thalamic infarction caused by deep venous system occlusion. He was treated with heparin and recovered with residual mild cognitive impairment. Our patient also showed bilateral thalamic involvement and had a favorable prognosis with anticoagulation therapy.

DCVT is considered to have a poor prognosis [1]. Review of the literature disclosed that 35% of isolated DCVT cases were fatal [1, 2, 4–9]. A prospective study of 59 patients also suggested that involvement of the straight sinus and the deep venous system may be associated with poor outcome [3]. In addition, the most recently published large CVT cohort study revealed that DCVT, mental status disorder, male sex, and age >37 years, all of which were observed in our patient, increased the risk of death and dependence [12]. However, a favorable prognosis has recently been reported in some cases [1, 4, 5, 7–9]. This may partly be because milder cases are increasingly diagnosed earlier by MRI [5, 9]. The early recovery and complete resolution of the abnormal MRI findings in our patient would suggest that he had a venous congestion or edema, but not an established venous infarction.

In our patient, steroid pulse therapy, which was administered before the diagnosis of DCVT, seemed to be effective. High-dose corticosteroids may have improved the venous congestion or vasogenic edema. An alternative explanation may be that spontaneous recanalization or an improvement of collateral blood flow occurred independently with steroid pulse therapy. If we had made an early diagnosis of DCVT, we might not have used corticosteroids. Although some reports suggested a good prognosis following steroid therapy administration [7, 8], the efficacy of corticosteroids against DCVT has not yet been systematically established [3].

In conclusion, DCVT should be considered in patients who show isolated mental changes. Symmetric signal intensity changes on MRI images of the deep brain structures and the blood flow defect detected by MRV will lead to the diagnosis. Therefore, DCVT can be important for the differential diagnosis of treatable dementia.

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# Transcortical Sensory Aphasia following Infarction in the Left Frontal Lobe

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#### Introduction

Nonfluent aphasias such as Broca's area and transcortical motor type aphasia are known to be caused by lesions of the anterior language area in the left frontal lobe [1], but few reports have described fluent aphasia caused by lesions in the frontal lobe [2–4]. We report a case of transcortical sensory aphasia characterized by fluent speech, poor comprehension and preserved repetition following infarction in the left frontal lobe.

#### Case Report

A 61-year-old, right-handed man suddenly developed speech disturbance on 18 February 2003 and was referred to our hospital for diagnosis. He had a history of hypertension but no prior history of neurological problems. He had had 12 years of education. Initially, the patient was completely conscious and fully oriented. No abnormalities were detected in his eyesight or field of vision, and no motor paresis or sensory disturbance could be demonstrated.

*Neuropsychological Findings.* The patient's spontaneous speech was fluent. He initiated speech without difficulty, articulated normally and did not have logorrhea. Echolalia was observed. For example, when we asked him, 'gokibun-wa ikagadesuka (how do you feel?)', he simply repeated the question. Verbal paraphasia was evident in substitutions such as 'isu' (chair) for 'tukue' (desk). Some perseveration was noted when he performed picture description tasks. By contrast, he had a fully preserved ability to repeat phonemes and short sentences of 5–6 words. He was able to read aloud in both Kanji (Chinese-based characters) and Kana (syllable-based characters) and read sentences aloud perfectly; however, he did not understand their meaning. Agraphia was almost total, except that writing to dictation of Kana letters and single words (Kanji and Kana) was partially pre-

