Effects of localised cerebral lesions and dysphasia on verbal memory

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SUMMARY Twenty-nine patients with unilateral left hemisphere lesions, 22 patients with unilateral right hemisphere lesions, and 19 neurological control patients with extracerebral lesions were assessed on verbal memory recall and recognition tests and on a battery of language tests. The left hemisphere group was significantly impaired in memory and language skills. Significant verbal memory impairment was found both in the subgroup of left hemisphere lesion patients whose lesions involved the temporal lobe and in the subgroup whose lesions did not. However, no significant differences between these left hemisphere subgroups' levels of performance on memory tasks emerged, even when dysphasia was taken into account. This study, therefore, fails to support the notion of a specific anatomical correlate of verbal memory impairment within the left hemisphere. Dysphasic subjects were significantly impaired on verbal memory tasks but displayed the same pattern of sensitivity to the effects of word frequency and word concreteness on verbal memory as control subjects, suggesting that the verbal memory of the dysphasic subjects was quantitatively rather than qualitatively impaired. This impairment could not be attributed to deficits in the comprehension or expression of the memory test items, and it is, therefore, proposed that language disturbances may hinder the efficient use of such language based procedures as may subserve verbal memory.

This study had two aims—to examine the effects of localised left hemisphere lesions on verbal memory and to investigate the relationship between verbal memory and dysphasia.

While it is well established that subjects with left temporal lobe lesions are frequently impaired on verbal memory tasks compared with normal subjects or those with right temporal lobe lesions (Meyer and Yates, 1955; Milner, 1958; Blakemore and Falconer, 1967; Milner and Teuber, 1968) it remains controversial as to whether or not left temporal lesions are of significantly greater detriment to verbal memory performance than lesions elsewhere in the left cerebral hemisphere. Newcombe (1969), using a variety of tasks, did not find any greater verbal memory deficits resulting from left temporal lesions than from other left hemisphere sites. In contrast, Milner (1964, 1969) has reported a specific intrahemispheric association between left temporal damage and verbal memory impairment. However, there would ap-

pear to be important differences in the populations studied. Newcombe's study included dysphasic subjects whereas at least one of Milner's studies (Milner, 1964) did not. Whether Milner's 1969 study included dysphasic subjects is not clear. It is possible that in a population containing dysphasic subjects effects of locus of lesion on verbal memory may be masked by the effects of language impairment. Nevertheless, dysphasia is a common consequence of left hemisphere lesions, and the exclusion of dysphasic subjects from localisation studies may result in lesion samples which are not typical of the cortical regions they purport to represent, and may also give rise to false impressions of the natural incidence of verbal memory deficits in subjects with localised damage. Rather than exclude dysphasic subjects from investigations into the effects of localised lesions on verbal memory it would thus seem appropriate to include them and, in comparing the verbal memory performance of each localisation group, to attempt to take the effects of language impairment into account by covariance analysis. This, therefore, is the approach I adopted in this present study.

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As regards the relationship between verbal memory and dysphasia, dysphasic subjects have frequently been shown to display impairment on verbal memory tasks, though at times conflicting accounts have emerged as to the specific types of task on which impairment is found, possibly because of differences in the natures and severities of the dysphasias studied or in the selection of control groups. Thus, poor rote learning of sentences but relatively good story recall (Zangwill, 1946), impaired rote learning and relearning of sentences (Ettlinger and Moffett, 1970), impaired story recall but unimpaired paired associate learning and nonsense syllable learning (Newcombe, 1969), impaired nonsense syllable learning but unimpaired word list learning (Wyke, 1966), and impaired serial anticipation learning (Carson *et al.*, 1968; Tikofsky, 1971) have all been reported for dysphasic subjects. The reasons for these various deficits are not well understood, though Tikofsky (1971) has suggested that dysphasic subjects may suffer increased vulnerability to interference effects, and Wyke (1966), in the case of poor nonsense syllable learning, has argued that dysphasic subjects have difficulty in forming rapid meaningful associations for use as verbal memory mediators. With the exception of Wyke's study it is also unclear to what extent the verbal memory performance deficits reported may be attributable to the impaired comprehension and expression of test materials rather than to any impairment in verbal memory skills. The extent to which disorders of comprehension and expression may hinder the comprehension and expression of memory test materials is likely to depend on the particular nature of these deficits and on the linguistic demands made by the materials, so in considering the verbal memory skills of dysphasic patients it is important to specify these factors. This study investigates the verbal memory of dysphasic subjects for whom language skills relevant to the test materials have been assessed. Both recall and recognition tasks are employed. Previous studies have employed only recall tasks, but recognition tasks, although still vulnerable to receptive deficits, provide a method of assessing verbal memory without relying on expressive skills. In addition, the effects of word frequency and word concreteness on the verbal memory performance of dysphasic subjects are examined. Performance of normal subjects on memory tasks is known to be sensitive to these parameters (Paivio, 1971) and the pattern of sensitivity displayed by dysphasic subjects may give some insight into the nature of the relationship between dysphasia and verbal memory.

