Radiat Environ Biophys (1983) 22: 61–73

Radiation and Environmental Biophysics

Bioelectric Properties of Frog Sciatic Nerves During Exposure to Stationary Magnetic Fields

C. T. Gaffey and T. S. Tenforde

Biology and Medicine Division, Lawrence Berkeley Laboratory, University of California, Berkeley, CA 94720, USA

Summary. The bioelectric properties of frog sciatic nerves have been measured during exposure to homogeneous, stationary magnetic fields. The action potential amplitude, conduction velocity, absolute refractory period and relative refractory period were found to be unaffected by a continuous 4-h exposure to perpendicular or parallel 2.0 T (1 T = 10^4 G) magnetic fields. These parameters also remained unchanged during a 1-h post-exposure period. The conduction velocity was similarly found to be unchanged when the field was applied continuously for 17 h. Exposure of sciatic nerves to a 1.0-T field led to no alteration in the threshold for neural excitation. The absence of magnetic field effects on nerve electrical activity observed in the present experiments contrasts with the positive findings reported previously by other investigators. These discrepancies may be attributable to an inadequate control of ambient temperature in the earlier studies.

1. Introduction

A large number of reports on the effects of stationary magnetic fields on the bioelectric properties of isolated nerve preparations have provided conflicting information. In an early study, McKendrick (1879) observed that contraction of the gastrocnemius muscle resulted from exposure of the frog sciatic nerve to magnetic fields. Drinker and Thomson (1921) and Erdman (1955) later reported that there were no effects of magnetic fields on the impulse conduction properties of nerve. Erdman (1955), however, did observe an alteration in the rheobase and chronaxie of nerves subsequent to magnetic field exposure, but Liberman et al. (1959) subsequently obtained evidence that there was no change in the excitation threshold of nerves exposed to magnetic fields.

A similarly conflicting body of experimental data has emerged from studies on the influence of magnetic field orientation relative to the nerve axis. Because of the vectorial nature of the Lorentz force exerted on ionic conduction currents, several investigators have examined the influence of magnetic fields applied

either parallel or perpendicular to the nerve axis. Reno (1969) reported a 30% increase in the impulse conduction velocity of frog sciatic nerves in a 1.16-T parallel magnetic field, but observed no effect when a field of the same strength was applied in a perpendicular orientation. Schwartz (1978) later reported that the conduction velocity of isolated lobster circumesophageal nerve was unaffected by a 1.2-T field applied in either a parallel or perpendicular orientation. Edelman et al. (1979) observed no effect of parallel magnetic fields on the amplitude of frog sciatic nerve action potentials. However, when the nerve was placed in a perpendicular configuration in fields ranging from 0.2–0.6 T, a time-dependent increase in action potential amplitude was observed. This effect appeared 15–20 min after the field was applied, and by 60 min the amplitude reached a value 80% above the control level.

The current study with isolated frog sciatic nerves was designed to resolve many of the questions raised by the divergent results of earlier studies. A field strength of 2.0 T was used in both parallel and perpendicular configurations relative to the nerve axis, and this field level exceeds that used in any previous studies. Similarly, the magnetic field exposure interval of 4–17 h exceeds that used in earlier experiments. Finally, measurements of the absolute and relative refractory periods for frog sciatic nerves in high magnetic fields are reported here for the first time.

2. Materials and Methods

2.1. Nerve Preparations

Adult frogs (*Rana pipiens*) were obtained from Western Scientific Co. (Sacramento, California). The frogs were decapitated, their spinal cords pithed, and the sciatic nerves were than carefully dissected from both legs. Each cut nerve ending was tied with surgical thread to prevent a loss of axoplasm. The nerves were then bathed for at least 1 h in frog Ringer solution at room temperature to achieve ionic equilibrium.

2.2. Electrophysiological Procedures

A. Electrode Chamber. An isolated nerve was placed across stimulating and recording electrodes within an airtight chamber. The chamber was constructed from a 25 mm × 90 mm × 65 mm block of acrylic plastic with a central 12 mm × 12 mm × 45 mm groove. Five Ag-AgCl electrodes (0.69 mm diameter) extended at right angles across the longitudinal groove. The anode-cathode separation in the stimulating electrode pair was 3 mm, and the distances from the cathode to the three recording electrodes were 10, 20, and 30 mm, respectively. The chamber contained a reservoir of Ringer solution on the floor of the groove to maintain a high relative humidity, and the moist chamber was sealed with a 3-mil Mylar sheet coated with vaseline. The chamber was maintained at an ambient temperature of approximately 21° C.

