Quantitative Analyses of EEG with Special Electrodes in TGA Patients

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Quantitative Analyses of EEG with Special Electrodes in TGA Patients

Directed by Professor Won Joo Kim

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This certifies that the Master's Thesis of
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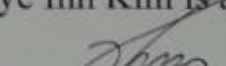

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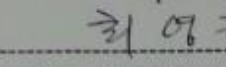

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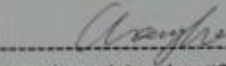

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ABSTRACT

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Transient global amnesia (TGA) is a clinical diagnosis of sudden onset amnesia resolving within 24 hours with no neurological deficits. Known routine EEG findings are mostly normal or temporal spike and slow waves. We compared EEG differences between TGA and normal patients through quantitative EEG (qEEG) and low-resolution brain electromagnetic tomography (LORETA). We retrospectively selected 37 TGA patients and 9 normal controls and analyzed the EEG with nasopharyngeal (PG1 and PG2) and anterior temporal (T1 and T2) electrodes. Spectral powers and current densities were computed in 6 different frequency bands by fast Fourier transform and by LORETA. Means were compared in 23 channels and 8 scalp regions. In TGA patients, mean delta and theta relative spectral power were significantly lower in bilateral temporal, central and parieto-occipital regions, respectively ($p<0.05$). Mean beta relative spectral power was significantly higher in right parieto-occipital ($p<0.05$) and insignificantly higher in left parieto-occipital region ($p=0.067$). In LORETA, beta2 and gamma frequency densities were significantly increased in both parieto-temporo-occipital and limbic regions ($p<0.01$). Statistical comparison of qEEG and LORETA showed significant differences in several frequencies, suggestive of bilateral parieto-occipital involvement. However, in qEEG relative power analysis, the significance was greater in right side. This is inconsistent to previous studies reporting bilateral or left dominance of qEEG in TGA patients. Also, beta power was significantly increased in TGA patients in our study, opposite to previous study.

Key words : transient global amnesia (TGA), quantitative EEG(qEEG), nasopharyngeal, beta frequency, hippocampus

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I. INTRODUCTION

Transient global amnesia (TGA) is a clinical diagnosis of sudden onset amnesia resolving within 24 hours with no neurological deficits. The association between TGA and various etiologies such as migraine, focal ischemia, venous flow abnormality and seizure has been suggested, but the exact cause and mechanism are still unknown.[1]

Most EEG findings in TGA patients were reported normal. Previous studies reported abnormal EEG findings in about 20-40% of TGA patients, mostly bilateral or left temporal spike and slow waves.[2-4]

In epilepsy, special electrodes such as nasopharyngeal and anterior temporal electrodes are useful in finding mediobasal temporal EEG abnormalities, when scalp EEG is normal.[5, 6] Reports on EEG with special electrodes such as nasopharyngeal and anterior temporal electrodes in TGA patients have rarely been reported.

Quantitative EEG (qEEG) is commonly used in behavioral science, psychiatry, and neurology in search for functional abnormalities.[7, 8] However, there are not many qEEG studies in TGA patients. Primavera et al. analyzed the qEEG of 13 TGA patients within 1 week from onset compared to 13 normal controls and reported the decrease of beta1 power in multiple channels with high level of statistical significance in the temporal and parietal locations of the left

hemisphere. The authors had suggested a persistent damage of the left temporo-parietal structures after the TGA attack as a possible mechanism.[9]
In this study, we will compare the EEG differences between TGA and normal patients through the spectral power analysis and the low-resolution brain electromagnetic tomography (LORETA) and investigate the mechanism of TGA.

