

Can you split this on section  
and subsection with numbers?

# Magnetic-resonant fields for the treatment of motor neuron disorders

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## Keywords

be more general NO

Amyotrophic lateral sclerosis, neuron voltage potentials,  $K^+$  and  $Na^+$  ion conductance, magnetic-resonant potential fields.

## Abstract

Explain what is the problem be brief (Read some Abstract or example)

A means of magnetic resonant coupling of modes from an external circuit device powering a loop antenna at a characteristic frequency such that energy is transferred to electrically-charged neuronal tissues by contact with a median metallic disc on the surface of the skin. A model of transfer of electrical energy at a stable amplitude on a potential-inductance vector, determined by the presence of neuronal tissues, without surgical invasion.

## Introduction

Treatment of disorders in the somatic nervous system due to progressive neurodegeneration, while dependent upon a clear aetiology, can be addressed symptomatically [1,2,3] in search for possible treatments [4-7]. While this paper does not condone apriori reasons for treatment paradigms based on the severe debilitation such disorders, it does approach the problem from a mechanistic interpretation of the clinical evidence surrounding the epidemiology of fundamental degradation characteristics surrounding amyotrophic lateral sclerosis.

The symptoms of amyotrophic lateral sclerosis are caused by degeneration of motor nerve cells (motor neurons) in the spinal cord, brainstem, and motor cortex. Median survival from first symptoms is a little more than two years [8]. The exact cause of this degeneration is unknown but it is thought that environmental exposures and genetic factors play a role in susceptibility to the disease. In 5-10% of patients the family history is positive for ALS. However, it is not always possible to establish the mode of inheritance in each pedigree. The hallmark of this disease is the selective death of motor neurons in the brain and spinal cord, leading to paralysis of voluntary muscles [9]. The paralysis begins focally and disseminates in a pattern that suggests that degeneration is spreading among contiguous pools of motor neurons. If the mechanism which causes ALS can be disturbed by the contribution of replenished neural energy directly transferred to the nerve cells and glial tissues, it is proposed that it could arrest the progress of ALS with the implication of its possible reversal.

The clinical features of ALS [10,11], are the primary means by which a diagnosis is carried out. Although not straightforward, the time between the onset of the disease and when the disease has progressed far in its course and involves many parts of the body, the patient's appearance and the findings on the

neurologic examination oftentimes provide sufficient evidence for the diagnosis [12]. One aetiologic assumption that neural tissues degenerate because of lack synthesis of macromolecules. In 75-80% of patients, symptoms begin with limb involvement and evolve into a loss of function and painless weakness to more regions of the body. Bulbar symptoms are evident in 20-25% of the cases. As the disease becomes more advanced, muscle atrophy and complete debilitation of the somatic nervous system is evident.

*(Open) Now on He three taken care for us & other word*

Some studies have been carried out which try to link a causal effect between the use of magnetic fields as a means of therapy for treatment of ALS disorders [13,14]. A further line of inquiry involves direct stimulation of the somatic nervous system to trigger the release of ATP from the somata of DTG neurons [15]. This paper will extend this concept by describing a treatment of neuron starvation by direct stimulation of nerve fibers and ganglia to increase the production of ATP neurotransmitter. It is the goal of this paper to present a contribution to medical science for the treatment of ALS using wireless currents.

DTG.  
ATD

This paper will provide a set of descriptions of inductive-capacitive coupling of an external circuit and neuronal tissues by potential linking, transfer of magnetic energy in the form of electrically-biased, leveled-power eddy current, and induced current and manifest voltage of currents transported to organic material without surgical invasion. The practicality of this is to stimulate neural tissues in order to treat neural disorders such as amyotrophic lateral sclerosis. This paper will discuss a machine designed with the expressed medical purpose is to show that neural tissues can be charged with compatible power (compatible in the sense that they utilize the energy at this characteristic frequency) and show greater activity of somatic tissues.

