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# Left dominance of EEG abnormalities in patients with transient global amnesia

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ABSTRACT

*Purpose:* Transient global amnesia (TGA) is a syndrome of unknown etiology. Electroencephalographic (EEG) abnormalities in TGA have been reported previously. We analyzed the frequency and characteristics of EEG abnormalities in patients with TGA.
*Methods:* We collected EEGs of patients with a clinical diagnosis of TGA who had visited the emergency room or the outpatient clinic over a period of 8 years and compared clinical and demographic characteristics of the patients with normal EEGs with those with abnormal EEGs.
*Results:* EEG abnormalities were found in 35 (22.9%) out of 153 patients and epileptiform discharges were seen in 26 (74.3%) out of these 35 patients. Spikes or sharp waves were detected on the left side only (48.6%) or on both sides (25.7%), but none of the patients showed spikes or sharp waves on right side only. In six patients the EEG had normalized within three months of presentation, in ten within six months, and in twelve by one year. The EEG remained abnormal in eleven out of the 23 patients one year after presentation.
*Conclusion:* In this largest consecutive EEG study at one center, the proportion of patients with TGA in whom epileptiform discharges were demonstrated within days of the episode of TGA was significantly higher than in the previous literature. EEG abnormalities such as spikes or sharp waves spontaneously disappeared in almost half of cases over one-year of follow-up. There was a clear left dominance of EEG abnormalities in patients with TGA.


## 1. Purpose

Transient global amnesia (TGA) is characterized by development of acute anterograde amnesia without loss of consciousness, lasting less than 24 h. Poser and Ziegler described seven cases with temporary amnesia as a manifestation of cerebrovascular insufficiency in 1960,[1] and one of these cases had EEG abnormalities without a detailed description. Fisher and Adams, who used the term of TGA for the first time in 1964 to describe typical amnesia,[2] found EEG abnormalities in five out of thirteen patients and bilateral focal spikes or sharp waves in two out of five patients. Since then, the EEG abnormality rate in TGA has been reported to be up to 76.9%[3] but some studies found no EEG abnormalities.[4–7] The exact etiology and mechanism of TGA are still unknown,[8–11] but epilepsy is thought to be a possible mechanism for TGA like ischemia, migraine, and venous insufficiency. TGA has often been focused on DWI or SPECT images during the acute episode. EEG results in patients with TGA are inconsistent and there are few systematic large-scale studies. In this study, we consecutively collected EEGs of total 217 patients with TGA and analyzed the frequency and characteristics of EEG abnormalities in patients with TGA.

## 2. Methods

### 2.1. Subjects

We retrospectively collected medical records of patients who had visited the emergency room or the outpatient clinic of our hospital from 2003 to 2011 and had been diagnosed as having TGA according to the Hodges & Warlow criteria.[5] Anterograde amnesia

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was witnessed by an observer and focal neurological or epileptic signs were not detected. We excluded the patients in whom the EEG was obtained after 4 weeks from the TGA attack. We compared the EEG findings with magnetic resonance imaging (MRI) and single photon emission computed tomography (SPECT) results. We counted the number of recurrences recorded in the notes before admission and in the outpatient clinic after discharge, although these data were not collected prospectively. The study protocol was reviewed and approved by the Seoul National University Institutional Review Board, and the requirement for informed consent was waived.

### 2.2. EEG measurement

We performed digital EEG (Grass Technologies) in a copper-walled room, with the patient in a resting state with eyes closed for 15–30 min to avoid interference. We attached a total of 29 electrodes on the frontal (F3–F4), central (C3–C4), temporal (T3–T4), parietal (P3–P4), and occipital (O1–O2) areas of the patient's scalp according to the 10–20 system devised by the International Federation of Clinical Neurophysiology, and on the midline of the forehead as a reference electrode. We set the impedance of all electrodes below 5 kΩ, and used a speed of sample selection of 200 Hz/channel, 16-bit A/D transducer, 1 Hz low filter, 70 Hz high filter, and 60 Hz notch filter.

