Journal of Animal and Veterinary Advances 10 (18): 2414-2420, 2011
ISSN: 1680-5593

The Cerebral Hemodynamic Correlates of Quantitative EEG to The Right Cranial Cervical Ganglion Block in Beagle Dogs

[1]Woo-Dae Park and [2]Young-Sook Lee
[1]Department of Veterinary Nurse and Pet Science, Seojeong College, 482-133 Yangju, Korea
[2]Department of Cosmetology, Seojeong College, 482-133 Yangju, Korea

Abstract: The sympathetic nerves act on changes in cerebral blood flow. These changes have been related to changes in brain waves. Therefore, we investigated the hypothesis that changes in brain waves could be affected by changes in cerebral blood flow following a block of the Cranial Cervical Ganglion (CCG) in dogs. A total of 25 healthy beagle dogs were divided into 3 groups. Group 1 (no. = 10) underwent a block of the right CCG (rCCGB) using 1% lidocaine. Group 2 (no. = 10) served as a control for group 1. This group was injected with 1% lidocaine into the right or left side digastric muscle. Group 3 (no. = 5) served as a control for groups 1 and 2. This group underwent rCCGB using saline. After injecting drugs into the digastric muscles or CCGB, the quantitative EEG (95% spectral edge frequency (95% SEF), the Median Frequency (MF) and the relative band power (δ, θ, α, β), α:β ratio) was measured at 5, 10, 15, 20, 25 and 30 min for each 1 min. There was a 95% SEF increase in group 1 at 20 min and the MF was significantly decreased at 5 min and significantly increased at 15 min compared to those of the other groups ($p<0.05$). In the relative band power, the low frequencies (δ, θ) were significantly increased while the high frequencies (α, β) were decreased at 5 min ($p<0.05$). Thereafter, the low frequencies (δ, θ) were significantly decreased and the high frequency (α, β) was significantly increased between 10 and 20 min ($p<0.05$). The α:β ratio showed that the value decreased in the right frontal lobe. The results suggest that the rCCGB is expected to be useful in the treatment of neural and cerebral disorders in dogs because the block has a significant effect on the change of the Cerebral Blood Flow Rate (CBFr).

Key words: Cerebral blood flow, cranial cervical ganglion, dog, nerve block, qEEG, Korea

INTRODUCTION

The regulation of the autonomic nervous system's innervations as distributed in the brain greatly depends on the hypothalamus but each part of the brain also controls the function of the autonomic nerves within the organic network. The regulation of the sympathetic nerves in the brain reveals that the Cranial Cervical Ganglion (CCG) has control of it (Choi *et al*., 2002; Seo *et al*., 1996).

From previous research, the influences and effects on the autonomic nervous system in a brain are already known. Sympathetic nerves are known to have regular effects on the control of Cerebral Blood Flow (CBF). The self regulation of CBF caused by metabolism or chemical factors and effective causes, possibly influence an increase or a decrease of CBF by the stimulation or the block of a sympathetic nerve. This results in various effects depending on the animals used for experiment and the given reports (Seo *et al*., 1996). As the reports revealed, the change of correlation between CBF and Electroencephalography (EEG) by Archer *et al*. (2003) for instance, the increase of slow wave activity, correlates with the decrease of Blood Oxygen Level Dependency (BOLD). The studies of Krakow *et al*. (2001) and Hamandi *et al*. (2004) reveals the increase of high-frequency activity causing an increase of BOLD and it is possible to suggest that the cerebral hemodynamics correlates with the EEG.

The purpose of this research is to evaluate possible effects on the changes of quantitative EEG (qEEG) related to the changes of the CBFr and the cerebral hemodynamics caused by the rCCGB.

MATERIALS AND METHODS

This experiment was carried out using the same conditions as Park *et al*. (2007)'s method.

Subjects: About 25 healthy Beagles (CJ kennel, Korea, 15 males, 10 females) were used for all experiments. The study was carried out in accordance with the guidelines set by the Animal Care and Use Committee of Seojeong College, Korea. The level of care provided to the animals

Corresponding Author: Woo-Dae Park, Department of Veterinary Nurse and Pet Science, Seojeong College, 482-777 Yangju, Korea

exceeded the basic requirements outlined in the NIH Guide for the Care and Use of Laboratory Animals. Subjects were clinically examined thoroughly through general health and nervous examinations (observation, hands-on examination) to verify their health. Selected subjects were domesticated for the experiment over a 1 week period before the experiment was performed. The environment of the animals was regulated and continuously monitored, room temperature was maintained (24~26°C), a daily photoperiod (12 h day^{-1} and night) was undertaken and a special cage which isolated the subjects from external noise was used each cage containing only one subject.