### The theoretical model

*what model? No model.*

Given the conceptual model for the treatment of ALS presented in the previous section, it is relevant to expand it into a theoretical principle to describe treatment. The theoretical model is based upon two critical assumptions and one side-effect hypothesis:

- what is the assumption?*
1. The assumption that the level of energy within the tissues affected by the disorder, including those atrophying, reflects a lack of self repair. For example, certain catalytic drugs are administered which excite the chemical structures of the neural tissues with either an increase of sodium for enhanced firing and linking or an increase of potassium for enhanced suppression.
  2. The degree of neural activity in tissue is determined by the degree which neurons are firing and the amount of contiguous firing between neurons. This implies the connectedness between pools of neurons, arbitrarily bounded over a given area and demonstrates the transfer of energy from one set to the next.
  3. The number of engaged neurons is represented by the amount of spiking relative to the applied energy and the potential voltage and electrical current passing through the ion conductance. At the limit of conductance, new extraneous linking by dendrite growth includes not only new links but new neurons [16-21].

Spiking behavior and its governing equations [22] form the basis of the measure of the effectiveness of energy transfer between the external device and neural tissues. Given the suggested ability of the

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#?

No clear what  
are the implications  
of your model

mammalian brain to grow neuronal tissues [23], it is an interesting aspect of this research to understand not only that we can charge neuronal tissues, but actually stimulate neurogenesis [24] for exposure times and cognitive training suitable for persons who have lost neurons due to health or accident.

OK

The machine under consideration applies magnetic-resonant fields to subcutaneous neural tissues by potential inking between the metallic disc surface in the form of eddy currents, and the electrical potential field in the peripheral nervous system—the neurons (which receive and transmit impulses) and neuroglia (which assist the propagation of impulses as well as provide nutrients). Comprised of nerves and ganglia, chemical reactions due to the interaction of induced electrical currents stimulate the production of neurotransmitters. The field effects the axon by increasing its output of action potential signals by the absorption of electrical energy via linked inductances forming a resonant circuit.

fine  
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a picture

### Neuronal somata and the transmission of energy via magnetic-resonant fields

explain what is the sedition all about

The unity potassium and sodium currents of Hodgkin-Huxley [25] and observation of analogous currents [26] for the electrical model of the machine. The conditions associated with sporadic ALS [27] finds that the metabolic disturbance of mitochondria can be addressed by influencing the metabolic process in direct energetic replenishment. As the connection between neuronal activity and glucose metabolism has been established [28], this paper will turn the idea on its head that introduction of energy alters the existing homeostasis of the localized tissues triggering a metabolic response from the brain, increasing the regulatory mechanism of systemic glucose metabolism.

No evidence  
from this  
paper?  
No neural  
experience

Like the Hodgkin-Huxley model, this paper will treat the component of an excitable cell as an electrical element, for the purposes of mathematizing the hypothesis. To begin, the original model [25] described the properties of an excitable cell by four continuous differential equations:

$$\begin{aligned} I_0 &= C_m \frac{dV_m}{dt} + \bar{g}_K n^4 (V_m - V_K) + \bar{g}_{Na} m^3 (V_m - V_{Na}) + \bar{g}_I (V_m - V_I), \\ \frac{dn}{dt} &= \alpha_n (V_m) (1-n) - \beta_n (V_m) n, \\ \frac{dm}{dt} &= \alpha_m (V_m) (1-m) - \beta_m (V_m) m, \\ \frac{dh}{dt} &= \alpha_h (V_m) (1-h) - \beta_h (V_m) h \end{aligned}$$

?

$V_m$      $V_K$      $V_{Na}$   
 $\alpha_n$      $\beta_n$      $\alpha_m$   
 $\alpha_m$      $\beta_m$      $\alpha_h$   
 $\beta_h$      $V_I$      $V_{Na}$  ??

where  $I_0$  is the current per unit area,  $\alpha_i$  and  $\beta_i$  are rate constants for the  $i^{th}$  ion channel dependent upon voltage,  $\bar{g}_i$  is the maximum value of conductance,  $n, m, h$  are dimensionless quantities between 0 and 1 associated with potassium channel activation, sodium channel activation, and sodium channel deactivation, respectively. Setting  $p = (n, m, h)$ ,  $\alpha_p$  and  $\beta_p$  take the form

from 25?

$$\alpha_p(V_m) = \frac{p_\infty(V_m)}{\tau_p}, \quad (2)$$

$$\beta_p(V_m) = \frac{1 - p_\infty(V_m)}{\tau_p},$$

where  $p_\infty$  and  $1 - p_\infty$  are the steady state values for activation and deactivation and are usually represented by the Boltzmann equations as functions of  $V_m$ . The values of  $\alpha$  and  $\beta$  are represented as

