

# Dysgraphia in two forms of conduction aphasia

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## Abstract

Recent clinical observations, in the absence of experimental data, appear to suggest that written expression in conduction aphasics parallels their speech (Goodglass, 1992). The current study undertakes an analysis of word level writing in two conduction aphasics, and attempts to explore the posited ‘parallel’ relationship between speech production deficits and deficits in written expression. JL, a 66-year-old female with left posterior parietal lobe lesion and PP, a 65-year-old female with a left posterior temporo-parietal lobe lesion served as subjects of this study. Their response patterns on Boston Naming Test (BNT) and written naming task (John Hopkins Dysgraphia Battery) were utilized to verify the parallel hypothesis. Although both cases have exhibited phonological and semantic paraphasias on BNT, PP’s overall performance was far superior to that of JL. JL produced numerous multiple responses to stimuli compared to PP’s occasional multiple responses. PP’s performance on the written naming task was far inferior to that of JL. JL’s predominant error pattern in writing was the production of phonologically similar words to the target words. This paper argues that such seemingly contradictory, unpredicted patterns can be parsimoniously better explained, not by the parallel hypothesis but by current cognitive-neuropsychological models of writing.

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## 1. Introduction

Wernicke’s, 1874 initial neuro-anatomical model of language representation suggested that an interruption of the fiber tract that is deep to the insular cortex that connects the center for acoustic images of speech and the motor center for speech production could result in a disconnection syndrome of aphasia. Wernicke predicted that such a syndrome would be characterized by fluent speech, good auditory comprehension but poor verbal repetition for auditorily presented speech. In the subsequent years, Wernicke reported two cases that matched his predictions and named the syndrome as ‘conduction aphasia.’ However, Wernicke did not offer post-mortem confirmation of site of lesion in either case. Over the decades that followed Wernicke’s conceptualization of conduction aphasia, this syndrome has evolved far beyond the limits of its original characterization.

This metamorphosis of conduction aphasia can be seen in changes in the areas of (1) lesion sites, (2) core symptom characteristics, and (3) classification of conduction aphasia, and the underlying mechanism responsible for the symptoms.

The issue of site of lesion associated with conduction aphasia remained elusive and problematic to the Wernicke–Lichtheim–Geschwind model of language. Geschwind’s (1970) account of conduction aphasia argues that the lesions typically occur in the arcuate fasciculus beneath the lower parietal lobe to disconnect the Wernicke’s area from Broca’s area. Once again, focal lesion occurring in the arcuate fasciculus in such cases has not been documented at autopsy. Furthermore, lesion studies on conduction aphasia have identified lesions in a number of different areas including the posterior portion of the superior temporal gyrus, supra-marginal gyrus, angular gyrus or insular cortex of the left hemisphere (Anderson et al., 1999; Green & Howes, 1978). It is also perplexing to note that frontal lobe lesion was reported in a case with conduction aphasia

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(Sherrmata, Andrews, & Pandya, 1974). Cases of conduction aphasia without lesions in the arcuate fasciculus have also been reported (Damasio & Damasio, 1980). More recent studies identified two predominant patterns of lesions: (1) posterior superior temporal gyrus, and (2) inferior parietal lobe of the left hemisphere (Axer, von Keyserlingk, Berks, & von Keyserlingk, 2001; Bartha & Benke, 2003). A few conduction aphasics were reported with more extensive lesions involving temporal and parietal lobes of the left hemisphere (Axer et al., 2001). The role of the posterior superior temporal gyrus in speech production was further confirmed by recent functional neuroimaging studies (Hickok, Erhard, Kassubek, Helms-Tillery, & Naeve-Velguth, 2000) and stimulation studies (Quigg & Fountain, 1999). Thus, the role of the arcuate fasciculus lesion in producing conduction aphasia appears to be attenuated by increasing number of studies that identified lesions in the posterior superior temporal lobe or inferior parietal lobe of the left hemisphere.

