

Language Deviations in Aphasia: A Frequency Analysis

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Thirty right-handed left hemisphere-damaged patients were taken and divided into five groups (transcortical motor, Broca, conduction, Wernicke, and anomic aphasia). Language deviations were scored and analyzed for the Picture Description (Plate No. 1, *The Cookie Theft*), Repetition (Words, High and Low Probability Sentences), and Naming (Responsive Naming, Confrontation, and Body-part naming) subtests of the Boston Diagnostic Aphasia Examination—Spanish version (Goodglass & Kaplan, 1979). A classification of paraphasias is proposed. Language deviations were scores for the following groups: Literal paraphasias (phoneme omissions, additions, displacements, and substitutions), verbal paraphasias (formal, morphologic, semantic, and unrelated), syntagmatic paraphasias, circumlocutions (object description and instrumental function), indefinite anaphors, and neologisms. Frequency of different types of language deviations is presented in the five aphasia groups. It was found that some paraphasic errors appeared in several aphasia groups; others were characteristics of specific aphasic syndromes. © 1993 Academic Press, Inc.

An aphasic deviation is any speech error produced by an aphasic patient (Ryalls, Valdois, & Lecours, 1988). Aphasic deviations were originally described by Wernicke (1874). Kussmaul (1877) coined the term "paraphasia" and distinguished literal and verbal paraphasias; in the former case there is a confusion between phonemes; in the latter, the two forms present some semantic resemblance to each other.

During the past few decades, a great deal of research in the aphasia realm has been devoted to the analysis of paraphasias. Jakobson (1964) interpreted paraphasias as a paradigmatic error in the process of selec-

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tion. Lecours and Lhermitte (1969) showed that there are many different types of errors in aphasic language deviations and several types of paraphasias can be distinguished. Blumstein (1973) observed that phoneme substitution represents the most frequent type of error in literal paraphasias, followed by simplification, environmental, and addition errors. Lecours (1975) presented a systematic analysis of transformations of language in aphasia and proposed the concepts of *paradigmatic distance*, the degree of similarity between replaced and replacing units, and *formal similarity index*, the number of phonemes common to both the replaced and the replacing words.

The distinction between "phonemic" and "phonetic" levels has been analyzed by different authors (Blumstein, 1973; Lecours & Caplan, 1975; Lecours & Lhermitte, 1969; Ryalls et al., 1988). Phonetic-level errors (e.g., [t] → [t']) are not usually referred to as "phonetic paraphasias," but rather as "phonetic distortions" (Ryalls et al., 1988). The name "paraphasia" is not applied to such phonetic errors because they are not high-level language disorders, but rather inappropriate realizations of the phonemes. As a general rule, in motor aphasias phonetic deviations are abundant. However, some of the presumed phonemic paraphasias found in motor aphasias could be the result of phonetic deviations so pronounced that phonemes are wrongly perceived (Buckingham, 1989a; Lecours & Caplan, 1975); in this sense, they would be phonemic changes for the listener, but phonetic distortions for the speaker (Buckingham & Yule, 1987). For the patient, it is a motor articulatory deficit and for the listener a phonemic paraphasia. Luria (1976) emphasizes that some literal paraphasias found in afferent motor aphasia (conduction aphasia) are perceived as phonemic substitutions, but are articulatory in origin.

Different types of paraphasias can be distinguished. Paraphasias can result from an inappropriate phoneme sequence. This type of paraphasia is named literal (or phonemic) paraphasia. Literal errors can be due to omissions, additions, displacement, or substitutions of phonemes (Blumstein, 1988; Buckingham, 1989a,b; Lecours, 1975; Ardila, Montañes, Caro, Delgado, & Buckingham, 1989).

A verbal paraphasia is interpreted as the erroneous use of a word belonging to an inventory of the language in place of another word which also belongs to one of the language inventories (Lecours, Trepagnier, Naesser, & Lavallo-Huvnh, 1983; Ryalls et al., 1988). Different subtypes of verbal paraphasias can be distinguished. A formal verbal paraphasia is a transformation in which the substituting word and the substituted word are similar in terms of their form but not their meaning (e.g., pear → dare (Blanken, 1990; Lecours & Rouillon, 1976). Formal verbal paraphasias might be considered a particular type of phonemic paraphasias (Lecours et al., 1983).

Morphemic verbal paraphasia refers to an inappropriate word that has

been, however, assembled using morphemes belonging to the language inventory (e.g., *summerly*) (Lecours & Lhermitte, 1972; Lecours, 1975). The resulting word can be acceptable from the point of view of the language, but unacceptable for the current context. These innovations (creation of a new word by combining existing morphemes in a new way) are particularly observed in Wernicke aphasia (Liederman, Kohn, Wolf, & Goodglass, 1983). When the resulting word is unacceptable from the point of view of the language (deviations sometimes named neologisms, but also blends, hybrids, or *telescopes*), this can be due to: (1) incorrect affixation, (2) simultaneous encoding of two phonologically related lexical items which do not have semantic similarity, (3) simultaneous encoding of two semantically related words which may also be phonologically associated; and (4) perseveration or anticipation of part of a word in the sentential string (Buckingham, 1981b).