II. MATERIALS AND METHODS

1. Participants

We retrospectively selected 40 TGA patients and 10 normal controls with normal EEG. Patients who had undergone EEG within 3 days of onset were selected for inclusion. We excluded the patients whose EEG data were not suitable for analysis due to the presence of artifacts related to electrical current, muscle contraction, drowsiness, or electrode motion. Total 36 EEGs from TGA group and 9 EEGs from normal control group were analyzed. The diagnosis was made due to the TGA diagnostic criteria suggested by Hodges and Warlow in 1990: (1) Witness attacks and information available from an observer, (2) Clear-cut anterograde amnesia during the attack, (3) No clouding of consciousness or loss of personal identity, (4) No focal neurological symptoms or epileptic features, (5) No recent history of head trauma or seizures, (6) Resolution of symptoms within 24 hours.[10]

2. Clinical Assessment

We evaluated the demographic and clinical characteristics between the TGA patients and normal controls by retrospectively reviewing the medical records. The onset age, sex, past history of hypertension, diabetes, hyperlipidemia, old cerebrovascular accident, coronary artery occlusive disease, psychiatric disease,

smoking, alcohol, previous TGA, precipitating event and whether the patient was taking psychiatric medications were assessed.

3. EEG recording

The EEG with anterior temporal and nasopharyngeal electrodes was recorded in a quiet and dim room with minimal background noise. The patients lied on a comfortable bed in a resting awake state. Nineteen EEG electrodes(Fp1, Fp2, F3, F4, F7, F8, Fz, T3, T4, T5, T6, C3, C4, Cz, P3, P4, Pz, O1, and O2) were attached to the scalp by international 10-20 system. The anterior temporal electrodes (T1 andT2) were placed lateral to the outer canthi superior to the zygomatic arch and the nasopharyngeal electrodes (PG1 and PG2) were inserted through both nostrils and placed in the nasal cavity. The EEG data were recorded on a computer based system with a Comet-PLUS EEG (Grass Products, Warwick, RI, USA) at a sampling rate of 200 Hz. The EEG data were filtered by 0.3-75 Hz band pass filter and 60 Hz notch filter. The ground electrodes were placed on the forehead and the reference electrode were at A1 and A2. We recorded the EEG for 40 minutes during awake state.

4. EEG Analysis

The recorded data were preprocessed using MATLAB 2009a (Mathworks, Inc., Natick, MA, USA). A specialized epileptologist reviewed the EEG by visual inspection. Epochs contaminated with artifacts, such as sweating, movement, EMG, EKG artifacts, were rejected. After the baseline correction to remove DC offset, any epochs with signals over ±75μV on any channel were excluded from the analysis. Twenty-five artifact-free 2-second epochs were used for the analyses. Before the analyses, the signals recorded from all electrodes except PG1 and PG2 were re-referenced to an average reference.

The fast Fourier transformation was performed on 23 electrode channels and spectral powers were calculated for 6 different frequency bands: delta (1-4Hz), theta (4-8Hz), alpha (8-14Hz), beta1 (14-20Hz), beta2 (20-25Hz), and gamma (25-70Hz). The band powers were normalized by dividing by the whole band power (1-70 Hz), and then log-transformed. We divided 23 channels into 8 scalp regions: right frontal (Fp2, F4, F8), left frontal (Fp1, F3, F7), right temporal (T2, T4, T6), left temporal (T1, T3, T5), right central (C4), left central (C3), right parieto-occipital (P4, O2), left parieto-occipital (P3, O1), right nasopharyngeal (PG2), and left nasopharyngeal (PG1). The log-transformed relative powers were calculated and the means were compared and the current source density of qEEG band power was calculated using LORETA.[11] LORETA method calculates the current distribution throughout the whole brain area under the hypothesis that neurons nearby activate simultaneously.

5. Statistical analyses

We compared demographic and clinical characteristics between TGA patients and normal controls by using Fisher's exact test for categorical variables, and the independent t-test or Mann-Whitney U test for continuous variables based on the normality tests. The threshold for statistical significance was set to $p<0.05$. We used SPSS 17.0 for analysis.

