CLINICAL OBSERVATIONS ON MAGNETIC MOLECULAR ENERGIZER IN PARKINSON'S DISEASE ~ A PILOT STUDY

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Context: Magnetic therapy has been used in medicine ranging from static to pulsed electromagnetic fields for wound healing, tissue regeneration, immune system stimulation, neuroendocrine modulation, treatment of osteoporosis, bone repair, electro-acupuncture, and nerve stimulation. High gauss (3000-5000) DC electromagnetic field therapy from the Magnetic Molecular Energizer (MME), a novel experimental device, has been used in over 600 patients in a pilot study under an independent IRB for neurodegenerative and osteo-degenerative diseases.

Objective: 1. To reduce the oxidative stress and remove heavy metals which have been implicated in Parkinson's disease. 2. To increase tissue regeneration by decrease in hyperhydration and apoptosis of dopanergic cells, and the up regulation of stem cells.

Design: A multicenter pilot open clinical trial of MME in patients with Parkinson's disease.

Participants: 14 patients with Parkinson's disease who presented to AMRI in PA, NC, CA and Canada.

Intervention: 3000-5000 gauss DC electromagnets were placed above and below the patient.

Main Outcome Measures: Global Physician Assessment consisted of hand writing, gait, posture, walking and temor. A Combined Symptoms Assessment (CSA) scoring system was used which combined the Global Physician Assessment with the Patients Self Assessment.

Results: Every patient noted improvement in his or her symptoms after the trial period, with an average increase of 3.1 CSA points above baseline, or moderate cessation of their initial symptoms. Treatment hours correlated strongly with the patient's final condition. Increases in MME therapy time lead to continuing improvement in symptoms and do not appear to lead to any dose related side effect or serious side effects at all. On average, patients first noted a significant improvement after thirty-one and a half hours of treatment, with an average CSA of 1.3 at that point.

Conclusions: MME appears to improve symptoms of Parkinson's disease and potentially influence biological events ranging from heavy metal removal, tissue regeneration by decrease in hyperhydration and apoptosis of dopanergic cells, and the up-regulation of stem cells and decreased oxidative stress. Sham controlled, masked multicenter trials of MME therapy in PD with several major medical universities are to commence in mid-2003. Further studies are underway to elucidate the mechanisms of patients' symptoms improvement.

Introduction:

A NIH Consensuses Conference on "Alternative Medicine, Expanding Medical Horizons" held in Chantilly Virginia in 1992, and published in December 1994 on "Bioelectromagnetic Therapy," suggested eight major new applications of the use of electromagnetic fields in medicine include:

Wound healing, Tissue regeneration, Immune system stimulation, Neuroendrocrine modulations, Treatment of osteoarthritis, Bone repair, Electro-acupuncture, and Nerve stimulation.

The MME (Magnetic Molecular Energizer) is a treatment method that is hypothesized to addresses all the areas above except acupuncture. It was developed by Dr. Dean Bonlie at the research laboratory at Magnetico, Inc. Calgary Alberta starting in 1996. The MME device resembles MRI equipment, which is used only for imaging. However, the MME is a novel treatment modality consisting of two very large and strong nonpulsed DC electromagnets (3000 to 5000 gauss) with the patient lying in the focal point between a negative pole below and the positive pole above (US patent #6,210,317)seen in Figure 1.

Figure1. Patient photographed while being treated with MME. It has been used on over 600 patients in North America under an independent Institutional Review Board as an experimental device for neurodegenerative and osteo-degenerative diseases.*

Hypothesis:

Parkinson's disease (PD) is a neurodegenerative disease resulting from degeneration of a subset of dopaminergic neurons in the brain stem located in the substantia nigra pars compacta. These cells also contain large amounts of melanin, and hence are heavily pigmented leading to the speculation that melanin may also contribute to their degeneration. In addition, PD preferentially affects the lateral parts of the substantia nigra (SN) suggesting that PD differs in a substantial way from normal aging, which affect the dorsal tier of the SN. In addition to cell loss, PD is associated with characteristic intracytoplasmic inclusions known as Lewy bodies, containing intermediate filaments and ubiquitin.[1] A substantial body of evidence implicates dopamine as an endogenous toxin in PD. Dopamine shows toxicity when applied to cultured neural cells and is more neurotoxic than MPP^+. [2] Dopamine toxicity is not receptor mediated, and dopamine oxidizes to produce hydrogen peroxide in contrast to glutamate toxicity, which occurs through the activation of specific glutamate receptors. Hydrogen peroxide acts as a free radical to damage cellular constituents including lipids and protein only protected by catalase. The more highly reactive superoxide and hydroxy radicals modify lipids and protein, damaging the cell. There is considerable evidence now indicating that PD involves oxidative stress caused by free radicals.[3,4,5] Oxidative stress may derive from several sources. Exogenous toxins can increase free radicals. Oxidative stress may also derive from endogenous toxins as a result of mitochondrial dysfunction.