B. Evoked Action Potentials. A multifunction stimulator (model S-8 with isolation units, Grass Instrument Co., Quincy, Massachusetts) sent rectangular pulses 0.01 ms in duration through the stimulating electrode pair in the nerve chamber. The anode of the stimulating electrode pair was placed adjacent to the end of the sciatic nerve which had been cut at the point where it emerged from the spinal cord. The evoked potentials then travelled in the natural, efferent direction along the nerve. The stimulating pulses, which were delivered at a 1-Hz frequency, were synchronized with the sweep of an oscilloscope (model RM 5621 storage oscilloscope with 3B4 time base and 3A3 dual trace differential amplifier; Tektronix, Portland, Oregon). Each rectangular stimulating pulse appeared as a "stimulus artifact" that defined the exact time at which the pulse was delivered. The voltage output of the stimulator was progressively increased to generate action potentials detected at the recording electrodes. The recording electrodes were connected to AC preamplifiers (model 511, Grass Instrument Co., Quincy, Massachusetts) which delivered the neural signals to the storage oscilloscope. Action potentials were recorded with a polaroid oscilloscope camera (model C-12, Tektronix, Portland, Oregon).

C. Maximal Action Potentials. As the stimulus strength was progressively increased, action potentials appeared at the recording electrodes which consisted of a wave of surface negativity with a duration of approximately 1–2 ms. To evoke maximal action potentials (MAP), the stimulus strength was increased to a level beyond which a further increase had no effect on the amplitude as measured from the zero baseline to the summit of the action potential. MAP amplitudes varied from 15–25 mV depending upon the diameter of the nerve trunk.

D. Conduction Velocity. The impulse conduction velocity was determined from the time required for a MAP summit to move from the first to the second recording electrodes, which were separated by 10 mm. The third recording electrode, which was adjacent to the distal nerve ending, served as a reference electrode.

E. Refractory Period. To determine the absolute and relative refractory periods, a pair of 0.1 ms rectangular pulses of independently variable strength was delivered to the nerve preparation. The time interval between these pulses, which are referred to as the conditioning and test stimuli, could be varied in a precise manner. A MAP was first evoked by the conditioning stimulus, and after a controlled time interval, a second test stimulus of the same intensity was delivered. If the interval between stimuli exceeded approximately 7 ms, two MAPs with identical amplitudes were generated. The amplitude of the second evoked potential decreased when the interval between the conditioning and test stimuli fell in the relative refractory period (RRP). If the test stimulus was delivered at very short intervals, i.e., within the absolute refractory period (ARP), then the second action potential was not evoked regardless of the strength of the test stimulus. In the measurements of ARP and RRP reported here, both the conditioning and test stimuli were adjusted to be twice the stimulus strength required to evoke a MAP.

F. Excitation Threshold. The sciatic nerve action potential represents a summation of a large number of action potentials from individual nerve fibers with diameters ranging from 7–22 μm and differing threshold for electrical excitation. When all of the fibers are excited, a MAP is achieved. However, when the stimulus strength is insufficient to excite all of the fibers in the sciatic nerve, then a submaximal action potential (SMAP) is observed. When the stimulus evoking a SMAP is repetitively applied at a constant strength, the stability of the SMAP amplitude as a function of time provides a sensitive index for detecting changes in the threshold for neural excitation. The constancy of SMAP amplitudes was therefore used in our experiments to assess the potential influence of a stationary magnetic field on the excitation threshold. The applied stimulus used for these studies was adjusted to give a SMAP amplitude that was 50% of the MAP amplitude.