III. RESULTS

1. Demographic and Clinical Characteristics

Demographic and clinical characteristics between 36 TGA patients and 9 normal controls showed no significant difference (Table 1). Among the TGA patients, 13.9% had the past history of psychiatric disease, but this was not significant compared to 11.1% of the normal control group. Also, 16.7% of

TGA patients were under psychiatric medications, such as benzodiazepine and selective serotonin receptor inhibitor, but showed no significant difference to 33.3% of normal control group.

Table 1. Clinical characteristics between TGA patients and normal controls

	Normal (n=9)	**TGA (n=36)**
Age in years, median±SD	51.00±23.46	60.00±10.27
Male sex	6 (66.7%)	10 (27.8%)
Hypertension	3 (33.3%)	13 (36.1%)
Diabetes	0 (0.0%)	3 (8.3%)
Hyperlipidemia	0 (0.0%)	3 (8.3%)
Old CVA	1 (11.1%)	1 (2.8%)
CAOD	1 (11.1%)	0 (0.0%)
Previous TGA	0 (0.0%)	4 (11.1%)
Psychiatric history	1 (11.1%)	5 (13.9%)
Psychiatric medications	3 (33.3%)	6 (16.7%)
Smoking	2 (22.2%)	3 (8.3%)
Alcohol	3 (33.3%)	10 (27.8%)
Precipitating event	0 (0.0%)	11 (30.6%)

2. Relative EEG spectral power analysis

We compared the relative EEG spectral powers of 23 channels between TGA patients and normal controls in each frequency band (Table 2). In TGA patients, the mean delta relative spectral power was significantly decreased in F7, T1, T3, T5, C3, P3, O1, T4, T6, Fp2, C4, P4, O2, Fz, Cz, Pz channels and the mean theta relative spectral power was significantly decreased in T1, T3, T2 channels ($p<0.05$). In contrast, the mean beta1 relative spectral power was significantly increased in P4 and O2 channels and the mean beta2 relative spectral power was

significantly increased in O2 channel in TGA patients ($p<0.05$). The anterior temporal and the nasopharyngeal electrodes also showed a decrease of the mean delta and theta frequencies and an increase of the meal beta1 and beta2 frequencies, but was not statistically significant.

