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CONTENTS

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Original Article

Effects of Pulsed Electromagnetic Field and Swimming Exercise on Rats with Experimental Sciatic Nerve Injury

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Abstract. [Purpose] The current study aimed to reveal the therapeutic effects of a pulsed electromagnetic field and swimming exercises on rats with experimental sciatic nerve injury, which was induced with crush-type neuropathy model damage, using electrophysiological methods. [Subjects] In the current study, the sample consisted of 28 adult male Wistar albino rats. [Methods] The rats were randomized into four groups (n=7). Swimming exercise and PEMF (2 Hz and 0.3 MT) were applied one hour a day, five days a week, for four weeks. Electroneuromyographic (ENMG) measurements were taken on day 7. [Results] When the data were evaluated, it was found that the 4 weeks of PEMF and swimming exercises led to an increase in motor conduction rates and a decrease in latency values, but the changes were not significant in comparison with the control and injury groups. The compound muscle action potential (CMAP) values of the left leg were lower in weeks 2, 3, and 4 in the swimming exercise group in comparison with the control group, although for the PEMF group, the CMAP values of the left leg reached the level observed in the control group beginning in week 3. [Conclusion] PEMF and swimming exercise made positive contributions to nerve regeneration after week 1, and regeneration was enhanced.
Key words: Pulsed electromagnetic field, Swimming exercise, Nerve regeneration

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INTRODUCTION

There are various etiologic factors that lead to peripheral nerve injury. Nerve lesions developing due to trauma are the most common ones in practice[1)].

Magnetic fields and swimming exercises have been shown to affect nerve regeneration, soft tissue, and particularly the growth of all nerve tissue in many studies done about electromagnetic fields and swimming[2–5)].

The aim of the this study was to reveal the effects of swimming exercise and a pulsed electromagnetic field applied after a crush-type injury, which is a neuropathy model, with electrophysiologic methods.

SUBJECTS AND METHODS

This study was conducted after ethics committee approval had been obtained from the Local Ethics Committee for Animal Experimentation at Adnan Menderes University (ADÜ-HADYEK 050.04/2010/090). Animal rights were protected during this study.

This study was conducted in the Department of Physiology of Veterinary Medicine, Adnan Menderes University, between 2010 and 2012. A total of 28 male Wistar albino rats aged 3 months were used in the study. The influences of swimming exercise and a pulsed electromagnetic field (PEMF) applied for 4 weeks on healing were detected by an electroneuromyographic method in the regeneration processes of the rat sciatic nerve after a crush-type injury, which was selected as a neuropathy model. Rats were randomly divided into four groups with 7 rats in each. Group 1 was the control group, Group 2 was the injury control group (sciatic nerve injury), Group 3 was the injury + PEMF group (sciatic nerve injury and PEMF), and Group 4 was the injury + swimming group (sciatic nerve injury and swimming exercise). Each group was kept in macrolon cages. Rats were kept in a semi-climatized room at 22±2 °C with a 12/12 h dark and light cycle and fed standard rat feed; they were given feed and water ad libitum.

First, body weight was measured before sciatic nerve injury. It was then recorded before PEMF once a week.

Rats were applied anesthesia with ketamine (90 mg/kg) + xylazine (10 mg/kg), and the left sciatic nerve was removed at the middle femoral level by blunt dissection under aseptic conditions. The sciatic nerve was squeezed and

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crushed with fine forceps for 30 sec as 3 notch (the severity of the clamping forceps). Afterwards, the skin in the incision region was closed routinely, and the operation was terminated. A superficial wound was created only on the skin surface of the animals in control group.

A regular magnetic field was formed in the axial center of two equal regular coils with diameter of 6.5 cm set 6.5 cm apart.

According to the known basic electronic principles, the formula for calculating the magnetic field $B= (8.99 \times 10^{-7}) NI/R$ is used[6]. Here, N is the number of wire winding of a coil, I is the effective value of serial, equal flow passing from the coils, and R is the mean diameter of the coils. At 2 Hz (120 ppm), when power stage is at 8, the effective value of the coil flow is 119 mA and feeding tension of the coils is 0,4 V. The measured and calculated homogenous magnetic field density are both 0.3 mT.