Experimental conditions: The laboratory was designed to minimize influences from external lights or noises. The laboratory had only one exit and double gates. External entrances were controlled and windows were blocked to intercept external noise and light, controlling this to minimum requirements. The temperature and humidity were controlled to maintain 24~26°C. In addition, equipment was removed that could possibly distract subjects and only required experiment equipment was retained. The experiment was conducted and limited to two personnel, one for blocking the cranial cervical ganglion and the other for measuring EEG.

The subjects' heads were shaved with animal clippers at least 2 days prior to the experiment (Oster, USA) and check ups were implemented for body temperature, heart rate, breathing rate to make sure the conditions were right before the experiment. The subjects were transferred to the laboratory and left for about 20 min to let them adjust to the new atmosphere.

After the adjustment period each subject's head was disinfected with 0.05% chlorhexidine for the adhering of EEG electrodes. After sterilization, disk-figured EEG electrodes (LXEL_SAF-Dk-T01®,Laxtha Inc., Korea) were adhered to the subject's head (Park *et al*., 2007).

Experimental group: In order to assess the effects caused by the block of the rCCG, experimental groups were divided into 3 groups. The Rt group (n = 10) were injected with 1% lidocaine (Lidocaine HCl 1%®, Korean medicine manufacturer, Korea) and 2 mL into the rCCG. The L group (n = 10, served as control 1) and were injected with lidocaine (2 mL) into the left or right digastric muscles. The N group (n = 5, control 2) were injected with 0.9% normal saline solutions (Korean saline solution®, Korean drug manufacturer, Seoul, Korea, 2 mL) into the rCCG.

The procedure for the rCCGB: Injection of the blocking agent into the CCG was applied using the Park (2008) method after placing EEG electrodes on the heads of subjects. The paratrachel approach was implemented with the right 1st cervical vertebrae as a marker. When blocking, a 25 gauge needle was used as a way of keeping the effects of stress in the brain down to a minimum. The occurrence of Horner's syndrome was considered successful if all three occurrences of blepharoptosis, 3rd eyelid prolapse and miosis occurred within 5 min after the blocker injection.

qEEG recording and analysis: The EEG signals were recorded using electrodes on the subject's head, Left Frontal (LF), Right Frontal (RF), Left Occipital (LO), Right Occipital (RO), Vertex (V) and ground (Bergamasco *et al*., 2003). The subjects were stabilized during 10 min after placing electrodes which used EEG receiving equipment to measure EEG for 1 min at each 5, 10, 15, 20, 25 and 30 min intervals after the blocker injection on the head. The raw EEG signal was recorded in epoch segments with QEEG-4 (LXE3204®, Laxtha Inc., Korea).

The raw EEG signals were analyzed by qEEG analyzing software Tele-scan Version 2.85 (CD-TS-2.2®, Laxtha Inc., Korea). Each epoch was converted to an amplitude spectrum of frequency domain by the fast Fourier transformation algorithm and then calculated to the power spectrum γ frequency (30.1-50 Hz). Muscle movement was filtered and eliminated by the fast Fourier transformation method. The EEG signals were calculated and the relative band powers divided (δ (0~3.0 Hz), θ (3.1~7 Hz), α (7.1~13 Hz) and β (13.1~30 Hz). About 95% Spectral Edge Frequency (SEF), Median Frequency (MF) and α:β ratio at each time period were also calculated.

Statistical analysis: The comparisons of data were done by repeated ANOVA test (SPSS for windows 12.0 k, SPSS Korea, Korea) and considered significant at $p<0.05$ followed by a Duncan's post-hoc test.

RESULTS AND DISCUSSION

The 95% SEF: The L group showed a significant decrease at 5 min and the Rt group showed significant differences between 5-20 min. No other significant differences were observed at other times compared to the values before the block ($p<0.05$, Table 1).

In comparison of 95% SEF among groups, a significant increase appeared in the Rt group rather than in the N group at 20 min ($p<0.05$).