Table 2. Relative EEG spectral power differences of 23 electrode channels

	Delta		Theta		Alpha		Beta1		Beta2		Gamma	
Channel band	P-value	Mean difference [TGA-Normal]	P-value	Mean difference [TGA-Normal]	P-value	Mean difference [TGA-Normal]	P-value	Mean difference [TGA-Normal]	P-value	Mean difference [TGA-Normal]	P-value	Mean difference [TGA-Normal]
F7	0.031	-1.40096	0.154	-0.64365	0.977	-0.19294	0.780	0.23032	0.916	0.09379	0.684	-0.4419
T1	0.009	-2.15645	0.003	-1.63483	0.196	-0.68876	0.826	0.16754	0.595	0.42271	0.390	0.84995
T3	0.025	-1.67415	0.042	-1.00152	0.963	-0.02846	0.195	1.04393	0.339	0.80247	0.518	0.66742
T5	0.002	-1.80786	0.054	-1.10275	0.349	0.07778	0.140	1.17819	0.129	1.23253	0.758	0.27722
Fp1	0.441	-0.82784	0.349	-0.61442	0.929	0.05947	0.478	0.65138	0.439	0.90237	0.465	1.18809
F3	0.115	-1.06306	0.329	-0.44225	0.753	0.19114	0.670	0.33879	0.547	0.53831	0.934	-0.08641
C3	0.019	-1.43578	0.227	-0.4309	0.551	0.40693	0.313	0.89214	0.253	1.0615	0.603	0.48747
P3	0.007	-1.46753	0.207	-0.54087	0.379	0.39336	0.105	1.50196	0.097	1.55714	0.498	0.59378
O1	0.001	-1.54626	0.278	-0.75073	0.100	0.6361	0.057	1.51949	0.055	1.60052	0.431	0.65336
F8	0.420	-0.74297	0.508	-0.38595	0.777	0.02235	0.735	0.2373	0.761	0.23989	0.791	0.29298
T2	0.084	-1.53563	0.006	-1.40661	0.600	-0.25039	0.730	0.22593	0.587	0.3822	0.545	0.55921
T4	0.040	-1.5015	0.12	-0.77566	0.953	0.03153	0.296	0.73902	0.473	0.56568	0.895	0.13787
T6	0.006	-1.64869	0.07	-0.96373	0.281	0.2074	0.078	1.31375	0.056	1.46933	0.507	0.567
Fp2	0.041	-1.40218	0.164	-0.65891	0.478	-0.1437	0.820	0.10121	0.670	0.48068	0.336	1.41285
F4	0.134	-1.10547	0.365	-0.41681	0.387	0.47747	0.730	0.27033	0.741	0.31823	0.688	0.47186
C4	0.030	-1.34238	0.14	-0.57088	0.563	0.38683	0.217	1.07811	0.211	1.15925	0.403	0.84692
P4	0.015	-1.43427	0.093	-0.80108	0.294	0.44149	0.036	1.52302	0.066	1.705	0.469	0.63669
O2	0.003	-1.4638	0.267	-0.69395	0.083	0.76975	0.041	1.63668	0.020	1.95598	0.378	0.71178
Fz	0.039	-1.45714	0.697	-0.21329	0.307	0.4437	0.714	0.30543	0.840	0.18274	0.700	0.42777
Cz	0.021	-1.27047	0.598	-0.21376	0.152	0.86208	0.284	0.9046	0.312	0.9687	0.424	0.72934
