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Phonological Transformations in Conduction Aphasia

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Different explanations and subtypes of conduction aphasia are analyzed. Characteristics of literal paraphasias in parietal–insular conduction aphasia are discussed, emphasizing that paraphasias in conduction aphasia are articulatory-based (articulatory literal paraphasias) and due mainly to phoneme substitutions and phoneme deletions; they result basically in switches in phoneme manner and place of articulation. Similarities between errors in ideomotor apraxia and conduction aphasia language deficits are presented. It is proposed that language deviations (in oral as in written language) in conduction aphasia can be understood as a segmentary apraxia of speech.

Conduction aphasia, initially described by Wernicke in 1874, represents one of the most controversial aphasic syndromes. It is usually defined as an aphasia characterized by relatively fluent spontaneous language, good comprehension, and poor repetition with the presence of literal paraphasias (e.g., Benson, 1979; Benson & Ardila, 1993; Kertesz, 1979, 1985). Benson et al. (1973) point to three basic and five secondary characteristics of conduction aphasia. Basic characteristics are (1) fluent, paraphasic (usually literal) conversational speech; (2) near normal comprehension; and (3) repetition disturbances of a significant degree. Conduction aphasia very often also includes (1) naming disturbances (from literal paraphasic contamination to total inability to produce the appropriate word), (2) reading disturbances (comprehension is much better than reading aloud), (3) writing disturbances (from mild spelling difficulties to profound agraphia), (4) ideomotor apraxia (buccofacial and

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limb), and (5) elementary neurological abnormalities (some right hemiparesis and cortical sensory loss).

This language disorder has been called central aphasia (Goldstein, 1948), repetition aphasia (Kleist, 1934), afferent or kinesthesic motor aphasia (Luria, 1966, 1976), efferent conduction aphasia (Kertesz, 1985), reproduction conduction aphasia (Shallice & Warrington, 1977), as well as conduction aphasia (Benson, 1979, 1988; Geschwind, 1965; Hécaen & Albert, 1978; Lecours, Poncet, Ponzio, & Ramade-Poncet, 1983; Wernicke, 1874).

The *sine qua non* of the syndrome is the repetition defect. Nevertheless, this defect has been explained in different ways. The oldest and most frequent account has been in terms of disconnection (e.g., Geschwind, 1965; Damasio & Damasio, 1983; Wernicke, 1874). Some other authors, however, prefer to interpret conduction aphasia in terms of an apraxic deficit (Ardila & Rosselli, 1990; Brown, 1972, 1975; Luria 1966, 1976; Vinarskaya, 1971). Given this second interpretation, conduction aphasia would represent a verbal apraxia, or an ideomotor apraxia of speech (Brown, 1975), or a kinesthesic apraxia of speech (Luria, 1976).

The possibility of several mechanisms, each of them capable of giving rise to deficient repetition, has led to the postulation of two different forms of conduction aphasia: efferent/afferent (Kertesz, 1979, 1985), or reproduction/repetition (e.g., Caplan, Vanier & Baker, 1986; Shallice & Warrington, 1977). The efferent–reproduction type involves the phonemic organization and representation of words and correlates with parietal and insular lobe damage, whereas the afferent–repetition conduction aphasia involves short-term memory, affects the repetition of large stretches of material, and arises from temporal damage (e.g., Caramazza, Basili, Koller, & Berndt, 1981). Luria (1976) considered that what has been referred to as conduction aphasia corresponds to two different types of linguistic defects. He used the term afferent motor aphasia to refer to the efferent–reproduction parietal conduction aphasia type mentioned above. Luria considered this to involve an inability to analyze, manipulate, or otherwise appreciate the featural composition of movements required to produce language sounds (Luria's *articulemes*). He observed that this aphasia is indeed a type of kinesthesic apraxia of speech. The second type of conduction aphasia (afferent–repetition) is associated with short-term verbal memory deficits, and was included in Luria's acoustic–amnesic aphasia. In this paper, I will deal exclusively with the first type of conduction aphasia, i.e., parietal–insular conduction aphasia, or afferent motor aphasia.

