

Alexia caused by a fusiform or posterior inferior temporal lesion

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Abstract

We evaluated the alexia and agraphia of three patients with different lesions using Japanese *kanji* (morphograms) and *kana* (phonograms) and made a lesion-to-symptom analysis. Patient 1 (pure alexia for both *kanji* and *kana* and minor agraphia for *kanji* after a fusiform lesion) made more paragraphic errors for *kanji*, whereas patient 2 (alexia with agraphia for *kanji* after a posterior inferior temporal lesion) showed severe reading and writing disturbances and more agraphic errors for *kanji*. Brodmann Area 37 was affected in both patients, but in patient 2 the lesion was located lateral to that in patient 1. Patient 3 showed agraphia without alexia after restricted lesion to the angular gyrus. We believe that pure alexia (patient 1) results from a disconnection between the medial fusiform gyrus and posterior inferior temporal area (the lateral fusiform and inferior temporal gyri), whereas alexia with agraphia for *kanji* (patient 2), corresponding to lexical agraphia in Western countries, results from damage to the posterior inferior temporal area, in which whole-word images of words are thought to be stored. Furthermore, restricted lesion in the angular gyrus (patient 3) does not produce alexia; the alexic symptom of “angular” alexia with agraphia may be the result of damage to the adjacent lateral occipital gyri. © 2000 Elsevier Science B.V. All rights reserved.

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1. Introduction

Alexia with agraphia caused by a lesion in the angular gyrus and pure alexia (alexia without agraphia) caused by lesion to the medial occipital gyri and the splenium were first described by Dejerine [1,2]. He thought that the visual images of letters and words were stored in the angular gyrus and that any lesion which interrupted visual input to the angular gyrus may cause alexia [1,3]. Geschwind [4] also stressed the importance of the angular gyrus in reading. In his view, the angular gyrus constitutes a cross-

modal association area that turns written language into spoken language and vice versa.

In addition to the classical (spleno–occipital) type of Dejerine’s pure alexia, a type of pure alexia subsequent to damage to the subcortical white matter [5–10] or to the cortical region [6,11–13] in the left temporo–occipital junction or the occipital lobe has been reported. Greenblatt [14] called this non-classical type [15] of pure alexia “preangular alexia”, and the alexia caused by subcortical lesion to the angular gyrus “subangular alexia”. He considered the subcortical white matter ventral to the angular gyrus to be an important structure in subangular alexia. The features of reading impairment in the non-classical type of pure alexia have only rarely been described in neurological studies [8,10].

On the other hand, because of the existence of two

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distinct writing systems in the Japanese; i.e., *kanji* (morphograms) and *kana* (phonograms), Japanese alexic patients can show dissociative disturbances in reading between *kanji* and *kana*. It is generally believed that patients with dyslexia with dysgraphia from a lesion in the angular gyrus can read *kanji* better than *kana*, whereas patients with dyslexia with dysgraphia from a posterior inferior temporal lesion can read *kana* better than *kanji* [16–18].

Kanji are graphically complicated and have meaning as well as two kinds of reading: *on*-reading which conveys the phonetic value; and *kun*-reading which conveys the meaning (e.g., 水 [sui, mizu], water). In contrast, *kana* are graphically simple and have one definite phonetic value and no intrinsic meaning (e.g., さ [sa]). We argued that *kanji* are an extreme form of irregular words (those that do not obey English spelling rules) in that a whole-word image is more concerned in reading and writing, whereas *kana* words (or non-words) are an extreme form of regular words (or pseudowords) in that grapheme-to-phoneme correspondence is almost complete [19]. Thus the relative difference of reading disturbances between regular words and irregular words in European languages can present as the definite dissociation of reading difficulties between *kanji* and *kana* in Japanese, which may help to understand the mechanism of reading in general.

We report three patients with reading and writing disturbances; pure alexia after a fusiform lesion, alexia with agraphia after a posterior inferior temporal lesion, and agraphia after an angular lesion (Fig. 1 shows the anatomical relations among gyri discussed in this paper). We examined the reading and writing abilities of these patients using *kanji* and *kana* materials and correlated the results

with the patients' lesions. The purpose of this study is to show a neuroanatomical basis for reading applicable not only to Japanese but to languages in general.

2. Case reports

2.1. Patient 1

The patient's acute phase observations are reported elsewhere [20].

In May 1995, a 73-year-old, right-handed man, who was a university graduate and engineer, experienced floating sensation in the head. He consulted a physician and underwent an MRI examination in September of that year. Cerebral infarction in the left medial occipital gyri was detected (Fig. 2). At this time, his reading ability was normal, and he was not aware of any visual field defect. In April 1996, he suddenly found that he could not read characters, and the next day realized that he could not read any Japanese *kana* symbol, whereas he could read numerals and some *kanji* symbols. He still could write *kana* symbols with ease. He entered a hospital in Yokohama, where a CT scan showed a fresh infarction in the left occipital lobe. Because of persistent reading impairment, in May 1996 he was admitted to the Department of Neurology of the Japanese Red Cross Medical Center, Tokyo.