Pz	0.031	-1.20362	0.258	-0.49773	0.112	0.65513	0.205	1.13397	0.146	1.27632	0.394	0.75366
PG1	0.562	-0.58074	0.181	-0.78706	0.481	0.51897	0.251	0.92223	0.268	0.93038	0.39	0.94584
PG2	0.071	-1.46673	0.206	-0.7498	0.086	1.18053	0.099	1.3583	0.123	1.3453	0.420	0.97249

We also compared the relative EEG spectral powers of 8 different scalp regions between TGA patients and normal controls (table 3). In TGA patients, the mean delta relative spectral power was significantly decreased in the

bilateral temporal, central and parieto-occipital regions and the mean theta relative spectral power was significantly decreased in the bilateral temporal region ($p<0.05$). Also, the mean beta2 relative spectral power was significantly increased in the right parieto-occipital region in the TGA patients ($p<0.05$). Although statistically insignificant, the mean beta1 relative spectral power was increased in the bilateral parieto-occipital region ($p=0.056$ and $p=0.071$, respectively) and the mean beta2 relative spectral power was increased in the right parieto-occipital region ($p=0.067$).

Table 3. Relative EEG spectral power differences in 8 scalp regions

	Delta		Theta		Alpha		Beta1		Beta2		Gamma	
Region	p-value	Mean difference [TGA-Normal]	p-value	Mean difference [TGA-Normal]	p-value	Mean difference [TGA-Normal]	p-value	Mean difference [TGA-Normal]	p-value	Mean difference [TGA-Normal]	p-value	Mean difference [TGA-Normal]
LF	0.057	-1.09729	0.245	-0.56678	0.970	0.01922	0.603	0.40683	0.575	0.51149	0.856	0.21993
RF	0.168	-1.08354	0.369	-0.48722	0.812	0.11871	0.775	0.20295	0.702	0.34626	0.544	0.72589
LT	0.005	-1.87949	0.005	-1.24637	0.693	-0.21315	0.271	0.79655	0.277	0.81924	0.513	0.59819
RT	0.024	-1.56194	0.016	-1.04867	0.993	-0.00382	0.233	0.75957	0.235	0.80573	0.634	0.42136
LC	0.019	-1.43578	0.227	-0.4309	0.551	0.40693	0.313	0.89214	0.253	1.0615	0.603	0.48747
RC	0.030	-1.34238	0.14	-0.57088	0.563	0.38683	0.217	1.07811	0.211	1.15925	0.403	0.84692
LPO	0.002	-1.5069	0.223	-0.6458	0.133	0.51473	0.071	1.51072	0.067	1.57883	0.46	0.62357
RPO	0.005	-1.44904	0.148	-0.74752	0.125	0.60562	0.056	1.57985	0.034	1.83049	0.421	0.67424

