

EFFORTFUL ECHOLALIA

Kazuo Hadano¹, Hikaru Nakamura^{2,3} and Toshihiko Hamanaka²

¹Department of Psychogeriatrics, National Institute of Mental Health (NCNP);

²Department of Neuropsychiatry, Nagoya City University School of Medicine;

³Department of Audiology and Logopedics, Japan College of Rehabilitation and Welfare Professionals, Nagoya)

ABSTRACT

We report three cases of effortful echolalia in patients with cerebral infarction. The clinical picture of speech disturbance is associated with Type 1 Transcortical Motor Aphasia (TCMA, Goldstein, 1915). The patients always spoke nonfluently with loss of speech initiative, dysarthria, dysprosody, agrammatism, and increased effort and were unable to repeat sentences longer than those containing four or six words. In conversation, they first repeated a few words spoken to them, and then produced self initiated speech. The initial repetition as well as the subsequent self initiated speech, which were realized equally laboriously, can be regarded as mitigated echolalia (Pick, 1924). They were always aware of their own echolalia and tried to control it without effect. These cases demonstrate that neither the ability to repeat nor fluent speech are always necessary for echolalia. The possibility that a lesion in the left medial frontal lobe, including the supplementary motor area, plays an important role in effortful echolalia is discussed.

Key words: echolalia, transcortical motor aphasia, compulsive behavior, frontal lobe.

INTRODUCTION

Echolalia (Romberg, 1857), the automatic repetition of speech of the partner in conversation, occurs in various conditions: (1) in normal children, occurring at a particular stage of language acquisition; (2) in pathological mental development, especially in early infantile autism and mental retardation; (3) as a catatonic symptom in schizophrenic patients; (4) as a partial symptom of dementia in diffuse brain damage, for example in patients with Alzheimer's or Pick's disease; and (5), in the subject of this paper, echolalia occurring in patients predominantly with transcortical aphasia caused by a circumscribed lesion of the left hemisphere.

Echolalia as a form of automatic speech (Poeck, 1989) is generally realized with fluent speech lacking in increased effort, dysarthria (anarthria), decreased phrase length (agrammatism), and dysprosody (Benson, 1979). The repetition ability of patients with transcortical aphasia, in which echolalia often occurs, is by definition well (or normally) preserved (Goldstein, 1915, 1948). However, we have recently observed nonfluent effortful echolalia combined with slight or moderate difficulty in repetition in three aphasic patients. Addressed by the conversation partner, they always repeated very laboriously the few words spoken to them before answering. Despite full awareness of their echolalia, they

were unable to stop it voluntarily, and they felt it forced on them. We present here a report of three cases with effortful echolalia and consider its implications in aphasiology.

CASE REPORTS

Case 1

MM, a 68-year-old right-handed housewife educated to the elementary school level, was hospitalized for rehabilitation therapy. She had been known to be hypertensive for three years. Three months before admission, she had suddenly developed a severe speech disturbance and mild right hemiparesis including the face, and was initially admitted to another hospital and medically treated under the diagnosis of cerebral infarction.

X-ray Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) revealed a large circumscribed lesion caused by cerebral infarction involving the medial frontal lobe including the anterior portion of the cingulate gyrus and the supplementary motor area (SMA), the superior and middle frontal gyrus, and the anterior and superior part of the basal ganglia in the left hemisphere. The infarct was located in the territories of the anterior and middle cerebral artery and the watershed area between them (Figure 1). The posterior part of the inferior

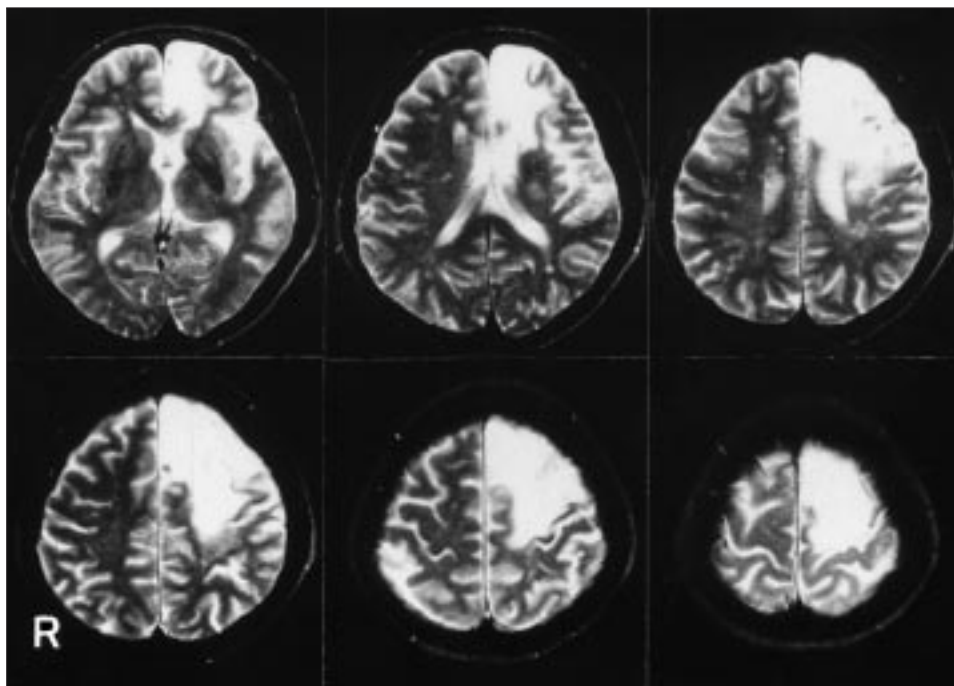


Fig. 1 – T2-Weighted Magnetic Resonance Imaging (MRI) of Case 1.

frontal gyrus containing Broca's area and the lower part of the pre- and post-central gyrus in the left hemisphere appeared to be spared. There were some small lacunar infarctions in the right hemisphere.

She was awake, alert, fully oriented and cooperative during each examination. She showed only slight emotional lability during the first few months. Neurological examination demonstrated a mild right hemiparesis including the face. Orofacial apraxia and severe aphasia were detected as neuropsychological symptoms. The performance intelligent quotient (IQ) was 66 on the Wechsler Adult Intelligence Scale (WAIS) and 54 with the Kohs Block Design Test. The Raven's Progressive Matrices score was 14/36 in the Colored and 17/60 in the Standard Version.