Oxidative stress has also been found to cause apoptosis (cell death) of neurons. PD is associated with genetic and environmental factors such as exposure to pesticides and heavy metals with subsequent depletion of the mitochondrial protective enzymes, superoxide dismutase and glutathione, as well as the neurohormones serotonin and melatonin.[6]

The facts that water is the dominant component of cell, and that it serves as the main medium where the major part of biochemical processes takes place, allow us to consider it as a common "second" messenger through which the effects of external and internal signals in cell metabolism are realized. Although the great physiological importance of intracellular water is widely accepted, investigators have not paid an adequate attention to its role as a messenger for generating various diseases.

It is known that cell pathology is accompanied by its swelling (hydration). Cell hyperhydration precedes cell death independent of any other reason causing apoptosis or necrosis as demonstrated by Bennett and Huxlin in 1996.[7] Ayrapetyan and his associate Danielyan demonstrated in 1999, that after 0.2 Tesla (T) or 2000 gauss Static Magnetic Field (SMF), there was a time dependent decrease of hydration and adaptation, followed by disadaptation, detected in brain and liver tissues in most rats after 3.5-5 hours of exposure.[8]

The Magnetic Molecular Energizer is hypothesized to treat Parkinson's diseases by increasing the velocity of valence electrons of atoms in response to the effects of the DC electromagnets. Antioxidant protective systems in the mitochondria of PD brains, such as superoxide dismutase (SOD) and reduced glutathione (GSH) are altered and are believed to be evidence of oxidative damage to lipids, protein and DNA. Magnetic fields have been shown to up-regulate the production of enzymes, and we suspect that this may also be occurring during therapy with MME in patients with PD to prevent further damage from oxidative stress. Furthermore, MME 0.3-0.5 T, is believed to decrease the hyperhydration of cells and prevent apoptosis in patients with PD.

* The Independent Institutional Review Board for Magnetic Molecular Energizer consists of all Advanced Magnetic Research Institute investigators, a medical university professor and registered nurse, a clinical psychologist, and two board certified medical physicians (neurology and family practice) who are experts in magnetic therapy and are not affiliated with AMRI. All minutes from meetings are on file with the FDA

Pacini and associates in 1999 have studied the effect of 0.2 T static magnetic fields on human neurons in vitro and found that after 15 minutes exposure, cells showed dramatic changes of morphology; they formed vortices of cells and exposed branched neurites featured synaptic buttons.[9] We also hypothesize that prolonged improvement seen in these patients and other neurodegenerative diseases such as Alzheimer's disease and multiple sclerosis implicates up-regulation of neural stem cells, vortices and synaptic button formation.

Since patients with Parkinson's in our pilot study of MME appear to detoxify, with symptoms of nausea, agitation, and the release of metallic odor during the first day, we suspect that heavy metals are released from brain tissues and eliminated. Both Alzheimer's and Parkinson's have genetic as well as environmental factors in response to heavy metals in particular. Iron, zinc, calcium, aluminum, and mercury have been implicated in AD[10,11,12] and iron and mercury in PD.[13,14] Heavy metals increase oxidative stress via the Fenton reaction and cadmium, lead and mercury have demonstrated uncoupling of mitochondrial oxidative phosphorylation.[15] MME is hypothesized to liberate heavy metals from the brain, up regulate cytoprotective enzymes and increase tissue regeneration. Tissue regeneration may be due to endogenous stem cell up- regulation and is currently being investigated at AMRI PA with collaboration at Whitaker Biomedical Engineering Institute at Johns Hopkins University.