Subjects and methods

The subjects were 29 patients with unilateral left hemisphere lesions (LH group), 22 patients with unilateral right hemisphere lesions (RH group), and 19 neurological control patients (control group) with extracerebral lesions. All were hospital inpatients who had recently undergone surgery or had been admitted for neurological investigation. All patients were native speakers of English and were in the age range 21–70 years. All patients with a cerebral lesion were right handed. The patients in each group were unselected except that three potential LH group members were not included because dysphasia was too severe to permit comprehension of simple test instructions. The presence and laterality of the lesions of the subjects in the RH and LH groups were established from positive findings on at least two reliable neurological techniques—*isotopic brain scan*, *angiography*, *pneumoencephalography*, or *CAT scan*—and, where available, from surgeons' post-operative reports. Most of these lesions were tumours. The patients in the LH group were also classified into mutually exclusive temporal (N=19) and non-temporal (N=10) localisation groups (the temporal group comprising all patients whose lesions included the temporal lobe and the non-temporal group comprising the remaining left hemisphere lesion patients) and also, irrespective of lesion site, into mutually exclusive dysphasic (N=20) and non-dysphasic (N=9) groups. For this second method of classification dysphasia was defined objectively as performance at or below the 5th percentile on any one of three tests, Object Naming, Description Naming—that is, naming objects from verbal description—or the Modified Token Test (after De Renzi and Vignolo, 1962).

There were no significant differences in mean number of years' education between the control, RH, and LH groups or between the LH subgroups. Control, RH, and LH groups were satisfactorily matched in terms of socioeconomic status, as were the LH subgroups.

PRELIMINARY TESTS

These tests were included to give an estimate of intellectual level and level of functioning in a variety of speech skills, and were administered a day or two before the verbal memory tests. Full descriptions of the preliminary test procedures are given by Coughlan and Warrington (1978).

Current IQ

An estimate of each patient's current level of

intellectual functioning was derived from performance on the B and C sets of Raven's Progressive Matrices (Raven, 1958). Scores on these sets were extrapolated to Full Matrices scores and then converted to equivalent Full Scale Wechsler-Bellevue IQs using a conversion table.

Modified Token Test

This test comprised a set of fifteen orally presented complex instructions and was a shortened version of the most complex part of the De Renzi and Vignolo (1962) Token Test for the assessment of receptive disturbances. Each instruction was presented once only. Subjects with a cerebral lesion who scored 12 or less out of 15 on this task were considered to be dysphasic.

Object Naming

Subjects were asked to name a set of 15 objects. An error was scored on any object not named within five seconds. Subjects with a cerebral lesion who scored 13 or less out of 15 on this task were considered to be dysphasic.

Description Naming

Verbal descriptions of objects were presented orally and subjects were required to name the object described each time within seven seconds. Fifteen descriptions were used and each description was presented once only. Subjects with cerebral lesions who scored 12 or less out of 15 on this task were considered to be dysphasic.

Phoneme Discrimination

In this test, subjects were presented with pairs of words (by tape recorder) and asked to say or indicate whether the two words in each pair were the same or different. Forty pairs of words were presented, 20 in which the words were the same—for example—fight-fight—and 20 in which the words differed by one consonant phoneme—for example, town-down. Each pair was presented once weekly.

Articulation

Subjects were asked to repeat the 32 monosyllabic words in the dysarthria/dyspraxia section of the Minnesota Test for the Differential Diagnosis of Aphasia (Schuell, 1965). Each word was presented once only and scoring was strict—that is, spontaneous correction of errors was not credited.

WAIS Vocabulary Subtest (Wechsler, 1955)

This test requires the patients to define words of increasing difficulty. The standard administration procedure was modified in that the words were

presented orally only, to conform with the oral presentation of other tests in this study. The words were repeated if necessary.