Another dimension of development of the concept of conduction aphasia syndrome pertains to the core symptoms with which it has been associated. The original conceptualization of conduction aphasia as a disconnection syndrome only predicted a core deficit of repetition in the absence of auditory comprehension impairments or speech production deficits (Wernicke, 1874). Wernicke's reasoning was based on the assumption that the center for auditory language (memory for acoustic images of words) and Broca's areas are essentially intact when the lesion occurs only in the fiber tract that interconnects these two language related cortical centers. However, Wernicke's two cases of conduction aphasia had an additional symptom of altered sound patterns of the target words, known as, literal or phonemic paraphasias. Both repetition deficits and literal or phonemic paraphasias have been reported in several studies on conduction aphasia. For the diagnostic identification of conduction aphasia, at least for some researchers, repetition deficit was not the defining characteristic. Phonemic paraphasia is the major indispensable feature of conduction aphasia (Goodglass, 1992). This particular view differs from Wernicke's disconnection theory of conduction aphasia that predicted the occurrence of repetition deficit as the central feature. Other researchers observed that each of these two symptoms may characterize different type of conduction aphasia (Shallice & Warrington, 1977).

From a historical perspective, it is interesting to note that conduction aphasia that was conceptualized as a unitary syndrome came to be further sub-divided in more than one way. The homogeneity of the syndrome of conduction aphasia was supported by some researchers (Bartha & Benke, 2003; Goodglass, 1992), whereas, heterogeneity of this syndrome received support from others (Axer et al., 2001; Caplan, Vanier, & Baker,

1986; Caplan & Waters, 1992; Caramazza, Basili, Koller, & Berndt, 1991; Demeurisse & Capon, 1991; Hillis & Caramazza, 1989; Kohn, 1992; Nadeau, 2001; Roeltgen, 1994, 2000; Shallice & Warrington, 1977). A classification of conduction aphasia that was based on the two symptoms of repetition deficits and phonemic paraphasias was introduced by Shallice and Warrington (1977) and was further elaborated by other researchers (Caplan et al., 1986; Nadeau, 2001), is as follows: (1) repetition conduction aphasia, and (2) reproduction conduction aphasia. Repetition conduction aphasia was characterized by failure to repeat verbal stimuli due to auditory short-term memory impairment. In such cases, spontaneous speech was said to be unaffected. In reproduction conduction aphasia, it was proposed that the problem in repetition was due to a deficit in encoding phonological form of words and the associated sequential articulatory gestures. Axer et al. (2001) identified two types of conduction aphasias on the basis of lesion sites: (1) suprasylvian and (2) infrasyllvian conduction aphasias. Axer et al. (2001) claim that these two forms are actually equivalent to reproduction and repetition conduction aphasias, respectively. The performance of suprasylvian conduction aphasics on auditory comprehension and repetition tasks was found to be better than that of the infrasyllvian conduction aphasics. However, the incidence of phonemic paraphasias was much higher in suprasylvian conduction aphasics than in infrasyllvian group. These researchers also report that lesions extending to both infra- and suprasylvian regions of the left hemisphere produced symptoms of both types. In addition, the mixed group's performance was found much inferior to that of the other two groups on tasks related to writing, naming, and comprehension (Axer et al., 2001).

Studies on conduction aphasia, as summarized above, have largely targeted phonemic paraphasias and repetition deficits. There were very few studies that looked at other modalities of language performance, such as auditory language comprehension (Caramazza & Zurif, 1976; Zurif & Caramazza, 1976), and written expression (Axer et al., 2001; Goodglass, 1992; Marcie & Hecaen, 1979). But, what is quite conspicuous in the literature on conduction aphasia is the lack of comprehensive, theoretically motivated studies on graphic performance of conduction aphasics. There are a few clinical observations on the occurrence of spelling errors. For instance, Roeltgen (1985) summarizes the clinical observations of other researchers (Marcie & Hecaen, 1979) thus: "Agraphia with conduction aphasia is characterized by misspelling and overwriting" (p. 78). Such errors were said to parallel the phonological production errors in conduction aphasics (Goodglass, 1992). In sum, only the modality of verbal language production in conduction aphasics was subjected to sophisticated cognitive neuropsychological and neurolinguistic

investigations. Although, most proponents of cognitive neuropsychological approach have strong reservations about the existence of aphasia syndromes (Caramazza, 1984; Schwartz, 1984) the application of cognitive neuropsychological methods in the study of aphasia syndromes such as Broca's aphasia, Wernicke's aphasia, and conduction aphasia continue to take place. Thus, the use of cognitive neuropsychological methods in the study of dysgraphia in conduction aphasia is entirely justifiable. Comprehensive, model oriented studies of written production in conduction aphasia still remain to be undertaken. The current study offers empirical data and argues for the expansion of the syndromic features of conduction aphasia to include types of deficits in written production.