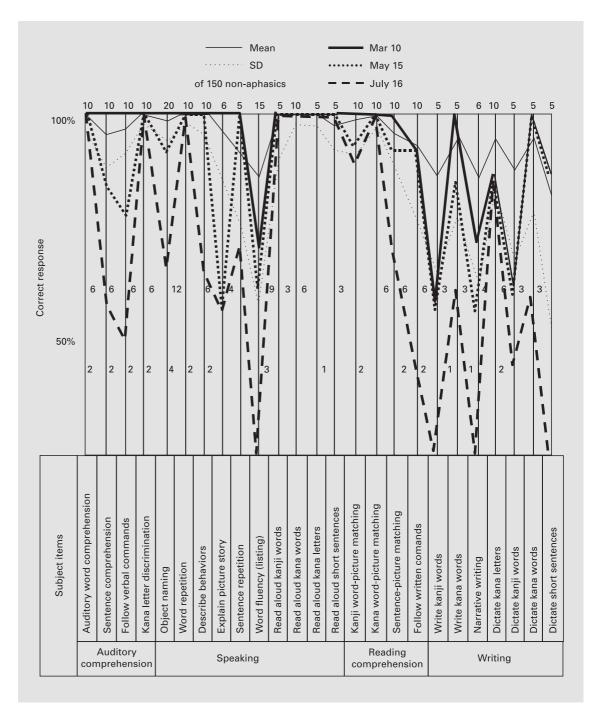
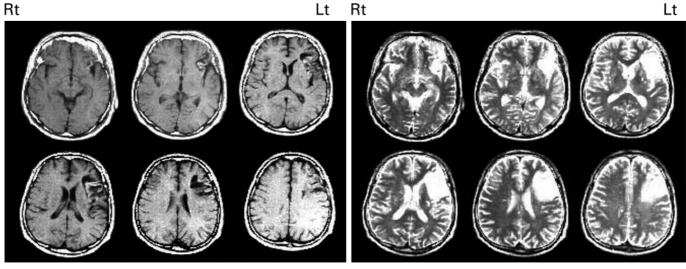


Fig. 1. Profile of SLTA.

served. These features, characterized by (1) fluent spontaneous speech with echolalia and verbal paraphasia, (2) good repetition, (3) moderately poor comprehension (oral and written), (4) agraphia, (5) failure of confrontation naming and (6) absence of articulate disorder, were considered to represent transcortical sensory aphasia according to the classic classification [5].

A standard language test for aphasia (SLTA) was performed 3 weeks after the onset of symptoms (results are shown in fig. 1). In addition to aphasia, the patient demonstrated constructional apraxia but not bucco-facial, ideomotor or ideational apraxia. On the Wechsler Adult Intelligence Scale-Revised, his performance IQ was 67. Verbal IQ could not be measured because of the aphasia.

Short Reports



T<sub>1</sub>-weighted image

T<sub>2</sub>-weighted image

**Fig. 2.** MRI revealed cerebral infarction in the left frontal lobe, involving the inferior frontal, middle frontal and precentral gyrus.

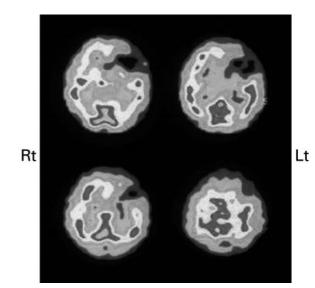
*Neuroradiologic Findings.* Magnetic resonance imaging (MRI) 2 days after the onset of symptoms revealed cerebral infarction involving the left inferior frontal, midfrontal and precentral gyri (fig. 2). Single-photon emission computed tomography (SPECT) 6 weeks after symptom onset revealed a localized area of low perfusion over the entire inferior frontal gyrus, extending to the middle frontal and precentral gyrus without involvement of the postcentral gyrus (fig. 3).

*Clinical Course.* Speech improved gradually 3 months after the onset of symptoms. Spontaneous speech was still limited. However, the patient improved his ability to identify and describe scenes. Paraphasia had disappeared. Auditory verbal comprehension had improved remarkably but was still poor at sentence level. Agraphia improved remarkably. Although he still had mild aphasia 6 months after symptom onset, functional MRI (fMRI) was performed to determine the dominant side for language. Word generation fMRI imaging showed activity to be almost entirely confined to the left hemisphere.

