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SHORT-TERM MEMORY IMPAIRMENT: EVIDENCE FROM APHASIA

To determine the mechanism of STM decline following brain damage, patients with left hemisphere damage and aphasia (LHD+), patients with left hemisphere damage without aphasia (LHD-), and persons with right hemisphere dysfunction (RHD) were examined. An auditory-verbal memory task, as well as a series of visual memory tasks, consisting of designs, objects and pictures, were used. Patients with LHD+ performed the auditory memory task significantly worse than those with LHD- and RHD. Although the groups did not differ with regard to nonverbal STM measurement, the visual task performance suggested a better memory in aphasics for sensory rather than meaningful features of the visual stimulus. There was no effect of localization of a lesion within the left hemisphere on short-term memory.

Memory deficits in aphasia have been described in the literature for many years. However, they have not been investigated systematically, perhaps because it is inherently difficult to differentiate memory dysfunctions and language disturbances. According to the traditional standpoint, the most severe memory disorders are observed in non-dysphasic subjects, especially with the amnesic syndrome or dementia (Squire & Butters, 1992). The foregoing position was questioned for the first time by Zangwill in 1946. He indicated that a marked defect of verbal learning appeared sometimes in mild aphasia (Ettlinger & Moffett, 1970). This observation is partially relevant to the data reported later by Cermak and Tarlow (1978). Whereas the Korsakoff patients performed significantly below the level of the aphasics on the nonverbal tasks, the aphasics performed significantly below the Korsakoffs on the verbal tasks. Furthermore, clinical practice also reveals that many language-impaired patients (like those with aphasia) complain of memory difficulties, and their families often confirm that the patients have troubles recalling previously known names and events, and learning new information. It seems that the complaints of poor memory in such patients are more significant than simply a manifestation of an underlying speech breakdown (Ween et al.,1996). The above opinion is supported by a large number of studies showing that aphasics also have problems in visual memory tasks not requiring

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a verbal answer (e.g., Dalla Barba et al., 1996; Gainotti et al., 1978; Goodglass et al., 1974; Gutbrod et al., 1987). These results are usually interpreted as a lack of verbal mediation in coding visual material which is caused by acoustic-semantic confusion of stimuli names. Ostergaard and Meudell (1984) maintained that only Broca's aphasics showed visual recognition memory difficulties. The same individuals demonstrated verbal memory deficits which could not be attributed to linguistic disturbances, while the memory deficiencies seen with the Wernicke's aphasics could be regarded as secondary to linguistic defects.

Unlike the above data, other authors observed selective, only auditory-verbal, memory deficit in aphasia (Cermak & Tarlow, 1978; Łuria, 1976; Martin & Saffran, 1997; Shallice & Warrington, 1977; Trojano & Grossi, 1995; Warrington & Shallice, 1969).

Because of the variety of findings and conclusions, the mechanism of memory disorders in aphasia remains unclear. In general, recent studies on forgetfulness in aphasia have taken two different approaches to defining the relationship of memory and language processes.

One area of research is based on structural models of human memory and aims to identify short-term (STM) and long-term (LTM) memory deficits in aphasia in relation to the site of brain damage. For example, Beeson et al. (1993) and Risse et al. (1984) reported that anterior lesions were associated with impaired long-term retention and posterior damages with STM deficits. In contrast, other studies failed to support this notion (Burgio & Basso, 1997; Coughlan, 1979; Ween et al., 1996).

A second, more recent, group of studies stems from neurolinguistics and seeks to identify the relationship between language and STM within a functional model of language/memory processing. This relationship has now been emphasized in a number of information processing theories and has been better defined in psycholinguistic terms than within the framework of a classical taxonomy of aphasic syndromes (Caplan & Waters, 1995; Caramazza, 1997; Martin & Lesch, 1995; Martin & Saffran, 1997; Trojano & Grossi, 1995). These theories have all considered "memory" as being a byproduct of the extent to which an individual analyzes the information during the time it is presented (processed). More specifically, patterns of STM performance appear to be related to the integrity of semantic and phonological capacities (McElree, 1996). In this sense, memory and language create an interactive system and are interdependent in several ways. For example, verbal memory is dependent upon language functions at least to some degree. Before an item can be stored in LTM, it must be decoded and recognized as a linguistic item with phonological and/or semantic characteristics. The ability to retrieve such an item from memory depends upon access to its verbal representation. The interdependence of memory and language can also be defined in a different manner. An obvious way in which language is dependent on verbal memory is that vocabulary is learned via verbal memory functions. It also seems that grammar can be considered a specific form of procedural memory on how to combine linguistic symbols into meaningful units. It is argued that morphological and syntactic rules are implicit in nature, and they are routinely applied without explicit conscious awareness (Crosson, 1992).