Background: Extremely Weak Pulsed Magnetic Fields

Sandyk reported a number of patients with Parkinson's disease (PD) who experienced significant improvement in symptoms following treatment with extra-cranial pulsed picotesla-range magnetic fields.[16] He also reported improvement in patients with multiple sclerosis with his device.[17] Sandyk reported that two AD patients experienced significant improvement in visual memory and drawing performance following external application of electromagnetic fields from 5 to 8 Hz. Improvement was also seen in other cognitive functions including spatial orientation, mood, short-term memory, and social interactions.[18] The disorganization of circadian rhythm, causally related to memory deterioration in old age, and possibly to Alzheimer's disease, when treated with magnetic fields could lead to memory improvement among the elderly by means of resynchronization, or resetting the circadian rhythms.[19] Bardasano, using a plastic helmet device housing a set of coils generating fields of 8Hz and 7.5 picotesla, produced beneficial effects in patients suffering with PD.[20]

Valentova used pulsed magnetic therapy at 25 Hz for 20 minutes over 10-12 exposures along with comprehensive spa therapy in patients with Parkinsonism and spasticity. At the end of treatment, the patient's motor tests improved significantly for walking ($p<0.001$) and for change of position ($p<0.01$). The subjective tension in muscles of the lower extremities was reduced in 84.6% of cases, back complaints reduced in 84.6% and general improvement was reported in 96%.[21]

Jacobson using low amplitude, extremely low frequency magnetic fields, reported his treatment of osteoarthritic knees in Alternative Therapies in Health and Medicine in 2001 and has received IDE market approval in Canada.[22]

Initial Assessment:

Patients with PD were first assessed in the office of the investigators after prior neurological evaluation and confirmed diagnosis by standard criteria as well as response to dopaminergic medication and or anticholinergics medication. After reading and signing an informed consent, all patients were positioned on the bed with their heads under the focal point between the two magnets for 3-5 hours initially, then for periods up to 20 hours per day. PD patients were treated for as many consecutive days as possible. The total number of hours varied for each patient. Patients were continued on the same dose of dopaminergics or anticholinergics. Patients were evaluated after each session with the treating MME physician and progress noted on a Global Physician Assessment. Hand writing before and after the completion of therapy was noted as well as arm swing, gait and tremor. One investigator (LP) video taped by digital camera all PD patients and upon completion of therapy.

Inclusion Criteria for Patient Enrollment:

Diagnosis of Parkinson's disease in patients required:

(1) The presence of at least two of the following signs; resting tremor, cogwheel rigidity, bradykinesia, and postural reflex impairment, at least one of which must be either resting tremor or bradykinesia.

(2) The Parkinsonism was not due to trauma, brain tumor, infection, cerebrovascular disease, and/or other known neurological disease or to known drugs, or chemicals.

(3) Patients had no or mild dyskinesia. Hahn-Yahr ratings in the off state did not exceeded 3.0. Patients had no greater than 3 point rating in any extremity on the UDPRS of bradykinesia, rigidity or temor in the off state. Patients will have no greater than a 3 rating for postural stability in the off state.

(4) Parkinson's patients were responsive to dopaminergic drugs and showed a 33% or greater improvement in their UPDR score over that measured in their worst OFF.

(5) Age of patients with PD ranged from 35 to 85.

All patients were able to read, understand and sign Informed Consent written in English.

Exclusion Criteria for Patient Enrollment:

(1) Patients with advanced PD were excluded from this study because of their inability to recline for long periods of time in a fairly close space and because of clinically significant behavioral symptoms (depression, agitation).
(2) Patients who meet criteria or carry the diagnosis of an atypical Parkinson syndrome including: progressive supranuclear palsy, multi-system atrophy (includes the Shy-Drager Syndrome, striatal nigral degeneration, olivo-ponto cerebellar atrophy), corticobasal ganglionic degeneration, and diffuse Lewy body disease were excluded.
(3) Patients with pacemakers, defibrillators, cochlear implants, aneurysm clips and metallic fragments in their eyes were excluded.
(4) Patients with any experimental PD surgical therapy such as fetal or porcine stem cells and patients who have had surgery for PD such as stereotaxic surgery of the globus pallidus, ventrolateral thalamus or implanted brain stimulators were excluded.

Clinical Notes:

Forty-one patients with Parkinson's (PD) disease have been treated with 32 definitely improving in symptoms (91-308 hrs); however, we have only collected sufficient data for the last fourteen patients to enable accurate tracking of their progress. A synopsis of these latter PD patients treated with MME follows below:

L.S.
Patient is a 64 year old white female with a diagnosis of Parkinson's disease for 11 years with slowly progressive tremor, rigidity, and bradykinesia bilaterally, but mainly on the left side. She has difficulty getting up and down out of chair, slowness of gait with some freeze-ups and frequent falls. She has hyperkinetic movements (jerking) with Sinemet dose. After 36 hours of MME therapy, she reported improvement in the jerking movements and freeze-ups and more energy. By 43 hours of MME therapy, she had improvement in tremor and was not falling as much and at 83 hours was significantly improved regarding tremor, hyperkinesis, and falling. She received a total of 125 ½ hours of MME treatment. Her tremor remained significantly improved for at least a year of follow-up.