There was little spontaneous speech, but she responded verbally to questions from the examiner with a strong tendency to echolalia. She always first repeated a few words in the question and then produced her response. Accordingly, her speech consisted of two parts, the echolalic repetition and her self initiated answer; these corresponded to the "mitigated form of echo" described by Pick (1924). Both the echolalic and intentional part were realized nonfluently with distorted articulation, simplification of grammatical forms (agrammatism), monotonous intonation (dysprosody), and increased effort (Benson, 1979). Figure 2 shows the profile of her speech characteristics evaluated with the Boston Diagnostic Aphasia Examination (BDAE, Goodglass and Kaplan, 1972). She was always aware of her echolalia, against which she often agonizingly struggled by closing her eyes tightly and shaking her head violently during her echolalising. She was nevertheless unable to control it.

Moderate word-finding difficulties and few phonemic paraphasias were observed on confrontation naming. Auditory comprehension was moderately disturbed at the semantic level. In the object pointing task, examining auditory comprehension, she always repeated the stimulus word correctly, but sometimes pointed to another object. The score on the Token Test (Japanese version) was 36/165. She showed marked difficulties in repeating utterances longer than those containing four words. She was able to repeat five-digit numbers forward. Reading aloud and reading comprehension of written words were well preserved, but those of written sentences were moderately or severely disturbed. She could write only a few Kana- and Kanji-words. Writing sentences was impossible. Figure 3 shows the Z-score profile of the Japanese Standard Language Test for Aphasia (JSLTA, Japanese Society of Aphasiology, 1977).

Her speech performance improved considerably with intensive speech therapy. Figure 3 shows the JSLTA profile after three months of speech therapy. The nonfluent speech and effortful echolalia, however, remained unchanged.

The type of aphasia in this patient is not consistent with any typical aphasia described in classical theory. Loss of speech initiative with echolalia can be regarded as components of Transcortical Motor Aphasia (TCMA), while nonfluent agrammatic speech, mildly impaired repetition and moderate disturbance of auditory comprehension led us to a diagnosis of Broca's aphasia. The diagnosis we finally reached was a mixed TCMA and Broca's aphasia.

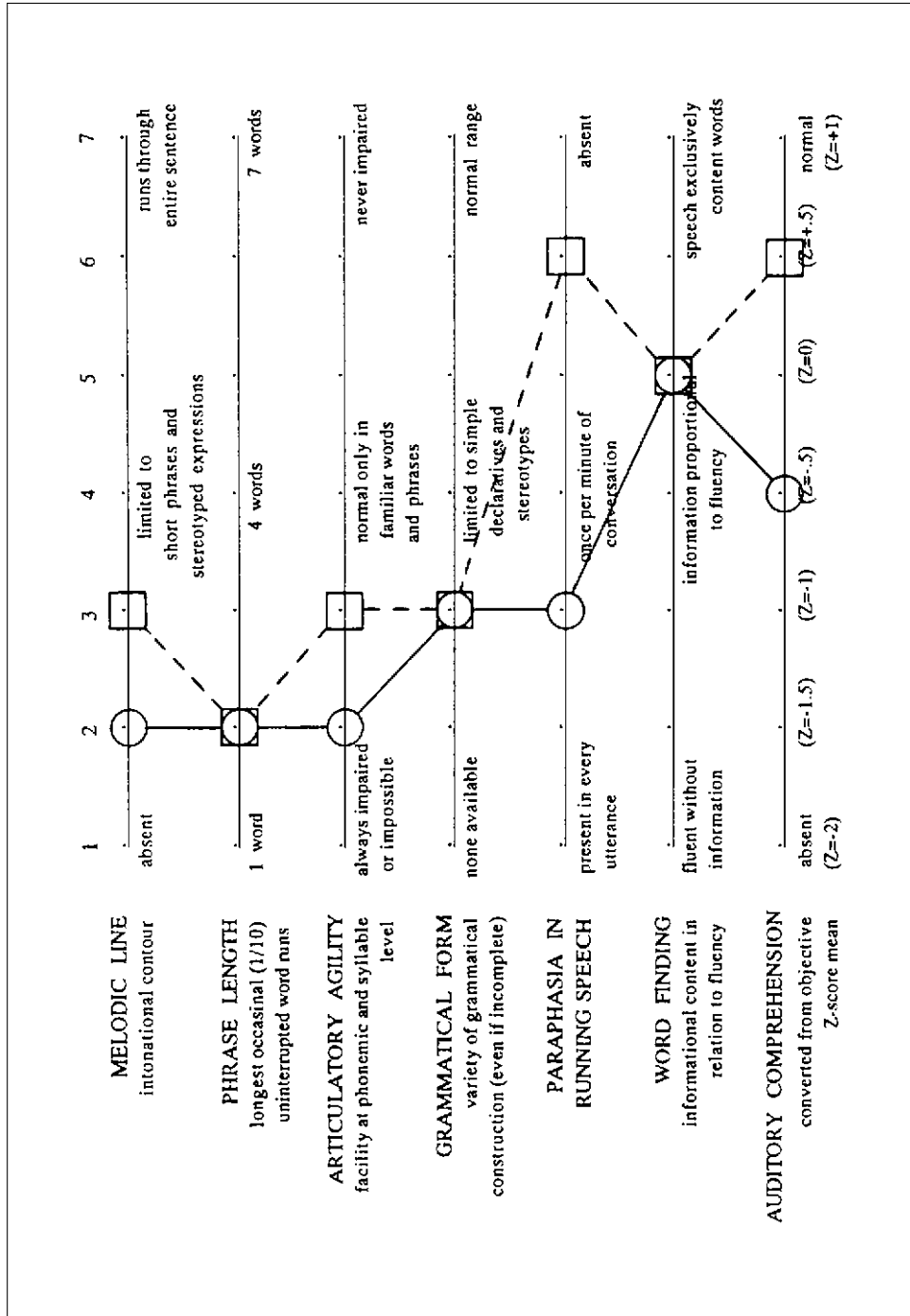


Fig. 2 – Speech profile ratings of Case 1 (shown by circles linked with solid lines) and Case 2 (by squares with broken lines). Rating scale profiles of speech characteristics in Boston Diagnostic Aphasia Examination (BDAE, Goodglass and Kaplan, 1972).

