matter is electrically conductive to some extent.

A simple analogy is useful. Imagine that iron filings are sprinkled on a sheet of paper, and that the paper is placed over a bar magnet. Tapping the paper gently, we may view the orientation of filings in correspondence to the lines of force created by the bar magnet. If all the filings are so oriented, except one, we then have 3 possibilities to consider when we tap a second time:

- A) If the paper is then tapped too hard, more of the iron filings will be displaced from the oriented magnetic lines of force;
- B) If the paper is tapped too gently there will be no change in the positions of the filings; or
- C) If the paper is tapped with precisely the right force, the displaced iron filing will then snap right into place, and all the filings would then be properly ordered and aligned with the magnetic lines of force. This represents an example of resonance. And this analogy is representational of the atoms in a molecule (which are themselves permanent spinning magnetics) or molecules in a living system (which exhibit magnetic moments subject to electromagnetic fields).

Selected Review of Research Projects

The Jacobson Resonance Theory is the first to predict that pico-Tesla range magnetic fields are physiologic, and that these fields are vital for renormalizing the structure and function of tissue. All of the following studies utilized Jacobson Resonators (the forerunner of the Magnesphere⁾ to provide electromagnetic field parameters and protocols, derived from Jacobson Resonance Theory, whether the studies were basic science (in vitro and in vivo) or double blind clinical studies.

1. Electromagnetic Modulation of Autonomic Nervous System (ANS) Tonicity Magnetic Resonance Therapy (MRT) Target-Specific Stimulation of Vagal and/or Sympathetic Innervation

A) Heart Rate Variability (HRV)

A human heart rate varies with every heartbeat, through electro physiologic adjustments to maintain homeostasis. Our bodies are a collection of trillions of atoms in mutual cooperation creating reversible processes such that all the interactions oscillate about a steady state system to regulate homeostatic function. Remarkably, any change to the body, e.g. stressor, is automatically reflected in each heartbeat. Heart rate variability represents the variation of beat-to-beat intervals, i.e. R-R intervals. Electrocardiogram (ECG) reveals the electric signaling that originates from the heart. The electrochemical tone of the heart is intrinsically stimulated by ganglionated plexi on the heart, whereas the ANS inherently regulates heart function; in conjunction with unconscious and conscious activity of the brain. The distinctive feature of the ECG is the QRS complex, representing electromechanical transduction systems originating in the ventricles. At the beginning of ventricular contraction, the tricuspid and mitral valves close, causing the first heart sound, "lub." When the pressure in the right ventricle exceeds the diastolic pressure in the pulmonary artery (10mmHg) and the left ventricular pressure exceeds the diastolic pressure in the aorta (80mmHg), then the pulmonary and aortic valves open and ventricular ejection begins. Under normal resting conditions, the pressure reaches 25mmHg on the right side and 120mmHg on the left side. The stroke volume ejected from either ventricle is 70-90mL, while about 50mL of blood remain in either ventricle at the end of systole. As the ventricles begin to relax, the pressure drops rapidly. The pulmonary and aortic valves close, preventing back-flow into the ventricles from the arteries and causes the second heart sound, "dub." Also, the tricuspid and mitral valves open, and blood begins to flow from the atria into the ventricles. Because the body is a good conductor of electricity, potential differences generated by the depolarization and repolarization of the myocardium can be detected on the surface of the body, and recorded as an ECG. The P marks the peak of the P wave, the signature of atrial depolarization (atrial conduction). The QRS complex is the record of ventricular depolarization: the T wave, of ventricular repolarization. The short flat segment between S and T represents the refractory state of the ventricular myocardium; that between P and Q, a non-conductive phase of the A-V node, during which atrial systole can be completed.



time, ms

The R-R interval is illustrated as follows:



HRV indicates a fluctuation of heart rate from beat to beat around an average heart rate. Thus, an average heart rate of 72 beats per minute (bpm) does not mean that the interval between successive heartbeats would be exactly 0.8571 sec. Instead, successive beats fluctuate or vary from approximately 0.5 sec to about 1.67 sec.

HRV is dependent upon many factors, e.g. aerobic fitness. A well-conditioned heart HRV is usually large at rest, athletes having a pulse rate of 60 bpm or less. Whereas, HRV may vary from about 0.5 Hz to about 2 Hz.

Age, genetic disposition, bodily position, time of day (diurnal curve) and health status contribute to HRV. As a person exercises, heart rate rises and HRV naturally decreases. As intensity of exercise increases, HRV will decrease as the refractory period decreases. Emotional stress, anxiety and tension will decrease HRV, as HRV is regulated by the

autonomic nervous system. Parasympathetic activity decreases heart rate and increases HRV, whereas sympathetic stimulation increases heart rate and decreases HRV. Indeed, when HRV changes as in athletic training or under stressful conditions, it is a good indicator of need for training load accommodation, or the need to modulate such conditions as environmental stressors. The alteration of HRV indicates electro physiologic altered states of equilibrium in your body, and is useful as a window into your level of health; and capacity for accommodation to stimuli including intrinsic and extrinsic factors.

HRV is a biomarker showing great promise as a broad indicator of overall health and fitness, adaptability and capacity to maintain equilibrium. Therein lies the beauty of HRV: it offers a glimpse into the workings of our autonomic nervous systems. And, the HRV represents an objective marker telling us whether a treatment has successfully ameliorated conditions of stress and strain, both physical and mental. This is the reason why cardiologists have been utilizing HRV for decades to track the health and recovery of patients. HRV is a predictive indicator of the health of our hearts, the potential risk of heart attack, and other physiologic events. For example, HRV has great predictive value in that low HRV is associated with development of coronary artery disease, as well as the multiple metabolic syndromes, including diabetes, hypertension and high cholesterol. HRV is also an indicator of longevity, because a high HRV is associated with a graceful pattern of aging. Endurance, training load and high performance athletics require HRV monitoring, because greater variability between heartbeats indicates the body's ability to restore itself, maintain equilibrium and balance. Tracking HRV with regularity after establishing a baseline value can alert one to possible insufficiency in some area of physiology. Perhaps degrees of stressful situations can be monitored and consciously adjusted when we become aware of such necessity. It can tell us to start breathing deeply, suppress the racing thoughts daily stressors bring and generally slow down. While some people have that dialed in intuition about how they feel, not everyone can accomplish this, and by getting the instant objective feedback through HRV measurement, they can learn more about how their body works.