PEMF application was performed with a two-channel Electromagnetic Therapy System (EMTS Digital BM 3006S).

The pulsed electromagnetic field system was composed of a pair of coils and a pulsed power source. PEMF was performed with 0,3 mT (3G) power with a frequency of 2 Hz and a sinusoidal wave form. PEMF application was performed by placing the rats between the coils in a specifically prepared apparatus with the rats in a restrainer. Application was performed for hour a day, five days a week, for four weeks and was performed concurrent to the swimming exercise in the swimming exercise group.

The rats in the swimming exercise group were put into individual pools with a depth of 30 cm and diameter of 41 cm containing water at 35±1 °C and were induced to swim. Swimming exercise was applied one hour a day, five days a week, for four weeks. This was performed concurrent to the PEMF application in the electromagnetic field treatment group.

Electrophysiologic measurements were repeated each week. Animals were administered anesthesia comprising a combination of 50 mg/kg ketamine (Alfamine®) + 10 mg/kg xylazine (Alfazine®). The field to be measured was shaved, cleaned with alcohol, and dried. The temperature of the environment was kept at 25 °C. Animals were placed on thermal pads in order to avoid potential effects of low body temperature on nerve conduction, and body temperatures were kept within normal physiologic ranges as best as possible[7].

The gastrocnemius muscle was used for EMG recording. An active electrode was placed in the middle part of the muscle, reference electrode was place in the tendon region, and ground electrode was placed on the tail. A stimulus electrode was placed at the trochanter major level of the sciatic nerve.

A VIASYS Nicolet Viking Quest (USA) two-channel EMG device was used for measurements. Data were analyzed using the VIASYS Nicolet Viking Quest software. H-reflex and M-wave responses were determined by elevating the stimulus intensity 1–2 V until reaching the maximum amplitude level.

The average stimulus intensity for the motor nerve conduction velocity (MNCV) was 12.03 V for the control group, 12.17 V for the injury group, 12.48 V for the injury + electromagnetic field group, and 12.50 V for the injury + swimming group.

The mean stimulus intensity for the H-reflex was determined to be 7.55 V for the control group, 7.83 V for the injury group, 7.73 V for the injury + electromagnetic field group, and 8.68 V for the injury + swimming group.

The latency period was taken as the duration between beginning of the stimulus and the beginning of the action potential.

The Shapiro-Wilk test was used to determine the normality of the distribution of data. Logarithmic or square root transformation was applied to data not showing a normal distribution. Significance of the difference between mean values was determined with two-way analysis of variance for repeated measurements (two-way ANOVA). Duncan's post hoc test was used to determine from which group a difference arose. Tukey's HSD post hoc test was performed to determine from when or from which group a difference arose in data with interaction8). One-way analysis of variance (one-way ANOVA) was performed using Duncan's post hoc test in order to determine from which time interval and from which group an effect arose in data where there was no time difference and group-time interaction but there was a group effect. The paired t-test was performed to compare data obtained from the right and left legs at the same measurement time points. A p level of ≤ 0.05 was taken as statistically significant. Data were analyzed using the SPSS 18.0 software. Data in tables are expressed as means ($\overline{X}$) and standard errors ($S\overline{x}$).

RESULTS

Although there were time-dependent variations in all groups in terms of body weights and left side MNCV and latency values compared with the output values for four weeks ($p<0.05$), there was no effect of any groups ($p>0.05$) or group-time interaction ($p>0.05$). The left MNCV values at the first week were lower in the injury group compared with the control group; however, the difference was not statistically significant ($p>0.05$). The increase in MNCV in the swimming and electromagnetic field groups were not statistically significant when compared with the control and injury groups. When the right and left MNCV values were analyzed for all groups, a significant reduction was detected in initial values at the time of injury in the PEMF and swimming groups and in initial values in the injury group and in the left leg compared with the right leg in week one measurements ($p<0.05$). No difference was detected in the control group in terms of right and left leg PEMF values during the experiment (Table 2).