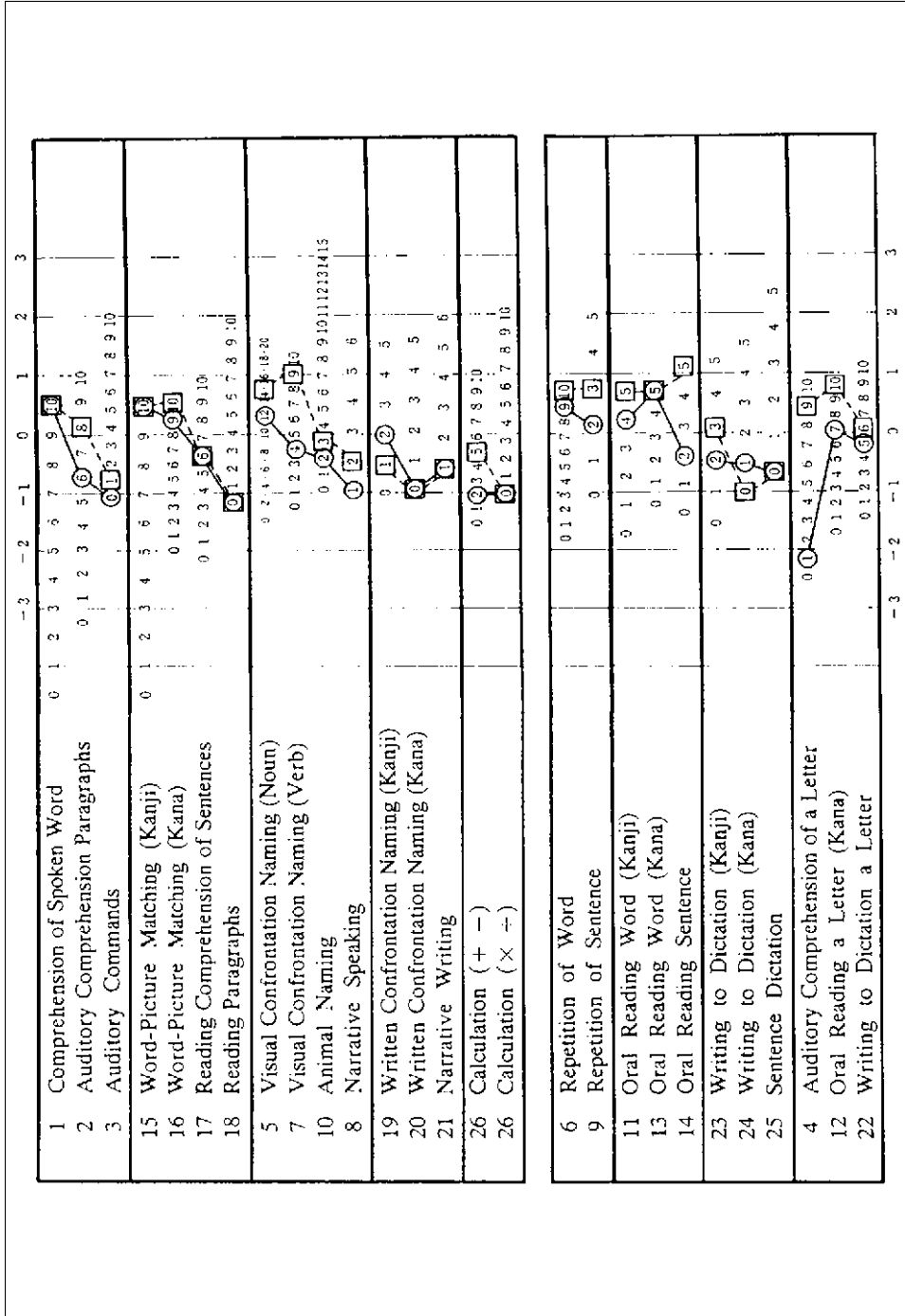


Fig. 3 - Z-score profile of Japanese Standard Language Test for Aphasia (JSLTA, Japanese Society of Aphasiology, 1977) on admission (shown by circles linked with solid lines) and three months later (by squares with broken lines) of Case 1.

Case 2

MY, an 80-year-old right-handed woman with a high school education, was hospitalized in order to rehabilitation therapy for aphasia and right sided motor disturbance. She had a long history of hypertension. A cerebral infarction at the age of 58 had caused a temporary mild right hemiparesis. At the age of 79, eleven months before admission, she had been suddenly affected by a second cerebral infarction, again developing a hemiparesis of the right side and severe speech disturbance.

X-ray CT and MRI revealed a large lesion in the left hemisphere, including the inner and anterior portion of the superior frontal gyrus, the subcortical white matter of the frontal lobe, and the upper part of the insula and the basal ganglia (Figure 4). The posterior part of the inferior frontal cortex (Broca's area) appeared to be largely spared. The left SMA cortex also seemed to be spared, but the underlying white matter was widely involved. There were no ischemic lesions in the right hemisphere with the exception of moderate so-called leuko-araiosis.

She was alert, fully oriented and cooperative. Neurological examination revealed a severe, incomplete, right hemiplegia with forced grasping. Neuropsychological evaluations detected a severe aphasia, orofacial and ideomotor apraxias, and constructive disabilities. The Mini-Mental State Examination (MMSE, Japanese version) score was 16/30 and the Performance

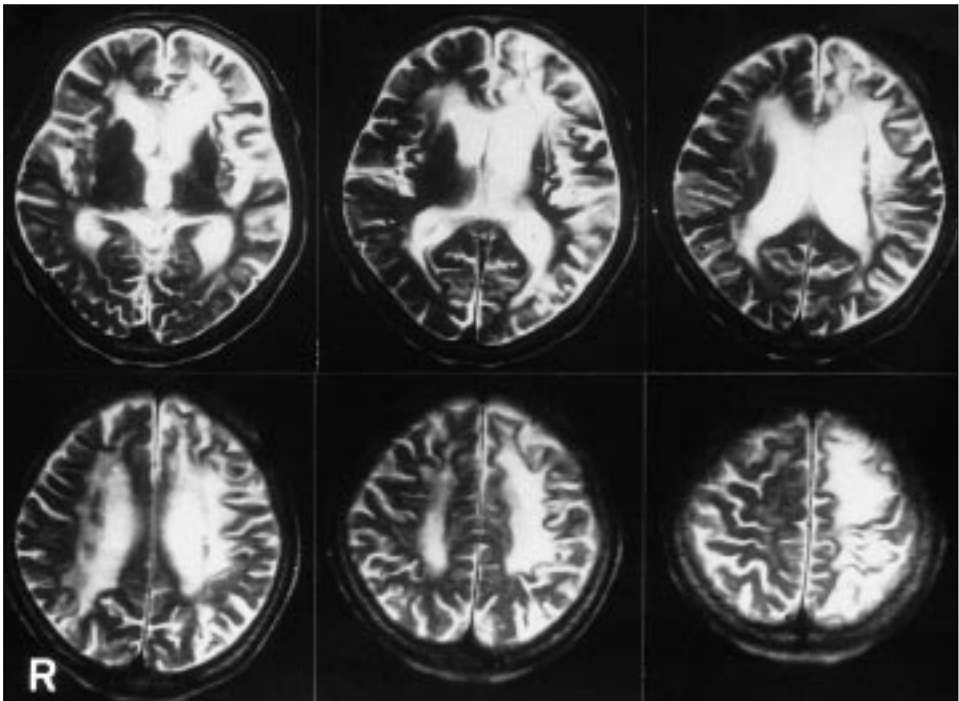


Fig. 4 – T2-Weighted MRI of Case 2.