The neurological examination on admission, showed right upper homonymous quadrantanopsia (later confirmed by Goldmann perimetry), reading and writing impairment, and minimal anomia. He read slowly and painfully even words that he had just written. He could read three of five

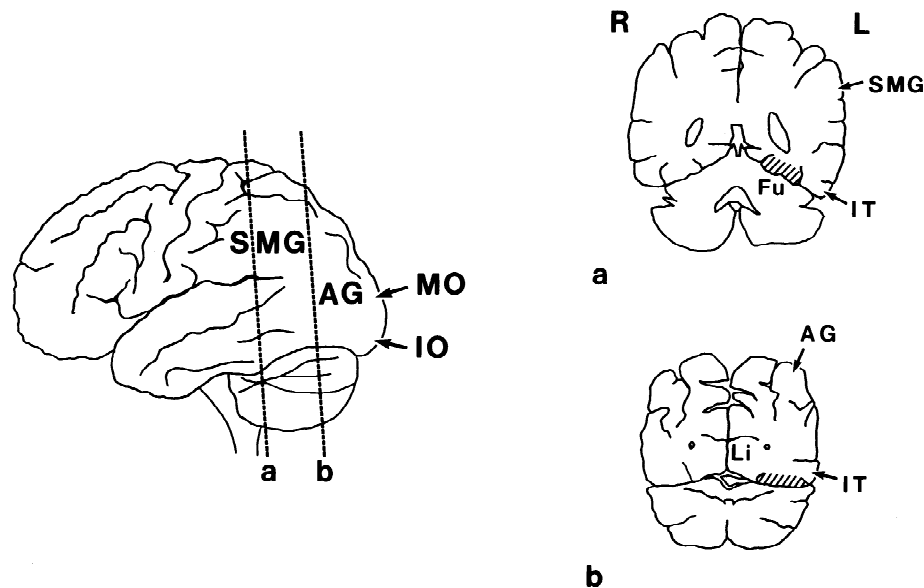


Fig. 1. Anatomical relations among gyri in the occipital and temporal lobes (Drawn from Damasio [21]). Nomenclature is based on Dejerine [22] and Talairach and Tournoux [23]. Coronal planes through the fusiform and inferior temporal gyri (Area 37; (a)) and the angular gyrus (b) are shown. An area with oblique lines denotes the fusiform gyrus. Middle occipital gyrus is located just posterior to the angular gyrus. Abbreviations. SMG, Supramarginal gyrus; AG, Angular gyrus; MO, Middle occipital gyrus; IO, Inferior occipital gyrus; Fu, Fusiform gyrus; IT, Inferior temporal gyrus; Li, Lingual gyrus.