Recently, the most promising framework for memory study in aphasia seems to be the working memory concept established originally in 1974 by Baddeley and Hitch (Baddeley, 1992). The essential subcomponent of the working memory is the phonological loop which stores and rehearses speech-based information. The phonological loop comprises a phonological store and an articulatory control process. Auditory information has direct access to this store, but visual information only has access through the articulatory control process, which allows visual material to be phonologically coded. In other words, visually presented material, such as words or nameable pictures, is registered in the phonological store by subvocalization. Some authors claim that interruption of the phonological loop is responsible for memory deficits in aphasia (Caplan & Waters, 1995; Rochon et al., 1990). Others, in contrast, postulate a multiplelanguage capacity rather than a single-capacity view of working memory. Martin et al. (1994) came to such a conclusion on the basis of two patients they studied. Both patients, one with worse retention for semantic than phonological information and other with reverse dissociations of disorders, presented similar reductions in memory span.

The existence of connections between language and memory disorders becomes more probable in the light of neuroanatomical studies. Even though many brain lesions that produce aphasia are located outside neural structures critical for memory (particularly limbic), it is now widely believed that language and memory deficits can be observed together following lesions located in such parts of the brain as the temporal lobes, thalamus or basal ganglia (Brockway et al., 1998; Crosson, 1992; Kądzielawa, 1997). However, a precise characterization and explanation of the coexistence of memory and language disorders is still a controversial issue.

The purpose of this study was to examine STM ability of aphasic individuals in relation to site of brain damage. The question of whether patients with aphasia show global or only a selective, modality-specific, STM deficit (for words) was analyzed as well. With the aim of further investigating the extent of STM impairment in aphasia, this study examined auditory and visual memory.

The last question was addressed to the contribution of perceptual and conceptual impairment to the visual memory deficit. There is evidence in the literature that perceptual and conceptual memory processes in the visual domain are subserved, at least in part, by different neurological structures in the brain. In general, results from several functional neuroimaging experiments testing both healthy participants and brain-damaged patients show that conceptual tasks activated the medial and lateral left hemisphere in frontal and temporal regions as well as the lateral aspect of the bilateral inferior parietal lobule. Perceptual tasks, in contrast, produced relatively greater activation in the right frontal and temporal cortex as well as bilateral activation in more posterior regions (Blaxton et al., 1996).

Method

Subjects

Thirty-six patients divided into three groups took part in the study. The subjects were 13 patients (10 male, 3 female) with unilateral left hemisphere lesions and aphasia (LHD+ group), 10 non-aphasic left brain-damaged patients (6 male, 4 female) (LHD- group), and 13 patients (11 male, 2 female) with unilateral right hemisphere damage (RHD group). Type of aphasia in LHD+ group was assessed according to patients' performances on the Aphasia Screening Test by Wepman and Halstead derived from the Halstead-Reitan Battery (Polish translation, Kądzielawa, 1990), and on selected tasks from Łucki's Set (1995). Nine aphasics were classified as nonfluent and 4 as fluent. All individuals were recruited from the Hospital of the Rehabilitation Center of Gdańsk in Dzierżążno. All patients were right-handed and native speakers of Polish. The subjects were in the age range 19-77 (mean 52, SD=14) and had an average of 12 years of education (SD=3). For all patients, cerebrovascular accident (CVA) had occurred, with an average of 194 days (SD=151) between CVA and current neuropsychological testing. The presence and laterality of the brain damages were identified by a review of the medical records and computerized tomography (CT) or magnetic resonance imaging (MRI) scans. Patients had no known history of other significant medical disease such as psychiatric disorder, dementia, substance abuse, or additional neurological events (e.g., head injury). Only patients demonstrating normal single-word comprehension, as well as with no evident behavioral symptoms of unilateral visual neglect were included in the study.

