Clinical Aphasiology, Vol. 22, 1994, pp. 53–65

Verbal Learning with the Right Hemisphere

Pelagie M. Beeson, Steven Z. Rapcsak,
Alan B. Rubens, and Kathryn A. Bayles

It is well documented that individuals with aphasia frequently exhibit verbal memory impairment (Albert, 1976; Butters, Samuels, Goodglass, & Brody, 1970; Goodglass, Gleason, & Hyde, 1970; Heilman, Scholes, & Watson, 1976). Short-term memory impairment, or reduced immediate serial recall, has been associated with all aphasia types (except the transcortical aphasias) and with lesions throughout the distribution of the left middle cerebral artery (DeRenzi & Nichelli, 1975; Gordon, 1983). A selective impairment of short-term verbal memory has been associated with lesions localized to the posterior parietal region (Shallice & Warrington, 1970; Warrington, Logue, & Pratt, 1971). Alternatively, deficits in verbal learning and long-term verbal recall have been associated with anterior lesions, particularly in the dorsolateral region of the left frontal lobe (Beeson, Bayles, Rubens, & Kaszniak, in press; Risse, Rubens, & Jordan, 1984). In the case of left hemisphere lesions that significantly damage the anterior ***and*** posterior regions served by the middle cerebral artery, impairment of both span memory and verbal learning would be expected.

In the process of testing 46 individuals with chronic aphasia as part of a larger study on memory impairment after stroke (Beeson, 1991), it was noted that two individuals with massive left hemisphere lesions performed surprisingly well on tests of verbal learning and long-term verbal memory. We report here on those two cases in relation to group data from Beeson (1991) and Beeson et al. (in press) that included normal control subjects and individuals with aphasia due to lesions confined to anterior or posterior brain regions.

METHODS

Subjects

Case 1. Subject GK was a 60-year-old male, 12.5 years post stroke at the time of testing. This previously right-handed man had undergone eleven years of formal education, had received a high school graduate equivalency diploma (GED), and had worked as a machinist and truck driver. Prior to his stroke, GK had no significant neurological history. His intelligence quotient estimated from demographic variables was 101.4 (Barona, Reynolds, & Chastain, 1984). There was no history of familial left-handedness.

Following a massive left hemisphere stroke at age 47, GK exhibited right hemiparesis, right hemisensory loss, right hemianopia, and global aphasia. His aphasia evolved to a Broca's type, and administration of the *Western Aphasia Battery* (WAB) (Kertesz, 1982; Shewan & Kertesz, 1980) yielded an Aphasia Quotient (AQ) of 69.5. GK's nonfluent, agrammatic speech consisted primarily of single words and short phrases of high communicative value.

Postacute CT and MRI scans revealed virtually complete destruction of GK's left hemisphere, including all regions served by the anterior, middle, and posterior cerebral arteries. A T_2-weighted MRI scan obtained close to the time of testing is displayed in Figure 1. The small islands of cortical tissue in the left hemisphere that are evident on the scan appear to be completely undercut and isolated and are of questionable viability.[1]

Case 2. Subject VH was a 73-year-old male, 5.5 years post stroke at the time of testing. He was previously right-handed, had 18 years of education, and was a retired U.S. Air Force colonel. He experienced a massive left hemisphere stroke at age 68 resulting in right hemiparesis, right visual field defect, and severe aphasia. During the 10 months preceding the stroke, VH had experienced intermittent right paresthesia without concomitant language difficulty. He has no known familial history of left-handedness other than one of his children, whose left-handedness was considered to be of maternal origin because of a documented history of left-handedness in his wife's family.

Administration of the WAB yielded an AQ of 60.7, with a profile of Broca's aphasia. VH's spontaneous speech was similar to GK in that it was nonfluent and agrammatic, but it yielded fairly successful communication with an active listener.

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1. The extent of left hemisphere damage experienced by GK aroused our interest with regard to many aspects of performance, and accounts of GK's reading, writing, and praxis appear elsewhere (Rapcsak, Beeson, & Rubens, 1991a, 1991b; Rapcsak, Ochipa, Beeson, & Rubens, in press).