### 2.3. EEG reading and follow-up

EEG reading was performed by two other epileptologists who were blind to the purpose of the study. The final EEG reading was categorized into epileptiform discharge, focal slowing, or diffuse cerebral dysfunction. Epileptiform discharge and focal slowing were categorized into left, right, or both-sided according to the lateralization. The patients underwent an EEG test during or after the TGA attack. If the EEG result was abnormal, EEG was performed again until it was normalized within one year.

### 2.4. Brain MRI

MRI imaging included diffusion weighted imaging (DWI), T1- and T2-weighted imaging, fluid-attenuated inversion recovery imaging, conventional gradient-echo imaging in the transverse plane, and T1-weighted imaging in the sagittal plane. Each DWI session included three different sequences using different *b*-values (seconds per $mm^2$) and section thicknesses (mm): 1000/5, 2000/3, and 2000/2. MRI was performed within 24 h and again at day 3 post-onset with the same imaging parameters.[12,13] We defined a positive finding on brain MRI as a lesion corresponding to a dot-like high signal lesion(s) in the hippocampus on DWI.

### 2.5. Brain SPECT

SPECT images were acquired for 30–35 min after intravenous injection of 30 mCi $^{99m}$Tc-ethylcysteine dimer. We defined a positive finding on brain SPECT as a lesion corresponding to decreased blood flow in the mesial temporal lobe.

### 2.6. Statistical analysis

We analyzed statistical data (*t*-test, Chi-square test, McNemar test) of the normal EEG group versus the abnormal EEG group by using SPSS 18.0 (IBM Corp., Armonk, NY, USA). We set the level for a statistically significant effect at a *p*-value below 0.05.

## 3. Results

We collected data of 217 patients who were clinically diagnosed as having TGA. One hundred and seventy-four EEGs were obtained during or immediately after the TGA episode, and 43 patients refused to undergo an EEG test. Because 21 patients, in whom the EEG was obtained after 4 weeks from the TGA attack, were excluded, EEGs of 153 patients were included in the final analysis. One hundred eighteen (77.1%) patients had normal EEGs. Thirty-five (22.9%) patients had EEG abnormalities and 26 (17.0%) out of these 35 patients had epileptiform discharges. Follow-up of abnormal EEGs was performed in 23 out of 35 patients for a period of one year. EEG tests performed after four weeks from the TGA attack revealed left temporal spikes or sharp waves in one of the 21 patients, which spontaneously disappeared within a month (Fig. 1).

We did not observe any significant gender or age difference between the normal EEG group and the abnormal EEG group. The average time interval between the attack and the EEG recording was $3.8 \pm 6.0$ days: $4.0 \pm 6.3$ days in the normal EEG group and $2.9 \pm 4.6$ days in the abnormal EEG group. The time interval between the TGA attack and the EEG recording was less in the abnormal EEG group, but the difference was not statistically significant. TGA recurred in 18/153 (11.8%): one TGA attack in thirteen patients, two TGA attacks in three patients, and three TGA attacks in two patients. TGA recurred in 12/118 (10.2%) of the normal EEG group and in 6/35 (17.1%) of the abnormal EEG group. However, the difference in recurrence between the two groups was not statistically significant (Table 1). Six patients with both recurring TGA attack and abnormal EEG included four patients with epileptiform discharges on the left side only, one patient with epileptiform discharges on both sides, and one patient with diffuse cerebral dysfunction. Three out of these six patients underwent follow-up EEG, and the EEG results were as follows: epileptiform discharges on the left side were normalized after 2 months, epileptiform discharges on the left side were converted to diffuse cerebral dysfunction after 6 months, and epileptiform discharges on both sides were normalized after 40 days.