Because the threshold for nerve excitation is extremely temperature dependant, a thermoregulating system was devised to provide control of the ambient temperature to within ± 0.1° C. The nerve chamber was placed within a 45 cm × 45 cm × 16.5 cm lucite box that was continuously flushed with a 5 l/min flow of temperature-regulated air (approximately 21° C) from a Wedco Life Science Environmental Cabinet (Wedco Incorporated, Silver Springs, Maryland). The temperature of the nerve chamber was monitored by means of three copper-constantin thermocouples connected to recording units. The accuracy of the thermocouples was unaffected by the presence of magnetic fields up to 2 T.

2.3. Magnetic Field Exposures

Two DC iron-core electromagnets were used in this study. One magnet produced a 2.15-T (maximum) stationary horizontal field between flat, 18.42-cm diameter pole faces separated by a 7.16-cm gap. A description of the field characteristics of this magnet has been presented previously (Gaffey and Tenforde 1981). The sealed chamber containing a sciatic nerve preparation was placed in the geometrical center of the magnet gap, and mounted with the nerve in either a parallel or perpendicular orientation relative to the lines of magnetic induction. In either configuration, the magnetic field strength was homogeneous to within 0.1% over the entire length of the nerve. Measurements of the action potential amplitude, conduction velocity and refractory period were made in this electromagnet at an ambient temperature of approximately 21° C.

A second, large-volume DC electromagnet was used to study the influence of stationary magnetic fields on the excitation threshold of nerve. The magnet produced a 1.60-T (maximum) vertical field between flat, 73.7 cm × 81.3-cm pole faces separated by a 19.4-cm gap. The field characteristics of this magnet have been described previously (Tenforde 1979; Tenforde et al. 1983). During measurements of the nerve excitation threshold, the thermoregulated lucite enclosure which held the sealed nerve chamber was placed in the geometrical center of the magnet gap. The nerve preparation was oriented perpendicular to the lines of magnetic induction, and the field was uniform to within 0.1% over the entire length of the nerve.

3. Results

3.1. Maximum Action Potential (MAP) Amplitude

An example of MAP amplitude measurements before, during and after a 4-h exposure of a frog sciatic nerve to a 2.0-T field is shown in Fig. 1. The nerve axis was oriented parallel to the lines of magnetic induction, and no significant variation in the 20-mV MAP amplitude resulted from application of the field. A similar experimental procedure was followed with a total of 16 nerve preparations, eight of which were exposed to the field with the nerve axis oriented parallel to the lines of magnetic induction, and the remainder with the axis perpendicular to the lines of induction. As shown by the data summary in Table 1, a 4-h exposure to a 2.0-T field in either the parallel or perpendicular configuration had no statistically significant effect on the MAP amplitude relative to the 1-h pre-exposure control interval. Similarly, no change in MAP amplitude occurred during the 1-h post-exposure interval.

3.2. MAP Conduction Velocity

Measurements of the velocity of MAP impulse transmission are shown in Fig. 2 for a sciatic nerve oriented with the long axis parallel to a 2.0-T field. The conduction velocity remained at a stable level of 23–24 m/s during a 0.5-h exposure to the field and a 0.5-h post-exposure interval. Similar measurements