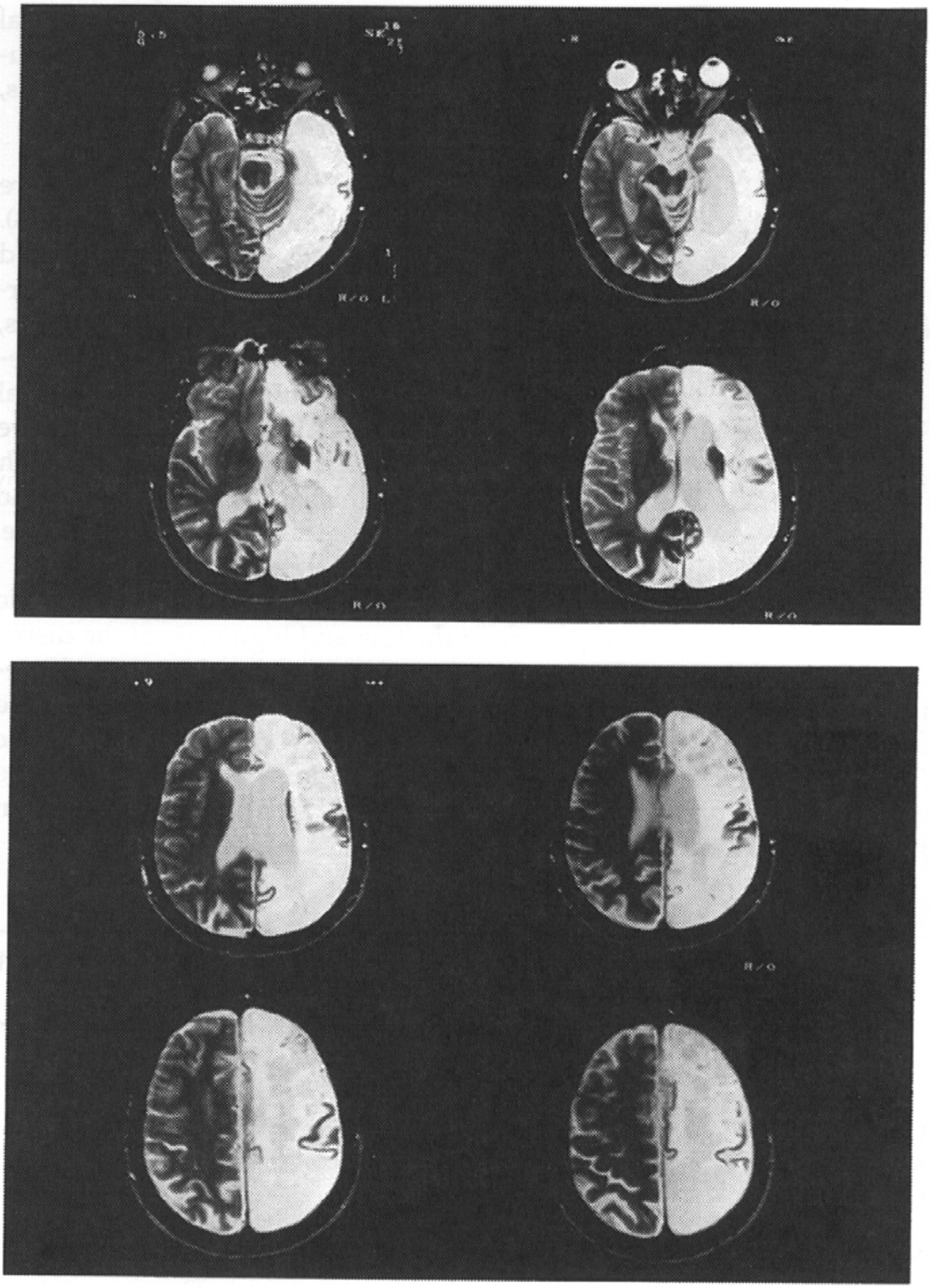

Figure 1. T_2-weighted MRI head scan of Subject GK demonstrating virtually complete destruction of the left hemisphere.

A recent CT head scan revealed an extensive left hemisphere lesion embracing essentially the entire distribution of the left middle cerebral artery, including the lenticulostriate branches, and most of the distribution of the anterior cerebral artery (Figure 2). Other than the left thalamus, the regions served by the left posterior cerebral artery were spared.

Group Data. The verbal memory performances of GK and VH were compared with group data from Beeson (1991) and Beeson et al. (1993). The subject groups comprised fourteen individuals who had experienced a single left hemisphere stroke resulting in persistent aphasia and fourteen demographically matched control subjects. Of the stroke patients, seven individuals had lesions of the frontal lobe (referred to as the anterior group) and seven individuals had lesions of the temporoparietal region (referred to as the posterior group). All subjects were native speakers of English and passed screening tests for vision and speech discrimination. The stroke patients were right-handed individuals who were neurologically normal prior to a single clinically documented stroke. All were at least six months post stroke at the time of testing.