P.K.
Patient is a 72-year-old white male with a diagnosis of Parkinson's disease for 13
years. Prior to MM|E treatments he was slowly progressing in all symptoms. His tremor was worsening on the left especially when the Sinemet "runs out." He has more difficulty with stability with turns and gait. Beginning at 13 hours of MME, he felt his tremor was much better, walking, energy and balance was improved. He maintained improvement in the above symptoms through the end of the treatment period. He received a total of 124 ½ hours of MME therapy.

L.M.
Patient is a 72-year-old white female with a diagnosis of Parkinson's disease for 13 years.
This patient prior to beginning therapy was experiencing increasing difficulty with her gait and balance, moderate tremor, difficulty getting up and down out of chairs. Her gait problem was complicated by bilateral hip replacement and significant hip pain. She also had low volume speech. After 14 hour of MME therapy, she was reporting feeling more energy and
improvement in her tremor. At 27 hours of MME treatment, she reported improvement in tremor, gait and balance, anxiety level and was sleeping better. At 51 hours of MME, her hip pain improved and she felt generally stronger. She was treated for 145 hours with maintained improvement in gait, tremor, and relief of hip pain. Three years later, her gait is still improved over baseline and she has progressed very little in her overall Parkinsonism status. Her hip pain was virtually eliminated and is just beginning to return to some degree.

D.G.
Patient is a 72-year-old white male with a diagnosis of Parkinson's disease for 17
years. His main problems have included mild to moderate resting tremor, slowing down of movement, postural instability, difficulty swallowing, low volume speech and depression. His depression has responded nicely to antidepressant medication, which he has been on for many years. Prior to MME therapy, his disease was slowly progressing with a major problem with swallowing which was felt to require gastric tube placement if it continued. At the end of 7 ½ hours of MME his stiffness had improved, tremor was better, mucous in throat was much less and his voice was more understandable. He maintained improvement over a total of 110 hours of MME treatment. The patient's swallowing and sensation of mucous in his throat improved significantly and was maintained so that he did not need a gastric tube.

K.C.
Patient is a 55-year-old white male with a diagnosis of Parkinson's disease for 11 years.
Just prior to MME treatments he felt his disease was progressing and elected to retire from his job as a high school coach. He had moderate tremor, increasing rigidity and freeze-ups when walking and turning, slowing down of movement, mild stuttering speech. He has a short steppage in his gait and occasional falling, especially when he is carrying something at the same time. He noted that his tremor stopped after about 3 hours under the MME therapy, but returned after completing the session. No change noted until the 44^{th} hour when he noted continuing improvement in his tremor, and at the 49^{th} hour, his gait improved. At the 50^{th} hour of MME, his treatment schedule was changed to 4 hours per week, which he has done over 3 years. He is stable, feels that he has not progressed, his medications are working better, and he has retained improvement from baseline in his tremor, postural stability and gait. He has had a total of 570 treatment hours of MME therapy.

S.D.
Patient is a 62-year-old white female with a diagnosis of Parkinson's disease for 11 years. She has a long history of insomnia, depression, and loss of smell in addition to resting tremor, slowing of movement and instability of gait, mild/moderate rigidity. She is fatigued much of the time. For the first 100 hours of MME therapy, there was very little change except she felt she was falling more. At 121 hours of MME, she noted some improvement in rigidity and her vision seemed clearer. Her balance and postural stability were somewhat worse. At 128 hours of MME treatment, she reported sleeping much better. At 133 hours of MME, she reported marked improvement in tremor and energy level but still had trouble with balance and gait. Her energy level remained better until end of treatment. Some weeks later she called back and said she was overall much improved, her energy level remained improved, and her sense of smell had returned. The total number of treatment hours using MME was 139 ½ hours.