$p_\infty$  linked with  $p$

$P$   $\dot{P}$ ?

$$\alpha_n(V_m) = \frac{0.01(V_m - 10)}{\exp\left(\frac{V_m - 10}{10}\right) - 1},$$

$$\alpha_m(V_m) = \frac{0.1(V_m - 25)}{\exp\left(\frac{V_m - 25}{10}\right) - 1},$$

$$\alpha_h(V_m) = 0.07 \exp\left(\frac{V_m}{20}\right),$$

$$\beta_n(V_m) = 0.125 \exp\left(\frac{V_m}{80}\right),$$

$$\beta_m(V_m) = 4 \exp\left(\frac{V_m}{18}\right),$$

$$\beta_h(V_m) = \frac{1}{\exp\left(\frac{V_m - 30}{10}\right) + 1}.$$

Generalized oftentimes [29] to

$$\frac{A_p(V_m - B_p)}{\exp\left(\frac{V_m - B_p}{C_p}\right) - D_p}. \quad (4)$$

Not an equation

In order to exhibit the behavior of (3) in the somata itself, a voltage clamp triggers neural firing. This paper will introduce a voltage potential directly to somata by wireless magnetic-resonant currents applied via eddy currents over the area of a metallic disc transferred through the skin. The mathematical components of transfer of power by a magnetic-resonant field relative to this application are the energy quotient, the inductance-link potential, and the coupling between the coil, a metallic disc, the surface of the skin, and the neuronal somata containing the  $K^+$  and  $Na^+$  ion conductance.

A time-varying current in a coil of wire induces an alternating magnetic field at right angles to it whose dynamics follow the curvature along the surface at the aperture. For a loop coil impressed with a current,  $I_1$ , the energy,  $U_\phi$ , as a summation of the forces form a radiation field with a hyperbolic structural

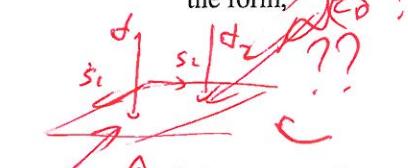
what is this?

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use a picture

↳ I mean we don't see  
the purpose! be clearer.

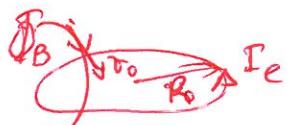
pattern. If the forces responsible for the magnetic field oscillate at a sufficiently low frequency, it is possible to accurately model them in a quasi-static context [30]. As such, the forces appear as energy in the form,



*what do you mean*

$$U_\phi = \frac{1}{2} \sum \iint \sigma_1 \sigma_2 r^{-1} \cos \epsilon_B ds_1 ds_2, \quad \leftarrow \text{from 30?} \quad (5)$$

where  $\sigma_1$  and  $\sigma_2$  are the forces in the plane  $s_1 s_2$ , given the radius of the coil,  $r$ , and the angle of the field,  $\epsilon_B$ , relative to the surface of the conductive material. Lenz's law states that the direction of the induced current is such as to oppose the applied current producing it. Given the material of the disc and its thickness, the eddy current takes the form of a polarization of the medium. The motion of the force of polarization is a vortex disturbance in the form of rotating conic sections converging to a point demarcated by the potential link [31]. The eddy current,  $I_e$ , is given by



$$I_e = \frac{-1}{r} \frac{d\Phi_B}{dt} \frac{v_0}{\mu_0},$$

[31]?

*why? show they or give eqn RL*

*picture*

where  $r$  is the radius and  $v_0$  is the velocity of the field  $\Phi_B$ . Generally speaking, eddy currents flow in closed loops within conductors, in planes perpendicular to the magnetic field. The energy stored in the electromagnetic field is confined within a boundary,  $\Omega$ , given the projection of the zero order electrical,  $E_0$ , and first order magnetic,  $H_1$ , fields [32], as,

$$\begin{aligned} & \int_{\Omega} \left( \sigma_0 E_0 \frac{\partial E_0}{\partial t} + \mu_0 H_0 \frac{\partial H_1}{\partial t} \right) d\Omega + \int_{\Omega} \frac{|J|^2}{\sigma} d\Omega \\ & - \int_{\Omega} E_i J d\Omega + \oint_{\Gamma} (E_0 \times H_0) d\Gamma = 0. \end{aligned} \quad (7)$$