Semantic verbal paraphasia designates the aphasic transformation in which the substituting and the substituted words are close in meaning (e.g., *table* → *chair*). Semantic verbal paraphasias observed in aphasics can fall under different headings: (1) the substituting and the substituted words belong to the same semantic field (e.g., *lion* → *tiger*); (2) they are antonymous (e.g., *big* → *small*); (3) the target word is replaced by a superordinate (e.g., *lion* → *animal*); as a matter of fact, aphasic language frequently recurs to words with a high level of generality but low content (e.g., *thing*, *stuff*, etc.); and (4) there is an environmental proximity between the substituting and the substituted words (e.g., *cigarette* → *matches*) (Ryalls et al., 1988).

In addition to these three types of verbal paraphasias (formal, morphemic, and semantic), at times a patient can introduce a word that, given the context, does not seem phonologically or semantically related to what appears to be required. This deviation is named unrelated to verbal paraphasia (Green, 1969; Buckingham, 1989a) (e.g., *it has been colorful to come to the hospital*).

It should be pointed out that a paraphasia does not always refer to a single word. Substitutions can appear among linguistic units more complex than words (e.g., *the acuarium of the fish* → *the cage of the lion*); this last type of substitution represents a syntagmatic paraphasia.

Some additional types of language deviations should be mentioned. Object description (e.g., *snow* → *soft, white, and cold*) and instrument function (e.g., *watch* → *for knowing the hour*) are often observed in aphasic language. The anaphor is a word that has an antecedent occurring before or after to which it refers (Buckingham, 1981a). Aphasics will often use anaphors for which a referent is nonexistent (indefinite anaphors (e.g., *I read it*. That is, if previously some word such as *book*, *letter*, or *newspaper* has not been mentioned, *it* will be an indefinite anaphor).

The neologism is a phonological form for which it is impossible to re-

TABLE 1
Substitution Errors Found in Aphasia

Phonetic distortions (perceived as such by the listener)
Literal paraphasias
Phonemic paraphasias
Omissions
Additions
Displacements
Substitutions
Articulatory paraphasias (perceived to be phonemic paraphasias)
Verbal paraphasias
Formal verbal paraphasias (phonological relation)
Morphologic verbal paraphasias
Semantic verbal paraphasias (semantic relation)
Same semantic field
Antonyms
Superordinate
Proximity
Unrelated verbal paraphasias
Syntagmatic paraphasias
Circumlocutions
Object description
Instrumental function
Indefinite anaphors
Neologisms

cover with any reasonable degree of certainty some single item or items in the vocabulary of the patient's language as it presumably existed before the onset of the disease (Buckingham & Kertesz, 1976). In other words, it is not possible to identify the target word; however, it is almost always possible to identify its grammatical category based on its position and inflection. Neologism may in some cases be due to a double error: a lexical selection error which, before it reaches phonetic materialization, is subsequently distorted phonemically.

Table 1 presents a summary of the main language deviations found in aphasia.

Jargonaphasia is a descriptive term that refers to fluent, well-articulated language that has no meaning from the point of view of the listener (Brown, 1981; Ryalls et al., 1988). The absence of meaning is due to the abundance of paraphasias and neologisms. Different types of jargonaphasia can be distinguished: phonemic jargon, semantic jargon, and neologistic jargon (Kertesz, 1985). However, the three types of deviations are usually found together, although one of them can predominate. Neologis-

tic and semantic jargon can be occasionally confused with schizophrenic language (Benson, 1979).

The purpose of this research was to carry out a further analysis of the frequency of different types of language deviations in an unselected group of right-handed left hemisphere-damaged patients.

METHOD

Subjects. Thirty right-handed, monolingual Spanish-speaking aphasic patients with left hemisphere damage were studied (12 women, 18 men; average age 37.56, age range 18–65). These subjects presented various etiologies (vascular = 20; tumoral = 7; traumatic = 3). The cerebral damage had evolved in a period varying from 1 to 4 months. Patients had no background of previous neurological or psychiatric illness. Average schooling was 8.26 years (range 4–16). All lesions were corroborated by means of computerized axial tomography (CAT). Table 2 presents the general characteristics of the sample.

The patients included in the sample were taken from the neuropsychology services of the San Juan de Dios Hospital and the Colombian Institute of Neurology (Bogotá, Colombia). The following criteria for inclusion were used: (a) a single focal CAT lesion; (b) no background of previous neurological or psychiatric illness; (c) age range between 16 and 65 years old; and, (d) right-handedness.

Procedure. In addition to the general neurological and neuropsychological exams, the following tests were given to each patient: (1) the Boston Diagnostic Aphasia Examination—Spanish version (Goodglass & Kaplan, 1979), and (2) the Token Test—shortened version (De Renzi & Faglioni, 1978). Patients were divided into five groups, and the following criteria were jointly considered: results on the Boston Diagnostic Aphasia Examination, the Token Test, and the general neurological and neuropsychological examination. The following groups were formed: (1) transcortical motor aphasia (left prefrontal damage) (TMA—five patients), (2) Broca's aphasia (BA—five patients), (3) conduction aphasia (CA—six patients), (4) Wernicke's aphasia (WA—11 patients), and (5) anomic or amnesic aphasia (AA—three patients).