Spikes or sharp waves were detected on the left side only (17 patients) or on both sides (9 patients), but they were not detected on the right side only in any of the patients. Diffuse cerebral dysfunction was detected in six patients. Focal slowing on the left frontotemporal area was observed in two patients, and focal slowing on both frontotemporal areas was observed in one patient.

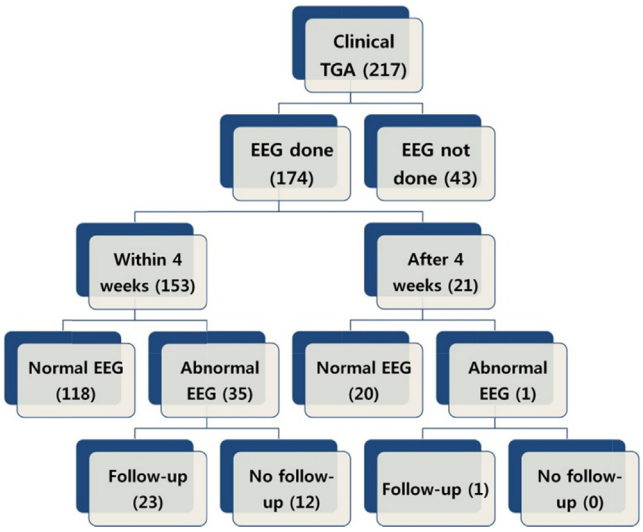


**Fig. 1.** Flow chart classifying the EEG abnormalities in TGA.

**Table 1**
Clinical characteristics of patients with TGA.

| | Normal EEG group | Abnormal EEG group | Total | *P* |
|---|---|---|---|---|
| No. of subjects (percent) | 118 (77.1%) | 35 (22.9%) | 153 (100.0%) | |
| No. of women (percent) | 80 (67.8%) | 26 (74.3%) | 106 (69.3%) | NS |
| Age (years) | 60.9 ± 8.1 | 61.0 ± 8.8 | 60.9 ± 8.2 | NS |
| Time interval between the TGA attack and the EEG recording (days) | 4.0 ± 6.3 | 2.9 ± 4.6 | 3.8 ± 6.0 | NS |
| Duration of symptoms (hours) | 5.6 ± 4.2 | 4.7 ± 3.4 | 5.4 ± 4.1 | NS |
| No. of recurrences (percent) | 12 (10.2%) | 6 (17.1%) | 18 (11.8%) | NS |

Data are expressed as mean (±standard deviation) values.
NS: not significant.
Age: no significant difference.
Sex: no significant difference.

However, focal slowing on the right frontotemporal area was not observed (Table 2).

None of the patients who underwent an EEG had a past history of epilepsy or febrile seizures. Six patients who underwent an EEG test during the ictal stage (symptomatic period) showed normal EEG results. The rate of EEG abnormalities according to the time interval between the TGA attack and the EEG recording was 24.6% within 7 days, 18.2% between 8 and 14 days, 14.3% between 22 and 28 days, and 4.8% exceeding 29 days.

Two hundred six patients underwent brain MRI, and 86 patients (41.7%) had a dot-like high signal lesion(s) in the hippocampus on DWI: 36 patients had a lesion in the left hippocampus, 34 patients had a lesion in the right hippocampus, and 16 patients had a lesion in both hippocampi. One hundred seventy-one patients underwent brain SPECT, and 119 patients (69.6%) had decreased blood flow in the mesial temporal lobe on brain SPECT: 55 patients had decreased blood flow in the left mesial temporal lobe, 42 patients had decreased blood flow in the right mesial temporal lobe, and 22 patients had decreased blood flow in both mesial temporal lobes (Table 3).

The EEG had normalized in six patients within three months, in ten within six months, and in twelve by one year. The EEG remained abnormal in eleven out of the 23 patients for a period of one year: six patients with the left epileptiform discharges, two patients with diffuse cerebral dysfunction, two patients with focal slowing, and one patient with both epileptiform discharges.