The first integral accounts for the rate of change of energy density of the electromagnetic field in the volume  $\Omega$ , the second integral energy dissipation, the third integral resonant energy by the input voltage, and the last integral the intensity of flow on the surface,  $\Gamma$ , of the Poynting vector. The total magnetic field energy,  $W_T$ , inside the volume  $\Omega$ , equal to the stored energy in the circuital elements  $L_1, L_2$  and  $M_{12}$  is,

$$W_T = \int_{\Omega} \frac{\mu_0 H^2}{2} = \frac{L_1 I_1^2}{2} + M_{12} I_1 I_2 + \frac{L_2 I_2^2}{2}, \quad (8)$$

where the average stored magnetic energy at resonance in the transmitter circuit is,

*) needs to explain thus*

*(in short that it is an equivalent circuit*

$$\begin{aligned}
 \langle W_m \rangle_{a_0} &= \frac{1}{4} \int_0^l L_1 |I_1|^2 dz \\
 &= \frac{1}{4} L_1 |I_1|^2 \int_0^l \cos^2\left(\frac{\pi z}{l}\right) dz \\
 &= \frac{1}{8} L_1 l |I_0|^2
 \end{aligned} \tag{9}$$

Considering the non-neuronal organic tissue as a periodic structure in an off-resonant state [33, 34] with implicit time-dependence  $e^{j\alpha_0 t}$  that have the spatial dependences  $e^{-j\beta_1 z}$  and  $e^{-j\beta_2 z}$ , due to the electrical potential of the field, respectively where  $\beta_1 \approx \beta_2$  are propagation constants due to the strength of the link. For a period,  $\Lambda$ , due to the frequency of the oscillator and the feedback dampening of the neural tissues, the coupling coefficient takes the form  $2\kappa_{12} \cos(2\pi z/\Lambda)$ .

#### Potential linking by inductance

The linking model, based on the notion of coupled modes [33, 35-39], is formulated around the strength of the potential field between the coil-oscillator combination as transmitter and neuronal somata as receivers, in the form circuits [25]. The definition of inductance, a primary feature of linking, revolves around a semi-classical interpretation that it does not exist purely for its own sake, rather, due to a tension between two resonant objects which, by a favorable geometric arrangement, are in near enough proximity to be considered coupled. The quantities required for potential linking and those representing its effect are the coupled force,  $F_\kappa$ , the potential of the field,  $A$ , its displacement,  $D$ , and the current density,  $J$ . The absorption taking place at a dielectric axon has the quantities of consequential electricity parallel to it at in terms of its electrical properties from (1), cumulate to  $p'$ , as,

$$\left. \begin{aligned}
 p' &= p + \frac{dn}{dt}, \\
 q' &= q + \frac{dm}{dt}, \\
 r' &= r + \frac{dh}{dt}.
 \end{aligned} \right\} \text{Not in bold} \tag{10}$$

Linear approximation?

The force of the link is dependent upon the capacitance,  $C_2$ , of the ions in the axon and its connective length,  $\ell_2$ , and the mutual inductance between the coil and somata,  $M_{12}$ ,

$$F_\kappa = \frac{2}{C_2 \ell_2} M_{12}, \tag{11}$$

given the consideration of momentum at any point in the field over the length of the axon connectivity in the plane,  $s$ ,

Not clear

Not an equation

$$\int \left( F \frac{dn}{ds} + G \frac{dm}{ds} + H \frac{dh}{ds} \right) ds. \quad (12)$$

related to the magnetic intensity, first from the magnetic force from the device,

$$\begin{aligned} \mu\alpha &= \frac{dH}{dy} - \frac{dG}{dz}, \\ \mu\beta &= \frac{dF}{dz} - \frac{dH}{dx}, \\ \mu\gamma &= \frac{dG}{dx} - \frac{dF}{dy}. \end{aligned} \quad ) \quad \text{in } \mathbb{R}^3 ? \quad (13)$$

define  $\alpha, \beta, \gamma, p, G, F$  ~~etc.~~

ref [1]