Modified Peabody Test

This was a shortened version of the 150 item Peabody Picture Vocabulary Test (Dunn, 1959) in which word comprehension is assessed by items which require the subjects to identify the meaning of a spoken word from a choice of four pictures. This shortened version employed only 60 items from the middle range of the original test. The words were repeated if necessary.

VERBAL MEMORY TESTS

Verbal memory was assessed by both recall and forced choice recognition of words in orally presented lists. In order to investigate the effects of word frequency and word concreteness, four separate experiments (each comprising recall and recognition tasks) were carried out, each using a different type of word: high frequency—high concreteness (HF-HC), high frequency—low concreteness (HF-LC), low frequency—high concreteness (LF-HC), and low frequency—low concreteness (LF-LC). Concreteness estimations were taken from the Brown and Ure (1969) seven-point scale ratings. High concreteness words were those rated above the mean concreteness value of 4.68, and low concreteness words were those rated below this value. High frequency words were those occurring more than 50 times/million, that is, A or AA frequency, and low frequency words were those occurring between 1 and 49 times/million (Thorndike and Lorge, 1944). For each word type the recall task used three 10 word lists and the recognition task used a 35 word list of stimuli followed by forced choice recognition of pairs of words consisting of one list item and one distractor.

In the recall task, presentation and recall of the first list was followed by presentation and recall of the second list, and so on. Each list was read out to the subject three times consecutively at a rate of one word per two seconds. The final presentation of a list was immediately followed by a 35 second interpolated task (adding one to single digits rapidly presented on cards) to minimise the effects of short-term memory, and then the subject was allowed 75 seconds to recall the words, in any order. Only precise reproductions of list item were accepted as correct responses—morphological modifications or paraphasic distortion were classed as errors even if it was obvious which list item was being referred to. After this recall period the subject was given

10 seconds to prepare for the next recall list. After finishing the recall task on a given word type, subjects were allowed a two minute break before beginning the recognition task for that word type, and the instructions for the recognition task were given in the latter part of this break. The recall task on a given word type, therefore, always preceded the recognition task on that word type.

In the recognition task the list of 35 words was read to the patient at a rate of one word per three seconds. Immediately after presentation of the list the subject was presented with successive pairs of spoken words, each comprising of a list item and a distractor of the same word type, and asked to indicate the list item by either speech or gesture. Pairs were repeated if necessary. Morphological modifications of paraphasic distortions were accepted if it was clear which item was being referred to. A minimum interval of 7.5 seconds was imposed between successive pairs. This gave most subjects ample time to answer, through some, mainly from the left hemisphere lesion group, required longer on some items. Nonetheless, this procedure afforded better control over the amount of time spent on the task than if each subject had been allowed to proceed at his or her own pace. Both recall and recognition tasks on a given word type were completed before examining performance on another word type.

For each word type most of the words used were of one or two syllables, although a small number of three syllable words (about 2%) were also included. The recall tasks were balanced for word syllable length (30% one syllable, 70% two syllables) across the different word types. Recognition stimuli were also balanced for word syllable length (50% one syllable, 50% two syllables, with occasional three syllables) across the different word types but the recognition distractors contained a relatively higher proportion of mono-syllabic words than the stimuli for the high frequency word types and a relatively higher proportion of disyllabic words than the stimuli for the low frequency word types. These differences, however, were not sufficient to provide syllable length clues that could assist performance in the recognition tasks. In each of the two experiments using high frequency words (HF-HC and HF-LC) both recall and recognition tasks each contained approximately 50% A and 50% AA frequency words. In each of the two experiments using low frequency words (LF-HC and LF-LC) the mean word frequencies for recall lists, recognition stimuli, and recognition distractors were all between 19.0 and 23.0 occurrences/million.

Each subject required two sessions to complete these memory experiments. Performance on two of the word types was assessed in each session with a break of ten minutes between the two word types. A restricted design was employed which involved only four of the 24 possible different orders of presentation of the four word types over the two sessions, as it was felt that with the small numbers in the groups a random design may have led to order-of-presentation differences between groups. The four orders used were as follows:

Session 1		1	2	3	4
		HF-HC	HF-LC	LF-HC	LF-LC
Session 2	First	LF-HC	LF-LC	HF-HC	HF-LC
	Second	LF-LC	LF-HC	HF-LC	HF-HC

Thus, in any one session the two word types with the same word frequency but differing in word concreteness were used.

