Pilot Study – Theramag™:

A Treatment of Parkinson's Disease

Objectives

To evaluate the safety and efficacy of a novel treatment of Parkinson's Disease with THERAMAG™.

Design

A pilot study of 15 patients without placebo controls was conducted. All patients were given treatments of active therapy and three sets of observations, which included the Karnofsky life quality. The 15 Parkinson's were quantified by neurological signs and Parkinson maintenance requirements with observations taken: initially, at mid therapy and at the end of therapy.

Setting

Out-patient clinic.

Patients

15 Parkinson's Disease: Age range 38-76 Years, 5 Females and 10 Males.

The TheraMag Device

This semi-portable device produces an electromagnetic field with variable field strength of 0-2000 gauss, and is powered by 120 VAC. The unit is one configuration with a cylindrical opening in the middle front. The cylindrical opening is bathed in a homogenous electromagnetic field, so that when a hand and forearm of the subject is inserted into the opening, the coil-induced field penetrated the patient's tissues and body. This may be measured by monitoring the changes in cardiovascular system. During the course of therapy there is a universal flushness beginning with the contra lateral arm, forearm and hand and at the concluding therapy *(1 hour)* there is a transient postural hypotension lasting about 2-3 minutes. Each patient, following evaluation and consent approval had a baseline evaluation and was placed in the TheraMag device for sixty minutes. At the end of each session repeat clinical evaluations were performed. Repeat sessions generally occurred every two weeks and each lasting one hour. The FDA has deemed that it is permissible to use magnets in specific studies, since there have been no reports of ill effects resulting from the external application of magnets, and therefore safety is not an issue.

The Pilot Study

For Parkinson's: General and neurological evaluations were conducted weekly, and the conventional signs of Parkinson's disease *(rigidity, tremor, bradykinesia, frequency of on/off effect, and confusion)* were rated on a 5-point scale of 0 to 4, both before the study began, and on a weekly basis while the therapy was undertaken.

Modified Karnofsky or General Performance Scale:

100	Normal, no complaints, no evidence of disease
90	Able to carry on normal activities; symptoms of disease
80	Normal activity with effort; some signs or symptoms of disease
70	Cares for self; unable to carry on normal activity or active work
60	Requires occasional assistance but is able to care for most needs
50	Requires considerable assistance and frequent medical care
40	Disabled, requires special care and assistance
30	Severely disabled, hospitalization indicated
20	Very sick, hospitalization necessary, active support necessary
10	Moribund, fatal processes progressing rapidly
0	Dead

Results

Parkinson's: The 15 Parkinson's patients improved functionally over the active study period showing an increase in the Karnofsky *(functional scoring)* from 48.5 to 67.5, while the clinical signs of the disease *(rigidity, tremor, bradykinesia)* and drug dependence were concurrently reduced. In particular the following chart indicated the pre-post therapy results.

Parkinson scale Mean 0 (none)-4 *(maximum)*

Symptom	Before	After
Karnofsky	48.5	67.5
Tremor	2.8	2.5
Rigidity	3.0	1.5*
Bradykinesia	3.5	1.2*
Dexterity	3.4	1.5*

*significant

Discussion

While originally described in a monograph by Sir James Parkinson, it was Lewy who first observed neuronal cell loss in the nucleus basalis of Meynert in patients with Parkinson's disease. It was later suggested that neuronal loss in this area was associated with a condition of mental slowness *(bradyphrenia)* that has been related to PD. Classical Parkinson patients will

appreciate the idiotypic symptoms of muscular rigidity *(i.e. stiffness)*, tremor *(typically of hands, arms and lips)* and Bradykinesia *(slowness of motion)* as well as spontaneous On/Off *(a side effect of antiparkinson drugs)* and mental confusion. Parkinson people have a large descriptive vocabulary for their many symptoms and conditions.

During the last several years, the magnocellular perikarya of the nucleus basalis have been shown to contain the specific cholinergic marker choline acetyltransferase *(ChAT)*, provide the major cholinergic innervation to the neocortex and amygdala, and to be under the influence of the trophic nerve growth factor *(NGF)*.

A specific link between nerve growth factors *(NGF)*, Parkinson's Disease *(PD)* and Alzheimer's Disease *(AD)* was first suggested by Hefti, as disturbances in the interaction between NGF and Basal Forebrain Cholinergic Neurons *(BFCNs)* could in theory occur at many different levels, including the synthesis and release of NGF by target neurons and/or glia, the uptake and retrograde transport of NGF by BFCNs, and the role of NGF in activating specific genes in the BFCN cell nuclei. Several authors have pointed out that there are different diseases demonstrating BFCN degeneration, including PD and AD. While there is no common factor at the initial stages of the pathological cascade, but it would seem that BFCNs are particularly susceptible to a wide range of insults. The precise mechanisms involved in BFCN vulnerability are not known, although abnormal cell stimulation could well result from diverse pathological events occurring in various parts of the brain *(both cortical and subcortical)* with inputs to BFCNs.

While Parkinson's disease results from degeneration of dopaminergic neurons in the pars compacta of the substantia nigra, these dopaminergic cells may require a trophic signal from their targets, primarily the nerve cells of the striatum, for differentiation and growth. In tissue culture, the presence of striatal neurons enhances the outgrowth and transmitter-synthesizing capabilities of dopaminergic neurons from the substantia nigra. Possible trophic factors have been isolated and partially purified from striatal neurons that enhance differentiation of the dopaminergic cells. In addition, brain derived neurotrophic factor, a protein that may be produced by striatal cells, enhances the survival of dopaminergic neurons of the substantia nigra when grown in cell culture. Thus, PD may result from a lack of neurotrophic factors. Administration of these neurotrophic factors in addition to TheraMag application may slow the progression of PD.

Conclusions

No undesirable side effects were observed. This pilot work indicates that the TheraMag treatment appears to be safe and efficacious in PD for bradykinesia, dexterity, rigidity and slowness of movement. The longest number of hourly treatments has been in only 5 patients and includes 12 therapy sessions. Each treatment appears to last 2-3 weeks in most cases.

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REFERENCES

There are 34 references available for this article. If you would like a copy of the References faxed to you, please fax or email your request to Explore Publications.