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INTERACTION OF STATIONARY MAGNETIC FIELDS
WITH THE NERVOUS SYSTEM AND VISION

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INTERACTION OF STATIONARY MAGNETIC FIELDS WITH THE NERVOUS SYSTEM AND VISION

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Introduction

Because the bioelectric properties of nerve are mediated by ionic conduction processes, a perturbing influence of stationary magnetic fields could occur through two physical mechanisms. The first type of interaction involves a distortion of the current patterns in nerve via the Lorentz force exerted on moving ionic charges. The second mechanism involves changes in nerve current patterns resulting from an inductive interaction between a stationary magnetic field and the moving current loops associated with a propagating action potential. Both of these interaction mechanisms have been modeled theoretically, and there is a substantial body of experimental data that can be compared to the predictions of these physical theories. Unfortunately, in many cases, contradictory results have emerged from experiments carried out in different laboratories using the same type of nerve, e.g., the frog sciatic nerve. A description is given here of the simple physical mechanisms through which stationary magnetic fields might alter the bioelectric activity of nerve, and the available experimental data are evaluated in the context of this theoretical framework. An effort is also made to assess inadequacies in experimental technique that may, in certain instances, have led to artifactual results that were incorrectly interpreted by the investigators as being indicative of a magnetic field influence on nerve function.

Theoretical aspects of magnetic field interactions with nerve tissues

An important aspect of the interaction of a magnetic field with nerve bioelectric activity is the vectorial nature of both the field and the ionic conduction currents responsible for impulse propagation. Using the Lorentz force law that describes the interaction between an applied magnetic field and

moving charges, Wikswo and Barach (1980) have calculated the magnitude of the field that would produce a measurable distortion in the conduction currents of an isolated nerve. The maximum effect of the field will occur when the ion drift velocity, $\vec{v}_d$, is orthogonal to the magnetic flux density, $\vec{B}$. Under this condition, the magnitude of the magnetic force, $\vec{F}_m$, experienced by the moving ion is equal to $|\vec{F}_m| = e|\vec{v}_d||\vec{B}|$, where e is the ion charge. The ion also experiences a strong electrical force, $\vec{F}_e$, imposed by the electric field, $\vec{E}$, of the nerve membrane. The magnitude of this force is $|\vec{F}_e| = e|\vec{E}|$. In order for the magnetic field to have a significant perturbing influence, $|\vec{F}_m|$ must obviously be a significant fraction of $|\vec{F}_e|$. Wikswo and Barach (1980) considered the specific case of $|\vec{F}_m| = 0.1|\vec{F}_e|$, under which condition the ion currents would be deflected by 10%. Using the above expressions for $|\vec{F}_m|$ and $|\vec{F}_e|$, the magnetic field strength producing this effect is equal to

$$|\vec{B}| = \frac{0.1|\vec{E}|}{|\vec{v}_d|} \tag{1}$$

The electric field $\vec{E}$ can be related to the nerve conductance properties by using Ohm's law, i.e., $\vec{E} = \rho\vec{J}$, where ρ is the electrical resistivity and $\vec{J}$ is the vectorial current density. The current density can be further related to the density of ionic charge carriers, N_c, by the relation $\vec{J} = N_c \cdot e \cdot \vec{v}_d$. Using this expression for $\vec{J}$ in Ohm's law leads to a final expression from which $\vec{v}_d$ can be calculated:

$$|\vec{v}_d| = \frac{|\vec{E}|}{\rho e N_c} \tag{2}$$

From values of ρ, N_c and $|\vec{E}|$ appropriate to a nerve conducting an action potential, Wikswo and Barach (1980) calculate that the maximum value of $|\vec{v}_d|$ is 3.3×10^{-2} m/s. Using this value for $|\vec{v}_d|$ and assuming $|\vec{E}| = 8$ V/m, which is

the approximate magnitude of the axial electric field during the propagation of an action potential, then the value of $|\vec{B}|$ calculated from eqn. (1) is 24 Tesla. Based on these simple physical concepts, Wikswo and Barach [1] concluded that the fields achieved by conventional iron-core electromagnets ($\lesssim$2 Tesla) should not measurably perturb the ionic current patterns during axonal impulse propagation. Phrased in slightly different terms, it could be stated that the transverse Hall effect potential and current induced in a conducting nerve by an applied magnetic field are negligible at field strengths below 2 Tesla. Based on a direct calculation of the Hall voltage, Liboff (1980) reached a conclusion entirely consistent with that of Wikswo and Barach (1980).