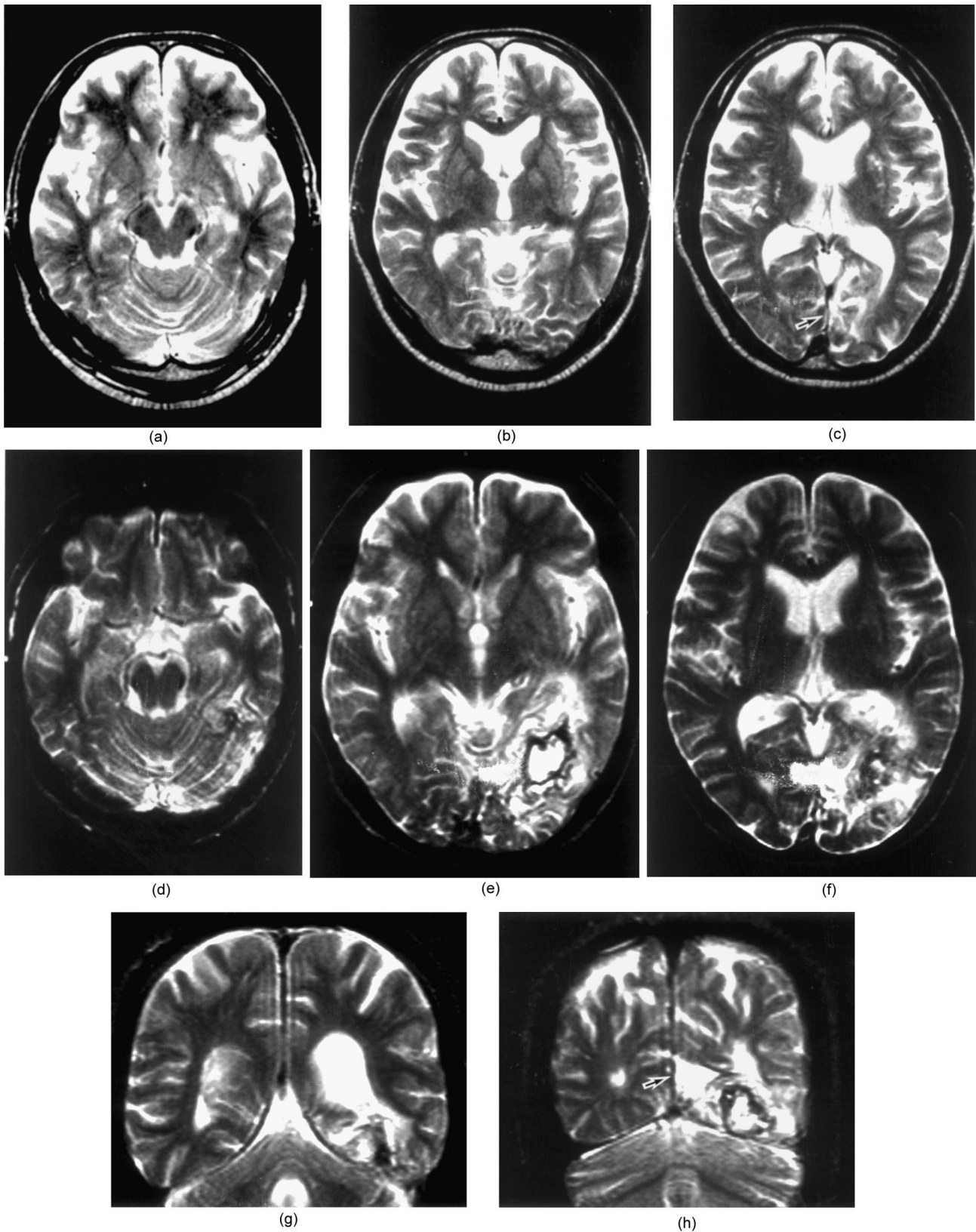


Fig. 2. MRI T2-weighted axial images of patient 1, 4 months after the first stroke (September 1995; a–c), and axial and coronal images 45 days after the second stroke (June 1996; d–h). The left side of each image is the patient's right side (also in Figs. 3 and 4). In the first stroke, the lesion is restricted to the left lingual gyrus and part of the cuneate gyrus (a–c; arrow, 0.5 tesla, TR/TE=3000/100, also seen in Fig. 3; arrow). In the second stroke, a high-intensity area with ringed low intensity inside, indicative of hemorrhagic infarction, is located mainly in the fusiform gyrus but extends laterally to the inferior temporal gyrus and posteriorly to the inferior occipital gyrus and subcortical area of the angular gyrus (d–h; 0.5 tesla, TR/TE=2500/100).

kana characters. Whenever he had difficulty reading, he used kinesthetic reading (i.e., tracing characters with the fingers). He made some verbal paraphasia in *kanji* when writing to dictation.

The Western aphasia battery (WAB; Japanese edition) administered 1 month after on-set showed fluent spontaneous speech without paraphasia (Table 1). Auditory comprehension, repetition, and naming were all perfect. The patient could not, however, read some *kanji* and *kana* symbols, including one- or two-character *kanji* nouns, one-character *kanji* verbs with *kana* suffixes, and *kana* function words, resulting in a poor performance for sentence comprehension. Kinesthetic reading was not always successful. His writing was nearly normal, but there was impaired recall and verbal paraphasia of some *kanji* symbols. In all, his language profile was rated as pure alexia (strictly speaking, alexia with minor agraphia for *kanji*). The digit span forward score was four. The copying of sentences, drawing of figures, and color naming were normal. The Panel D15 test showed intact color vision.

Forty days after on-set, his reading impairment gradually began to improve, and he became able to read newspapers, although with some effort.

2.1.1. Neuroradiological examination

Lesion location was determined from T1- and T2-weighted axial and coronal MRI images with a center-to-center slice distance of 7–12 mm. Each gyrus was identified using the atlases of Damasio [21], Dejerine [22], and Talairach and Tournoux [23]. T2-weighted images at the time of the first stroke showed a high-intensity area in the left medial occipital gyri, including the lingual gyrus

and part of the cuneate gyrus (Fig. 2a–c). T1- and T2-weighted axial images 45 days after the second attack showed an abnormal intensity lesion with low-(T2) and high-(T1) intensity rings in the left occipito-temporal and occipital areas, indicative of a hemorrhagic infarction (Fig. 2d–f). T2-weighted coronal images showed that this lesion involved the left lingual (Brodmann Areas 17 and 18), fusiform (Areas 18, 19, and 37), parahippocampal, inferior temporal, and inferior lateral occipital gyri, probably affecting the optic radiation and the inferior longitudinal fasciculus, and that the lesion extended posteriorly and upward to the subcortical white matter under the angular gyrus (Fig. 2g and h). Single photon emission computed tomography done with iodoamphetamine labeled with ^{123}I (^{123}I -IMP-SPECT) showed hypoperfusion throughout the left occipital lobe, in the posterior part of the middle and inferior temporal gyri, and in the angular gyrus.