Compared with other aphasia types, conduction aphasia patients

display a particularly high number of literal paraphasias, specially during language repetition tasks. Spontaneous language can fluctuate, in the sense that sometimes language is fluent and easily produced while on other occasions it is nonfluent, effortful, and paraphasic. The patient can easily produce one or several sentences, but, arriving at a particular word, he/she becomes totally unable to continue.

Ardila and Rosselli (1992) analyzed repetition errors in a large sample of aphasic patients. Literal errors, self-corrections, and approximations to the target word were evident in conduction aphasia individuals. A very notorious difference between high-probability and low-probability sentence repetition scores was observed. While scores in word repetition and high-probability sentence repetition were better than in Broca patients, scores in low-probability condition were only half of Broca patients' scores. When meaningful words or sentences were presented, their performance in repetition tests was very close to Broca and Wernicke patients.

CHARACTERISTICS OF PARAPHASIAS

Although conduction aphasia patients can present some phonetic deviations and verbal paraphasias, the majority of the switches in oral language correspond to literal paraphasias. Literal paraphasias are more frequently observed during repetition, and especially in pseudoword (logotome) repetition. Ardila and Rosselli (1993) calculated the ratio literal paraphasias/verbal paraphasias in different aphasic groups. The ratio observed was 4.63 for Broca's aphasia patients and 8.16 for conduction aphasia subjects, implying a neat predominance of literal over verbal paraphasias. In Wernicke's aphasics this literal paraphasias/verbal paraphasias ratio was only 0.94; that is, the amount of literal and verbal paraphasias was roughly equivalent in this last group of aphasic patients. Relatively, among aphasia subgroups, the highest amount of literal paraphasias was observed in conduction asphasia patients.

Some important language characteristics of conduction aphasia patients should be emphasized: (1) Conduction aphasia patients present successive phonological approximations to target words, and self-corrections (e.g., /kandado/ (lock) → /kardado/, /karbado/, /kaldado/, /kandado/), pointing to the fact that the acoustic image of the word is preserved; (2) the patient can easily recognize correctly and incorrectly produced words; (3) sometimes the subject is totally unable to produce a particular word in his/her spontaneous of repetitive language, and a moment later

he/she can produce the same word without any apparent effort. Language production is strongly context-dependent.

Some general rules in phonological switches in conduction aphasia have been observed. These switches has been found to be virtually identical even in quite different languages such as Russian and Spanish (Ardila, Montañes, Caro, Delgado, & Buckingham, 1989b; Ardila & Rosselli, 1990; Vinarskaya, 1971):

1. Simplification: Word phonemes tend to be replaced by more primary and easily produced language sounds. In this sense, Jakobson's rule would be applied: Earlier in life acquired phonemes (and consequently, simplier from the articulatory point of view) will tend to replace later acquired phonemes (e.g., /r/ → /l/); in a certain sense, phonological aphasic regression mirrors the phoneme acquisition in the child (Jakobson, 1956). Some 90% of the errors are due to phoneme substitutions and phoneme deletions.
2. Switches in phonemes are mainly a result of changes in the manner of articulation [e.g., /marina/ (marine) → /barina/] and the point of articulation [/bocado/ (bite) → /bocabo/]. In Spanish-speaking aphasics, these two types of switches (manner and point of articulation) account for about 90% of the paraphasias.
3. At least in Spanish language, consonantic changes are maximal (about 95% of the total number of switches), while vocalic changes are minimal (only some 5%). Vocalic changes are approximately six times more frequent in fluent aphasics (Wernicke's and anomics) and twice as frequent in Broca's aphasia than in conduction aphasics.
4. Phoneme substitutions [e.g., /maleta/(suitcase) → /paleta/] represent about 50% of the total number of literal errors; phoneme deletions [e.g., /marina/(marine) → /maina/] associated with articulatory simplification, represent about 25%; reduplicative substitutions [e.g., /lapis/(pencil) → /papis/] are about 15% of the total number of literal paraphasic errors. Phoneme additions [e.g., /tomate/ (tomate) → /tormate/] are the minimal (about 4%) among aphasic subgroups, and exchanges [e.g., /telefono/(phone) → /telenofo/] are virtually nonexistent (Ardila et al., 1989b).
5. As it is also observed in other aphasic syndromes, the majority of the phoneme substitutions include changes in only one or two phonemic features.