GK and VH fell within the range of the group data for the demographic variables. However, they represented the low and high ends of the distribution, respectively, with regard to age, education, and estimated premorbid intelligence (Table 1). Whereas VH fell within the range of time post stroke for the group data, GK was tested at a later postonset time than any other subject. Both GK and VH obtained WAB AQ that were lower than the group means but still within one standard deviation from the mean of the anterior group.

Table 1. Subject Characteristics

Group	*Age (years)*	*Education (years)*	*IQ (est.)*	*AQ (WAB)*	*Post CVA (years)*
Subject GK	60	11	101.4	69.5	12.5
Subject VH	73	18	122.4	60.7	5.5
Anterior CVA (N = 7)[a]	63.5(7.0)	11.6(1.8)	105.1(6.7)	74.8(16.7)	4.8(5.9)
Posterior CVA (N = 7)[a]	67.0(7.2)	13.5(4.5)	108.6(11.8)	80.1(12.9)	2.5(2.7)
M	67.0	13.5	108.6	80.1	2.5
(SD)	(7.2)	(4.5)	(11.8)	(12.9)	(2.7)
Control (N = 14)[a]	68.7(8.6)	15.3(3.1)	113.4(5.9)	N/A	N/A

Note: AQ = Aphasia Quotient; WAB = Western Aphasia Battery.
[a]Data for anterior CVA, posterior CVA, and control groups are given as mean (standard deviation).