IQ was 62 evaluated with the Wechsler Adult Intelligence Scale-Revised (WAIS-R) and 49 with the Kohs Block Design Test.

She had virtually no spontaneous speech, but she responded verbally to questions. Her conversation was limited to simple subjects of daily life, and this was not easy even with the help of the partner. Her speech was non-fluent with mild dysarthria, monotonous dysprosody, telegrammatic agrammatism, and greatly increased effort (Benson, 1979). Speech characteristics were assessed by the rating scale profile of BDAE (Figure 1). She always repeated laboriously a few words of high informative value contained in the partner's question, and subsequently, she made her own answer with a word or two also produced laboriously (mitigated echolalia, Pick, 1924). She often shook her head with an agonized look during the echolalic repetition, and appeared to be trying to fight it. She appeared to feel it forced against her own will. Indeed, she explained laboriously that she was fully aware of her echolalia but was unsuccessful in trying to stop it.

The JSLTA profile on admission is shown in Figure 5. Auditory comprehension was mildly disturbed. The Token Test (Japanese version) score was 117/165. Repetition was limited to words and to short sentences containing four words despite echolalia; JSLTA showed moderate disturbance of repetition.

In the confrontation naming test, she demonstrated moderate word-finding difficulty with some semantic paraphasias. No phonemic paraphasia was observed either in the test situations or in conversation. Reading aloud and comprehension of written material, including Kanji- and Kana-words, was relatively well preserved. Writing both on written confrontation naming and to dictation was severely impaired. She could write only a few Kanji-words and several Kana-letters.

Regular speech therapy had little effect, and after two months she complained of a general malaise with further deterioration of her speech. Although there were no changes in the MRI-findings, the cause was thought to be further cerebral ischemia. Loss of speech initiative and nonfluent effortful echolalia was almost unchanged, but her performance on language tests, including the JSLTA, showed some deterioration on retesting after six months (Figure 5).

The clinical picture of aphasia with loss of speech initiative and echolalia is compatible with Type 1 TCMA (Goldstein, 1915) with the exception of nonfluent speech and moderate impairment of repetition that may belong to Broca's aphasia syndrome.

Case 3

BT, a 63-year-old right-handed man who was a retired official with an elementary education and a four-year history of diabetes mellitus, developed acute right-sided motor weakness and aphasia with impairment of consciousness. Consciousness cleared after a few days, but severe aphasia and mild right hemiparesis became apparent. He was admitted to our institution two weeks after the onset and was diagnosed as having a cerebral infarction on the basis of neurological and neuroradiological findings.

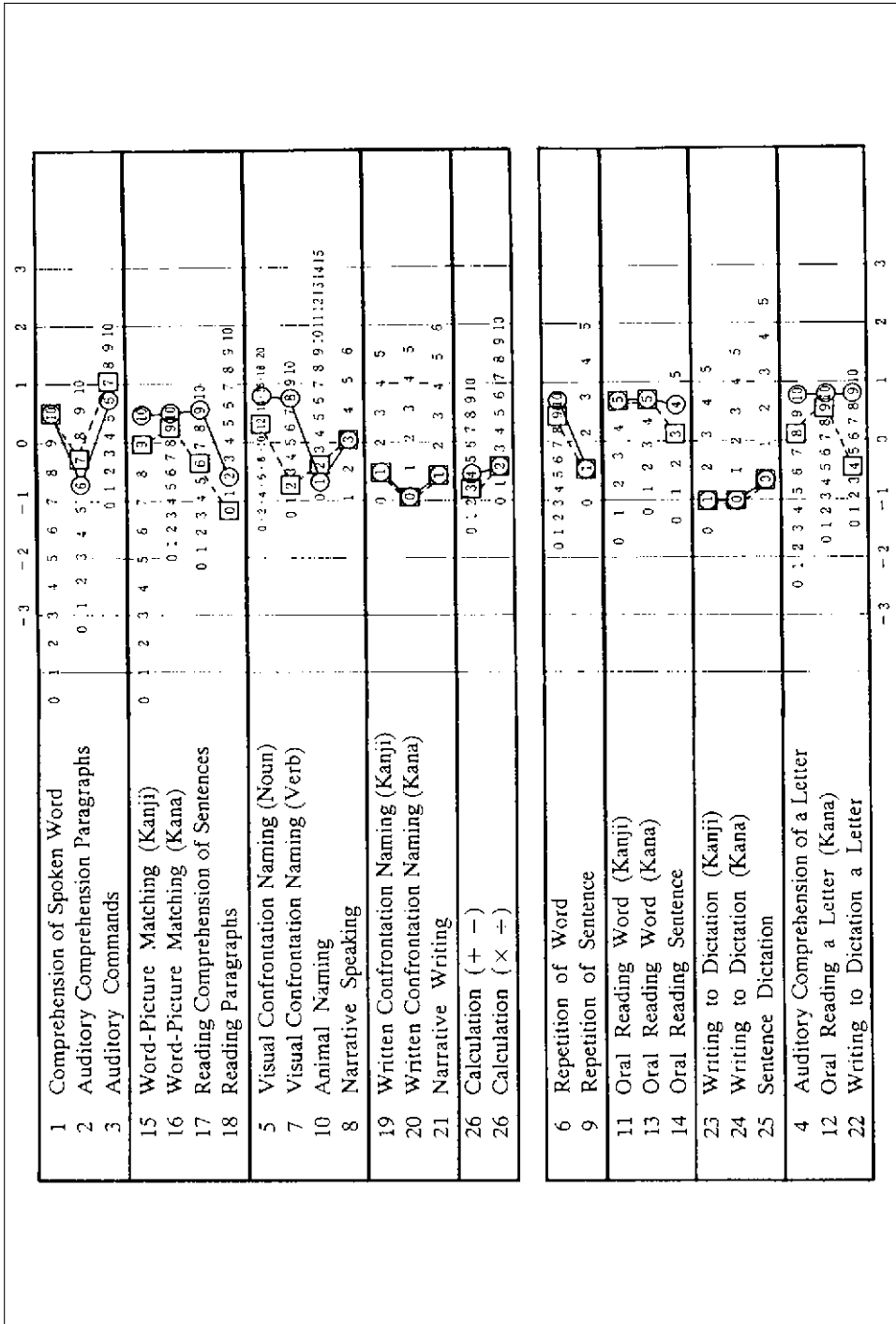


Fig. 5 – Z-score profile (JSLTA) on admission (shown by circles linked with solid lines) and six months later (by squares with broken lines) of Case 2.