There were no significant group differences for age [$F(2;33)=.64$, $p>.05$], years of education [$F(2;29)=.64$, $p>.05$], and time post CVA [$F(2;33)=2.54$, $p>.05$].

Procedure

The assessment of memory in aphasia presents a methodological challenge. Speech comprehension problems and verbal expression difficulties may contribute to the failure to recall list items. For this reason, most of the popular memory tests are not useful in cases of aphasia, since direct recall tasks rely upon the patient's ability to verbalize the material aloud (which he often cannot do). Many patients with aphasia suffer also from right hemiplegia or hemiparesis and they are not able to answer in written or graphic form.

Four immediate recall tasks (one auditory and three visual) were administered to examine short-term memory. The material consisted of words (read aloud), graphic designs, real objects, and pictures of common objects. Memory was tested only with immediate recognition tasks. Memory for objects and for pictures were examined on different days in order to avoid intrusion errors across tasks. The interval between tasks was 7 days.

Auditory-verbal memory task

The task procedure was designed to measure memory span for words and required auditory recognition of 12 words immediately after their oral presentation. The task consisted of two presentations. The second presentation, which followed directly the first one, was used to eliminate potential attention problems. High frequency concrete nouns selected from the Rey Auditory-Verbal Learning Test (Lezak, 1995) were presented at a rate of one word every 2 sec (e.g., *drum*, *curtain*, *belt*, *coffee*, *school*, *parents*, *sun*, *garden*, *cap*, *man*, *house*, *river*). Immediately following the second list presentation, subjects were asked to recognize them on a list consisting of 12 target and 12 other words (derived from an alternative version of the AVLT). The words were displayed in random order. In the recognition test, on each trial the examiner read aloud a word and the subject's task was to identify whether the item had been previously presented. Each person was given 5-7 seconds between words to indicate with a raised hand (Yes) or a lowered hand (No) his recognition response. Nonverbal Yes or No hand signs were used until the patient responded satisfactorily. Thus, the test procedure assumed two presentations of the same word list and one recognition trial following the second exposure. The total score for the task performance was the difference between correct recognitions and false-positive errors.

Visual memory task for graphic designs

The task was administered to assess the patients' short-term memory for visuospatial material. The examiner showed simultaneously a set of nine designs (3 designs × 3 rows) and the subject was asked to remember them during 30 seconds. The task material was adapted from the DUM test (Weidlich & Lamberti, 1996). Each design was a simple schematic figure composed of five straight lines. Following the presentation, a new set of 18 designs (including target and lure items proportionally) was presented and the patient was required to point to the target items. As earlier, false-positive errors were subtracted from the number of correct choices.

Visual memory task for objects

In this task, twelve real objects, well-known and of everyday use, were presented for 30 seconds in two rows (e.g., *scissors*, *watch*, *button*, *spoon*, *matches*, *bulb* / *comb*, *cup*, *pencil*, *key*, *envelope*, *glasses*). The recognition trial was conducted immediately. The patient was asked to recognize the objects given on 24 schematic pictures (each 6×6 cm) arranged in four rows. Because the pictures were schematic and black and white, the patient was precisely instructed to identify the same category on the picture to which the object belongs, regardless of the perceptual similarity between them (i.e. object seen earlier, and the picture). False recognition were also taken into account in scoring. Such test conditions were constructed to assess the effect of interference on visual recognition memory. The interference was caused by perceptual difference between presentation (acquisition) and recognition (recall) conditions.

Visual memory task for pictures

The procedure was similar to that used in the memory task for objects. In contrast to the previous task, the perceptual conditions of presentation and recognition were consistent. Twelve pictures of common objects were presented for 30 seconds in two rows (e.g., *knife*, *table*, *plate*, *flower*, *hammer*, *horse* / *candle*, *pipe*, *ear*, *duck*, *shoe*, *book*). The patient was then instructed to identify the target pictures presented together with 12 new pictures. As in all tasks, the number of incorrect recognitions was also recorded to compute the total score.

To determine whether memory deficits might be attributable to problems of speech comprehension, subjects also performed a short task of auditory understanding (Speech Comprehension Task) derived from Łucki's Set (1995). The task material consists of several geometric figures which differ with regard to size, color and shape. The subject was requested to pick the figure described by the examiner (for example, "show me a big blue square"). The maximum score was 10.