The second class of physical interactions between a stationary magnetic field and a conducting nerve axon results from the time-varying flux linkage of the field with the closed loops that define the paths of ionic conduction currents. This change in flux linkage gives rise to an induced potential, $V(\theta)$, which can be calculated from Faraday's law:

$$V(\theta) = -\frac{\partial}{\partial t} \oiint \vec{B} \cdot d\vec{S} \qquad (3)$$

where the integral is taken over the surface of an ionic conducting loop and θ is the angle between $\vec{B}$ and the normal to the loop defined by the surface area vector, $\vec{S}$. The induced current, I_{ind}, associated with $V(\theta)$ is equal to $G \cdot V(\theta)$, where G is the net conductance of the nerve axon.

It is evident from eqn. (3) that this form of magnetic interaction also depends upon the relative orientation of the field and the ionic currents involved in impulse propagation. It also follows immediately from the integration of eqn. (3) that the induced potential will be zero if the ionic

conduction loops have complete rotational symmetry about the nerve axis. Liboff (1980), however, has provided a more general theoretical framework to predict the inductive interaction of a magnetic field under conditions where the nerve conduction loops have an asymmetric pattern. By modeling the current loops as a network and integrating eqn. (3), he arrives at the following expression for the induced current:

$$I_{ind} = \frac{\alpha G B a v}{\pi} \tag{4}$$

where a is the axon radius and v is the action potential conduction velocity. The parameter α was introduced by Liboff (1980) to describe the degree of asymmetry of the ionic conduction loops. If $\alpha = 0$, the axonal current loops have rotational symmetry and there is a complete cancellation of the induced currents around the nerve. If $\alpha = 1$, the axonal current loops have a maximal asymmetry, i.e. the conduction currents are restricted to one half of the cylindrical axonal membrane. Under this condition, I_{ind} achieves a maximum value. If the parameters in eqn. (4) are assigned values appropriate to the squid giant axon, then the induced current achieves the same value as the axonal current when $B = 0.26/\alpha$ Tesla. For values of α between approximately 0.13 - 1.0, Liboff's theory therefore predicts that a measurable perturbation of action potential current flow should occur in fields of less than 2 Tesla strength. It must be noted, however, that there is no experimental evidence indicating that such a large degree of asymmetry exists in the conductance properties of axonal membranes.

The inductive interaction of a stationary magnetic field with axonal currents has also been considered by Valentinuzzi (1965), who concluded that this effect should be very small (and possibly undetectable) for individual nerves because of the symmetry of their current loops. However, for

interconnected networks of neurons such as those of the central nervous system, he argued that a multiplication of small magnetic inductive effects on the individual neuroelements could occur along a complex neural pathway, thereby producing a measurable electrophysiological effect. Valentinuzzi's discussion of this multiplicative phenomenon was of a qualitative nature, and no prediction was made of the threshold magnetic field strength required to produce an experimentally detectable effect on the central nervous system.

Bioelectric activity of isolated neurons in stationary magnetic fields

A. Excitation threshold. The threshold for neural excitation has been examined for both intact frog sciatic nerves and single myelinated sciatic nerve fibers during exposure to a homogeneous, stationary magnetic field (Liberman et al., 1959; Gaffey and Tenforde, 1983). In both studies, the field orientation was transverse to the nerve axis. No evidence was obtained in these experiments for a magnetic field effect on the minimum electrical stimulus strength required to evoke action potentials in either signle fibers or intact sciatic nerves.

An important observation that has a direct bearing on other studies described below was made by Gaffey and Tenforde (1983), who determined the temperature coefficient of the frog sciatic nerve excitation threshold. In experiments with 29 intact nerve preparations, they found that the submaximal action potential elicited by a threshold stimulus exhibited a 2.7 mV rise per 1.0 °C increase in the environmental temperature. This increase was 12.8% of the maximal action potential amplitude that could be evoked by suprathreshold stimuli. From this observation, they concluded that considerable care must be taken to maintain the ambient temperature within 0.1 °C when examining the threshold for nerve bioelectric activity.