It is important to emphasize that the types of switches and the responsible mechanisms are the same in spontaneous as in repetitive lan-

guage. But in repetitive language, and particularly in the repetition of pseudowords, their frequency is highest.

Table I presents a summary of the types of changes and the mechanisms utilized in conduction aphasia literal paraphasias. Percentages are approximate, and are taken from Vinarskaya's (1971) Russian-speaking patients and Ardila et al.'s (1989b) Spanish-speaking patients. The percentages both authors presented in these two so different languages (Slavic and Latin) are virtually identical, except that Russian possesses a phonemic opposition nonexistent in Spanish (or English) between soft and hard production for some phonemes; Russian-speaking conduction aphasia patients show a great deal of error in regard to this soft–hard opposition.

THEORETICAL INTERPRETATIONS OF CONDUCTION APHASIA

Two main theoretical interpretations of conduction aphasia syndrome have been proposed: (1) According to the first explanation conduction aphasia is considered a verbal apraxia; (2) the second theoretical explanation emphasizes that conduction aphasia represents a disconnection syndrome. Both theoretical points of view will be briefly examined.

Table I. Characteristics of Literal Paraphasias in Conduction Aphasia; Relative Frequency of Different Types of Switches, and Mechanisms Utilized, According to Vinarskaya (1971) and Ardila et al. (1989b, 1990)

	Relative frequency
Type of change	
Manner of articulation	55%
Place of articulation	35%
Voiced/voiceless (oral stops)	5%
Vocalic changes	5%
Mechanisms utilized	
Substitution	52%
Deletion	25%
Reduplicative substitution	15%
Addition	4%
Reduplicative addition	3%
Exchange	1%

Conduction Aphasias as a Verbal Apraxia

The characteristics of linguistic errors found in conduction aphasia patients have led some authors to propose that errors in conduction aphasia correspond to apraxic-type errors. Conduction aphasia, according to such an interpretation, would represent a "segmentary ideomotor apraxia," or ideomotor apraxia of speech" (Brown, 1975), or a "kinesthesic apraxia of speech" (Luria, 1976).

Some clinical evidence seems to support this interpretation.

The defects in the movements usually found in ideomotor apraxias in general include the inability to perform the movement, simplifications or incompleteness, onomaopoeic responses, use of the hand (instead of the mouth in buccofacial apraxia), and self-cuing (Geschwind & Damasio, 1985); approximations and self-corrections should be also included. The movements performed by the patient are diffuse, amorphous, shortened, and deformed; two or more movements are incorporated into one; gestural enhancement is observed as are pantomimed context, body-part as object, and vocal overflow (Hécaen & Albert, 1978). Some of these apraxic characteristics are quite evident in the language production of conduction aphasia patients (e.g., simplifications, incompleteness, self-cuing, approximations, self-corrections, gestural enhancement, and verbal overflow). Others are not applicable for obvious reasons (e.g., body-part as object).