Goodglass' (1992) observation about the parallels between the phonological production deficits and dysgraphia characteristics in conduction aphasia deserves further exploration, especially, in the context of recent developments in cognitive neuropsychological studies of dysgraphia and the models of writing that prompted and benefited from such studies. The 'parallel deficit' view appears to be based on the assumption that writing requires phonological mediation. Contrary to this assumption, current research in cognitive neuropsychology supports the autonomy of lexical orthography (Miceli, Benvegnu, Capasso, & Caramazza, 1997; Rapp, Benzing, & Caramazza, 1997). There is a robust body of clinical literature that supports the autonomous or semi-autonomous status of lexical orthography (Basso, Taborelli, & Vignola, 1978; Hier & Mohr, 1977; Miceli et al., 1997). Selective impairment or preservation of lexical orthography has been reiterated in these studies. Contemporary account of the cognitive architecture of spelling recognizes (1) lexical, (2) post-lexical, and (3) sub-lexical processes (Rapp, 2002; Rapcsak & Beeson, 2002). These processes can be selectively impaired in brain damaged cases. Models of word level writing in response to dictation, written naming of pictures, and copy encoding explain spelling performance in terms of these processes and their components.

The objectives of the current study are (1) to undertake an analysis of word level writing in two participants with conduction aphasia, and (2) to verify the claim that there are parallels between phonological production errors and writing errors in conduction aphasics.

## 2. Method

### 2.1. Subjects

The participants of the current study were two persons with conduction aphasia in their chronic stage: JL and PP. JL, a 66-year-old Caucasian female and a former bank accountant suffered a non-hemorrhagic

stroke and subsequently developed symptoms of conduction aphasia (see Fig. 1, BDAE profile of JL). She had exhibited an urge to self-correct her production errors (*conduit d'approche*). Phonemic and semantic paraphasias were the predominant characteristics of speech production.

At 2 years post-onset, JL was tested on Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). As JL's BDAE profile (Fig. 1) indicates, her auditory and reading comprehension performance was much better than her verbal performance as revealed by her patterns of errors on measures of fluency, naming, repetition, and paraphasias. She had also produced neologistic responses on confrontation naming and repetition tasks. JL underwent CT scan examination twice in her course of stay at a hospital. According to the medical report, the second CT scan examination done 2 weeks post-onset revealed a resolving cortical infarction in the posterior temporo-parietal lobe (see Fig. 3). In addition, there was bilateral white matter atrophy of the cortex.

PP, a 65-year-old Caucasian female suffered an ischemic stroke. According to her medical report, a CT scan examination revealed a lesion in the left temporal-parietal regions (see Fig. 4). However, PP's lesion in the left temporo-parietal cortex was more extensive than that of JL. Additionally, PP's brain did not evidence bilateral white matter atrophy. PP had fluent aphasic symptoms at 2 weeks post-onset. At 3 years post-onset, PP was tested on Boston Diagnostic Aphasia Examination and her performance was suggestive of patterns of conduction aphasia (see Fig. 2). PP's auditory comprehension was much better than her performance on verbal tasks as revealed in her fluency, repetition, and paraphasia raw scores and percentiles.

A comparison of the BDAE profiles of JL and PP indicates the following: (1) on naming task, PP's performance was far superior to that of JL, (2) JL produced greater number of paraphasias than did PP, with no neologistic paraphasias in the verbal production of PP, and (3) repetition was found impaired in both cases to variable degree. At word level, PP's repetition was much better than that of JL. Yet another feature that distinguishes JL from PP is the tendency exhibited by JL to produce multiple responses to produce target words resulting in '*conduit d'approche*.'

### 2.2. Materials

The present study used two test batteries: (1) Boston Naming Test (Goodglass & Kaplan, 1983), and (2) The John Hopkins Dysgraphia Battery (Goodman & Caramazza, 1986).