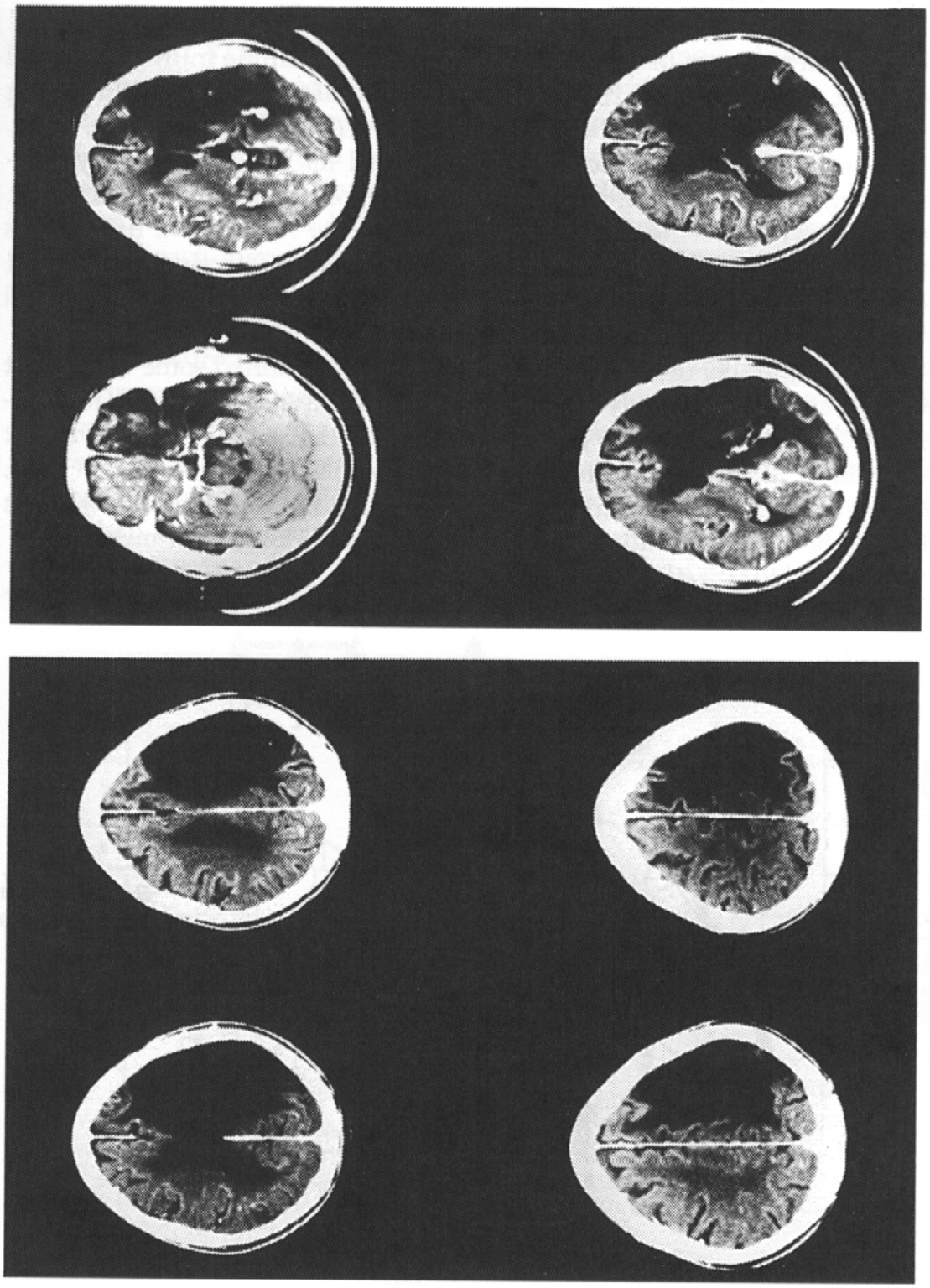

Figure 2. CT head scan of Subject VH demonstrating massive left hemisphere lesion affecting the entire distribution of the left middle cerebral artery and most of the distribution of the left anterior cerebral artery.

PROCEDURE AND RESULTS

Two verbal memory tests were administered to examine multitrial verbal learning and recall. One test was a replication of the selective reminding test (SRT) used by Risse, Rubens, and Jordan (1984) (after Buschke, 1973), which consisted of free recall of a nine-word list. High-frequency, concrete nouns selected from three semantic categories were presented orally. Immediately following list presentation, subjects were asked to recall as many words as possible, in any order. Subjects were selectively reminded of omitted words on 10 recall trials. GK was given a delayed recall trial (Trial 11) following a filled 15-minute delay.

The total number of words recalled on each trial included some words that were recalled immediately following list presentation or selective reminding (i.e., short-term memory) and some words that were recalled without reminding. According to the convention established by Buschke (1973), words that were recalled on two successive trials (i.e., recalled once without reminding) were thereafter considered recall from long-term memory.

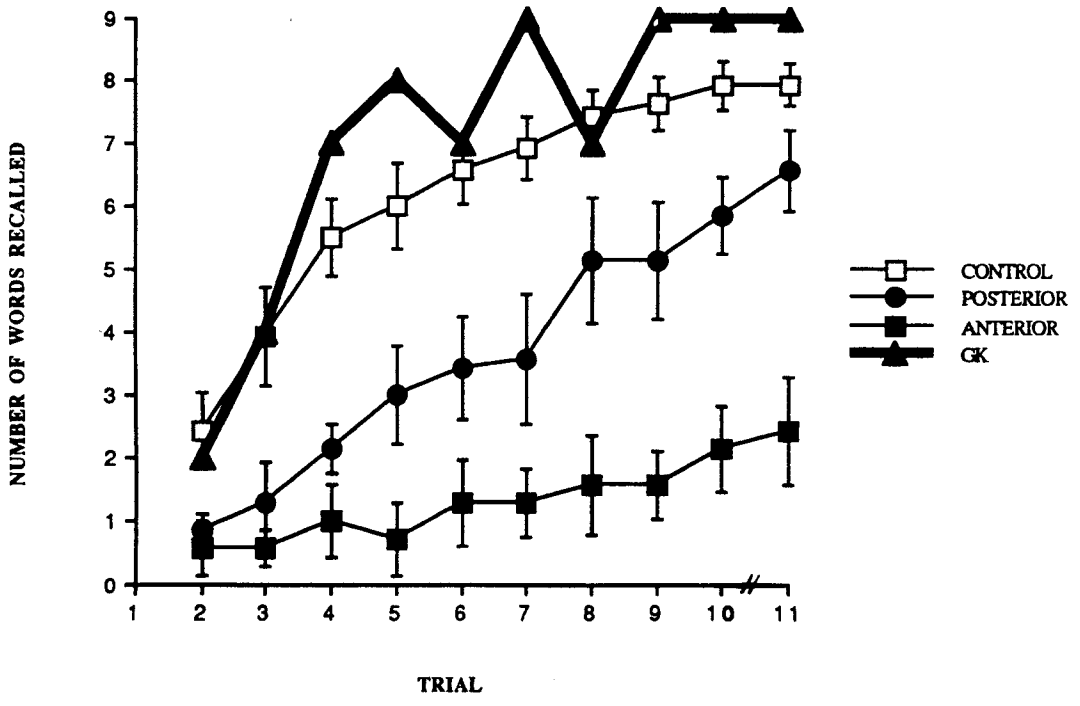

Figure 3. Recall from long-term memory on nine-word selective reminding test. (Subject GK's performance is shown along with the mean performance of control group and individuals with aphasia grouped by lesion location. Note that a 15-min delay was imposed between Trials 10 and 11. Anterior = frontal lesions; posterior = temporoparietal lesions. Vertical bars represent the standard error of the group mean.)