MF: In comparisons for each time before and after the block in each group, the Rt group only showed significant differences that indicated a significant decrease at 5 min

Table 1: Time courses of changes in 95% SEF and MF obtained by qEEG by before and after CCGB with normal saline or 1% lidocaine

Groups	EEG (Hz)	Time (min) Pre	5	10	15	20	25	30
N	SEF	23.64±2.98	23.36±3.13	24.91±4.05	23.19±4.59	22.86±4.41	22.79±4.60	23.39±4.85
	MF	7.72±3.92	8.38±4.58	8.59±5.72	8.57±4.95	9.85±6.13	7.08±4.14	7.73±3.24
L	SEF	25.08±2.32^{b}	22.18±3.39^{a}	24.99±3.16^{b}	23.16±4.16^{ab}	24.30±3.04^{b}	24.94±3.31^{b}	25.08±2.22^{b}
	MF	8.34±5.07	6.40±3.15	8.32±4.63	7.55±4.84	8.99±5.92	7.81±3.41	9.04±2.68
Rt	SEF	24.45±4.04^{ab}	23.44±3.34^{a}	23.95±3.30^{ab}	24.45±3.28^{ab}	25.64±3.29^{b}	24.28±2.95^{ab}	24.70±2.83^{ab}
	MF	7.87±4.51^{ab}	6.86±3.27^{a}	8.20±3.91^{ab}	10.98±5.32^{c}	9.19±4.51^{bc}	8.11±3.97^{ab}	8.66±4.36^{ab}

Table 2: Comparison of relative δ band power among groups

Groups	Ch	Time (min) Pre	5	10	15	20	25	30
N	1	0.748±0.186	0.847±0.092	0.872±0.0420	0.761±0.133	0.748±0.2204	0.756±0.180	0.810±0.107
	2	0.714±0.188	0.796±0.125	0.856±0.0302	0.758±0.145	0.777±0.1430	0.764±0.162	0.850±0.042
	3	0.597±0.246	0.689±0.167	0.737±0.7480	0.653±0.847	0.694±0.8720	0.747±0.761	0.727±0.714
	4	0.705±0.810	0.741±0.787	0.789±0.7140	0.678±0.796	0.707±0.8560	0.784±0.758	0.795±0.777
L	1	0.741±0.188	0.794±0.100	0.797±0.1090	0.824±0.082	0.737±0.1940	0.768±0.124	0.735±0.029
	2	0.753±0.091	0.771±0.133	0.823±0.1880	0.835±0.061	0.741±0.1630	0.735±0.185	0.704±0.014
	3	0.705±0.130	0.709±0.162	0.707±0.1660	0.774±0.107	0.732±0.1470	0.630±0.216	0.631±0.125
	4	0.734±0.127	0.723±0.130	0.651±0.2290	0.714±0.162	0.660±0.1940	0.623±0.167	0.619±0.015
Rt	1	0.742±0.099^{ab}	0.811±0.055^{b}	0.720±0.1350^{ab}	0.701±0.078^{ab}	0.621±0.2200^{a}	0.727±0.094^{ab}	0.682±0.229^{ab}
	2	0.809±0.109	0.826±0.047	0.747±0.1210	0.800±0.090	0.704±0.1740	0.767±0.139	0.705±0.132
	3	0.733±0.106	0.726±0.154	0.660±0.1620	0.628±0.202	0.595±0.2101	0.665±0.115	0.604±0.152
	4	0.677±0.175^{ab}	0.752±0.083^{b}	0.620±0.2020^{ab}	0.642±0.153^{ab}	0.584±0.1950^{ab}	0.556±0.148^{a}	0.528±0.180^{a}

The control data (N, L) are the same as Park *et al.* (2007)'s data; the data is reported as the mean±SD; Pre: before CCGB; 95% SEF: 95% Spectral Edge Frequency; MF: Median Frequency (50% spectral edge frequency); N: CCGB with normal saline; L: inject into paratracheal muscle with lidocaine; Rt: rCCGB with lidocaine; $^{a-c}$Means with different subscripts are significantly different within the same row ($p<0.05$); values with different superscripts are significantly different from the baseline ($p<0.05$); 1) statistical significances were tested by one way analysis of variances among groups; 2) the same letters indicate non-significant difference between groups based on Duncan's multiple comparison test

and significant increase after 15 min following the block ($p<0.05$, Table 1). Comparisons among groups in each time indicated a significant decrease in the L group at 5 min. The Rt group showed a significant increase to the L group at 15 min ($p<0.05$).