The criteria for inclusion in the different groups, taken from Goodglass and Kaplan (1979), Lezak (1983), and Ardila and Ostrosky-Solis (1991) were:

1. TMA: Auditory Comprehension (except for complex material), Naming, and Repetition subtests in the BDAE present positive Z scores. Comprehension according to the Token Test is normal, or reduced only for complex commands. There is a noticeable reduction in spontaneous language, and the Narrative Writing subtest has a score below 2. In the

TABLE 2
General Characteristics of the Sample

Patient	Sex	Age	Years of education	Etiology	Category	BDAE auditory comprehension	BDAE naming	BDAE repetition	Token
1	M	55	6	Vascular	TMA	+0.75	+1.00	+0.50	30
2	M	20	11	Vascular	TMA	+1.00	+1.00	+1.50	27
3	F	56	16	Tumor	TMA	+0.50	+0.75	+1.00	26
4	M	45	4	Vascular	TMA	+0.50	+1.00	+1.50	24
5	F	23	11	Tumor	TMA	+1.00	+1.00	+1.50	26
6	M	26	16	Vascular	Broca	+0.50	+1.00	-0.25	26
7	M	36	16	Vascular	Broca	+0.50	+0.75	-0.25	30
8	F	25	5	Vascular	Broca	0.00	-1.00	-1.00	18
9	F	46	5	Vascular	Broca	-0.25	-1.00	-1.00	12
10	F	22	5	Vascular	Broca	-0.75	+0.25	-0.75	14
11	M	35	5	Tumor	Conduction	+0.50	+1.00	-0.25	26
12	F	61	8	Vascular	Conduction	+0.75	-1.25	-1.25	22
13	F	39	5	Vascular	Conduction	+0.75	+0.50	-1.00	31
14	M	33	8	Vascular	Conduction	+1.00	+0.50	-0.25	32
15	F	43	5	Tumor	Conduction	+1.00	+1.50	-0.25	31
16	F	26	11	Vascular	Conduction	+1.00	+1.50	-0.50	27
17	M	63	6	Vascular	Wernicke	-0.50	0.00	-1.00	17
18	M	34	11	Tumor	Wernicke	-0.25	0.00	+0.25	18
19	M	31	8	Vascular	Wernicke	-1.00	0.00	-0.50	14
20	F	48	5	Vascular	Wernicke	-1.00	-0.50	-1.00	18
21	M	18	9	Trauma	Wernicke	-1.00	-1.00	-1.00	15
22	F	53	4	Vascular	Wernicke	-1.50	-1.00	-1.50	5
23	M	28	5	Trauma	Wernicke	-1.00	-0.50	+0.25	22
24	M	63	9	Vascular	Wernicke	-1.00	-0.50	-0.25	14
25	M	46	16	Tumor	Wernicke	-1.00	-0.50	0.00	8
26	M	28	5	Trauma	Wernicke	-0.75	-0.25	-1.50	18
27	M	46	4	Vascular	Wernicke	-0.25	+0.50	+0.25	24
28	F	43	7	Tumor	Anomic	+1.00	-0.25	+0.25	30
29	M	65	11	Vascular	Anomic	+0.50	0.00	+1.50	28
30	M	30	11	Vascular	Anomic	0.00	-0.50	+0.75	24

neuropsychological evaluation perseveration and/or attentional defects are observed.

2. BA: Fluency scores are notoriously lower than Auditory Comprehension and Naming scores. Repetition *Z* scores are negative. Comprehension defects according to the BDAE and the Token Test are present in a variable degree. Some degree of right hemiparesis is observed.

3. CA: Auditory Comprehension scores are higher than Repetition scores. Auditory Comprehension scores present positive *Z* score values, while Repetition *Z* scores are negative. Fluency *Z* scores are zero or positive. Mild right hemiparesis and sensory loss can be observed. At least some degree of ideomotor apraxia is observed.

4. WA: Fluency scores are positive while Auditory Comprehension is impaired (negative *Z* scores). *Z* scores for Naming subtests are negative or around zero. Repetition is variable, but at least some moderate defects are observed. General neurological examination can be normal.

5. AA: Repetition scores are normal. Naming *Z* scores are negative. Auditory Comprehension scores can be decreased, but they are higher than Naming scores. Verbal memory can be impaired. The general neurological examination can be normal.

Paraphasia analysis. Paraphasias were scored and analyzed for the Picture Description (Plate No. 1, *The Cookie Theft*), Repetition (Words, High and Low Probability Sentences), and Naming (Responsive Naming, Confrontation, and Body-part naming) subtests of the Boston Diagnostic Aphasia Examination. Classification of paraphasias was performed according to the classification system presented in Table 1. Language samples were both audiotaped and transcribed in written form. However, phonetic deviations were not taken into account. Phonemic literal paraphasias and articulatory literal paraphasias were taken together; as a matter of fact, this division represents a more theoretical than clinical distinction. When a single word contained different types of errors (e.g., a phoneme substitution in a semantic verbal paraphasia) each type of error was counted independently. When a single word contained the same error repeatedly (e.g., two phoneme additions) it was counted as a single paraphasia. Paraphasias were independently scored by the two authors of this research. Eight hundred thirty-three language deviations were analyzed; agreement in classification was found in 817 (98.1%) cases. For the 16 nonagreement cases, a detailed analysis was carried out until an agreement was reached.

Neuroimaging. In order to transcribe the patients' lesions, a template was designed with 10 standard scanner cuts from the base of the brain to the highest region. The lesions corresponding to the different patients in the same category or group were then superimposed onto the template, following a combination approach derived from the methods described by Kertesz (1983), Naeser (1983), and Damasio and Damasio (1989) in order to identify the critical zone in the appearance of the deficit analyzed. Figures 1–5 present the CT scans superimposition for the different aphasia groups.