**Table 2**
EEG lateralization in patients with TGA.

| EEG abnormalities | Left side | Right side | Both sides | Total |
|---|---|---|---|---|
| Epileptiform discharge | 17 (48.6%) | 0 (0.0%) | 9 (25.7%) | 26 (74.3%) |
| Focal slowing | 2 (5.7%) | 0 (0.0%) | 1 (2.9%) | 3 (8.6%) |
| Epileptiform discharge or focal slowing | 19 (54.3%) | 0 (0.0%) | 10 (28.6%) | 29 (82.9%) |
| Diffuse cerebral dysfunction | | | 6 (17.1%) | |
| Total | | | 35 (100.0%) | |

**Table 3**
MRI and SPECT lateralization in patients with TGA.

| | Left side | Right side | Both sides | Total |
|---|---|---|---|---|
| MRI | 36 (17.5%) | 34 (16.5%) | 16 (7.8%) | 86/206 (41.7%) |
| SPECT | 55 (32.2%) | 42 (24.6%) | 22 (12.9%) | 119/171 (69.6%) |

## 4. Conclusion

We found that 26/153 (17.0%) showed epileptiform discharges within four weeks after the TGA event. The rate of EEG abnormalities in TGA has been reported from 0% to 76.9%, and spikes or sharp waves have been described only in 4.8% of 19 studies (Table 4).[1–7,14–25] Epileptiform discharge rate in EEGs of the patients was significantly higher than in the previous literature, possibly because of less time interval between the TGA attack and the EEG recording. The rate of EEG abnormalities tended to decrease as the time interval between the TGA attack and the EEG recording increased, as has been previously reported.[16,18]

Epileptiform discharge or focal slowing was detected on the left side only (54.3%) or on both sides (28.6%), but not on the right side only at all, therefore, there was a clear left dominance of EEG abnormalities in patients with TGA. There are few previous studies on lateralization of EEG in TGA. Yang suggested that lateralized abnormalities in brain functioning may be an important component of the pathophysiology of TGA, as evidenced by a significantly decreased regional cerebral blood flow on the SPECT scans in the superior temporal, precentral, and postcentral gyri with a relative left dominance.[26] Mark reported that patients with left hippocampal sclerosis differ from normal subjects in the distribution of memory-encoding activity between left and right hippocampus.[27] The lateralization of MRI and SPECT in the patients was not significant, unlike that of the EEG (Table 3).

The relationship between TGA and epilepsy is unclear. However, epilepsy has been considered as a mechanism for TGA. In this study, we found several notable EEG findings in patients with TGA. First, EEG abnormalities such as spikes or sharp waves in the initial EEG spontaneously disappeared in almost half of cases over one-year of follow-up. Continuous EEG abnormalities might reflect metabolic or diminished blood-flow disturbances.[18] Secondly, EEGs obtained during the ictal stage were normal in all of the six patients. Andrew reported a case of a middle-aged man who experienced onset of TGA while undergoing an EEG (which showed normal results).[28] Bartsch insisted that the EEG obtained during the acute TGA attack does not show abnormalities suggestive of epileptiform discharge.[9] Finally, none of the patients had a history of epilepsy, and this finding is in agreement with that in a previous study.[29] TGA is often confused with TEA (transient epileptic amnesia), which has clinical features of epilepsy such as lip-smacking or olfactory hallucinations and tends to recur frequently.[30,31] Epileptiform discharges in the EEG might indicate an epileptic process caused by damage to the medial temporal lobe. A total 26/153 (17.0%) patients and 5/18 (27.8%) patients with recurrences had epileptiform discharges in the EEG, which is in line with that reported in the literature (36.7% of patients with TEA had epileptiform EEG abnormalities[32]). Considering the retrospective