A.S.
Patient is a 73-year-old white female with a diagnosis of Parkinson's disease for 8 yrs. When first seen she was mainly confined to chair, walking only with support. Severe bradykinesia, poor speech, moderate to severe tremor, moderate rigidity was observed. The patient did not readily communicate. After 10 ½ hours of MME therapy, she was able to push with her feet and help with body weight as moved from chair to bed. At 48 hours of MME treatment, she was stronger, was able to move from wheelchair to bed, sleeping better. At 81 hours her vision had improved, speech was more effective. At 91 hours of MME, she could walk unassisted with a walker, stand up on her own, and pull herself up from bed and chair. The total number of treatment hours of MME was 91 ½ hours.

C.J.
Patient is a 71year old white male with a diagnosis of Parkinson's disease for 14 years. He has had an increasing problem with his gait over the past 6 years to the point that he is ambulatory only with assistance. He has moderate resting tremor, rigidity and akinesia, poor memory and stooped posture. The patient turns en bloc with postural instability. At 38 hours of MME therapy, patient said he didn't feel "as heavy." At 53 hours of MME treatment, he noted improvement in balance, which continued to improve. Speech was improved in volume. Rigidity and tightness in muscles improved. The number of total MME treatment hours was 125 hours.

F.B.
Patient is a 68-year-old white female with a diagnosis of Parkinson's disease for 1-½ years. Tremor began in the left hand, slowing of gait and difficulty getting up and down out of chair. She tends to veer to the side when walking. She exhibited decreased word finding. At onset of treatment with MME, her condition was progressively worsening. After 13 hours of MME therapy, she reported improvement in tremor, left side rigidity and drawing, and numbness. By 47 hours of MME, she had better balance, gait, coordination and tremor. At the end of 100 hours she had sustained improvement in tremor, gait, rigidity and memory. She went home at the end of the study period and maintained good improvement generally but after 5 months was worsening but not back to the prior level. She came back for another 110 hours of MME and again improved in balance, coordination and tremor. She maintained improvement. Some 10 months later she returned and received another treatment, again making more progress. The patient has received a total of 327.5 hours of MME treatment and remains improved in regard to gait, getting up and down out of chair, rigidity, tremor and memory.

C.A.
Patient is a 71-year-old white male with a diagnosis of Parkinson's disease for 2 years. Additionally he has diabetes mellitus treated with insulin and hypoglycemic agents. Problems prior to treatment include difficulty with handwriting, stooped posture with slowing of gait, low volume speech, resting tremor, swallowing difficulty, choking at times, trouble with buttons, slowed thinking, and slowing down in most activities. At 52 hours of MME therapy, patient felt he was walking better, with improved range of movement right arm, picking his feet up better, and not dragging left leg now. At 62 hours of MME treatment, his tremor was "a lot less."

J.S.
Patient is a 57-year-old Asian female with a 10-year history of Parkinson's disease. Her main problems at initiation of treatment were stiffness and slowing down of movement, tremor inside but not outside, difficulty with postural instability and gait, low volume speech,
swallowing difficulty and dyskinesia with Sinemet. At 65 hours of MME therapy, patient noted less tightness in muscles, improved balance and gait. At 131 hours of MME treatment, stiffness, balance, coordination of movements and walking were improved. The patient had difficult time with treatment because she did not tolerate lying on the tables well, and felt stressed.

L.S.
The patient is a 61-year-old white female with Parkinson's disease for 4 years. Her main problems at initiation of treatment were slowing down of movement, mild tremor of the left hand, and slowed shuffling gait. There was also difficulty getting up and down out of chair, low volume speech, poor memory, and handwriting difficulty. At 30 hours of MME therapy, she began noting improvement in walking, energy level, tremor, and handwriting. She stated that she was able to go longer without medication at 54 hours. At 82 hours of MME treatment, improvement noted were continuing and she felt that her hands work better. At 95 hours of MME, she was much better in gait, tremor, and handwriting, able to get up and down more easily, and had more energy and felt better. The patient had improved even further at 101 hours. The total treatment hours of MME were 101.

V.K.
The patient is a 69-year-old white female with a diagnosis of Parkinson's disease but without tremor and a history of chemical sensitivities. She developed initially weakness of her right hand in 1988, which progressed to difficulty initiating movement and agraphia. After 29 hours of MME therapy, the patient was observed to be walking better with improved arm swing. At 59 hours, her balance, gait and arm swing had improved. Patient, however, was not satisfied with the results, and counting time after the study period, spent a total of 203 hours of MME treatment without much more improvement.