Long-term memory recall is depicted in Figures 3 and 4 for GK and VH, respectively, in relation to the anterior, posterior, and control groups. Both GK and VH exhibited verbal learning that most closely approximated the performance of the monaphasic control subjects.

The second verbal memory test required free recall and cued recall of an 18-word list presented using a guided semantic encoding task modeled after Grober and Buschke (1987). Labeled pictures were initially presented in groups of three for an auditory comprehension task that associated each item with its semantic category. For example, when presented with labeled pictures of a *trumpet*, a *foot*, and a *horse*, subjects had to point and say *"trumpet"* when asked *"Which one is the musical instrument?"* The stimuli were removed after all three items were identified, and an immediate cued recall trial was given wherein subjects were asked to recall the items in response to their semantic category (e.g., *"Which one was the musical instrument?"*). All subjects successfully performed the comprehension task, and the immediate recall task was accomplished with no more than three opportunities given. Thus, it was documented that all subjects comprehended the list items, were capable of producing the item names, and had associated each item with its semantic category.

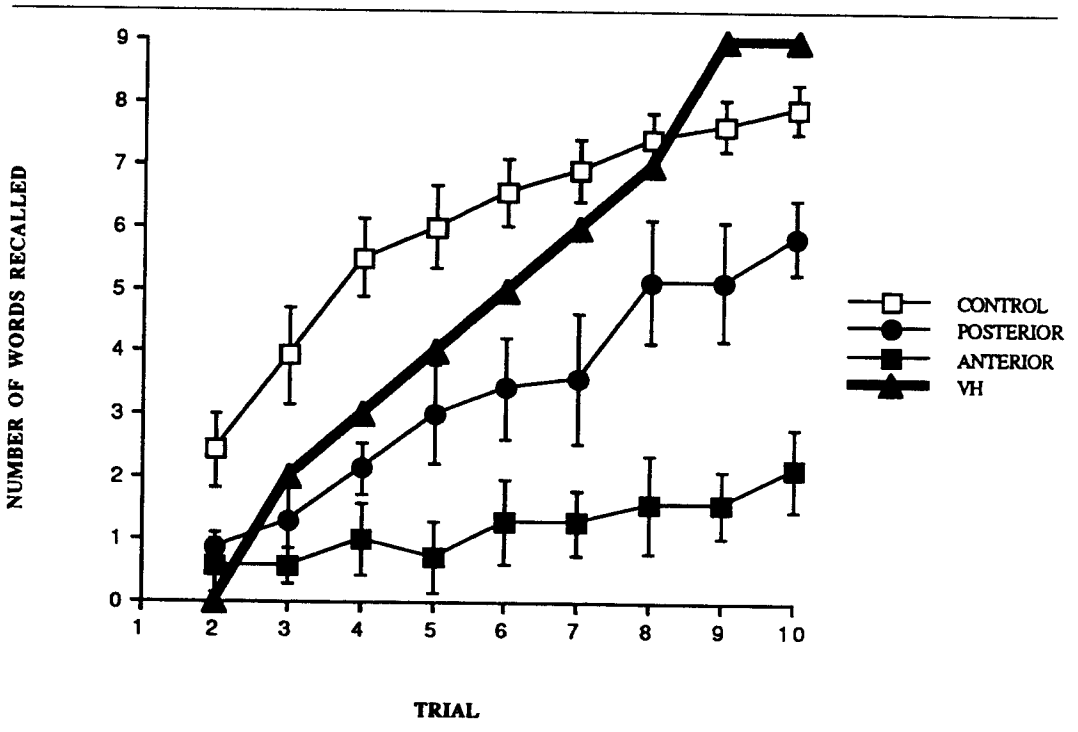

Figure 4. Recall from long-term memory on nine-word selective reminding test. (Subject VH's performance is shown along with the mean performance of control group and individuals with aphasia grouped by lesion location. Anterior = frontal lesions; posterior = temporoparietal lesions. Vertical bars represent the standard error of the group mean.)