It is well-known the motion of a magnetic pole in the electromagnetic field in a closed circuit cannot generate work unless the circuit which the pole describes passes around via electric current. Hence, except in the space occupied by the electric currents,

$$\alpha dx + \beta dy + \gamma dz = d\varphi,$$

$d\varphi$  is differential form  
 $d\varphi \in \Lambda^1(\mathbb{R}^3) ??$

describes a differential of the scalar potential,  $\varphi$ . When in the proximity of the current  $p'$ , completely around the circuit  $4\pi$ , yields,

$$\begin{aligned} 4\pi p' &= \frac{d\gamma}{dy} - \frac{d\beta}{dz}, \\ 4\pi q' &= \frac{d\alpha}{dz} - \frac{d\gamma}{dx}, \\ 4\pi r' &= \frac{d\beta}{dx} - \frac{d\alpha}{dy}, \end{aligned} \quad \left. \begin{array}{l} \\ \\ \end{array} \right\} \quad \begin{array}{l} \text{where thus } \varphi \\ \text{come from?} \end{array} \quad (15)$$

which is Maxwell's equation of currents, contextualized here for the axon and glial cells of the neuronal somata. Therefore, the displacement force,  $F_D$ , acting on the axon and glial cells, demarcated as an element of length,  $ds$ , in proximity to the magnetic field is

$$F_D = \int_{n=0}^{xy} \left( P \frac{dx}{ds} + Q \frac{dy}{ds} + R \frac{dz}{ds} \right) ds. \quad (16)$$

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explain/  
re-formula-

When an electromotive force acts upon a dielectric, the dielectric's state is transformed into a polarized condition where currents oscillate along its length. A feedback force reflects energy back upon the potential link. The link is related closely to the formalism for  $\mathbf{A}$  described by the geometry of moving forces within the magnetic field. Consequently, it is more relevant to discuss the potential link as a vector decomposition for the time-harmonic case, where the changing magnetic field induces electrical currents in the metallic disc,

{ can not converge -

what is this?

$$\mu_r \epsilon_0 \frac{\partial \mathbf{A}}{\partial t} + \nabla \times \sum_{i=1}^{n_s} (\mu_0^{-1} \nabla \times \mathbf{A}) = \mu_0 \mathbf{J}_s + \mu_0 \epsilon_0 n_s \frac{V_1}{2\pi r}, \quad (17)$$

over the neuronal somata,  $n_s$ , where  $\mu_0$  is the permeability of the vacuum,  $\mu_r$  the relative permeability of the organic tissues lying on the path of the field,  $\sigma_0$  the electric conductivity of the tissues, and  $V_1$  the voltage contained in the primary circuit. The magnitude of the potential link,  $F_k$ , is defined by the inductance properties of the circuit and of the neuronal somata coupled to the circuit via the link. The potential link is related to the solution for the vector potential,  $\mathbf{A}$ , expressed in terms of the geometry of the motion at the disc, e.g., a line integral over the region encompassed by the disc of the parametric form  $x^2 + y^2 = r$ , where  $r$  is the radius of the area under the loop. For the case of a current point-source where charges are manifest in the eddy currents as point-vortices, is derived in terms of the potential between the boundary of the coil and the area immediately under it, and between this area and the distance to the first neural tissues. The displacement of energy from the electromagnetic forces form a contour to the limit at the dispersion of the field as oscillating ordered fields described mathematically by family time [40],

$$\mathbf{A}_{B_{LOOP}} = \frac{\mu_0 I}{4\pi} \oint \frac{d\vec{l}}{r}, \mathbf{A}_{D_{SOMATA}} = \frac{\mu_0 \mathbf{I}}{4\pi} \oint_C \frac{e^{-j\omega r}}{r} dl, \quad (18)$$

where  $\mathbf{I} = I \cos \omega_0 t$ . The eddy currents coupled to the somata take the form a current density because of the assumption of a contour, the propagation of the eddy currents as collections of potential vortices,

$$\mathbf{A}_e = \frac{\mu_0}{4\pi} \oint \frac{j d\vec{S} \cdot d\vec{l}}{r}, \quad (19)$$

