The effects of qi-gong and acupuncture on human cerebral evoked potentials and electroencephalogram

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Abstract : Although a number of studies on traditional Chinese medicine, such as qi-gong (QG), acupuncture (AC), moxibustion and Chinese herbal drugs, have been reported in recent years, there are few reports on human cerebral evoked potentials (EPs), especially relating only to QG and AC. In the present study, we examined the changes in EPs and electroencephalogram (EEG) by QG, and by AC stimulation to the point called "Zusanli" on the left lower leg, with one healthy male adult. 1.With regard to the effects of QG, significant changes in EP-components originated from the cortex suggest both facilitating and inhibitory effects of QG on the cortex. However, no significant changes in EP-components originated from the subcortex and no significant changes in EEG power% suggest that QG does not affect the subcortex. 2.With regard to the effects of AC, significant changes in EP-components originated from the cortex suggest facilitating and inhibitory effects of AC stimulation on the cortex. Furthermore, it is suggested that AC stimulation has few effects on the somatosensory and the visual pathways up to the cortex, while it has complicated effects on the auditory pathway up to the cortex. J. Med. Invest. 44 : 163-171, 1998

Key Words : *traditional Chinese medicine, qi-gong, acupuncture, cerebral evoked potentials, electroencephalogram*

INTRODUCTION

Enthusiastic efforts have been made to keep or promote the psychosomatic condition in China for centuries, that is traditional Chinese medicine. In recent years, a number of studies on traditional Chinese medicine have been reported using modern scientific methods and techniques (1〜5).

QG, originating from Chinese traditional culture has a history of more than four-thousand-years. Since the 1950s, QG has been formally used in Chinese medicine. As science has developed, there have been more studies on QG (1, 2, 4, 6〜10), and there are some studies on the electrophysiological effects of QG. However, most electrophysiological studies are about the EEG (11〜14), and there are few reports on the human cerebral EPs (15〜17), leaving the effect of QG unclarified.

AC treatment has also been commonly used in traditional Chinese medicine. There have been many studies on AC stimulation (5, 18〜23). Although there are some reports on the somatosensory evoked potential (SEP) and the auditory evoked potential (AEP), there are few studies on the visual evoked potential (VEP) (24〜29).

In the present study, we investigated the changes in EPs and EEG before, during and after QG or AC stimulation, with one healthy male adult.

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SUBJECT

The subject was a healthy male adult, 32 years old, 180cm in height, 65kg in weight, right-handed, without EEG abnormalities, and had not used psychotropic drugs. He was a postgraduate student from China who had trained QG Dazhoutain for 15 years, and who was qualified as an official AC therapist in China. The subject gave his consent to participate in this study.

METHODS

1. Recording methods of EPs and EEG

Recording electrodes were placed on the subject's scalp according to the10-20 international electrode system. During the recording, the subject was relaxed with his eyes closed, lying in a reclining chair at 70 degrees backward in a dark shielded room at 24-25°C.

Electric stimuli of square waves, with 0.5 msec pulse duration (30), at the voltage of twitching threshold of the thumb (QG ; 87.9±10.5V, AC ; 97.5±29.7V), were applied percutaneously to the median nerve at the right wrist. Flash stimuli from a XENON tube of acoustically shielded Retinogragh (MSP-2R, Nihon Kohden, Tokyo, Japan. The same if not specified), were presented from a distance of 30cm from closed eyes. Click stimuli, at 110 dBSL through

the two speakers (Foster SH10, 8ohm), were presented from a distance of 80cm from each ear at the same time. During the recording, electric stimuli were followed by flash stimuli one sec later, and click stimuli three sec later, with the next electric stimuli five sec later. A sequence of these stimuli were repeated in a cycle, and EEG containing SEP, VEP and AEP was recorded for about ten minutes.

In each experimental session, SEP, VEP, and AEP were derived from the 4 th (C3'→A 1+2)(31), the 2nd (O1→A 1+2) (32) and the 3rd channel (Cz→A1+2)(33), respectively. EPs were recorded onto magnetic tapes (Data Recorder RX-50L, TEAC, Tokyo, Japan).

For the experiment of QG, EEG containing SEP, VEP and AEP was recorded in ten experimental sessions. After the first recording (before QG, for ten minutes) as the control in the first time period, the subject started QG, and ten minutes later, the second recording (during QG) was carried out in the second time period for ten minutes, and after the termination of QG, at an interval of five minutes, the third recording (after QG) was carried out in the third time period for ten minutes.