2.2. Patient 2

This patient also has been described elsewhere [24]. Here we summarize the case report and give follow-up data

In June 1991, a 71-year-old, right-handed man, a senior high school graduate, suddenly realized that he could not remember the names of objects or his family and could not read newspapers or write anything but his own name. A subcortical hemorrhage in the left posterior inferior temporal area was diagnosed by CT. Neuropsychological examination 3 months after on-set showed alexia with agraphia that was preferentially disturbed for *kanji*, and severe anomia. The patient read character-by-character sentences written in *kanji* and *kana* in the WAB test. He could not read *kanji* words and showed phonemic paralexia for *kana* verbs and verbal paralexia for *kanji* verbs with *kana* suffixes. Of note is that he could point to objects correctly, even though he could not read aloud the names of the objects written in *kanji* (understanding without phonology), characteristic of deep dyslexia [25]. He produced four semantic paralexia errors in a 100-*kanji* character reading test (described later). The digit span forward score was five. Copying of sentences, drawing of figures, and color naming were intact. Because the patient could explain how to use objects and could correctly point to them on command almost all the time, visual agnosia was ruled out as a cause of anomia. MRI done 4 months after on-set showed the lesion involved the left lateral fusiform and inferior temporal gyri (Area 37) and extended subcortically to the anterior third of the temporal lobe. Follow-up MRI 6 years later showed the same lesion with additional periventricular hyperintensity (Fig. 3; see also the figures in Sakurai et al. [24]). Alexia with agraphia for *kanji* and anomia persisted at the time of the re-evaluation, 5 years after on-set, but character-by-character reading had disappeared (Table 1). In the WAB test of sentence reading, he still could not read some *kanji* words, sometimes

Table 1
Neuropsychological test scores for the patients 1 and 2

Time after on-set (months)	1	64
	Patient 1	Patient 2
WAIS-R		
Verbal IQ	113	–
Performance IQ	101	–
WAB		
Spontaneous speech		
Information content	10/10	9/10
Fluency	10/10	9/10
Naming total	10/10	3.5/10
Repetition	10/10	9.4/10
Comprehension total	10/10	8.9/10
Reading total	4.55/10	7.12/10
Comprehension of sentences	6/40	30/40
Spelled <i>kanji</i> character recognition	2/6	0/6
Oral spelling of <i>kanji</i> characters	0/6	0/6
Writing total	9.8/10	6.95/10
Copying	10/10	10/10
<i>Kanji</i> writing from dictation	5.5/6	0/6
<i>Kana</i> writing from dictation	6/6	4.5/6

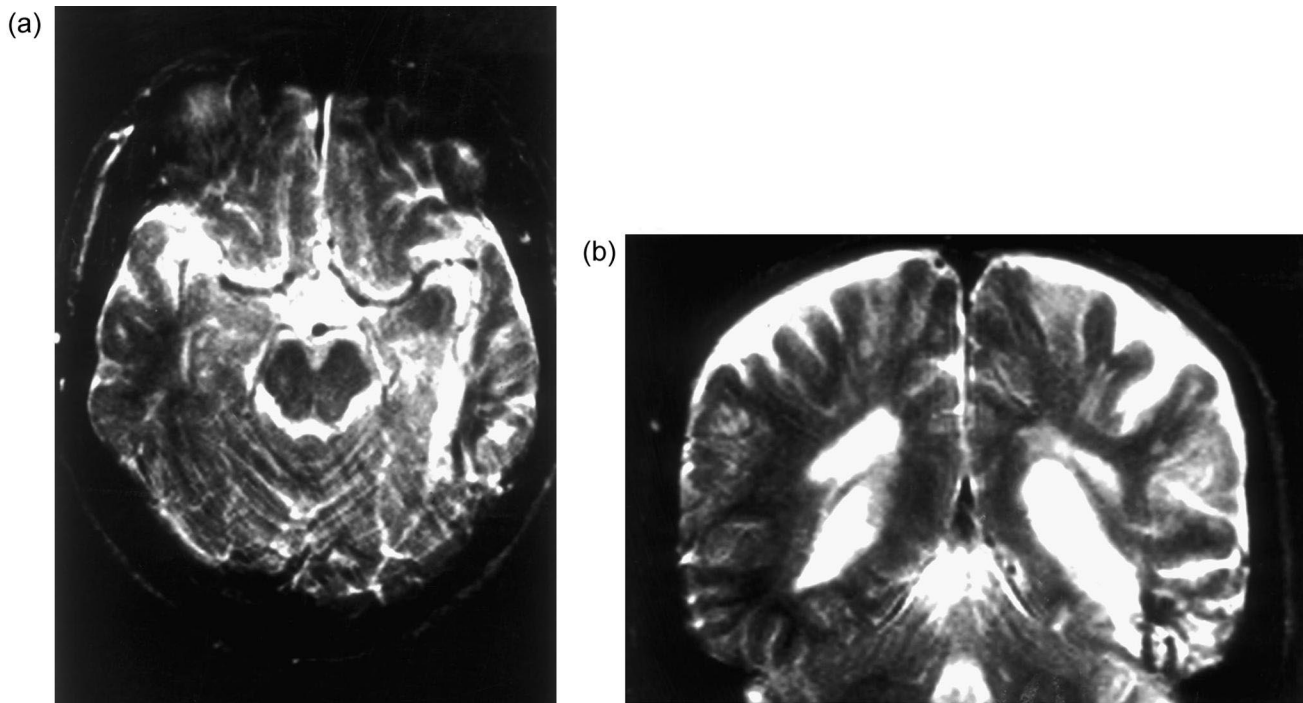


Fig. 3. MRI T2-weighted axial (a) and coronal (b) images of patient 2, 6 years after on-set. A low-intensity area, suggestive of hemosiderin deposition, with medial high-intensity is present in the left inferior temporal gyrus and lateral side of the fusiform gyrus and extends forward in the temporal lobe. Note that this lesion is located more laterally than the lesion in patient 1 (0.5 tesla, TR/TE=2500/120).