and this value is taken to be  $-0.0002820196456 \text{ V*s/m}$  when  $r = 2.5 \text{ cm}$ . The total electromagnetic force acting on region of this space  $\Omega$  can be obtained by integrating Maxwell's stress tensor and the Larmor force on the delimiting boundary  $\partial\Omega$ ,

$$\mathbf{F} = \int_{\partial\Omega} T \mathbf{n} dS. \quad (20)$$

Therefore, the spatial equations are,

$$\begin{aligned} \frac{d}{dz} a_1 &= -j\beta_1 a_1 \left[ \mu_r \epsilon_0 \frac{\partial \mathbf{A}}{\partial t} + \nabla \times \sum_{i=1}^{n_s} (\mu_0^{-1} \nabla \times \mathbf{A}) \right] \cdot F_k j 2\kappa_1 \cos\left(\frac{2\pi z}{l}\right) a_2, \\ \frac{d}{dz} a_2 &= -j\beta_2 a_2 \left[ \mu_r \epsilon_0 \frac{\partial \mathbf{A}}{\partial t} + \nabla \times \sum_{i=1}^{n_s} (\mu_0^{-1} \nabla \times \mathbf{A}) \right] \cdot F_b j 2\kappa_2 \cos\left(\frac{2\pi z}{l}\right) a_1, \end{aligned} \quad (21)$$

given a with a weak time dependence of amplitudes  $a_1$  and  $a_2$  because the link is passive,

?

$$\begin{aligned}\frac{d}{dt}a_1 &= j\omega_1 a_1 + j\kappa_1 a_2, \\ \frac{d}{dt}a_2 &= j\omega_2 a_2 + j\kappa_2 a_1,\end{aligned}$$

know equation  
so Not sure  
what is all  
about - (22)

relative to the coupled steady-state,

$$\kappa_1 = \kappa_2 = \frac{M_{12}}{\sqrt{L_1 L_2}}. \quad (23)$$

Where the utilized energy is given by the energy transferred to the somata by the conductance potential.

#### Inductive-reactive feedback between the coil and neural somata to regulate energy flow

The circuit-based approach of [25] for neural somata allows the consideration of power transfer in the context of this paper from a primary circuit to a secondary circuit. To accomplish this, a load impedance  $Z_L$ , defines the relation of secondary voltage  $V_2$ , to secondary current  $I_2$ ,

$$I_2 Z_L = V_2 = j\omega_0 L_2 I_2 - j\omega_0 M_{12} I_1, \quad (24)$$

and relates the secondary current as function of primary current,  $I_1$ , as

$$I_2 = -\frac{j\omega_0 M_{12}}{Z_L - j\omega_0 L_2} I_1, \quad (25)$$

where  $\omega_0$  is the resonant frequency. This establishes the relation of primary voltage to primary current,

$$V_1 = \left[ j\omega_0 L_1 + \frac{\omega_0^2 M_{12}^2}{Z_L - j\omega_0 L_2} \right] I_1. \quad (26)$$

Therefore, the apparent primary power,  $P_{IN}$ , and secondary power,  $P_{OUT}$ , relations are,

$$\begin{aligned}P_{IN} &= V_1 I_1 = \left[ j\omega_0 L_1 + \frac{\omega_0^2 M_{12}^2}{Z_L - j\omega_0 L_2} \right] I_1^2, \\ P_{OUT} &= I_2^2 Z_L = \left( -\frac{j\omega_0 M_{12}}{Z_L - j\omega_0 L_2} \right)^2 I_1^2 Z_L.\end{aligned} \quad (27)$$

Apparent power contains effective power, able to produce ion conductance and reactive power from the neuronal somata back to the coil. Energy transfer can be considered by the ratio of secondary output per primary input apparent power, as,

Figure to explain?

$$\frac{P_{OUT}}{P_{IN}} = \frac{\omega_0 M_{12} Z_L}{\omega_0 (2L_1 L_2 Z_L + M_{12}^2 Z_L) + j [L_1 Z_L^2 - \omega_0^2 (L_2 M_{12}^2 + L_1 L_2^2)]}. \quad (28)$$