B. Action potential amplitude and conduction velocity. Several groups of investigators have studied the properties of evoked action potentials in isolated nerve preparations during exposure to stationary magnetic fields oriented either parallel or perpendicular to the nerve axis. Schwartz (1978) exposed the circumesophageal connective nerve of the lobster to stationary fields with a maximum strength of 1.2 Tesla. The nerve preparation was maintained in an L-shaped chamber, and the field gradients along the sections of nerve oriented parallel and perpendicular to the field lines were 2 and 15 Tesla/m, respectively. No effects of either the parallel or perpendicular fields applied for periods up to 30 min were observed on the nerve conduction velocity. Gaffey and Tenforde (1983) conducted similar measurements on intact frog sciatic nerves exposed to either parallel or perpendicular 2.0 Tesla stationary fields that were homogeneous to within 0.1% over the entire length of the nerve. With both field configurations, they found no effects of a continuous 4-h exposure on either the amplitude or the conduction velocity of maximal evoked action potentials. Extending the duration of exposure to 17 h was also found to have no influence on the impulse conduction velocity.

Schwartz (1979) has used the double sucrose gap technique to measure under voltage-clamp conditions the membrane potentials and transmembrane currents in lobster circumesophageal connective nerves exposed to a 1.2 Tesla stationary field. Both parallel and perpendicular field orientations relative to the nerve axis were used in these experiments, and the field gradients were identical to those described above in the discussion of Schwartz's studies on nerve conduction velocity. No effect of the parallel or perpendicular magnetic fields was observed on either the action potentials or the transmembrane currents during nerve excitation.

In contrast to the negative results of the studies described above, two

other investigations have yielded apparent positive effects of stationary magnetic fields on nerve bioelectric activity (Reno, 1969; Edelman et al., 1979). In studies with intact frog sciatic nerves, Reno (1969) found that the application of a homogeneous 1.16 Tesla field oriented parallel to the nerve axis led to a measurable increase in the impulse conduction velocity beginning after approximately 5 min of exposure. After 20 min in the field, the nerve conduction velocity reached a level that was 30% above the pre-exposure control value. Upon removal of the field, a progressive increase in conduction velocity continued for an additional 20 min, and reached a maximum value that was 70% above the control level. The conduction velocity then began to decline towards the initial control value.

Two aspects of Reno's observations suggest that the apparent effect of an applied magnetic field may, in fact, have resulted from an elevation in the ambient temperature due to the heat dissipated from the coils of his electromagnet. First, the physical interaction of a stationary magnetic field with the conduction currents involved in impulse propagation would be expected to have an immediate effect on nerve bioelectric activity, rather than producing an effect that is manifest only after 5 min of exposure. Secondly, the physical influence of the field on conduction currents would not be expected to persist after termination of the exposure. On the other hand, the increase and subsequent decay of the magnetic field effect observed by Reno is fully consistent with the effect on nerve conduction velocity that would be expected to result from the heating and cooling trends that occur during and after the excitation of electromagnet coils to high power levels.

In another series of experiments that produced positive effects, Edelman et al. (1979) reported that the application of 0.10 - 0.71 Tesla stationary magnetic fields perpendicular to the frog sciatic nerve axis produced a gradual

increase in the amplitude of evoked action potentials. This effect appeared 15-20 min after the field was applied, and the action potential amplitude reached levels as high as 80% above the pre-exposure control level after 1 h of exposure. When the field was removed, the action potential amplitude declined at a slower rate than it had risen during the field exposure. This apparent magnetic field effect thus had a time course that was qualitatively similar to the effect on nerve conduction velocity previously described by Reno (1969). As discussed above, the delayed emergence of a magnetic field effect on nerve bioelectric activity, and a persistence of the effect after termination of the exposure, would not be expected if the influence of the field resulted from a direct physical interaction with ionic conduction currents. However, the results reported by Edelman et al. (1979), like those of Reno (1969), are fully consistent with thermal effects associated with the dissipation of heat from electromagnet coils under conditions where no provision is made for temperature regulation within the magnet gap. It should also be noted that Edelman et al. (1979) used electrical stimuli that produced submaximal action potentials with amplitudes of 7 - 10 mV. As discussed above, Gaffey and Tenforde (1983) have demonstrated that such submaximal action potentials are extremely temperature sensitive.

C. Absolute and relative refractory periods. Following the passage of a maximal action potential, an isolated peripheral nerve enters an absolute refractory period of 1-2 ms duration during which a second impulse cannot be evoked. Subsequent to the absolute refractory period, the nerve enters a relative refractory period during which action potentials of progressively increasing amplitude can be evoked by electrical stimulation. After a period of approximately 4-6 ms, the second action potential reaches the same maximal amplitude as the impulse elicited by the initial stimulus, thus denoting the

end of the relative refractory period. The characteristics of both the absolute and relative refractory periods have been examined by Gaffey and Tenforde (1983) during the exposure of frog sciatic nerves to a homogeneous 2.0 Tesla field. Using both parallel and perpendicular configurations of the magnetic field relative to the nerve axis, these workers found no influence of the field on the duration of either the absolute or the relative refractory periods. In addition, the amplitudes of impulses evoked during the relative refractory period were unaffected by the magnetic field exposure.