The two sessions needed to complete the memory tests were usually carried out on different days. In cases where the two sessions occurred on the same day they were separated by an interval of several hours. The word lists used in these memory tasks can be obtained from the author on request.

Comparisons between groups were made using Student's *t* test and, where appropriate, one-way analysis of covariance. Confidence levels of 5%, 2%, 1%, and 0.1% are used in discussing the results. Unless otherwise stated all confidence levels reported refer to two-tailed tests.

Results

Means and standard deviations for age and scores on the preliminary tests are presented in Table 1 for the control, RH, and LH groups, and for the left hemisphere temporal and non-temporal subgroups and for dysphasic and non-dysphasic subgroups.

The RH group did not differ significantly from the control group in age, current IQ nor, apart from impairment on the Modified Token Test ($P<0.05$), in performance on language tests. The LH group did not differ significantly from the control or RH groups in age or current IQ, but was significantly impaired compared with both of these groups on tasks of word retrieval—that is, Object Naming (controls v LH, $P<0.01$; RH v LH, $P<0.001$) and Description Naming (controls v LH, $P<0.001$; RH v LH, $P<0.001$), and on complex tasks of comprehension and expression—that is, the Modified Token Test (controls v LH, $P<0.001$; RH v LH, $P<0.01$) and the WAIS

Table 1 Means and standard deviations for age and preliminary test scores for each group

	Control (N=19)	RH (N=22)	LH (N=29)	LH temporal (N=19)	LH non-temporal (N=10)	LH dysphasic (N=20)	LH non-dysphasic (N=9)
Age (yr)	45.9 (10.9)	45.7 (13.1)	44.9 (11.3)	47.8 (10.1)	33.2 (11.6)	44.8 (10.8)	45.0 (12.8)
Current IQ	111.1 (12.0)	104.7 (15.1)	105.9 (16.9)	109.2 (15.5)	93.6 (18.5)	103.4 (16.5)	111.6 (17.5)
Phoneme Discrimination (Max=40; chance=20)	36.5 (2.2)	36.6 (2.4)	36.9 (1.7)	36.8 (1.4)	37.0 (2.2)	37.0 (1.7)	36.9 (1.2)
Articulation (Max=32)	31.9 (0.3)	31.7 (0.7)	31.3 (1.2)	31.1 (1.4)	31.7 (1.3)	31.1 (1.4)	31.9 (0.2)
Object Naming (Max=15)	14.8 (0.4)	14.7 (0.6)	11.6 (4.3)	11.2 (4.3)	12.2 (4.4)	10.2 (4.5)	14.6 (0.5)
Description Naming (Max=15)	14.5 (0.9)	14.0 (1.7)	10.6 (4.4)	9.7 (4.8)	12.3 (3.2)	9.1 (4.5)	14.0 (0.9)
Modified Peabody Test (Max=60; chance=15)	58.4 (1.6)	57.0 (4.9)	55.2 (5.3)	53.9 (6.1)	57.3 (1.6)	53.7 (5.8)	58.2 (1.1)
WAIS Vocabulary Subtest (Age scaled)	11.6 (2.2)	11.9 (2.4)	9.3 (2.6)	9.3 (2.9)	9.3 (2.0)	8.3 (2.1)	11.6 (2.1)
Modified Token Test (Max=15)	14.7 (0.7)	13.9 (1.5)	10.0 (5.0)	10.1 (5.4)	9.8 (4.5)	8.1 (5.0)	14.1 (0.8)

Vocabulary Subtest (controls v LH, $P<0.01$; RH v LH, $P<0.01$). The LH group was also significantly impaired compared to the control group on word meaning comprehension—that is, the Modified Peabody Test ($P<0.01$), and on the Articulation Test ($P<0.02$). However, despite the significant impairment shown by the LH group as a whole, only five LH patients scored below normal limits on the Modified Peabody Test (that is, scored less than 53 out of 60, see Coughlan and Warrington, 1978). As this task predominantly samples words with a Thorndike-Lorge (1944) frequency of less than 20/million (the average frequency of the low frequency words in the memory tests) it seems that semantic comprehension of the memory test items was adequate for the majority of the LH patients. On the Articulation Test the LH group achieved a near ceiling level mean score despite very strict scoring in which spontaneous corrections were not credited. Only four LH patients scored below normal limits on this task (that is, less than 31 out of 32; see Coughlan and Warrington, 1978), and even in these patients the articulatory deficits were mild and appeared insufficient to hinder substantially the production of the memory test items. Within the left hemisphere, temporal and non-temporal groups differed significantly in age ($P<0.05$) but not in current IQ or in performance on language tasks. There was a higher incidence of dysphasic patients in the non-temporal (90%) than in the temporal group (58%) but the latter tended to contain those patients with the relatively more severe dysphasic symptoms.