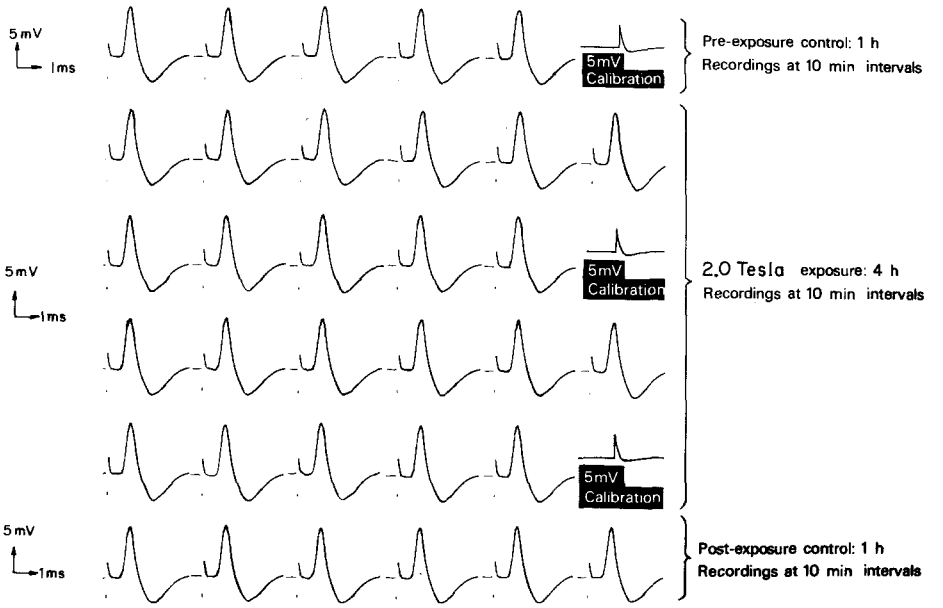

Fig. 1. Evoked action potentials in a frog sciatic nerve are shown as a function of time before, during and after exposure to a 2.0-T field

Table 1. Frog sciatic nerve maximal action potential (MAP) amplitudes and conduction velocities before, during and after exposure to a 2.0-T field in either a parallel or perpendicular orientation

Orientation relative to field	No. nerve preparations	Experimental condition	Duration (h)	MAP amplitude ± 1 SD (mV)	MAP conduction velocity ± 1 SD[a] (m/s)
Parallel	8	Pre-exposure	1	20.8 ± 2.8	
		B = 2.0 T	4	21.3 ± 2.8	
		Post-exposure	1	20.9 ± 2.6	
Perpendicular	8	Pre-exposure	1	20.9 ± 2.6	
		B = 2.0 T	4	21.3 ± 2.8	
		Post-exposure	1	20.8 ± 2.6	
Parallel	8[b]	Pre-exposure	1		26.0 ± 2.8
		B = 2.0 T	4		26.2 ± 2.9
		Post-exposure	1		26.2 ± 2.8
Perpendicular	8[b]	Pre-exposure	1		26.7 ± 2.4
		B = 2.0 T	4		26.8 ± 2.6
		Post-exposure	1		26.7 ± 2.6

[a] Based on a one-way analysis of variance, the differences between the exposure and control intervals were not statistically significant at the level $p = 0.05$

[b] Four of the eight nerves used for conduction velocity measurements were also used for measurements of action potential amplitudes

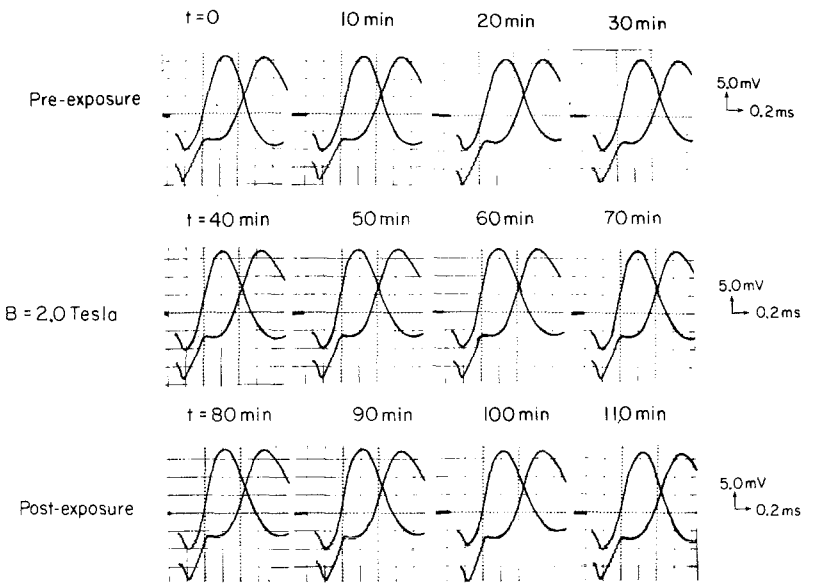

Fig. 2. The conduction of an evoked action potential along a frog sciatic nerve is shown before, during and after exposure to a 2.0-T field. Each action potential was recorded at two points separated by a distance of 1.0 cm along the nerve axis

Table 2. Maximal action potential conduction velocities of frog sciatic nerves exposed to a 2.0-T field for 17 h in either a parallel or perpendicular orientation