Central nervous system response to stationary magnetic fields

Several reports have been made of profound changes in brain electrical activity during the exposure of experimental animals to stationary fields ranging from approximately 0.1 - 9.1 Tesla. These studies, which have produced a number of conflicting observations on the central nervous system response to magnetic fields, will next be briefly summarized. An emphasis will be placed on describing the apparently dichotomous findings of excitatory and inhibitory responses by different groups of investigators.

In a series of electroencephalogram (EEG) measurements on squirrel monkeys, Beischer and Knepton (1966) observed that exposure to stationary magnetic fields produced a significant increase in the amplitude and frequency of brain electrical signals recorded with silver electrodes inserted below the scalp in the frontal, parietal, temporal, occipital and median cranial regions. Recordings of the EEG were made in homogeneous fields produced by a Bitter magnet with intensities ranging from 1.47 - 9.13 Tesla. EEG measurements were also made in the strong gradient fields at the periphery of the magnet gap. During exposures ranging from 3 - 45 min, it was found that the predominant EEG frequencies shifted from their pre-exposure range of 8 - 12 Hz to 14 - 50 Hz,

independent of the field intensity or homogeneity. The amplitude of the signals also increased from the control level of 25 - 50 μV to 50 - 400 μV. These changes were uniformly observed in the different cranial regions that were simultaneously monitored, and there was no latency in the response upon application of the field. When the field was removed, both the amplitude and frequency spectrum of the EEG signals returned to their pre-exposure levels.

In analyzing the results of these experiments, Beischer and Knepton (1966) considered several potential sources of artifacts, including ripple currents from the magnet power supply, animal movements associated with heart contractions and breathing, pick-up of stray 60-Hz fields by the EEG electrodes and leads, and skeletal muscle tremors. All of these factors except for muscle tremors could be excluded because their characteristic frequencies were outside of the frequency range observed for the predominant EEG signals in the presence of a stationary magnetic field. However, the characteristics of the EEG tracings obtained for monkeys in the magnetic field suggest that "myographic noise" from the movement of skeletal muscles may have been superimposed on the brain electrical signals. It is also possible that environmental factors present only during excitation of the magnet coils, including mechanical vibrations, audible noise and an increased ambient temperature, could have led to an altered pattern of brain electrical activity. The possible existence of such extraneous variables brings into question the validity of the finding of an apparent increase in the excitatory state of the central nervous system during exposure to large stationary magnetic fields.

In direct contrast to the above findings with monkeys, it has been reported that the exposure of rabbits to relatively weak, 0.08 - 0.10 Tesla stationary fields produces an EEG signature that is characteristic of a general inhibitory state in the central nervous system (Kholodov, 1964, 1966; Kholodov

et al., 1969). The major changes observed in the EEG during magnetic field exposure were slow waves and high-amplitude spindles that appeared in the electrical activity recorded from various regions of the brain. These phenomena were not uniformly exhibited in all of the experimental tests. In a series of 100 field exposures on 12 rabbits, Kholodov (1966) observed the occurrence of spindles in 30% of the tests, and an increase in the number of slow waves with frequencies less than 4 Hz in 19% of the tests. Both phenomena occurred with a latency of approximately 15 s after the field was turned on, and reached maximum levels after 45 s of exposure. The increased number of spindles and slow waves persisted during exposures to a 0.1 Tesla field for 3 min, and decreased immediately after the field was turned off. However, 15 - 25 s after the exposure was terminated, a transient increase in the number of spindles and slow waves occurred with a duration of approximately 20 - 30 s.

Kholodov (1966) has presented evidence that the EEG alterations observed in his experiments with rabbits were not artifacts resulting from the induced e.m.f.'s that occur during the switching on and off of an electromagnet. This possibility was excluded on the basis of trials in which the magnet was energized and de-energized at varying rates, with no resulting change in the character of the observed EEG alterations. However, Kholodov has not discussed the possibility that other extraneous factors such as low-frequency mechanical vibrations and acoustic noise within the magnet gap, irregularities in the breathing rate or the rate of cardiac contraction, sporadic muscle tremors or episodic shivering of the experimental subject could have led to spurious signals in the EEG record. Another factor that must be considered in evaluating these studies is the lack of data on the field strength dependence of the reported effects. Such information might lend insight into the existence of variables other than the magnetic field that could have produced

abnormal features in the EEG recordings.