omitted one *kana* or *kanji* character in a word and sometimes added a *kana* functor. Anomia and reading scores for both *kanji* and *kana* were almost unchanged, whereas his *kana* writing was worse than at re-examination made 3 years before (from 77% to 58% on the 100-word writing test). In addition, he had difficulty reading single *kana* characters, and misread seven of 46 *kana* characters.

2.3. Patient 3

We have reported the patient in another context (patient 6) [26]. A short summary is given here.

In June 1996, a 61-year-old, right-handed man, a university graduate and president of an advertising company, found that he could not remember the names of well-known persons and things. A cerebral infarction in the left parietal lobe was diagnosed by CT. The neuropsychological examination showed Gerstmann syndrome (agraphia, acalculia, finger agnosia, and left-right disorientation) and slight anomia that converged to agraphia, acalculia, and minimal anomia 2 months after on-set. The digit span forward score was four. Copying of sentences, drawing of figures, and color naming were intact. MRI done 40 days after on-set showed the lesion involved the left angular gyrus with the subcortical white matter and the inferior part of the supramarginal gyrus, and that it

extended to the posterior horn of the lateral ventricle (Fig. 4; see also the figures in Sakurai et al. [26]).

3. Methods and results

3.1. Special tests

To evaluate the patients' reading and writing ability quantitatively, we administered a special test (Table 2), the same as that used in a previous study [24]. The task was to read aloud and write from dictation 100 *kanji* (one-character words) and their corresponding *kana* (readings of *kanji*, or *kana* transcription of *kanji* words), all of which are taught in the first 3 years of Japanese primary school. The *kanji* and *kana* materials consisted of an equal number of concrete nouns, abstract nouns, adjectives, and verbs. Because *kanji* characters usually have *on*- and *kun*-readings (described in the introduction), and the *on*-readings contain many homophones, we required the patients to identify *kanji* characters by the *kun*-reading, the transcription of which was adopted in the *kana* reading test.

Table 2 shows scores and times required for reading for each patient. Errors were classified into non-response, visual, phonological, semantic, etc. (Table 3).

Results show that for patient 1, reading improved to the normal range, although the act of reading took a long time.