Right side latency values were prolonged in the electromagnetic field group compared with the injury and swimming groups during the entire experiment ($p<0.05$). When the right and left leg latency values were analyzed for all measurement time points, significant elongation was detected in left side latency values compared with right side

Table 1. Left leg latency (msec) and CMAP (mV) values of rats

Group	n	Weeks Output values (0)	1st week	2nd week	3rd week	4th week
		$\overline{X} \pm S\overline{x}$	$\overline{X} \pm S\overline{x}$	$\overline{X} \pm S\overline{x}$	$\overline{X} \pm S\overline{x}$	$\overline{X} \pm S\overline{x}$
				Left leg latency (msec)		
Control*	7	1.19±0.01	1.14±0.01	1.21±0.02	1.17±0.02	1.15±0.02
Injury control*	7	1.20±0.01	1.14±0.01	1.19±0.02	1.18±0.02	1.16±0.02
PEMF*	7	1.20±0.01	1.18±0.01	1.18±0.02	1.19±0.02	1.18±0.02
Swimming*	7	1.19±0.01	1.18±0.01	1.20±0.02	1.16±0.02	1.18±0.02
				Left leg CMAP (mV)		
Control	7	7.69±6.01†	8.64±5.16†	10.98±4.62†	8.24±3.36†	11.02±6.28†
Injury control	7	1.91±1.38$^{\neq}$	2.47±1.64$^{\neq}$	4.61±2.54$^{\neq}$	4.04±2.39$^{\neq}$	4.63±2.26$^{\dagger,\neq}$
PEMF	7	4.22±3.50$^{\dagger,\neq}$	4.33±2.04$^{\dagger,\neq}$	2.40±2.82$^{\neq}$	3.33±2.46$^{\dagger,\neq}$	5.03±4.45$^{\dagger,\neq}$
Swimming	7	2.51±1.74$^{\dagger,\neq}$	4.70±3.50$^{\dagger,\neq}$	4.01±2.18$^{\neq}$	3.27±2.20$^{\neq}$	2.06±2.17$^{\neq}$

†, $^{\neq}$Different symbols within a column indicate statistical significance ($p<0.05$), *Statistically significant (there were time-dependent variations in all groups) ($p<0.05$).
Control, control group (Group 1); injury control, injury control group (Group 2); PEMF, injury + PEMF (Group 3); swimming, injury + swimming (Group 4).

Table 2. Left and right leg MNCV (m/sec) and latency (msec) ratios of rats

Group/ Week	n	MNCV (m/sec) Output values (0)		1st week		2nd week		3rd week		4th week	
		Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Control	7	32.70±1.60	33.59±0.50	38.07±1,05	36.06±1.46	33.58±1.50	34.73±0.70	32.14±073	34.20±1.35	34.72±1.23	35.01±1.07
Injury control	7	29.23±1.21*	33.89±1.02	34.37±1.48*	37.14±1.30	34.58±1.99	35.63±1.12	34.68±0.94	35.66±0.73	34.46±1.39	34.44±0.64
PEMF	7	29.74±1.54*	33.44±1.21	34.07±0.66	33.69±1.17	31.95±1.93	32.67±0.73	33.26±0.99	34.92±0.79	33.05±1.75	33.77±1.76
Swimming	7	30.28±1.57*	34.90±1.06	33.62±0.76	34.55±1.36	33.12±1.19	34.19±1.29	36.56±0.35	35.88±1.55	33.22±1.86*	36.24±0.97
						Latency (msec)					
Control	7	1.41±0.02	1.33±0.04$^{\dagger,\neq}$	1.30±0.03	1.33±0.41$^{\dagger,\neq}$	1.45±0.02	1.35±0.02$^{\dagger,\neq}$	1.36±0.01	1.41±0.02$^{\dagger,\neq}$	1.31±0.03	1.16±0.02$^{\dagger,\neq}$
Injury control	7	1.45±0.02*	1.31±0.03$^{\neq}$	1.30±0.03	1.34±0.02$^{\neq}$	1.42±0.03*	1.31±0.02$^{\neq}$	1.40±0.05	1.27±0.02$^{\neq}$	1.35±0.04	1.15±0.03$^{\neq}$
PEMF	7	1.43±0.04	1.42±0.04†	1.38±0.02	1.35±0.02†	1.40±0.06	1.44±0.01†	1.42±0.05	1.32±0.02†	1.40±0.03	1.17±0.03†
Swimming	7	1.41±0.03*	1.30±0.03$^{\neq}$	1.40±0.02	1.34±0.03$^{\neq}$	1.44±0.03*	1.32±0.042$^{\neq}$	1.35±0.01*	1.24±0.01$^{\neq}$	1.40±0.065	1.16±0.03$^{\neq}$