RESULTS

Table 3 presents the general results obtained in the different aphasia groups. Literal paraphasias are strongly associated with perisylvian aphasia syndromes (BA, CA, and WA). The frequency of literal paraphasias in other groups is very low. In general, standard deviations are very high; this high dispersion can result from the unavoidable heterogeneity of patients: some of them presented just a mild language disorder, while others presented with a very severe aphasic syndrome.

In TMA only some formal verbal paraphasias are observed. One patient presents a semantic verbal paraphasia, and another patient a syn-

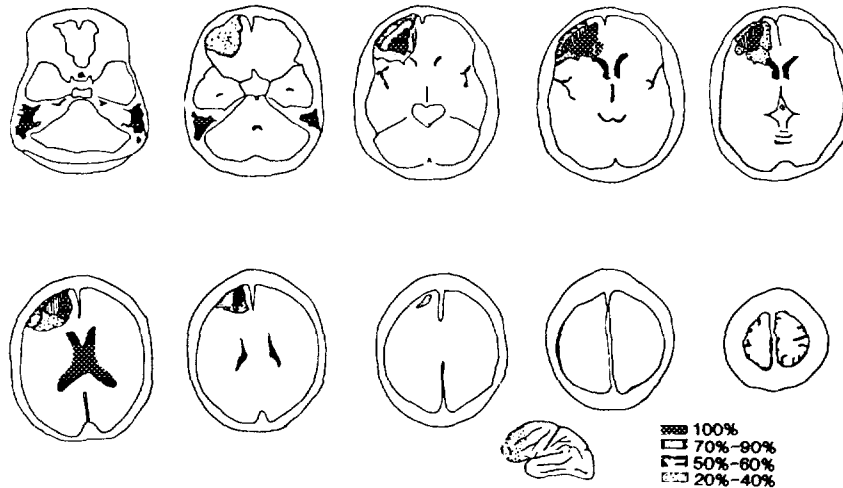


FIG. 1. CAT superimposition: Transcortical motor aphasia. (Reproduced with permission from Ardila, Rosselli, & Pinzón, 1989.)

tagmatic paraphasia. Three of the patients do not present any language deviation.

In BA subjects, the frequency of literal paraphasias is very high (average = 18.2 per patient), especially due to phoneme omissions (46% of the literal errors) and substitutions (40% of the literal errors). Although these patients present some other types of paraphasias, their frequency is low and they do not appear in all but only in some individuals.

In CA frequency of literal paraphasias is particularly high (average =

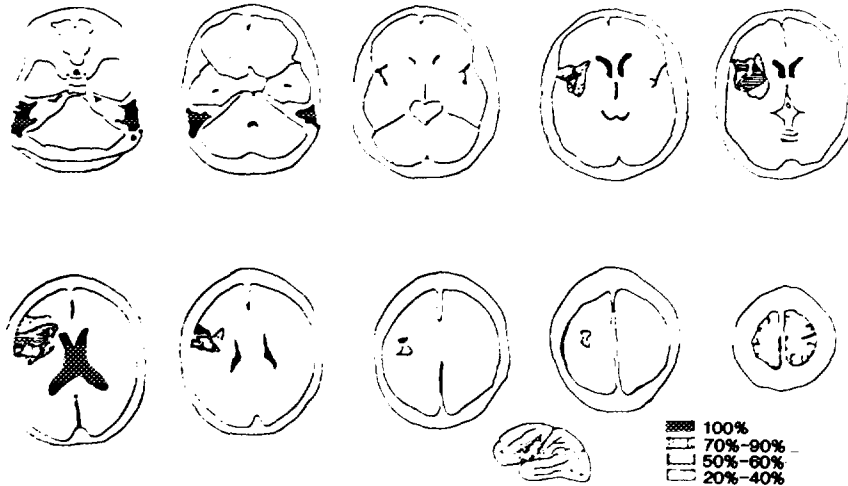


FIG. 2. CAT superimposition: Broca's aphasia. (Reproduced with permission from Ardila, Rosselli, & Pinzón, 1989.)

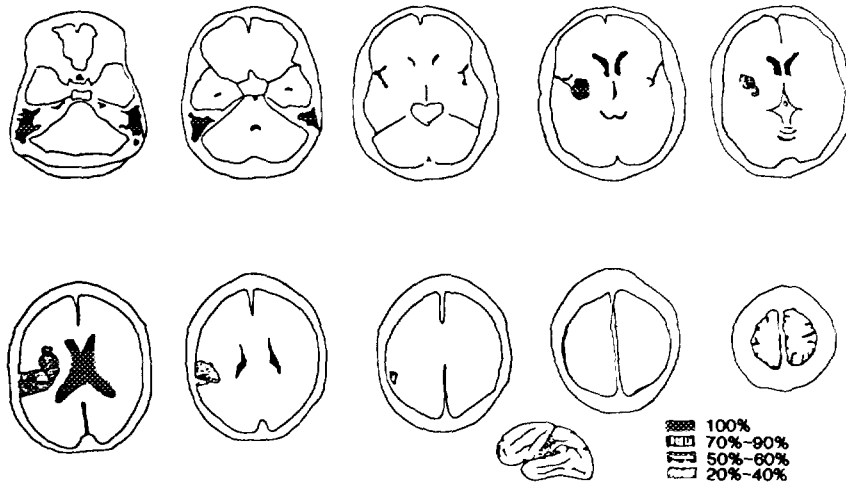


FIG. 3. CAT superimposition: Conduction aphasia. (Reproduced with permission from Ardila, Rosselli, & Pinzón, 1989.)