Results

(A) STM and site of brain damage within the left hemisphere

In order to study the influence of locus of the brain damage on memory within LHD groups, only patients whose lesions were classifiable as predominantly anterior or predominantly posterior to the central sulcus on the basis of CT or MRI scans were included in the analysis. Because of the small number of patients, the LHD+ and LHD- groups were considered together. This way, two groups of patients were separated out: the anterior lesion group (N=6), and the posterior lesion group (N=7). However, statistical analysis (t-test) revealed no significant differences between the groups on the basis of means obtained on all the memory tasks (*p* >0.1).

(B) Extent of STM impairment in aphasia

The second aim of the study was to determine whether patients with aphasia show a general, or only a selective, modality-specific defect in STM (for example, only for words presented auditorily). Table 1 shows the mean scores of left and right brain-damaged patients obtained on the memory tasks.

Table 1. Mean performance of the groups on the memory tasks

Memory task	Group* LHD + Mean (SD)	LHD– Mean (SD)	RHD Mean (SD)
Words	5.61 (3.20)	9.40 (2.67)	9.30 (1.65)
	a	b	b
Figures	6.69 (1.03)	6.60 (1.89)	5.61 (1.60)
	a	a	a
Objects	7.07 (2.81)	9.10 (1.59)	9.38 (2.18)
	a	ab	b
Pictures	9.38 (2.36)	9.50 (2.46)	8.76 (2.31)
	a	a	a

* (LHD +) left hemisphere damage and aphasia; (LHD -) left hemisphere damage without aphasia; (RHD) right hemisphere damage

One-way ANOVA revealed significant between-group differences in two cases: in the memory task for words [$F(2;33)=8.67$, $p<.001$] and in the memory task for objects [$F(2;33)=3.78$, $p<.05$]. *Post hoc* analysis is included in Table 1 as well. Means sharing ‘a’ subscript, and means sharing ‘b’ subscript do not differ significantly at p<.05 (Tukey’s multiple range test).

(C) Perceptual and conceptual mechanisms of visual STM impairment

Comparison within subject groups (performed separately for each group) was carried out to determine the differences between the performances on the memory task for objects and the memory task for pictures. Results are displayed in Table 2.

Table 2. Between-task comparison for pictures and objects memory task

Group	Pictures Mean (SD)	Objects Mean (SD)	t value	df	p
LHD +	9.38 (2.36)	7.07 (2.81)	2.79	12	0.01
LHD -	9.50 (2.46)	9.10 (1.59)	0.60	9	0.56
RHD	8.76 (2.31)	9.38 (2.18)	- 0.92	12	0.37

Simple effect analysis performed with t-test showed that only the aphasics (LHD+ group) had more problems recalling objects than pictures. Reverse dissociation was not seen.

The next step of the statistical analysis aimed to determine whether recognition memory deficits can be attributed to speech (comprehension) disturbances. For the purpose of calculating the correlations, r-Pearson correlation analysis was employed twice. Because of the small sample of aphasics (N=13), an analysis was performed additionally in relation to all subjects (N=36). As shown in Table 3, the only bivariate correlations that reached significance were found in the entire patients' group.

Table 3. Correlations between speech comprehension task and memory tasks

	Pearson r			
	Memory for words	Memory for figures	Memory for objects	Memory for pictures
Speech comprehension (N=13)	0.228	0.118	0.452	0.164
Speech comprehension (N=36)	0.563*	- 0.155	0.588*	0.034

* Significant at p <.001

With regard to the speech comprehension task, the bivariate correlations were the strongest with Memory for Words and Memory for Objects (r=0.563, r=0.588; p<.001).