Orientation relative to field	No. nerve preparations	Experimental condition	Duration[a] (h)	MAP conduction velocity ± 1 SD[b] (m/s)
Parallel	7	Non-exposed	0	29.7 ± 1.9
			17	28.2 ± 2.0
	7	B = 2.0 T	0	28.2 ± 1.5
			17	27.3 ± 1.7
Perpendicular	7	Non-exposed	0	29.8 ± 1.7
			17	28.7 ± 1.9
	7	B = 2.0 T	0	29.1 ± 1.6
			17	28.3 ± 3.3

[a] The durations marked as 0 indicate that measurements of conduction velocity were made immediately before the experimental condition was established

[b] Based on a one-way analysis of variance, the differences between the exposed and non-exposed nerve preparations were not statistically significant at the level $p = 0.05$

of conduction velocity were made for a total of 16 nerve preparations, half of which were oriented parallel to the lines of magnetic induction and the remainder in a perpendicular configuration. Velocity measurements were made at 10-min intervals during a 1-h pre-exposure control period, a 4-h exposure to a 2.0-T field, and a 1-h post-exposure period. As shown by the data summary in Table 1, no statistically significant alteration in conduction velocity occurred during the field exposure or the post-exposure interval.

The influence on nerve impulse conduction velocity of a 17-h continuous exposure to a 2.0-T field was also evaluated for the sciatic nerves from a total of 14 frogs. In these experiments, one sciatic nerve from each frog was exposed to the field while bathed in Ringer's solution within a petri dish, and the other sciatic nerve from the same frog was maintained under similar conditions in a control environment approximately 30 m away from the magnet gap. Measurements of MAP conduction velocities were made at the beginning and end of the 17-h exposure interval. As shown by the data summary in Table 2, a 17-h exposure to a 2.0-T field with the nerve axis oriented either parallel or perpendicular to the lines of magnetic induction had no influence on the impulse conduction velocities exhibited by exposed sciatic nerves relative to their pair-matched controls.

3.3. Absolute and Relative Refractory Periods

When a conditioning stimulus is delivered to a nerve, a conditioning action potential (CAP) travels along the nerve length and leaves behind it a trail of refractoriness to a second test stimulus. If the test stimulus is applied within approximately 1.5 ms following the onset of the CAP stimulus, the nerve is in an absolute refractory state and no test action potential (TAP) is observed. When

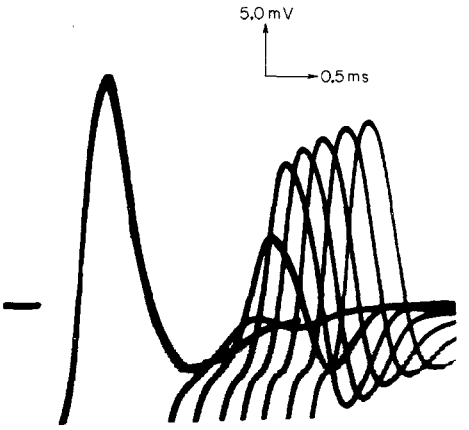

Fig. 3. A series of evoked action potentials in a frog sciatic nerve is shown during the relative refractory period. The horizontal bar at the left defines the zero-potential baseline

Table 3. Frog sciatic nerve absolute refractory periods before, during and after exposure to a 2.0-T field in either a parallel or perpendicular orientation

Orientation relative to field	No. nerve preparations	Experimental condition	Duration (h)	Absolute refractory period ± 1 SD[a] (ms)
Parallel	8	Pre-exposure	0.5	1.40 ± 0.02
		B = 2.0 T	0.5	1.41 ± 0.02
		Post-exposure	0.5	1.41 ± 0.03
Perpendicular	8	Pre-exposure	0.5	1.42 ± 0.02
		B = 2.0 T	0.5	1.43 ± 0.03
		Post-exposure	0.5	1.43 ± 0.03

[a] Based on a one-way analysis of variance, the differences between the exposure and control intervals were not statistically significant at the level $p = 0.05$

the interval between the conditioning and test stimuli is lengthened beyond the absolute refractory period (ARP), the nerve enters a relative refractory period (RRP) during which the TAP can be observed. As the interstimulus interval is further increased, the TAP amplitude becomes progressively larger and reaches the same magnitude as the CAP amplitude at the end of the RRP. These concepts are illustrated in Fig. 3 by a series of frog sciatic nerve action potentials during the RRP.