12.8 per patient). Numerous literal paraphasias are observed in all the patients. These individuals present mostly substitutions (55% of the literal errors), but also omissions (21%), additions (10%), and displacements (14%). Three patients (50% of the cases) present one or two verbal semantic paraphasias, and three patients present irre recognizable words (neologisms). The rest of the deviations have a very low frequency.

All the language deviation types are observed in WA patients. Literal paraphasias (average = 16.8 per patient) and neologisms (average = 9.9 per patient) clearly predominate. In literal paraphasias, substitutions



FIG. 4. CAT superimposition: Wernicke's aphasia. (Reproduced with permission from Ardila, Rosselli, & Pinzón, 1989.)

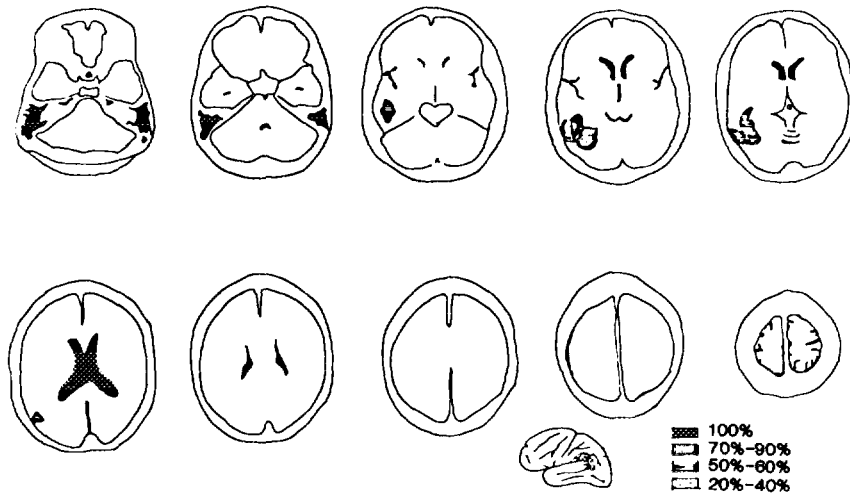


FIG. 5. CAT superimposition: Anomic aphasia. (Reproduced with permission from Ardila, Rosselli, & Pinzón, 1989.)

predominate (57% of literal errors). The total number of verbal paraphasias (average = 18.9 per patient) and literal paraphasias are roughly similar in frequency. Semantic confusions within the same semantic field (average = 5.2 per patient), formal verbal (average = 4.6 per patient) and unrelated verbal paraphasias (average = 4.5 per patient), are maximum within the verbal paraphasic errors.

AA patients present mostly semantic verbal paraphasias, due to substitutions within the same semantic field (average = 7.0 per patient). Occasionally, superordinate words are used (average = 1.0 per patient). One patient presents two unrelated verbal paraphasias, and each one of the three subjects presents one circumlocution. Only one patient presents two literal paraphasias resulting from phoneme substitutions.

The ratio "literal paraphasias/verbal paraphasias" was calculated for the different aphasic groups. This ratio was 4.63 for BA patients and 8.16 for CA subjects, implying a neat predominance of literal over verbal paraphasias. However, in WA this ratio was 0.94; that is, the amount of literal and verbal paraphasias was roughly equivalent. In TMA paraphasias were virtually absent; however, sporadically some verbal paraphasias were disclosed; no literal paraphasia was observed. In AA group a noticeable amount of verbal, especially semantic, paraphasias was observed. Literal paraphasias were virtually absent.

DISCUSSION

First, it is important to bear in mind that although all the patients presented a restricted focal brain damage, there is an unavoidable hetero-

TABLE 3
Mean Frequency of Paraphasias in Different Aphasic Groups

	TMA	BA	CA	WA	AA
Literal paraphasias					
Omissions	—	8.4 (5.6)	3.0 (2.3)	4.5 (4.3)	—
Additions	—	1.8 (1.2)	1.3 (1.1)	1.4 (1.2)	—
Displacements	—	1.0 (1.2)	1.7 (0.9)	1.0 (1.2)	—
Substitutions	—	7.4 (6.2)	9.5 (8.4)	9.6 (5.4)	0.6 (0.4)
Verbal paraphasias					
Formal verbal paraphasias	0.6 (0.8)	1.0 (1.0)	1.5 (0.9)	4.6 (4.5)	1.0 (0.8)
Morphologic verbal paraphasias	—	0.8 (0.4)	—	2.4 (1.5)	—
Semantic verbal paraphasias					
Same semantic field	0.2 (0.4)	2.2 (1.7)	0.8 (0.8)	5.1 (2.4)	7.0 (0.8)
Antonyms	—	—	0.2 (0.3)	0.5 (0.9)	—
Superordinate	—	—	—	1.5 (1.9)	1.0 (1.4)
Proximity	—	—	0.2 (0.3)	0.5 (0.4)	—
Unrelated verbal paraphasias	—	—	0.2 (0.3)	4.5 (6.0)	3.7 (5.1)
Syntagmatic paraphasias	0.2 (0.4)	—	—	0.5 (0.8)	—
Circumlocutions					
Object description	—	—	0.2 (0.3)	1.2 (2.0)	0.6 (0.4)
Instrumental function	—	—	—	1.5 (1.9)	0.6 (0.9)
Indefinite anaphors	—	—	0.3 (0.7)	0.2 (0.5)	—
Neologisms	—	1.0 (0.9)	2.3 (3.5)	10.9 (13.8)	—

Note. Standard deviations are in parentheses.

generality within and among the different aphasia groups. Any comparison across groups may therefore be only tentative. It is virtually impossible to match the different aphasic groups with regard to not only the severity of the language disorder, but also other potentially important variables, such as etiology, postonset time, educational level, etc. Our results can be considered just preliminary. However, some general trends are evident.