*Statistical significance compared with the right leg ($p<0.05$), $^{\dagger,\neq}$ Different symbols within a column indicate statistical significance ($p<0.05$).
Control, control group (Group 1); injury control, injury control group (Group 2); PEMF, injury + PEMF (Group 3); swimming, injury + swimming (Group 4).

latency values at the beginning and in the second week in the injury group and at the beginning and in the second and third weeks in the swimming group ($p<0.05$) (Table 2). In the control and PEMF groups, no difference was detected in terms of the rigt and left leg latency values ($p>0.05$) (Table 2).

A group effect was detected in terms of the left leg compound muscle action potential (CMAP) ($p<0.001$). A statistically significant decrease was detected in the injury group in terms of the CMAP value compared with the control group at output and in the first week ($p<0.05$). In the second week, the CMAP values of the control group were higher than those in all other groups ($p<0.01$). The CMAP values were significantly lower in the injury and swimming groups compared with the control group in week 3 and were significantly lower in the swimming group only in week 4 ($p<0.01$) (Table 1). The right and left leg CMAP values were found to be significantly different from each other at all measurement times points in all groups ($p<0.05$) (Table 3).

When data in the injury field were analyzed in terms of H-reflex latency values, time ($p<0.001$), group ($p<0.001$), and group-time interaction found ($p<0.001$). In within-group comparison for the PEMF and swimming groups, while there was a significant improvement in H-reflex la-

Table 3. Left and right amplitudes of CMAP (mV) and Hmax/Mmax ratios of rats

Group/ Week	n	CMAP (mV)									
		Output values (0)		1st week		2nd week		3rd week		4th week	
		Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Control	7	3.21±0.35*	6.82±1.22	7.86±1.65*	7.89±0.61	16.09±3.04*	5.86±0.33	8.12±1.18*	9.63±2.14	12.50±1.77*	12.59±1.77
Injury control	7	1.89±0.84*	5.65±2.93	2.22±0.44*	3.58±0.52	4.94±1.01*	11.17±2.31	4.12±0.08*	14.22±4.13	5.32±0.85*	6.15±0.52
PEMF	7	6.25±0.68*	34.30±2.34	3.99±0.31*	5.01±1.61	1.40±0.46*	13.07±0.88	3.56±0.75*	14.47±4.19	3.30±1.03*	5.30±1.24
Swimming	7	2.65±0.47*	14.31±4.49	2.00±0.80*	21.20±4.40	4.78±0.64*	26.61±3.13	4.03±0.60*	2.35±0.56	1.32±0.52*	1.47±0.50
		Hmax / Mmax ratios									
Control	7	0.18±0.04†	0.22±0.08	0.22±0.11	0.25±0.11	0.17±0.08†	0.22±0.06	0.14±0.09†	0.44±0.65	0.18±0.11†	0.20±0.12
Injury control	7	*0.49±0.20$^{\neq}$	0.23±0.01	*0.43±0.14	0.23±0.05	*0.70±0.28$^{\neq}$	0.24±0.03	*0.57±0.35$^{\neq}$	0.26±0.05	*0.54±0.22$^{\neq}$	0.21±0.03
PEMF	7	*0.56±0.18$^{\neq}$	0.23±0.04	*0.45±0.17	0.28±0.13	*0.78±0.30$^{\neq}$	0.23±0.07	*0.39±0.20$^{\dagger,\neq}$	0.22±0.09	*0.37±0.13$^{\dagger,\neq}$	0.20±0.04
Swimming	7	*0.63±0.26$^{\neq}$	0.18±0.04	*0.44±0.23	0.17±0.03	*0.79±0.33$^{\neq}$	0.21±0.07	*0.39±0.30$^{\dagger,\neq}$	0.18±0.06	*0.31±0.09$^{\dagger,\neq}$	0.19±0.08