## Discussion

In transcortical aphasia, repetition classically has been considered to be carried out by the spared left perisylvian area, i.e. Heschl's gyrus, Wernicke's area, Broca's area, the inferior left precentral gyrus and their interconnections [6]. In support of this view, several neuropathological and radiological studies have shown that transcortical aphasia can result from acute brain lesions surrounding the speech area, thus potentially disconnecting the intact perisylvian language zone from the peripheral brain areas that may be essential for meaning and intention [7]. However, in our case of transcortical sensory aphasia, the lesion was situated in the left frontal lobe including the inferior frontal lobe (Broca's area).

Basso et al. [8] studied the frequency of exceptions to classic aphasia localizations by correlating clinical and computerized to-



**Fig. 3.** SPECT revealed a localized area of low perfusion over the entire inferior frontal gyrus extending to the middle frontal and precentral gyrus without involvement of the postcentral gyrus.

mography data. Out of 207 aphasics, 7 were reported to have fluent aphasia with 'anterior' CT lesion. Some authors have suggested that repetition in transcortical aphasia is carried out either by the nondominant (usually right) hemisphere or by joint activity of uninvolved left hemispheric structures and the intact right hemisphere [9]. Based on Wada test findings in a patient with transcortical sensory aphasia, Bando et al. [10] suggested a fundamental role of the nondominant (right) hemisphere in the repetition of language. Berthier et al. [11] argued that the spared contralateral hemisphere may explain preserved repetition in some transcortical aphasia patients, irrespective of whether the lesion is within or outside the speech-dominant perisylvian area. Although we did not perform an amytal test, the fMRI of our patient suggested that the left hemisphere was dominant for language functions. Nonetheless, we concluded that repetition could be preserved despite a lesion in the left inferior frontal gyrus in the perisylvian speech area.

Anarthria, the core symptom of nonfluent aphasia, does not occur when damage is limited to Broca's area. Instead, it occurs when the lower part of the precentral gyrus [12–14] or pre- and postcentral gyri are involved [15].

The few reports of fluent aphasia caused by lesions in the frontal lobe including Broca's area that have described cerebral blood flow (CBF) findings [3, 16] suggested that nonfluent characteristics did not appear because CBF in the precentral gyrus was maintained. Our present case shows cortical hypoperfusion in the inferior frontal gyrus extending to the middle frontal and precentral gyrus without involvement of the postcentral gyrus. We suggest that some individual variation may occur in the frontal lobe site potentially responsible for anarthria.

Although our case had a cortical and subcortical lesion in the frontal lobe, comprehension of spoken and written language was poor. A positron-emission tomography study by Peterson et al. [16] found an increase in the blood flow in the frontal lobe to be necessary for understanding words. Schaffler et al. [17] reported a case where electrical stimulation of Broca's area caused poor comprehension. Caplan et al. [18] insisted that syntactic processing involves an extensive neural system, whose most important region is the left perisylvian cortex. Stromswold et al. [19] showed that one aspect of sentence processing is centered in the pars opercularis of Broca's area. Enokido et al. [20] reported that the posterior parts of the superior, middle and inferior frontal lobes may play different roles in the expressive processes of language. The superior frontal lobe be may responsible for spontaneous speech production, the middle frontal lobe may take a role in identifying the contextual sentence structure, and the inferior frontal lobe could be responsible for grammar. Based on clinical observations, Otsuki et al. [21] reported that in aphasia due to lesions located in the frontal lobe, the ability to understand deteriorates more as the lesion spreads inferiorly and laterally from the superior frontal gyrus. Such findings underscore the importance of lesions in the midfrontal gyrus cortex and subcortex for semantic understanding disturbances. Aphasic symptoms in our case, mainly poor comprehension, improved gradually over several weeks. Transcortical sensory aphasia caused by frontal lobe lesions may typically be followed by a relatively rapid recovery of comprehension, although this is not true for all cases.

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