Modifications of HRV by specific interventions include: Beta-adrenergic blockade, antiarrhythmic drugs, scopolamine, thrombolysis, exercise training, biofeedback and wind instrument training.

Lowered HRV values from baseline may predict conditions such as myocardial infarction, congestive heart failure, diabetic neuropathy, depression, emotional arousal, post cardiac transplant issues, and poor survival in premature babies. High frequency (HF) activity associated with higher parasympathetic tone has been found to decrease under condition of acute time pressure, emotional strain, daily worry and elevated state anxiety (attributable to focused attention and motor inhibition); and post traumatic stress disorder (PTSD). PTSD reveals a low frequency (LF) HRV, indicative of high sympathetic tonicity. Respiration gives rise to waves in heart rate mediated primarily via the PSNS. It is thought that the lag in the baroceptor feedback loop may produce ten second waves in heart rate, associated with Myer waves of blood pressure. Various factors influencing input are the baroreflex, thermoregulation, hormones, sleep-wake cycles, meals, physical activity and stress. Thus, a decrease in parasympathetic activity or increased sympathetic activity reduces HRV.

High frequency (HF) activity (0.15 to 0.4 Hz) has been linked to PSNS activity. In this range, activity is associated with respiratory sinus arrhythmia (RSA), which is a vagally mediated modulation of heart rate, such that it increases during inspiration and decreases during expiration. Somewhat less is known about the physiologic inputs of low frequency, (LF) activity, the range of which is (0.04 to 0.15Hz); and is associated with high sympathetic tone. However, the foregoing may reflect mixtures of SNS and PSNS. In other words, inspiration slows the heart while expiration speeds the heart.

Methods of Analysis

Time domain methods are based on the beat to beat or NN (normal) intervals, that are analyzed to yield variables such as: 1) SDNN-The standard deviation of NN intervals, usually five minutes. SDANN is a measure due to cycles longer than five minutes, usually 24 hours. 2) RMSSD, is the root mean square of successive differences; the square root of the mean of the squares of the successive differences. 4) NN50- Is the number of pairs of successive NNs that differ by more than 50 ms. 5) pNN50 Is the proportion of NN50 divided by the total number of NNs. 6) NN20- Is the number of pairs of successive NNs that differ by more than 50 ms. 5) pNN50 Is the proportion of NN50 divided by the total number of NNs. 6) NN20- Is the number of pairs of successive NNs that differ by more than 20 ms. 7) EBC- Is the estimated breath cycles.

NN intervals also can be converted into a geometric pattern, such as the sample density distribution of NN interval durations; sample density distribution of differences between adjacent NN intervals. Resulting patterns of variability are judged via graphical analysis or geometric properties of resulting patterns. Sequence domain methods include power spectral density (PST), providing power distributions across frequencies. Finally, given the complexity of mechanisms regulating heart rate, it is reasonable to apply HRV analysis based upon methods of non-linear dynamics. Although HRV cannot be described as a chaotic process, application of chaotic globals to HRV have been shown to predict diabetes. The Poincare plot is the most commonly used non-linear method of analysis.

We note that the parasympathetic influence on heart rate is mediated by release of acetylcholine by the vagus nerve. Muscarinic acetylcholine receptors respond by an increase in cell membrane potassium conductance, to therein inhibit the hyperpolarization- activated "pacemaker" current. Whereas, the sympathetic influence on heart rate is mediated by epinephrine and norepinephrine, activating beta-adrenergic receptors. This results in cyclic AMP-mediated phosphorylation of membrane proteins to increase calcium currents. The result is acceleration of slow diastolic depolarization. Importantly, under resting conditions, vagal tone prevails. Vagal and sympathetic activities constantly interact. The SA node quickly hydrolyzes acetylcholine due to its being rich in acetylcholinesterase, but PNS effects exceed SNS effects, mostly because there is a cholinergic attenuation of responses to adrenergic stimulation, and there is cholinergic induced reduction of norepinephrine released in response to SNS activity.

B) Review of Experimental Studies

In accord with the diversity of possibilities presented herein, concerning biological effects secondary to application of non-ionizing extremely low intensity and low frequency EMF's, we point to studies conducted at the University of Oklahoma Health Sciences Center, Arrhythmia Research Institute.

Truly, it appears that a distinctive, and quite unique potential is unfolding from the radiological sciences for ameliorating the aging process and the effects therefrom.

In our initial experimental study we used 2 different sized Helmholtz coils to apply micro Gauss (μ G) levels of electromagnetic fields (EMFs) either to the vagosympathetic trunks or across the chest of anesthetized dogs. From previous reports on frequency analysis of heart rate variability, the sympathetic activity averaged 0.043 Hz. Using the Jacobson

(mc²=q_J v BL) and Cyclotron Resonance ($f = \frac{q B}{2 \rho m}$) equations, we calculated the

correspondent EMF amplitude value of 2.87×10^{-6} Gauss for parasympathetic activity. Applying these EMFs at the vagal trunks invasively or across the chest non-invasively, we found enhanced parasympathetic effects on the heart rate and atrioventricular conduction (AVC), both properties influenced by parasympathetic innervation. The maximal heart rate change in the experimental versus control groups was 29% versus 12% (P = 0.03). The same EMF stimulation decreased the voltage applied to the vagal trunks by 60% in the experimental group versus a 5% increase in the control group (P = 0.005). We note the right and left femoral veins were cannulated for delivery of fluids and anesthetics, and for the insertion of an electrode catheter which was advanced and positioned against the lateral atrial wall in the low right atrium for atrial pacing. Using another level of EMF, (amplitude, 0.34 μ G and 2 kHz) determined empirically, applied as above, there was a significant increase in atrial arrhythmias, including atrial fibrillation (AF) atrial premature depolarization, and atrial tachycardia, which could be suppressed by applied EMF, (2.87 μ G at 0.043 Hz). It should be pointed out that 2kHz is a non-physiologic frequency and 0.34 micro gauss is sympathomimetic.