For the experiment of AC, EEG containing VEP and AEP was recorded in four experimental sessions, and EEG containing SEP, VEP and AEP in another ten sessions. After the first recording (before AC stimulation, for ten minutes) as the control in the first time period, an AC needle of 2 inch in length (Hatuo, made in China) was inserted into a point called "Zusanli" on the left lower leg, and the second recording (during AC stimulation) was carried out in the second time period for ten minutes. After that, the AC needle was removed and the third recording (after AC stimulation) was carried out in the third time period for ten minutes.

2. Methods of data processing

2•1 Data processing of EPs

Reproducing EEG containing SEP, VEP and AEP from magnetic tapes, relating to trigger pulses, individual EPs (SEP, VEP, AEP) derived from the three derivations were obtained, averaging 100 responses (ATAC-210, 1024 address$\times 2^{20}$ bit) for 1024 msec of analysis time. Using a mini-computer (PANAFACOM U-1100, FUJITSU, Tokyo, Japan), these EPs were recorded onto floppy disk, and then subjected to the computation. Individual EPs were adjusted by a least squares method, so that sums of squares of instantaneous values from the baseline were a minimum.

2•1•1 Group mean EPs

Referring to the components in the group schematic EPs (SEP, VEP, AEP) (34～36), derived from 100 healthy male adult subjects on CRT monitor, the components (P1～P8, N1～N8) in the group mean EPs (SEP, VEP, AEP) for each time period were identified, and the changes in the waveforms of the group mean EPs were studied.

2•1•2 Component analysis of individual EPs

Referring to the components in the group mean EPs (SEP, VEP, AEP) for each time period on the CRT monitor, those with individual EPs were identified. Subsequently, the peak latencies and the interpeak amplitudes were subjected to the component analysis, and the differences in the peak latencies and the interpeak amplitudes between the data obtained before, during and after QG or AC stimulation were tested by Wilcoxon signed-ranks test.

2•2 Frequency analysis of EEG

Absolute power values of EEG were calculated by the program (QP-130B "RHYTHM") of quantitative frequency analysis. Eight time periods (32sec) with 128Hz sampling rate and 512 points were analyzed every 0.25 Hz by fast Fourier transformation (Dell 333 s/L), divided into six frequency bands : δ (2.0—3.75 Hz), θ (4.0—7.75 Hz), $\alpha 1$ (8.0—9.75 Hz), $\alpha 2$ (10.0—12.75 Hz), $\beta 1$ (13.0—19.75 Hz) and $\beta 2$ (20.0—30.0 Hz). Then, the differences in power % between the data obtained before, during, and after QG or AC stimulation were tested by Wilcoxon signed-ranks test.

RESULTS

1. Group mean EPs

For QG, the components (P 1～P 8, N 1～N 8) could be identified in the group mean SEP, VEP and AEP. Compared with the control recorded before QG, the amplitude after 100 msec in latency in SEP decreased during and after QG, and P6 shifted in the negative direction during and after QG in VEP, while there were no obvious changes in AEP (Figure 1).

For AC, the components (P1～P8, N1～N8) could be identified in the group mean SEP, VEP and AEP. Compared with the control recorded before AC stimulation, the amplitudes after 100 msec in latency in SEP decreased during and after AC, while there were no obvious changes in the group mean VEP and AEP (Figure 2).

2. Individual EPs

2•1 Changes in peak latencies

For QG, in SEP, N6, P7, N7, P8 and N8-latencies significantly decreased during QG, and there were no significant changes after QG, compared with the control. In VEP, N6, N7 and N8-latencies significantly decreased during QG, and there were no significant changes after QG. In AEP, P4, N5, N6, P8 and N8-latencies significantly decreased during QG, and there were no significant changes after QG (Table 1).

For AC, in SEP, the P 5-latency significantly decreased during AC, and there were no significant changes after AC, compared with the control. In VEP, N 8-latency significantly decreased during AC, and there were no significant changes after AC. In AEP, the P 4-latency significantly decreased during AC, and there were no significant changes after AC (Table 2).