3. LORETA analysis

The log of ratio averages of LORETA beta2 frequency densities were significantly increased in the bilateral parieto-occipital, temporal and limbic regions ($p<0.01$): superior temporal gyrus, subgyral temporal lobe, angular gyrus, supramarginal gyrus, middle occipital gyrus, superior occipital gyrus, cuneus, precuneus, lingual gyrus, supramarginal gyrus, subgyral parietal lobe, inferior parietal lobule, angular gyrus, superior parietal lobule, postcentral gyrus, precuneus, posterior cingulate, subgyral limbic lobe and insula (Figure 1). The

log of ratio averages of LORETA gamma frequency densities were significantly increased in the bilateral parieto-occipital, temporal and limbic regions ($p<0.01$): angular gyrus, middle temporal gyrus, cuneus, middle occipital gyrus, lingual gyrus, superior occipital gyrus, precuneus, superior parietal lobule, inferior parietal lobule, subgyral parietal lobe, postcentral gyrus, paracentral lobule, precuneus, and subgyral limbic lobe (Figure 2). The convergence analysis of log of ratio averages of LORETA beta2 and gamma frequency densities also showed a significant increase bilateral parieto-occipital, temporal and limbic regions ($p<0.01$; Figure 3A, B).

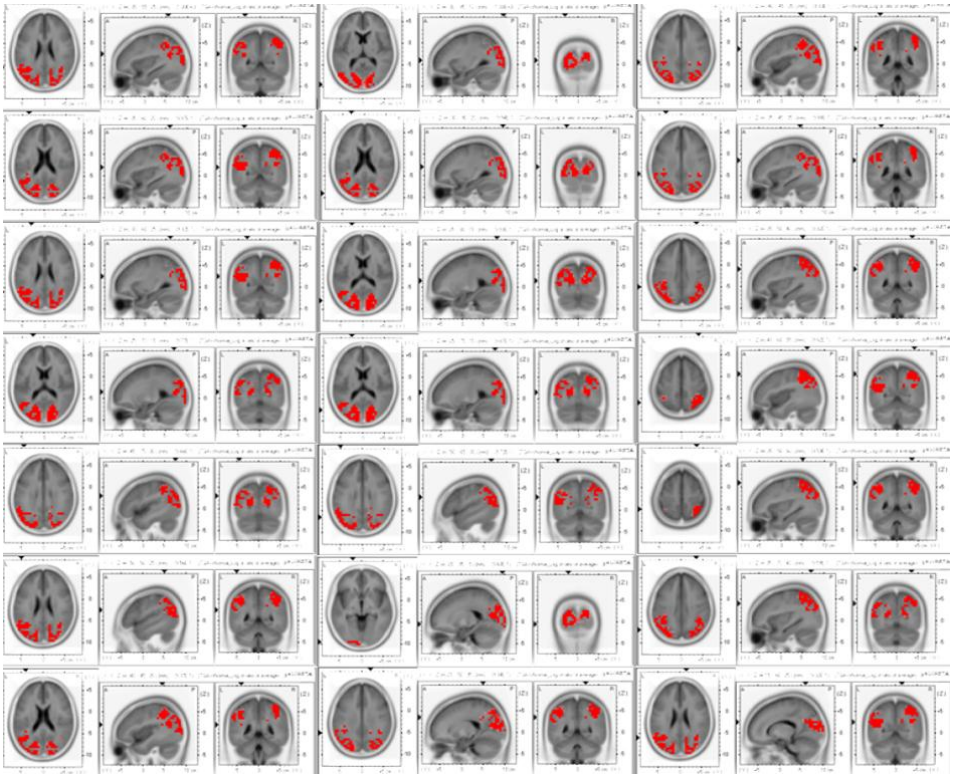

Figure 1. Log of ratio averages of LOREATA beta2 frequency densities. The figure shows significant increase of LORETA beta2 frequency densities in the bilateral parieto-occipital, temporal and limbic regions ($p<0.01$).

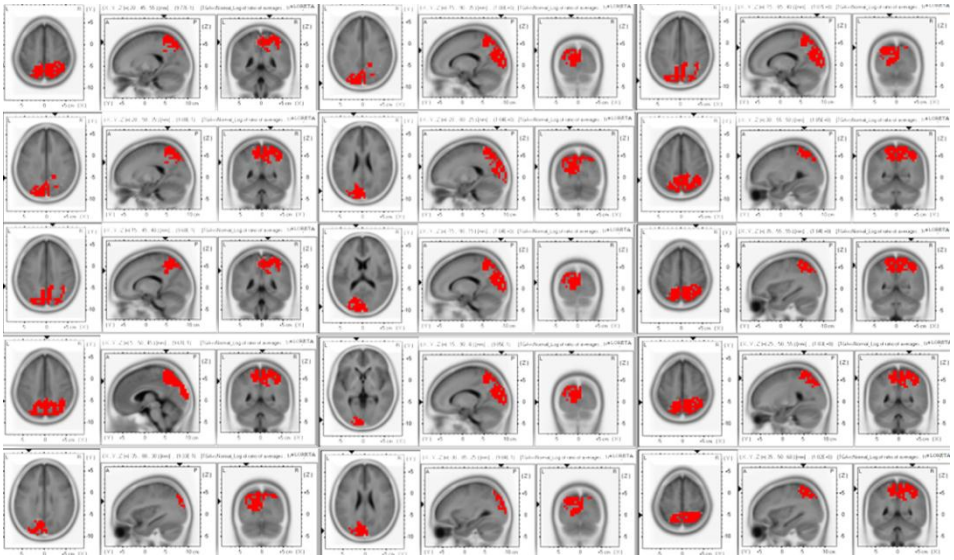

Figure 2. Log of ratio averages of LORETA gamma frequency densities. The figure shows significant increase of LORETA gamma frequency densities in the bilateral parieto-occipital, temporal and limbic regions ($p<0.01$).