Relative δ band power (δ): In the comparison of each group before and after the block, none of the groups showed significant differences except the Rt group. The Rt group showed an increase in Channel (Ch) at 5 min but Ch 1 also showed low values between 10 and 30 min compared to 5 min and showed significant increases at 5 min compared to 20 min ($p<0.05$, Table 2). Ch 3 in the Rt group showed that the values after the block were decreased from the value before the block but there were no significant differences.

In Ch 4, the increased value was observed at 5 min however, it showed a decrease at 10 min hence, the values at 5 min indicated a significant increase compared to the values at 25 and 30 min ($p<0.05$, Table 2). In the comparisons of each time among groups, Ch 1 in the Rt group showed a significant decrease compared to the N group at 10 min and L group at 15 min ($p<0.05$, Table 2). In Ch 2, the Rt group indicated a significant decrease compared to the N group in 10 min ($p<0.05$, Table 2). Ch 3 did not show a significant difference but the Rt group in Ch 4 showed a significant decrease compared to N group at 25 min ($p<0.05$, Table 2).

Relative band power (θ): The values of θ in each group only showed significant differences in the Rt group shown in Table 3. Ch 1 and 2 in the Rt group did not show a significant difference but showed high values at 5 and 10 min and low values at 15 and 20 min compared to the value before the block. At 25 and 30 min, the values were higher than the values before the block. Ch 3 maintained a higher value compared to the value before the block at 5 and 10 min.

However, the value at 25 min was lower than the value before the block ($p<0.05$, Table 3). In Ch 4, the frequencies showed higher values at 5 and 10 min compared to the value before the block and also showed lower values at 15 and 20 min compared to the value before the block. The value at 30 min showed significant difference compared to the values at 15 and 20 min ($p<0.05$, Table 3).

In the comparisons among groups, the Rt group showed a significant difference compared to the N group in Ch 1 at 10 min. However, none of the statistical differences were found in Ch 2-4 ($p<0.05$, Table 3).

Table 3: Comparison of relative θ band power among groups

		Time (min)						
Groups	Ch	Pre	5	10	15	20	25	30
N	1	0.072±0.029	0.069±0.047	0.053±0.011	0.091±0.052	0.072±0.044	0.079±0.023	0.074±0.034
	2	0.078±0.027	0.080±0.043	0.072±0.014	0.090±0.037	0.077±0.031	0.099±0.036	0.076±0.023
	3	0.082±0.039	0.079±0.039	0.084±0.028	0.107±0.088	0.079±0.030	0.100±0.052	0.084±0.033
	4	0.081±0.036	0.090±0.033	0.097±0.044	0.128±0.081	0.099±0.043	0.100±0.038	0.087±0.044
L	1	0.071±0.020	0.103±0.035	0.082±0.039	0.076±0.036	0.081±0.067	0.084±0.028	0.089±0.041
	2	0.092±0.036	0.099±0.016	0.072±0.023	0.077±0.027	0.084±0.064	0.089±0.037	0.079±0.027
	3	0.095±0.033	0.119±0.043	0.084±0.025	0.089±0.035	0.083±0.032	0.105±0.038	0.106±0.063
	4	0.106±0.036	0.116±0.042	0.099±0.063	0.093±0.044	0.099±0.068	0.104±0.032	0.120±0.053
R	1	0.090±0.026	0.101±0.036	0.106±0.054	0.088±0.029	0.078±0.040	0.100±0.048	0.127±0.091
	2	0.091±0.040	0.102±0.027	0.112±0.056	0.078±0.035	0.083±0.041	0.108±0.059	0.125±0.072
	3	0.096±0.028^{ab}	0.116±0.032^{ab}	0.119±0.072^{ab}	0.097±0.043^{ab}	0.086±0.033^{a}	0.112±0.049^{ab}	0.148±0.081^{b}
	4	0.115±0.045^{ab}	0.123±0.047^{ab}	0.131±0.063^{ab}	0.092±0.039^{a}	0.089±0.034^{a}	0.143±0.055^{ab}	0.166±0.125^{b}