*Statistical significance compared with the right leg ($p<0.05$), $^{\dagger,\neq}$Different symbols within a column indicate statistical significance ($p<0.05$)
Control, control group (Group 1); injury control, injury control group (Group 2); PEMF, injury + PEMF (Group 3); swimming, injury + swimming (Group 4).

Table 4. Left and right leg H-reflex latency (msec) values

Group/ Week	n	H-reflex latency values (msec)									
		Output values (0)		1st week		2nd week		3rd week		4th week	
		Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Control	7	5.29±0.15	5.05±0.10	5.25±0.22	4.95±0.13	5.20±0.06	4.87±0.10	5.38±0.09	4.97±0.08*	5.14±0.10	4.94±0.08
Injury control	7	6.92±0.19	4.71±0.07*	6.20±0.23	4.87±0.08*	5.78±0.16	4.84±0.19*	5.78±0.22	5.07±0.14	5.48±0.15	4.71±0.05*
PEMF	7	7.26±0.21	4.75±0.08*	6.01±0.12	4.91±0.11*	5.50±0.13	4.91±0.09*	5.46±0.16	4.81±0.08*	5.45±0.18	4.80±0.09*
Swimming	7	6.85±0.11	4.70±0.14*	6.01±0.15	4.50±0.11*	5.72±0.09	4.67±0.13*	5.26±0.14	4.94±0.05	5.47±0.18	4.67±0.08*

*Statistical significance compared with the right leg ($p<0.05$)
Control, control group (Group 1); injury control, injury control group (Group 2); PEMF, injury + PEMF (Group 3); swimming, injury + swimming (Group 4).

tency values beginning from the first week, this improvement was detected beginning from the second week in the injury group. When groups were compared with each other, the treatment application groups reached the control group values in the second week, and the injury group reach the control group values in the third week. In within-group comparisons for the injury, PEMF, and swimming groups in terms of the right and left leg H-reflex latency values, the lef-side H-reflex latency values were seen to decrease in the injury, PEMF, and swimming groups ($p<0.05$); however, this decrease was found in the right-side H-reflex latency values (healthy side) (Table 4).

While there were no significant differences in left Hmax/Mmax values for time and group factors ($p<0.001$), group-time interaction was not found ($p>0.05$). The left Hmax/Mmax values of the control group were lower than those of the other groups at output and in the second week ($p<0.01$). The left Hmax/Mmax values of the control group were lower than those of the other groups in the first week ($p<0.05$). The left Hmax/Mmax values in the third and fourth weeks were lower than those of the groups; however, this difference was only significant in the injury group ($p<0.05$). When the rigth and left leg Hmax/Mmax ratios were compared, while the left leg Hmax/Mmax ratios of the injury, PEMF, and swimming groups were higher than those of the right leg Hmax/Mmax ratios during the experiment ($p<0.05$), no difference was found in the control group ($p>0.05$) (Table 3).

DISCUSSION

Statistically significant weight gain was seen in all groups during the four weeks. There was, however, no significant difference in weight gain between groups. Weight gain in the injury group was found to be particularly more prominent after the second week. This result suggests that injured animals could have stayed immobile and therefore desired to move less, and their weight gain may have arise

from this.

Van Meeteren et al.[9] revealed that chronic-pulsed stress application reduced live weight gain in sciatic nerve injury-induced rats. Similarly, the live weight gains in the magnetic field and swimming groups being less than that of the control group indicates that both swimming exercise and PEMF application act as stress factors in animals. In addition, the reduction in live weight in the PEMF-applied animals during the first two weeks suggests that magnetic field application is a more effective stress factor compared with swimming in animals.