2•2 Changes in interpeak amplitudes

For QG, in SEP, N4-P5, P6-N6 and N7-P8 significantly decreased during QG, and N1-P 2 and N2-P3 significantly increased, while P4-N4, N4-P5 and P6-N6 significantly decreased after QG, compared with the control. In VEP, P3-N3 significantly increased during QG, and N3-P4 and P8-N8 significantly increased, while P5-N5 significantly decreased after QG. In AEP, P3-N3, N3-P4 and N7-P8 significantly decreased during QG, and P3-N3 and N3-P4

significantly decreased after QG (Table 3).

For AC, in SEP, N4-P5 and P6-N6 significantly decreased during AC, and P3-N3 and P4-N4 significantly increased, while N4-P5 and P6-N6 significantly decreased after AC, compared with the control. In VEP, P3-N3 significantly increased during AC, and N6-P7, P7-N7, N7-P8 and P8-N8 significantly increased after AC. In AEP, N1-P2 and N7-P8 significantly increased, while P3-N3 and N3-P4 significantly decreased during AC, and N2-P3 significantly increased, while P1-N1, P3-N3 and N3-P4 significantly decreased after AC (Table 4).

3. Changes in EEG power%

For QG, there were no significant changes in EEG power%, during and after QG, compared with the control (Table 5).

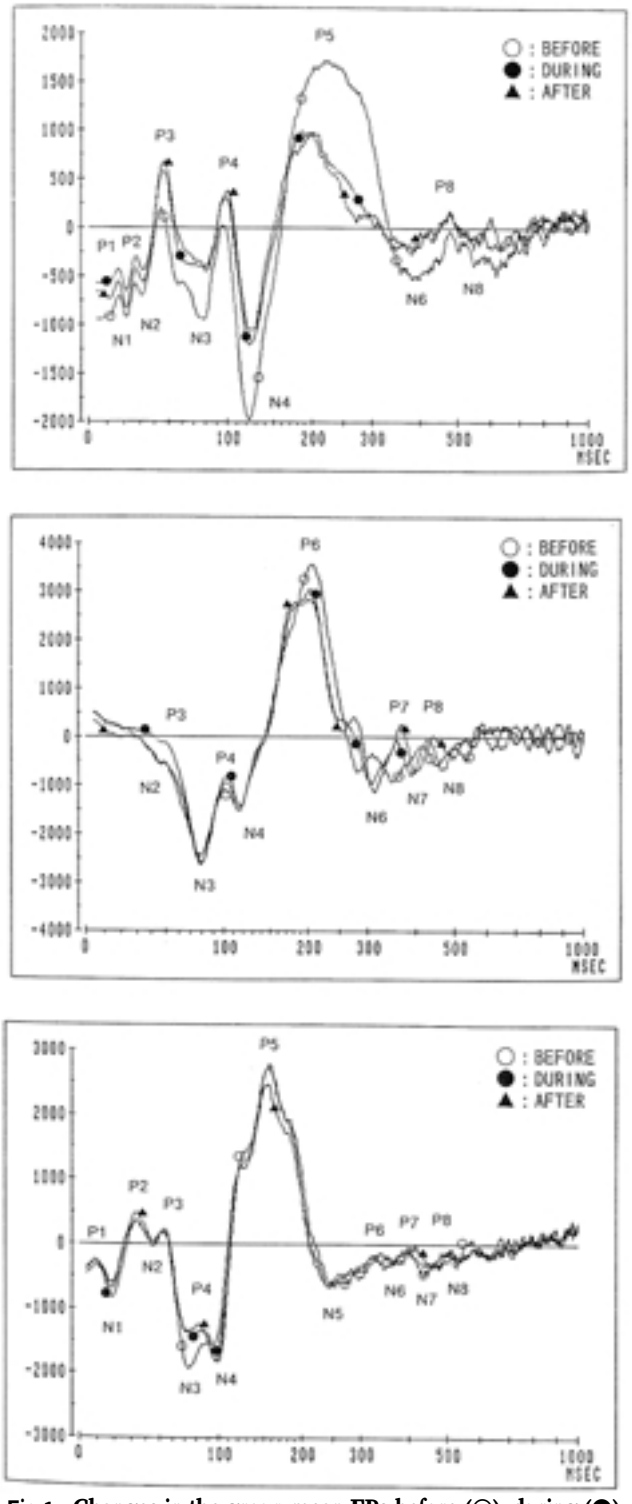

Fig.1. Changes in the group mean EPs before (○), during (●) and after (▲) qi-gong. The top, middle and bottom figures show SEP, VEP and AEP, respectively. Scales of time (horizontal) are logarithmical, and of amplitude (vertical) are comparative, 12870 corresponding to 50 μV.