A shortcoming of these studies was the lack of a mechanism underlying these responses to low level EMFs. Subsequently, a series of experimental studies have been published in which we used low-level vagosympathetic trunk electrical stimulation at levels 10% and 50%, which did not slow the heart rate or slow atrio ventricular, conduction (AVC). In an experimental model of induced AF, we found that the nerve clusters called ganglionated plexi (GP) found in specific vulnerable sites in the atria became hyperactive under the influence of excessive release of cholinergic (parasympathetic) and adrenergic (sympathetic) neurotransmitters. In this regard, Smith et al. tested the function of the GP, weeks after separation of the vagal and sympathetic nerves from the aforementioned structures. Not only did the intrinsic GP neurons remain viable but their responsiveness was enhanced. To emphasize this point, we severed the neural connection from the brain to the GP in experimental animals and found after 10 weeks there was a progressive increase in the occurrence of paroxysmal atrial fibrillation. Low-level vagal nerve stimulation markedly attenuated the hyperactive state of the GP, thereby suppressing AF. In a recent experimental study, we recorded the neural activity of the GP and found that several hours of induced AF caused a significant increase in the amplitude and frequency, whereas low level vagal nerve stimulation not only suppressed

AF propensity but also the increased amplitude and frequency of the hyperactive GP. A recent clinical report from our group has confirmed that low-level vagal nerve stimulation can mitigate AF in patients with the paroxysmal form of this arrhythmia. Since electrical stimulation of nerves induces its actions via release of chemicals called neurotransmitters, we found that a specific peptide, vasostatin-1 was released at low-levels of vagal nerve stimulation (50% below the voltage that causes slowing of the heart rate) and even at very low levels of vagal nerve stimulation (80% below the slowing threshold). Indeed, further studies in our experimental model of induced AF, showed that vasostatin-1 suppressed AF by inhibiting GP hyperactivity by an anti-autonomic action mediated by nitric oxide.

Returning to the earlier studies using low level EMFs to affect heart rate and rhythm, we inserted the molecular weight value for vasostatin-1 into the Jacobson and Cyclotron Resonance equations to derive the amplitude (0.034μ G) and frequency (0.952 Hz), respectively. Applying these EMFs at the vagal trunks and across the chest we found that these low level fields significantly suppressed AF and also decreased the amplitude and frequency of the neural activity of the hyperactive GP.

A fundamental question is: Why do these neural tissues (GP) on the heart become hyperactive in some of the population, in particular, disproportionately in persons from 60–80 years old? An early report by Kaijser and Sachs studied healthy women and men comprising groups, age 20-40, 40-60, and those between ages 60–80. Using simple procedures, such as handgrip and the response to the dive reflex test on heart rate and blood pressure, they found, "There seems to be only a moderate attenuation of autonomic cardiovascular responses to about 60 years, after which there is a more rapid decline." Since these cardiovascular responses are mediated by the autonomic innervation from the brain to the heart, these studies suggest that with age the control of the GP on the heart by the higher centers is markedly attenuated allowing these lower centers to become independently hyperactive. This would help to explain the increased incidence of AF in the elderly population compared to younger cohorts.

Now, although parasympathetic stimulation has been proposed as a generic approach to anti-aging, it must be pointed out that for various indications, e.g. obesity, fibromyalgia, hypertension in cases usually refractory to pharmacological intervention, esophageal reflux, autism, and diabetes...etc., sympathomimetic electromagnetic field intervention may be useful. Understanding physiologic mechanisms underlying various clinical symptomatology is important, and requires extensive ongoing research. Einstein once defined the grand aim of science as, "To cover the greatest number of empirical facts by logical deduction from the smallest possible number of hypotheses or axioms." He believed in a basic universal field in which the multifarious manifestations are merely particular ephemeral forms or conditions of state. Indeed, Lincoln Barnett said, "The urge to consolidate premises, to unify concepts, to penetrate the variety and particularity of the manifest world to the undifferentiated unity that lies beyond is not only the leaven of science, it is the loftiest passion of the human intellect.

Clinical Perspective

"Despite the superiority of catheter ablation to drug therapy, recurrence of atrial fibrillation (AF) or atrial tachycardia is still common for patients with paroxysmal AF who underwent AF ablation. The critical study described a noninvasive therapy to treat AF in a canine model of AF. When an extremely low-level electromagnetic field (0.034 micro gauss at 0.0952 hertz) was applied through a pair of Helmholtz coils across the canine chest wall, the atrial effective refractory period was prolonged, AF inducibility was reduced, and the neural activity in the ganglionated plexi was suppressed. The study demonstrates that AF can be potentially controlled noninvasively by PTEMF's within 2-3 hours after it was initiated. Early termination of AF by PTEMF's, if proven by clinical studies, will have a significantly positive impact on the use of antiarrhythmic drugs and oral anticoagulation therapy, particularly for patients in the earlier stage of paroxysmal AF. This approach, if effective, has a unique advantage that will not destroy any myocardium and perhaps avoid introducing iatrogenic atrial tachycardia's." [10]

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2. Effect of Magnetic Fields on Excised Mice Sciatic Nerves In-Vitro

Two studies were conducted at the Weill Medical College of Cornell University and then replicated at Fairleigh Dickson University, Department of Biological Sciences. These studies tested the effect of pT magnetic fields on excised sciatic nerves of mice, in-vitro. The Principle Investigator at Cornell was Prof. Brij B. Saxena, Director of Reproductive Endocrinology, Department of Obstetrics/Gynecology. The Principal Investigator at Fairleigh Dickinson was Professor Emeritus of Neuroscience, Anjali Saxena. In the first experiment four segments of the sciatic nerve, 1.5cm in length and 1.0 mm in width, were surgically excised under aseptic conditions from mice under ether anesthesia. Nerve segments were maintained in flasks and incubated. Growth medium was changed three times a week. A set of two nerve segments was exposed to 14 magnetic field settings 35 minutes each day for 5 days. The other set served as the unexposed control. Controlled cultures of excised nerve segments were removed from the incubator and also placed in between the Resonator coils (having the coils turned off).

In the second set of experiments, both sciatic nerves of 12 mice were excised aseptically to yield a total of 24 nerve segments. Six nerve segments served as the control. The remaining 18 nerves were divided into three experimental groups of 6 nerve segments each and exposed to magnetic fields daily for 15 days. Experimental groups were selected to determine (a) the effect of increased time of exposure on the dimensional and structural change of exposed nerves, (b) the effect of multiple versus single exposure on the same, and (c) a frequency and amplitude window that promotes greater effect on the growth and structure of the nerve segments. Three control and three experimental nerve segments were randomly chosen for DNA analysis. DNA was extracted for gel electrophoresis.

