

Naming difficulties in alexia with agraphia for *kanji* after a left posterior inferior temporal lesion

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Abstract

The case is described of a patient with alexia and agraphia for *kanji*, and severe anomia after a subcortical haemorrhage in the left posterior inferior temporal area. Magnetic resonance imaging at four months after onset showed a lesion in the inferior temporal and fusiform gyri, extending from the temporo-occipital junction toward the anterior third of the temporal lobe. Comparison with other reported cases of alexia with agraphia and anomia made it clear that when accompanied by severe anomia, the lesions extended either forward to the anterior part of the middle temporal gyrus or medially to the parahippocampal gyrus. It is suggested that the disconnection of association fibres between the parahippocampal, fusiform, middle, and inferior temporal gyri, especially between the parahippocampal gyrus and the other temporal gyri, or the cortical damage to the posterior part of these gyri is essential for the production of anomia.

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Since Iwata¹ first described a case of alexia with agraphia only for *kanji* (Japanese morphograms) due to a left posterior inferior temporal lesion, there have been many similar case reports stressing the importance of this area in the reading and writing of *kanji*.²⁻¹⁷ Alexia with agraphia for *kanji* due to a left posterior inferior temporal lesion has two main features. Firstly, it is often accompanied, to a greater or lesser degree, by naming difficulties (anomia). Little is known as to why anomia is caused by this lesion. Secondly, in some patients *kana* reading is more impaired than *kanji* reading, though *kanji* writing disturbance is always worse and persists longer than *kana* writing. For this reason it is called pure agraphia for *kanji* by some workers.^{2,15}

In this paper we describe a Japanese man with alexia and agraphia for *kanji* accompanied by severe naming difficulties due to a left posterior inferior temporal lesion, and discuss the mechanism underlying the production of anomia by comparing this patient with reported cases.

Case report

Clinical details of the patient during the acute phase have been described elsewhere.¹⁷ The

patient, a 71-year-old right-handed man, was educated to senior high school level. He had no history of serious illness. On 22 June 1991 he suddenly felt a slight left-sided headache. He then noticed that he could not remember the names of objects or his family and could not read newspapers or write anything but his own name. He was admitted to the Japanese Red Cross Medical Center on 22 June with a diagnosis of cerebrovascular disease.

He was fully conscious on admission. Cranial nerve function, motor system, cutaneous and deep sensation, and coordination were all intact except for a mild hearing loss in both ears. There was no visual field defect. Blood chemistry was normal. Audiometry showed mild hearing loss in both ears for speech discrimination (right ear, 75%; left ear, 80%; 80-85 dB).

NEUROPSYCHOLOGICAL EXAMINATION

The patient's verbal IQ with the Wechsler adult intelligence scale—revised (WAIS-R) was 80, and his performance IQ was 86.

The Western aphasia battery (WAB; Japanese edition¹⁸ (table 1)) at two months showed that his spontaneous speech was fluent with preserved prosody. He showed marked word finding difficulty and overused demonstrative pronouns, but there were few paraphasias. He made three correct responses out of 20 in object naming and, given phonemic cues, made another four correct responses. Circumlocution was observed in most of the incorrect responses. He could explain how to use objects and could point to

Table 1 Scores for the Western aphasia battery

	August 1991	October 1992
Spontaneous speech		
Information content	7/10	9/10
Fluency	8/10	9/10
Naming total	2-4/10	5-7/10
Object naming	13/60	31/60
Repetition	8-6/10	9-6/10
Comprehension total	7/10	9-45/10
Yes/no questions	51/60	54/60
Auditory word recognition	41/60	59/60
Sequential commands	48/80	76/80
Reading total	3-9/10	8-05/10
Comprehension of sentences	4/40	40/40
Reading commands	7-5/20	13/20
<i>Kanji</i> to object matching	3/3	3/3
<i>Kana</i> to object matching	3/3	3/3
<i>Kanji</i> to picture matching	3/3	3/3
<i>Kana</i> to picture matching	3/3	3/3
Writing total	6-55/10	7-5/10
Copying	10/10	10/10
<i>Kanji</i> writing to dictation	0/6	2/6
<i>Kana</i> writing to dictation	4/6	5/6
Sentence writing to dictation	7/10	9/10
Praxis total	9-8/10	9-8/10
Construction total	8/10	7-9/10