The Boston Naming Test is a test that targets picture naming ability of subjects. It consists of 60 stimulus pictures that represent names of high, mid, and low fre-

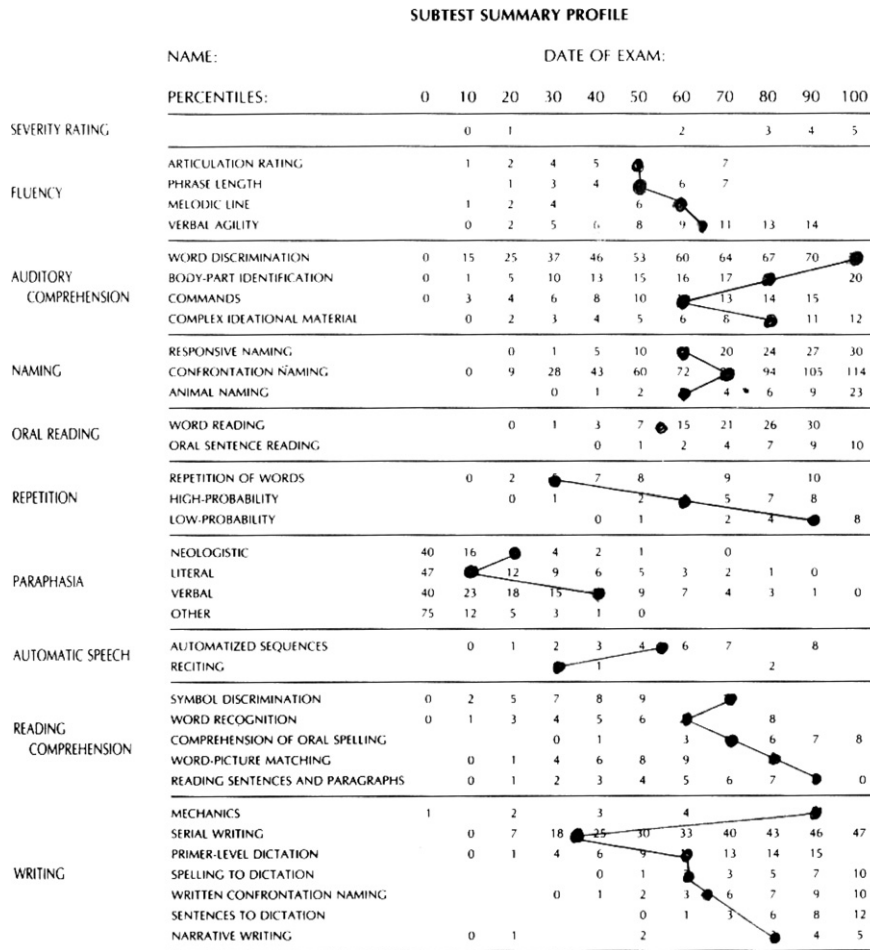


Fig. 1. JL's BDAE profile.

quency of occurrence. These pictures are arranged sequentially in a descending order of frequency. Thus, the test would prove to be increasingly more challenging to the examinees, as the test progresses. Norms are available only for adults of 18–59 years of age.

The Johns Hopkins University Dysgraphia Battery is an unpublished, nonetheless popular, test among clinicians, and researchers for assessing written skills. This battery consists of several sub-tests that assess the cognitive processes (lexical, post-lexical, and sub-lexical) underlying graphic performance at word level. Five sub-tests that were utilized in the present study include: (1) concreteness, (2) parts of speech, (3) word length, (4) written naming, and (5) copy transcoding. At this point, there are no norms available for the experimental version of JHUDB.

### 2.3. Procedures

JL was tested at home. PP was tested at a University Speech–Language and Hearing Clinic. Boston Naming Test and The John Hopkins University Dysgraphia Battery (JHUDB) were administered over 10 sessions. Both

subjects were encouraged to let the examiner know when they needed a break. First, the Boston Naming Test was administered to both subjects, followed by the JHUDB.

In administering the Boston Naming Test (BNT) the following procedures were used. For each stimulus picture, when a subject fails to give any response, a stimulus cue was given to facilitate the naming process. If the stimulus cue (semantic in nature) did not facilitate the production of target name, a phonemic cue in the form of the initial sound of the target name was given. A subject's score on Boston Naming Test will include the number of spontaneously given correct responses plus the number of correct responses following phonemic cues. The maximum possible score on BNT is 60. Paraphasic responses (literal, neologistic, and verbal) were also identified.

Three of the five sub-tests of JHUDB involved writing to dictation (concreteness sub-test, parts of speech sub-test, and word length sub-test). The copy transcoding sub-test involved copying the target words. The written naming task required production of written names for stimulus pictures. Responses to all target words in each sub-test were graded either correct or incorrect.