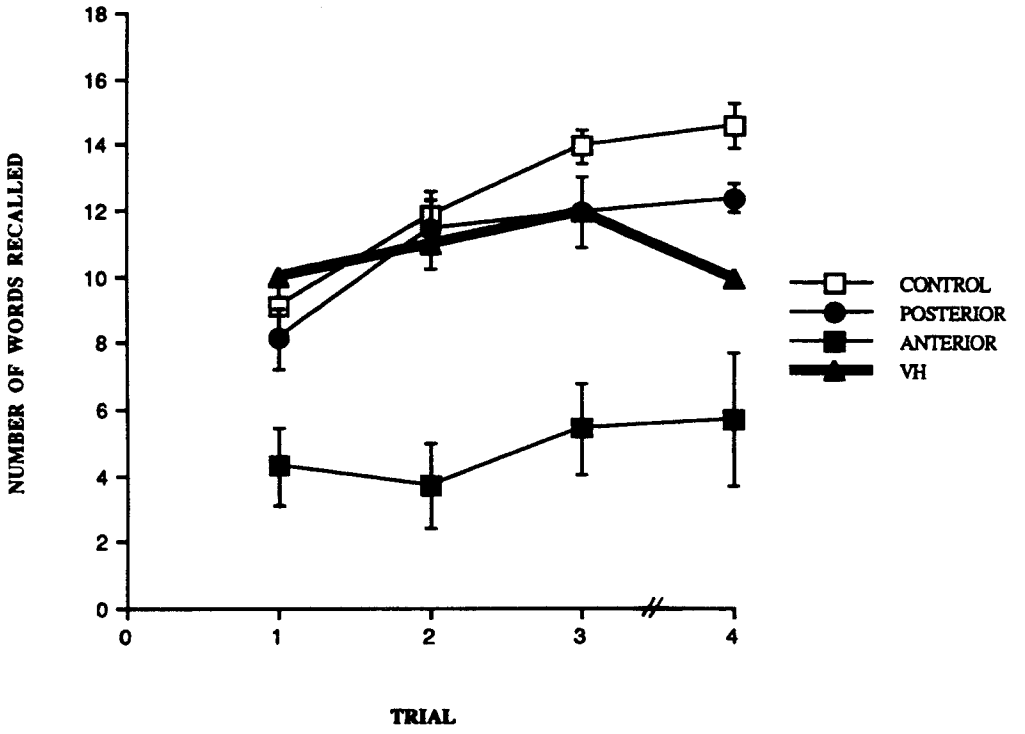

Figure 5. Free recall of 18-word list initially presented with guided semantic encoding procedure. (Subject GK's performance is shown along with the mean performance of control group and individuals with aphasia grouped by lesion location. Note that a 15-min delay was imposed between Trials 3 and 4. Anterior = frontal lesions; posterior = temporoparietal lesions. Vertical bars represent the standard error of the group mean.)

After all 18 list items were presented and a 30-second distraction task was performed, three free recall trials were presented. Each free recall trial was followed by semantically cued recall for unrecalled items and another 30-second distraction task to eliminate sustained subvocal rehearsal of list items. A delayed recall trial was administered following a 15-minute filled interval.

Figures 5 and 6 display free recall on the verbal-learning test with guided semantic encoding for GK and VH, respectively, in relation to the mean group performance. Their performance approximated the posterior and control groups on the initial recall trials and continued to approximate the performance of the posterior group over the repeated recall trials. Free recall by GK and VH was notably superior to the anterior aphasia group. Cued recall performance was high for all groups. The total number of words recalled by free recall plus cued recall was essentially undifferentiated for GK, VH, the posterior group, and the control group.

Short-term verbal memory was tested using the Digits Forward subtest from the *Wechsler Memory Scale–Revised* (WMS–R) (Wechsler, 1988). GK