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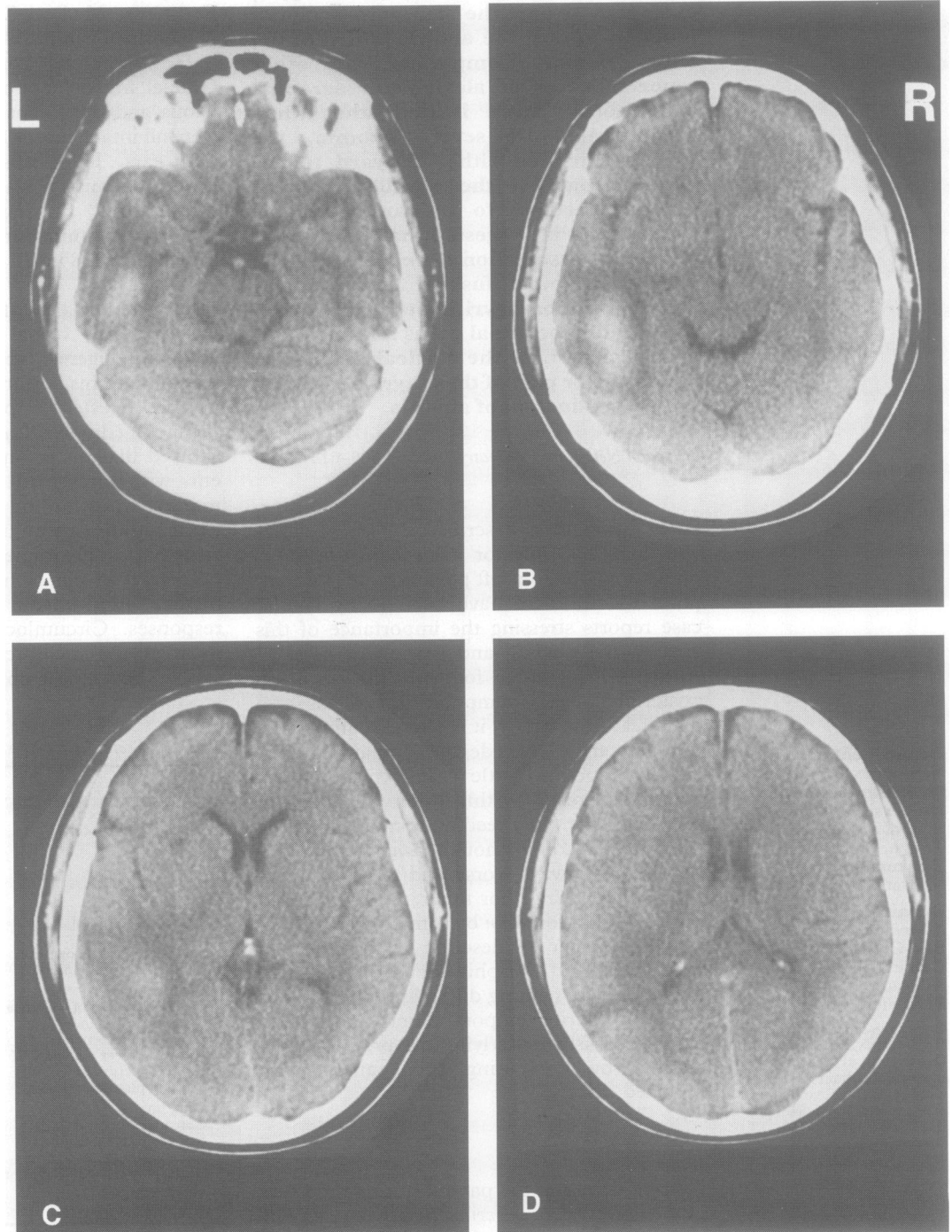
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objects on command fairly well (17/20). Repetition was almost normal. Auditory comprehension was slightly impaired. *Kanji* reading was poor, which resulted in a low score for comprehension of written language, whereas *kana* reading was generally good, though with a few phonemic paraphasias (this difference was made clear in the reading of sentences including both *kanji* and *kana*, but was not reflected in the WAB score). In *kanji*-to-object matching he could point to the appropriate objects correctly, though he could only read one of six *kanji* word items. This implies that semantics were preserved even when he could not read *kanji* words, characteristic of deep dyslexia.¹⁹ Also, in writing to dictation, *kanji* was inferior to *kana*. Agraphia of *kanji* due to word recall difficulty and phonemic paraphasias of *kana* were noted. The patient's

speech profile was rated as one of anomic aphasia and alexia with agraphia, relatively selective for *kanji*. He did not show apraxia, a construction disturbance, nor colour agnosia.