Measurements of the ARP for a total of 16 sciatic nerve preparations are summarized in Table 3. These data demonstrate that no statistically significant change in the ARP occurs during exposure to a 2.0-T field for periods of either 0.5 or 4.0 h. Eight of the nerve preparations were exposed with the nerve axis oriented parallel to the lines of magnetic induction, and eight with the axis perpendicular to the lines of induction. In all cases, no statistically significant change in the TAP amplitude relative to the CAP amplitude occurred in response to the field exposure, or during a 0.5-h post-exposure interval. These observations indicate that the nerve impulse characteristics during the RRP are unaffected by exposure to a 2.0-T field for periods up to 4 h.

Table 4. Test action potential (TAP) amplitudes during the relative refractory period following a maximal conditioning action potential (CAP) for frog sciatic nerves before, during and after exposure to a 2.0-T field in either a parallel or perpendicular orientation

Orientation relative to field	No. nerve preparations	Experimental condition[a]	Duration (h)	CAP amplitude ± 1 SD (mV)	TAP amplitude relative to CAP ± 1 SD[b, c]			
					1.5 ms	2.0 ms	3.0 ms	4.0 ms
Parallel	4	Pre-exposure	0.5	21.5 ± 2.9	0.176 ± 0.055	0.512 ± 0.042	0.819 ± 0.034	0.953 ± 0.012
		B = 2.0 T	0.5		0.182 ± 0.051	0.514 ± 0.042	0.823 ± 0.023	0.956 ± 0.014
		Post-exposure	0.5		0.180 ± 0.045	0.508 ± 0.029	0.824 ± 0.020	0.954 ± 0.011
	4	Pre-exposure	0.5	22.1 ± 2.0	0.182 ± 0.084	0.508 ± 0.060	0.879 ± 0.094	0.980 ± 0.025
		B = 2.0 T	4.0		0.166 ± 0.073	0.489 ± 0.072	0.797 ± 0.050	0.936 ± 0.045
		Post-exposure	0.5		0.168 ± 0.064	0.497 ± 0.071	0.808 ± 0.050	0.941 ± 0.036
Perpendicular	4	Pre-exposure	0.5	19.2 ± 2.9	0.121 ± 0.080	0.452 ± 0.123	0.785 ± 0.064	0.937 ± 0.023
		B = 2.0 T	0.5		0.114 ± 0.064	0.466 ± 0.086	0.774 ± 0.050	0.926 ± 0.019
		Post-exposure	0.5		0.110 ± 0.058	0.462 ± 0.086	0.766 ± 0.047	0.924 ± 0.016
	4	Pre-exposure	0.5	19.8 ± 1.6	0.189 ± 0.050	0.550 ± 0.046	0.848 ± 0.054	0.968 ± 0.029
		B = 2.0 T	4.0		0.173 ± 0.043	0.522 ± 0.027	0.836 ± 0.045	0.953 ± 0.050
		Post-exposure	0.5		0.161 ± 0.038	0.508 ± 0.022	0.815 ± 0.048	0.947 ± 0.049

[a] Measurements made during a 4-h application of the field were carried out during the 0.5-h interval immediately preceding termination of the exposure

[b] The indicated times represent the interval between the application of the conditioning and the test stimuli

[c] Based on a one-way analysis of variance, the differences between the exposure and control intervals were not statistically significant at the level $p = 0.05$

