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Pre-lexical disorders in repetition conduction aphasia

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ABSTRACT

At the level of clinical speech/language evaluation, the repetition type of conduction aphasia is characterized by repetition difficulties concomitant with reduced short-term memory capacities, in the presence of fluent spontaneous speech as well as unimpaired naming and reading abilities. It is still unsettled which dysfunctions of the pre-lexical processing stage of spoken word recognition contribute to this syndrome and whether there is any relevant top-down impact of the mental lexicon upon the phonetic/phonological level of speech perception. In order to further specify the underlying pathomechanisms, a comprehensive battery of psycholinguistic tests was applied to a patient suffering from repetition conduction aphasia. The obtained results point at a pre-lexical disorder in this subject. To further specify the assumed pre-lexical dysfunction, computer simulations of single-word processing, based upon an interactive activation model (IAM), were conducted. An attenuation of the features-to-phonemes inhibition value was found to simulate the observed profile of psycholinguistic deficits. Conceivably, these pre-lexical disorders interfere with the task-dependent adjustment of the temporal windows of signal analysis, giving rise to compromised sequencing of auditory-verbal information.

1. Introduction

As a rule, psycholinguistic models of spoken word recognition assume a pre-lexical processing stage during speech perception, acting upon sets of phonetic features (speech sound "elements") and/or phonemes (speech sound categories). Phonetic features might be defined in terms of articulatory gestures (see, e.g., *The Motor Theory of Speech Perception*, Liberman & Mattingly, 1985) or acoustic properties (e.g., McClelland & Rumelhart, 1981). Some speech perception models suppose that a bundle of distinct phonetic features determine phonemes or longer segmental units like syllables (Cutler, Mehler, Norris, & Segui, 1987), and the latter sound structures then are mapped onto wordforms stored in a listener's mental lexicon (for a review, see Moore, Tyler, & Marslen-Wilson, 2007; Wright et al., 1997). Neural networks such as the interactive activation model (IAM; McClelland & Rumelhart, 1981; Martin, Lesch, & Bartha, 1999) allow for computer simulations of the phonetic/phonological and lexical operations engaged in auditory language comprehension.

The syndrome of conduction aphasia usually is characterized – at the level of clinical speech/language evaluation – by markedly impaired repetition of polysyllabic words and nonwords, in the presence of – by and large – preserved spontaneous speech production and auditory language comprehension (see, e.g., Benson, 1973). Based upon psycholinguistic investigations, Shallice and Warrington (1970,1977) were able to differentiate two variants of this constellation: the reproduction and the repetition type. These authors suggested an exclusive deficit of auditory-verbal short-term memory in repetition conduction aphasia whereas the other variant was assumed to reflect disrupted phonological encoding mechanism, afflicting confrontation tasks such as repetition, reading and naming in a similar manner.

The following paragraphs focus on repetition conduction aphasia, the constellation of speech/language pathology displayed by the patient examined in this study. At least three different pathomechanisms of this syndrome have been proposed in the literature:

(i) impaired sequencing capabilities of the central-auditory system (Albert, 1972);
(ii) deficits of auditory-verbal short-term memory such as limited buffer capacities (Caramazza, Basili, Koller, & Berndt, 1981; Shallice & Warrington, 1970; Shallice & Warrington, 1977),

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(iii) accelerated decay of speech sound representations (Martin, Breedin, & Damian, 1999; Martin, Lesch et al., 1999).

Other authors argue, however, for a more generalized dysfunction in conduction aphasia, encroaching also upon output devices (e.g., McCarthy & Warrington, 1984) or central phonological representations (Allport, 1984).

Based upon IAM computer simulations, a recent case study tried to further specify the pathomechanisms of repetition conduction aphasia (Martin, Breedin et al., 1999). The present investigation also utilizes this approach, providing a platform for the formulation and evaluation of specific hypotheses on the computational, especially phonological disorders involved in repetition conduction aphasia. In general, these models (e.g., Dell, 1986; Dell & O'Seaghdha, 1992; McClelland & Rumelhart, 1981) propose four hierarchically ordered domains of speech processing, organized as networks of distinct nodes each: phonetic features, phonological segments, lexical representations and semantic data structures. Each computational stage exclusively interacts with its preceding and subsequent level, and these directed interconnections specify the relationships between individual nodes. For example, the phonological segments of long-term memory can be activated both by representational units of the input phonological buffer and top-down by lexical nodes. These operations are assumed to keep the content of long-term memory in an active state and to facilitate the lexical selection process (for further details, see Fig. 1). Besides excitatory relationships, inhibitory connections between the phonetic feature and phonological segment nodes and between the phonological segment level and the lexical layer are proposed as well. In addition to top-down excitatory connections, these models also encompass inhibitory feedback connections from the word to the phonological segment level. Finally, there are within-level inhibitory connections between competing units of each distinct layer.