R.J.
The patient is a 68-year-old male with a diagnosis of Parkinson's disease for 2 years. Prior to treatment he was unable to get out of chairs and took baby steps. The patient had difficult with stiffness, took small steps, and had a light tremor. Following treatment it was possible to reduce his Sinemet treatments by one third, and the patient was able to go without this medication entirely for a day and a half, with stiffness following withdrawal and ceasing with a return to the (lowered) dosage.

Methods:

Patients were evaluated using the Combined Symptoms Assessment (CSA) scoring system. Every Parkinson's related symptom and its severity were recorded on this scale as the particular patient's baseline index. Patients were asked to note at what time(s) they experienced significant changes in these symptoms and to rank their new severity level. These descriptions were translated into –5 to +5 point scale for that symptom and the various symptom scores averaged to produce the CSA rating for that hour level of treatment. Broadly, with "0" set as the baseline, scores 1-5 were considered to be mild, marked, moderate, major, or complete in terms of either improving (+) or worsening (-) condition. The final rankings were assigned and by the physician and an independent observer. In terms of agreement, their ratings varied only three times by one point, with only a difference of one in total points assigned.

Heavy metals appeared to be liberated from patients during the first several days of MME and the protocol now requires the administration of DMSA (Chemet 500 mg) before each treatment session to lessen side affects. Both Alzheimer's and Parkinson's have genetic as well as environmental factors in response to heavy metals in particular. Iron, calcium, aluminum, and mercury have been implicated in AD and iron and mercury in PD. Heavy metals increase oxidative stress via the Fenton reaction and cadmium, lead and mercury have demonstrated uncoupling of mitochondrial oxidative phosphorylation. MME is hypothesized to liberate heavy metals from the brain, up regulate cytoprotective enzymes and increase tissue regeneration.

Results:

Every patient noted improvement in his or her symptoms after the trial period, with an average increase of 3.1 CSA points above baseline, or moderate cessation of their initial symptoms. Treatment hours correlated strongly with the patient's final condition. Increases in MME therapy time lead to continuing improvement in symptoms and do not appear to lead to any dose related side effect or serious side effects at all. On average, patients first noted a significant improvement after thirty-one and a half hours of treatment, with an average CSA of 1.3 at that point.

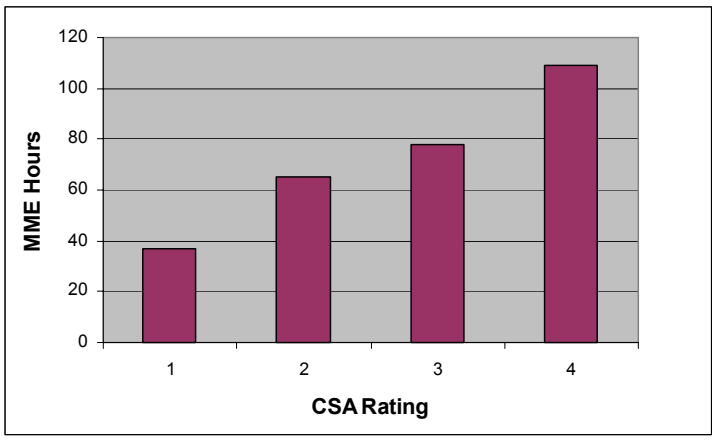

Figure 2. Correlation between improved CSA scores and increasing treatment times.

Given below in Fig 3, are the CSA ratings of the patients with quadratic trend line for the CSA score at the conclusion of their treatment and at the points when they noted significant improvement.

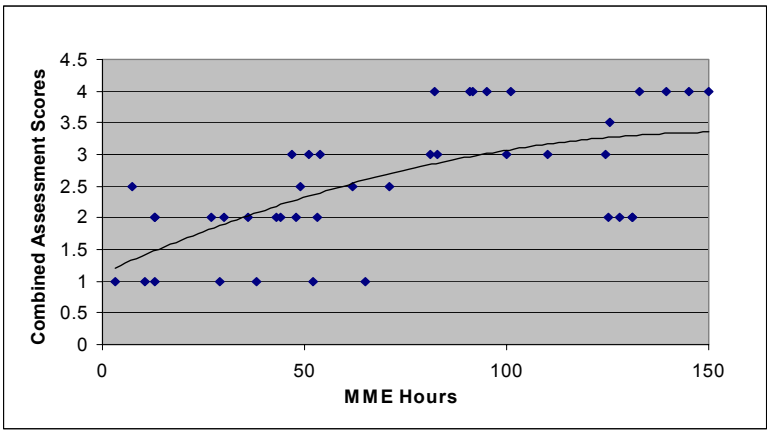

Figure 3. Generalized recovery trajectory.