Table 4: Comparison of relative α band power among groups

		Time (min)						
Groups	Ch	Pre	5	10	15	20	25	30
N	1	0.036±0.0510	0.018±0.011	0.011±0.0060	0.027±0.019	0.024±0.028	0.025±0.014	0.020±0.009
	2	0.039±0.0401	0.029±0.020	0.014±0.0050	0.032±0.022	0.017±0.015	0.028±0.020	0.015±0.005
	3	0.055±0.0600	0.061±0.043	0.029±0.0140	0.046±0.035	0.033±0.027	0.048±0.038	0.031±0.023
	4	0.054±0.0660	0.052±0.034	0.026±0.0130	0.051±0.031	0.034±0.025	0.042±0.037	0.030±0.013
L	1	0.021±0.0130	0.034±0.026	0.028±0.0290	0.024±0.025	0.027±0.024	0.031±0.019	0.046±0.027
	2	0.028±0.0220	0.037±0.023	0.022±0.0140	0.020±0.017	0.019±0.011	0.030±0.028	0.026±0.023
	3	0.038±0.0280	0.064±0.060	0.042±0.0400	0.034±0.029	0.048±0.063	0.059±0.054	0.074±0.046
	4	0.037±0.0300	0.048±0.038	0.045±0.0430	0.039±0.028	0.072±0.067	0.057±0.034	0.060±0.054
Rt	1	0.032±0.0230	0.021±0.008	0.0501±0.039	0.036±0.019	0.037±0.027	0.047±0.034	0.042±0.033
	2	0.024±0.0170	0.022±0.012	0.047±0.0360	0.021±0.009	0.033±0.022	0.039±0.037	0.048±0.038
	3	0.035±0.0200	0.033±0.029	0.069±0.0560	0.046±0.026	0.048±0.026	0.058±0.039	0.057±0.032
	4	0.051±0.0320^{ab}	0.031±0.021^{a}	0.055±0.0550^{ab}	0.047±0.030^{ab}	0.053±0.030^{ab}	0.078±0.036^{b}	0.075±0.062^{b}

The control data (N, L) are the same as Park *et al.* (2007)'s data; the data is reported as the mean±SD; Pre: before CCGB; 95% SEF: 95% Spectral Edge frequency; MF: Median Frequency (50% spectral edge frequency); N: CCGB with normal saline; L: inject into paratracheal muscle with lidocaine; Rt: rCCGB with lidocaine; $^{a-c}$Means with different subscripts are significantly different within the same row ($p<0.05$); values with different superscripts are significantly different from the baseline ($p<0.05$); 1) statistical significances were tested by one way analysis of variances among groups; 2) the same letters indicate non-significant difference between groups based on Duncan's multiple comparison test

Relative band power (α): None of the groups has significant differences in the assessment of α value in each group as shown in Table 4 except the Rt group. In the Rt group, the value of all Ch showed a decrease at 5 min and showed an increase in all Ch at 10 min. In Ch 4, statistical differences were found at 5, 25 and 30 min ($p<0.05$, Table 4).

In periodic comparison among the groups, the Rt group showed a significant increase compared to the N group in Ch 1 and 2 at 10 min after the block ($p<0.05$). No particular change in other time periods was found in Ch 3 and 4 ($p<0.05$, Table 4).

Relative band power (β): In the comparisons of values in each group before and after the block, none of the significant differences was found except in the Rt group (Table 5). Statistical differences in Ch 1 in the Rt group was recognized at 5, 15 and 20 min ($p<0.05$, Table 5). Although, none of the significant differences were observed in Ch 2-4, the values of all Ch were decreased at 5 min and then increased in all Ch after 10 min (Table 5). In the comparisons of the values among the groups at each time, Ch 1 in the Rt group showed a significant increase compared to the N group at 10 min and showed a significant increase compared to the N group and L group at 15 min ($p<0.05$). In Ch's 2-4, significant differences were not found (Table 5).

α:β ratio: In the brain map that shows the change of α:β ratio, the Ch 1 and 3 of the Rt group showed increased ratio values while Ch 2 showed a decreased ratio value. Ch 2 of N group and the Ch 3 of L group showed increased ratio values (Fig. 1).

The CCG that controls the activation of sympathetic nerves in the brain is located at both sides of the craniocervical junction (Cottrell and Turndorf, 2003). Among the various reports on the changes in CBFr caused by the block of sympathetic nerves, Skinhoj (1971) suggested that the blocking of sympathetic nerves does not have much effect on the increase of CBFr and