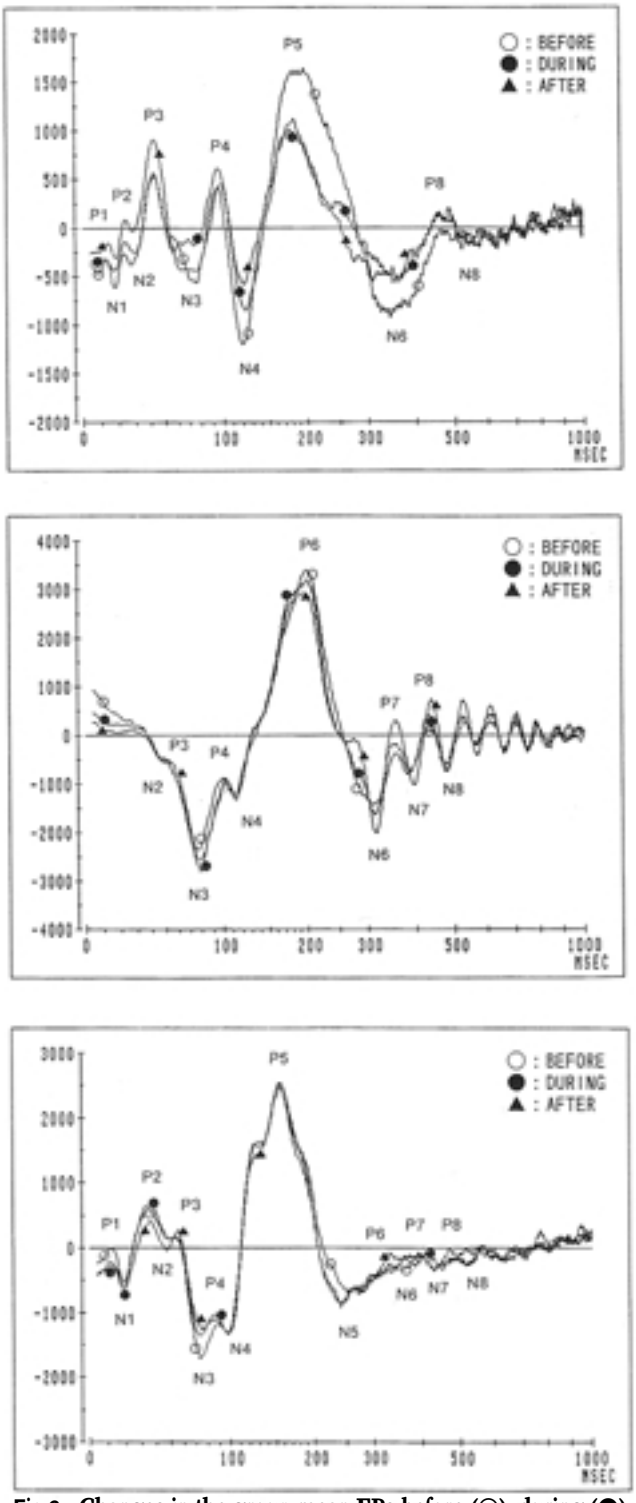

Fig.2. Changes in the group mean EPs before (○), during (●) and after (▲) acupuncture stimulation. The top, middle and bottom figures show SEP, VEP and AEP, respectively. Scales of time (horizontal) are logarithmical, and of amplitude (vertical) are comparative, 12870 corresponding to 50 μV.

Table 1. The changes in the peak latencies by QG

	SEP (C3'→Al+2)			VEP (O1→Al+2)			AEP (Cz→Al+2)		
	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)
P 1	16.5	101	98	15.6	102	80	11.4	88	83
N 1	21.1	101	101	20.0	108	95	17.9	102	104
P 2	26.8	102	104	28.0	102	96	32.6	107	105
N 2	30.8	103	103	42.3	103	104	44.3	103	104
P 3	44.2	103	104	48.9	99	109	53.1	101	99
N 3	76.1	100	103	78.2	102	101	74.3	99	96
P 4	95.7	102	102	99.7	101	103	86.8	94*	96
N 4	122.8	99	102	114.1	102	101	97.4	99	99
P 5	199.9	97	98	136.0	99	102	155.1	100	99
N 5	237.8	100	100	141.9	99	102	254.0	95**	97
P 6	253.8	94	103	203.3	97	97	317.9	97	101
N 6	358.4	90**	93	281.2	95*	98	359.1	95*	100
P 7	409.5	92*	97	313.9	94	100	401.9	99	101
N 7	434.2	93**	97	351.0	94*	98	442.5	98	102
P 8	466.7	92**	100	393.2	93	97	491.3	96**	102
N 8	493.3	92**	101	433.3	93*	98	525.7	95**	100