Changes in CSA rating did not correlate strongly with age or sex. In three instances, where patients expressed unhappiness or stress concerning the treatment, their scores were lower. There is, however, a general correlation between the length of time a patient has been diagnosed with Parkinson's, and the efficiency in terms of the time it took them to achieve their final CSA standing. Figure 4 shows these two groups which diverge at a juncture of roughly ten years after diagnosis. The one spike value is likely explainable in that it comes from the youngest patient by fourteen years, although there was no further general association with age. It does hint that there may indeed be one in a larger study, since the other patients in this study are tightly clustered in a higher range.

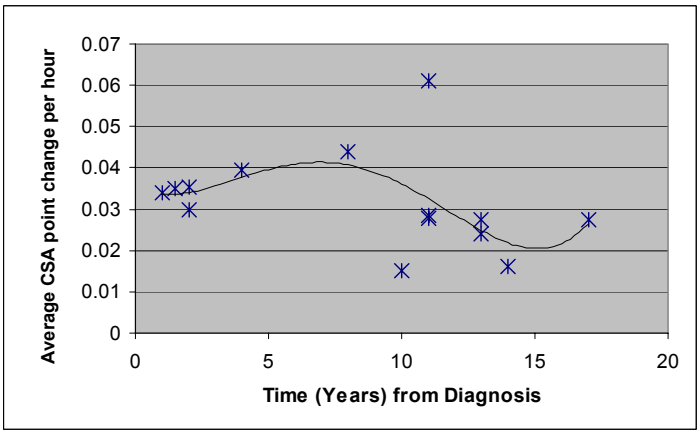

Figure 4. MME efficiency and disease term

Discussion:

Other researches have shown regeneration with electromagnetic therapy starting with Robert O. Becker, MD who demonstrated differentiation from frog red cells with microcurrent. Along with Andy Bassett MD, they pioneered accelerated bone repair with electrical current, which then lead to pulsed electromagnetic field therapy (PEMF) for non-union of fractures.[23] Walleczek in "Magnetokinetic effects of Radical Pairs" has pointed out that there is a resonance temporal type (intensity window) for enzymatic reactions. He reviews previous work by others, demonstrating that the interaction mechanism with magnetic fields with chemical reactions in living systems is explained on "biochemical reactions involving species having unpaired electrons (e.g., processes involving the transfer of electrons along the chain of cytochromes and associated reactions of oxidative phosphorylation), many enzymatic reactions, the oxidation of non-heme iron by oxygen and certain stages of photosynthesis". He lists a 35% increase in redox activity in cytochrome *c*-oxidase with a static magnetic field of 0.3 millitesla and a 40% increase with a 50-Hz sinusoidal magnetic field.[24]

When treating conditions involving the brain, the MME produces approximately 5000 gauss at the surface with a minimum of 3000 in the brain. The patient is placed in the same position as he or she normally sleeps because the existing tissue atoms are orientated to the earth's magnetic field. As clinical evidence that supports the efficacy of MME in the treatment of human disease accumulates, an understanding of the underlying molecular mechanisms will be required to facilitate long-term advances in the field. The body, while mainly composed of materials that are essentially non-magnetic, does contain a wealth of charged particles that have the potential to interact with an applied field. These particles include molecules, individual atoms, and electrons and protons, the subatomic components that comprise each atom. When a patient is placed in the MME device, there is a temporary increase in the magnetic force at the focal point similar in magnitude (~0.5T) to that employed in EPR, a technique widely used to analyze the properties of molecules containing unpaired electrons (free radicals).[25,26,27] Consequently, the effects of applied magnetic fields on free radicals are well known and are beginning to offer biophysical explanation for the therapeutic effects of MME. Briefly, an electron can be considered to spin on its axis, but it also normally orbits the nucleus. An applied

magnetic field changes the angular momentum of orbit and causes precession (wobble) of the orbit. These two parameters, angular momentum and precession, combine to give each electron a particular "spin state." For two unpaired electrons to form a new chemical bond, they must have complementary spin states wherein the spin of one electron exactly cancels the spin of the other electron. Given that applied magnetic fields are able to influence the rate of interconversion between spin states, it follows that those chemical reactions that proceed via free radical mechanism will be affected by MME.