Fig. 6 – T2-Weighted MRI of Case 3.

X-ray CT and MRI performed on admission demonstrated a circumscribed lesion localized to the anterior superior part of the medial frontal lobe, the anterior part of the basal ganglia, the insula, the posterior part of the inferior frontal gyrus including Broca's area, and the lower part of the central gyri in the left hemisphere (Figure 6). The left SMA was spared.

He was alert, oriented in time and space, and cooperative to examination. Neuropsychological examination on admission revealed mild orofacial and ideomotor apraxia, transient constructional disability, and severe aphasia. Performance IQ (WAIS-R) was 64 and the score on Raven's Colored Progressive Matrices was 31/36.

The patient produced hardly any spontaneous utterances. Speech was limited exclusively to nonfluent repetition of several words contained in the examiner's question during the first two weeks after admission (complete echolalia). Auditory comprehension was severely disturbed on admission, but markedly recovered four months later. As the speech disturbance gradually improved, his echolalic repetition came to be nearly always followed by self initiated speech (mitigated echolalia). His speech, including echolalia, was produced laboriously with occasional articulatory distortions, short phrase length and monotonous intonation, and was contaminated with sporadic verbal paraphasias. The rating profile of his speech characteristics (BDAE) is shown in Figure 7. In spite of the

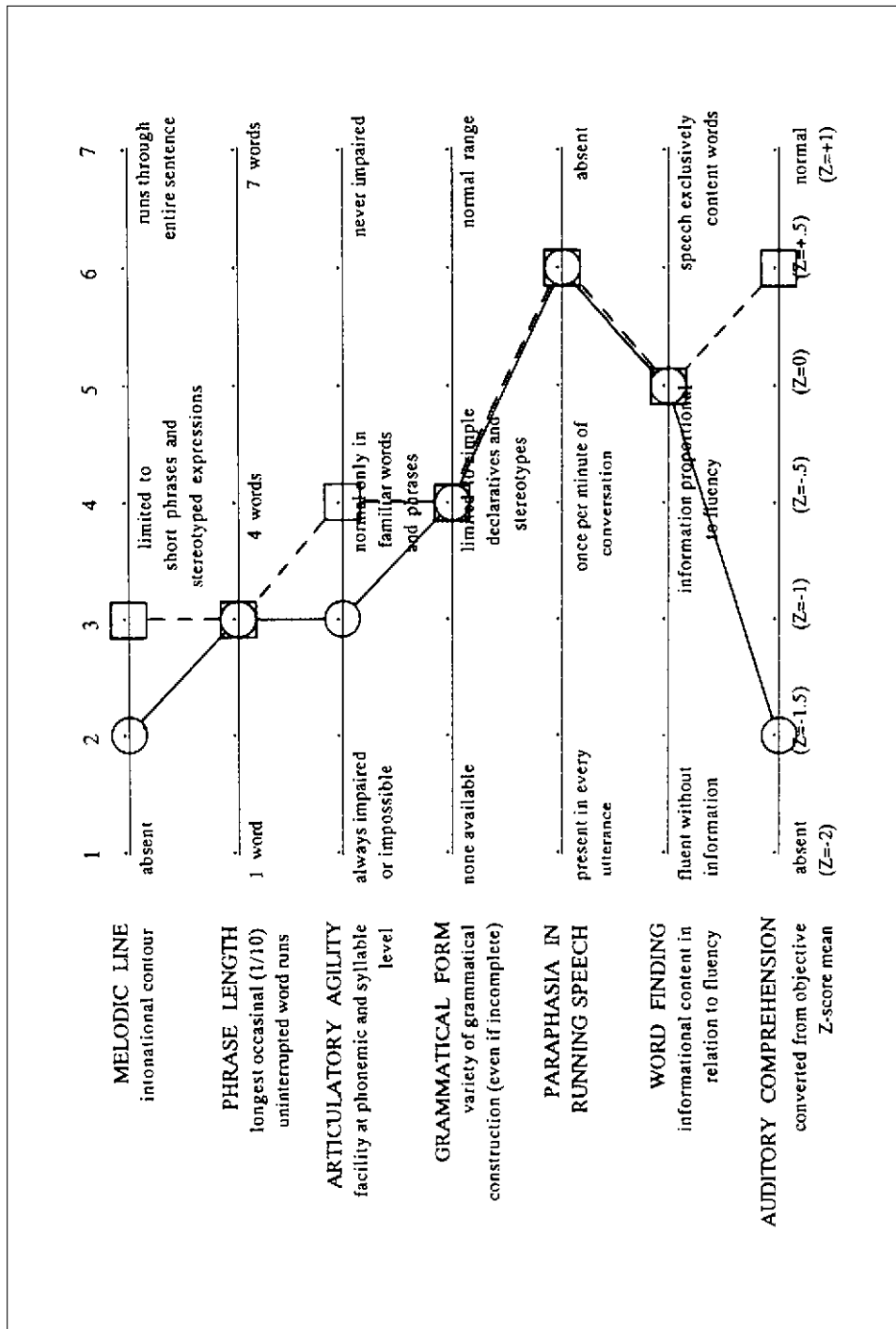


Fig. 7 – Speech profile ratings (BDAE) on admission (shown by circles linked with solid lines) and four months later (by squares with broken lines) of Case 3.

