Magnetic-resonant fields for the treatment of motor neuron disorders

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Keywords

Amyotrophic lateral sclerosis, magnetic-resonant coupling, potential fields, neuronal somata.

**Abstract**

A means of magnetic resonant coupling of modes from an external circuital 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.

1. **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.

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.

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.

1. **The theoretical 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:

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 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.

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.

1. **Neuronal somata and the transmission of energy via magnetic-resonant fields**

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.

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



where  is the current per unit area,  and  are rate constants for the  ion channel dependent upon voltage,  is the maximum value of conductance,  are dimensionless quantities between 0 and 1 associated with potassium channel activation, sodium channel activation, and sodium channel deactivation, respectively. Setting   and  take the form



where  and  are the steady state values for activation and deactivation and are usually represented by the Boltzmann equations s functions of  The values of  and  are represented as



Generalized oftentimes [29] to



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.

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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,  the energy,  as a summation of the forces form a radiation field with a hyperbolic structural 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,



where  and  are the forces in the plane  given the radius of the coil,  and the angle of the field,  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,  is given by



where  is the radius and  is the velocity of the field  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,  given the projection of the zero order electrical,  and first order magnetic,  fields [32], as,



The first integral accounts for the rate of change of energy density of the electromagnetic field in the volume  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,  of the Poynting vector. The total magnetic field energy,  inside the volume  equal to the stored energy in the circuital elements  and  is,



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



Considering the non-neuronal organic tissue as a periodic structure in an off-resonant state [33, 34] with implicit time-dependence  that have the spatial dependences  and  due to the electrical potential of the field, respectively where  are propagation constants due to the strength of the link. For a period,  due to the frequency of the oscillator and the feedback dampening of the neural tissues, the coupling coefficient takes the form 

1. **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,  the potential of the field,  its displacement,  and the current density,  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 , cumulate to  as,



The force of the link is dependent upon the capacitance,  of the ions in the axon and its connective length, and the mutual inductance between the coil and somata, 



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



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



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,



describes a differential of the scalar potential,  When in the proximity of the current  completely around the circuit  yields,



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



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 fordescribed 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,



over the neuronal somata,  where  is the permeability of the vacuum,  the relative permeability of the organic tissues lying on the path of the field,  the electric conductivity of the tissues, and  the voltage contained in the primary circuit. The magnitude of the potential link, 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, 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  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],



where  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,



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



Therefore, the spatial equations are,



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



relative to the coupled steady-state,



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

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1. **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  defines the relation of secondary voltage  to secondary current 



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



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



Therefore, the apparent primary power,  and secondary power,  relations are,



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,



For the case of negligible secondary yielding a very high impedance,  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,



where  is the voltage transformation ratio  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.

1. **Experimental work**

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.

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 µV.

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.

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.

1. **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.