BEFORE : Mean latency (msec) before QG, BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01 (Wilcoxon signed-ranks test)

Table 2. The changes in the peak latencies by AC

	SEP (C3'→Al+2)			VEP (O1→Al+2)			AEP (Cz→Al+2)		
	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)
P 1	13.6	78	106	17.9	123	85	11.5	97	97
N 1	17.9	92	104	25.3	99	83	18.6	97	106
P 2	23.7	98	98	31.4	100	98	34.2	102	106
N 2	29.0	99	100	46.2	99	102	48.6	101	95
P 3	43.8	104	99	51.7	98	103	55.1	99	100
N 3	67.8	96	103	76.8	102	102	73.7	99	103
P 4	98.8	100	99	98.8	101	104	89.3	96*	99
N 4	119.9	103	98	110.9	100	101	99.2	100	98
P 5	184.7	94**	97	135.8	100	101	154.6	100	99
N 5	238.8	99	101	142.6	99	100	254.2	97	96
P 6	256.7	103	106	196.7	98	99	302.0	99	99
N 6	352.5	98	95	293.4	101	97	339.2	97	98
P 7	417.5	97	101	332.2	98	95	394.6	99	97
N 7	445.0	98	101	361.6	99	97	438.9	98	99
P 8	484.1	101	105	403.4	100	98	491.9	99	99
N 8	523.0	104	105	449.1	97*	97	534.2	99	98

BEFORE : Mean latency (msec) before AC, BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01 (Wilcoxon signed-ranks test)

Table 3. The changes in the interpeak amplitudes by QG

	SEP (C3'→Al+2)			VEP (O1→Al+2)			AEP (Cz→Al+2)		
	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)
P 1 - N 1	262.5	106	253	260.7	64	111	490.8	134	115
N 1 - P 2	374.4	110	159*	142.0	339	851	1120.5	144	134
P 2 - N 2	162.9	245	376	664.9	32	178	404.0	195	177
N 2 - P 3	952.8	133	165*	201.7	556	284	274.0	943	1486
P 3 - N 3	1255.0	87	95	2123.1	140**	109	2246.0	79*	81*
N 3 - P 4	1090.1	80	97	1562.7	130	131*	656.6	39*	44*
P 4 - N 4	2086.0	77	76*	770.4	111	139	500.2	178	92
N 4 - P 5	3647.5	59**	62**	1778.1	88	98	4745.2	100	90
P 5 - N 5	720.4	199	273	300.2	100	50*	3617.4	99	97
N 5 - P 6	524.2	35	72	4062.3	89	83	825.9	105	121
P 6 - N 6	2446.2	40**	42**	4695.8	86	88	586.8	92	116
N 6 - P 7	467.1	113	173	1142.4	237	366	676.5	110	132
P 7 - N 7	443.6	88	121	1714.1	80	112	735.4	117	144
N 7 - P 8	629.0	80*	169	1691.9	95	104	707.2	76*	119
P 8 - N 8	511.8	96	126	1164.6	120	145*	525.3	101	166

BEFORE : Mean amplitudes (50 μV=12870) before QG, BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01(Wilcoxon signed-ranks test)