It is important to underline that characteristics of an ideomotor apraxia have been found to be present in patients with expressive language disorders. Liepmann (1908) showed an association between motor aphasia and buccofacial apraxia. Liepmann considered the defects in verbal expression as an apraxia in the use of the speech muscles. Several authors have emphasized the strong association between buccofacial apraxia and conduction aphasia (e.g., De Renzi, Pieczuro, & Vignolo, 1966; Geschwind, 1965); the presence of literal paraphasias in buccofacial apraxia (e.g., Poeck & Kerschensteiner, 1975; Tognola & Vignolo, 1980); and the correlation between suprasylvian conduction aphasia and ideomotor apraxia (Benson et al., 1973). Common mechanisms underlying both conduction aphasia and ideomotor apraxia could be supposed. In conduction aphasia, repetitive language is impaired, whereas spontaneous language is much better preserved, and even can be relatively normal. In ideomotor apraxia, the execution of movements is impaired only when they are required out of their natural context either on verbal command or by having the patient imitate a movement performed by the examiner (Geschwind & Damasio, 1985; Poeck, 1986). Spontaneous performance is always superior to performance under command, and this holds true

not only for conduction aphasia, but also for ideomotor apraxias in general. If conduction aphasia is interpreted as a verbal apraxia, it becomes understandable that spontaneous language is much better preserved than repetitive language.

Luria (1966, 1976) introduced the concept of *articuleme* to explain literal errors in conduction (afferent motor) aphasia. According to Luria, damage to the inferior postcentral left-hemisphere area will be associated with a kinesthesic apraxia for the movements used in language production. Articuleme refers to the specific articulatory manoeuver required to produce a specific language sound (phoneme). The inability to determine the positions of the mouth, lips, and tongue used for the production of language sounds represents the basic feature of the kinesthesic apraxia of speech. The patient does not know how to put and move correctly the tongue and lips to articulate language sounds. The patient will present deviated language sounds, distant from the acoustic point of view, but close from the articulatory point of view. Literal paraphasias in kinesthesic apraxia of speech or afferent motor aphasia would represent a result not from phoneme confusions, but from articuleme confusions. Since errors are articulatory-based and not phonologically based, literal paraphasias in conduction aphasia should be called articulatory literal paraphasias, to distinguish them from the phonemic literal paraphasias found in Wernicke's type of aphasia.

Buckingham (1981, 1989) has illustrated the existence of motor articulatory-based disorders as a source of phonological transformations in nonfluent aphasia: In motor aphasics there is a phonetic-level breakdown that alters articulation such that acoustic cues are altered, leading hearers to falsely identify phonemes as intended by the aphasic; voice onset time (VOT) errors would be the clearest instance of this in Broca's aphasia. Therefore, some literal paraphasias that appear as phonemic for the hearer are, according to Buckingham, articulatory-based.

Apraxia has been usually associated with left-parietal damage, particularly the lower parietal region in the angular and supramarginal gyri (Geschwind & Damasio, 1985). However, the most crucial areas in oral apraxia include the frontal opercula, the central (Rolandic) opercula, the first central convolution, and the anterior portion of the insula (Heilman, Rothi & Kertesz, 1983; Tognola & Vignolo, 1980). In consequence, not only parietal lobe, but insula, damage can also be associated with some forms of apraxia.

Conduction Aphasia as a Disconnnection Syndrome

Alternative explanations of conduction aphasia have been proposed. The initial and most frequent account has been in terms of disconnection (e.g., Damasio & Damasio, 1983; Geschwind, 1965; Wernicke, 1874). Wernicke originally proposed that the crucial locus of damage in conduction aphasia was the insula. Dejerine (1901, 1914), however, proposed the arcuate fasciculus as the main connecting pathway for the sensory and motor language areas. Supramarginal and/or parietal operculum damage involving the arcuate fasciculus was thus postulated as crucial to the syndrome of conduction aphasia. This topography has come to be generally accepted (Damasio & Damasio, 1980, 1983; Geschwind, 1965). Damasio and Damasio proposed that the repetition defect in conduction aphasia is based on the disconnection of posterior sensory pathways from anterior motor areas and further suggested that the repetition deficit follows damage in the subinsular pathways. Most reports suggest that brain damage in conduction aphasia follows pathology in the left parietal lobe (lower postcentral and supramarginal gyri) and/or the insula (Benson, 1988; Benson & Ardila, 1993; Benson et al., 1973; Kertesz, 1979, 1985; Damasio & Damasio, 1980, 1983; Demesurisse & Capon, 1991). A disconnection disorder resulting from the interruption between posterior auditory and anterior motor language areas would be observed.

