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# Apraxia of speech due to the left postcentral gyrus lesion

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**Key Clinical Message**
Apraxia of speech (AOS) due to a postcentral infarction differs from conventional precentral AOS with respect to phonemic errors (phoneme substitution) which are more common than phonetic errors (phoneme distortion) and preserved accent and intonation.

**Abstract**
Clinical features of apraxia of speech caused by lesions in the postcentral gyrus have not yet been elucidated. Here, we report a patient with this lesion and show how postcentral apraxia of speech differs from the hitherto known precentral apraxia of speech. A 54-year-old man developed Broca′s aphasia with apraxia of speech that resolved into pure apraxia of speech within 3 weeks following infarction of the postcentral gyrus. The diagnosis of apraxia of speech was based on the patient's effortful, slow speech and inconsistent phonetic distortions with phonemic paraphasia. The Western Aphasia Battery was used to examine the patient's speech samples. Speech was recorded using a digital voice recorder and transcribed into a narrow transcription of the International Phonetic Alphabet. The error types were categorized phonologically. The results revealed that (a) phonemic errors (vowel and consonant substitutions, also known as phonemic paraphasia) were more common than phonetic errors (vowel and consonant distortions). Similar to conduction aphasia, phonemic errors were more pronounced in confrontation naming than in repetition, accompanied by self-correction, and (b) word accent and sentence intonation were preserved, although the speech was slow. These two features are characteristic of postcentral apraxia of speech, which can be differentiated from conventional precentral apraxia of speech.

**KEYWORDS**
conduction aphasia, dysprosody, phonemic paraphasia, phonetic distortion

## 1 | INTRODUCTION

Apraxia of speech (AOS) is a neurological speech impairment characterized by reduced ability to plan or program sensorimotor commands required for directing normal speech. The cardinal features of AOS are slow speech rate, lengthened segment (prolonged vowels and consonants) and intersegment (word segregation) durations, phonetic

distortions, and error variability.[1–3] AOS can exist without clinically apparent impairments in the speech muscles for nonspeech tasks.[4] Darley et al.[5] established an AOS to distinguish it from dysarthria or aphasia. However, AOS manifests in isolation or with Broca's aphasia or dysarthria, making it difficult to diagnose symptoms and identify the responsible lesion.

The neuroanatomical association with AOS is controversial. The responsible lesion was identified in the left posterior inferior frontal gyrus[6] and in the lower portion of the precentral gyrus.[7] Recent lesion–symptom mapping studies have shown a strong association between AOS and the premotor and motor cortices[8] or the motor and somatosensory areas[9] and in particular, a restricted region of the lower precentral gyrus at the level of the inferior frontal sulcus.[10]

Here, we present a case of AOS caused by a lesion in the left postcentral gyrus, and show how AOS symptoms differ from typical AOS symptoms caused by damage to the precentral gyrus.

## 2 | CASE PRESENTATION

In March 2018, a 54-year-old right-handed native Japanese man with a history of depression experienced difficulty uttering. The patient was referred to our hospital's Department of Neurology with a diagnosis of cerebral infarction. Magnetic resonance imaging (MRI) performed 16 days after disease onset indicated a focal infarction in the postcentral gyrus that had spread subcortically to the precentral gyrus (Figure 1). A minor degree of right facial and limb hemiparesis, hypoesthesia, disturbances in deep sensation and cortical combined sensation (stereognosis,

graphesthesia, and two-point discrimination) of the right hand, and limb-kinetic apraxia of the right hand were observed on neurological examination. No buccofacial apraxia was observed. The patient had a digit span forward score of four. The Western Aphasia Battery (WAB; Japanese edition[11]) was used to evaluate language function six days after symptom onset (Table 1). The patient was cooperative and completed the test. Based on the patient's effortful slow speech rate, inconsistent phonetic distortions with phonemic paraphasia, and minimal auditory comprehension deficits, mild Broca's aphasia with AOS was diagnosed.

Writing was moderately impaired (Figure 2), and phonological agraphia of kana (Japanese phonetic writing) and lexical agraphia of kanji (Japanese morphograms) were observed. Reading and writing were further evaluated using the 100 single-character kanji and kana transcription test[12] 22 days after symptom onset (Table 1). Kanji materials were selected from those taught during the first 3 years of primary school in Japan. Scores for reading were close to normal levels, although the reading time was much longer than normal for both kanji and kana. The writing was poor and illegible, which lowered the scores for kanji and kana, in particular kanji that is graphically complicated. The deficit in writing was attributable to somesthetic dysgraphia owing to impaired cortical sensation.[13] In addition, phonological errors (kana substitution) and no response (impaired kana character recall) further lowered the score for kana (Table 1).