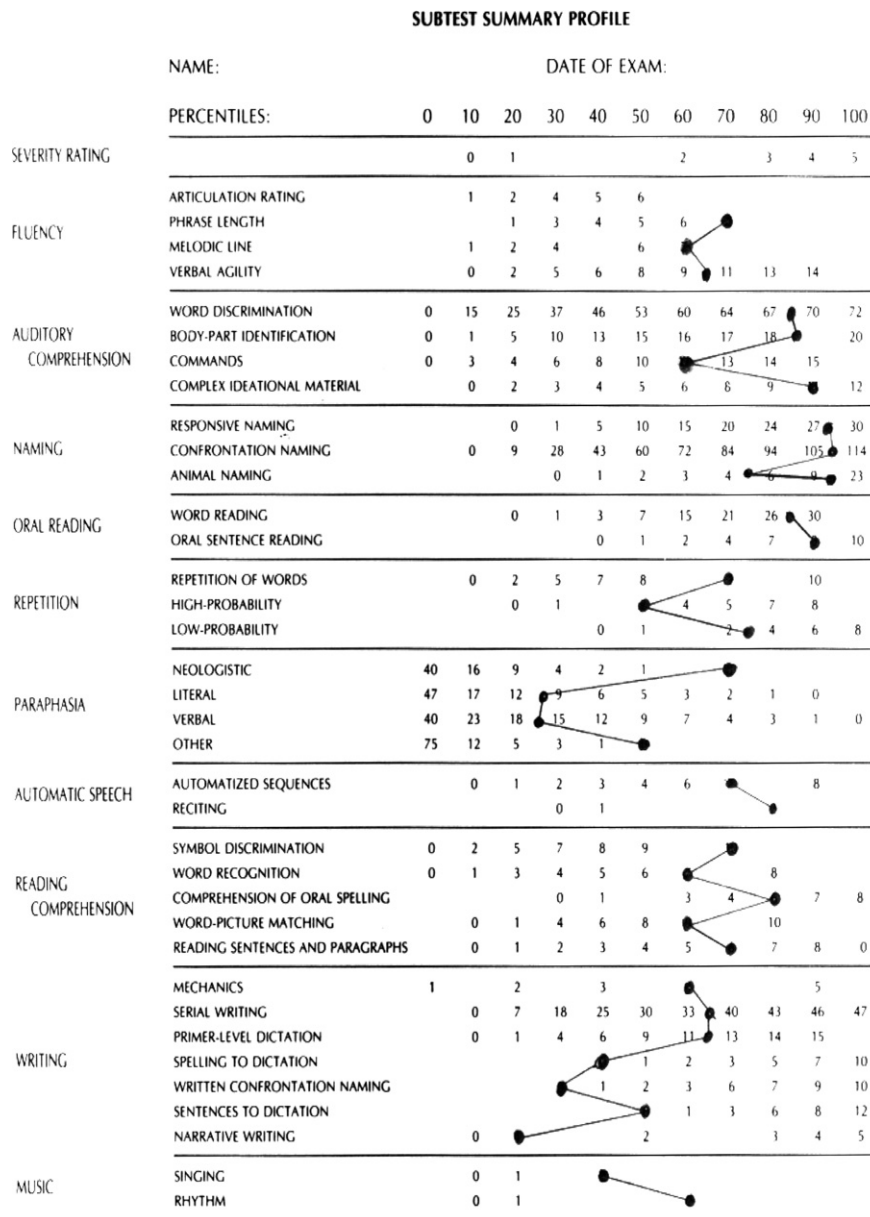


Fig. 2. PP's BDAE profile.

A correct written response is one without errors. Each correct response was given one point, and incorrect ones were given a zero point. Maximum possible scores for each of the JHADB sub-test are as follow: concreteness sub-test (42), part of speech sub-test (138), word length sub-test (70) written naming (51), copy transcribing (62), and phoneme-grapheme conversion list (110).

**3. Results and discussion**

JL's performance on BNT indicated that she produced 17 correct responses, 13 of those were spontaneously correct, and four responses followed the phonemic cues. In other words, JL performed at

28.3% accuracy level in naming a total of 60 pictures. Error analysis of JL's responses revealed that she produced multiple responses for 20 target stimuli. These responses were mostly in the form of phonological approximates to the target names. Thus, they constitute what is known as conduit d'approche. In her multiple attempts to produce the target names, JL also produced names that were semantically related to the target. JL also produced neologistic jargons in response to the picture naming task. These features are unique to JL.