Results:

In the first experiment, the initial dimensions of both the control and experimental nerve segments were 15mm in length and 1mm in width. At the end of the experiment the ends of the exposed nerve segments showed significantly more dendritic growth than the control. Control segments remained at their initial length, while the exposed segments appeared to grow in length. The final dimensions of exposed nerve segments were 20mm in length and 1.5mm in width, a 33% increase in length and a 50% increase in width.

The response of the nerve segments in the second set of experiments to magnetic fields was similar to the first: the length and width of exposed nerve segments increased.

The light microscope observations revealed a normal and regular distribution of the axons in the exposed segments. In contrast the axons in the control nerve segments were fewer in number, had an irregular and abnormal shape, and had a very narrow band of myelination. Under the electron microscope, the exposed segments exhibited myelin sheaths with a normal distribution of microtubules and neurofilaments, Schwann cells with normal configuration, and mitochondria with condensed conformation indicative of anabolic activity. In contrast, nerve segments in the control group showed fragmented and disintegrated myelin sheath suggestive of lack of myelin synthesis, highly vacuolated Schwann cells, and mitochondria with inactive and orthodox conformation.

Results of the electrophoresis of DNA extracted from control and experimental nerves showed a similar single band of DNA in 0.8% and 2.0% agarose minigel, which suggest that the magnetic fields used in these experiments did not cause DNA degradation. Both the exposed and control nerves also stained negative for the MIB-I marker, implying that the magnetic fields used in this experiment did not lead to uncontrolled cell proliferation. (11)

3. Restoration of Nerve Ultrastructure and Recovery from Motorneuropathy in Mice by

Electromagnetic Field

The effect of electromagnetic fields (EMFs) on the restoration of forelimb grip strength and radial nerve ultrastructure was studied in mice with motorneuropathy induced by the administration of neurotoxin, 0.62% 3,3'Iminodipropionitrile (IDPN), in drinking water for 9 ½ weeks. Forelimb grip strength (lb) of mice as measured by a force gauge meter declined to 47% compared to the control group (p<0.004). The IDPN treated group without any EMF exposure persisted to have a 56% decrease in grip strength; and radial nerve electronmicrographs showed axonal demyelination, mitochondria in an orthodox state of conformation (nonfunctional) and uneven dispersion of neurofilaments and microtubules. In contrast, one IDPN treated group was treated with applied EMF (electromagnetic field) intensities and frequencies that were calculated on the basis of the mass of molecules vital to nerve function using $mc^2=bvLq$ and $f=qB/2 \square m$. During EMF exposure mice were held in a perforated Lucite box which was placed in a Resonator that generated the EMF between the centers of two 18" discs. 9" apart. containing copper coils in Helmholtz configuration. EMF was applied twice weekly for 8 $\frac{1}{2}$ weeks that resulted in as much as 87% recovery (p<0.05) of grip strength. This was sustained after the termination of exposure at an 82% level until the 27th week of observation. The EMF exposed group also exhibited axonal remyelination, functional condensed state of mitochondria, and evenly dispersed neurofilaments and microtubules consistent with grip strength recovery.

These results are the first to demonstrate a biological effect of EMF in vivo on the restoration of subcellular structures required for nerve impulse conduction and metabolism in nerves, and consequently a grip strength recovery from motorneuropathy, under controlled experimental conditions.

The studies were conducted at the Weill Medical College of Cornell University, and replicated at Fairleigh Dickinson University, School of Natural Sciences. (Saxena, A., Jacobson, JI, Saxena, B., et al, 2003) (6)

Radial Nerve Ultrastructure Presented In Electron Micrographs

Fig. 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 m.





Fig. 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), Mitrochondria (MT), Myelin sheath (MY), Neurofilament (NF), Schwanna cells (SC). A. (Top) MY, AXA, B. (Bottom) MY, AXM, MT, NF. EM Magnification x 10,000. Scale Bar = 1.





Fig. 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, $C \times 4,800$. Scale Bar = 1.





4. Efficacy and Safety of Low Level Electromagnetic Field Treatment in Parkinson's

Disease

Background: Small case series suggest extremely low level (Tesla) electromagnetic fields (EMF) may be useful in the treatment of Parkinson's disease (PD). No controlled studies have been previously reported.

Design/Methods: A single center, double-blind, randomized, placebo-controlled trial of EMF as an adjuvant to standard medical therapy in PD patients with motor fluctuations was performed in 12 subjects (6 per group). 24 sessions of 1.5 hour of total body EMF were administered over 8 weeks. Standardized motor and non-motor assessments were performed prior to treatment, at endpoint, and monthly for 3 months.