In 3 weeks, comprehension (8.0 → 9.3) and repetition (7.0 → 9.4) on the WAB test at 22 days after symptom onset recovered to a nearly normal range; therefore, we considered that Broca's aphasia with AOS resolved into pure
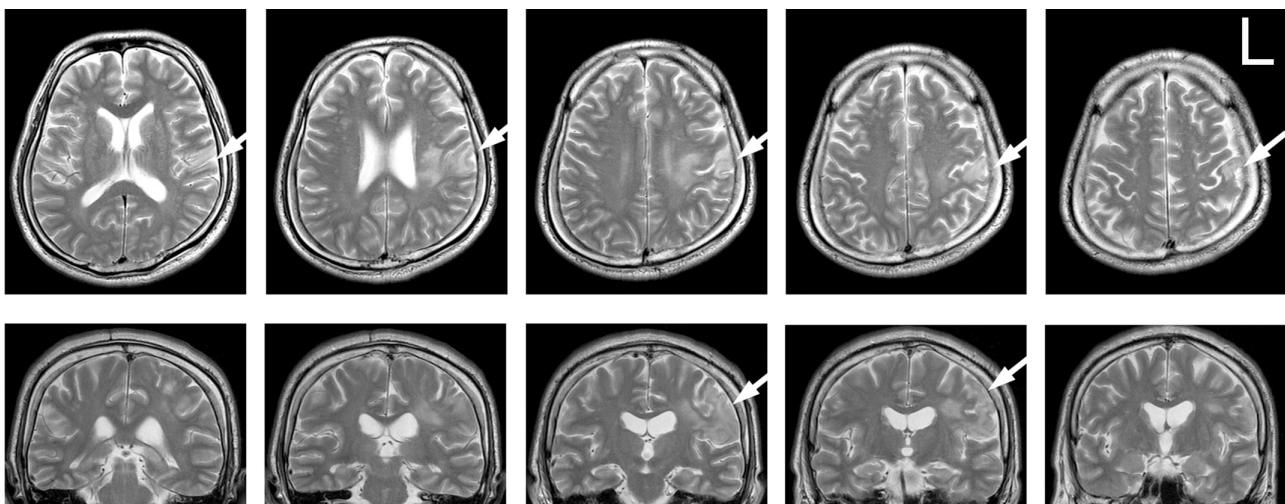


**FIGURE 1** Magnetic resonance imaging T2-weighted axial and coronal images of the patient at 16 days after disease onset. A high-intensity area was noted in the lower half of the left postcentral gyrus, which spread subcortically to the precentral gyrus (arrows).

**TABLE 1** Scores for the Western Aphasia Battery (WAB; Japanese Edition) and 100-single-character kanji and kana transcription test.

| | Patient | | Controls[a] |
|---|---|---|---|
| | Time after onset | | Mean (SD) |
| | 6 days | 22 days | |
| WAB | | | |
|   Spontaneous speech | | | |
|     Information content (/10) | 6[b] | 8[b] | 9.7 (0.6) |
|     Fluency (/10) | 5[b] | 6[b] | 10.0 (0.0) |
|   Comprehension total (/10) | 8.0[a] | 9.3[b] | 9.8 (0.1) |
|   Repetition (/10) | 7.0[b] | 9.4 | 9.87 (0.3) |
|   Naming total (/10) | 6.1[b] | 8.8 | 9.5 (0.6) |
|     Confrontation naming (/60) | 43[b] | 57 | 59.2 (2.4) |
|   Reading total (/10) | 8.9 | 9.4 | 9.5 (0.8) |
|   Writing total (/10) | 4.4[b] | 6.3[b] | 9.6 (1.0) |
|     Kanji word writing (/6) | 4.5 | 5 | 5.3 (1.5) |
|     Kana word writing (/6) | 1[b] | 4 | 5.7 (1.0) |
|   Praxis (/10) | rt. 10, lt. 10 | rt. 10, lt. 10 | 9.97 (0.1) |
|   Construction total (/10) | 9.4 | 9.5 | n.d. |
| **100 single-character kanji and kana transcription** | | | |
| | | Score, Time | |
| Kanji reading (/100) | | 99[c], 9 min 30 s[b] | 99.6 (1.2), 1 min 32 s (32 s) |
| Kana reading (/100) | | 96[b,c], 6 min 36 s[b] | 99.6 (0.5), 1 min 13 s (24 s) |
| Kanji writing (/100) | | 65[b,d], 12 min 44 s | 95.9 (3.0), 10 min 11 s (136 s) |
| Kana writing (/100) | | 55[b,e], 15 min 12 s[b] | 99.3 (0.9), 8 min 7 s (107 s) |