Discussion

The results of this study indicate that individuals with anterior or posterior sites of lesion in the left cerebral hemisphere exhibit comparable STM abilities. These findings do not suggest a significant lesion site effect on any of our memory measures. In contrast, immediate recall deficiency following mainly posterior brain dysfunction was found, for example, by Beeson et al. (1993), Łuria (1976), and Risse et al. (1984). Nonetheless, the absence of association between STM and the specific locus of brain damage found in this study is consistent with other investigators' observations (Burgio & Basso, 1997; Ween et al., 1996). There are several possible explanations for this result. Firstly, our left brain-damaged population was small (only 13 left brain-damaged patients' lesions could be classified as anterior or posterior), and both aphasic and non-aphasic patients were included in the analysis. This way, each group was homogeneous only with respect to the anatomical, but not to the functional (presence of aphasia) criteria. This could result in the picture of memory impairment being less clear. As the current study also demonstrated, aphasia might play a certain role in the immediate recall. Such an effect occurred in the auditory-verbal memory task which was performed defectively by patients with aphasia. Secondly, it is difficult to state whether all tasks we used were equally sensitive to memory deficits. Besides, unlike other studies, only the recognition paradigm of memory examination was used. According to a third explanation, which is contradictory to the earlier one but compatible with modern models of language processing, STM capacity depends on different language processes (phonological, lexical-semantic) distributed in only partially overlapping neural systems (Martin & Saffran, 1997; Martin et al., 1994). Thus the disruption in various parts of the brain may result in STM deficits, decreasing the probability of the lesion site effect. However, the interaction between language and STM remains controversial.

The second aim of the study was to investigate the extent of STM impairment in aphasia. Results of the present research demonstrate that aphasics mainly suffer from auditory-verbal STM deficit. Recognition memory for words was considerably worse in aphasics than in patients without language problems regardless of laterality of the brain damage. Unfortunately, it is difficult to be absolutely certain about the modality specificity of the defect. On the one hand, patients with aphasia also had the problem of reporting objects presented visually; on the other, the difference was significant only in comparison with right hemisphere damaged patients. From this observation it might be argued that individuals with aphasia are impaired in memory tasks if the material is verbal or easy to verbalize. This standpoint is further supported by the fact that there was no between-group difference in the nonverbal recognition memory task. However, it is impossible to entirely reduce the verbal mediation here. It is known that even test stimuli which consist of quite complex and unfamiliar designs or nonsense figures (like those used in this study) do not fully escape verbal labeling (Lezak, 1995). The hypothesis assuming only a verbal character of the memory impairment in aphasia is questionable also by the fact that there was no between-group difference in performances on the memory task consisting of meaningful pictures equally easy to verbalize. According to Lezak (1995), because of the verbalizability of pictures and real objects, they both may be used to test span of visual retention, as well as verbal memory. As further discussion about the discrepancy between those visual-verbal memory measures is attractive for theoretical reasons, it may help to explain the mechanism of the visual memory disorders in aphasia. For this purpose, between-task comparison between object and picture retention was conducted in each group separately. It was discovered that only patients with aphasia had greater problems to recognize objects than pictures presented earlier. The above dissociation may reflect a distinct contribution of sensory and meaningful processes in visual short-term memory. Namely, in the memory task for objects, the previously presented objects were recognized in the pictures. In such conditions, the patient might not rely only on sensory features of the material but would have to detect its meaningful characteristics. In other words, searching for the perceptual similarity between object and picture was not a sufficient way of recognizing the former. This finding supports the view that a visual memory defect in aphasia can be explained in terms of semantic breakdown.

In considering the possible origins of verbal memory deficits in aphasia our first impression may be that such deficits were attributable to problems with speech comprehension. At this point, our results are relevant to the data reported by Ostergaard and Meudell (1984). A significant relationship was found between speech comprehension and two measures of memory: for words and for objects. However, such a conclusion is formulated on the basis of an entire sample, and is far more probable for the auditory than for the visual memory. That the visual memory problems were not entirely attributable to defective receptive abilities (understanding the test instruction) may be inferred from the finding that a relationship did not occur on the other visual tasks. Alternatively, difficulties with speech comprehension can be considered as a measure of general severity of aphasia. In this sense, STM impairment would reflect the severity of aphasia. However, the empirical support for this prediction is rather inconclusive (Cermak & Tarlow, 1978; Goodglass et al., 1974).

Summing up, this investigation did not confirm the effects of localized left cerebral hemisphere lesions on short-term memory. The results of the study suggest that aphasia may be accompanied by memory deficits that are not always simply a consequence of the speech difficulties. The most frequent STM disorders concern auditory-verbal material. Patients also show no evidence of verbal mediation in memory tasks when presentation is visual. The possible pathomechanism is seen in the breakdown of sub-vocal repetition (rehearsal). Nevertheless, in certain circumstances aphasics' retention ability of verbal material in the visual domain is spared. The distinction may be conceptualized in terms of perceptual and meaningful coding.

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