3.4. Neural Excitation Threshold

Initial studies with a total of 29 sciatic nerve preparations indicated that the threshold for excitation is extremely temperature dependent, with the submaximal action potential (SMAP) exhibiting an average increase of 2.7 ± 1.4 (SD) mV for a 1.0° C rise in the ambient temperature. An increase of this magnitude represents approximately a 25% change in the SMAP amplitude under our experimental conditions. The thermoregulatory system described in the methods section was therefore devised to counteract a temperature rise introduced by heat from the magnet coils. SMAP amplitudes were recorded for five consecutive min during which the ambient temperature in the nerve chamber remained within ± 0.1° C of the initial value (approximately 21° C). The magnet was then energized to produce a 1.0-T field, and the recording of SMAP amplitudes was continued until the ambient temperature rose by 0.1° C. A total of 22 sciatic nerves were studied by this procedure, with the long axis of each nerve oriented perpendicular to the lines of magnetic induction. Experiments were not conducted with nerves oriented parallel to the lines of induction because this configuration could not be achieved in the magnet gap when the nerve preparation was enclosed within a large thermoregulated chamber. The average MAP amplitude of the 22 nerves was 21.1 ± 3.6 (SD) mV, and the average SMAP amplitude 0.53 ± 0.12 (SD) of the MAP amplitude, i.e., 11.2 ± 2.5 mV. The average duration of the 1.0-T magnetic field exposure, during which the temperature in the nerve chamber varied by less than 0.1° C, was 8.7 min with a range of 4–12 min. During the application of the 1.0-T field, the SMAP amplitudes of all 22 sciatic nerves remained within 0.1 mV of their values during the 5-min pre-exposure control period. Acute exposure to a field of this magnitude therefore had no effect on the threshold for neural excitation.

Attempts were also made to study the effects of exposure to field levels greater than 1.0 T on the nerve excitation threshold. These efforts were unsuccessful, however, because of our inability to maintain the temperature of the nerve chamber constant to within 0.1° C for several min when the magnet coil current was increased to levels that produced fields significantly above 1.0 T.

4. Discussion

From theoretical considerations, neural bioelectric activity could be influenced by stationary magnetic fields as the result of ionic current distortion and/or inductive effects. Liboff (1980) calculated the magnitude of the Hall effect on ionic charge carriers, and concluded that a field of 10^5 T would be required to produce distortions in the current pattern associated with nerve action potentials. Wikswo and Barach (1980) considered the magnitude of the Lorentz force on ions moving through a nerve membrane in a magnetic field, and concluded that fields exceeding 24 T would be required to significantly perturb nerve impulse conduction.

A second class of magnetic phenomena considered by Liboff (1980) was neural current alteration due to an inductive interaction. He concluded that cylindrical symmetry of the action potential current pattern under normal physiological conditions would lead to a cancellation of magnetic field inductive effects. If, however, the ion flow mechanisms associated with the generation of an action potential are highly asymmetric, then a properly oriented magnetic field in excess of 0.26 T could theoretically disturb the action potential current flow.

In the present series of experiments, a continuous 4-h exposure of isolated frog sciatic nerves to a homogeneous 2.0 T magnetic field was found to have no effect on the action potential amplitude, conduction velocity or refractory period. The absence of any biomagnetic effect was observed when the nerve was oriented with its axis either parallel or perpendicular to the lines of magnetic induction. These results are consistent with previous observations by Erdman (1955), who exposed frog sciatic nerves to a transverse 1.7-T field for 10 min without adverse effects on the impulse conduction velocity. Similarly, Schwartz (1978, 1979) found no effect of a 30-min exposure to a 1.2-T field on the conduction velocity, membrane potential or transmembrane currents in the giant axon of the lobster circumesophageal connective. The absence of biomagnetic effects on the lobster axon were demonstrated when the nerve was oriented in both parallel or perpendicular configurations relative to the field.

The experimental findings reported here have also demonstrated that exposure of the frog sciatic nerve to a 1.0-T transverse field for several min produces no alteration in the threshold for neural excitation. This finding is consistent with the observations of Liberman et al. (1959), who exposed both intact frog sciatic nerves and single myelinated nerve fibers to a transverse 1.0-T field and found no effect on the excitation threshold.

Previous reports of significant magnetic field effects on the amplitude and conduction velocity of frog sciatic nerves have been presented by Reno (1969) and Edelman et al. (1979). Reno observed that when the sciatic nerve axis was oriented parallel to a 1.16-T field, the impulse conduction velocity exhibited a measurable change after 5 min of exposure, and rose 30% above the control value by 10 min. When the field was removed, the conduction velocity continued to increase for approximately 15 min, and then declined towards the pre-exposure value. Reno (1969) suggested that the increase in conduction velocity may have resulted from a change in temperature within the recording chamber as the result of heat dissipated from the magnet coils.