Table 4. The changes in the interpeak amplitudes by AC

	SEP (C3'→Al+2)			VEP (O1→Al+2)			AEP (Cz→Al+2)		
	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)	BEFORE	DU/BE (%)	AF/BE (%)
P 1 - N 1	354.5	98	124	292.6	120	116	669.2	96	87*
N 1 - P 2	448.2	108	162	156.5	252	207	1252.4	126*	97
P 2 - N 2	235.0	130	171	794.7	145	114	590.1	150	119
N 2 - P 3	1144.7	92	102	149.3	241	206	201.6	150	318*
P 3 - N 3	1337.9	88	129*	2106.4	127*	111	1989.3	86*	87*
N 3 - P 4	1271.4	97	95	1790.5	129	115	655.6	78*	52**
P 4 - N 4	1978.9	613	325*	761.1	101	75	356.0	154	118
N 4 - P 5	3246.3	85*	88**	1950.6	111	95	4093.7	101	103
P 5 - N 5	1562.4	201	197	218.0	138	120	3432.9	107	108
N 5 - P 6	310.6	238	275	3361.4	101	88	737.1	158	186
P 6 - N 6	1893.8	70*	79*	5221.6	99	91	520.5	113	207
N 6 - P 7	1088.6	98	138	1202.5	141	181*	733.9	108	123
P 7 - N 7	365.9	146	161	993.2	179	299**	620.0	130	140
N 7 - P 8	700.6	102	123	1694.8	117	148*	649.0	133*	107
P 8 - N 8	602.0	128	140	1167.8	130	161**	593.5	117	114

BEFORE : Mean amplitudes (50 μV=12870) before AC, BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01 (Wilcoxon signed-ranks test)

For AC, there were no significant changes in EEG power% during AC, and the α1 and α2 bands power% significantly increased in the 4th channel (C3'→A1+2), and the α1 band power% significantly increased in the 3rd channel (Cz→A1+2) after AC, compared with the control (Table 6).

DISCUSSION

Traditional Chinese medicine, such as QG, AC, moxibustion and Chinese herbal drugs, has been studied in recent years. Regarding QG and AC, there are some studies on EPs. However, all of the studies are concerned with the changes in short latency components (SLC) or middle latency components (MLC), and the effects of QG or AC stimulation on EPs remain unclarified. In the present study, we investigated the changes in SLC, MLC and long latency components (LLC).

With regard to the origin of EPs, in SEP, SLC up to approximately 20 msec in latency (P1 and N1 in our data) originated from the subcortex (37). Components more than 20 msec in latency were divided into MLC (20～100 msec, P2～P4 in our data) and LLC (100 msec～, N4～N8 in our data), and are thought to have originated from the association cortex other than the somatosensory area, and to be related to higher levels of brain function such as recognition, memory and fear (38). In VEP, SLC up to approximately 70 msec (P1～P3 in our data) are thought to have originated from the special sensory pathway up to the primary visual cortex (39), and LLC (N3～N8 in our data) are thought to have originated from the cortex. In AEP, the components are divided into SLC (up to 8 msec ; auditory brainstem response) (40), MLC (8～50 msec, P1～P3 in our data) and LLC (N 3～N 8 in our data). Although there are several theories about the origin of the components, P2 and P3-components are thought to originate from the subcortex such as brainstem, reticular formation and thalamus (41, 42). Therefore, the components from N3 to N8 in our data are thought to have originated from the cortex, and to reflect the higher levels of brain function.

With regard to the changes in EPs by QG, Zhang et al. (17) reported that during QG, the amplitudes of VEP increased in a training group (in which subjects had been

Table 5. The changes in EEG power % by QG

	δ (2.0～3.75)	θ (4.0～7.75)	α 1 (8.0～9.75)	α 2 (10.0～12.75)	β 1 (13.0～19.75)	β 2 (20.0～30.0)
C3'→Al + 2						
DU/BE (%)	105	101	128	111	95	105
AF/BE (%)	103	101	118	115	100	103
O1→Al + 2						
DU/BE (%)	103	98	114	118	87	102
AF/BE (%)	106	110	104	109	101	113
Cz→Al + 2						
DU/BE (%)	105	100	131	108	98	103
AF/BE (%)	99	95	126	117	102	104

BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01 (Wilcoxon signed-ranks test)

Table 6. The changes in EEG power % by AC

	δ (2.0～3.75)	θ (4.0～7.75)	α 1 (8.0～9.75)	α 2 (10.0～12.75)	β 1 (13.0～19.75)	β 2 (20.0～30.0)
C3'→Al + 2						
DU/BE (%)	99	102	116	110	93	104
AF/BE (%)	103	90	143*	113*	96	99
O1→Al + 2						
DU/BE (%)	94	99	94	103	99	112
AF/BE (%)	89	91	114	110	92	102
Cz→Al + 2						
DU/BE (%)	94	98	112	106	100	103
AF/BE (%)	94	90	143*	109	103	100

BE : before, DU : during, AF : after, * : P<0.05,** : P<0.01 (Wilcoxon signed-ranks test)

trained for 0.5 to 5.5 years), while they decreased in a professional group (a QG therapist group) compared with the control group (a non trained group). From these results, they concluded that the differences might be attributed to the differences in the individual methods of practicing QG.