To evaluate the extent of the reading and writing difficulties for *kanji* compared with *kana* more precisely, at three months the patient was given reading aloud and writing to dictation tasks involving 100 *kanji* and the corresponding *kana* (readings of *kanji*), all of which are taught in the first three years of primary school. The results showed that in both reading (correct response in *kanji*, 20%; *kana*, 75%; $p < 0.001$ on the two-tailed Fisher's exact method) and writing to dictation (correct response in *kanji*, 4%; *kana*, 59%; $p < 0.001$ on the two-tailed Fisher's exact method), *kana* function was significantly superior to *kanji* function. Most of the errors

Figure 1 CT scan on admission showing a large high density area in the left temporal subcortical structure extending to the anterior part of the temporal lobe. The left is the patient's left side.



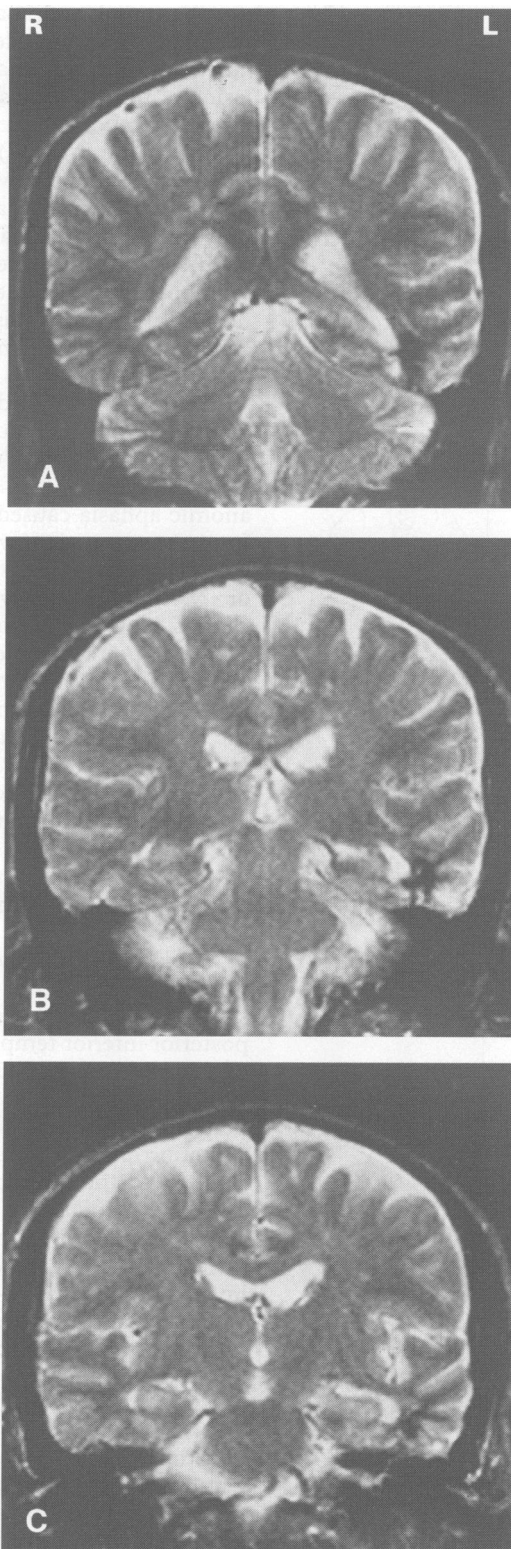
comprised no response in *kanji* tasks and phonemic paralexias or paraphrasias in *kana* tasks. A similar tendency was observed on re-examination 15 months after onset with significant improvement (reading: correct response in *kanji*, 46%, $p < 0.005$; *kana*, 91%, $p < 0.05$; writing to dictation: correct response in *kanji*, 12%, $p < 0.005$; *kana*, 77%, $p < 0.005$, on the McNemar test). On the whole, he could read in *kana* more than 90% of the items he read correctly in *kanji*, as well as in writing. He could also read almost

all *kanji* or *kana* he could write in the re-examination, though there were some *kanji* or *kana* which he could not read but could write, or vice versa, in the first test (number of items with both correct reading and correct writing were two for *kanji* and 45 for *kana*).