*Note*: In the WAB subtests, higher score denotes better performance. Upper limit is displayed in parentheses next to the specific subtest name.

Abbreviations: lt., left; n.d., not done; rt., right.

[a]Data on normal controls for the WAB subtests are retrieved from the WAB test manual (Japanese edition),[11] and data on normal controls for the 100 single-character kanji and kana transcription test are from Sakurai et al.[12]

[b]Two SDs below or over the normal mean.

[c]Kanji and kana reading errors included all phonological errors (phonemic paralexia).

[d]Kanji writing errors: Most characters were illegible because of somesthetic dysgraphia. No response (impaired character recall) was noted in two characters.

[e]Kana writing errors were phonological (one or more characters of a kana word were substituted for another kana word; $n = 28$), no response ($n = 8$), constructional (omitting or adding a kana component; $n = 4$), partial (one or more characters of a kana word were not written; $n = 2$), phonological/partial (combination of a phonological error and a partial error; $n = 2$), and semantic (changeing to another kana word semantically associated with the correct word; $n = 1$).

AOS. Phonemic paraphasia became more evident than phonetic distortions.

# 3 | METHODS AND RESULTS

## 3.1 | Neuroimaging study

We used single-photon emission computed tomography with $^{99m}$Tc-ethylcysteinate dimer ($^{99m}$Tc-ECD-SPECT) 16 days after disease onset to determine the localization and extent of hypoperfusion. SPECT data were converted into the Analyze format and normalized, smoothed, and corrected for inter-laboratory differences using a three-dimensional conversion map with the easy Z-score Imaging System version 1.1.0.[14] The corrected data were compared with the normal subject database of the same generation provided by the National Center for Neurology and Psychiatry in Tokyo ($n = 30$ for men and women aged 40–59 years). Statistical significance was evaluated using a two-sample $t$ test (uncorrected $p < 0.001$) in Statistical Parametric Mapping version 12 (https://www.fil.ion.ucl.ac.uk/spm/). The extent threshold was set to be equal to the expected voxels per cluster to avoid noise. The left anterior superior frontal gyrus, posterior middle frontal gyrus, triangular part of the inferior frontal gyrus, lower half of the postcentral gyrus, and perisylvian supramarginal gyrus showed a significant reduction in blood flow (Figure 3).

| | | | |
|---|---|---|---|
| 切手<br>kitte (stamp) | | きって<br>kitte (stamp) | |
| 時計<br>tokei (watch) | | とけい<br>tokei (watch) | |
| 新聞<br>shinbun<br>(newspaper) | | しんぶん<br>shinbun<br>(newspaper) | |
| 毛糸<br>keito (wool) | | けいと<br>keito (wool) | |
| 灰皿<br>haizara (ashtray) | | はいざら<br>haizara (ashtray) | |
| 鉛筆<br>enpitsu (pencil) | | えんぴつ<br>enpitsu (pencil) | |

**FIGURE 2** Writing two-character kanji words and kana transcriptions from dictation in the WAB test. Poor and illegible writing was evident in both kanji and kana words (somesthetic dysgraphia). Lexical kanji agraphia and phonological kana agraphia were also noted. Kanji (left column) and *hiragana* (right column, cursive form of kana) with the printed writing style are shown.

## 3.2 | Speech analysis

Spontaneous speech, repetition, and naming on the WAB were analyzed 22 days after disease onset. Speech was recorded using a digital voice recorder. A speech-language pathologist (N. M.) with 20 years of practical experience transcribed the voice samples into a narrow transcription of the International Phonetic Alphabet.