PP's performance on BNT was far superior to that of JL. The total number of correct responses was 48, 41 of those responses were spontaneously correctly produced and the remaining seven were produced following phonemic cuing. Thus, PP performed at 80% accuracy level



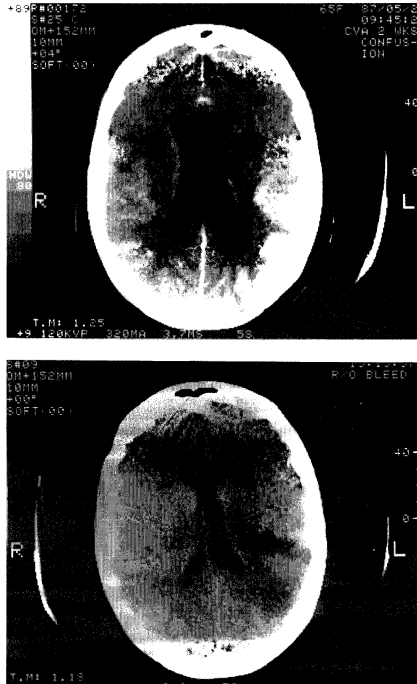


Fig. 3. JL's CT scans indicate lesion in the left posterior parietal lobe.

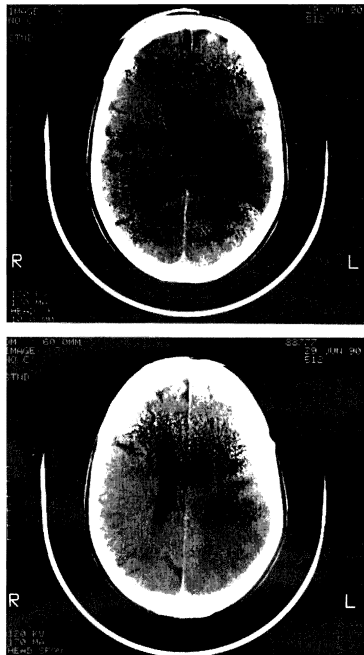


Fig. 4. PP's CT scans indicate lesion in the left temporo-parietal lobes.

in naming a total of 60 pictures of BNT. Her error patterns included both phonemic and semantic paraphasias. In the process of picture naming, PP received fewer stimulus cues and phonemic cues than did JL.

Both PP and JL produced phonemic (literal) paraphasias, verbal paraphasias, and neologistic responses on BDAE tasks (see Figs. 1 and 2). The BNT task added more information about their verbal speech characteris-

tics. JL's conduction aphasia is of 'conduit d'approche' type, whereas PP evidenced conduction aphasia without such a tendency to produce multiple responses to each stimulus. Besides, BDAE and BNT profiles suggest that PP's performance on all speech related tasks was superior to that of JL.

According to Goodglass (1992), dysgraphia in conduction aphasia will parallel their speech characteristics. One may predict that JL's graphic performance will be parallel to her verbal characteristics. In other words, JL's written production will be far more seriously impaired than that of PP. One may also expect multiple responses for each stimulus on writing tasks and phonologically related responses in written production.

On the other hand, if the lexical orthography is autonomous and not phonologically mediated in writing, as claimed by cognitive neuropsychologists, then, there will be no parallel deficits in writing in conduction aphasia. These predictions can be tested by examining the performance of both JL and PP on various sub-tests of JHUDB.

On the sub-tests of JHUDB, JL's scores and percent correct responses are as follow: (1) written naming: 37.2% (19/51), (2) word length: 30% (21/70), (3) concreteness: 19% (8/42), (4) part of speech: 17.8% (26/138), (5) copy transcoding: 93.5% (58/62), and (6) phoneme-grapheme conversion: 43.6% (48/110).

PP's scores on five sub-tests of JHUDB are as follows: (1) written naming: 3.9% (2/51), (2) word length: 0% (0/70), (3) concreteness: 0% (0/42), (4) parts of speech: 0% (0/138), (5) copy transcoding: 100% (62/62), and (6) phoneme-grapheme conversion list: not administered.

The patterns of performance of JL and PP on these sub-tests of JHUDB are not in accord with the predictions based on verbal characteristics that were uncovered by BNT and BDAE. PP's written production was profoundly impaired in all sub-tests of JHUDB, except copy transcoding sub-test. On the other hand, JL's performance showed moderate-severe impairment on five out of six sub-tests of JHUDB. In contradistinction to the predictions made above, JL's performance is actually better than that of PP on these sub-tests, except the copy transcoding sub-test. These results are puzzling and confusing to the adherents of the neoclassical localizationists. In order to shed more light on the nature of dysgraphia in these two forms of conduction aphasia, an error analysis of responses given to written naming task was undertaken (see Figs. 5A and B).