Severe naming difficulties and alexia with agraphia for *kanji* still persisted at 16 months, though there was some improvement (table 1).

CT on admission showed a high density area in the left temporal subcortical structure, thus the patient was diagnosed as having a cerebral haemorrhage (fig 1). There was also a low density area suggesting an old infarction in the anterolateral portion of the left putamen. MRI at the same time confirmed these lesions and there was no other change in intensity affecting the left thalamus. In MRI at four months the lesion was evident in the cortico-subcortical tissue of the left inferior temporal and fusiform gyri, probably affecting the inferior longitudinal fasciculus and extending from the temporo-occipital junction toward the anterior third of the temporal lobe (fig 2). Single photon emission CT with iodoamphetamine labelled with iodine-123 (^{123}I -IMP-SPECT) at four months showed decreased blood flow in the lower and medial parts of the left temporal lobe, including the hippocampal region, whereas the regional cerebral blood flow in the left basal ganglia and thalamus was preserved.

Figure 2 MRI T2-weighted coronal view (0.5 T; TR = 2500 ms; TE = 120 ms) through the atrium of the lateral ventricle (A) and hippocampus (B, C) at four months. The right is the patient's left side. A low intensity area suggesting haemosiderin deposition is present in the cortico-subcortical tissue of the left fusiform, inferior temporal gyri from the occipital to the anterior third of the temporal lobe.



Discussion

The patient presented with alexia with agraphia for *kanji* and severe naming difficulties after a subcortical haemorrhage of the left posterior inferior temporal area. Anomia can also be observed in a thalamic lesion.²⁰ The haematoma and its surrounding oedema in the acute phase could have some effect on the left thalamus, but MRI at this time showed that there was no change in intensity in the thalamus. In addition, the patient's severe anomia was still present at 12 months. Therefore, we cannot attribute anomia to the acute dysfunction of the thalamus.

The patient had an old infarction in the anterolateral portion of the left putamen. A basal ganglia lesion can also produce atypical aphasia.²¹ In such a case, however, the lesion affects more widespread areas including the head of the caudate nucleus, the putamen, and the internal capsule. The patient's putamenal lesion was too small to cause aphasia, and the regional cerebral blood flow of the left basal ganglia was preserved. Furthermore, the patient did not show anomia until admission, thus, the old infarction in the putamen cannot cause the anomia.

There have been more than 20 case reports of alexia with agraphia due to a left posterior inferior temporal lesion. Severe naming difficulties often occurred in those patients in whom *kanji* reading was more impaired, whereas in patients with more impaired *kana* reading naming difficulties were, if present, only slight. Thus to correlate the lesion with

Table 2 Classification of alexia with agraphia for kanji with significant difference in reading between kanji and kana. Values are mean (%) scores

Patient group*	Naming	Reading		Lesion†
		kana	kanji	
Group A				
Shiota <i>et al</i> ³	sl.	20/36(56)	20/24(83)	PITs‡
Kawamura ²				
Patient TN	sl.	263/416(63)	402/416(92)	Fu, inf T, T-O‡‡
Mochizuki and Ohtomo ⁶	N	8/48(17)	74/100(74)	Inf T, O‡‡
Group B				
Oka <i>et al</i> ⁹	16/20(80%)	752/800(94)	674/800(84)	Inf T, Fu‡‡
Kawahata <i>et al</i> ¹⁰	18/20(90%)	24/25(96)	2/25(8)	PIT‡‡
Group C				
Imamura <i>et al</i> ¹²	0/20(0%)	28/30(93)	9/30(30)	HC, OR, inf T-Os‡‡
Notoya <i>et al</i> ¹³	11/20(55%)	20/20(100)	12/20(60)	HC, inf T, Fu‡
Soma <i>et al</i> ¹⁵	33/50(66%)	219/221(99)	201/221(91)	Mid T, inf T‡‡
Patient 2				
Notoya <i>et al</i> ¹⁶	7/20(35%)	20/20(100)	14/20(70)	Mid T, inf T, T-O‡
This patient	3/20(15%)	75/100(75)	20/100(20)	Inf T, Fu‡‡