### 3.2.1 | Articulation

There were 417 syllables (or moras consisting of consonants and vowels, exceptionally with consonant-only and vowel-only) articulated in spontaneous speech, repetition, and naming, including 380 vowels and 370 consonants (Table 2). The analysis excluded interjections like "Um" and cliched phrases like "I'm sorry." Duffy's classification[4] was used to determine error types. Consonant substitution (phonemic paraphasia; S-C in Table 2), vowel prolongation (P-V), consonant distortion (D-C), and consonant-distorted substitution (e.g., [dakedo] → [r̃akedo]; Ds-C) were common types of errors. The total number of distorted substitutions (Ds) and substitutions (S) was 189. Among these, consonant errors were substantially more common than vowel errors in distorted substitutions (Ds-C 83% vs. Ds-V 17%) and substitutions (S-C 87% vs. S-V 13%). There was no

transposition (changing the sequential order of phonemes in a word) in consonant substitutions (S-C), few distorted voicing distinctions (blurring of voiced-voiceless boundaries; Dv), and no distorted addition or distorted prolongation.

There were 161 attempts to correct the incorrect articulation of 107 words spoken in the test (sum of attempts related to spontaneous speech, repetition, object naming, word fluency, sentence completion, and responsive speech in the WAB test), with 142 failed attempts. In these corrections, 68 phonemes were incorrectly articulated in the same way, 83 were incorrectly articulated again but differently, and 31 were incorrectly articulated despite being correctly articulated before rephrasing. Of note, multiple incorrect articulations were observed in specific attempts; therefore, the total incorrect articulations exceeded the 142 failed attempts.

There were 73 utterances in the speech (sum of utterances during spontaneous speech, repetition, object naming, word fluency, sentence completion, and responsive speech in the WAB test), and 17 (23.3%) had a false articulatory start and restart. While attempting to correct articulatory errors, the patient frequently grimaced, sometimes flushed, and strained his voice. There were 29 transient cessations of articulatory movements during the speech.

Further evaluations included the frequency of phonetic and phonemic errors regarding word length. The
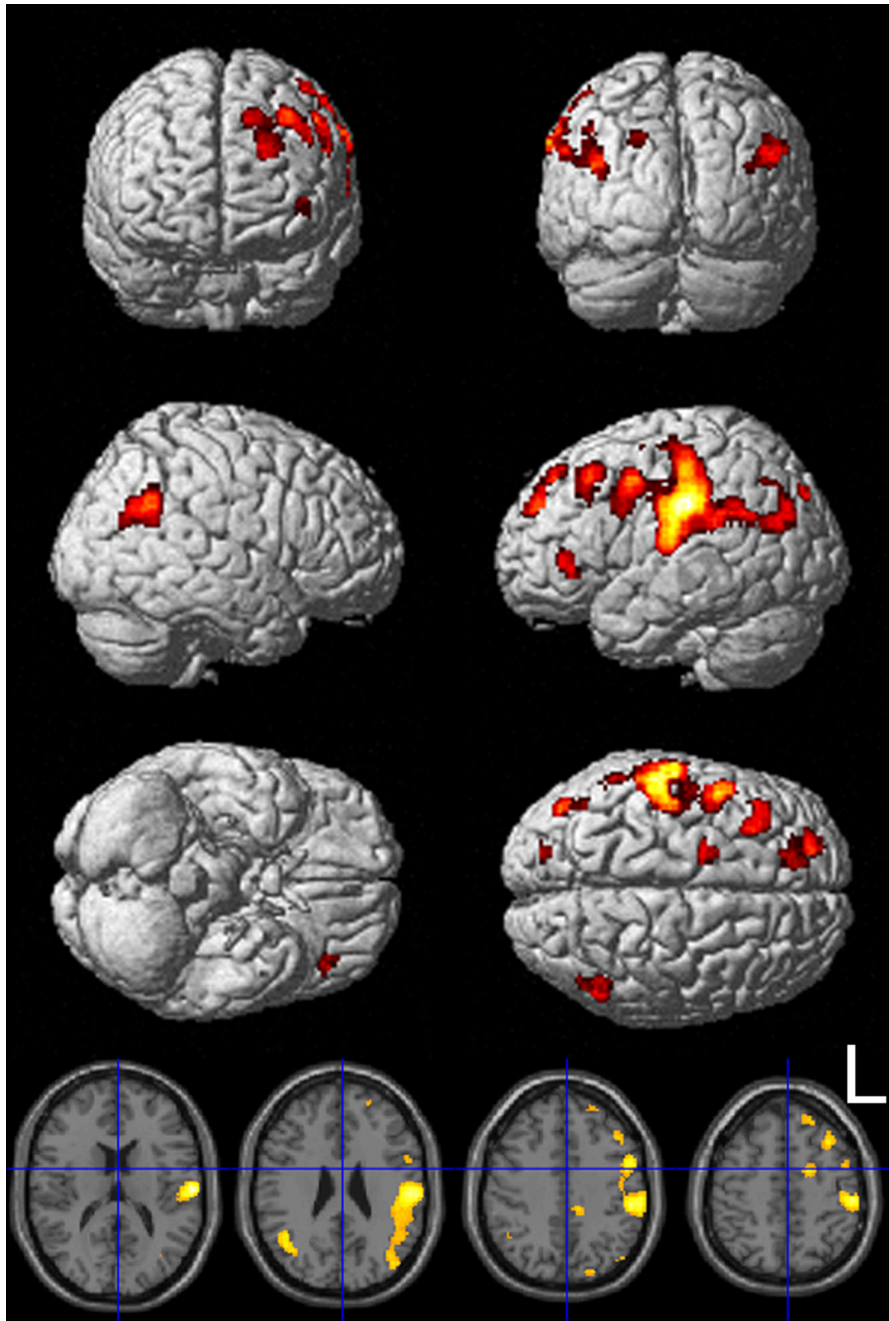
**FIGURE 3** ECD-SPECT images of the patient at 16 days after onset. Comparison with the normal database of the same generation using the easy Z-Score Imaging System and Statistical Parametric Mapping version 12 revealed significant (uncorrected $p < 0.001$) hypoperfusion in the left anterior superior frontal gyrus, posterior middle frontal gyrus, triangular part of the inferior frontal gyrus, lower half of the postcentral gyrus, and the perisylvian supramarginal gyrus.