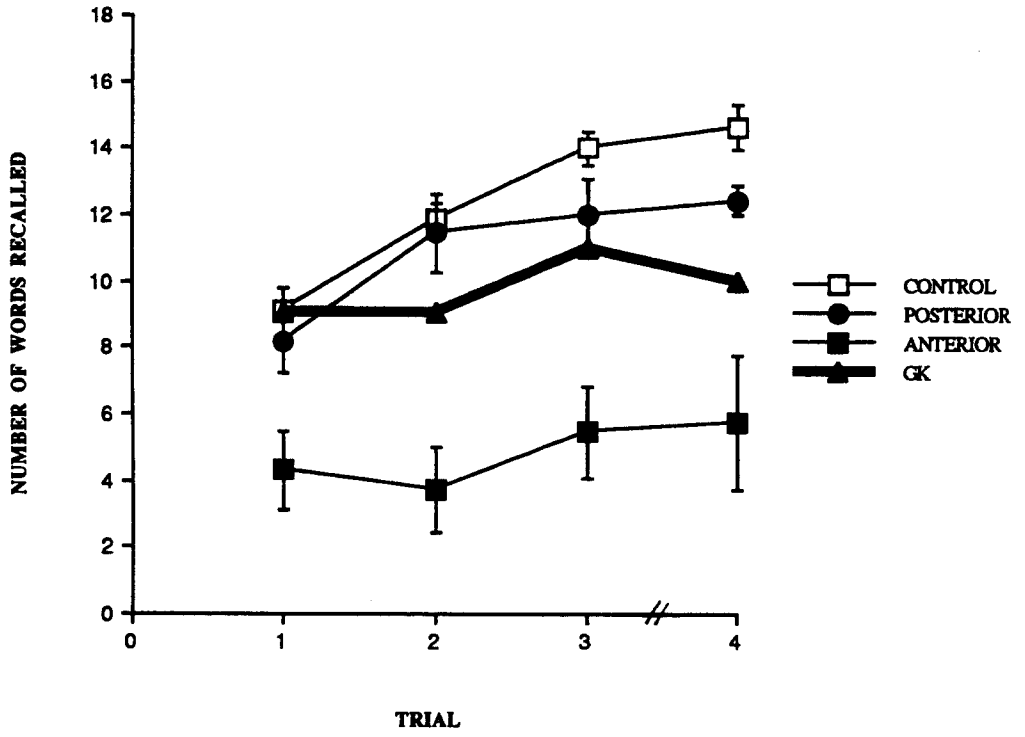

Figure 6. Free recall of 18-word list initially presented with guided semantic encoding procedure. (Subject VH's performance is shown along with the mean performance of control group and individuals with aphasia grouped by lesion location. Note that a 15-min delay was imposed between Trials 3 and 4. Anterior = frontal lesions; posterior = temporoparietal lesions. Vertical bars represent the standard error of the group mean.)

and VH each received raw scores of 3 for digit span, indicating spans of about three digits. The mean raw scores for the anterior (M = 4.7, SD = 2.4), posterior (M = 2.3, SD = 1.4), and control groups (M = 7.9, SD = 1.4) represented digit spans of approximately four to five digits, three digits, and six digits, respectively (Figure 7).

Visual memory span was assessed using the Tapping Forward subtest from the WMS–R (Figure 7). GK and VH exhibited visual memory spans of approximately 4–5 (raw scores = 6 and 7, respectively); the anterior, posterior, and control groups exhibited visual memory spans of 5–6 (raw score Ms = 8.3, 7.7, 8.5, respectively).

DISCUSSION

The relatively unimpaired, or mildly impaired, performance of GK and VH on tests of verbal learning and recall was quite surprising, consider-