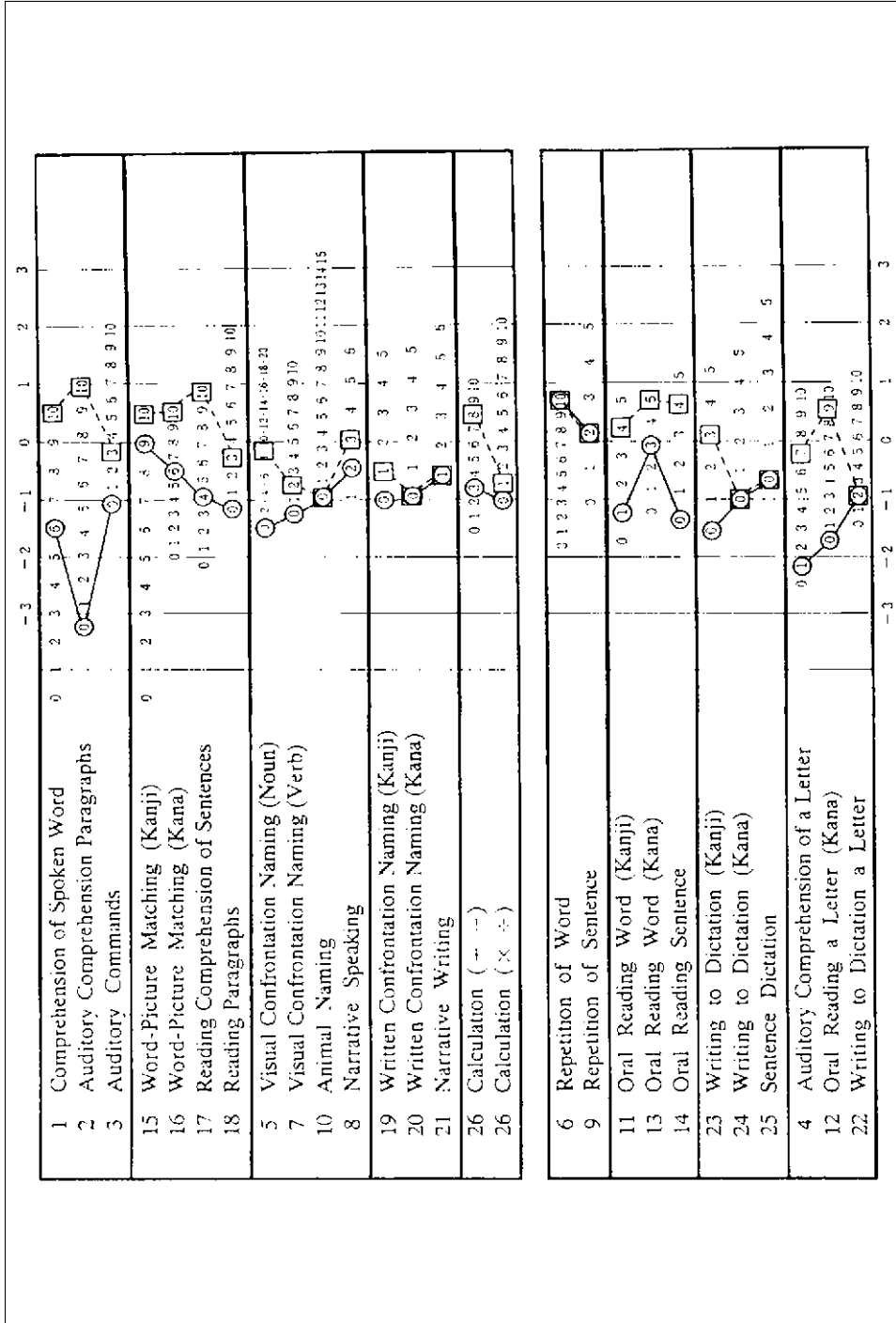


Fig. 8 – Z-score profile (JSLTA) on admission (shown by circles linked with solid lines) and four months later (by squares with broken lines) of Case 3.

echolalia, his repetition was not complete and was limited to sentences containing six words. He was fully aware of his own echolalic behaviour and tried to check it, accompanied by expressions of frustration, to little effect. Figure 8 shows his JSLTA Z-score profile.

The aphasia improved with intensive speech therapy. Figures 7 and 8 show his speech performance after four months. While auditory and reading comprehension, confrontation naming and reading aloud improved considerably, the dominant characteristics of his speech disturbance, i.e. nonfluency and mitigated echolalia, did not substantially recover.

The clinical picture of the language disturbance is almost consistent with a mixed transcortical aphasia in the acute stage and Type 1 TCMA subsequently.

DISCUSSION

The speech disturbance demonstrated in the three patients reported here can be summarized as mitigated echolalia (Pick, 1924) realized with nonfluent speech (Benson, 1979). In echolalia, as well as in self initiated speech, they spoke only a few words of high informative value which were produced laboriously with distorted articulation (dysarthria, anarthria), short phrase length, simplification of grammatical forms (agrammatism), and monotonous intonation (dysprosody). Except for the echolalia, pathological automatic expressions including so-called completion phenomena (Stengel, 1947) were not observed in the patients. They were also completely aware of their own echolalic repetition, against which they openly struggled without being unable to control it.

Echolalia is a form of automatic speech (Poeck, 1989), and is therefore manifested as non-effortful fluent speech in general. We propose here the term "effortful echolalia" for the echolalic behaviour observed in our patients. Although the combination of the terms "effortful" and "echolalia" may appear contradictory, it nevertheless fits the phenomenon observed clinically.

If this terminology sounds strange to some aphasiologists, it may be as a result of excessive acceptance of the theoretical concept of disconnection, for example, the Wernicke-Lichtheim schema in aphasiology. Disconnectionists have asserted that echolalia corresponds to selectively preserved repetition, and consequently, they have identified mixed transcortical aphasia with predominant echolalia as a syndrome of "isolation of the speech area" (Goldstein, 1915; Geschwind, Quadfasel and Segarra, 1968). Goldstein (1915, p. 456) described two necessary conditions for the occurrence of echolalia: first, the presence of an intact, prompt functioning speech apparatus, and second, an isolation of this from the remaining areas of the brain, i.e., from the so-called concept field (Begriffsfeld). He asserted consequently that true echolalia is never caused by a lesion in the speech area itself. Geschwind et al. (1968) modified Goldstein's "isolation" hypothesis concerning the connecting pathway (the arcuate fasciculus) between the anterior and posterior speech area, and applied this explanation to their case of CO-intoxication with mixed transcortical aphasia exhibiting marked echolalia. Although this hypothesis has been widely accepted in modern aphasiology (Whitaker, 1976; Assal, Regli, Thuillard et al., 1983;

Bogousslavsky, Regli, and Assal, 1988; Grossi, Trojano, Chiacchio et al., 1991), some have raised objections to it (Brown, 1975; Liebaltd, Scheller und Schleip, 1975; Rapcsak, Krupp, Rubens et al., 1990).

Our three cases with effortful echolalia provide evidence against the "isolation" hypothesis. Disability of repetition and nonfluency indicate that their "speech apparatus" was far from a state of "intact, prompt functioning". Accordingly, we conclude that the ability to repeat and to produce fluent speech are not always necessary conditions for echolalia. Effortful echolalia undermines a basic premise of the "isolation" hypothesis.