Although the subatomic effects, mentioned above, forms a solid theoretical basis to explain how MME can influence chemical reactivity, the exact effects of these forces on specific molecules within a cell are largely unknown. First, the identities of the molecules and chemical reactions that are affected have not been catalogued in detail. In theory, any reaction that utilizes a free radical mechanism is a candidate; there are already more than 20 such enzymatic reactions known. A few of these reactions are those catalyzed by cytochrome-C oxidase, ethanolamine ammonia lyase, the photosynthetic reaction center, and ornithine decarboxylase.[28,29,30,31]

A confounding factor in deciphering the molecular effects of MME is that applied magnetic fields are able to either increase or decrease the rate of a chemical reaction; they also have the potential to change the potential to change the composition of the products of a reaction[32]

Another possibility is that the effects of MME are not realized through effects on specific enzymatic reactions, but rather through alterations in cellular pools of free radicals. Free radicals such as hydrogen peroxide, superoxide, and hydroxyls are produced during normal cellular metabolism and have been implicated as causative factors in DNA damage, degenerative disease and aging.[33,34,35] It also possible that MME can ameliorate the deleterious effects of these molecules by decreasing the chemical reactions that causes damage to DNA, proteins and lipids. Alternatively, applied magnetic fields may increase the desired fates of these molecules; specifically, by increasing their rates of degradation by reaction with protective enzymes such as catalases and superoxide dismutase.[33] Furthermore, nitric oxide, a molecule that plays a role in many cell signaling events, is also a free radical and therefore is a candidate for MME intervention. In summary, although much work at a molecular level remains, the picture is now coming into focus of how Magnetic Molecular Energizer can potentially influence biological events ranging from heavy metal removal, tissue regeneration by decrease in hyperhydration and apoptosis of dopaminergic cells and the up regulation of stem cells and decreased oxidative stress. Some of these hypotheses will be tested in a double blind sham cross over controlled trial of MME in patients with early to mid Parkinson's diseases in a multicenter trial with major medical centers to commence in mid 2003. These results will be submitted to the FDA for IDE Market Approval.

Limitations of this Study:

Since this is pilot research of MME, the statistical analysis of the results was not possible. Disease rating scales such as the United Parkinson's Rating Scale (UPDRS) were not used but will be used prospectively in our next trial. Controlled masked multicenter clinical trials of sham compared to MME complete with stratification of PD, UPDRS and Schwab and England Activities of Daily Living scales, biochemical measures of oxidative stress and apoptosis with statistical analysis will be performed on Parkinson's patients before MME and at periodic interval following treatment, starting in mid 2003.

Furthermore, the exact number of hours needed to treat each patient's condition was not known initially and since all patients are genetically and phenotypically different, the number of hours needed, will always vary. Therefore at the early stages of research, success rates were not as favorable as later ones. Since MME is treatment-time related, some patients became discouraged and left before their recommended minimum numbers of hours were received. Also since no grant money or venture capital was available, most patients had to pay out of pocket which caused a number of them to discontinue therapy before the window of healing occurred.

Long-term results were more difficult to obtain. Phone calls were made to as many patients as possible by this investigator at a one-year interval. Some lessons learned are that patients who respond to MME more rapidly are those with a "will to heal". They are more aware of nutrition and self-actualizing. They have not abused their bodies with alcohol, street drugs, or tobacco. Such patients were also more likely to complete the recommended minimum number of hours of MME.

Future of Medicine:

Andy Bassett, MD made this prophetic statement in a 1992 article:

In the decade to come, it is safe to predict bioelectromagnetics will assume a therapeutic importance equal to, or greater than, that of pharmacology and surgery today. With proper interdisciplinary effort, significant inroads can be made in controlling the ravages of cancer, some forms of heart disease, arthritis, hormonal disorders, and neurological scourges such as Alzheimer's disease, spinal cord injury, and multiple sclerosis.[36] This prediction is not pie in the sky. Pilot studies (recent clinical trials and experiments with MME) and biological mechanisms already in primordial terms form a rational basis for such a statement.

Perhaps in the future we will relearn what Paracaleus, a famous physician, said in the Dark Ages: **"Magnetism is the King of All Secrets."**

Acknowledgments:

Kevin Yarema PhD., Whitaker Biomedical Engineering Institute, Johns Hopkins Univ. helped in editing the manuscript concerning biophysics. Christopher Gaiteri participated with preparation of this manuscript and data collection and the Adv. Magnetic Research Institute Int. supplied the Magnetic Molecular Energizers.

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