In our study, the mean MNCV values of the control group were 32–38 m/sec at all measurement time points. In the study of Hüseyinoğlu et al.[10] investigating nerve regeneration in Wistar albino rats, the mean sciatic nerve MNCV value was found to be 59.08 m/sec. A significant difference was seen between conduction velocities in both studies. Hüseyinoğlu et al.[10] obtained records by stimulating the nerve from two different points using a bipolar needle electrode. In our study, a bipolar superficial electrode was used for nerve stimulation. The distance between the anode and cathode of this electrode was about 2 cm. Reference points that could most easily reach the nerve were used in accordance with the literature[11]. The anatomic injury and measurement technique made determination of MNCV using a reference point mandatory. In other words, MNCV was calculated using the distance between the stimulus point and record point through only distal latency. This difference in methods also caused a lower nerve conduction velocity to be obtained than the mean nerve conduction velocity reported in the literature. Data were also found to be similar in the experiment groups, as measurements were performed similarly in all groups.

In this study, squeezing the sciatic nerve for 30 sec led to a reduction in nerve conduction velocity. However, this reduction was not significant in the intergroup comparison. On the other hand, when data obtained from healthy and injured legs of rats were compared, the difference was significant five days after injury. This difference remained in the next measurement interval in the injury group (on day 12 after injury). However, the MNCV is seen to normalize within the day seven after the fifth day that injury is determined in the swimming and PEMF groups. This finding indicates that the swimming and PEMF applications caused normalization of the MNCV one week earlier compared with the control group.

The first measurement was performed on the fifth day after injury. This five-day period, wound healing in rats, and is necessary for the formation of Wallerian degeneration. In addition, previous studies revealed that Wallerian degeneration started within the hours following injury, and this process could change depending on the type and duration of injury formation[12–14].

Myelin destruction after axotomy in rats begins from the second day, and the number of myelin-phagocytosing macrophages reaches the maximum on day 7. The myelin formation process begins on day 5 after axotomy. Both destruction and renewal processes take approximately two weeks[15, 16]. Considering that the process results in axonotmesis, a similar process may also be possible in a squeeze injury model in which cell bodies stay durable. Given that the injury was limited to only the myelin sheath, the healing process takes an average of two weeks without any interventions.

In the swimming and PEMF groups, this significance disappeared after the first measurement time point, and the MNCV values reached normal values. These findings show that swimming and PEMF have positive effects. Functional recovery after sciatic nerve injury is similar in mice and rats, and sensory and motor functions normalize within approximately three weeks in both types of injury[17].

The myelin sheath thickness is significantly increased in injured axons at the end of 6 weeks of swimming exercise started just after sciatic nerve injury and beginning 14 days after injury[18].

A histopathologic examination was not performed for the injury in our study. However, functional evaluations revealed that conduction velocities returned to normal in the injury group within two weeks.

Regarding the latency values, no significance was detected when the injury and control groups were compared to each other at the first measurement time point. When the right and left leg latency values were analyzed for all measurement times points, significance was found between the initial measurements and those at 2 weeks in the injury group and between the measurements for the second and third weeks. No change was detected in the control and PEMF groups in terms of latency values.

English et al.[19] found that M-response and H-reflex latencies were longer compared with control groups after incision. Navarro and Valero Cabre[20] also found similar results. In their study, the latency values were less prolonged in the crush group compared with the incision and implant groups. When recovery rates were evaluated, the latency values were found to be long in the crush group compared with the control even after 90 days. These findings are different from the data in our study. The latency values reached the same values as the controls in all groups at the end of 4 weeks.