Error responses were classified following the categories of errors described in the JHUDB. The categories include (1) phonologically plausible errors (PPE), (2) phonologically implausible non-words (PINs), (3) don't know (DK), (4) semantic errors (SEMANTIC), (5) visually/phonologically similar words (VSW), (6) semantic/visually similar, (7) partial responses, and (8) other/mis-

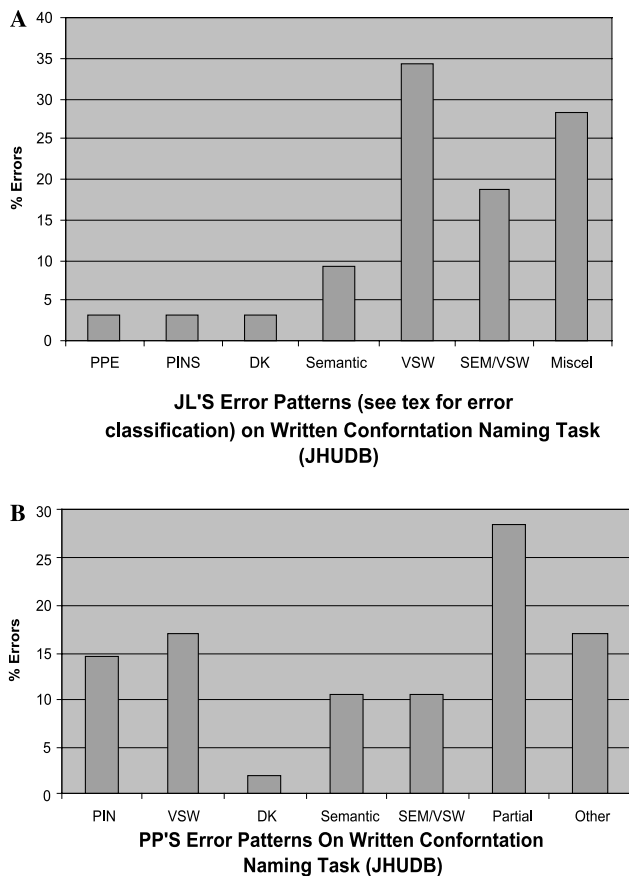


Fig. 5. (A and B) Present error patterns in written performance of JL and PP, respectively.

cellaneous responses (see Figs. 5A and B). A closer look at the error patterns bring out some of the commonalities and differences between JL and PP in their performance on written confrontation naming task (JHUDB). In JL's written production errors, the visually/phonologically similar word responses (VSW) was the major error category accounting for nearly 35% of her errors on this particular task. PP's error patterns included large number of partial responses that included the first letter of the target word and graphemes that are not part of the target word. PP also produced many more phonologically implausible non-words than did JL. Both JL and PP produced nearly same number of semantically related errors. Yet another unique feature of PP's written error production was the complete absence of the category called 'phonologically plausible errors' (PPE). Thus, PP's written naming is more seriously impaired than JL's. The absence of PPE category of error, large number of PIN and partial responses attest to this observation. However, it seems that both JL and PP are impaired, to varying degrees, in lexical, post-lexical, and sub-lexical processes of writing.

The present study explored the clinical observation that writing errors parallel the verbal production errors in conduction aphasia. Data obtained from PP do not

offer support to the 'parallel' view, whereas data obtained from JL appear to offer some support to the parallel view. However, JL's data can be explained in terms of the impaired modular cognitive processes such as the lexical, post-lexical, and sub-lexical processes.

It is also important to recognize that PP with temporo-parietal lesion has experienced greater problems in writing. This finding accords well with previous studies that reported severe problems in writing (Axer et al., 2001).

Lastly, the present study offers data that appear to warrant a revision of the symptom complex of conduction aphasia syndrome to reflect on the characteristics of dysgraphia noted in JL and PP. However, before a decision is made in favor of syndrome expansion, more case studies need to be carried out to fully explore the dysgraphia characteristics in conduction aphasics. In the current study, important tasks such as oral spelling could not be carried out because both JL and PP were unavailable for further testing. As reviewed in Section 1 of this paper, the syndrome of 'conduction aphasia' has undergone revisions before and it is hard to say that this syndrome will not undergo further modification in future.

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