Considering LH subjects purely as dysphasic or non-dysphasic, irrespective of lesion site, it was found that the dysphasic group did not differ significantly from the non-dysphasic group in age, current IQ, or in performance on Articulation and Phoneme Discrimination Tests. Significant dysphasic group impairment was, however, found on the WAIS Vocabulary Subtest ($P<0.001$) and the Modified Peabody Test ($P<0.05$) and, naturally, on the Object Naming, Description Naming, and Modified Token Tests (all $P<0.01$), the three tests used in the designation of dysphasic patients.

VERBAL MEMORY TESTS

The mean recall and recognition scores of the control, RH, and LH groups, and of the left hemisphere temporal and non-temporal subgroups and dysphasic and non-dysphasic subgroups are shown in Table 2.

The performance of the RH group did not differ significantly from that of the control group on either recall or recognition but that of the LH group was very significantly impaired compared with the control group ($P<0.001$) on each task. Clear-cut recall and recognition deficits, defined as scores at least two standard deviations below the control group's mean scores (scores no better than 30 out of 120 on recall, and 95 out of 140 on recognition), were displayed by 15 (52%) of the LH group—11 displaying deficits on both recall and recognition, two on recall only, and two on recognition only. The association between recall and recognition deficits in the LH

Table 2 Overall recall and recognition performances: mean scores and standard deviations for each group

	Control (N=19)	RH (N=22)	LH (N=29)	LH temporal (N=19)	LH non-temporal (N=10)	LH dysphasic (N=20)	LH non-dysphasic (N=9)
Recall total (Max=120)	62.1 (11.9)	63.5 (15.3)	33.3 (18.4)	34.2 (18.1)	45.3 (17.0)	33.6 (19.6)	49.0 (8.9)
Recognition total (Max=140; chance=70)	116.5 (10.5)	113.3 (12.6)	99.9 (16.1)	95.5 (16.3)	105.2 (14.2)	95.5 (16.0)	107.4 (14.3)

group was strongly significant ($\chi^2=12.31$, $P<0.001$). Significant negative Pearson correlations ($P<0.05$, one-tailed tests) were found between age and recall in the control, RH, and LH groups and between age and recognition in the RH and LH groups. Correlations between current IQ and recall and recognition were very small and did not approach significance in any of the three groups.

VERBAL MEMORY AND LOCUS OF LESION WITHIN THE LEFT HEMISPHERE

Both LH temporal and LH non-temporal groups were significantly impaired on recall and recognition compared with the control group (control v LH temporal, recall $P<0.001$, recognition $P<0.001$; control v LH non-temporal, recall $P<0.01$, recognition $P<0.05$).

On inspection, the verbal memory deficits appeared greater in the LH temporal group than in the LH non-temporal group (Table 2) but comparisons between these groups failed to reach significance on either recall or recognition (recall, $df=27$, $t=1.78$, NS; recognition, $df=27$, $t=1.58$, NS). Further comparison between these groups incorporating covariance adjustments for age and degree of dysphasia (using performances on Object Naming, Description Naming, and the Modified Token Test as covariates) also failed to reveal significant differences in either recall or recognition (recall, $df=1, 23$, $F=0.13$, NS; recognition, $df=1, 23$, $F=0.00$, NS).

VERBAL MEMORY AND DYSPHASIA

The LH dysphasic group was very significantly impaired compared with the control group on both recall ($P<0.001$) and recognition ($P<0.001$); the LH non-dysphasic group was significantly impaired compared with controls on recall only ($P<0.01$). The LH dysphasic group performed at lower levels than the LH non-dysphasic group on recall and recognition, but only on recall was the difference between these two groups significant (recall, $df=27$, $t=2.24$, $P<0.05$; recognition, $df=27$, $t=1.77$, NS). The LH dysphasic and LH non-dysphasic groups were not, however, well matched