PARALEXIAS AND PARAGRAPHIAS

When reading aloud, conduction aphasia patients present a considerable number of paralexias, while reading comprehension can be almost perfect or at least considerably better (Ardila, Rosselli, & Pinzón, 1989a; Benson, 1979; Kertesz, 1985). Errors in reading aloud parallel errors found in spoken language. Errors are observed specially in reading pseudowords. Letter substitution errors [e.g., VENTANA/bentana/(window) − > /benkana/] are the most common type of error and represent about 30% of the total number of reading errors. Anticipation errors [e.g., DROMEDARIO /dromedaryo/ (dromedary) − > /dromeraryo/]; literal reading [e.g., SOL/sol/(sun) − > /s/, /o/, /l/]; substitution of pseudowords for meaningful words [e.g., ASRILO (no meaning) − > /anijo/ (ring)]; letter omissions in reading (e.g., BRA − > ba); and letter additions [e.g., TU/tu/(you) − > /tus/] represent each one about 10% of the total number of errors found in conduction aphasia reading (Ardila et al., 1989a). Other types of errors are minimal.

In writing, letter substitution errors [e.g., VENTANA (window)

− > mentana] (about one-third of the total number of errors), letter omission errors [e.g., LIBRO (book) − > libo] (about one-fourth), and neologisms (incomprehensible words) (about one-fourth) clearly predominate. Other types of error are less frequently observed. Table II presents a summary of the main types of errors found in conduction aphasia patients' reading and writing (Ardila et al., 1989a).

Agraphia can be particularly severe in conduction aphasia. Sometimes conduction aphasic agraphia is associated with an apraxic agraphia (inability to perform the movements required to write letters). Usually copying is very superior to writing from dictation. Luria (1966) has named the writing disorder associated with conduction (afferent motor) aphasia as *afferent motor agraphia*. According to Luria, when writing we usually rely (to a certain extent) upon the repetition of the words to ourselves; this is particularly the case when writing complex and not very well-known words (or pseudowords). Therefore, if there is an apraxia in producing words and the patient tends to confuse close articulatory movements, when writing there will appear literal paragraphias, omission, and similar defects. In some cases, there is even a total inability to write from dictation. Occasionally the patient writes a word incorrectly and recognizes it as incorrect, but is totally unable to correct it.

CONCLUSIONS

Literal paraphasias observed during repetitive language in conduction aphasia patients have been explained in two different ways: as a

Table II. Main Types of Reading and Writing Errors Found in Conduction Aphasia Patients (According to Ardila et al., 1989a)

	Relative frequency
Reading	
Letter substitutions	30%
Anticipations	10%
Literal reading	10%
Pseudowords to meaningful words	10%
Literal omissions	10%
Literal additions	10%
Writing	
Letter substitutions	30%
Letter omissions	25%
Neologisms	25%

result of an apraxic articulatory-based deficit and as a disconnection syndrome resulting from the interruption between posterior auditory and anterior motor language areas. Current evidence might be interpreted as supporting the first theoretical explanation.

Conduction aphasia patients preserve the acoustic image of the word, and easily recognize phonologically distorted words. Their language production presents a large number of approximations to the target word. These patients present errors in the articulatory manuevers (basically in the manner and place of articulation) interpreted by some authors as a segmentary ideomotor apraxia. Kohn (1984) has pointed out that speech production in conduction aphasia involves a dysfunction at an early stage of sound-encoding and paraphasias represent phonologically oriented sequences.