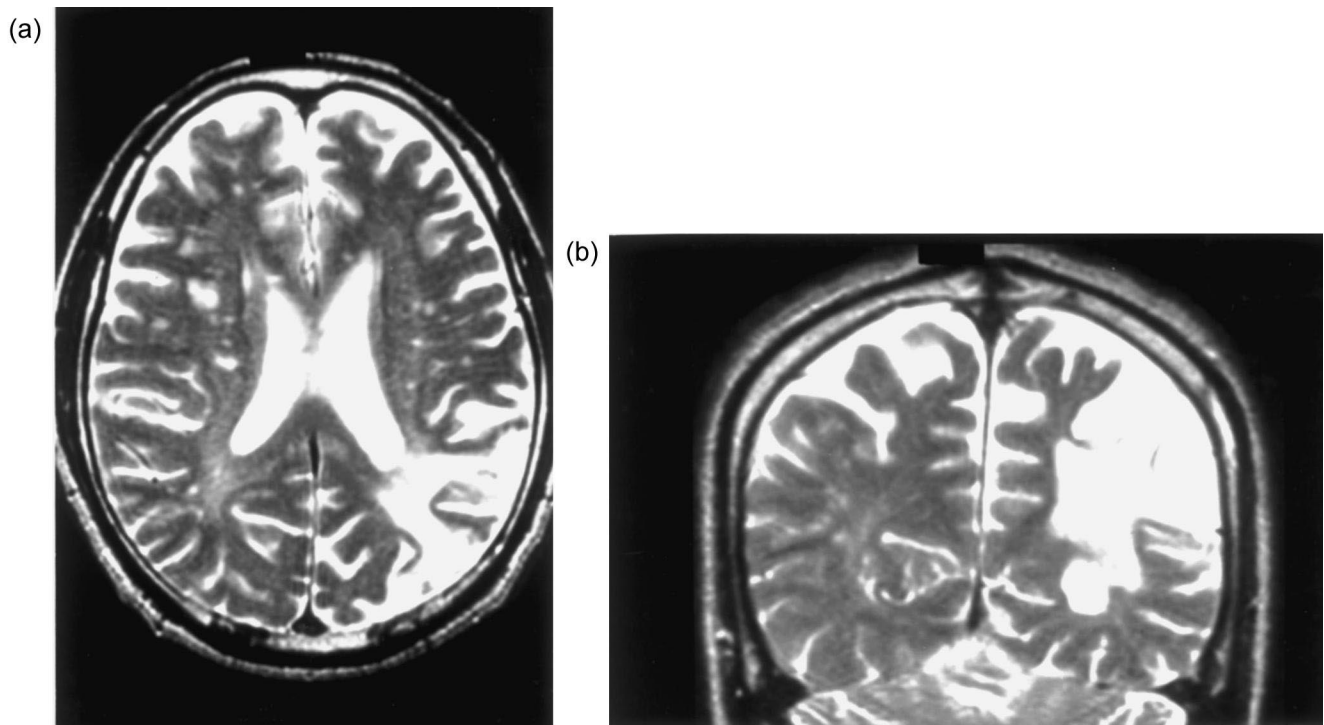


Fig. 4. MRI T2-weighted axial (a) and coronal (b) images of patient 3, 40 days after on-set. The lesion is restricted to the cortico-subcortical structures of the left angular gyrus and to part of the inferior supramarginal gyrus (1.5 tesla, TR/TE=3000/102).

Errors consisted of two concrete nouns and one verb for *kanji* and two abstract nouns and one adjective for *kana*. He actively used the strategy of kinesthetic facilitation and succeeded in reading most of the time. In contrast, his writing scores were lower in the *kanji* task than in the *kana* task ($P < 0.0001$ by Fisher's exact method). Half of the errors were visually similar to the target (Table 4). He could write all of the three *kanji* correctly that he could not read.

Patient 2 showed severe reading and writing impairment of *kanji* with relative sparing of *kana* reading and writing (reading, $P < 0.0001$; writing, $P < 0.0001$ between *kanji* and *kana* by Fisher's exact method). Kinesthetic reading was not always successful. Most *kanji* reading and writing

errors were the non-response type (impaired word recall). All the circumlocution and semantic errors in *kanji* reading were associated semantically with the correct answer: e.g., "white" (incorrect) for "color" (correct), and "something to eat" for "drink". In *kanji* reading, the number of correct responses for concrete nouns was 13/25, abstract nouns 13/25, adjectives 11/25, and verbs 8/25. In addition, this patient could not write *kanji* words that he could not read.

Patient 3 read both *kanji* and *kana* very well, although he spent more time reading than the control subjects (time was not measured). In contrast, he had lower scores for writing *kanji* words than *kana* words ($P < 0.0001$ by Fisher's exact method) because of impaired word recall

Table 2

Special tests for reading and writing (100-single character *kanji* and the corresponding *kana*)

Patient	1	2	3	Controls ($n=11$) ^a	
Symptom	Pure alexia (recovery phase)	Alexia with agraphia	Agraphia	mean score (S.D.)	
Time after on-set (months)	2	61–64	2	Mean time for reading	
<i>Kanji</i> reading	97 (7 min; 5/8) ^b	45 (22 min; 7/59)	100 ^c	99.6 (1.2)	1 min 32 s
<i>Kana</i> reading	97 (5 min)	95 (4 min)	99	99.6 (0.5)	1 min 13 s
<i>Kanji</i> writing	77 (35 min)	6 (12 min)	29	95.9 (3.0)	10 min 11 s
<i>Kana</i> writing	100 (12 min)	58 (15 min)	97	99.3 (0.9)	8 min 7 s

^a All normal volunteers (ten men and one woman, ages 61–78, mean 68-years-old) gave their informed consent.

^b Values are % correct (and time spent in the task; correct responses with kinesthetic reading). The fraction denotes the number of correct responses versus the total attempts at kinesthetic reading: e.g., 5/8 means that five of the eight kinesthetic reading trials were correct.

^c For patient 3, time for reading was not measured.

Table 3

Types of reading errors in 100-character *kanji* or 100-word *kana* test (values are the number of errors)

Patient	1	2	3
<i>Kanji</i> (n=100)			
Non-response	3	34	
On response ^a		6	
Circumlocution		5	
Visual ^a		3	
Semantic ^a		2	
Unrelated ^a		5	
Total errors	3	55	
<i>Kana</i> (n=100)			
Phonological ^b	2	3	1
Phonological and/or visual ^b	1	1	
Unrelated ^b		1	
Total errors	3	5	1

^a On response: the patient read the *kanji* with its *on*-reading (the reading that conveys its phonetic value according to the Chinese pronunciation) but could not give its *kun*-reading (the reading that conveys its meaning in the Japanese language). Visual response: changing the *kanji* into the reading of another *kanji* that has a visual resemblance to the correct answer; e.g., 聞 ([kiku], hear)→開 ([hiraku], open). Semantic errors: those associated semantically with the target word; e.g., 色 ([iro], color)→白 ([shiroi], white). Unrelated response: the changing of a *kanji* into another *kanji* that has no visual or phonological similarity to the correct answer.

^b Phonological response: one or more characters in a *kana* word were substituted for other *kana* (phonemic paralexia). Phonological and/or visual response: changing one character into another visually similar to the target character; e.g., あおい ([aoi], blue)→おおい ([ooi], numerous). Unrelated response: the changing of a *kana* word into another *kana* word that has no visual or phonological similarity; e.g., はやい ([hayai], fast) → あおい ([aoi], blue).