*Group A, more impaired kana reading; group B, more impaired kanji reading with slight or no anomia; and group C, more impaired kanji reading with profound anomia. †Based on the authors' evaluation. ‡p < 0.05, ‡‡p < 0.01 on the two-tailed Fisher's exact method between kanji reading and kana reading. Abbreviations: PIT = posterior inferior temporal; inf = inferior; sl. = slightly disturbed; N = normal; T = temporal; O = occipital; s = subcortical; Fu = fusiform; HC = hippocampus; OR = optic radiation.

the symptom we divided the reported cases of alexia with agraphia due to a posterior inferior temporal lesion into three groups: group A, patients with more impaired kana reading³⁻⁷; group B, patients with more impaired kanji reading and slight or no anomia (80% or more correct in confrontation naming)⁸⁻¹¹; and group C, patients with more impaired kanji

reading and severe anomia (less than 80% correct in confrontation naming).¹²⁻¹⁷

We determined which was more impaired, kanji or kana, in each patient based on the scores for the individual kanji and kana reading test and the authors' evaluation. We then chose patients with significantly more impaired kana or kanji reading from each group (p < 0.05 or less on the Fisher's exact method; table 2) and traced their lesion on CT axial slices. Figure 3 shows the superimposed lesion images of each group. We can see from this figure that in group A patients the lesion extends from the left posterior inferior temporal area backward to the lateral occipital gyrus. In alexia with agraphia for kanji without profound anomia (group B) the lesion is mostly localised in the left posterior inferior temporal area (almost corresponding to Brodmann area 37). When severe anomia accompanies alexia with agraphia for kanji (group C), the lesion affects the left posterior inferior temporal area, extending either forward as far as the anterior part of the middle temporal gyrus or medially to the parahippocampal gyrus. As this inference is based on a small number of patients, we need further evidence to confirm it.

This patient showed severe anomia, but he could explain how to use objects and point to objects almost always correctly on command. Such a symptom is characteristic of pure anomic aphasia caused by a localised lesion.²² In contrast, a patient with anomia with a cortical degenerative disease such as Pick's disease or Mesulam's slowly progressive aphasia²³ cannot explain how to use objects, recognise them, or point to objects on command.²⁴ As this patient did not improve with tactile cues and could copy and match figures to sample, the naming disturbance was not caused by visual agnosia. This difference is probably because in anomia caused by a localised lesion, lexical information is still preserved in the cortex but its use is impaired, whereas in anomia observed in a cortical degenerative disease lexico-semantic information itself is affected.

Comparison of the lesions of group B patients with those of group C patients showed that the localised damage to the left posterior inferior temporal area (posterior part of the middle and inferior temporal area and fusiform gyri) did not produce anomia and that severe anomia occurred only when the posterior inferior temporal lesion extended forward to the anterior part of the temporal lobe or medially to the parahippocampal gyrus. We identified the extent of the lesion in our patient in more detail on MRI coronal images. It affected the point at which fibres from the fusiform, middle, and inferior temporal gyri seemed to converge, extending from the temporo-occipital junction to the anterior third of the temporal lobe. It was continuous along almost the whole length of the parahippocampal gyrus as a result, separating the parahippocampal gyrus from the other temporal gyri (fig 2). A similar lesion was reported in a patient with severe anomia and alexia with