Control $24.52 + /-6.93$ $22.70 + /-6.84$ (-7%) $17.96 + /-8.94$ (-27%) PDQ-39 (Mobility)-motor Treatment $25.00 + /-24.75$ $13.33 + /-13.48$ (-47%) $12.92 + /-15.69$ (-48%) Control $35.42 + /-12.19$ $33.33 + /-16.56$ (-6%) $24.58 + /-16.00$ (-31%) PDQ-39 (ADL)-activities of daily living Treatment $25.00 + /-18.07$ $9.03 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) Control $22.92 + /-5.74$ $20.83 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-4.08$ (-56%) $5.17 + /-3.17$ (-47%) Control $11.67 + /-4.27$ $8.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + $	Kev Finding	<u>PIIOLILE</u> Baseline	Endpoint Week 8	8 10 1:1101 % Chg	Washout Week 16	% Chg
Control $24.52 + /-6.93$ $22.70 + /-6.84$ (-7%) $17.96 + /-8.94$ (-27%) PDQ-39 (Mobility)-motor Treatment $25.00 + /-24.75$ $13.33 + /-13.48$ (-47%) $12.92 + /-15.69$ (-48%) Control $35.42 + /-12.19$ $33.33 + /-16.56$ (-6%) $24.58 + /-16.00$ (-31%) PDQ-39 (ADL)-activities of daily living Treatment $25.00 + /-18.07$ $9.03 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) Control $22.92 + /-5.74$ $20.83 + /-6/97$ (-9%) $18.06 + /-3.40$ (-21%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-4.08$ (-56%) $5.17 + /-3.17$ (-47%) Control $11.67 + /-4.27$ $8.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) C	PDQ-39 (SI)-si	ummary index QoL				
PDQ-39 (Mobility)-motor Treatment 25.00 +/-24.75 13.33 +/-13.48 (-47%) 12.92 +/-15.69 (-48%) Control 35.42 +/-12.19 33.33 +/-16.56 (-6%) 24.58 +/-16.00 (-31%) PDQ-39 (ADL)-activities of daily living Treatment 25.00 +/-18.07 9.03 +/-6.67 (64%) 17.36 +/-14.05 (-31%) PDQ-39 (ADL)-activities of daily living Treatment 22.92 +/-5.74 20.83 +/-6/97 (-9%) 18.06 +/-3.40 (-21%) PDQ-39 (BD)-body discomfort/pain Treatment 27.78 +/-15.52 19.45 +/-6.80 (-30%) 16.67 +/-13.95 (-40%) Control 22.22 +/-14.59 25.00 +/-20.41 (+13%) 25.00 +/-22.98 (+13%) Back Depression Inventory II-depression Treatment 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%)	Treatment	20.01 +/-12.26	11.61 +/-7.44	(-42%)	10.86 +/-9.47	(-46%)
Treatment $25.00 + /-24.75$ $13.33 + /-13.48$ (-47%) $12.92 + /-15.69$ (-48%) Control $35.42 + /-12.19$ $33.33 + /-16.56$ (-6%) $24.58 + /-16.00$ (-31%) PDQ-39 (ADL)-activities of daily living Treatment $25.00 + /-18.07$ $9.03 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) Control $22.92 + /-5.74$ $20.83 + /-6/97$ (-9%) $18.06 + /-3.40$ (-21%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $29.67 + /-7.94$ $23.83 + /-11.44$ (-20%) $27.67 + /-11.55$ (-7%) FSS-fatigue Treatment $34.44 + /-10.23$ $26.67 + /-9.29$ (-22%) $28.00 + /-8.99$ (-18%)	Control	24.52 +/-6.93	22.70 +/-6.84	(-7%)	17.96 +/-8.94	(-27%)
Control $35.42 + /-12.19$ $33.33 + /-16.56$ (-6%) $24.58 + /-16.00$ (-31%) PDQ-39 (ADL)-activities of daily living Treatment $25.00 + /-18.07$ $9.03 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) Control $22.92 + /-5.74$ $20.83 + /-6/97$ (-9%) $18.06 + /-3.40$ (-21%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $21.67 + /-7.94$ $23.83 + /-11.44$ (-20%) $27.67 + /-11.55$ (-7%) SES-fatigue Treatment $34.44 + /-10.23$ $26.67 + /-9.29$ (-22%) $28.00 + /-8.99$ (-18%)	PDQ-39 (Mob	ility)-motor				
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Treatment $25.00 + /-18.07$ $9.03 + /-6.67$ (64%) $17.36 + /-14.05$ (-31%) Control $22.92 + /-5.74$ $20.83 + /-6/97$ (-9%) $18.06 + /-3.40$ (-21%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-4.08$ (-56%) $5.17 + /-3.17$ (-47%) Control $11.67 + /-4.27$ $8.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $29.67 + /-7.94$ $23.83 + /-11.44$ (-20%) $27.67 + /-11.55$ (-7%) FSS-fatigue Treatment $34.44 + /-10.23$ $26.67 + /-9.29$ (-22%) $28.00 + /-8.99$ (-18%)	Control	35.42 +/-12.19	33.33 +/-16.56	(-6%)	24.58 +/-16.00	(-31%)
Control $22.92 + /-5.74$ $20.83 + /-6/97$ (-9%) $18.06 + /-3.40$ (-21%) PDQ-39 (BD)-body discomfort/pain Treatment $27.78 + /-15.52$ $19.45 + /-6.80$ (-30%) $16.67 + /-13.95$ (-40%) Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depression Treatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily living Treatment $9.83 + /-6.34$ $4.33 + /-4.08$ (-56%) $5.17 + /-3.17$ (-47%) Control $11.67 + /-4.27$ $8.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motor Treatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $21.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $29.67 + /-7.94$ $23.83 + /-11.44$ (-20%) $27.67 + /-11.55$ (-7%) FSS-fatigue Treatment $34.44 + /-10.23$ $26.67 + /-9.29$ (-22%) $28.00 + /-8.99$ (-18%)	PDQ-39 (ADL)	-activities of daily	living			
PDQ-39 (BD)-body discomfort/pain Treatment 27.78 +/-15.52 19.45 +/-6.80 (-30%) 16.67 +/-13.95 (-40%) Control 22.22 +/-14.59 25.00 +/-20.41 (+13%) 25.00 +/-22.98 (+13%) Back Depression Inventory II-depression Treatment 12.33 +/-4.76 6.50 +/-5.32 (-47%) 5.83 +/-4.02 (-53%) Control 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Treatment	25.00 +/-18.07	9.03 +/-6.67	(64%)	17.36 +/-14.05	(-31%)
Treatment 27.78 +/-15.52 19.45 +/-6.80 (-30%) 16.67 +/-13.95 (-40%) Control 22.22 +/-14.59 25.00 +/-20.41 (+13%) 25.00 +/-22.98 (+13%) Back Depression Inventory II-depression (+13%) 25.00 +/-22.98 (+13%) Back Depression Inventory II-depression (-47%) 5.83 +/-4.02 (-53%) Control 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Control	22.92 +/-5.74	20.83 +/-6/97	(-9%)	18.06 +/-3.40	(-21%)
Control $22.22 + /-14.59$ $25.00 + /-20.41$ $(+13\%)$ $25.00 + /-22.98$ $(+13\%)$ Back Depression Inventory II-depressionTreatment $12.33 + /-4.76$ $6.50 + /-5.32$ (-47%) $5.83 + /-4.02$ (-53%) Control $12.17 + /-7.25$ $12.00 + /4.94$ (-1%) $11.50 + /-5.89$ (-6%) UPDRS II (On)-activities of daily livingTreatment $9.83 + /-6.34$ $4.33 + /-4.08$ (-56%) $5.17 + /-3.17$ (-47%) Control $11.67 + /-4.27$ $8.33 + /-2.25$ (-28%) $8.83 + /-2.48$ (-24%) UPDRS III (On)-motorTreatment $23.50 + /-12.99$ $14.00 + /-7.56$ (-40%) $15.67 + /-6.83$ (-33%) Control $29.67 + /-7.94$ $23.83 + /-11.44$ (-20%) $27.67 + /-11.55$ (-7%) FSS-fatigueTreatment $34.44 + /-10.23$ $26.67 + /-9.29$ (-22%) $28.00 + /-8.99$ (-18%)	PDQ-39 (BD)-	body discomfort/pa	ain			
Back Depression Inventory II-depression Treatment 12.33 +/-4.76 6.50 +/-5.32 (-47%) 5.83 +/-4.02 (-53%) Control 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Treatment	27.78 +/-15.52	19.45 +/-6.80	(-30%)	16.67 +/-13.95	(-40%)
Treatment 12.33 +/-4.76 6.50 +/-5.32 (-47%) 5.83 +/-4.02 (-53%) Control 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Control	22.22 +/-14.59	25.00 +/-20.41	(+13%)	25.00 +/-22.98	(+13%)
Control 12.17 +/-7.25 12.00 +/4.94 (-1%) 11.50 +/-5.89 (-6%) UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Back Depressi	on Inventory II-dep	pression			
UPDRS II (On)-activities of daily living Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Treatment	12.33 +/-4.76	6.50 +/-5.32	(-47%)	5.83 +/-4.02	(-53%)
Treatment 9.83 +/-6.34 4.33 +/-4.08 (-56%) 5.17 +/-3.17 (-47%) Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Control	12.17 +/-7.25	12.00 +/4.94	(-1%)	11.50 +/-5.89	(-6%)
Control 11.67 +/-4.27 8.33 +/-2.25 (-28%) 8.83 +/-2.48 (-24%) UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	UPDRS II (On)	-activities of daily	living			
UPDRS III (On)-motor Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Treatment	9.83 +/-6.34	4.33 +/-4.08	(-56%)	5.17 +/-3.17	(-47%)
Treatment 23.50 +/-12.99 14.00 +/-7.56 (-40%) 15.67 +/-6.83 (-33%) Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Control	11.67 +/-4.27	8.33 +/-2.25	(-28%)	8.83 +/-2.48	(-24%)
Control 29.67 +/-7.94 23.83 +/-11.44 (-20%) 27.67 +/-11.55 (-7%) FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	UPDRS III (On)-motor				
FSS-fatigue Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Treatment	23.50 +/-12.99	14.00 +/-7.56	(-40%)	15.67 +/-6.83	(-33%)
Treatment 34.44 +/-10.23 26.67 +/-9.29 (-22%) 28.00 +/-8.99 (-18%)	Control	29.67 +/-7.94	23.83 +/-11.44	(-20%)	27.67 +/-11.55	(-7%)
	FSS-fatigue					
Control 35.17 +/-8.93 33.33 +/-9.65 (-5%) 35.33 +/-19.29 (+.5%)	Treatment	34.44 +/-10.23	26.67 +/-9.29	(-22%)	28.00 +/-8.99	(-18%)
	Control	35.17 +/-8.93	33.33 +/-9.65	(-5%)	35.33 +/-19.29	(+.5%)