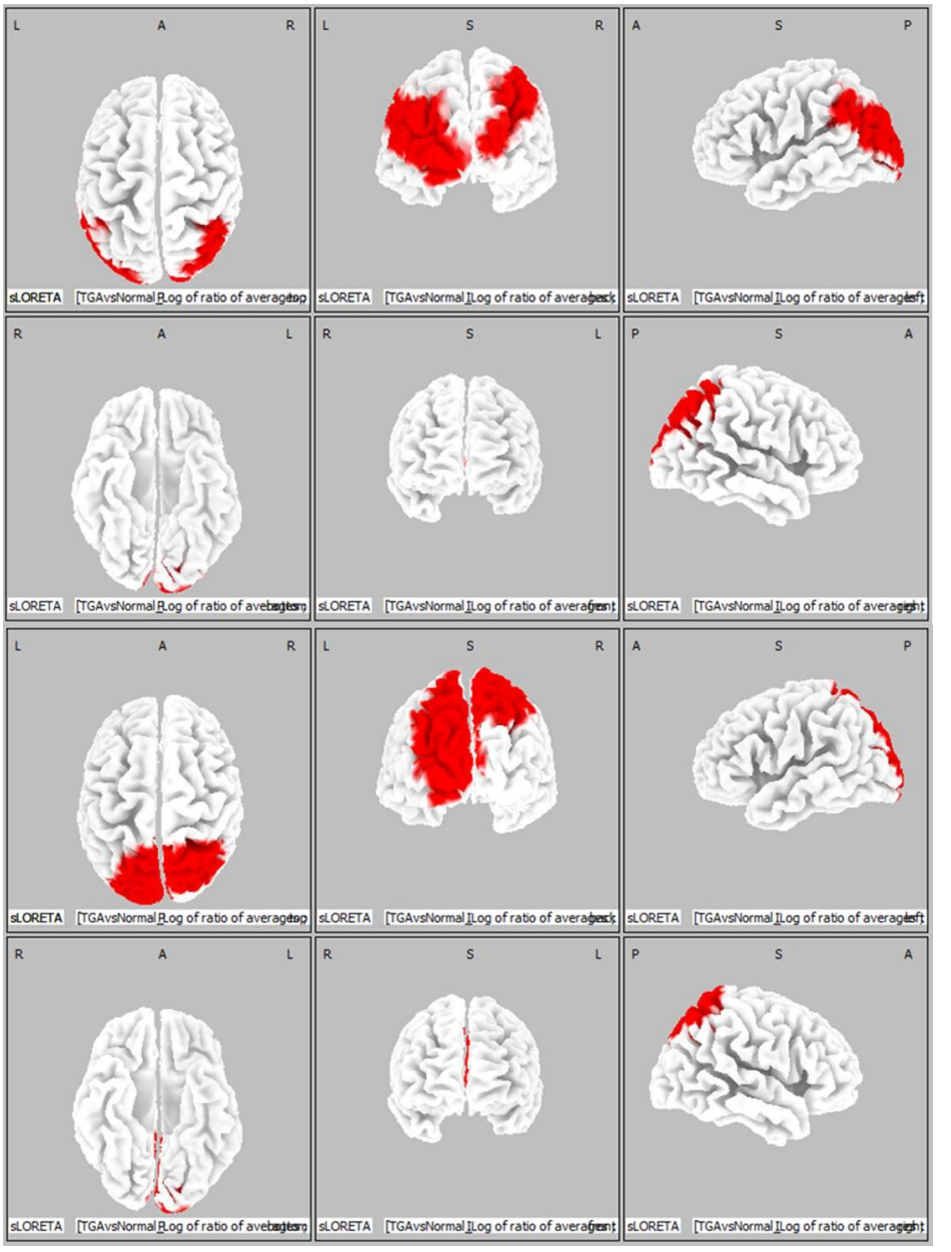

Figure 3. Convergence analysis of log of ratio averages of LORETA beta2 and gamma frequency densities. The figure shows significant increase in the bilateral parieto-occipital, temporal, and limbic regions ($p<0.01$).

IV. DISCUSSION

The present study investigated EEG differences between TGA patients compared to normal controls by two approaches: spectral analysis of the EEG power values and computation of intracortical spatial distribution of the EEG generators using LORETA.

The spectral analysis showed significant decreases in the mean delta and theta relative spectral powers and increase of mean beta relative spectral power. To our knowledge, this study is the first to demonstrate the increase of beta power in TGA, in contrast to a previous study.

In TGA, it has suggested that the increased glutamatergic transmission and calcium influx in response to the preceding stresses might modulate CA1 synaptic mechanisms, which is selectively vulnerable to metabolic and oxidative stresses.[9, 12] Previous quantitative EEG studies had showed that relatively slower EEG frequencies, such as delta and theta, correspond to cerebral hypometabolism and relatively faster EEG frequency, such as beta, correspond to cerebral hypermetabolism.[13-15] We think that the decrease of the mean delta and theta frequencies and the increase of the mean beta relative powers may reflect comparatively increased metabolic activities in TGA patients, thus might result in the affect hippocampal CA1 neurons and following derangement of the memory pathways. We think that, the decrease of delta and increase of beta relative spectral powers in the bilateral parieto-occipital regions in addition to the significant decrease of the delta and theta relative spectral powers, suggest not only the dysfunction of the mesial temporal area and but also the involvement of the subsequent cortico-parahippocampal-hippocampal pathway.