Visuosensory response to magnetic fields

One of the most extensively studied magnetic effects in living systems is the induction of magnetophosphenes, in which a flickering illumination within the visual field occurs in response to stimulation by pulsed or oscillating magnetic fields with frequencies less than 100 Hz (d'Arsonval, 1896; Thompson, 1909-1910; Dunlap, 1911; Magnusson and Stevens, 1911-1912; Barlow et al., 1947; Lövsund et al., 1980). The locus of the magnetic field interaction that leads to phosphenes has been shown to be the retina on the basis of several lines of evidence: (1) magnetophosphenes are produced by time-varying fields applied in the region of the eye, and not by fields directed towards the visual cortex in the occipital region of the brain (Barlow et al., 1947); (2) pressure on the eyeball abolishes sensitivity to magnetically-induced phosphenes (Barlow et al., 1947); (3) the threshold magnetic field intensity required to elicit phosphenes in human subjects with defects in color vision was found to have a different dependence on the field frequency than that observed for subjects with normal color vision (Lövsund et al., 1980); (4) in a patient in whom both eyes had been removed as the result of severe glaucoma, phosphenes could not be induced by time-varying magnetic fields, thereby precluding the possibility that magnetophosphenes can be initiated directly in the visual pathways of the brain (Lövsund et al., 1980).

Although the available evidence strongly implicates the retina as the site of magnetic field action leading to phosphenes, it is not as yet clear whether the photoreceptors or the neuronal elements of the retina are the sensitive substrates which respond to the field. In a series of experiments on in vitro frog retinal preparations, Lövsund et al. (1981) have made extracellular

electrical recordings from the ganglion cell layer of the retina immediately following termination of exposure to a 20-Hz, 60-mT field in the presence or absence of broad-spectrum background light. It was found that the average latency time for response of the ganglion cells to a photic stimulus was increased from 87 to 92 ms ($p < 0.05$) in the presence of the magnetic field. In addition, the ganglion cells that exhibited electrical activity during photic stimulation ("on" cells) ceased their activity during magnetic field stimulation (i.e., they became "off" cells). The converse behavior of ganglion cells was also observed. These observations indicate that stimulation of the retina by light and by a time-varying magnetic field elicits responses in similar post-synaptic neural pathways.

An important electrophysiological finding by Lövsund et al. (1981) was the observation that the electrical response of frog retinal ganglion cells to both photic stimuli and time-varying magnetic fields was blocked when either sodium aspartate or cobalt chloride was added to the Ringer's solution in the eyecup preparation. These compounds inhibit the transfer of information from the photoreceptors to the neuronal elements of the retina. The electrophysiological observations on chemically-blocked retinal preparations appear to implicate the photoreceptors per se as the locus of magnetic field stimulation. The origin of magnetic field responses within the receptors is consistent with the hypothesis of Knighton (1975) that a transretinal electric current may act to polarize the photoreceptor synaptic membrane, and thereby alter the post-synaptic transmission of electrical information. One experimental observation made by Lövsund et al. (1981) which appears to be inconsistent with this hypothesis was the ability of an applied magnetic field to induce phosphenes in a patient with Retinitis pigmentosa, in whom the photoreceptors and pigment epithelium were defective but the bipolar and ganglion cell layers

of the retina were conserved. The disparity in these observations, however, may be attributable to a smaller number of functional photoreceptors within the otherwise degenerated retina of the Retinitis patient. In this context, it is of interest to note that Kato et al. (1983) found that electrophosphenes could be generated in patients with pigmentary retinal dystrophy, but a substantially larger stimulus intensity was required over the entire frequency range of 7 - 80 Hz than with subjects that had normal vision. Lövsund et al. (1981) have also speculated that sensitivity to time-varying magnetic fields may exist within both the photoreceptor and the neuronal elements of the retina, but that the former are stimulated with greater ease.

Although the psychophysical phenomenon of phosphenes has not been reported by human observers during exposure to stationary magnetic fields, there are two potential interaction mechanisms between these fields and elements of the retina that are involved in the visual response to photic stimulation. First, the photoreceptor outer segments are subject to orientation in a stationary magnetic field as the result of their large diamagnetic anisotropy (Hong, 1980). Second, the initial photoisomerization event elicited by photon absorption in the retinal photopigments is followed by a series of ionic fluxes that lead to excitation of the retinal neurons, and ultimately the visual cortex via a complex neural pathway. This component of the phototransduction process could be influenced by stationary magnetic fields as the result of ionic current distortion and/or inductive effects, as discussed in an earlier section of this chapter.