In the present study, the peak latencies of LLC in EPs significantly decreased only during QG. The interpeak amplitudes of LLC significantly increased or decreased during QG. After QG, those of MLC and LLC significantly increased or decreased. For components that originated from the cortex, it is suggested that QG had facilitating effects on the peak latencies, while it had both facilitating and inhibitory effects on the interpeak amplitudes. And then, in AEP, the amplitudes of LLC (P3-N3 and N3-P4) significantly decreased continuously during and after QG, suggesting a persisting inhibitory effect on the primary auditory cortex. For components that originated from the subcortex, there were no significant changes in the peak latencies and the interpeak amplitudes during and after QG, suggesting that QG did not affect the subcortex. In QG training, the trainer imagines a shape like a "ball" in his mind, and then "senses" it going through all parts of the body during meditation. This procedure in QG might explain our results, because imaging belongs to the cortical function.

For the changes of EEG, Kawano (11) reported that α-rhythm was accelerated during QG. However, in the present study, there were no significant changes in EEG power% during and after QG. EEG rhythm is controlled by the thalamocortical pathway, where the activity is controlled by either brainstem reticular formation or the forebrain basal region (43). Therefore, our results also suggest that QG did not affect the subcortex such as the brain stem or thalamus.

Since Dimond (44) reported that the amplitudes evoked by pain stimulation in the rat cortex were inhibited, there had been some reports on the effects of AC stimulation on SEP. In the several studies on SEP, inhibition of the amplitudes in MLC by AC stimulation (45, 46) has been observed. Besides SEP, Liao et al. reported that the amplitudes of MLC in AEP increased during AC stimulation (27).

In the present study, we chose "Zusanli" for its higher safety, and AC stimulation to that point is reported to have relaxing effects on the subjects mind from the electrophysiological point of view (47). From our results and the theories of the origins of EPs, we can summarize that peak latencies of LLC that originated from the cortex significantly decreased during AC stimulation in SEP, VEP and AEP. As for the changes in the interpeak amplitudes, the amplitudes of shorter MLC in AEP that originated from the subcortex significantly increased or decreased. Among the components that originated from the cortex, the amplitudes of MLC in SEP significantly increased, and those of LLC in SEP and the shorter LLC in AEP significantly decreased, but those of longer LLC in VEP and AEP significantly increased during and after AC. From the results that P3-N3 and N3-P4 of shorter LLC in AEP were strongly inhibited continuously during and after AC, an inhibitory effect on the primary auditory cortex is suggested.

For components that originated from the subcortex, the effects of AC stimulation are thought to be different among SEP, VEP and AEP due to the different neural pathways. In the present study, no significant changes were found in the amplitudes of SLC in SEP and VEP. However, in AEP, N1-P2 significantly increased during AC, and P1-N1 significantly decreased, N2-P3 significantly increased and also P2-N2 tended to increase after AC. These results suggest that AC stimulation to "Zusanli" does not affect the somatosensory and visual pathways up to the cortex, while it does affect the auditory pathway continuously and in a complicated manner.

Regarding EEG, Yano et al. (47), using AC-electric stimulation to the upper extremity points ("Hergu", "Shousanli", "Qiuchi") and lower extremity points ("Sanyinjao", "Zusanli"), found two types of cases : theθ band-increased type and the α band-increased type in EEG power%. In addition, these changes were not limited locally, but tended to extend to the entire cortex, whenever the stimulation was applied to the points of the upper or lower extremities. As a result, they concluded that an increase in the θ and α bands in EEG power% indicated the relaxing effects of the AC-electric stimulation. In the present study, we found no significant changes in EEG power% during AC, while the α 1 and α 2 bands power% increased significantly after AC. From these results, the subject is thought to be an α band-increased type. Considering the findings of the latency decrease in EPs that indicated the subject's wakefulness, our results are attributed to an "arousal sedative effect" of AC stimulation. Future research might explain its mechanisms.

In conclusion, QG has facilitating and inhibitory effects on the cortex, but not on the subcortex. AC has facilitating and inhibitory effects on the cortex, but few effects on the somatosensory and the visual pathways up to the cortex, while it has complicated effects on the auditory pathway up to the cortex.

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