For the case of negligible secondary yielding a very high impedance,  $Z_L$ , there is still a secondary voltage present in the  $K^+$  and  $Na^+$  ion channels containing an associated current over its inductance in order to regulate the amount of absorption of energy. In this case, the apparent power will be only reactive, appearing as such to the primary side of the circuit,

$$\left. \frac{P_{OUT}}{P_{IN}} \right|_{for I_2 \ll} = \left| \frac{-I_1^2 j \omega_0 M_{12}}{I_1^2 j \omega_0 L_1} \right| \rightarrow \frac{M_{12}}{L_1} = K_{12} \sqrt{\frac{L_2}{L_1}} = \frac{K_{12}}{T_{12}}, \quad (29)$$

where  $T_{12}$  is the voltage transformation ratio  $V_2/V_1$ . Therefore the neuronal somata will only absorb the amount of energy required to elevate its energy level to its steady state while the external circuit will not send energy above this limit.

### Experimental work

*Needs some context? Real subject? age etc...*

The first set of experiments was coupling to the central nervous system near to the base of the spinal cord and see if the power transfer behaved in the manner described by the hypothesis in the previous section. The idea of the experiment is to see if a method of energy transfer to neuronal tissues could be accomplished in an area populated with more neurons, e.g., in the dorsal root ganglia. The experiment was to determine two things: 1. If the apparatus could transfer a steady flow of energy to the muscle group by measuring after fifteen minutes of exposure the neurons under the area of the metallic disc if the neurons manifest a fast spiking (with oscillatory) pattern, and, 2. Measure how long the neurons under the area of the disc which had been exposed they continued manifesting the fast spiking pattern. By defining the threshold of the experiment in this manner, it could be determined how efficient the apparatus is at energy transfer and demonstrate the neural response to applied magnetic-resonant fields. Data sets from the EEG device are taken for an interval of five minutes.

*How did you conduct the experiments?*

The area, as measured by four EEG probes, before exposure is shown in Fig. 7. The figure shows the two channels mixed over a center rest value of 0. EEG probes numbered one (in red) and two (in blue), each with a positive and negative polarity show that the pattern in exhibiting a regular spiking pattern with a maximum average level of  $60 \mu V$ .

Next, the area was exposed to magnetic resonant fields. The area, as measured by four EEG probes, immediately after fifteen minutes of exposure by the apparatus is shown in Fig. 8. The figure shows the same measurement points in the graph but in this instance they are exhibiting a fast spiking pattern. It is also shown in the figure that each channel is measuring an oscillatory pattern in the firing of the neurons.

The area, as measured by four EEG probes, 76 minutes after exposure is shown in Fig. 9. The figure shows each channel split so the pattern can be better shown. The pattern shown that the Psoas Minor is still exhibiting a fast spiking pattern while the Psoas Major has returned to its original pattern before exposure.

*No it's very clear.*

*real exposure?*

*Recent does not make any sense*

EEG? ?

The area, as measured by four EEG probes, 149 minutes after exposure is shown in Fig. 10. The pattern shows that the Psoas Minor has slowed its oscillatory pattern yet is still exhibiting a fast spiking pattern while the Psoas Major has remained in its original pattern before exposure.

The area, as measured by four EEG probes, 160 minutes after exposure is shown in Fig. 11. The pattern shows that the Psoas Minor has slowed its oscillatory pattern and is not exhibiting a fast spiking pattern while the Psoas Major has slowed compared to its original pattern before exposure.

The area, as measured by four EEG probes, 180 minutes after exposure is shown in Fig. 12. The pattern shows that the Psoas Minor has slowed its oscillatory pattern and is returning to its original pattern before exposure while the Psoas Major has settled to its original signal level before exposure.

The second set of experiments was coupling to the peripheral nervous system for neurons located in the right hand and see, again, if the power transfer behaved in the manner described by the hypothesis in the previous section. The idea for an experiment like this is to see if the technique works: 1. in an area external to the central nervous system serving as a communication relay, and, 2. in an area most often associated noted as the beginning point of ALS, e.g., the extremities.

## Conclusion

This paper has discussed a method of transfer of energy by magnetic-resonant fields to neuronal somata by coupling through the magnetic potential of the antenna and the somata groups facilitated by the novel concept of potential linking. This paper has presented a method whereby the energy can be used to induce electrical currents in  $K^+$  and  $Na^+$  conductance channels in somata increasing their firing pattern and facilitating the growth of new axons and dendrites. This method is best served as a treatment for ALS.

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