evaluation of the samples from repetition and naming tests revealed 9/12 (75%) errors in two-mora words, 8/14 (57%) errors in three-mora words, 9/10 (90%) errors in four-mora words, and 6/7 (86%) errors in words with five or more moras. Although errors occurred only once in a word, the trend of more errors in longer words suggested a word-length effect.

Phonetic and phonemic errors in spontaneous speech, repetition, and naming encompassed morphemes,

numbers, and function words with no semantic errors (substitution with a semantically associated word).

### 3.2.2 | Naming versus repetition

Table 2 shows that errors in confrontation naming were 6.5 times higher than those in repetition (286.6% vs. 43.6%). Specifically, phonemic paraphasia (substitution

**TABLE 2** Articulation errors in the WAB Test.

| | D | | Ds | | Dv | | P | | S | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | V | C | V | C | V | C | V | C | V | C | O | A | Total |
| Spontaneous speech (161) | 11 | 12 | 5 | 11 | 0 | 0 | 18 | 0 | 6 | 29 | 1 | 1 | 94 (58.4%) |
| Repetition (110) | 7 | 13 | 0 | 9 | 0 | 3 | 2 | 0 | 0 | 11 | 1 | 2 | 48 (43.6%) |
| Object naming (67) | 12 | 14 | 3 | 22 | 0 | 5 | 59 | 0 | 11 | 64 | 1 | 1 | 192 (286.6%) |
| Word fluency (31) | 0 | 2 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 10 | 0 | 0 | 15 (48.4%) |
| Sentence completion (20) | 0 | 5 | 0 | 0 | 0 | 0 | 6 | 1 | 0 | 3 | 0 | 1 | 16 (80.0%) |
| Responsive speech (28) | 0 | 8 | 0 | 0 | 0 | 1 | 4 | 0 | 1 | 1 | 2 | 1 | 18 (64.3%) |
| Total (417) | 30 | 54 | 9 | 44 | 0 | 9 | 89 | 1 | 18 | 118 | 5 | 6 | 383 (91.8%) |

*Note*: The number of articulated syllables is denoted in parentheses. The error types were based on the classification of Duffy.[4]

Abbreviations: A, additions; C, consonants; D, distortions; Ds, distorted substitutions; Dv, distorted voicing distinctions (blurring of voiced–voiceless boundaries); P, prolongations; S, substitutions; O, omissions; V, vowels.

errors) in naming (75/67) was much higher than in repetition (11/110). Frequent error types in object naming included consonant substitutions and vowel prolongations. Similarly, vowel substitution, which was not noted in repetition, was observed in naming.