**Table 4**
EEG abnormalities in patients with TGA reported in the previous literature.

| Author (year) | Patients (No) | EEG abnormalities (percent) | Spikes or sharp waves (percent) | Generalized slowing (percent) | Focal slowing (percent) | Lateralization of spikes or sharp waves (No) |
|---|---|---|---|---|---|---|
| Poser and Ziegler (1960)[1] | 7 | 1 (14.3%) | 1 (14.3%) | 0 (0.0%) | 0 (0.0%) | ? |
| Fisher and Adams (1964)[2] | 13 | 5 (38.5%) | 2 (15.4%) | 2 (15.4%) | 1 (7.7%) | Both (2) |
| Jaffe and Bender (1966)[14] | 27 | 1 (3.7%) | 0 (0.0%) | 0 (0.0%) | 1 (3.7%) | Left (1) |
| Heathfield (1973)[15] | 16 | 7 (43.8%) | 1 (6.3%) | 4 (25.0%) | 2 (12.5%) | Left (1) |
| Mathew and Meyer (1974)[3] | 13 | 10 (76.9%) | 1 (7.7%) | 1(7.7%) | 8 (61.5%) | Both (1) |
| Rowan and Protass (1979)[16] | 10 | 4 (40.0%) | 2 (20.0%) | 2 (20.0%) | 0 (0.0%) | Left (1), right (1) |
| Shuping (1980)[17] | 35 | 11 (31.4%) | 0 (0.0%) | 6 (17.1%) | 5 (14.3%) | |
| Logar (1981)[18,a] | 30 | 18 (60.0%) | 17 (56.7%) | 1 (3.3%) | 0 (0.0%) | ? |
| Fisher (1982)[19] | 42 | 13 (31.0%) | 3 (7.1%) | 10 (23.8%) | 0 (0.0%) | ? |
| Cattaino (1984)[20] | 30 | 7 (23.3%) | 0 (0.0%) | 1 (3.3%) | 6 (20.0%) | |
| Crowell (1984)[4] | 12 | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | |
| Kushner and Hauser (1985)[21] | 18 | 4 (16.7%) | 1 (5.6%) | 1 (5.6%) | 2 (11.1%) | Right (1) |
| Miller (1987)[22] | 120 | 47 (39.2%) | 1 (0.8%) | 13 (10.8%) | 33 (27.5%) | Left (1) |
| Jacome (1989)[23] | 47 | 17 (36.0%) | 5 (10.6%) | 7 (14.9%) | 5 (10.6%) | Left (4), both (1) |
| Hodges and Warlow (1990)[5] | 8 | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | |
| Lampl (2004)[6] | 16 | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | |
| Quinette (2006)[24] | 106 | 21 (20.0%) | 0 (0.0%) | 21 (20.0%) | 0 (0.0%) | |
| Agosti (2008)[25] | 130 | 11 (8.5%) | 0 (0.0%) | 0 (0.0%) | 11 (8.5%) | |
| Auyeng (2011)[7] | 27 | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | |
| Total | 707 (100.0%) | 177 (25.0%) | 34 (4.8%) | 69 (9.8%) | 74 (10.5%) | Left (8), both (4), right (2) |