Kev Findings of Pilot II results: Baseline to Endpoint:

Results:

The treatment group demonstrated significant improvement over placebo after 8 weeks of therapy as follows: Scale, absolute point reduction, % improvement vs % improvement placebo (unless noted all results p < .05): UPDRS II(ON) 5.5, 56% vs 28%: UPDRS III(ON) 9.5, 40% vs 20%, p5.054; PDQ-39(SI) 8.4, 42% vs 7%; PDQ-39(MOB) 11.67, 47% vs 6%; PDQ-39(ADL) 15.97 pts, 64% vs 9%; PDQ-39(BD) 8.33 pts, 30% vs -13%; Beck Depression Inventory II 5.73 pts, 47% vs 1%; Fatigue Severity Scale 7.66pts, 22% vs 5%, p5.12; Finger Taps (ON) 67 taps. 25% vs -5%. It is important to note that improvement on several scales persisted up to 2 months post treatment. No treatment related adverse events were reported.

Conclusions:

Low-level EMF may improve motor and non-motor features of PD beyond that achieved with standard medical therapy.

These effects are long-lasting. Larger placebo-controlled studies have been undertaken to confirm and further investigate the benefit of this unique, noninvasive and potentially-promising therapy.

5. Effects of Low Intensity and Low Frequency Electromagnetic Field Stimulation (EMFS) on Thoracic Spinal Neurons Receiving Noxious Cardiac and Esophageal Inputs

Summary:

Many patients suffering from angina pectoris are refractory to surgical and pharmacological treatments. In order to improve the quality of life, alternative methods must be developed to relieve the pain in these patients. Because low intensity electromagnetic fields provide substantial levels of pain relief in patients with different sources of chronic pain, it was proposed that electromagnetic field stimulation might be used to reduce the pain of angina pectoris. To test the effects of EMF stimulation in an animal model, small Helmholz coils were placed on both sides of the chest of anesthetized rats. A mixture of algesic chemicals that are usually released during ischemic episodes of the heart was injected into the pericardial sac to activate cardiac nociceptive afferent fibers. During the injections, extracellular action potentials were recorded from cells in the upper thoracic spinal cord. Increased cell activity during the chemical injection was interpreted as a response to a nociceptive cardiac stimulus. We found that the responses of a population of thoracic spinal neurons to the nociceptive stimulus were reduced when pico-Tesla EM field stimulation (Jacobson Resonancederived signal parameters) was applied for 40 minutes. The activity often remained suppressed for up to two hours after terminating the stimulations.

These results show that EM field stimulation can modify nociceptive cardiac afferent information that affects the processing of spinal neurons. This leads to the suggestion that this technique might be used in the future to reduce cardiac pain in patients who are refractory to surgical and pharmacologic treatments. (8, 24)

6. Low Amplitude, Extremely Low Frequency Magnetic Fields for the Treatment of Osteoarthritic Knees: A Double-Blind Clinical Study

Participating Institutions:

Institute of Theoretical Physics and Advanced Studies for Biophysical research, The Perspectivism Foundation Jupiter Fla.

National Medical Research Institute, Department of Medical Physics and Neuromagnetics Boca Raton, Fla.

Department of Medicine, Cardiac Arrhythmia Research Institute, University of Oklahoma Health Sciences Center, Oklahoma City, Okla.

Department of Obstetrics/Gynecology Division of Reproductive Endocrinology, Cornell University Weill College of Medicine, New York, NY.

Prototyping Laboratory, John C. Stennis Space Center, Miss.

Context:

Noninvasive magnetotherapeutic approaches to bone healing have been successful in past clinical studies.

Objective:

To determine the effectiveness of low-amplitude, extremely low frequency magnetic fields on patients with knee pain due to osteoarthritis.

Designs:

Placebo-controlled, randomized, double-blind clinical study.