### 3.2.3 | Speech rate and prosody

Except for cliched phrases, speech was generally slow with discernible speed variance. As previously stated, vowels were frequently prolonged, particularly in object naming, with a variable rate. This change was quantitatively confirmed by measuring the duration of each syllable or mora (consonant–vowel pair, exceptionally with consonant-only or vowel-only) in words chosen from spontaneous speech, repetition, and naming components of the WAB test using Praat software version 6.3.20 (https://www.fon.hum.uva.nl/praat/). Nouns preceding a particle were selected from the samples. Those at the end of the phrases were excluded from analysis because of the inherent instability of syllable duration. There were a total of 31 nouns ranging from 2 to 7 syllables (median, 3 syllables). Speech rate was defined as the number of syllables per second. The comparison of the distribution patterns of words with low and high speech rates revealed that there were more words with low speech rates (Figure 4). The mean speech rate was 4.4 (95% confidence interval, 3.9–5.0) syllable/s, which was lower than the normal speech rate (6.1 syllable/s) in older controls.[15]

Next, the duration of each consonant–vowel syllable (mora) constituting a word was measured to examine the variance of the syllable duration (Table 3). Seven clinically diagnosed vowel prolongations were identified. The mean speech rate was faster in words with vowel prolongation than in words without apparent vowel
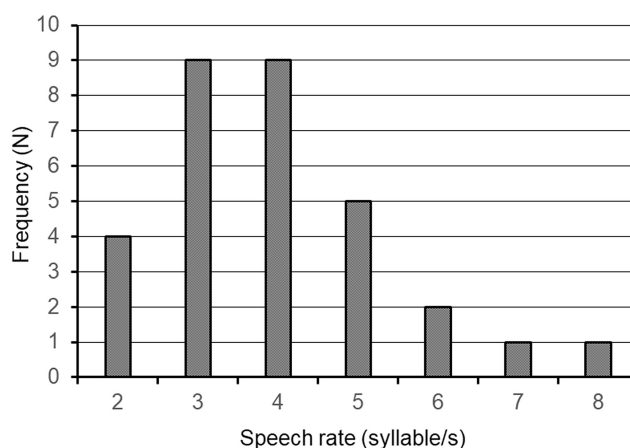


**FIGURE 4** Frequency of words with varied speech rate. Speech rate was defined as the number of syllables in a word divided by the duration of the word's pronunciation. The mean speech rate was 4.4 syllable/s, which was lower than the normal speech rate (6.1 syllable/s) in older controls ($n=13$).[15]

prolongation, but the difference was not significant. The mean syllable duration and the variance were nearly equal between words with vowel prolongation and those without vowel prolongation. We then calculated the ratio of each syllable duration/minimum syllable duration in a word to evaluate a variance of duration within a word. The mean syllable duration ratio for words with vowel prolongation was significantly larger (over 1.5) than that for words without vowel prolongation. This difference implied that the syllable duration was within 1.5 times the minimum duration for words without vowel prolongation and that the syllable duration exceeded this threshold for words with apparent prolongation. That is, words without vowel prolongation were pronounced slowly with a lower variance in duration, whereas those containing prolonged syllables were pronounced faster to nearly the normal level (not significant) but with a

**TABLE 3** Comparison between words with vowel prolongation and words without vowel prolongation.

| | Vowel prolongation (+) | Vowel prolongation (−) | p Value |
|---|---|---|---|
| Speech rate, syllable/s | 5.6<br>($n=4$ nouns) | 4.3<br>($n=27$ nouns) | 0.076 |
| Syllable duration (SD), s | 0.22 (0.07)<br>($n=7$ syllables) | 0.24 (0.07)<br>($n=95$ syllables) | 0.509 |
| Duration ratio (each syllable/shortest syllable) | 1.72<br>Range 1.53–1.93<br>($n=7$) | 1.14<br>Range 1.00–1.44<br>($n=64$) | <0.0001 |

*Note*: *p*-Values were based on Student's *t*-test or Welch's *t*-test.

Abbreviations: s, second; SD, standard deviation.

higher variance in duration. However, the absolute syllable duration was not different between the words with and without vowel prolongation.