Effortful echolalia may be analogous to a forced grasping reaction induced in an incompletely paretic hand, which is an automatic and compulsive movement evoked against the limitation of the motor disturbance. Although forced grasping occurred only in Case 2, this analogy leads us to believe that this type of echolalia is not only a form of automatic speech, but is also a compulsive speech phenomenon (Scheller, 1969; Kornyei, 1975). Indeed, all our patients expressed facial and gestural resistance to their echolalic repetition, and two commented that it occurred against their will.

Some recent neuropsychological studies of compulsive behaviour have emphasized its relationship with frontal lobe damage, for example, "compulsive manipulation of tools" (Mori and Yamadori, 1982), and "utilization" and "imitation behaviour" (Lhermitte, 1983, Lhermitte, Pillon and Serdaru, 1986). Scheller (1969) developed an understanding of echolalia as forced behaviour in speech and named this situation "loss of freedom" (Freiheitsverlust). Based on Kleist's (1934) investigations, he asserted that the syndrome of "loss of freedom," including forced grasping, sucking (oraler Einstellreflex), gaze (Zwangsblicken), reading (Zwanglesen) as well as repeating (echolalia), is caused by a pathological process in the frontal lobe. Lhermitte et al. (1986) proposed the term "environmental dependency syndrome" as a special manifestation of the loss of personal autonomy caused by frontal lobe damage: this may correspond to Scheller's concept of "loss of freedom" (1969). The imitation behaviour (Lhermitte et al., 1986) seems to us to be a modified form of echopraxia that is a forced imitation of gesture or behaviour in classical symptomatology. Echolalia as a special form of "loss of freedom" is generally of a compulsive nature. We consider that this attribute may be essential for the occurrence of echolalia and that the effortful echolalia in our patients demonstrated this situation most clearly.

It is not easy to diagnose the type of aphasia in our patients. Loss of speech initiative is a definitive characteristic of TCMA, while nonfluency and impaired repetition are generally considered as indispensable symptoms constituting Broca's aphasia syndrome. Goldstein (1915) described two types of TCMA in his voluminous monograph on transcortical aphasias: Type 1 TCMA can be regarded as an insufficient form of Broca's aphasia with relatively preserved repetition, better than spontaneous speech, caused by partial dysfunction of the "motor field"; and Type 2 is characterized by severely impaired speech initiation and intact repetition without dysfunction of the "motor field," probably corresponding to Luria's "dynamic aphasia." Accordingly, Type 1 TCMA in itself may be interpreted as intermediate between Type 2 TCMA and Broca's

aphasia with respect to the symptom of aphasia as well as to the site of lesion. Furthermore, no clear boundary can be fixed between Type 1 TCMA and Broca's aphasia, and a continuum is more likely (Rubens, 1976). Rubens (1975) stated, on the basis of his own observations and descriptions in the literature, that repetition improves disproportionately to spontaneous speech, despite initial total speechlessness, if the lesion is limited mainly to the mesial frontal area, but that, when there is a combination of superficial mesial and deep capsular lesions, repetition falls to the level of spontaneous speech and the disturbance is not distinguishable from severe Broca's aphasia.

Our patients with effortful echolalia may also indicate the presence of this continuum. The Z-score JSLTA profile confirmed that auditory comprehension and repetition were disturbed to a mild or moderate degree in each of our patients. Case 3, with mild impairment of repetition in the chronic stage, falls within Type 1 TCMA, because the severely disturbed comprehension in the acute stage improved remarkably. We are unable to reach a diagnosis of Type 1 TCMA in Cases 1 and 2, because comprehension in Case 1 and repetition in Case 2 were moderately impaired. We conclude that Cases 1 and 2 lie between Type 1 TCMA and Broca's aphasia on the above-mentioned continuum.

The cerebral lesion common to our patients was in the inner side and subcortical white matter of the frontal lobe and the anterior portion of the basal ganglia in the left hemisphere. Traditional localization theory in neuropsychology, in which aphasic nonfluency is attributed to Broca's area in the posterior part of the left inferior frontal gyrus (Dejerine, 1914), can explain the nonfluent speech in case 3 only. Another hypothesis, which locates the nonfluency in the lower part of the left pre- and post-central gyri (Knopman, Selnes, Niccum et al., 1983), could not account for the findings in Cases 1 and 2. Naeser, Palumbo, Helm-Estabrooks et al. (1989) hypothesized that persistent nonfluency is caused by combined lesions of two subcortical white matter areas in the left hemisphere: the most rostral portion of the medial subcallosal fasciculus, located in the lateral angle of the frontal horn (extremely deep to Broca's area), and the periventricular white matter near the body of the lateral ventricle, deep to the lower motor/sensory cortex area for the mouth. This subcortical hypothesis may explain the nonfluent speech in our patients.

A lesion of the medial portion of the left frontal lobe seems to be of importance in the occurrence of effortful echolalia. Some authors have claimed that damage to the supplementary motor area (SMA) in the left medial frontal lobe is associated with echolalia (Kornyei, 1975; Rubens, 1975), but others have not observed echolalia in cases with lesions including the SMA (Racy, Jannotta and Lehner, 1979; Masdeu, Schoene and Funkenstein, 1978). Ross (1980) reported two cases (his Cases 2 and 3) with TCMA due to damage to the left SMA, but he observed echolalia only in Case 3. There are also some reports of patients with the left medial frontal lesion including the SMA who exhibited TCMA, forced grasping or grasp reflex, and the "alien hand" sign, i.e. apparently purposeful movements dissociated from conscious volition (Goldberg, Mayer and Togliola, 1981; McNabb, Carrol and Mastaglia, 1988). These authors suggested an association between these compulsive movements or behaviours independent of conscious volition and damage to the SMA and related areas in

the left medial frontal lobe. In our patients the MRI-findings showed that the left SMA was damaged cortically in Case 1 and subcortically in Case 2, but was spared in Case 3, so that the lesion responsible for effortful echolalia does not appear to lie solely in the left SMA. Our experience and the literature indicate that the medial frontal lobe, including the SMA, may play an important role in the occurrence of the echolalia of a compulsive nature that we have described here as an effortful type, but we are still unable to limit the responsible lesion solely to the left SMA.