in terms of their lesion site compositions as the LH non-dysphasic group contained a much higher incidence of subjects with lesions involving the temporal lobe than did the LH dysphasic group. A clearer testament to the relationship between verbal memory impairment and dysphasia was obtained by comparison of groups of dysphasic and non-dysphasic subjects with a left hemisphere lesion which were more closely matched in terms of lesion site composition, namely groups in which all subjects exhibited lesions involving the temporal lobe, ie LH temporal-dysphasic ($N=11$) and LH temporal-non-dysphasic ($N=8$) groups. The LH temporal-dysphasic group was significantly impaired ($df=17$, $t=3.56$, $P<0.01$) compared with the LH temporal-non-dysphasic group on recall (mean scores 24.4 and 47.6 respectively) and ($df=17$, $t=2.62$, $P<0.02$) on recognition (mean scores 89.3 and 106.5 respectively). Similar comparisons between dysphasic and non-dysphasic groups with non-temporal left hemisphere lesions were not valid as only one of the nine subjects with such a lesion was non-dysphasic.

The relationship between verbal memory impairment and dysphasia was further attested by the findings of significant correlations in the LH group between performances on recall and recognition and those on a variety of language tasks (see Table 3). Articulation and Phoneme Discrimination performances, however, yielded only small correlations with recall and recognition which did not approach significance, indicating

Table 3 Correlation between preliminary test scores and recall and recognition total scores in the LH group

	Recall total	Recognition total
Phoneme Discrimination	0.11	0.00
Articulation	0.18	0.17
Object Naming	0.58§	0.38*
Description Naming	0.64§	0.51‡
Modified Peabody Test	0.48‡	0.51‡
WAIS Vocabulary Subtest (Age-scaled)	0.56‡	0.43‡
Modified Token Test	0.49‡	0.31

* $P<0.05$, † $P<0.02$, ‡ $P<0.01$, § $P<0.001$, (one-tailed tests).

that memory deficits among the LH patients were not the result of any impairments in these relatively peripheral language skills. Recall and recognition performances were themselves significantly correlated in the LH group (Pearson correlation coefficient=0.67, $P<0.001$, one-tailed test).

WORD FREQUENCY AND WORD CONCRETENESS EFFECTS

The recall and recognition performances described so far consisted of four separate recall and recognition tasks each of which employed a specific type of word, either high frequency-high concreteness (HF-HC), high frequency-low concreteness (HF-LC), low frequency-high concreteness (LF-HC) or low frequency-low concreteness (LF-LC) words. On recall tasks control and RH groups scored at similar levels on each type while the LH group performed at lower levels throughout. Both the LH non-dysphasic and the LH dysphasic groups also yielded lower mean scores than the control or RH groups throughout, those of the LH dysphasic group always being the lowest (Table 4). However, all five groups displayed the same order of mean scores, namely, HF-HC>LF-HC>HF-LC>LF-LC.

On recognition, the LH group and the LH dysphasic and LH non-dysphasic groups again all displayed lower mean scores than the control and RH groups on each type of word, those of the LH dysphasic group being the lowest throughout (Table 4). All five groups performed best on LF-HC words but the order of mean scores on the

other three types varied from group to group. These variations seemed attributable to the very small differences in mean levels of performance on each type of word that were found *within* each group, rather than the fundamental differences in pattern of performance between the groups.

The overall effects of word frequency and word concreteness on recall and recognition in the control, RH, and LH groups and in the LH subgroups were examined by summing over concreteness and frequency conditions respectively. Inspection of these summed scores showed that in recall, all groups performed better on high frequency words (HF-HC+HF-LC) than on low frequency words (LF-HC+LF-LC), and better on high concreteness words (HF-HC+LF-HC) than on low concreteness words (HF-LC+LF-LC). In recognition, all groups performed better on low frequency words than on high frequency words and better on high concreteness words than on low concreteness words. These findings are consistent with those previously reported for normal experimental subjects (Paivio, 1971).