In the experiments of Edelman et al. (1979), a 0.10–0.71-T field perpendicular to the sciatic nerve axis was observed to produce a gradual increase in the action potential amplitude, which reached levels as high as 80% above the control value after approximately 1 h of exposure. When the field was removed, the action potential amplitude declined at a slower rate than it had risen during application of the field. These experiments utilized electrical stimuli that produced submaximal action potential amplitudes of 7–10 mV, and SMAP were found in our studies to be extremely temperature sensitive. Unfortunately, Edelman et al. (1979) have not provided details of their magnetic field exposure

conditions or data pertaining to temperature changes during application of the field. However, the time course of the changes in SMAP amplitude observed by these investigators during and after magnetic field exposure follows the thermal transients that would be expected to occur in an electromagnet gap if no provision was made for temperature regulation. The divergence of the experimental results of Edelman et al. (1979) from those reported here may therefore be explained by a lack of adequate thermoregulation in the former studies.

In summary, the following conclusions can be drawn regarding the interaction of stationary magnetic fields with peripheral nervous tissue: (*1*) The highest field levels achieved by conventional iron-core electromagnets have no effect on the bioelectric properties of isolated nerve preparations. (*2*) The experimental observation that fields up to 2.0 T do not significantly perturb nerve electrical properties is consonant with theoretical predictions, which indicate that under normal physiological conditions, fields in excess of 20 T would be required to alter the ionic current patterns associated with nerve impulses. (*3*) Previous reports of alterations in the action potential amplitude and conduction velocity during exposure to fields on the order of 1.0 T may be attributable to thermal effects on nerve bioelectric behavior, rather than to electrodynamic interactions between the field and the ionic currents involved in the generation of nerve impulses.

Acknowledgements. The exellent secretarial assistance of L. Hawkins is gratefully acknowledged. Research support was received from the Office of Energy Research, Health and Environmental Research Division, of the U.S. Department of Energy under Contract No. DE-AC03-76SF00098 with the Lawrence Berkeley Laboratory.

References

Drinker CK, Thomson RM (1921) Does the magnetic field constitute an industrial hazard? J Indust Hyg 3:117–129

Edelman A, Teulon J, Puchalska IB (1979) Influence of magnetic fields on frog sciatic nerves. Biochem Biophys Res Commun 91:118–122

Erdman GM (1955) The problem of the effects of a constant magnetic field on nerve. Akad Nauk SSSR Inst Biol Fiziki Trudy 1:35–39

Gaffey CT, Tenforde TS (1981) Alterations in the rat electrocardiogram induced by stationary magnetic fields. Bioelectromagnetics 2:357–370

Liberman EA, Vaintsvaig MN, Tsofina LM (1959) The effect of a constant magnetic field on the excitation threshold of isolated frog nerve. Biofizika 4:505–506

Liboff RL (1980) Neuromagnetic thresholds. J Theor Biol 83:427–436

McKendrick M (1879) Observations on the influence of an electromagnet on some of the phenomena of a nerve. J Anat 13:219–223

Reno VR (1969) Conduction velocity in nerve exposed to a high magnetic field. NASA report NAMI-1089, Naval Aerospace Medical Institute, Pensacola, FL

Schwartz J-L (1978) Influence of a constant magnetic field on nervous tissues: I. Nerve conduction velocity studies. IEEE Trans Biomed Eng 25:467–473

Schwartz J-L (1979) Influence of a constant magnetic field on nervous tissues: II. Voltage-clamp studies. IEEE Trans Biomed Eng 26:238–243

Tenforde TS (1979) Dosimetry, instrumentation and exposure chambers for DC magnetic field studies. Lawrence Berkeley Laboratory Report No. 9554, University of California, Berkeley, CA

Tenforde TS, Gaffey CT, Moyer BR, Budinger TF (1983) Cardiovascular alterations in Macaca monkeys exposed to stationary magnetic fields: experimental observations and theoretical analysis. Bioelectromagnetics 4: 1–9

Wikswo JP Jr, Barach JP (1980) An estimate of the steady magnetic field strength required to influence nerve conduction. IEEE Trans Biomed Eng 27: 722–723

Accepted February 12, 1983