Kohn and Smith (1990) have recently presented an analysis of phonological errors in a conduction aphasia patient. Their observed frequency of different types of errors in general agreed with the error frequency we have observed in our Spanish-speaking aphasics: Exchanges are nonexistent while most of the errors are copy errors (corresponding to substitutions, additions, reduplicative substitutions, and reduplicative additions). However, they found a similar distribution for vocalic and consonantic errors. We found in our Spanish-speaking patients that most errors were consonantic errors (Ardila et al., 1989b; Ardila & Rosselli, 1990), and Vinarskaya (1971) found a similar distribution with her Russian-speaking patients. This could result from the simplicity of the Spanish and Russian vowel systems, and the notorious complexity of the English vowel system.

Based on an apraxic interpretation, literal errors and clinical characteristics observed in the parietal–insular type of conduction aphasia seemingly become more understandable and comprehensible. However, much more research, particularly cross-linguistic research, is required.

REFERENCES

Ardila, A., Montañes, P., Caro, C., Delgado, F., & Buckingham, H. W. (1989b). Phonological transformations in Spanish-speaking aphasics. *Journal of Psycholinguistic Research, 18,* 163–180.

Ardila, A., & Rosselli, M. (1990). Conduction aphasia and verbal apraxia. *Journal of Neurolinguistics, 5,* 39–48.

Ardila, A. & Rosselli, M. (1992). Repetition in aphasia. *Journal of Neuorlinguistics, 7,* 1–11.

Ardila, A., & Rosselli, M. (1993). Language deviations in aphasia: A frequency analysis. *Brain and Language.*

Ardila, A., Rosselli, M., & Pinzón, O. (1989a). Alexia and agraphia in Spanish speakers: CAT correlations and interlinguistic analysis. In A. Ardila & F. Ostrosky (Eds.), *Brain organization of language and cognitive processes*. (pp. 147–175). New York: Plenum Press.

Benson, D. F. (1979). *Aphasia, alexia, and agraphia*. New York: Churchill Livingstone.

Benson, D. F. (1988). Classical syndromes in aphasia. In F. Boller, J. Grafman, G. Rizolatti, H. Goodglass (Eds.), *Handbook of neuropsychology* (Vol. 1, pp. 267–280). Amsterdam: Elsevier.

Benson, D. F. & Ardila, A. (1993). Conduction aphasia. In H. Kirshner (Ed.), *Handbook of speech and language disorders*. New York: Marcel Dekker.

Benson, D. F., Sheremata, W. A., Bouchard, R., Segarra, S. M., Prince, D., & Geschwind, N. (1973). Conduction aphasia: A clinicopathological study. *Archives of Neurology, 28*, 339–346.

Brown, J. W. (1972). *Aphasia, apraxia, and agnosia*. Springfield, IL: Thomas.

Brown, J. M. (1975). The problem of repetition: A case study of conduction aphasia and the "isolation" syndrome. *Cortex, 11*, 37–52.

Buckingham, H. W. (1981). Explanations for the concept of apraxia of speech. In M. T. Sarno (Ed.), *Acquired aphasia* (pp. 271–302). New York: Academic Press.

Buckingham, H. W. (1989). Mechanisms underlying aphasic transformations. In A. Ardila & F. Ostrosky (Eds.), *Brain organization of language and cognitive processes* (pp. 123–146). New York: Plenum Press.

Caplan, D. M., Vanier, M., & Baker, C. (1986). A case study of reproduction conduction aphasia I: Word production. *Cognitive Neuropsychology, 3*, 99–128.

Caramazza, A., Basili, A. G., Koller, J. J. & Berndt, R. S. (1981). An investigation of repetition and language processing in a case of conduction aphasia. *Brain and Language, 14*, 235–271.

Damasio, H., & Damasio, A. (1980). The anatomical bases of conduction aphasia. *Brain, 103*, 337–350.