Setting:

4 outpatient clinics

Participants:

176 patients were randomly assigned to 1 of 2 groups: the placebo group (magnet off) or the active group (magnet on).

Intervention:

6-minute exposure to each magnetic field signal using 8 exposure sessions for each treatment session, the number of treatment sessions totaling 8 during a 2-week period, yielded patients being exposed to uniform magnetic fields for 48 minutes per treatment session 8 times in 2 weeks. The magnetic fields used in this study were generated by a Jacobson Resonator, which consists of two 18-inch diameter (46-cm diameter) coils connected in series, in turn connected to a function generator via an attenuator to obtain the specific amplitude and frequency. The range of magnetic field amplitudes used were in the pico-Tesla range.

Outcome Measures:

Each subject rated his or her pain level from 1 (minimal) to 10 (maximal) before and after each treatment, and 2 weeks after treatment. Subjects also recorded their pain intensity in a diary while outside the treatment environment for 2 weeks after the last treatment session (session 8) twice daily: upon awakening (within 15 minutes) and upon retiring (just before going to bed at night).

Results:

Reduction in pain after a treatment session was significantly (P<.001) greater in the magnet-on group (46%) compared to the magnet-off group (8%).

Conclusion:

Low-amplitude, extremely low frequency magnetic fields are safe and effective for treating patients with chronic knee pain due to osteoarthritis. (25)

7. Effects of pico-Tesla electromagnetic field treatment on wound healing in rats

The following study was executed at the College of Veterinary Medicine, Mississippi State University. Jacobson's Protocols and the Jacobson pico-Tesla electromagnetic therapy unit were supplied by Jacobson Resonance Enterprises, Inc.
C. Todd Trostel, DVM: Ron M. McLaughlin, DVM, DVSc; John G. Lamberth, PhD; Robert C. Cooper, DVM, MS; Steven H. Elder, PhD; Roy R. Pool, DVM, PhD; Cheng Gao, DDS, MS; Joseph A. Cromiak, PhD; Carolyn R. Boyle, PhD.

Objective:

To evaluate the effects of a pico-Tesla electromagnetic field (PTEF) on healing of sutured and open skin wounds and clinicopathologic variables in rats.

Animals:

64 male Fischer 344 rats

Procedure:

An incision made in the dorsal aspect of the neck was sutured (n= 32) or left open to heal (32) in each group. 16 rats were not PTEF treated (controls). Wound treatment consisted of exposure to a PTEF once daily. Rats in each group were euthanatized at days 2, 4, 7, and 14. Wounds were evaluated via tensiometry (sutured wounds), digital planimetry (open wounds), laser Doppler perfusion imaging, bacteriologic culture, and histologic examination. Blood samples were collected from all rats for analysis.

Results:

At day 14, sutured wounds in PTEF treated rats were stronger (ultimate stress) and tougher (strain energy) than were sutured wounds in control rats. Open wounds in PTEF treated rats contracted more quickly at days 2 and 4 than did those in control rats. Compared with control wounds, histologic changes (indicative of improved healing) in sutured and open wounds in PTEF treated rats were detected as early as day 4. Laser Doppler perfusion measurements, results of CBCs, serum biochemical analyses, and bacteriologic cultures were not different between groups.

Conclusions and Clinical Relevance:

Exposure to the PTEF caused no adverse effects on clinicoathologic, histologic, or bacteriologic variables tested in this study.

It appears that PTEF is a safe form of adjuvant treatment for wounds and speeds contraction of open wounds. (9)

In Vitro Cancer Cell Studies

8. The following studies were conducted from (2000), by the Department of Basic

Sciences, Veterinary Medical Research, Mississippi State University

The principle investigator was Prof. Cody Coyne. The Jacobson Resonator was utilized, and pico-Tesla electromagnetic field (PTEMF) signal parameters were derived from Jacobson's Equation (in collaboration with Prof. Jerry Jacobson).

Preliminary Investigations and Experimental Findings:

Preliminary Investigations: During the first replicate study a total of twelve multifrequency PTEMEF schedules were screened for their ability to their alter the viability and/or proliferation rate of human mammary carcinoma cell populations (HTB-126 & MCF-7) in multi well tissue culture plates. Of these twelve techniques, two were found to compromise the viability and/or proliferation rate of HTB-126/MCF-7 cell types relative to untreated negative reference controls. Over the course of subsequent replicate studies (n= 7) a total of three out of twenty-three (n=3/23) multi-frequency PTEMEF schedules were observed to consistently inhibit the viability and/or proliferation rate of HTB-126/MCF-7 populations between 31% to 35% compared to untreated negative reference controls.

Subsequent investigations identified membrane associated complexes that are expressed at elevated or decreased levels in MCF-7 populations following exposure to multi-frequency PTEMEF schedules. Subsequent investigations have detected several mRNA sequences (n=3) that are expressed at higher levels (n=1) or uniquely expressed (n=2) in populations of MCF-7 human mammary carcinoma following exposure to multifrequency PTEMEF schedules. It is our intention to publish these experimental findings in the future. However, due to the very nature of PTEMEF technology and instrumentation, there are numerous combinations of the variables of EM intensity, EM frequency, exposure duration, and exposure number that need to be evaluated in order to fully delineate the potential anti-neoplastic properties provided by this modality. Subsequent investigations attempted to determine if vitality staining, protein fraction, and/or mRNA transcription preparations performed immediately after exposure of MCF-7 human mammary carcinoma cell populations to PTEMEF schedules influenced experimental findings. The source of these biological samples were MCF-7 human mammary carcinoma cell populations processed approximately four hours after the fifth and final PTEMEF exposure period. In addition, an attempt was made to determine the relative biological influence of individual single-frequency PTEMEF techniques contained within multi-frequency PTEMEF schedules.