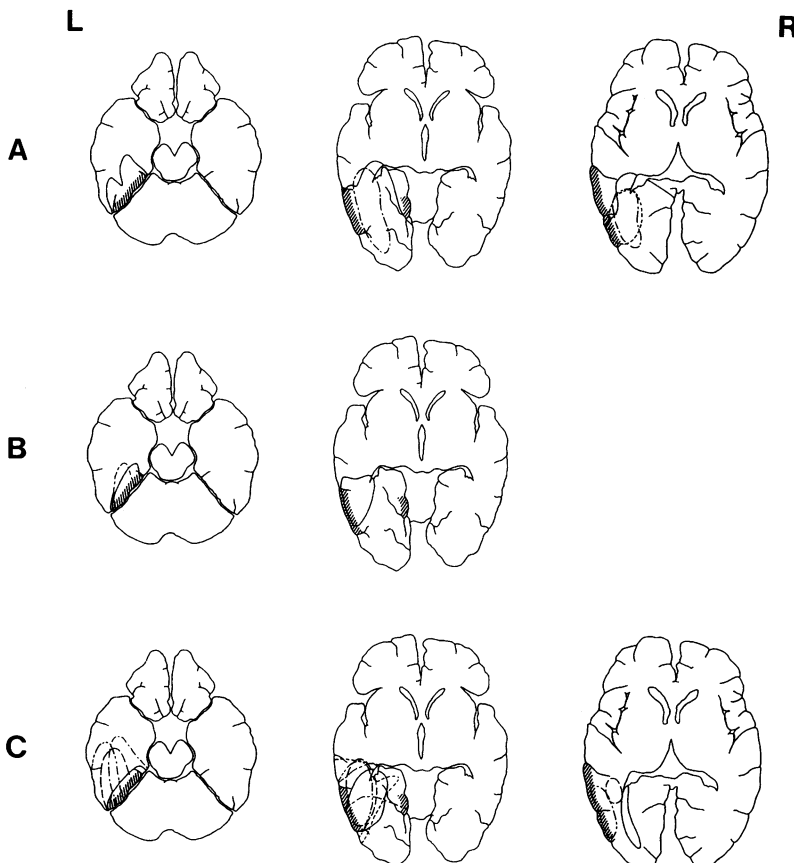


Figure 3 Superimposed schematic CT images of alexia with agraphia due to a left posterior inferior temporal lesion. The left is the patients' left side. Areas with oblique lines denote the left posterior inferior temporal area (almost corresponding to Brodmann's area 37). (A) Patients with significantly more impaired kana reading.^{3,5,6} (B) Patients with significantly more impaired kanji reading and slight or no anomia.^{9,10} (C) Patients with significantly more impaired kanji reading and severe anomia, including our patient.^{12,13,15-17} The lesion in alexia with agraphia for kanji is usually localised in the left posterior inferior temporal area (B). When kana reading impairment is more pronounced, the lesion affects the lateral occipital area (A). If it accompanies severe anomia, the lesion extends forward to the anterior part of the temporal lobe or medially to the parahippocampal gyrus (C).

agraphia for *kanji* due to cerebral infarction.¹⁴ These findings suggest that anomia occurs when the association fibres are disconnected between the parahippocampal, fusiform, middle, and inferior temporal gyri, or when the lesion affects the parahippocampal gyrus. The patient of Mochizuki and Ohtomo⁶ (group A, delineated by solid lines in fig 3A) does not seem to fit our view in that there was no anomia though the lesion extended forward in the temporal lobe. In this patient, however, the damage was, according to the MRI finding, restricted to the occipital lobe and the inferior temporal gyrus, whereas the lingual, fusiform, parahippocampal, superior and middle temporal gyri were spared. This patient shows that anomia does not appear if the posterior part of the parahippocampal gyrus is intact.

In our patient the parahippocampal gyrus and hippocampus were apparently spared, but SPECT showed the decreased blood flow in the left inferior and medial temporal area, including the hippocampal region. This implies that the lesion had some effect on the hippocampal memory system.

In monkeys the parahippocampal gyrus and perirhinal cortex have afferent and efferent connections with several association areas in the frontal, temporal, and parietal lobes.²⁵ Given these findings, it is assumed that the disconnection of association fibres between the parahippocampal, fusiform, middle, and inferior temporal gyri, especially between the parahippocampal gyrus and the other temporal gyri, or the cortical damage to the posterior part of these gyri is essential for the production of pure anomic aphasia.

We found in a review of published work¹⁷ that the more severe the naming difficulties were, the more impaired *kanji* writing seemed to be, and this was dependent on the extent of the lesion. The combination of anomia and alexia with agraphia for *kanji* is observed only in Japan. The same lesion may result in anomia and reading and writing difficulties of irregular words in English-speaking countries.

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