Table 5: Comparison of relative β band power among groups

		Time (min)						
Groups	Ch	Pre	5	10	15	20	25	30
N	1	0.044±0.055	0.023±0.023	0.021±0.015	0.043±0.057	0.090±0.107	0.060±0.093	0.037±0.038
	2	0.070±0.075	0.042±0.056	0.022±0.015	0.037±0.038	0.067±0.093	0.046±0.055	0.025±0.018
	3	0.103±0.090	0.087±0.073	0.075±0.071	0.083±0.096	0.085±0.090	0.038±0.024	0.064±0.085
	4	0.082±0.091	0.067±0.049	0.045±0.042	0.068±0.058	0.088±0.089	0.034±0.023	0.038±0.014
L	1	0.058±0.082	0.030±0.031	0.040±0.025	0.035±0.036	0.074±0.095	0.054±0.040	0.078±0.039
	2	0.044±0.040	0.038±0.051	0.036±0.023	0.030±0.033	0.070±0.079	0.059±0.059	0.050±0.003
	3	0.069±0.053	0.055±0.051	0.080±0.071	0.051±0.050	0.066±0.061	0.104±0.097	0.125±0.006
	4	0.057±0.060	0.065±0.073	0.119±0.134	0.089±0.088	0.115±0.136	0.122±0.084	0.152±0.028
Rt	1	0.062±0.045^{ab}	0.027±0.016^{a}	0.065±0.039^{ab}	0.092±0.038^{b}	0.105±0.093^{b}	0.055±0.024^{ab}	0.064±0.087^{ab}
	2	0.034±0.034	0.022±0.009	0.049±0.031	0.051±0.051	0.064±0.062	0.035±0.023	0.034±0.042
	3	0.069±0.048	0.057±0.062	0.087±0.049	0.127±0.096	0.114±0.090	0.071±0.049	0.080±0.046
	4	0.093±0.084	0.061±0.067	0.107±0.089	0.136±0.096	0.145±0.117	0.125±0.115	0.141±0.118

The control data (N, L) are the same as Park *et al.* (2007)'s data; the data is reported as the mean±SD; Pre: before CCGB; 95% SEF: 95% Spectral Edge Frequency; MF: Median Frequency (50% spectral edge frequency); N: CCGB with normal saline; L: inject into paratracheal muscle with lidocaine; Rt: rCCGB with lidocaine; $^{a-c}$Means with different subscripts are significantly different within the same row ($p<0.05$); values with different superscripts are significantly different from the baseline ($p<0.05$); 1) statistical significances were tested by one way analysis of variances among groups; 2) the same letters indicate non-significant difference between groups based on Duncan's multiple comparison test

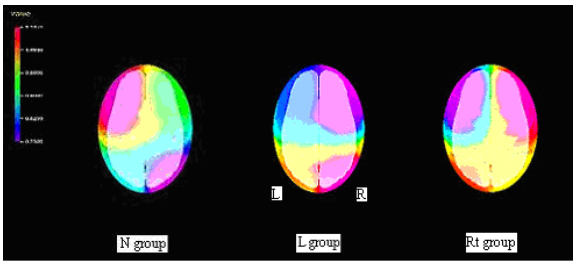

Fig 1: The brain map showing the comparison of α:β ratio among groups. The color scale indicates EEG amplitude (color change toward dark red indicates higher amplitude and change toward dark violet indicate lower amplitude)

therefore, the tension of the brain veins is insignificant. Also, Bevan *et al.* (1987) insists that the sympathetic nerve in the brain has a much greater effect on the change of the CBFr in the large cerebral blood veins which contains many adrenergic recepters but less effect on the small veins due to a slight effect caused by the limited adrenergic recepters (decreased affinity by small veins). However, Heistad *et al.* (1978) questioned the action of peripheral sympathetic nerves which has an effect on the CBFr because there are different responses when the sympathetic nervous system of animals is blocked by local anesthetic.

Several reports insist that the blocking of the sympathetic nervous system distributed into the brain caused the increase of the CBFr (Hernandez-Perez *et al.*, 1975). Alborch *et al.* (1977) announced that there was a temporary increase of the CBFr when the CCG was removed. Thereafter, it was stabilized to a regular state after 15-25 days. That means blood flow is under the control of the CCG in both sides. Moreover, Seo *et al.* (1996) reported that sympathetic nerves are obligated to have regular roles in controlling the CBFr by increasing ipsilateral CBFr through evaluating the change of mesencephalic artery's blood flow velocity change after blocking the stellate ganglion. The research into correlation between CBFr and EEG shows that qEEG can change due to the changes of CBFr (Kilner *et al.*, 2005; Kraaier *et al.*, 1992). However, questions regarding the CBFr and the brain's electronic activities have been somewhat revealed, since Schmidt and Hendrix (1938) suggested correlations between CBFr and electronic activities of the brain cortex. The decrease of the CBFr increases activities of low frequencies (δ and θ wave) and decreases activities of high frequencies (α wave) (Archer *et al.*, 2003; Kraaier *et al.*, 1992). Conversely, the increase of the CBFr decreases activities of low frequencies and increases activities of high frequency (Kraaier *et al.*, 1992).