**References**

1. Piemonte and Valle d’Aosta Register for Amyotrophic Lateral Sclerosis (PARALS). “Incidence of ALS in Italy: evidence for a uniform frequency in Western countries,” *Neurology*, Vol. 56, pp. 239-44, 2001.
2. H. Hodkinson. “More favourable prognosis of motor neurone disease in old age,” *Age Ageing*, Vol. 1, pp. 182-84, 1972.
3. E. Cellura, R. Spataro, A.C. Taiello, V. La Bella. “Factors affecting the diagnostic delay in amyotrophic lateral sclerosis,” *Clinical Neurology and Neurosurgery*, Vol. 114, pp. 550-54, 2012.
4. J.R. Williams, D. Fitzhenry, L. Grant, D. Martyn, and D.A. Kerr. “Diagnosis pathway for patients with amyotrophic lateral sclerosis: retrospective analysis of the US Medicare longitudinal claims database,” *BioMedCentral Neurology*, Vol. 13, No. 1, pp. 160-67, 2013.
5. L.C. Wijesekera and P.N. Leigh. “Amyotrophic lateral sclerosis,” *Orphanet Journal of Rare Diseases*, Vol. 4, No. 1, pp. 1-22, 2009.
6. C. Armon. Epidemiology of ALS/MND, In: Shaw P and Strong M, eds. *Motor Neuron Disorders*. Elsevier Sciences, pp. 167-206, 2003.
7. C. Armon. *ALS 1996 and Beyond: New Hopes and Challenges. A manual for patients, families and friends*, 4th Edition. California: LLU Department of Neurology, 2007.
8. M.R. Turner, J. Scaber, J.A. Goodfellow, M.E. Lord, R. Marsden, and K. Talbot. “The diagnostic pathway and prognosis in bulbar-onset amyotrophic lateral sclerosis,” *Journal of the Neurological Sciences*, Vol. 294, No. 1, pp. 81-85, 2010.
9. D.W. Mulder, “Clinical limits of amyotrophic lateral sclerosis,” *Advances in Neurology*, Vol. 36, pp. 15-22, 1981.
10. J.F. Kurtzke. “Epidemiology of amyotrophic lateral sclerosis,” *Advances in Neurology*, Vol. 36, pp. 281-302, 1981.
11. R.B. Forbes, S. Colville, and R.J. Swingler. “The epidemiology of amyotrophic lateral sclerosis (ALS/MND) in people aged 80 or over,” *Age and Ageing*, Vol. 33, No. 2, pp. 131-4, 2004.
12. C. Wood-Allum and P.J. Shaw. “Motor neurone disease: a practical update on diagnosis and management,” *Clinical Medicine*, Vol. 10, No. 3, pp. 252-8, 2010.
13. G. Null. *Biomagnetic healing*, http//www.garynull.com/Documents/magnets.htm, 1998.
14. A. Bellossi and R. Berget. “Pulsed Magnetic Fields: A Glimmer of Hope for Patient Suffering from Amyotrophic Lateral Sclerosis,” *In Electricity and Magnetism in Biology and Medicine*, pp. 891-93. Springer, 1999.
15. X. Zhang, Y. Chen, C.L. Wang, and L.-Y.M. Huang. “Neuronal somatic ATP release triggers neuron–satellite glial cell communication in dorsal root ganglia,” *Proceedings of the National Academy of Sciences*, Vol. 104, No. 23, pp. 9864-9, 2007.
16. P. Fromherz, “Interfacing neurons and silicon by electrical induction,” *Berichte der Bunsengesellschaft für physikalische Chemie*, Vol. 100, Iss. 7, pp. 1093–1102, Jul. 1996.
17. S. Vassanelli and P. Fromherz, “Neurons from rat brain coupled to transistors,” *Applied Physics* *A*: Materials Science & Processing, Vol. 65, No. 2, pp. 85-88, Jun. 1997.
18. E. García-Pérez, M. Vargas-Caballero, N. Velazquez-Ulloa, A. Minzoni, and F. De-Miguel, “Synaptic Integration in Electrically Coupled Neurons,” *Biophysics Journal*, Vol. 86, No. 1, pp.646–55, Jan. 2004.
19. J. Altman, “Are new neurons formed in the brains of adult mammals?” *Science*, Vol. 30, No. 135, pp.1127-28, Mar. 1962.
20. S. Pacini, G. Vannelli, T. Barni, M. Ruggiero, I. Sardi, P. Pacini, M. Gulisano, “Effect of 0.2T static magnetic field on human neurons: remodeling and inhibition of signal transduction without genome instability,” *Neuoscience Letters*, Vol. 267, 185-88, Apr. 1999.
21. D. Formica and S. Silvestri, “Biological effects of exposure to magnetic resonance imaging: an overview,” *Biomedical Engineering*, Vol. 3, No. 11, Apr. 2004.
22. E. Izhikevich, “Simple model of spiking neurons,” *IEEE Transactions on Neural Networks*, Vol. 14, No. 6, pp. 1569-72, Nov. 2003.
23. C. Walton, E. Pariser, F. Nottebohm, “The Zebra Finch Paradox: Song Is Little Changed, But Number of Neurons Doubles,” *Journal of Neuroscience*, Vol. 32, Iss. 3, pp. 761-774, Jan. 2012.
24. M. Kaplan, “Environment complexity stimulates visual cortex neurogenesis: death of a dogma and a research career,” *Trends in Neuroscience*, Vol. 24, Iss. 10, pp. 617-20, Oct. 2001.
25. A.L. Hodgkin and A.F. Huxley, “A quantitative description of membrane current and its application to conduction and excitation in nerve,” *The Journal of Physiology,* Vol. 177, No. 4, pp. 500-44, 1952.
26. J.A. Connor and C.F. Stevens, “Voltage clamp studies of a transient outward membrane current in gastropod neural somata,” *The Journal of Physiology*, Vol. 213, No. 1, pp. 21-30, 1971.
27. S. Sasaki and M. Iwata, “Mitochondrial alterations in the spinal cord of patients with sporadic amyotrophic lateral sclerosis,” *Journal of Neuropathology & Experimental Neurology*, Vol. 66, No. 1, pp. 10-16, 2007.
28. B. Göbel, K.M. Oltmanns, and M. Chung, “Linking neuronal brain activity to the glucose metabolism,” *Theoretical Biology and Medical Modelling*, Vol. 10, No. 1, 2013.
29. M.E. Nelson, *Electrophysiological Models In: Databasing the Brain: From Data to Knowledge*, Wiley, 2004.
30. J. Larmor, “A dynamical theory of the electric and luminiferous medium. Part II: Theory of electrons,” *Proceedings of the Royal Society London*, Vol. 58, pp. 222-28, No. 347-52, Jan. 1895.
31. J.T. Cacioppo, L.G. Tassinary, and G.G. Berntson, ed. *Handbook of psychophysiology, Third edition*, New York: Cambridge University Press, p. 121, 2007.
32. R. Alder, L. Chu, and R. Fano. *Electromagnetic Energy Transmission and Radiation*, John Wiley & Sons, Inc., Cambridge, 1969.
33. C.A. Tucker, “Magnetic resonant modes in a wireless-powered circuit,” *IEEE Telecommunications Forum (TELFOR), 2011 19th*, pp. 977-980.
34. C.A. Tucker, K. Warwick, and W. Holderbaum, “A contribution to the wireless transmission of power,” *International Journal of Electrical Power and Energy Systems,*  Vol. 47, pp. 235-42, 2013.
35. J.D. Kraus, *Antennas for all application*, *3rd Edition*, New York, McGraw-Hill, 2002.
36. H. Haus and W. Huang, “Coupled-mode theory,” *Proceedings of the IEEE*, Vol. 79, No. 10, pp.1505 – 1518, Oct. 1991.
37. B.L. Cannon, J. Hoburg, D. Stancil, and S. Goldstein, “Magnetic resonant coupling as a potential means for wireless power transfer to multiple small receivers”, *IEEE Transactions on Power Electronics*, Vol. 24, No. 7, Jul. 2009.
38. C.A. Tucker, K. Warwick, and W. Holderbaum, “Efficient wireless power delivery for biomedical implants,” *IET Wireless Sensor Systems,* Vol. 2, No. 3 pp. 176-82, 2012.
39. J. Ho et al., “Wireless power transfer to deep-tissue microimplants,” *Proceedings of the National Academy of Sciences*, 2014.
40. R. Fano, L. Chu, and R. Alder, R. *Electromagnetic Fields, Energy, and Forces*, John Wiley & Sons, New York, 1969.