ERROR ANALYSIS

An analysis of the incorrect responses made on all the recall tasks was carried out for the control, RH, and LH groups and for the LH dysphasic and LH non-dysphasic groups. Inspection of the data showed that the mean total error scores of control, RH, and LH non-dysphasic groups were similar (11.1, 10.5 and 10.8 respectively), but that that of the LH dysphasic group was much higher

Table 4 *Word frequency and word concreteness effects: means and standard deviations for each group on recall and recognition*

	Control (N=19)	RH (N=22)	LH (N=29)	LH dysphasic (N=20)	LH non-dysphasic (N=9)
<i>Recall</i>					
HF-FC	18.6 (4.2)	16.7 (3.9)	11.3 (6.0)	10.1 (6.0)	13.9 (4.9)
HF-LC	14.1 (3.5)	14.0 (4.4)	8.8 (4.8)	7.3 (4.4)	12.1 (3.8)
LF-HC	17.9 (4.3)	16.4 (4.6)	10.2 (5.7)	9.1 (6.1)	12.7 (3.4)
LF-LC	11.5 (3.3)	13.4 (4.1)	8.1 (4.6)	7.1 (5.0)	10.3 (2.1)
(Max=30 for each type)					
<i>Recognition</i>					
HF-HC	27.9 (3.8)	28.3 (4.0)	24.1 (4.2)	23.3 (4.3)	26.1 (2.8)
HF-LC	28.1 (3.3)	28.3 (4.2)	24.8 (4.6)	23.9 (4.7)	26.8 (3.5)
LF-HC	31.6 (2.6)	28.9 (3.6)	26.6 (5.4)	25.5 (5.1)	28.9 (5.2)
LF-LC	28.8 (3.7)	27.9 (4.3)	24.4 (4.9)	23.9 (4.9)	25.7 (4.5)
(Max=35; chance=17.5 for each type)					

(19.1). Errors that appeared to be either random words or words semantically related to those in the list being recalled at the time were the most common in each group and their proportion of the total errors in each group amounted to 57% for the control group, 64% for the RH group, 75% for the LH non-dysphasic group, and 79% for the LH dysphasic group. Intrusions from previous lists were the second most common type of error in each group, their proportion of the total errors in each group amounting to 37% for the control group, 34% for the RH group, 21% for the LH non-dysphasic group, and 13% for the LH dysphasic group. Derivative errors—that is, morphological variants of list items—ranged between 6% (control group) and 2% (RH group) of each group's total error score. Neologistic and paraphasic errors occurred only in the LH dysphasic group and accounted for 5% of this group's total errors.

Discussion

This investigation has been concerned with the effects of localised left cerebral hemisphere lesions on verbal memory and with the relationship between verbal memory and dysphasia. The effects of localisation of a lesion within the left hemisphere will be discussed first. In order to investigate such effects the subjects with a left hemisphere lesion were classified into mutually exclusive temporal and non-temporal lesion groups according to whether or not their lesions displayed any temporal lobe involvement. Both groups displayed significant impairments of recall and recognition verbal memory, and although on inspection these impairments appeared greater for the temporal group than the non-temporal group, the differences between these groups did not reach significance. Such findings would seem more in accord with those of Newcombe (1969), who found little relationship between locus of lesion and verbal memory deficits, than with those of Milner (1964, 1969), who reported a specific intrahemispheric relationship between verbal memory impairment and temporal lobe damage. However, both the present study and that of Newcombe (1969) included dysphasic subjects and it is possible that underlying effects of the locus of the lesion were obscured by the deleterious effect of dysphasia on verbal memory performance. Milner's 1964 study excluded dysphasic subjects, though it is not clear whether this was so in her 1969 study. Accordingly, further comparisons of the temporal and non-temporal groups' verbal memory performances

in the present study were carried out taking into account the language abilities of the subjects in each group, but again no significant differences emerged on either recall or recognition. These findings, based on an unselected series of patients with left hemisphere lesions, therefore, fail to support the notion of a specific anatomical correlate of verbal memory impairment within the left hemisphere.

In the investigation of the relationship between verbal memory and dysphasia, the subjects with left hemisphere lesions were classified into mutually exclusive dysphasic and non-dysphasic groups, irrespective of the site of the lesion within the left hemisphere. The dysphasic group displayed significant impairment on both recall and recognition and the non-dysphasic group displayed significant impairment on recall. The dysphasic group performed at lower levels than the non-dysphasic group on both recall and recognition, but differences in the composition of the lesion sites between the two groups (the non-dysphasic group contained a higher proportion of subjects with lesions involving a temporal lobe than did the dysphasic group) undoubtedly obscured the true extent of the deleterious effects of dysphasia on verbal memory. When the performances of dysphasic and non-dysphasic subjects with lesions involving only the temporal lobe were compared—that is, groups more closely matched in terms of lesion site compositions—the dysphasic subjects were found to be significantly inferior on both recall and recognition.