Based on these theoretical backgrounds, the hypothesis was established that through the block of the rCCG, an increase of the CBFr is caused and then consequently, the increase in the CBFr can lead to a decrease of low frequency activities and increase of high frequency activities.

The 95% SEF and MF as the parameter of EEG changes caused by the rCCGB were used in this experiment. The Rt group and L group showed a decrease of both 95% SEF and MF at 5 min. However, the Rt group showed significant increase of both 95% SEF and MF from 15-20 min. These results mean that the block of rCCG has an arousal effect. The results verify that the blocking of sympathetic nerves leading to the eye occur in the temporarily increase of the pressure by the increase of the blood flow rate in the afferent arteriole within the eye globe. Subsequently by the compensation mechanism, the

pressure in the efferent vein is lowered by the decrease of resistance. Moreover, those results also go with Wakusugi (1991)'s report that the blocking of sympathetic nerves leading to the brain improves the CBFr which has a similar effect in relieving the tensioned sympathetic nerve. This also accords with Krakow *et al.* (2001)'s result that the increase of CBFr increases the high frequencies related to wakening. It is possible to understand the effects of the changes of CBFr by the rCCGB.

Each relative band power is calculated from the ratio taken by each frequency out of all the frequencies to minimize deviation and the weak point of qEEG. It was possible to observe the increase of δ value in the other lobes except for the right occipital lobe in the Rt group at 5 min. This result is considered to occur due to the failure of immediately re-compensating for the decreased blood efferent rate and the increased vein resistance on the temporarily increased artery blood pressure after blocking the sympathetic ganglion. Also, the right frontal lobe and the right occipital lobe showed the lowest δ value at 20 min. The results are explained by the decrease of low frequency due to the increase of CBFr.

θ value indicated an increase until 10 min and then subsequently decreased and indicated the lowest value at 20 min. These results are thought to occur because the θ is less sensitive to the changes of the blood flow rate than δ.

α and β were increased in proportion to the increase of the blood flow rate. In the Rt group, α and β values were decreased in all lobes at 5 min then α was increased at the left and the right frontal lobe at 10 min. The β value at the frontal lobe and right occipital lobe was increased from 10-20 min .The results were that the right occipital lobe maintained higher values between 10 and 30 min compared to the basal value. The results also showed that the block of rCCG has an effect on the right occipital lobe and its effect time is short.

Roh *et al.* (1991)'s results showing that the change of CBF could be caused by the block of the sympathetic ganglion comply with the results that the decreases of both high and low frequencies at 5 min were due to the improper re-compensation of the CBF caused by the increased outflow resistance. After 5 min, the decrease of low frequency and the increase of high frequency were due to the increase of CBFr. In the comparisons of α:β ratio, the ratio of the right frontal lobe by the rCCGB decreased while the ratios of the control groups increased. These results suggest that the right brain is affected by the rCCGB and that the sympathetic nerve is more predominant than the parasympathetic nerve (Hagemann *et al.*, 2003; Wittling *et al.*, 1998a, b). The effect time in the rCCGB was short within 20 min.

Wakusugi (1991) confirms the time of increased CBFr by observing the change of the blood flow in the inner carotid after the CCGB with a local anesthetic in accord with the action time of the local anesthetic. However, the inner carotid's blood flow was temporarily increased for 30 min (Seo *et al.*, 1996). This can be interpreted as the brain having outstanding self-regulation ability which increases the threshold of blood flow to maintain the regular CBFr and which also makes it possible for the brain to maintain rapid homeostasis (Tamaki and Heistad, 1986).

CONCLUSION

According the results, the study suggested that rCCGB changes qEEG value and furthermore changes in the CBFr are caused by changes of the EEG value indirectly. Also, the study suggested that the action time affecting the brain after the rCCGB is short (about 20 min).

REFERENCES

Alborch, E., B. Gomez, G. Dieguez, J. Marin and S. Lluch, 1977. Cerebral blood flow and vascular reactivity after removal of the superior cervical sympathetic ganglion in the goat. Circ. Res., 41: 278-282.

Archer, J.S., D.F. Abbott, A.B. Waites and G.D. Jackson, 2003. fMRI deactivation of the posterior cingulate during generalized spike and wave. Neuroimage, 20: 1915-1922.