In an effort to elucidate whether stationary magnetic fields perturb the photically elicited electrical activity of the retina, Raybourn (1983) has recorded the external electroretinogram (ERG) of isolated turtle retinas during photic stimulation in the presence of magnetic fields with graded intensities.

When the in vitro retinal preparations from light-adapted or dark-adapted eyes were studied, no changes in the ERG occurred in fields up to 1.0 Tesla. However, the amplitude of the ERG b-wave, which results from electrical activity of postsynaptic retinal cells in the outer plexiform layer, was consistently suppressed in retinas prepared during the light-to-dark transition phase of the diurnal 12-h light/12-h dark cycle. During this transition phase, which extends for approximately 2 h after the onset of darkness, the photoreceptor cells undergo rapid changes in both their physiological and metabolic activities. The magnetic field effect during the transition phase was observed with intensities as low as 2-3 mT, and was rapidly reversible following termination of the field exposure. This effect was observed in both the cone-dominant retinas of Pseudemys scripta turtles, and the mixed rod-cone retinas of Chelydra serpentina turtles, thus suggesting that it is independent of the photoreceptor cell type. The mechanism underlying the magnetic field sensitivity of turtle retinas during one brief phase of the light/dark diurnal cycle has not been determined. The magnetic field strengths that produce a b-wave response compression are well below the levels that could exert orientational effects on photoreceptor disk membranes, and this fact suggests that charge translocation mechanisms in the retina may be involved in the magnetic field interaction.

In a recent series of electrophysiological experiments using mammalian subjects, Gaffey and Tenforde (1984) have recorded the ERG in cats (Felis domestica) and monkeys (Macaca) exposed to stationary magnetic fields with strengths up to 1.5 Tesla. Oscillographic ERG recordings were made of the negative a-wave (receptor field potential) and the larger positive b-wave (postsynaptic potential) following light flashes with relative intensities of 1, 4, 16, 48 and 80, where the highest intensity flash was approximately 240

lumen-s/m^2. Acute exposures to magnetic fields up to 1.5 Tesla had no effect on either the a-wave or b-wave amplitude in the ERG recordings from both cats and monkeys. Ophthalmic examinations also revealed no alterations associated with magnetic field exposure. These in vivo findings with experimental animal subjects are therefore consistent with the observation that stationary magnetic fields exert no measurable influence on the visual processes of humans.

Summary and conclusions

The majority of the experimental studies that have been conducted to date indicate that stationary magnetic fields with intensities up to 2 Tesla have little or no influence on the bioelectric properties of isolated neurons. This finding conforms quite well to the theoretical predictions discussed at the beginning of this chapter. The few instances of reported effects of magnetic fields on the electrical activity of peripheral nerves may have resulted from an inadequate control of temperature within the magnet gap.

Studies on the response of the central nervous system to stationary magnetic fields with a wide range of intensities have produced conflicting results that are currently difficult to interpret. Both excitatory and inhibitory responses of the central nervous system to magnetic fields have been observed by different investigators, and the potential sources of artifacts in these experiments cannot be adequately analyzed from the information that is available. As discussed in an earlier section of this chapter, Valentinuzzi (1965) has pointed out that a summation of magnetic field inductive effects could occur in complex neural networks. This additive field effect might conceivably lead to alterations in the bioelectric activity of the brain that would not be seen in studies with individual neurons. However, the information that is available at the present time is clearly inadequate for making an

unequivocal judgment as to the existence of magnetic field effects on the central nervous system.

From a number of studies on the visuosensory system, current evidence favors the view that the production of magnetophosphenes occurs via the interaction of time-varying magnetic fields with retinal photoreceptors, rather than with the neuronal elements of the retina or the complex neural pathway leading to the visual cortex. In several species of mammals, including humans, stationary magnetic fields have been shown to have no detectable influence on visual phototransduction or the processing of visual information within the central nervous system. The recently observed circadian dependence of visual sensitivity to stationary magnetic fields in isolated turtle retinas remains as an area that requires further study to elucidate the mechanism underlying this phenomenon, and to assess its potential relevance to magnetic field interactions with *in vivo* mammalian retinas.

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