Collective interpretation of experimental findings reveals an ability of a multifrequency PTEMEF schedule to induce alterations in viability/proliferation rate and expression profiles of (i) cytosol-soluble and membrane associated protein fractions; and (ii) genetic transcription of mRNA sequences compared to negative (non-exposed) reference controls. In this context, these alterations appeared to be of a different pattern when experimental samples were immediately processed following MCF-7 exposure to the fifth and final PTEMEF schedule. In contrast, slightly different and slightly more subtle differences were appreciated when an intentional delay of several hours was implemented between the final PTEMEF schedule exposure and sample preparation. Appreciation of this observation implies that maximum alteration in protein expression and mRNA transcription may occur during or shortly after periods of PTEMEF exposure. In addition, there was also a relative difference in the biological affect exerted by individual single-frequency PTEMEF techniques contained within the "master" multi-frequency PTEMEF schedule. Ultimately, these laboratory findings will serve as an experimental foundation for future research investigations devoted to delineating (i) time-frames that PTEMEF exert a biological affect; (ii) duration PTEMEF induced molecular/ genetics alterations; (iii) identity of PTEMEF that selectively exert specific biological affects in living systems; and (iv) identity of molecular/genetic "targets" that PTEMEF interact with in a manner that creates a biological affect. (26)

9. Alleviation of Chronic Pain (1999): Case-Controlled and Double-Blind Clinical Studies

Hospital Costa Del Sol & Hospital Serrania de Rhonda, under the supervision of Hospital Clinico de Malaga, Spain (Dr. Pedro Alonso Atienza)Institute of Theoretical Physics and Advanced Studies for Biophysical Research, Jupiter, Florida, USA (Prof. Jerry I. Jacobson)Status:

More than 300 patients were treated with Jacobson Resonance therapy (1999) and more than 90% of same experienced an alleviation of chronic pain. During the fourth quarter of 1999, 86 patients, with arthrosis of the knee were treated in a double-blind, randomized, placebo-controlled study. A total of 84 patients (97.7%) experienced a statistically-significant immediate reduction of pain. Furthermore, a week later, approximately the same level of pain reduction had been maintained in 52 patients (60.5%).

Jacobson Resonators have been successfully guided through CETECOM homologation tests (technical standards certified by the European Union), have complied closely with Direccion de Productos Sanitarios y Farmaceuticos (The Spanish Health Ministry), and have satisfied the European Union's Medical Device Directive. Jacobson Resonators were judged to be non-hazardous.

CE-Mark approval was granted on January 10, 2001.



10. Fibromyalgia: Double-Blind Placebo-Controlled Randomized Clinical Study

Fibromyalgia is a syndrome involving chronic diffuse widespread aching and stiffness of muscles and soft tissues as well as lack of stage IV sleep. Diagnosis conventionally requires 11 of 18 specific tender pints including the occiput, neck, shoulders, chest, elbows, gluteus, greater trochanter, and knees.

4/kg touch pressure elicits a painful response. Tender points are found on both sides of the body, and above and below the waist. Fibromyalgia has become an ever-increasing, often-debilitating condition throughout the world. Its etiology is unknown, although it has been linked to physical and emotional trauma, as well as viral infection.

A double-blind, placebo-controlled and randomized clinical study was performed with three groups: A (n=5), B (n=4) and C (N=4). Groups A and B were treated with different pTEMF protocols to establish optimization of signal parameters, while group C served as the Placebo. All subjects sat in the Magnesphere under the same ambient conditions, except that Group C did not experience exposure to pTEMF's. Scales utilized to determine outcome included NPI (pain scale/subject rated), FIG (quality of life scale), MOS (pain sleep scale), and ACR (pain scale/clinician rated).

Group A Treated

- The (NPI) baseline (for the last 3 Tx) average was 7.86 and it went to 4.168, a 46.97% decrease in pain/stiffness as determined by the patient.
- The (normalized) FIQ average baseline was 71.35, and at endpoint it was 37.48, a 47.47% decrease, showing a significant improvement in quality of life.
- The average baseline for MOS was 64.154, and at the end (day 14) the MOS was 32.034, a 50.07% improvement in sleep.
- The average for ACR/pain at baseline was 14, and at the end (day 14) it was 6.6, a 52.86% improvement.

Group B Treated

- The NPI baseline average was 7.4975, and the average of the last 3TX at endpoint was 4.5, a 39.98% improvement.
- The FIQ subtotal (normalized) baseline was 57.9885, and the endpoint (normalized) was 37.79435, a 34.82% improvement in quality of life. The MOS baseline subtotal average was 52.075, and at the endpoint (day 14) it was 34.195, a 34.34% improvement in sleep.
- The subtotal average ACR/Pain at baseline was 16.25, and at the endpoint (day14) it was 8.5, a 47.69% improvement.

Group C Untreated Control

- The NPI baseline was 8.88, and the endpoint was 7.00, a 21.21% improvement.
- The FIQ average baseline was 76.84, and at endpoint it was 56.38, a 26.62% improvement.
- The MOS average baseline was 68.74, and at endpoint it was 53.23, a 22.57% improvement.
- The ACR/Pain average at baseline was 16.25, and at endpoint it was 11.00, a 32.31% improvement.
 (Each TX exposure was 60 min 10 TX were performed)

% Responders in Groups A, B and C

30% represents clinical significance.

<u>(NPI) % Of individual responders for each group</u>
 Group A- 80%
 Group B- 50%
 Control Group C- 25%

Groups A and B combined: 66.67% Group C (Control) – 25%

· (FIQ) % individual responders for each group

Group A- 60.00% Group B- 75.00% Control group C- 50.00%

• (MOS) % of individual responders for each group

Group A- 80% Group B- 50% Control Group C- 25%

> These data indicate that pT EMF's may diminish pain/stiffness and that it may improve quality of sleep and life for fibromyalgia sufferers. These effects are longlasting in most cases. Larger placebo-controlled studies need to be undertaken to confirm and further investigate the benefits of this unique, noninvasive and promising therapy.

Prof. Dr. Jerry Jacobson, theoretical physicist, biophysicist and medical researcher, is a world-renowned pioneer in the field of bio-electromagnetics. Inventor of 40+ patents and author of more than 100 scientific publications, he has lectured on the theory and practice of Jacobson Resonance throughout the world for more than 30 years. He is listed in Who's Who in the World, Who's Who is America, Who's Who in Science and Engineering, and Who's Who of American Inventors.

He currently serves as Chief Science Officer for Pico-Tesla Magnetic Therapies, LLC and Magneceutical Health, LLC. He is recipient of the Albert Einstein Genius Dedication by the American Biographical Institute and a member of the International Order of Merit, a distinction awarded by the International Biographical Centre, Cambridge, England.

For more information please see: www.magneceutical.com

Also, his latest book, "Reason for Life," a compendium of his art, science, poetry and philosophy, is available through <u>www.AbbottPress.com</u> as well as other booksellers.



Dr. Jacobson is seen here lecturing to the Oncologic Society of Sweden, at the City Conference Center in Stockholm, Sweden.

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