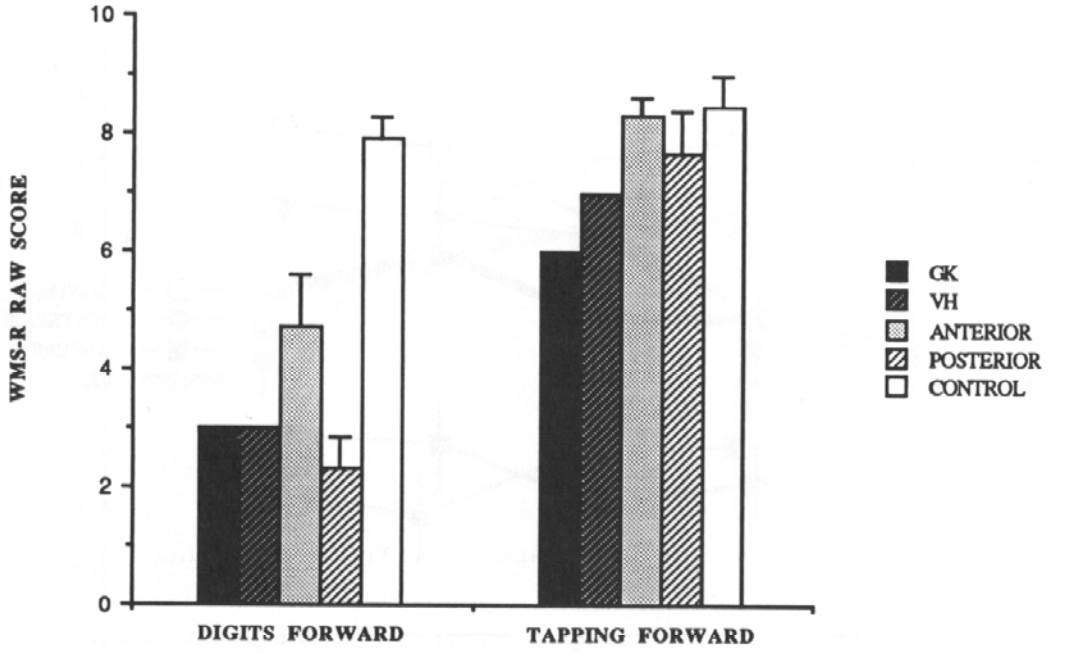

Figure 7. Raw scores from *Wechsler Memory Scale–Revised* Digits Forward and Tapping Forward subtests for Subjects GK and VH along with the mean performance of control group and individuals with aphasia grouped by lesion location. (Anterior = frontal lesions; posterior = temporoparietal lesions. Vertical bars represent the standard error of the group mean.)

ing the massive left hemisphere damage each had experienced. GK and VH consistently performed in a superior fashion when compared to the verbal learning of aphasic individuals with smaller lesions confined to the left frontal region. Their performance was either better than or similar to the mean performance of individuals with temporoparietal lesions. The findings could not be accounted for by aphasia severity because the AQs for GK and VH were lower than the mean AQs for the comparison groups. Furthermore, verbal-learning performance was independent of digit span, which was significantly limited for GK and VH, illustrating the dissociation of short- and long-term verbal memory noted by others (Risse, Rubens, & Jordan, 1984; Shallice & Warrington, 1970).

In view of the extent of the left hemisphere lesions in GK and VH, it is reasonable to conclude that their right hemispheres were responsible for accomplishing the verbal memory tasks. In the case of GK, the virtually complete destruction of his left hemisphere scarcely leaves an alternative explanation. A right hemisphere explanation is also favored for VH, rather than attributing his language and verbal memory abilities to the remaining tissue of the left frontal and occipital poles.

The linguistic demands of the verbal-learning tasks used in this study were relatively well suited to right hemisphere processing. The stimuli

were highly imageable concrete nouns that are optimal for the lexical-semantic system of the right hemisphere, as revealed in studies of split-brain and left hemispherectomized patients (Joanette, Goulet, & Hannequin, 1990; Zaidel, 1985). A digit span of about 3 ± 1 is also consistent with previously reported right hemisphere capacity (Zaidel, 1985).

We hypothesize a shift to right hemisphere processing may be explained as a release of the right hemisphere from left hemisphere inhibition, thus allowing it to manifest its competence for verbal learning. Landis and colleagues similarly reported evidence of a release of right hemisphere linguistic processing with large left hemisphere lesions (Landis, Regard, & Serrat, 1980; Landis, Regard, Graves, & Goodglass, 1983). They claimed a correlation between left hemisphere lesion size and the release of the right hemisphere for verbal processing. We infer from our findings that the tenacity of the partially damaged left hemisphere in maintaining its role as the verbal processor may impede the right hemisphere's assumption of certain language-related functions.

It is important to acknowledge that large individual differences have been noted in the literature with regard to the verbal capacity of the right hemisphere (Gazzaniga, 1983; Sidtis, Volpe, Wilson, Rayport, & Gazzaniga, 1981; Zaidel, 1985). Clearly, GK and VH represent particularly well developed right hemisphere verbal ability, and all individuals with extensive left hemisphere lesions may not benefit from the shift of verbal processing to the right hemisphere to the extent that GK and VH did. Nevertheless, these two subjects demonstrate that in some cases, the right hemisphere is capable of accomplishing verbal learning with considerable competence.

ACKNOWLEDGMENTS

Preparation of this paper was supported in part by the National Institute of Deafness and Other Communication Disorders grant DC-01409 to the Institute for Neurogenic Communication Disorders at the University of Arizona.

The authors would like to acknowledge the cooperation of the Tucson Veteran's Administration Medical Center, Dr. Enrique Labadie of the Department of Neurology, in particular, and the Rehab Institute of Tucson. We also express our thanks to GK, VH, and Mrs. H for their cheerful participation in this research effort.

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