Most previous MRI, EEG, and neuropsychological studies have reported

abnormalities in bilateral or left side. [16-20] However, this study showed significant increase of beta2 relative spectral power only in the right hemispheric region. Although the handedness of the subjects was not available, we suspected that the fast conduction of signals through the hippocampal commissure, as in the temporal lobe epilepsy, may have resulted this.[21, 22] The results of the LORETA analysis revealed similar findings with the spectral analysis, with significant increase of log of ratio averages of LORETA beta2 and gamma frequency densities in the bilateral parieto-occipital, temporal, and limbic regions. The LORETA method calculates the current distribution of the surface of the scalp and reconstructs a three-dimensional images, it can visually show a spectral power map with significant differences.[23] Thus, the LORETA results of the present study correlate with the spectral analysis, also suggesting the involvement of the mesial temporal lobe memory system throughout the brain.

There are several limitations to this study. First, although we selected only the patients within the 3 day period from the symptom onset, there still is a time gap between the amnesic event and the EEG. Thus, whether the patient was amnestic or had recovered at the time of EEG was not considered for analysis and may have resulted in less accurate elucidation of the the TGA mechanism. Second, the neuropsychological tests were not available and, therefore, the cognitive function could not be correlated with the qEEG results. Third, although the baseline characteristics showed no difference of psychiatric history and medications, the possibility that TGA patients might have been more anxious during the EEG due to their amnestic symptoms and thus resulted in the increased beta frequency still cannot be excluded. Lastly, since our study only contained a small number of patients, a confirmation study with a large number is needed to verify this finding.

V. CONCLUSION

To summarize, in the present study of the spectral analysis and LORETA analysis in TGA patients revealed similar findings. Although the increase of beta2 relative spectral power was detected in contrast to a previous study, we think that this suggests the perturbation of the cortico-parahippocampal-hippocampal pathway precipitated by the emotional stress and subsequent glutamate release in TGA. Further investigation is needed in the future.

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일과성전체기억상실 환자에서 특수 전극을 이용한 뇌파의 정량적 분석

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일과성전체기억상실(TGA, transient global amnesia)은 특이 신경학적 결손 없이 갑자기 발생하여 24시간 이내에 호전되는 기억상실을 특징으로 하는 질환이다. 알려진 뇌파 소견은 대부분 정상 혹은 측두엽 이상소견이 있다. 저자들은 TGA 환자와 정상인에서 정량적 뇌파 분석 및 LORETA 분석을 이용하여 뇌파의 차이를 비교하였다. 37명의 TGA 환자와 9명의 정상인에서 비인두 전극과 전측두 전극을 이용한 뇌파를 시행하였다. 주파수 스펙트럼은 6개의 주파수에서 fast Fourier transform과 LORETA 방법을 이용하여 분석하였고 23개의 채널과 8개의 두피 영역에서 비교를 하였다. TGA 환자에서 평균 델타 상대적 스펙트럼 파워가 양측 측두, 두정, 후두, 중심 영역에서, 평균 세타 상대적 스펙트럼 파워가 양측 측두엽에서 의미있게 낮았다. 평균 페타 상대적 스펙트럼 파워가 우측 두정, 후두엽에서 의미있게 높았다. 이는 TGA가 해마를 포함한 양측 측두엽 뿐만 아니라 두정엽과 후두엽 등을 포함한 피질-피질하-해마 경로와의 과대사와의 관련성을 시사하는 소견이라 생각된다.

핵심되는 말 : 일과성전체기억상실(TGA), 양적 뇌파 분석, 비인두, 베타파, 해마