Syllable segmentation within words was observed for five words, and each syllable was separated only once. The word accent and sentence intonation, which are important in natural Japanese speech, were intact. Overall, the speech was slow, and the rhythm of a sentence was affected by vowel prolongation, but word accent and sentence intonation were preserved.

# 4 | DISCUSSION

The patient exhibited sensory-modified motor speech disorder characterized by abundant phonetic distortion, phonemic paraphasia, and vowel prolongation, which was consistent with the diagnosis of AOS. The symptoms first appeared as Broca's aphasia with AOS, which resolved into pure AOS within 3 weeks. Phonemic paraphasia was more evident at this stage. MRI and ECD-SPECT revealed extensive damage to the lower half of the postcentral gyrus.

The patient had two important features that should be differentiated from those of conventional AOS. First, phonemic errors (vowel and consonant substitutions, or phonemic paraphasia) were more common than phonetic errors (vowel and consonant distortions). Although both types of errors are observed in AOS, phonetic errors are usually predominant, particularly in pure AOS.[16,17] Second, word accent and sentence intonation were preserved. This is in contrast to typical AOS profiles in which prosody impairments are common.[3]

## 4.1 | Phonemic paraphasia in postcentral AOS

The patient repeatedly attempted to correct his phonetic and phonemic errors. This kind of self-correction (or conduit d'approche in the French literature) is typically observed in conduction aphasia. However, preserved repetition and abundant phonetic errors from the disease onset are unlikely to diagnose this state as conduction aphasia. Phonemic paraphasia accompanied by conduction aphasia can be differentiated from Broca's aphasia with AOS as follows:

(a) Errors occur more frequently in confrontation naming than repetition.[18] As a potential mechanism, the phonetic information of a word is given in repetition, which serves to pronounce the target word, whereas the phonetic value of an object must be recalled in confrontation naming, a process that is interrupted in conduction aphasia. In Broca's aphasia with AOS, impairment lies in the later stages of vocalization. Thus, naming and repetition are also affected.[18]

(b) Errors are noted equally in consonants and vowels, in contrast to Broca's aphasia with AOS, in which consonant errors are predominant.[19]

(c) Transposition errors occur as often as substitution errors do.[19]

Our patient showed (a) but not (b) or (c). Frequent errors in confrontation naming suggested that auditorily inputted phonetic information about a word aided in the precise pronunciation of the target word. Because confrontation naming was nearly perfect at this stage (Table 1), impairment of the phoneme recall was ruled out.

Consonant substitutions were much more frequent, and no transposition was observed. However, it should be noted that some of the transposition errors of Monoi et al.[19] (e.g., [tamago] (egg)→[tamamo] and [tagamo]) were regarded as substitution errors in our classification. Their transposition errors were defined as "the production of a phoneme, in place of the target phoneme, that was a constituent elsewhere in the target word." Nevertheless, transposition errors are more frequently observed in conduction aphasia. These findings suggested that the patient's phonemic paraphasia had features similar to those observed in Broca's aphasia with AOS or conduction aphasia.

Additionally, the presence of a word-length effect cannot differentiate AOS from conduction aphasia because the length effect was observed not only in conduction aphasia but also in Broca's aphasia with AOS.[20] However, phonetic and phonemic errors occurring in morphemes, numbers, and function words with no semantic errors were more characteristic of AOS.[21]

Although the involvement of the postcentral gyrus in AOS has been indicated,[9] there have been no detailed reports of pure AOS caused by postcentral gyrus damage. On the other hand, atypical conduction aphasia with nonfluent speech features mimicking Broca's aphasia has been reported,[22,23] with lesions commonly involving the postcentral gyrus or subcortex. However, the role of the postcentral gyrus in speech is unclear. We previously reported activation of the inferior postcentral gyrus, including the parietal operculum, precentral gyrus, supplementary motor area, and insula, during vocalization (reading aloud minus covert reading) in a positron emission tomography study.[24] The somatosensory cortex (Brodmann Areas 3a, 3b, 1, and 2) receives proprioceptive sensations (position and motion) from the lips, tongue, and laryngeal muscles via the thalamus and connects to the motor cortex (Area 4) via U-fibers.[25] Furthermore, a recent neurophysiological study[26] has suggested that the motor cortex transmits efferent copy signals directly to the somatosensory cortex. These feedback information streams may contribute to the fine control of voice movement, and damage to the somatosensory cortex disrupts these feedback streams, resulting in different types of AOS.