Literal paraphasias were associated with perisylvian aphasic syndromes (BA, CA, and WA). In other aphasia groups, literal paraphasias were virtually nonexistent. Semantic verbal paraphasias, unrelated verbal paraphasias, and circumlocutions appeared in AA and in to a lesser degree WA. In other aphasia groups (TMA, BA, and CA), these language deviations were rarely observed. As expected, neologisms were characteristics of WA; 84% of the neologisms found in our sample were found in WA patients. Syntagmatic paraphasias were only occasionally observed in aphasic patients; in our current sample, only one TMA and one WA patient presented this type of paraphasic error.

Difficulties in producing the words are observed in cases of frontal damage, particularly damage to Broca's area, the left supplementary motor area, and the area in front of and above Broca's area (Alexander, Benson, & Stuss, 1989; Alexander & Benson, 1991; Ardila & López, 1984; Novoa & Ardila, 1987; Stuss & Benson, 1986). Difficulties can involve different levels of word production: the selection of the word, the initiation of motor activity, the production of the required articulatory movements to produce the names, and the correct sequencing of phonemes in the word. This deficit has been named word production anomia (Benson, 1979, 1988). Kohn and Goodglass (1985) found phonemic paraphasias, irre recognizable words (nonwords), and even semantic paraphasias in picture naming tasks in BA. BA patients present an articulatory deficit characterized by slow and effortful speech, simplification of syllabic clusters, and phonemic assimilations (a previously produced phoneme wrongly appears in a following syllable; or a phoneme corresponding to a future syllable is anticipated). There is a reduction in the phonemes and syllable repertoire output. Phonetic deviations are evident. Irre recognizable words can be the result of multiple distortions of the target word. In our sample, literal paraphasias resulting from omissions and substitutions of phonemes were clearly the most frequently found types of errors. However, some verbal paraphasias were also observed.

In CA spontaneous language can be preserved but language deviations are abundant. Literal paraphasias are apparent when naming and especially when repeating, although the patient attempts to self-correct the deviations and produces progressive approximations to the target word (Berndt, 1988; Ardila & Rosselli, 1990, 1992). Automatized language is well produced without apparent effort. The target name, impossible to produce during the naming task, can be easily produced during casual conversation or when included in an automatized sequence. The paraphasia can have the correct number of syllables but contains one or more literal switches. The patient can state that he knows the name, but does not know how to say it (Benson, Sheretama, Bouchard, Segarra, Prince, & Geschwind, 1973; Ardila & Rosselli, 1990). This deficit has been named paraphasic anomia (Benson, 1988). Approximately 60% of literal errors

corresponded to phoneme substitutions. Omissions, additions, and displacement of phonemes were observed to a lesser degree.

Phonemic desintegration of language is observed in WA (Luria, 1966, 1976; Kertesz, 1985; Blumstein, 1988). The sequence of phonemes is inappropriate and literal paraphasias are abundant. The memory of the correct phonological sequence of lexical units is seriously impaired (Bachman & Albert, 1988). The sequence of phonemes is inappropriate and literal paraphasias are abundant. The memory of the correct phonological sequence of lexical units is seriously impaired (Bachman & Albert, 1988). In our sample of WA patients, an equivalent number of literal and verbal paraphasias was observed (ratio literal/verbal paraphasias around 1). WA patients have, however, in general, good use of inflections. Affixes are preserved, and because of this, frequently it is possible to recognize the target word or at least its linguistic category. Plurals and derivated affixes are recognizable. Morphologic (average = 2.4 per patient) and formal verbal (average = 4.6 per patient) paraphasias were evident.

Difficulties in distinguishing the semantic fields of words are especially prevalent in posterior aphasia, particularly in AA (Kremin, 1988). These patients cannot determine the semantic boundaries of a word; they even express great surprise that a single word can eventually have two different meanings (inability to understand polysemia) (Ardila, Lopez, & Montañes, 1983). Kudo (1987) observed that hierarchical semantical categorization is generally impaired in aphasia: subjective category domains are more diffusive in aphasics than in normals, due to an increase of peripheral members but not of central members. However, fluent aphasics present a profound desintegration of semantic boundaries; the semantic limits of words are lost. It has been observed, however, that fewer semantic paraphasias are produced in confrontation with operative items (Feyereisen, Van der Borgh, & Seron, 1988). In general, the underlying representation of semantic categories is preserved in BA but seriously disrupted in fluent WA and AA (Goodglass & Baker, 1976; Grober, Perceman, Kellar, & Brown, 1980). Word-finding difficulties and verbal paraphasias, particularly semantic but also unrelated verbal paraphasias, were abundant (see Table 3).