Damasio, H., & Damasio, A. (1983). Localization of lesions in conduction aphasia. In A. Kertesz (Ed.), *Localization in neuropsychology* (pp. 231–244). New York: Academic Press.

Dejerine, J. (1901). *Anatomie des centres nerveux*. Paris: Rueff.

Dejerine, J. (1914). *Semiologie des affections du systeme nerveoux*. Paris: Masson.

Demesurisse, G., & Capon, A. (1991). Brain activation during a linguistic task in conduction aphasia. *Cortex, 27*, 285–294.

De Renzi, E., Pieczuro, A., Vignolo, L. A. (1966). Oral apraxia and aphasia. *Cortex, 2*, 50–73.

Geschwind, N. (1965). Disconnection syndromes in animals and man. *Brain, 88*, 237–294.

Geschwind, N., & Damasio, A. (1985). Apraxia. In J. A. M. Frederiks (Ed.), *Handbook of clinical neuropsychology, Vol. 45: Clinical neuropsychology* (pp. 423–432). Amsterdam: Elsevier.

Goldstein, K. (1948). *Language and language disturbances*. New York: Grune & Stratton.

Hécaen, H., & Albert, M. (1978). *Human neuropsychology*. New York: Wiley.

Heilman, K. M., Rothi, L., & Kertesz, A. (1983). Localization of apraxia-producing lesions. In A. Kertesz (Ed.), *Localization in neuropsychology* (pp. 371–392). New York: Academic Press.

Jakobson, R. (1956). Two aspects of aphasia and two types of aphasic disturbances. In

R. Jakobson & M. Halle (Eds.), *Fundamentals of language* (pp. 99–143). The Hague: Mouton.

Kertesz, A. (1979). *Aphasia and associated disorders*. New York: Grune & Stratton.

Kertesz, A. (1985). Aphasia. In J.A.M. Frederiks (Ed.), *Handbook of clinical neurology, Vol. 45: Clinical neuropsychology*. (pp. 287–332). Amsterdam: Elsevier.

Kleist, K. (1934). *Gehirnpathologie*. Leipzig: Barth.

Kohn, S. E. (1984). The nature of the phonological disorders in conduction aphasia. *Brain and Language, 23*, 97–115.

Kohn, S. E., & Smith, K. L. (1990). Between-word speech errors in conduction aphasia. *Cognitive Neuropsychology, 7*, 133–156.

Lecours, A. R., Poncet, M., Ponzio, J., & Ramade-Poncet, M. (1983). Classifications of the aphasias. In A. R. Lecours, F. Lhermitte, & B. Bryans (Eds.), *Aphasiology* (pp. 243–268). London: Bailliere Tindall.

Liepmann, H. (1908). *Drei aufsatze aus dem apraxiegebiet*, Berlin: Karger.

Luria, A. R. (1966). *Higher cortical functions in man*. New York: Basic Books.

Luria, A. A. (1976). *Basic problems of neurolinguistics*. The Hague: Mouton.

Poeck K. (1986). The clinical examination for motor apraxia. *Neuropsychologia, 24*, 129–134.

Poeck, K., & Kerschensteiner, M. (1975). Analysis of the sequential events in oral apraxia. In K. J. Zulch, O. Creutzfeldt, & J. C. Galbraith (Eds.), *Cerebral localization* (pp. 191–232). New York: Springer-Verlag.

Shallice, T., & Warrington, E. K. (1977). Auditory short-term memory impairment and conduction aphasia. *Brain and Language, 4*, 479–491.

Tognola, G., & Vignolo, A. L. (1980). Brain lesions associated with oral apraxia in stroke patients: A cliniconeuroradiological investigation with CT scan. *Neuropsychologia, 18*, 257–272.

Vinarskaya, E. N. (1971). *Clinical problems of aphasia*. Moscow: Meditsina (in Russian).

Wernicke, C. (1874). *Der aphasiche symptomen complex*. Breslau: Taschen.