*Poser and Ziegler*: Of the seven patients reported by Poser and Ziegler, six patients had normal EEGs.
*Fisher and Adams*: EEG abnormalities were found in five out of thirteen patients and bilateral focal spikes or sharp waves in two out of five patients.
*Jaffe and Bender*: Occasional bursts of irregular 3–5 c/s activity in temporal derivations on the left side alone or bilaterally, but more on the left side than the right side
*Healthfield*: Resolving left posterior temporal theta and sharp wave abnormality in serial EEGs recorded during the six months following four attacks of transient amnesia, each of which lasted about 3 h. All of the attacks occurred in a two-month period. The EEG abnormality was considered to be compatible with infarction in the distribution of a posterior temporal branch of the left posterior cerebral artery.
*Mathew*: In ten patients, the EEG was considered abnormal. Bitemporal and bioccipital slow waves, often paroxysmal in nature, were the most common abnormalities. They were often asymmetrical with left temporal areas showing more prominent slow waves in four of six cases. In one patient, an EEG recording at 12 h after the TGA attack showed bitemporal sharp wave activity which eventually disappeared. None of these patients showed spike activity or well-defined epileptiform activity in any of the serial EEGs performed.
*Rowan and Protass*: Routine tracings were normal in six patients, and abnormal in four patients. Two patients demonstrated a small number of spikes or sharp-wave discharges in one or the other temporal region. Recording from nasopharyngeal electrodes was performed in seven patients. Five recordings showed independent discharges at or near the nasopharyngeal electrodes, usually predominating on one side. Two NP recordings were normal. Of the five patients with nasopharyngeal spike discharges, the routine EEGs were normal in two patients. Two patients showed intermittent bitemporal slowing in the theta or delta ranges, and one patient showed very rare temporal spikes.
*Shuping*: Diffuse slowing (6), focal slowing (5).
*Logar*: The EEG findings from 30 patients with TGA of vascular origin are described. A pathological result was noted in 18 cases, of which 17 patients had predominantly focal changes. Twelve EEG's were normal.
*Fisher*: Mildly abnormal (10), clearly abnormal because of temporal slow or sharp wave activity (3)
*Cattaino*: Bitemporal slow wave (4), left temporal slow wave (2), diffuse slow wave (1)
*Crowell*: EEGs were normal in all of the cases.
*Kushner and Hauser*: Right temporal small sharp spikes (1), left asymmetrical α depression (1), intermittent rhythmic slowing (generalized 1, left temporal 1).
*Miller*: Normal (73), minimal abnormality (32), moderate abnormality (8), other abnormality (7): other abnormality included SREDA (subclinical rhythmic electrographic discharges of adults) (2), alpha asymmetry (1), focal abnormality unrelated to TGA (3), epileptiform abnormality of left temporal sharp wave (1).
*Jacome*: Diffuse (7) focal (7), paroxysmal specific (5) paroxysmal non-specific (8), sharp wave discharges, single or in combination, were considered paroxysmal specific.
*Hodges and Warlow*: Routine EEG recordings performed after the presenting TGA episode with a subsequent diagnosis of epilepsy were normal in all of the cases (8). EEGs performed after the presenting attacks (non-TGA patients who developed epilepsy) were abnormal in five patients, but only two patients showed clear-cut epileptic activity (runs of bitemporal spikes or sharp waves), and the other patients had non-specific focal slow waves.
*Lampl*: EEG was normal in all of the 16 patients.
*Quinette*: One hundred and six EEGs were performed during or after the episode. Eighty-five (80) EEGs were unremarkable. The remaining EEGs revealed minor abnormalities but with no epileptic features.
*Agosti*: At EEG recording, 11 out of 13 TGA-b (TGA patients with brain lesions) patients showed frontotemporal mild slowing of a specific pattern, bilateral or lateralized, without critical features. No EEG abnormality was found in TGA-p (primary TGA patients).
*Auyeng*: The EEG showed no epileptiform discharges in any of the 27 patients with TGA.
[a] The authors think that the 'predominantly focal changes' (17/30) in the paper presented by Logar might not necessarily indicate epileptiform discharges because they were very frequent than those reported in the other papers.

nature of this study, clinical data related to the patients with epileptiform EEGs may be incomplete, and it is possible that some of these patients may have in fact suffered from temporal lobe epilepsy, in particular TEA, rather than TGA.

This is the largest consecutive EEG study performed in patients with TGA at one center. The limitations of this study need to be mentioned. First, the time interval between the TGA attack and the EEG recording was not constant. Secondly, follow-up was not systematic and the follow-up period was relatively brief. Finally, the presence of electrophysiological abnormalities in TGA is likely to be underestimated because of the relative insensitivity of EEG to medial temporal lobe pathology. However, this study is valuable because it suggests left dominance, which has not been reported in previous studies of TGA. More studies are needed on EEG lateralization in TGA.

## Conflict of interest

None declared.

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