Table 4

Types of writing errors in the 100-character *kanji* and 100-word *kana* tests (values are number of errors)

Patient	1	2	3
<i>Kanji</i> (n=100)			
Non-response	5	90	67
Constructional ^a			2
Visual ^a	9		1
Visual and/or semantic ^a	1		
Phonological ^a		1	1
Semantic ^a	2	1	
Unrelated ^a	4	2	
Neologism	2		
Total errors	23	94	71
<i>Kana</i> (n=100)			
Constructional ^b			1
Phonological ^b		31	2
Phonological and/or visual ^b		9	
Unrelated ^b		2	
Total errors		42	3

^a Constructional response: the omission or addition of a component of a *kanji*; e.g., 鳥 ([tori], bird)→鳥. Visual response: substitution of another *kanji* that has a visual resemblance to the correct answer; e.g., 音 ([oto], sound) →音 ([mukashi], old times). Some visual errors were semantically associated with the target (visual and/or semantic); e.g., 顔 ([kao], face)→頭 ([atama], head). Phonological response: substitution of another *kanji* with a phonetic value similar to the correct answer; e.g., 数 ([kazu], digit)→活 ([katsu], active). Semantic errors: those associated semantically with the target word; e.g., 速 ([hayai], fast)→急 ([isogu], hurry). Unrelated response: substitution of another *kanji* that has no visual or phonological similarity to the correct answer.

^b Constructional response: the omission or addition of a component of a *kana*. Phonological response: one or more characters of a *kana* word were substituted for other *kana* (phonemic paraphasia). Phonological and/or visual: changing one character into another visually similar to the target character; e.g., くろい ([kuroi], black)→くらい ([kurai], dark). Unrelated response: substitution of another *kana* word with no visual or phonological similarity; e.g., ふゆ ([huyu], winter)→うみ ([umi], sea).

and made a few phonological errors in writing *kana* words (Table 4).

4. Discussion

Patient 1 presented with pure alexia (alexia for both *kanji* and *kana* and minor agraphia for *kanji*). Anatomically, he is classified as having non-classical type preangular alexia [15,27]. In Japan, pure alexia is frequently accompanied by slight agraphia for *kanji* [28], but why this agraphia coexists with pure alexia remains unknown. For our patient, it was because the lesion involved the adjacent lateral side of the fusiform gyrus and the inferior temporal gyrus (posterior inferior temporal area); the type of lesion which causes alexia with agraphia for *kanji* [24,29], or pure agraphia for *kanji* [30]. His condition was semiologically similar to what is called group A alexia with agraphia produced by a posterior inferior temporal lesion, in which *kana* character (or word) reading is more impaired than *kanji* character (or word) reading [24].

Patient 2 had alexia with agraphia for *kanji* accompanied by severe anomia caused by a posterior inferior temporal lesion (group C alexia with agraphia, as described in Sakurai et al. [24]). Alexia with agraphia for *kanji* corresponds to the category of lexical agraphia [30,31] in European countries, in which irregular word spelling is selectively impaired. As this symptom is thought to derive from deficient visual images of words, we originally designated it “morphologic” agraphia, but because “morphologic” has a specific usage in linguistics, hereafter we use “orthographic” agraphia to refer to this state. He could write his own name at the on-set, either because the whole-word image of his name was so robust, or because he did it almost automatically (with little access to the orthography).

Patient 3 first showed Gerstmann syndrome with anomia, with agraphia, acalculia, and minimal anomia remaining. A lesion in the angular gyrus is known to cause alexia with agraphia, but he did not show alexia throughout the clinical course (discussed later). Agraphia was more pronounced for *kanji* words than for *kana* words.

Next we correlate the alexic symptoms of the three patients with their lesions and discuss kinesthetic reading in alexia.

4.1. Neuroanatomical basis of alexia

According to Greenblatt’s classification [27], patients 1 and 2 showed preangular alexia. Their lesions were similar to spleno–occipital type alexia without hemianopsia [7], except that there was no damage to the splenium. Patient 1 had his first stroke in the left medial occipital area (mainly the lingual gyrus). At that time, right upper quadrantanopsia may have occurred, even though he was not aware of it, and visual information arriving in the left lingual gyrus

may not have been processed further. Early visual analysis in the lingual gyrus may have been completely compensated for by the right homologous area. His second attack was within the occipital lobe. This time the lesion was located mainly in the left fusiform gyrus and extended to the inferior temporal and lateral occipital gyri, including the optic radiation. Neurological and neuropsychological examinations showed right upper homonymous quadrantanopsia and pure alexia. Some patients with pure alexia also have a lesion restricted to the fusiform gyrus, which lesion sometimes extends to the lateral occipital gyri (Henderson et al. [8]; Kawamura [15], Case 2; Buxbaum et al. [32]). This preangular type of pure alexia is interpreted as being within the framework of disconnection theory [7], but the classic disconnection theory cannot account for alexia with agraphia caused by a posterior inferior temporal lesion (patient 2). The fact that visual information from the right homologous area did not compensate for the damage to the left fusiform gyrus suggests there is functional localization for letters or words in the fusiform gyrus.