In general, AA is associated with a normal language function except for word-finding pauses, circumlocutions, and sheer failure to name. Repetition is normal, comprehension is fairly normal, and the language deficit is only observed when naming. Pointing to objects when given the name is better; the patient can describe the use of the object, indicating that this is not a visual agnosic deficit. Almost invariable structural pathology involves the posterior inferior portion of the temporal lobe (Brodmann area 37) and/or the angular gyrus (Benson, 1988; Goldfarb, & Halpern, 1989; Hart & Gordon, 1990). This anomia would be correlated with a memory retrieval deficit; the patient understands the word when it is

presented, that is, there is word memory storage, but cannot spontaneously retrieve it. Cueing notoriously can help in naming. In this group of patients, the overwhelming majority of paraphasias corresponded to semantic verbal paraphasias resulting from confusions within the same semantic field.

In summary, the relationships between language deviations and aphasia type are complex and multiple. Despite the fact that some language deviations clearly tend to predominate in specific forms of aphasias, this relationship is far from being simple. The analysis of language deviations not only assists, but also is a prerequisite for the understanding of aphasic language disturbances.

REFERENCES

- Alexander, M. P., Benson, D. F., & Stuss, D. T. 1989. Frontal lobes and language. *Brain and Language*, **37**, 356-391.
- Alexander, M. P., & Benson, D. F. 1991. The aphasia and related disturbances. In R. J. Joynt (Ed.), *Clinical neurology*. Philadelphia: Lippincott.
- Ardila, A., & Lopez, M. V. 1984. Transcortical motor aphasia: One or two aphasias? *Brain and Language*, **22**, 350-353.
- Ardila, A., & López, M. V., & Montañes, P. 1983. Afasia amnésica: Implicaciones para la psicología cognoscitiva [Amnesic aphasia: Implications for cognitive psychology]. *XIX Interamerican Congress of Psychology*. Quito (Ecuador).
- Ardila, A., Montañes, P., Caro, C., Delgado, R., & Buckingham, H. W. 1989. Phonological transformations in Spanish-speaking aphasics. *Journal of Psycholinguistic Research*, **18**, 153-180.
- Ardila, A., & Ostrosky, F. 1991. *Diagnóstico del daño cerebral: Un enfoque neuropsicológico* [Assessment of brain damage: A neuropsychological approach]. Mexico: Trillas.
- Ardila, A., Rosselli, M., & Pinzón, O. 1989. Alexia and agraphia in Spanish speakers. In A. Ardila & F. Ostrosky (Eds.), *Brain organization of language and cognitive processes*. New York: Plenum Press. Pp. 147-175.
- Ardila, A., & Rosselli, M. 1990. Conduction aphasia and verbal apraxia. *Journal of Neurolinguistics*, **5**, 1-14.
- Ardila, A., & Rosselli, M. 1992. Repetition in aphasia. *Journal of Neurolinguistics*, **7**, 1-11.
- Bachman, D. L., & Albert, M. L. 1988. Auditory comprehension in aphasia. In F. Boller, J. Grafman, G. Rizzolati, & H. Goodglass (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier. Vol. 1, Pp. 281-306.
- Benson, D. F. 1979. *Aphasia, alexia, and agraphia*. New York: Churchill Livingstone.
- Benson, D. F. 1988. Anomia in aphasia. *Aphasiology*, **2**, 229-236.
- Benson, D. F., Sheretama, W. A., Bouchard, R., Segarra, S. M., Prince, D. N., & Geschwind, N. 1973. Conduction aphasia: A clinicopathological study. *Archives of Neurology*, **28**, 339-346.
- Berndt, R. S. 1988. Repetition in aphasia: Implications for models of language processing. In F. Boller, J. Grafman, G. Rizzolati, & H. Goodglass (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier. Vol. 1, Pp. 329-348.
- Blanken, G. 1990. Formal paraphasias: A single case study. *Brain and Language*, **38**, 534-554.
- Blumstein, S. 1973. *A phonological investigation of aphasic speech*. The Hague: Mouton.
- Blumstein, S. E. 1988. Approaches to speech production deficits in aphasia. In F. Boller,