## 4.2 | Prosodic deficits in AOS

The patient exhibited a slow speech. Vowels were frequently prolonged, particularly in object naming, and syllable segmentation within words was occasionally observed. Nevertheless, word accent and sentence intonation remained intact. It should be noted that, if the syllable duration varies within a certain range, slow speech has little effect on prosody, or at least accent and intonation. This may be evident when we consider that elderly people are generally slow in speech[27] but their prosody is minimally affected, although prosody in normal aging has been investigated to a lesser extent.[28] An unsolved problem is how vowel prolongation and syllable segmentation within a word can affect rhythm, another element of prosody. Because Japanese is a syllable (mora)-timed language,[29] these word-level deficits may interfere with the rhythm of a sentence to a certain extent.

The existence of AOS with dysprosody and AOS without dysprosody (accent and intonation) implies that dysprosody can be distinguished from AOS depending on the lesion site. When the neural substrates of these two syndromes are considered, dysprosody appears to be associated with the prerolandic areas. In this context, network overlap in the bilateral middle and lower precentral gyrus and medial frontal cortex has been found in foreign accent syndrome, an isolated prosodic disorder.[30] The left premotor and motor cortices (Brodmann Area 6) toward the end of the inferior frontal sulcus were identified as lesions responsible for foreign accent syndrome.[31] It should be noted that this area was very close to the average AOS lesion site.[10] These results, together with the present findings, suggest that prosody is preserved if the lesion does not involve the premotor and motor cortices.

## 4.3 | Accompanying agraphia

The patient exhibited somesthetic dysgraphia and kana and kanji agraphia. AOS is accompanied by frontal agraphia, given that the lesions are in close proximity. Somesthetic dysgraphia due to lesions in the postcentral gyrus is a type of peripheral agraphia that features abnormal grapheme formation, caused by interrupted proprioceptive feedback from the writing hand. Kanji writing was affected to a greater extent because of its orthographic complexity.

Kana agraphia is comparable to phonological agraphia in Western countries.[32] Japanese phonological agraphia occurs with lesions in the left posterior middle frontal gyrus,[33] subcortical inferior central sulcus area,[32] and supramarginal gyrus.[34] Phonological errors (kana substitutions) occur with lesions in any of these three areas, whereas transposition errors occur with lesions of the subcortical central sulcus[31] and supramarginal gyrus.[34] However, abundant character recall impairment occurs only in lesions with damage to the posterior middle and inferior frontal gyri.[33] In summary, the presence of kana agraphia did not discriminate the lesion location in the present case. However, the lack of transposition and abundant kana recall impairment suggested the involvement of the precentral area.

## 5 | CONCLUSIONS

Apraxia of speech due to postcentral gyrus lesions is characterized by the presence of abundant phonemic paraphasia, rather than phonetic distortion, and preserved word accent and sentence intonation. Phonemic paraphasia is accompanied by self-correction and is more pronounced in confrontational naming than in repetition, which is a characteristic of conduction aphasia. This symptomatic

similarity to conduction aphasia is an important feature of postcentral AOS. Furthermore, preserved accent and intonation, despite slow speech with vowel prolongation and syllable segmentation, is another feature distinct from precentral AOS. This case is clinically important in that if the above two features are observed, it indicates the involvement of the postcentral area.

Although the patient's primary lesion was located in the postcentral gyrus, it spread subcortically to the precentral gyrus. Therefore, it is unknown whether a restricted lesion in the postcentral gyrus yields AOS. Moreover, detailed phonological differentiation from conventional precentral AOS is yet to be elucidated. Further studies are needed to determine the characteristics of postcentral AOS.

## AUTHOR CONTRIBUTIONS

**Naoko Mitani:** Writing – original draft. **Yasuhisa Sakurai:** Writing – review and editing.

## FUNDING INFORMATION

## CONFLICT OF INTEREST STATEMENT

The authors report there are no competing interests to declare.

## DATA AVAILABILITY STATEMENT

All data generated or analyzed in this study are included in this published article. Further inquiries can be directed to the corresponding author.

## ETHICS STATEMENT

This study was conducted in accordance with the Declaration of Helsinki of the World Medical Association for Experiments involving Humans. The study protocol was reviewed and approved by the Research Ethics Committee of our hospital (No. C51).

## CONSENT

Written patient consent has been signed and collected in accordance with the journal's patient consent policy.

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