REFERENCES

- ASSAL, G., REGLI, F., THUILLARD, F., STECK, A., DERUAZ, J.-P., et PERENTES, E. Syndrome d'isolement de la zone du langage. Etude neuropsychologique et pathologique. *Revue Neurologique*, 139: 417-424, 1983.
- BENSON, D.F. *Aphasia, Alexia, and Agraphia*. New York: Churchill Livingstone, 1979.
- BOGOUSLAVSKY, J., REGLI, F., and ASSAL, G. Acute transcortical mixed aphasia. A carotid occlusion syndrome with pial and watershed infarcts. *Brain*, 111: 631-641, 1988.
- BROWN, J.W. The problem of repetition: A study of "conduction" aphasia and the "isolation" syndrome. *Cortex*, 11: 37-52, 1975.
- DEJERINE, J. *Sémiologie des Affections du Système Nerveux*. Paris: Masson et Cie, 1914.
- GESCHWIND, N., QUADFASSEL, F.A., and SEGARRA, J.M. Isolation of the speech area. *Neuropsychologia*, 6: 327-340, 1968.
- GOLDBERG, G., MAYER, N.H., and TOGLIA, J.U. Medial frontal cortex infarction and the alien hand sign. *Archives of Neurology*, 38: 683-686, 1981.
- GOLDSTEIN, K. *Language and Language Disturbances*. New York: Grune and Stratton, 1948.
- GOLDSTEIN, K. Die transkortalen Aphasien. *Ergebnisse der Neurologie und Psychiatrie*, 2: 349-629, 1915.
- GOODGLASS, H., and KAPLAN, E. *The Assessment of Aphasia and Related Disorders*. Philadelphia: Lea and Febiger, 1972.
- JAPANESE SOCIETY OF APHASIOLOGY (Ed.) *Standard Language Test for Aphasia*. Tokyo: Hohmeido, 1977.
- GROSSI, D., TROJANO, L., CHIACCHIO, K., SORICELLI, A., MANCI, L., POSTIGLIONE, A., and SALVATORE, M. Mixed transcortical aphasia: Clinical features and neuroanatomical correlates. A possible role of the right hemisphere. *European Neurology*, 31: 204-211, 1991.
- KNOPMAN, D., SELNES, O.A., NICCUM, N., RUBENS, A.B., YORK, D., and LARSON, D. A longitudinal study of speech fluency in aphasia: CT-correlates of recovery and persistent nonfluency. *Neurology*, 33: 1170-1178, 1983.
- KORNYEY, E. Aphasie transcorticale et écholalie: Le problème de l'initiative de la parole. *Revue Neurologique*, 131: 347-363, 1975.
- LHERMITTE, F. "Utilization behaviour" and its relation to lesions of the frontal lobe. *Brain*, 106: 237-255, 1983.
- LHERMITTE, F., PILLON, B., and SERDARU, M. Human autonomy and the frontal lobe. Part 1: Imitation and utilization behaviour: A neuropsychological study of 75 patients. *Annals of Neurology*, 19: 326-334, 1986.
- LHERMITTE, F. Human autonomy and the frontal lobe. Part 2: Patient behaviour in complex and social situations: The "environmental dependency syndrome". *Annals of Neurology*, 19: 335-343, 1986.
- LIEBALDT, G.T., SCHELLER, H., und SCHLEIP, I. Zur Frage der anatomischen Lokalisation echolalischer Phänomene in ZNS. Ein Beiträge zur Pathologie der Spontaneität der menschlichen Sprache. *Nervenarzt*, 46: 422-433, 1975.
- MASDEU, J.C., SCHOENE, M.D., and FUNKENSTEIN, H. Aphasia following infarction of the left supplementary motor area. *Neurology*, 28: 1220-1223, 1978.
- MENON, A.W., CARROL, W.M., and MASTAGLIA, F.L. "Alien hand" and loss of bimanual coordination after dominant anterior cerebral artery territory infarction. *Journal of Neurology, Neurosurgery and Psychiatry*, 51: 218-222, 1988.
- MORI, E., and YAMADORI, A. Compulsive manipulation of tools and pathological grasp phenomenon. *Clinical Neurology*, 22: 329-335, 1982.
- NAESSER, M.A., PALUMBO, C.L., HELM-ESTABROOKS, N., STIASSNY-EDER, D., and ALBERT, M. Severe nonfluency in aphasia. Role of the medial subcortical fasciculus and other white matter pathways in recovery of spontaneous speech. *Brain*, 112: 1-38, 1989.
- PICK, A. On pathology of echographia. *Brain*, 47: 417-429, 1924.

- POECK, K. *Klinische Neuropsychologie* (2. Auflage). Stuttgart: Georg Thieme Verlag, 1989.
- RACY, A., JANNOTTA, F.S., and LEHNER, L.H. Aphasia resulting from occlusion of the left anterior cerebral artery. Report of a case with an old infarct in the left rolandic region. *Archives of Neurology*, 36: 221-224, 1979.
- RAPCZAK, S.Z., KRUPP, L.B., RUBENS, A.B., and REIM, J. Mixed transcortical aphasia without anatomic isolation of the speech area. *Stroke*, 21: 953-956, 1990.
- ROMBERG, M.H. *Lehrbuch der Nervenkrankheiten der Menschen* (Dritte Auflage), Berlin: Verlag von August Hirschwald, 1857.
- ROSS, E.D. Left medial parietal lobe and receptive language functions: Mixed transcortical aphasia left anterior cerebral artery infarction. *Neurology*, 30: 144-151, 1980.
- RUBENS, A.B. Aphasia with infarction in the territory of the anterior cerebral artery. *Cortex*, 11: 239-250, 1975.
- RUBENS, A.B. Transcortical motor aphasia. In H. and H.A. Whitaker (Eds.), *Studies in Neurolinguistics, Vol. 1*. New York: Academic Press, 1976, pp. 293-303.
- SHELLER, H. Zur Anthropologie der Verhaltensstoerungen bei Stirnhirnprozessen (Aspontaneitaet, Greifen, Sperren, Echosymptome als Freiheitsverlust). *Nervenarzt*, 40: 557-560, 1969.
- STENGEL, E. A clinical and psychological study of echo-reactions. *Journal of Mental Science*, 93: 598-612, 1947.
- TONKONOGY, J., and GOODGLASS, H. Language function, foot of the third frontal gyrus, and Rolandic operculum. *Archives of Neurology*, 38: 486-490, 1981.
- WHITAKER, H. A case of the isolation of the language function. In H. and H.A. Whitaker (Eds.), *Studies in Neurolinguistics, Vol. 2* New York: Academic Press, 1976, pp. 1-58.
- Kazuo Hadano, M.D., Department of Psychogeriatrics, National Institute of Mental Health (NCNP), 1-7-3 Kohnodai, Ichikawa, Chiba, 272, Japan. E-mail: hadano@ncnp-k.go.jp

(Received 15 February 1997; accepted 15 July 1997)