- J. Grafman, G. Rizzolati, & H. Goodglass (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier. Vol. 1, Pp. 349–366.
- Brown, J. W. 1981. *Jargonaphasia*. New York: Academic Press.
- Buckingham, H. W. 1981a. Lexical and semantic aspects of aphasia. In M. T. Sarno (Ed.), *Acquired aphasia*. New York: Academic Press. Pp. 183–214.
- Buckingham, H. W. 1981b. Where do neologisms come from? In J. Brown (Ed.), *Jargon-aphasia*. New York: Academic Press. Pp. 39–62.
- Buckingham, H. W. 1989a. Mechanisms underlying aphasic transformations. In A. Ardila & F. Ostrosky (Eds.), *Brain organization of language and cognitive processes*. New York: Plenum. Pp. 123–145.
- Buckingham, H. W. 1989b. Phonological paraphasia. In C. Code (Ed.), *The characteristics of aphasia*. London: Taylor & Francis. Pp. 89–110.
- Buckingham, H. W., & Kertesz, A. 1976. *Neologistic jargon aphasia*. Amsterdam: Zwets and Zeitlinger.
- Buckingham, H. W., & Yule, G. 1987. Phonemic false evaluation: Clinical and theoretical aspects. *Clinical Linguistics and Phonetics*, 1, 113–125.
- Damasio, H., & Damasio, A. R. 1989. *Lesion analysis in neuropsychology*. New York: Oxford Univ. Press.
- De Renzi, E., & Faglioni, P. 1978. Normative data and screening power of a shortened version of the Token Test. *Cortex*, 13, 424–433.
- Feyereisen, P., Van der Borgh, F., & Seron, X. 1988. The operativity effect in naming: A re-analysis. *Neuropsychologia*, 26, 401–415.
- Goldfarb, R., & Halpern, H. 1989. Impairments in naming and word fluency. In C. Code (Ed.), *The characteristics of aphasia*. London: Taylor & Francis. Pp. 33–52.
- Green, E. 1969. Phonological and grammatical aspects of jargon in an aphasic patient. *Language and Speech*, 12, 103–118.
- Goodglass, H., & Baker, E. 1976. Semantic field, naming, and auditory comprehension in aphasia. *Brain and Language*, 3, 359–374.
- Goodglass, H., & Kaplan, E. 1979. *Evaluación de las afasias y de trastornos similares* [Assessment of aphasia and related disorders]. Buenos Aires: Editorial Médica Panamericana.
- Grober, E., Perecman, E., Kellar, L., & Brown, J. W. 1980. Lexical knowledge in anterior and posterior aphasics. *Brain and Language*, 10, 318–330.
- Hart, J., & Gordon, B. 1990. Delineation of single-word semantic comprehension deficits in aphasia, with anatomical correlation. *Annals of Neurology*, 27, 226–231.
- Jakobson, R. 1964. Towards a linguistic typology of aphasic impairments. In A. De Reuck & M. O'Connor (Eds.), *Disorders of language*. London: Churchill.
- Kertesz, A. 1983. Localization of lesions in Wernicke's aphasia. In A. Kertesz (Ed.), *Localization in neuropsychology*. New York: Academic Press. Pp. 209–230.
- Kertesz, A. 1985. Aphasia. In J. A. M. Frederiks (Ed.), *Handbook of clinical neurology*. Amsterdam: Elsevier. Vol. 45, Pp. 287–331.
- Kohn, S. E., & Goodglass, H. 1985. Picture-naming in aphasia. *Brain and Language*, 24, 226–283.
- Kremin, H. 1988. Naming and its disorders. In F. Boller, J. Grafman, G. Rizzolati, & H. Goodglass (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier. Vol. 1, Pp. 307–328.
- Kudo, T. 1987. Aphasics' appreciation of hierarchical semantic categories. *Brain and Language*, 30, 33–51.
- Kussmaul, A. 1877. *Die storungen der Sprache*. Leipzig: Vogel.
- Lecours, A. R. 1975. Methods for the description of aphasic transformation of language. In E. H. Lenneberg & E. Lenneberg (Eds.), *Foundations of language development*. New York: Academic Press. Vol. 2, Pp. 75–94.

- Lecours, A. R., & Caplan, D. 1975. A review of "A phonological investigation of aphasic speech" by S. E. Blumstein (1973). *Brain and Language*, **2**, 237-254.
- Lecours, A. R., & Lhermitte, F. 1969. Phonemic paraphasias: Linguistic structures and tentative hypotheses. *Cortex*, **5**, 193-228.
- Lecours, A. R., & Lhermitte, F. 1972. Recherches sur le langage des aphasiques: 4. Analyse d'un corpus de néologismes (notion de paraphasie monémique). *Encephalé*, **61**, 295-315.
- Lecours, A. R., & Rouillon, F. 1976. Neurolinguistic analysis of jargonaphasia and jargon-agraphia. In H. Whitaker & H. Whitaker (Eds.), *Studies in neurolinguistics*. New York: Academic Press. Vol. 2, Pp. 95-144.
- Lecours, A. R., Trepagnier, C., Naesser, C. J., & Lavalle-Huvnh, G. 1983. The interaction between linguistics and aphasiology. In A. R. Lecours, F. Lhermitte, & B. Bryans (Eds.), *Aphasiology*. London: Bailliere Tindall. Pp. 292-320.
- Lezak, M.D. 1983. *Neuropsychological assessment*. New York: Oxford Univ. Press. Second ed.
- Liederman, J., Kohn, S., Wolf, M., & Goodglass, H. 1983. Lexical creativity during instances of word-finding difficulty: Broca's versus Wernicke's aphasia. *Brain and Language*, **20**, 21-32.
- Luria, A. R. 1966. *Higher cortical functions in man*. New York: Basic Books.
- Luria, A. R. 1976. *Basic problems of neurolinguistics*. The Hague: Mouton.
- Naeser, M. A. 1983. CT scan lesion size and lesion locus in cortical and subcortical aphasias. In A. Kertesz (Ed.), *Localization in neuropsychology*. New York: Academic Press. Pp. 63-119.
- Novoa, O. P., & Ardila, A. 1987. Linguistic abilities in patients with prefrontal damage. *Brain and Language*, **30**, 206-225.
- Ryalls, J., Valdois, S., & Lecours, A. R. 1988. Paraphasia and jargon. In F. Boller, J. Grafman, G. Rizzolati, and H. Goodglass (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier. Vol. 1, Pp. 367-373.
- Stuss, D. T., & Benson, D. F. 1986. *The frontal lobes*. New York: Raven Press.
- Wernicke, K. 1874. *Der Aphasische Symptomencomplex* [Aphasic symptomatology]. Breslau: Cohn & Weigert.