Udina et al.[21] found that active and passive exercise caused prolongation of the latency value at the beginning of reinnervation and that this difference decreased with time, and they did not detect a difference between control and exercise groups in terms of latency. Based on this finding, they advocated that exercise did not increase the reinnervation ratio and that it only shortened this process. When the data of these researchers were evaluated, the results were found to be similar to ours in terms of the latency values, although they applied a different exercise. In our study, the latency values were found to be prolonged but the difference was not significant compared with the control. However, when healthy and injured legs of the same animals were compared, latency prolongation was found to be important at the first measurement time point after injury for the injury and swimming groups. Latency may change due to differences in anatomic factors like different sizes and extremity lengths of the animals and variations in measurement site (stimulus point and record point). This may be an important

factor, particularly in mild changes. However, performance of measurement for both the right and left legs of the same animal causes these variations to affect the groups by the same degree and consequently gives us the opportunity to see the effect of the intervention. Significant prolongation was seen at day 5 after injury in the swimming and injury groups when compared with healthy legs. These changes are more prominent and longer compared with the latency values when evaluated in terms of conduction velocity data and H-reflex latency. This prolongation was not found to be significant, although it was also observed in the PEMF group. These results support the results of Udina et al[21]. Detection of changes in latency values at week 3 in animals in the swimming group suggests that one hour of exercise may be a mild stress factor in animals. Vam Meeteren et al.[9] indicated that varying degrees of stress impaired nerve degeneration. The severity of the stress factor was also found to be important in this regression.

Regarding the CMAP values, amplitude values of the healthy leg were seen to be higher than in the injured leg when the right and left leg CMAP values were analyzed. The second week in the injury group, first week in the control group, initial value and second week in the PEMF group, and initial value and first and second weeks in the swimming group were found to be significant. However, the variations among the data were large (not normally distributed), making statistically significant differences difficult to find.

The H-reflex is a monosynaptic or oligosynaptic spinal reflex involving motor and sensory fibers. Normally, H-reflex latency varies depending on age and extremity length[22]. When data for the injured region were evaluated in terms of H-reflex latency values, time factors being statistically significant and the presence of group-time interaction indicated that the treatment applications were effective. In the within-group comparisons for the PEMF and swimming groups, it was found that the H-reflex latency values showed significant improvement beginning from the first week, and this improvement was seen beginning from the second week in the injury group. In the intergroup comparison, the control group values were reached in the second week in the treatment application groups and in only third week in the injury group. So it can be seen that the H-reflex latency values were normalized one week earlier compared with the control group and other groups, which is similar to the findings for the MNCV values.

The Hmax/Mmax ratios were found to be higher in all other groups compared with the control group beginning from the first measurement time point, and this continued to be the case for all measurement time points. However, the difference was less in the injury group, and it decreased compared with the injury group in the PEMF and swimming groups and approached that of the control group. Even this change was found to be more prominent in the fourth week in the swimming group. The H-reflex is highly facilitated in the early stages of the reinnervation process. As a result, the Hmax/Mmax amplitude ratio increases three- or four-fold. This is the indicator of increased synaptic responses of motor neurons to electrical stimulation of healthy afferents[20]. This increase in the reinnervation of the muscle reverses this facilitator effect, and the M wave amplitude increases, enabling the Hmax /Mmax ratio to return to normal values. Severity and form of injury is important for this return[20].

In this study, it was found that electromagnetic field application and swimming caused mild stress in animals within the first two weeks and that this was reflected in the Hmax/Mmax values as a negative effect on recovery. However, it was also found that the values of the PEMF and swimming groups approached those of the control group rather than those of the injury group within the next two weeks, but the presence of no significant difference between the swimming and PEMF groups and both the control and injury groups indicates that complete recovery could not be achieved. In conclusion, the Hmax/Mmax values indicate that the regeneration period after sciatic nerve injury is longer than four weeks.

The present study revealed that PEMF and swimming exercise after sciatic nerve injury had positive effects on the recovery process and increased regeneration. When evaluated in terms of application time, a four-week treatment period was no found to be enough for complete regeneration in both adjunctive treatment options. However, swimming exercise provided better results than PEMF application. All these data indicate that swimming exercise may be a better option due to its positive effects and due to findings about the biologic effects and side effects of electromagnetic fields being still a source of debate. This study also showed that both treatment applications could be a treatment option. In addition, it revealed that the duration of application and application protocol of a rehabilitation program may be as important as the treatment option.

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