Because of a dense interconnectivity between the various IAM components, "local" dysfunctions may give rise to more extensive disorders, encroaching upon "more distant" processing stages. A genuine short-term memory deficit, e.g., should yield insufficient activation of segmental features and, therefore, give rise to a rapid decay of activation at the level of phonological segments. On the other side, activation both of the phonological input-buffer and the appropriate lexical entities must be expected to be compromised in case of a change of the inhibition or activation values of phonemic data structures.[1] Most noteworthy, moderate deficits at the level of phonological segment nodes might initiate repair mechanisms, e.g., top-down (lexical nodes) or button-up (phonological buffer) activations which compensate for the lack of computational specification at the level of phonological segment nodes. Because of such feedback loops, disrupted phonemic representations not necessarily develop into deficits of speech sound perception.

As a prerequisite to such IAM computer simulations, a patient suffering from repetition conduction aphasia underwent a comprehensive series of psycholinguistic and neuropsychological tests. At the time of behavioral investigations, about 10 months after stroke onset, spontaneous speech and reading skills were found, as expected, unremarkable. Formal testing of repetition capabilities, a critical element in the evaluation of conduction aphasia, relied on lists of nonwords and lexical items, strictly controlled for length and frequency of occurrence. In order to assess the functional integrity of the phonological and lexical IAM layers, a phoneme discrimination task (evaluating the intactness of sound categories within long-term store) and an auditory lexical decision task (investigating lexical access within long-term store) were administered. As concerns the latter test, all items had been matched, besides word length and frequency of occurrence, for the number of lexical competitors (density). Lexical density must be expected to influence both the speed and the accuracy of identification tasks in healthy subjects (Luce, 1986; Luce & Pisoni, 1998). Consolidation of nonwords within the long-term store and their processing within the phonological input-buffer were assessed separately, using a standardized verbal learning (Sturm & Wilmes, 1999) and a letter probe test (MacAndrew, Klatzky, Fiez, McClelland, & Becker, 2002; Paulesu, Frith, & Frackowiak, 1993), respectively. A variety of studies based upon the probe task paradigm has demonstrated that items of a similar sound structure (e.g., B[be:], T[te:], C[tse:]) elicit more pronounced interferences – both during a visual as well as an auditory mode of presentation – than phonologically disparate units (e.g., S[ɛs:], H[ha:] W[we:]). An absent similarity effect within the auditory modality is assumed to indicate deficient phonological short-term memory functions or, within the visual domain, a disruption of the rehearsal process. As a consequence, these tests were expected to allow for a further specification of the nature of the subject's deficits, based upon the IAM framework proposed by Martin, Lesch et al. (1999).

Taken together, we used IAM to further elaborate the pathomechanisms of a distinct variant of conduction aphasia, i.e., the repetition type of this constellation, traditionally assumed to reflect a disorder of auditory short-term memory. At first, an extensive battery of psycholinguistic and neuropsychological tests, evaluating repetition capabilities, phoneme discrimination, differentiation of lexical items and pseudowords, and phonological short-term memory, was applied to a patient suffering from this syndrome of speech/language pathology. Second, the specific profile of the subject's cognitive deficits was reproduced by IAM computer simulations. Two main research questions were addressed:

(i) Which dysfunctions of the pre-lexical processing stage of spoken word recognition are associated with the subject's repetition conduction aphasia? The role of pre-lexical operations is a critical issue in any discussion of the pathomechanisms of this syndrome (see below).
(ii) Is there evidence for any top-down impact of the mental lexicon on the phonetic/phonological level of speech perception in this patient? Within an IAM framework, these feedback mechanisms have a crucial influence upon phoneme and lexical selection. However, behavioral data obtained from healthy Dutch subjects engaged in a phonetic categorization test argue against such top-down interactions (McQueen, Cutler, & Norris, 2003).

2. Materials and methods

2.1. Case report

In May 2003, a 66-year-old former electrical engineer (patient NP) was admitted to the Department of Neurology, Medical Center of the University of Tübingen. During local lumbosacral infiltration of an anaesthetic agent in another hospital, he had begun to experience difficulties in understanding spoken language and to display mild deterioration in speech production, concomitant with error awareness as well as self-corrections. At the initial clinical examination in the Department of Neurology, a right-sided central facial paresis could be noted. Cranial computerized tomography (CCT) documented a left-hemisphere temporo-parietal ischaemic infarction (Fig. 2). In addition, the CCT scans showed the sequels of an older – so far unnoticed – contralateral parieto-occipital cerebrovascular accident. Several preceding studies had reported lesions of a similar location in subjects with conduction aphasia (Damasio & Damasio, 1980; Kertesz & McCabe, 1977; Palumbo, Alexander, & Naeser, 1992; Mendez & Benson, 1985). Unfortunately, none of these patients had undergone detailed neurolinguistic and neuropsychological examinations.

[1] According to the terminology of IAM phonemic processing takes place at the level of "phonological segment nodes" (see also Fig. 1).