In considering the possible origins of the verbal memory deficits associated with dysphasia it may be noted that such deficits were not attributable to a general intellectual deterioration among the dysphasic population. The dysphasic group was not significantly inferior in current IQ to the control group, and correlations between current IQ and recall and recognition were very small and not significant among the patients with left hemisphere lesions. It is also implausible that the verbal memory deficits of dysphasic subjects were merely the consequence of larger lesions. Most left hemisphere lesions were progressive tumours and it is unlikely that such lesions reached a greater size before detection in dysphasic than in non-dysphasic subjects. It would, therefore, appear that the dysphasia itself is at least a contributory factor to the impaired verbal memory performance of dysphasic subjects. It is thus relevant to ask whether this impaired performance arises chiefly from difficulties in the comprehension and/or expression of the test materials, or whether it reflects a more fundamental disruption of verbal memory

skills. That it is not entirely attributable to impoverished expressive abilities may be inferred from the finding that deficits occur concurrently on both recall and recognition tasks even though recognition makes only minimal demands on expressive skills. However, since expressive disorders may exaggerate existing recall deficits it is appropriate to assess their possible contribution.

Impaired verbal memory performance was clearly not attributable to language impairments at the relatively peripheral receptive and expressive levels of phoneme discrimination and articulation as these skills were either intact or only minimally impaired among the dysphasic subjects, and were not significantly correlated with recall or recognition performance. Substantial language deficits occurred among the dysphasic subjects at the more central receptive and expressive levels of comprehension of word meaning and word retrieval, and significant correlations between performances on these skills and on verbal memory tasks indicated that such deficits may have influenced verbal memory performance. Even so, it is not clear that any such influence arose directly from an impairment in the comprehension or expression of the test items. Indeed, two observations suggest that it did not. First, comprehension of word meaning, although reduced among dysphasic subjects, appeared in most cases to be adequate for the words used in the verbal memory tasks. Secondly, the relative difficulty in recall of low frequency words as compared with high frequency words was no greater for dysphasic than for control subjects (mean recall scores; controls, high frequency words 32.7/60, low frequency words 29.4/60; LH dysphasic group, high frequency words 17.4/60, low frequency words 16.2/60); as low frequency words are more vulnerable to impairments of comprehension of word meaning and word retrieval than are high frequency words (Schuell *et al.*, 1961; Newcombe *et al.*, 1965; Rochford and Williams, 1965), it might be expected that, if direct impairment of the comprehension and/or expression of the words used in the recall tasks took place, dysphasic subjects would show an exaggerated difficulty in recall of low frequency words compared with high frequency words, but this was clearly not the case. It, therefore, seems likely that the language deficits of the dysphasic subjects in this study gave rise to verbal memory impairments in a more subtle way than by direct impairment of the comprehension and/or expression of test materials.

Further examination of word frequency and word concreteness effects in recall and recognition

revealed that dysphasic subjects displayed the same pattern of sensitivity to these parameters, though at lower levels of performance, as control or non-dysphasic brain-damaged subjects, suggesting that dysphasic patients retain the qualitative characteristics of normal verbal memory but operate at a reduced level of efficiency. A similar conclusion, after a study of the serial learning abilities of dysphasic patients, was also drawn by Tikofsky (1971) who further suggested that impaired verbal memory among dysphasics may derive from an increased susceptibility to interference effects. This would not appear to have been the case in the present study as, despite yielding greater numbers of incorrect responses on recall tasks than did the control subjects, the dysphasic subjects yielded fewer numbers of intrusions from prior lists (ie interference) errors. A more plausible explanation of the present findings would seem to be that as a result of central language disturbances dysphasic subjects have difficulty in the use or development of language based procedures for the storage, organisation, or retrieval of information in verbal memory—that is, that they are unable to employ effectively such verbal processes as are necessary to maintain an efficient verbal memory. Such a broad hypothesis may conceal a variety of discrete interactions between specific language deficits and verbal memory processes but more refined speculation is not appropriate when considering data derived from an unselected series of dysphasic patients. The question of whether or not specific types of dysphasia give rise to corresponding specific types of verbal memory disorder clearly warrants further investigation, but at present it seems appropriate to conclude that an impairment in central language skills frequently leads to a reduced ability to operate verbal memory efficiently, over and above any difficulty which may arise in the comprehension and/or expression of materials to be remembered.

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