Moreover, the main affected site was Brodmann Area 37 in both patients, but the lesion in patient 2 with alexia with agraphia was located more laterally in the fusiform and inferior temporal gyri. Compared with patient 1, patient 2 had severer reading and writing disturbances for *kanji*, and made more agraphic errors (non-responses due to impaired recall) for *kanji*, whereas patient 1 made paragraphic errors (visual and semantic responses) as well as agraphic errors for *kanji* (Table 4).

These findings suggest that both the fusiform gyrus and the inferior temporal gyrus constitute a contiguous (but different in detail) functional localization in reading and writing, visual images of individual *kana* being stored medially in the fusiform gyrus, and visual images of individual *kanji* and whole-word images of both *kana* and *kanji* words stored more laterally in the fusiform and the inferior temporal gyri. If so, the reason why patient 1 could write *kanji* that he could not read, although with some paragraphic errors, was because the visual images of the *kanji* remained relatively intact in the lateral fusiform and the inferior temporal gyri (posterior inferior temporal area), and he used them in writing. Thus pure alexia (patient 1) results from a disconnection between the medial fusiform gyrus and the posterior inferior temporal area, in which access to orthography for words is interrupted, whereas alexia with agraphia for *kanji* (patient 2) results from damage to the posterior inferior temporal area, in which orthography per se is impaired. This hypothesis contradicts the disconnection theory, in which the angular gyrus is regarded as the storage site [1,3] or cross-modal association area [4].

Furthermore, it should be noted that patient 3 with the angular lesion showed not alexia but agraphia throughout the clinical course, although he did have minimal difficulty in reading *kanji* words in the acute phase [26]. His lesion

was restricted to the angular gyrus and the inferior part of the supramarginal gyrus. The findings for this patient suggest that restricted lesion of the angular gyrus causes not alexia, but agraphia. The concept of alexia with agraphia caused by an angular lesion has been established [33–36], but even in Dejerine's original patient there was a lesion extending posteriorly to the lateral occipital gyri [1]. Some subsequent studies on angular alexia were of lesions affecting the occipital lobe [37]. CT and MRI studies from the 1970s also showed involvement of the lateral occipital gyri just posterior to the angular gyrus in alexia with agraphia [18,38]. The accompanying inability to copy words [1,39] suggests the existence of a more widespread lesion that involves the parieto-occipital and occipital areas. When a lesion is confined to the angular gyrus, the alexic symptom disappears within a few months [40], or apparently does not appear as in our patient 3. Yamadori [41] examined five cases of "angular" alexia with agraphia and concluded that clinical data were still insufficient to attribute alexia with agraphia to damage to the restricted area of the angular gyrus. It is our view that involvement of the lateral occipital gyri (more precisely, the middle occipital gyrus just posterior to the angular gyrus) is essential for the production of "angular" alexia. Kleist [42] first ascribed the alexic symptom in alexia with agraphia to lesion extension from the angular gyrus to the middle occipital gyrus.

This view is supported by our activation studies with positron emission tomography (PET) [43,44] (for the location of individual region-of-interest, see Sakurai et al. [45]). In the reading aloud tasks for *kanji* words, *kana* words and *kana* non-words, as compared with the fixation task, we found that the lateral occipital gyri posterior to the angular gyrus were activated in the *kana* word and non-word tasks, whereas the angular gyrus was deactivated in all the tasks. We reanalyzed our data using Statistical Parametric Mapping [46] and confirmed the above findings [47]. This fact may explain why *kana* reading is more impaired in "angular" alexia with agraphia (described in Section 1). Deactivation of the angular gyrus in reading also has been reported in Western countries [48].

4.2. Kinesthetic reading in alexia

Kinesthetic reading (shreibendes Lesen in the German literature [49]) is often used by Japanese patients with pure alexia as a strategy for overcoming reading difficulty. Kinesthetic reading clearly was less helpful for patient 2 with alexia and agraphia than for patient 1 with pure alexia. This reading strategy is known to be effective for patients with pure alexia. Although it is not clear whether kinesthetic reading is effective in alexia with agraphia for *kanji*, published reports on alexia with agraphia that refer to this strategy show that it is not helpful for reading *kanji* and *kana* together (e.g., Kawahata et al. [50]) or *kanji* reading alone (Kawamura [15]; Patient TN). This probably

is because in pure alexia the visual image of a word is mostly preserved access to it being interrupted; whereas, in alexia with agraphia for *kanji*, the visual image of the word itself is damaged, as described in Section 4.1. The fact that Brodmann Area 37 (a lesion in which causes alexia with agraphia for *kanji*) was activated in a PET study not only by word processing but by tactile reading [51] suggests this area is important in kinesthetic reading.

The above discussion is based on a small number of case and activation studies. Further studies therefore are required to determine the roles of the angular gyrus and the lateral occipital gyri in reading, and the mechanism of kinesthetic reading.

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