Bergamasco, L., A. Accatino, L. Priano, G. Neiger-Aeschbacher, S. Cizinauskas and A. Jaggy, 2003. Quantitative electroencephalographic findings in beagles anaesthetized with propofol. Vet. J., 166: 58-66.

Bevan, J.A., J. Duckworth, I. Laher, M.A. Oriowo, G.A. McPherson and R.D. Bevan, 1987. Sympathetic control of cerebral arteries: Specialization in receptor type, reserve, affinity and distribution. FASEB. J., 1: 193-198.

Choi, Y.G., J.H. Song, H.K. Lim, J.W. Han and S.K. Lee *et al.*, 2002. The effects of stellate ganglion block on the level of plasma adrenocorticotrophic hormone (ACTH) and cortisol. J. Korean Pain. Soc., 15: 58-62.

Cottrell, J.E. and H. Turndorf, 2003. Anesthesia and Neurosurgery. 2nd Edn., Mosby, St Louis, USA.

Hagemann, D., S.R. Waldstein and J.F. Thayer, 2003. Central and autonomic nervous system integration in emotion. Brain Cogn., 52: 79-87.

Hamandi, K., A. Salek-Haddadi, D.R. Fish and L. Lemieux, 2004. EEG/functional MRI in epilepsy: The queen square experience. J. Clin. Neurophysiol., 21: 241-248.

Heistad, D.D., M.L. Marcus and P.M. Gross, 1978. Effects of sympathetic nerves on cerebral vessels in dog, cat and monkey. Am. J. Physiol., 235: H544-H552.

Hernandez-Perez, M.J., M.E. Raichle and H.L. Stone, 1975. The role of the peripheral sympathetic nervous system in cerebral blood flow autoregulation. Stroke, 6: 284-292.

Kilner, J.M., J. Mattout, R. Henson and K.J. Friston, 2005. Hemodynamic correlates of EEG: A heuristic. Neuroimage, 28: 280-286.

Kraaier, V., A.C. van Huffelen, G.H. Wieneke, H.B. van der Worp and P.R. Bar, 1992. Quantitative EEG changes due to cerebral vasoconstriction. Indomethacin versus hyperventilation-induced reduction in cerebral blood flow in normal subjects. Electroenceph. Clin. Neurophysiol., 82: 208-212.

Krakow, K., L. Lemieux, D. Messina, C.A. Scott, M.R. Symms, J.S. Duncan and D.R. Fish, 2001. Spatio-temporal imaging of focal interictal epileptiform activity using EEG-triggered functional MRI. Epileptic Disord., 3: 67-74.

Park, W.D., 2008. The development of cranial cervical ganglion block in beagle dogs. J. Life Sci., 18: 91-95.

Park, W.D., C.S. Bae, S.E. Kim, S.H. Lee and J.S. Lee *et al*., 2007. The analysis of quantitative eeg to the left cranial cervical ganglion block in beagle dogs. J. Vet. Clin., 24: 514-521.

Roh, S.J., Y.S. Cheon, B.W. Min, J.S. Goh, H.C. Kim and K.S. Kim, 1991. The changes of intraocular pressure by stellate ganglion block in glaucoma patients. J. Korean Pain Soc., 4: 133-136.

Schmidt, C.F. and J.P. Hendrix, 1938. The action of chemical substances on cerebral blood vessels. Res. Publ. Ass. Nerv. Ment. Dis., 18: 229-276.

Seo, Y.S., S.H. Kim, C.R. Hur, K.J. Lee and S.Y. Lee *et al*., 1996. Changes in blood flow velocity of middle cerebral artery after stellate ganglion block. J. Korean Pain Soc., 9: 57-62.

Skinhoj, E., 1971. The sympathetic nervous system and the regulation of cerebral blood flow. Eur. Neurol., 72: 190-192.

Tamaki, K. and D.D. Heistad, 1986. Response of cerebral arteries to sympathetic stimulation during acute hypertension. Hypertension, 8: 911-917.

Wakusugi, B., 1991. New Application of stellate ganglion block. J. Korean Pain. Soc., 4: 1-7.

Wittling, W., A. Block, E. Schweiger and S. Genzel, 1998b. Hemisphere asymmetry in sympathetic control of the human myocardium. Brain Cogn., 38: 17-35.

Wittling, W., A. Block, S. Genzel and E. Schweiger, 1998a. Hemisphere asymmetry in parasympathetic control of the heart. Neuropsychologia, 36: 461-468.